DELHI PSYCHIATRY JOURNAL

(Official Publication of Delhi Psychiatric Society)

Vol. 17 No. 1 April, 2014

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Mental Health of Mental Health Professionals – need to care

M.S. Bhatia
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Medicine is a stressful profession. Some of its branches, including Psychiatry is much more stressful[1]. The research has shown that psychiatrists score high in neuroticism and reported greater work-related emotional exhaustion and depression.[2,3]

In psychiatry, the stress is contributed by the need to rescue the patient, a sense of failure and frustration when the patient's illness progress or does not respond, feelings of powerlessness against illness and its associated losses, grief, fear of becoming ill oneself or a desire to avoid patients to escape these feelings. The emotions are likely to be intensified from personal type of relationship with patient.There is external stress due to rapidly changing ways of service delivery, widening gap between the training and way of practice and increasing complex administrative and legal frame-works. The main stressors in psychiatric practice are overworking, demands of job interfering in family and social life, 24-hour responsibility for suicidal and homicidal patients, day on call,dealing with difficult and hostile relatives, relationship problems with other staff, performance-related stress, organizational problems, inadequate resources and threats to self-esteem.[4-6]

The outcome of chronic exposure to emotional and interpersonal stressors and external stressors lead to *burnout*[1], which is defined as, "an exposure of physical, emotional and mental exhaustion caused by long-term involvement in situations that are emotionally demanding". It is characterized by signs of emotional exhaustion (tiredness, somatic symptoms, decreased emotional resources and a feeling that one has nothing left to give to others), depersonalization (developing negative cynical attitudes and impersonal feelings towards others and treating them as objects) and lack of feelings of impersonal accomplishment (feeling of incompetence, inefficaciousness and inadequacy). It has a negative effect on patient care and individual's health. Negative changes in attitudes (reduced work goals, loss of idealism, heightened self-interest, increasing emotional detachment from clients, negative characteristics of patients, administrative hassles, resource deficits and staff conflicts, long hours and lack of positive feedback are also contributory factors.[7-9] "*Compassion fatigue"*(comparing with battle fatigue in war combatants) refers to the stressor that can affect clinicians who care for victims of severe trauma[10]. Empathy is a double-edged sword. It can precipitate conflicts in a mental health professional. Psychiatrists are prone to stress, burnout and suicide.[10] Even adjustment problems and divorce are reported to be common. Junior doctors report even high burnout than senior consultants.[11]

The burnout in psychiatrists is due to fear of violence, limited resources, crowded inpatient wards, and increasing culture of blame, high work demand without adequate resources, poorly defined role of consultants, responsibility without authority, inability to effect systemic change, conflict between responsibility towards employers versus towards its patients, isolation of consultants in community mental health care team, litigation threats and lack of feedback.[1,7-11] There is an inverse relationship between stress and job satisfaction in rehabilitation workers.[12]

Negative coping strategies are worrying, sleep problems, angry, irritable, blaming self, driving themselves harder in career, smoking, taking alcohol, bad driving, emotional symptoms (anxiety, fatigue, low mood, headaches, malaise) whereas positive coping strategies are support from partner, media entertainment, socializing, hobbies and exercise.[3] Exercise is specially helpful in coping with stress and preventing life style disorders. In a study,[3] a majority considered change in job, change in specialty, early retirement, leaving medicine, planning suicide and using self- prescribed medication. Junior staff used more coping devices whereas women used more negative coping.

There are no studies on the effectiveness of specific stress management techniques for mental health professionals. Relaxation training, emotional self-management, mastery (means use or overuse of one's usual methods of control of problematic situations) and distancing (means escape or an outlet, including meditation or sports) techniques and attitude adjustment programs are useful.[13,14] Intervention studies for burnout of mental health professionals are lacking. There is need to identify at risk professionals and a systemic review of stress, burnout and coping. Social skills training, stress management, social support and time management and organisational support are useful.[15] Support from family, friends and colleagues may be effective in handling burnout. If one develops a problem, it is ideal to seek treatment from a professional colleague rather than resorting to self-medication.

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Review Article

Depression and Periodontal Diseases

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Introduction

Periodontal disease is an immune-inflammatory response of tooth supporting structures which if left untreated will result in loss of tooth. Bacteria and their toxins are known to be etiological agents of periodontal disease but their presence itself is not capable of producing advance tissue destruction in all individuals. This means that there is an individual response and adaptation ability to have a certain amount of bacterial plaque with, a little or no progression of the disease. Onset of periodontal disease depends upon various factors like poor oral hygiene, smoking and systemic diseases such as diabetes mellitus, cardiovascular diseases which act together to create favorable conditions for the development of the disease in the individual.[1] In view of such concerns, it is important to identify risk factors that influence the development and progression of the periodontal disease.

Depression and its mechanism affecting periodontal health

Depression refers to a symptom as well as a cluster of syndromes.As a symptom, depression is generally equated with sadness, a feeling state that is protean in its manifestation, ranging from mild, transient disappointment to severe, confusing, enduring melancholy.

Depression is generally viewed as a subjective experience; an objective observer is likely to notice dysfunction in one or more of the following areas:

- **Thinking:** Poor concentration, worthlessness, hopelessness, and sometimes delusions and hallucinations
- **Behavior:** Crying, psychomotor retardation or agitation and suicidality
- **Physical functioning:** Alterations of sleep, appetite, libido, and/or energy

Major depression persists at least two weeks but can last for months or years.Some patients recover fully from each episode,particularly with treatment, and may remain well for years, other recovers only partially or endures frequent relapses despite treatments with medication and psychotherapy.Upto 13% of people with major depression remain in a chronic, severe state of illness.

As it is well known that there are two major groups of stress hormones catecholamines (adrenaline and nor-adrenaline) and glucocorticoids (cortisol). These catecholamines and glucocorticoids regulate pro- and anti-inflammatory cytokines which are produced by many cells in the body, and they also regulate one another. As catecholamines can increase pro-inflammatory cytokine production, glucocorticoids generally inhibit the production of cytokines (but not always).

The parasympathetic nervous system also plays an important regulatory role in allostasis.Since it generally opposes the sympathetic nervous system by slowing the heart and inducing anti-inflammatory effects means when any one mediator is increased or decreased, compensatory changes in other mediators occur.

This complex web of interactions ensures much "adaptive plasticity", so that in most situations, stress induces brain changes that are the transient and routinely normalized. At other times, through interactions among genetics, the intensity and duration of stress, along with previous life experiences lead to **dysfunction and depression**. This results in increased catecholamines secretion which in turn leads to increased pro-inflammatory cytokine production that enhance the inflammatory reactions in periodontium that may lead to periodontal diseases.

According to Friedlander and Norman[1], depression is also related to increased anticholinergic activity. There are several studies regarding depression and decreased salivary flow is given in the literature. Strongin and Hinsie[2] compared parotid gland flow rates in six manic-depressive patients with flow rates in normal control subjects and found that in the manic group flow rates fell within the normal range, whereas in the depressed subjects flow rates were below the lowest rate of the normal group.

We know that behavioral factors plays pivotal role in disease progression. Depressed patients have more tendency of impaired health behaviors like they do not maintain oral hygiene properly, increased consumption of cigarettes and alcohol, disturbed sleeping patterns, poor nutritional intake. All these factors individually as well as in combination may affect the periodontal health.

Taking antidepressants has another side of story. Along with positive effects on mental health it imposes negative effects on periodontal health. Reduced saliva secretion as a consequence of antidepressant medication is one of the main reasons which make the oral environment susceptible to periodontal diseases.[3,4]

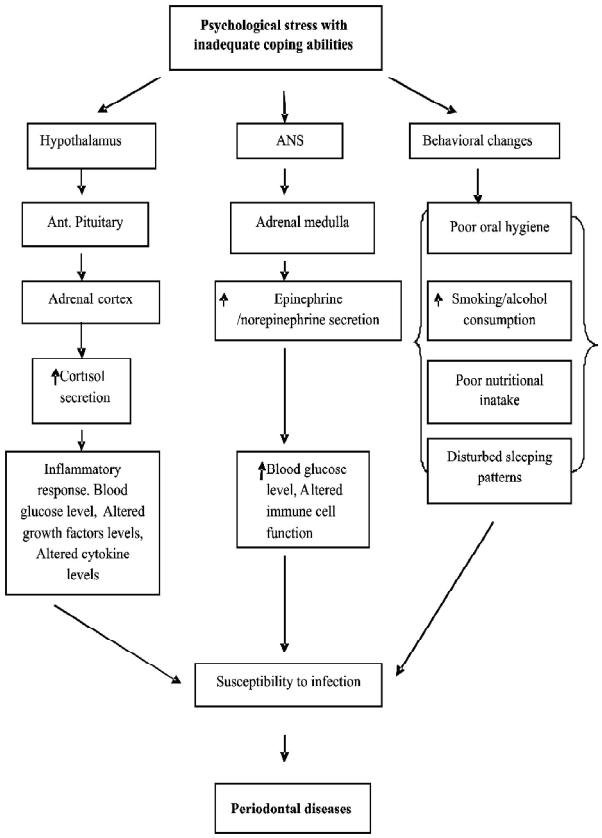
Depression and periodontal diseases

As depression influences the immune system and also modify the behavioral habits, it is extremely important to analyze its influence on the different forms of the periodontal diseases. Several human and animal studies suggests an association between stress and periodontal disease[5-8] but there are relatively few studies on the relationship between depression and periodontal conditions.

However, the concepts of stress and depression are biologically related, and there is substantial evidence that alteration of the stress hormone system plays a major causal role in the development of depression.

Gingivitis

Axtelius[5] confirmed the presence of cortisol in gingival crevicular fluid. Cortisol is a hormone released in body during stressed conditions. Its role has been proven in breaking down the overall good health of the periodontium, which can lead to periodontal disease. Cortisol can also suppress the



Source: Pathophysiology of stress response[9]

immune system, which promotes bacterial growth. Rosania et al[10] reported the cortisol level changes in depression patients. Hugo et al[11] reported that depression and high cortisol levels are risk indicators of elevated plaque and gingivitis levels in individuals aged 50 years and older. Cortisol hypersecretion has been shown to be a biological risk indicator for depression and may play a role in cognitive and emotional processing and depression. Tse and Bond[12] reported that cortisol secretion was significantly associated with both depression and poor social functioning. These findings suggest that poor social functioning is the mediator and elevated cortisol secretion is the predisposing factor in depression.

Johanssen et al[13] evaluated the relationship betweendental plaque, gingival inflammation and elevated levels of interleukin-6 and cortisol in gingival crevicular fluid from women with stress related depression and exhaustion. Borkowska and coworkers[14] also reported the positive association between gingival bleeding and HAD (Hospital anxiety and Depression scale) scores.

Chronic periodontitis

Genco et al[6] investigated the relationship of periodontal disease in adults with stress, distress, and coping and found that depression was associated with greater levels of clinical attachment loss (CAL) and alveolar bone loss. Ng and Leung[15] examined the relationship of periodontal disease to depression and found that there were trends of more severe psychological symptoms of depression in those with more severe CAL. Moss et al[16] explored the role of depression as a host factor which could influence the progression of chronic periodontitis and found that an individual's case status and presence of *T. forsythus* were associated with higher levels of depression.

Recurrent progressive periodontitis (RPP)

The term refers to destructive periodontal diseases in patients who, when longitudinally monitored, demonstrate additional attachment loss at one or more sites, despite well-executed therapeutic and patient efforts to stop the progression of disease. Brown and co-workers[17] reported that people who felt themselves depressed were at a higher risk for periodontal disease progression. Monteiro da Silva and co-workers[18] found that depression measured on a HAD scale, was significantly increased in the group of RPP patients compared to the routine chronic adult periodontitis and control groups.

Aggressive periodontitis

Page et al[19] described the aggressive periodontitis as a particular disease and established a link between aggressive periodontitis and psychosocial factors and loss of appetite. Further, Monteira da Silva[18] and Ababneh et al[20] showed that people with aggressive periodontitis were more depressed and more socially isolated than people with chronic periodontitis or a control group.

Necrotic periodontitis

Necrotizing periodontal diseases include ulcerated and necrotic interdental papillary and marginal gingiva covered by a yellowish-white or grayish slough or pseudomembrane, blunting and cratering of papillae, bleeding on provocation or spontaneous bleeding, pain, and fetid breath. These diseases may be accompanied by fever, malaise and lymphadenopathy.

It is well established in literature that psychosocial factors are predisposing factors for the development of necrotic periodontitis. Cohen-Cole et al[21] found higher scores on the MMPI (Minnesota Multiphasic Personality Inventory) depression scale among necrotic periodontitis patients compared to controls.

Periodontal treatment outcome

Generally depressed patient pay less attention to the oral hygiene practices and also to keep them motivated is difficult as they are found to be ignorant. According to Kloostra et al[22] more depressed a patient was, the more pain they had post-periodontal surgery, the stronger the pain medication they used, and the more delayed their wound healing was after surgery.

Genco and coworkers[6] evaluated the associations of stress, distress, and coping behaviors with periodontal diseases and found that the depression measured with BSI (Brief Symptom Inventory) scale was associated with a higher level of clinical attachment loss after adjustment for the known risk factors for periodontal disease. Likewise, a statistically significant association was found between financial strain and clinical attachment loss as well as bone loss. Elter et al[23] also found that depression was significantly associated with poor periodontal treatment outcome.

Stratification of the subjects according to their coping behavior showed that the risk for clinical attachment loss among the subjects with financial strain was modifiable by coping style in such a way that highly emotion-focused coping (poor coping), which is suggested to correlate with depression[24,25], increased the risk, while problem-focused coping (good coping) decreased it.

Conclusion

It is well documented that depression has impact on psycho-neuroimmunology, endocrino-logy and also on human behavior. Hence depression may influence periodontal disease but this interrelationship has not been well documented yet. Further studies are required to confirmits role as a risk factor for periodontal diseases. People who are feeling depressed or people having a passive coping strategy may be at risk. It is important to understand the

patients' physical and psychological state both to help in maintaining a healthy periodontium.

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Review Article

Inhalation Agents and its Abuse : Neurobiological Considerations

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Introduction

Recent years have witnessed growing reports of inhalant abuse by adolescents and young adult population throughout the world, especially in India. Traditional as well as non-traditional abuse of inhalants in teenage groups is reported from time to time. These organic inhalants are easily available chemical vapors that produce mind altering effect? These inhalants/ solvents are easily accessible to young people, as they are found in numerous readily available household and commercial products (for example, paint products, glues, petrol, correction fluid, lighter fuel and aerosols), and are cheap and legal, when inhaled in to the lung they get easily absorbed into blood and cause action on brain and other structures of the central nervous system.

These substances of daily use, are inexpensive and can be had for negligible cost especially among school age children and teenagers, and are considered to be a stepping stone to the long way of substance abuse, it is often called the 'gateway' drug,leading to the abuse of alcohol and other addictive substances. In spite of their low cost, legal availability and few or no barriers and widespread use, very little is known about the effects produced by inhalants on the central nervous system.

The behavioral effect and clinical feature produced by inhalant and inhaled anesthetics have a lot of similarities, since both types of chemicals are halogenated or aromatic hydrocarbons there seems to be same inhalant action on anesthetic target and GABA synapses and volatile anesthetics also have a history of abuse potential.[1-3] Also like anesthetics, abused inhalants exhibit non-selective actions on a number of neurotransmitter and voltage gated ion channels.[4-6] Synaptic sites of action of inhalant compound play a major role for inhalational anesthetic effects on the central nervous system.[1,7]

Compounds abused

Inhalants encompass a broad range of volatile compounds (see Box 1), such as nitrites, anesthetic gases and organic solvents. Such products typically contain a mixture of solvents, including aliphatic hydrocarbons (for example, isobutane,n-butane, n-hexane and propane), aromatic hydrocarbons (for example, toluene and xylene), chlorinated hydrocarbons (for example, tetrachloroethylene, 1,1,1-trichloroethane and trichloroethylene) and ketones (for example,acetone, butanone and methyl iso-butyl ketone).[8] However, it is toluene (also known as methylbenzene or phenylmethane), a clear and colourless flammable liquid commonly used as an industrial solvent in the manufacturing of paints, chemicals, pharmaceuticals and rubber, that is considered to have the highest potential for abuse.

Methods typically used for Inhalation

People typically abuse volatile substances by deliberately inhaling available vapors 15–20 times over a relatively brief period (for example, 10–15 min). That results in very high concentrations being inhaled (46000 p.p.m.), although the exact concentration typically varies by compound.[10]

Sniffing involves direct inhalation from a container or a piece of clothing sprayed with the substance.

Huffing when some users attempt to increase the amount of available vapors by heating the

Inhalant and their common Chemical Constituents[9]

Volatile Solvents

Correction fluids (1,1,1-trichloroethane)

dry-cleaning fluids (trichloroethylene, 1, 1, 1-trichloroethane)

glues (n-hexane, toluene, xylene)

nail polish remover (acetone, esters)

paint thinners and removers (dichloromethane, toluene, xylene)

petrol (benzene, n-hexane, toluene, xylene) — spray paints, glues, cement, paint remover, thinner, gasoline

Aerosol (may contain chlorofluorocarbons and fluorocarbon propellant)

Deodorants and hairspray

Fabric protector sprays

Spray paints(toluene,methyl isobutyl ketone)

Vegetable oil spray

Gases

Bottled gas (propane)

Cigarette lighter fluid (butane)

Medical anesthetics (ether, chloroform, nitrous oxide)

Whipped cream (nitrous oxide)

Nitrites

Amyl nitrite

substance first, or by holding a soaked cloth over the nose or mouth.

Bagging further increases the concentration of inhaled vapors, and involves breathing from a paper or plastic bag containing the volatile substance.

Typically, experimental use begins with the sniffing of inhalants, gradually progressing through huffing and bagging as theirmisuse escalates in the person.[11] Being lypophilic agent it causes rapid entry to brain,thus leading to high brain/blood ratio for long time.

Neurobiological effects

As compared to other substance of abuse, inhalants are the least well known. There has been little work/ knowledge about action of inhalant and understanding neurobiology of inhalant misuse. But over the past decade, there are preclinical research conducted for investigating the effects of acute inhalant exposure in adult animals.[10]

- Neuropharmacological effects of inhalants: cellular level
- Neuropharmacological effects of inhalants: receptor expression/neurochemistry
- Neuropharmacological effects of inhalants: behavior

Neuropharmacological effects of inhalants: cellular level

Psychopharmacological profile of commonly abused inhalants differ, but evidence till date shows that they act on common molecular receptors The majority of behavioral effects occur at micromolar inhalant concentrations, and results because of changes in receptor activity and/or membrane ion channel.[10]

During Acute solvent exposure it seems to produce NMDA (N-methyl-D-aspartate receptor) receptor inhibition, with the (NR1–NR2B) subunit combination the most sensitive to inhibition.[10] Cruz et al[5] also reported that toluene produces a rapid, non-competitive, almost complete and reversible inhibition of the cationic currents through NMDA receptors, Bale et al[7] demonstrated similar acute inhibition of NMDA receptors in cultured rat hippocampal neurons (IC50¼1.5mM). Findings were suggestive of hyperexcitability/hyper-glutamatergic state during withdrawal following chronic exposure, which similarly occurs during withdrawal from alcohol.[12] In vitro preparations, an IC50 of 1.5mM (as described above) equals to an inhalation exposure of approximately 4600 ppm (the molecular weight of toluene being 92.14MDL), which equals with binge concentrations inhaled by most human users.

There is also evidence that toluene and perchloroethylene acutely inhibit nicotinic ACh receptors (particularly alpha 4 beta 2, alpha 3 beta 2 and alpha 7 subunits),[4,7] in animal model explains that acute toluene exposure regulate hippocampal muscarinic receptor binding.[13] Toluene (0.5mM) also inhibits calcium-dependent potassium channels and G-protein-coupled, inwardly rectifying potassium channel-mediated currents in oocytes, an effect that is opposite to ethanol within this system.[6]

Neuropharmacological effects of inhalants:receptor expression/neurochemistry

Acute toluene exposure was shown to increase

m-opioid receptor protein in brain stem nuclei, including the dorsal raphe and periaqueductal grey.[14] Williams et al[15] exposed rats to toluene (8000 ppm) for 10 days (30 min day_1) and demonstrated increased NR1 and NR2B receptor subunits in the medial prefrontal cortex and NR2B subunits in the nucleus accumbens, suggesting anincrease in neuronal excitability with prolonged exposure. Chronic exposure was also found to increase GABAA alpha1 subunit levels in the medial prefrontal cortex, but decrease expression in the ventral mesencephalon.[15] Although such findings highlight the potential for excitotoxic neuronal damage with chronic inhalant exposure, most human studies have reported relatively more damage to white matter structures and the lipid component of the myelin sheath.[16,17] However, the cellular mechanisms causing this toxic damage remain undetermined. Animal studies that have attempted to elucidate the pathophysiological mechanisms of toluene encephalopathy tend to suggest that gliosis and activation of astrocytes in white matter, as opposed to neuronal death, is the main mechanism responsible.[18-20] In line with this notion, Aydin et al[21] used magnetic resonance spectroscopyto demonstrate decreased levels of N-acetylaspartate (a metabolite produced within neuronal mitochondria that reflects neuronal density and functional viability) and increased myoinositol-containing compounds in a sample of chronic users. These findings suggest that inhalant abuse does not cause active demyelination or breakdown in the neuronal membrane, but rather, may lead to impaired functional viability as a result of diffuse axonal injury.

Neuropharmacological effects of inhalants: behavior

Like other drugs of abuse, inhalants have reinforcing effect as they modulate mesolimbic dopaminergic activity. Toluene has been shown to induce c-fos activation in both the ventral tegmental area (VTA) and nucleus accumbens.[22] Perfusion of toluene directly into the VTA increased dopamine concentrations within both the VTA and the nucleus accumbens, suggesting that there are increases in somatodendritic dopamine release within the VTA as a consequence of increased neuronal firing.

Further, microdialysis revealed that toluene was more effective in increasing dopamine concentrations within the nucleus accumbens when infused directly into the posterior compared with the anterior VTA., and these findings are consistent with other research examining the neuropharma-cological effects of other psychoactive drugs within the mesolimbic system.[23-25] In human beings such studies are still in scarcity but the animal model clearly demonstrate neurotoxicity due to toluene exposure.

Similar to other CNS depressants, inhaled toluene tends to produce a dose-related continuum of effects that progress from motor excitation at low concentrations (that is, 500–4000 ppm) to sedation, motor impairment and anesthesia at higher concentrations (6000–15 000 ppm).[26,27] Prolonged exposure to high concentrations of toluene can result in coma and subsequent death (as a result of respiratory depression). Although some inhalants have proconvulsant properties (for example, the GABAA antagonist flurothyl), most of the commonly abused solvents have anticonvulsant effects[26,27] probably as a consequence of their activity on NMDA and GABAA receptors.

Acute inhalant exposure in human subjects (consistent with animal data) results in short-lived excitation, as well as subjective feelings of euphoria and light-headedness.[28] Intoxicated users feel less inhibited, making them more likely to act impulsively or take risks. Continued use leads to dizziness, sleepiness, slurred speech, blurred vision and headaches. At this stage, users may appear confused, ataxic or begin responding to hallucinations. With higher doses, further CNS depression occurs, which mayresult in seizures, coma and even cardiopulmonary arrest.[28] Users are at risk of suffocation or burns from exploding solvents, although deaths among young people are largely associated with 'sudden sniffing death' or accidental injury as a result of impulsive risk taking and impaired motor skills while intoxicated. There is no apparent safe level of use, with even first-time experimental users at risk of sudden sniffing death as a result of cardiac arrhythmias (particularly after abuse of toluene, chlorofluorocarbons and butane).[29] Inhalants appear to sensitize the myocardium to endogenous catecholamines, which may result in fatal ventricular arrhythmias if the user is startled or agitated.[28] Cruz et al[5] recently demonstrated that toluene reversibly inhibits cardiac voltage-activated sodium channels in a concentration-dependent

manner, which may partially explain their arrhythmogenic effects. The practice of spraying inhalants directly into the mouth is also potentially fatal, as the cooling agents within aerosols can produce death by asphyxiation.

Indian Perspective for Inhalant Abuse

There are few studies reporting inhalant abuse/ dependence in India. Few studies and case series resulting in paucity of literature thus result in lack of awareness in general population and health professionals and planning of health related strategies.

There is a growing trend of use of inhalants among adolescents especially in the lower economic groups like street children[30]. Benegal et al[31] showed in their study that, street based children start off with tobacco use when they are 10-11 years when they are little older they graduate to use inhalants. Other studies in special populations like juvenile delinquents also show that inhalants are one of the commonly used drugs of abuse[32]. They described as gateway drugs, which supposedly causes its users to move on to harder drugs. All the gate way drugs like inhalants are easily available to the children and can cause severe addiction.

In another clinical study[32] done at PGI Chandigarh of 21 consecutive inhalant abuser revealed that 100% were unmarried male of 19 year of mean age group with 76% belonging to middle socioeconomic status and having poor social support (62%). Duration of inhalant abuse has mean of 16 month with sniffing (67%), huffing (19%), curiosity (62%) and under peer pressure (24%). Around half of cases have positive family history of substance abuse.

How can Inhalant Abuse be recognized

Early identification and intervention are the best ways to stop inhalant abuse before it causes serious health consequences. Parents, educators and other health care practitioner role seem to be very important in early detection and prevention.

Early Recognition of signs of Inhalant Abuse

- Chemical odors on breath or clothing.
- Paint or other stains on face, hands, or clothes.
- Hidden empty spray paint or solvent containers and chemical soaked rags or clothing.
- Drunk or disoriented appearance, Slurred speech, Inattentiveness, lack of coordination, irritability and depression.

Short and Long Effects of Inhalant Use

Effects produced by inhalant varies from rapid high, with initial excitation followed by drowsiness, disinhibition, lightheadedness and agitation. If sufficient amounts are inhaled gases or solvent gases may produce a loss of sensation and which can further lead to unconsciousness.

Soon after intake effects like belligerence, apathy, impaired judgment and impaired functioning in work or social situations. Nausea and vomiting are other common side effects. Inhalant abuser may experience dizziness, drowsiness, slurred speech, lethargy, depressed reflexes, general muscle weakness and stupor. Asphyxiation, suffocation, convulsion, coma, choking and fatal injuries can also take place.

In addition cardiac arrhythmias, pulmonary (bronchitis), renal (renal tubular acidosis), hepatic (elevated liver function enzymes), hematological (bone marrow suppression). Immune (reduced cellular immunity) and teratogenic (fetal solvent syndrome) effects have also been noted.[33,34]

Impact and burden of inhalant abuse

There is paucity of literature and research on inhalants in India. Abuse of inhalants starts at young age causing a huge burden on society.In a crucial developmental period like childhood and adolescence, this could lead to progressive neglect of studies, extracurricular activities and interpersonal relationships placing these youth many paces behind their contemporaries.

Addiction to substance abuse especially among the vulnerable populations can directly contribute to high risk behaviors.[35] Activities like gambling, drug peddling, pick pocketing, stealing, fighting, rape and self harm are common among youth when intoxicated. They are at a higher risk of depression and suicide. Children and adolescents at times indulge in or are forced into sex in exchange for drugs. Youth substance abusers are prone to delinquent behavior and anti-social activities. They are at risk of contracting blood borne and sexually transmitted infections like AIDS, when they share

used needles or have unprotected sex under the influence of substance. Younger ages at the onset of substance use and dependence like that of opioids are associated with a higher severity of substance use, a higher lifetime use of other substances, higher sensation seeking and higher global psychopathology.[36] The financial burden of substance use poses on the substance users, their families and the nation will also be significant.

Preventive measures

There is limited evidence on the treatment and preventive strategies of inhalant abuse with very few studies from the developing nations and India in particular. The need of the hour is to investigate the efficacy of these interventions in the Indian setting and to formulate policies based on an evidence base.

Interventions

Inhalant misuse is a complex problem, involving many factors such as social and economic disadvantage, youth disengagement, and cultural issues as well as being associated with polydrug use. For this reason, the best approach to minimize the harms from inhalant misuse requires an holistic range of interventions that address all aspects of the problem from the user's point of view, and encourages active cooperation at the community level[37].

Diversion

The Children and adolescents of impressionable age should not be allowed to work at places were these inhalants are easily available like motor mechanic , painting shops etc.

Boredom and disengagement in society have been highlighted as key factors in the uptake of inhalant use.[37] To address this issue, communities should be encouraged to provide a range of sporting, recreational, artistic and educational activities to engage and provide meaningful opportunities for youth. Programs need to be appropriate to the gender and age of the participants, particularly in Indigenous communities[37] and youth to be brought in main stream of which education to be backbone since childhood leading the child to less chances of having inhalant abuse.

Product reformulation

Products need to be reformulated so that they are not appropriate to be inhaled

Retail control

The fact that inhalant products are so readily available and inexpensive is one of the major factors in their appeal to many users and there should be education regarding their rights and legal responsibilities in selling products that are used as inhalants.

Treatment

There are several factors that need to be considered in the treatment of chronic inhalant users: age and cultural background; family involvement; social and educational engagement; involvement in the justice system; physical and mental health and poly-drug use.

Community engagement

Successful responses to inhalant misuse require widespread community engagement and support to implement a range of intervention strategies, there should be close ties with governments; law enforcement agencies; health, community and education services; and retailers to provide a coordinated approach.[37]

Training

It is important that frontline healthcare workers, teachers, police, and youth and community. Counselors have appropriate training in recognizing the signs of inhalant misuse and are aware of the process for handling intoxicated persons and referral to appropriate services.

Harm reduction practices

For these users, harm reduction strategies may help to minimize harmful outcomes.

Media

The media has an important role to play in public education.

Conclusion

Substance use among adolescents in India is a major health concern associated with many co-morbidities and complications. Start of intake of substance use in childhood almost always results in

continuation of the substance into later ages. Therefore it is important to plan preventive strategies aimed at target population to reduce the burden caused by substance abuse in a developing nation like India. There are no large scale studies on adolescent substance use in the country as of date. Most of the research data available is from studies like small school based surveys, data from treatment and rehabilitation centers data or studies on special populations like street children. Further research in substance abuse in adolescents especially in the avenues of treatment and prevention are lacking and is the need of the hour.

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Review Article

Current Understanding of Organic Delusional Disorder — A Recent Update

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Introduction

Delusion disorders earlier known as paranoid disorders are known since ancient times. Emil Kraepelin described paranoia as stable, non bizarre, well-systematized delusions with a chronic course, although the course lacked the typical deterioration of dementia praecox. The difference between paranoid disorder and paranoid schizophrenia was maintained by Eugen Bleuler. This view was expressed in the second edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-II), in which paranoid states were regarded as possible variants of schizophrenia.[1]

Winokur renamed this illness as delusional disorder (DD). Kendler elaborated Winokur's criteria and suggested a division into simple delusional disorder which are without hallucina-tions and hallucinatory delusional disorder.[2,3]

The topic of validity of this diagnosis is also a matter of debate but recently Marneros et al, has validated DD as separate entity stable over fourteen year follow up study.[4]

Organic delusional disorders are syndromes produced by neurological disease or toxic metabolic disorders and are associated, mainly with limbic system and basal ganglia dysfunction.[5]

Epidemiology

Delusional disorders are not uncommon and according to epidemiological studies, rate for DD in inpatient admissions was reported as 0.3-0.5%[2] and as 0.5-9%.[6] In India 1% of the total outpatients have delusional disorder including half with delusional parasitosis.[7]

Epidemiology of organic delusional disorder is not very well studied and according to Lo Y et al prevalence of organic delusional disorders is 0.4 % of total admissions and 2.9% of organic mental disorders.[8]

Methodology

Relevant articles were searched using the words "Organic Delusional Disorder" using Google Scholar, Pubmed and standard textbooks of Psychiatry and Neurology. 241 articles were retrieved on pubmed. Additional search was carried out to find out the etio-pathogenesis and treatment.

Nosological Status

The nosological status of delusion disorder has been a matter of debate. The revised third edition of the DSM (DSM III R) and fourth edition of the (DSM IV) recognized the Kraepelin's concept. Table 1 shows the difference between criteria of delusional disorder in various classificatory systems.[9-12]

In ICD 10 the following types may be specified, if desired: persecutory type, litigious type, self referential type, grandiose type, hypochondriacal (somatic) type, jealous type, erotomanic type while In DSM IV erotomania, persecutory, somatic, jealous, grandiose, mixed, unspecified are the subtypes.

According to ICD 10 the diagnosis code for organic delusional disorder is F06.2 named as Organic delusional [schizophrenia like] disorder. It requires general criteria for F06 to be met, which are objective evidence from physical, neurological examination and laboratory tests and/or history of

Table-1: Features of delusion disorders (functional) in various classificatory systems. [9-12]

Features	DSM III R (297.10) and DSM IV (297.10)	DSM 5 (297.1)	ICD 10 (F22)
Non Bizarre	+	—	+
Hallucination	Olfactory and tactile hallucination can be present if Related to Delusion Theme	If present not prominent and related to Delusion Theme	Persistent hallucinations in any modality must not be present (but transitory or occasional auditory hallucinations that are not in the third person or giving a running commentary), may be present.
Duration	1 Month	1 Month	3 Months
Intact Functioning	+	+	—
Mood Disorders	If present, relatively should be for brief duration	If present, relatively should be for brief duration	Depressive symptoms or even a depressive episode may be present intermittently, provided that the delusions persist at times when there is no disturbance of mood.

cerebral insult or of systemic disorder known to cause cerebral disease. There should be a presumed relationship between development of the organic disease and mental disorder. Recovery of mental symptoms should be there on improvement of underlying cause and alternative causation of mental disorder should be absent. For F06.2 clinical picture should be dominated by delusions and consciousness should be clear.[11] In DSM III R there was a separate category named organic delusional disorder, code (293.81)[9] but in DSM IV and DSM 5 it is diagnosed as psychotic disorder due to another medical condition with delusions (293.81).[10-12]

Characteristics of Organic Delusional Disorders

Organic delusional disorders provide insight into the biological causation of psychiatric disorders. Delusions present in Organic delusional disorders share characteristics of the functional delusional disorders but these organic syndromes differ from delusional disorders in various important aspects. There are studies done in this area, which compare characteristic features of these two groups.[8,13] Table 2 shows comparison between organic delusional disorder and delusional disorder whereas Table 3 shows comparison between organic delusional disorder and schizophrenia.

Etiology

Genetics

Catalano et al studying genotype of schizophrenia, normal and subjects with delusional disorder found that involvement of genetic variation in the Dopamine D4 receptor gene confirmed susceptibility

Table 2. Comparison of organic delusional disorders and delusional disorders/Schizophrenia

	Organic delusional disorder	Delusional Disorders[8]
Age of onset	Late onset	Relatively early onset
Family history	Present less often	Present more often
Hospital stay	Longer	Shorter
Treatment	Require less dosage of anti psychotics	Require comparatively more dosages of antipsychotic

Table 3. Comparison between organic delusional disorder and schizophrenia

	Organic delusional disorder	Schizophrenia[13]
Intellectual impairment	More common	Less common
Sensorium	May be altered	Intact
Hallucinations	Hallucinations of Smell, taste or touch are more prevalent	Less common
Affect	Preserved	Flat
Thought process	Intact	Disorganized

to delusional disorder.[14] In a molecular genetic study of delusional disorder, Morimoto et al found genotype frequency of the DRD2 gene Ser311Cys to be higher in cases with persecutory type DD (21%), compared to schizophrenia cases and controls (6% each).[15] In other studies, a strong association was observed HLA A3 and A11. [16]

Neuroimaging

There are various neuroimaging studies done to localize the brain area involved in delusions and it has been that various areas like orbitofrontal cortex, amygdala, striatum, thalamus are affected.[17] Delusional misidentification syndromes have been seen to be associated with impairment of face recognition which is a condition appears to be associated with lesions affecting limbic structure and both frontal and parietal lobes with predomi-nance of right sided lesions.[18]

In Table 3, neuroimaging findings of few studies has been given which shows the evidence that certain brain areas are affected in patients with delusions.

Psychodynamic theories

According to psychodynamic theories persecutory delusion are protective psychological responses to conflicts that are threat to self. Delusions are seen as a personal unconscious inner state or conflict which is turned outwards and attributed to the external world. Freud considered that latent homosexual tendencies especially formed the basis of paranoid delusions. Later, psychoanalytical theorist suggested that delusions might be a compensation for any, not necessarily sexuality-related kind of mental weakness, e.g. lack of self-confidence, chronic anxiety or identity disturbances. [1, 18]

Other Theories

Cognitive and experimental psychologists suggests that person with persecutory delusions selectively attend to threatening information; attribute negative events to external personal causes. In shared psychotic disorders etiological theories are based on family and interpersonal dynamics.[40]

Table-3: Neuroimaging findings of studies in patients with various delusions

Type of Delusion	Brain areas involved	Authors
Delusion of parasitosis	MRI showed damaged striatum and it was found that dopamine transporter functioning was also decreased	Huber M et al (2007)[19]
Somatic Delusion	Hypoperfusion of left temporo parietal cortices	Wada et al (1999)[20]
Cotard Delusion	Non dominant temporoparietal cortex involvement	Pearn J et al (2002)[21]
Misidentification syndromes	MRI showed right frontal white matter lesions	Luca M et al (2013)[22]
Persecutory Delusions	fMRI showed involvement of Left frontal, right caudate and cingulated gyrus	Blackwood NJ et al (2000)[23]

Medical causes

It is not uncommon for medical diseases to present as delusional disorder.[24] The various important causes implicated in the etiology of Organic delusional disorder are as follows:

Infectious diseases (e.g Creutzfeldt-Jakob disease[25]), Neurodegenerative disorders (e.g. Alzheimer's disease,[26] Huntington's disease[27]), Brain tumors,[28] leptomeningeal metastases from a systemic cancer[29], Head trauma[30], Vascular disease (e.g. atherosclerotic vascular disease, hyper-tension)[31], Metabolic and endocrinal causes (e.g Hypercalcemia, hyperparathyroidism,[32] hypothyroi-dism[33]), Vitamin deficiencies (e.g Vitamin B_{12} deficiency)[34], Medications (e.g corticosteroids,[35] disulfiram[36]), Substances (e.g cocaine,[37] alcohol[38]) and Toxins (e.g. Mercury).[39]

Differential Diagnosis

Many different conditions may present with suspiciousness and persecutory delusions among the elderly. Differential, in addition to above mentioned medical conditions are dementia, persistent delusional disorder, depression with psychotic features, schizophrenia, shared psychotic disorder and personality disorders.

Management

Assessment

As there are numerous conditions that can

present with delusion disorder, it requires thorough investigation and hence outline of the required work up is presented.

Detailed psychiatric history taking, review of substance use, past treatment and current medication, forms the backbone of any psychiatric casework up and rule out known causes.

Detailed general physical, systemic examination and central nervous system examination will provide us with the clue towards the organic cause.

Mental status examination and higher mental function with detailed cognitive function assessment will establish delusion and rule out any delirium, dementia or other organic disorders.

Investigations would include complete blood count, erythrocyte sedimentation rate, liver function tests, kidney function tests, serum electrolytes, Thyroid function tests, urine examination, toxicology screening, Vitamin B_{12} levels, Brain imaging.

Treatment

Much of the literature on treatment of organic delusional disorder is in the form of reports on individual or very small series of cases. Treatment would mainly depend upon the cause detected. For persistent symptoms various treatment modality are used. The introduction of oral Pimozide[1] and its use in the treatment of delusional disorder has led some researchers to claim its therapeutic specificity for delusional disorder, which is not shared by other antipsychotics. From last two decades antipsychotics remain the main stay of the therapy but currently with the increasing research in psychosocial interventions, highly structured, integrated programs like cognitive behavioral and psycho education are being used. Here we are discussing about newer treatment modalities.

Non-Pharmacological

1. Cognitive-behavioral therapy (CBT) has been effective in treating delusions, both in schizophrenia and delusional disorder. CBT produced more impact on the Maudsley Assessment of Delusions Schedule (MADS) dimensions for Affect Relating to Belief, Strength of Conviction, and Positive Actions on Beliefs.[41]
2. A significant improvement has been seen in individuals with persistent delusion disorder with cognitive behavioral worry intervention, a specialized form of CBT targeting worry, which, according to research is associated with distressing paranoia.[42]
3. The Maudsley Review Training Program me: It aimed to target reasoning processes, particularly the 'Jumping to Conclusions' (JTC) bias and belief flexibility, which are thought to play a part in maintaining delusional conviction. It is a computerized programme comprising a general introduction to JTC and five training tasks. It was designed to be completed together with a therapist, who emphasized key messages and provided feedback on participants' comments, for example by reinforcing useful insights and normalizing JTC. When comparing the average baseline and post-intervention periods there were significant improvements in belief flexibility and delusional conviction in sample that is resistant to change using either traditional CBT for psychosis and/or antipsychotic medication.[43]

Pharmacological

With the advent of newer antipsychotics, more and more research is going on in measuring the efficacy of these medications in resolving persistent delusional disorders. Among the newer antipsychotics, Blonanserin[44], Paliperidone[45] have been successfully used in the treatment of the delusional disorder. Even all the currently available long acting injectable antipsychotics are used in the treatment of delusional disorder as first line treatment.[46]

In addition to antipsychotics, other forms of therapy such as Selective serotonin reuptake inhibitors, Monoamine oxidase inhibitors, Clomipramine, and Electroconvulsive therapy have also been shown to have beneficial effects in patients with chronic delusions.[47] In anecdotal case reports it has been seen that apart from the neurosurgical interventions, there is a need for antipsychotics[47,48] and other medical intervention.[49,50]

Conclusion

Delusional disorders forms a separate entity as evident through research carried in last few decades and it finds a place of its own in currently used classificatory systems, though more research is required to refine the nosological status of organic

disorders. Organic delusional disorders provide a great opportunity to study biological aspects of schizophrenia and other psychotic disorders as specific findings in each delusions are being discovered. Organic causes are not uncommon for the delusions disorders and diagnosis requires a detailed assessment through history, physical examination, mental status and detailed cognitive assessments followed by required investigations. We should thoroughly assess for potentially treatable neuro - medical causes to prevent morbidity and mortality and improve overall quality of life.

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Review Article

Group Interventions for Teachers and Parents of Children with Scholastic Backwardness

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Introduction

Scholastic backwardness is one of the common problem with which the children are referred to a child guidance clinic. Poor scholastic performance causes distress among the parents.It is estimated that 5-15% of school-going children are having scholastic backwardness[1].This has to be recognized and resolved at the earliest or it may lead to chaos in a child's life.It's not only affects school completion but it has life -long impact on the child's self esteem, higher education, interpersonal relationships, prospects for employment, marriage etc[2,3]. Indian school surveys in the past decade have recorded prevalence rates that range between 20 and 50 percent[4]. A child is considered to have scholastic backwardness if he or she failed regularly in all subjects or had class failure in the previous year[5]. According to Rutter[6] Scholastic backwardness was operationally defined in terms of poor overall scholastic performance as reflected by the overall mark percentage below 35%, and was assessed in response to question I on Performa A of the children's Behavior Questionnaire (CBQ). According to Krishnakumar et al[1] this can be also defined as repeated failures in all subjects or academic performance two classes below the class in which the child was studying at the time. Multi disciplinary approach needed as every scholastic backward child's problem is different and it has a multi-factorial etiology. This has to be tackled very effectively as it is a concern not only for the children but also to their family and teachers.

Criteria for identifying children with scholastic backwardness[3]

- Child who fails in one or more subjects
- Child who fails in one or more classes
- Child in lower 10th percentile marks in his/her class
- Child identified by parent or teacher to be difficult to teach.

The manifestation of academic difficulty varies as the causes vary from neuro developmental disorder, emotional and behavioral disorder, environmental factors to medical factors. A study done by Jayaprakash[7] on 100 children of age group ranging from 4 years - 12 years; who are attending child guidance clinic with history of poor scholastic performance. They were divided into 2 groups. (Group I: Non failure, Group II: Failure). Their finding revealed that psychiatric morbidity was present in 42%, developmental disorder in 34%, non psychiatric medical diagnosis in 25%, and abnormal psycho social situation in 31%. Multiple diagnosis were present in 16%. Comparison shows that prevalence of psychiatric co morbidity was more in the failure group than the non failure group. First and foremost; the parent should have realistic academic goal for the child. High expectation beyond the child's ability will lead to stress for both child and the parent. There is a felt-need for scaling-up strategies for the recogni-tion and management of scholastic backwardness. The role of family is also vital in improving scholastic performance apart from the teachers. Scholastic backwardness contributes to school dropout, especially after the primary school years and should be recognized and remedial measures initiated, in the primary classes itself for best results.[5] If scholastic backwardness is not managed properly, it will cause significant academic stress in children leading on to somatoform disorders, anxiety disorders, depression and even deliberate self harm.[8] Empathetic approach needed for children with scholastic backwardness. Once the diagnosis is made after IQ assessment, management depends on the cause of scholastic backwardness. Psychosocial intervention generally provided for

individuals with scholastic backwardness includes individualized education program, child counseling and family interventions, life skills training, social skills training, parent-child–teacher interaction training, family education, behavioral management, assertiveness training, self esteem enhancement and pharmacological/other structured psychosocial interventions etc.

Group Interventions

Group interventions can be done for homogenous teachers group or parents group however, it's also required for heterogeneous groups including teachers and parents of children with poor scholastic backwardness. Each group interventions can have different purpose including how to recognize poor scholastic backwardness, etiology and management of childrens' scholastic backward-ness and also identifying co- morbid psychiatric or medical diagnosis. At times, the group process can deal with the burden and distress caused while managing children with scholastic backwardness. The combined session would encourage healthy participation of teachers and parents in overall improvement of the child. The open communication can channelize the better utilization pattern of resources such as help from child guidance clinic, psycho social rehabilitation unit of various agencies in their locality. The demand of utilization would encourage professional to give priority to this concerns and would in turn lead to better mental health system in our country.

Need of the hour

1. Periodic group interventions for the parents / teachers / childrens.
2. Child guidance clinic in every Pediatric Department and Psychosocial Rehabilitation Unit in Psychiatry Department.
3. Workshop for teachers / parents to be conducted by clinicians (Pediatricians / Child Psycholo-gists and Psychiatrists / Psych Rehab Consultants)
4. Recruiting psychologist / student counselor to every schools.
5. Inter-disciplinary CME's and Conferences (Pediatric and Psychiatric)
6. Researches need to be focused on new management strategies and scaling–up existing services.

Conclusion

Though scholastic backwardness is common among school going children, psycho social intervention strategies has been not recorded much. Hence blaming game and secluding the child also still exists. A comprehensive evaluation of psycho-social role functioning, environment, evaluation of teachers' and parents' feedback shall be considered for goal setting. Group interventions would facilitate the interaction of the child-parent-teacher participation in improving child's scholastic performance and psychosocial role functioning. Most important is to show consistent un-conditional love and acceptance to the child. This could be feasible by conducting group interventions for teachers and parents of children with scholastic backwardness; by the help of child guidance clinic and psycho social rehabilitation unit.

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Review Article

Psychotherapy Client Centered Way

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Introduction

Psychotherapy is a general term referring to therapeutic interaction or treatment contracted between a trained professional and a client, patient, family, couple, or group. The problems addressed are psychological in nature and can vary in terms of their causes, influences, triggers, and potential resolutions. Accurate assessment of these and other variables depend on the practitioner's capability and can change or evolve as the practitioner acquires experience, knowledge, and insight. Psychotherapy includes interactive processes between a person or group and a qualified mental health professional. Its purpose is the exploration of thoughts, feelings and behavior for the purpose of problem solving or achieving higher levels of functioning. Psychotherapy aims to increase the individual's sense of his/her own well-being. Psychotherapists employ a range of techniques based on experiential relationship building, dialogue, communication and behavior change that are designed to improve the mental health of a client or patient, or to improve group relationships (such as in a family).

Carl Rogers has described his "*Person of Tomorrow*" as the one who taps vast inner resources for self-understanding and for self-directed behavior. This person is free, harmonious, wise, and peaceful, and is able to deal creatively with whatever changes may come. Trusting inner guidance, instead of relying on science, technology or institutions, the Person of Tomorrow relies on personal authority, is caring, capable of intimacy, open to others as well as to inner experience. Life is whole, guided by an integration of the varied energies rather than being motivated solely toward material gains or status symbols.[1] Furthermore, the Person of Tomorrow is guided by a spiritual search. Rogers doesn't promise any happiness and joy that will compete with the gods; but a person with these characteristics cannot fail to have a rich and fulfilling life. He says that if the therapist is empathic, congruent, prizes, and cares for, the client, a climate will be created which facilitates the journey. He also describes something of what the journey is like, when he delineates the characteristic directions of the process of psychotherapy.[2]

Also, his hypothesis on the outcomes in personality and behavior which result from therapy may be taken as a description of what lies ahead. The relationship with therapist still remains an important tool to bring a change in client's functioning. Generally the role of the therapist is to help the client to make his/her own decisions in various walks of life. Flexibility and creativity in the approach is also very important because at times the therapists have to intervene through Family meetings, Group therapy and Cognitive Behavioral approach; other than Individual Psychotherapy[3].

Psychotherapy in the Indian Context

We would also like to talk about one of our ancient scriptures – **'Bhagvad Gita'**. It has been considered as one of the Psychotherapeutic apparatus. Chapter – 4 Verse 34 …….. Krishna instructs Arjuna to become humble so that the knowledge is received by him. In his questioning, he has been fighting with his teacher instead of fighting the battle. Sri Krishna is teaching Arjuna not to resist but to become humble. Then clarity of mind is easily attained. Resistance is also one of the greatest barriers to change found in psychotherapy. The therapist is often in a peculiar position: if he gives advice, client ignores it. Therapist can keep his suggestions to himself, thereby helping the client to sort out his confusion and conflict and to come to

his own conclusions. Progress occurs only to the extent that one goes beyond his resistance, for resistance is holding onto that which one already is[4]. Neki talked about social dependency as a faulty personality and that parents foster dependency while themselves modeling dependable and this assists in the personality development of the Indian Child. Many including Neki have suggested that the relationship of a therapist and his patient is that of Guru and Chela (disciple) in the Indian Context and is without the western transference. Guru is a self disciplined person who helps directly in decision making process. Vidyasagar another well known clinician of North India also reported a great success using this therapeutic mode.[5]

Client-centered therapy—also referred to as person-centered therapy and Rogerian therapy—is a major approach to counseling and psychotherapy developed by Carl R. Rogers (1902-1987). A fundamental clinical framework informing the humanistic psychology movement, the client-centered approach maintains that human beings can only be understood as wholes, through their individual subjective experiences. Another difference between client-centered therapy and most approaches to psychotherapy is that client-centered therapy "definitely rejects the medical model which involves looking for pathology and developing a specific diagnosis, or thinking of treatment in terms of cure". Client-centered therapy posits specific attributes of a "good life" beyond the relative absence of psychopathology; it was designed to provide the conditions that encourage natural human growth and development toward the realization of the individual's potentials. Throughout his lengthy career, Rogers contributed numerous theoretical constructs and propositions that marked the evolution of his perspective. However, the central concept that serves as the core of his approach is the *actualizing tendency.*[6]

We think at present westernization is occurring at a great pace and the boundaries of Indian Psychotherapy and Western Psychotherapy are getting blurred slowly with the passage of time.

Definitions

Psychotherapy has been defined in many different ways by various workers, some of the popular definitions are as follows –

1. Winnicott[7] described Psychotherapy as, "two people playing together" and this recognizes the shared nature of effective psychotherapy.
2. Frank[8] described it as, "A type of social influence exerted by a trained and socially sanctioned healer on a person or persons who suffer and are seeking relief, through a series of defined contacts".
3. Werry and Andrews[9] defined it as, "A treatment that ameliorates psychopathologic conditions, functional impairments, and developmental disturbances by means of psychological processes and a therapeutic relationship with a trained therapist".
4. Butler and Strupp[10] described it as systematic use of a human relationship for therapeutic purposes. According to them it is a modality of treatment in which the therapist and patient(s) work together to ameliorate psychopathological conditions and functional impairment through focus on

- The therapeutic relationship;
- The patient's attitudes, thoughts, affect and behavior; and
- Social context and development.

The third set of domains, social context and development, are particularly salient for research in child and adolescent psychotherapy.[11-13] Contextual domains include parental psychopathology, family interaction, school, peers, neighborhood and service system, and each of these contribute to variance in outcome.

History

History is incomplete without mentioning the name of **Sigmund Freud (1856-1939)**. His orientation to therapy was so completely "physician – directed" that he would not appear to belong in any history of non directive thought. On the other hand, a great debt is owed to Freud by all schools of Psychotherapy for the work he did in establishing the interview as a recognized therapeutic measure and for his theoretical contributions in the fields of unconscious mechanism, childhood, and the emotions, which have made human behaviour far more understandable.[14]

What subject – matter the treatment begins with is on the whole immaterial, whether with the

patient's life story, with a history of the illness or with recollections of childhood; but in any case the patient must be left to talk, and the choice of subject left to him in the field of interpretation. Freud most clearly tends towards non direction as a result of bad luck with directive techniques. Even in later stages of the analysis one must be careful not to communicate the meaning of a symptom or the interpretation of a wish until the patient is already close upon it, so that he has only a short step to take in order to grasp the explanation himself. Premature communication of interpretations brought the treatment to an untimely end, both on account of the resistances suddenly aroused thereby and also because of the relief resulting from the insight so obtained. The intent of the above quotations is not to make Freud out as a nondirective therapist but to demonstrate that a therapist with his fundamentally authoritative orientation found it necessary to reckon more and more with the attitudes of the patient and to depend less and less upon the will of the analyst, in order to make therapeutic progress.[15]

Otto Rank[16]: Rank (1884-1939), long Freud's closest associate and disciple, first rebelled openly against classical Freudian theory and practice in 1924 with the publication of "The Trauma of Birth". In this work, birth replaced castration as the original trauma and the breast took precedence over the penis as the first libido object. In addition, Rank identified the origin of fear with the birth process. Rank is responsible for the initiation in psychotherapy of several extremely significant ideas:

1. The individual seeking help is not simply a battleground of impersonal forces such as id and superego, but has creative powers of his own, a will. When the individual is threatened, when a strange will is forced on him, this positive will becomes counter-will.
2. Because of the dangers involved in living and the fear of dying, all people experience a basic ambivalence, which may be viewed in various aspects. Thus, there is a conflict between will-to-health and will-to-illness, between self-determination and acceptance of fate, between being different and being like others, etc. This ambivalence is characteristic not just of neurotics, but is an integral part of life.
3. The aim of therapy, in the light of the above, becomes the acceptance by the individual of himself as unique and self-reliant, with all his ambivalences, and the freeing of the positive will through the elimination of the temporary blocking which consists of the concentration of creative energies on the ego.
4. In order to achieve this goal, the patient rather than the therapist must become the central figure in the therapeutic process. The patient is his own therapist, he has within him forces of self-creation as well as of self-destruction, and the former can be brought into play if the therapist will play the role, not of authority, but of ego-helper or assistant ego, not of positive will but of counter-will to strengthen the patient's positive will, not of total ego but of any part of the ego felt by the patient to be disturbing and against which he may battle; in sum, the therapist "becomes in the course of treatment a dumping ground on which the patient deposits his old neurotic ego and in successful cases finally leaves it behind him."
5. The therapist can be neither an instrument of love, which would make the patient more dependent, nor of education, which attempts to alter the individual, and so would inhibit the positive will by arousing the counter-will.
6. The goals of therapy are achieved by the patient not through an explanation of the past, which he would resist if interpreted to him, and which, even if accepted by him, would serve to lessen his responsibility for his present adjustment, but rather through the experiencing of the present in the therapeutic situation, in which he learns to will in reaction to the therapist's counter-will, in which he is using all of his earlier reaction patterns plus the present.
7. The end of therapy, the separation of patient from therapist, is a symbol of all separations in life, starting with the separation of fetus from womb in birth, and if the patient can be made to understand the will conflict present here, the conflict over growth

towards independence and self-reliance, and if he can exercise the separation as something which he wills himself, despite the pain of it, then it can symbolize the birth of the new individual.[16]

Hypothesis behind PCT (Person Centered Therapy) – a self directed growth process

Three core therapeutic conditions — "The first element could be called genuineness, realness, or congruence. The more the therapist is himself or herself in the relationship, putting up no professional front or personal facade, the greater is the likelihood that the client will change and grow in a constructive manner. This means that the therapist is openly being the feelings and attitudes that are flowing within at the moment.

The term "*transparent*" catches the flavor of this condition: the therapist makes himself or herself transparent to the client; the client can see right through what the therapist is in the relationship; the client experiences no holding back on the part of the therapist. As for the therapist, what he or she is experiencing is available to awareness, can be lived in the relationship, and can be communicated, if appropriate. Thus, there is a close matching, or congruence, between what is being experienced at the gut level, what is present in awareness, and what is expressed to the client.[17]

Unconditional Positive Regard

The second attitude of importance in creating a climate for change is acceptance, or caring, or prizing—what I have called 'unconditional positive regard.' When the therapist is experiencing a positive, acceptant attitude toward whatever the client is at that moment, therapeutic movement or change is more likely to occur. The therapist is willing for the client to be whatever immediate feeling is going on— confusion, resentment, fear, anger, courage, love, or pride. Such caring on the part of the therapist is non possessive. The therapist prizes the client in a total rather than a conditional way.[1]

Empathy

The third facilitative aspect of the relationship is empathic understanding. This means that the therapist senses accurately the feelings and personal meanings that the client is experiencing and communicates this understanding to the client. When functioning best, the therapist is so much inside the private world of the other that he or she can clarify not only the meanings of which the client is aware but even those just below the level of awareness. This kind of sensitive, active listening is exceedingly rare in our lives. We think, we listen, but very rarely we listen with real understanding, true empathy. Yet listening, of this very special kind, is one of the most potent forces for change.[1]

Evolution of client/person-centered approach to psychotherapy – As told by Rogers, that it was when he unlearned the how-to's and formalized theory of much of his professional training that he could sit down facing a client or student, with his head and eyes clear of clutter, look into the eyes of another person, feel their presence and be free to enter into the experience of the other person, undistracted. It was a fresh and energizing experience. So he had put aside a whole heap of techniques, therapies, expectations, rules, how-to's, methods, strategies, tests, case studies, case histories, theories, etc. They are all in here, somewhere, a part of him; but he does not pull them out for use, one by one, choosing which one is at the moment appropriate, or will work on this person or in that situation.

From Non-directive technique to Client-centered Psychotherapy to a Person-centered way of being

Over a period of 40 years, the growth that began with the non-directive technique evolved first to client-centered therapy, to a person-centered approach to interpersonal relationships. Emphasis on non-directive technique in the early 1940s, although still rooted in the diagnostic-prescriptive approach, was one of the early buds on this new plant.[1] He began to realize that he was saying something new, perhaps even original, about counseling and psychotherapy. He started recording therapeutic interviews, helping to focus his interest on the effects of different responses in the interview. This led to a heavy emphasis on technique-the so-called *non-directive technique*. It was during those years that the word "reflective" was rather widely used in describing the non-directive response of the therapist. Both terms were sometimes seen as passive and indicative of a person

who put himself away and repeated accurately what the client said and nodded or said um hum" to let the client know he was listening. What happened next is for him part of a pattern of growth that has characterized the evolution of the client/person-centered approach. Concluding the earlier quotation, Rogers continues, "I found that I had embarked not on a new method of therapy but a sharply different philosophy of living and relationships".[1] Here the two metaphors come into focus, that of the growing plant and that of the journey, the two metaphors best characterizing the client-centered/person-centered approach: the potato sprout growing toward the light even in a dark cellar and the therapist walking beside the client as they set out on a journey into self and toward becoming a fully functioning person, more fully functioning within the self and more fully functioning as a responsible resident of the planet Earth. Rogers, in "A Theory of the Fully Functioning Person",[18] wrote: Certain directional tendencies in the individual and certain needs have been explicitly postulated in the theory. This is already implicit in what has been given a concept of the ultimate in the actualization of the human organism. This ultimate hypothetical person would be synonymous with "the goal of social evolution-the end point of optimal psychotherapy," - the fully functioning person. It should be evident that the term "the fully functioning person" is synonymous with optimal psychological adjustment, optimal psychological maturity, complete congruence and complete openness to experience, as though such a person "had arrived." All the characteristics of such a person are process characteristics. The behaviors would be adequately adaptive to each new situation, and that the person would be continually in a process of further self-actualization. Within the context of evolution or organismic growth, its quite illuminating, a statement by Rogers in 1983[19] on the same subject: Here, then, is a theoretical model of the person who emerges from therapy, from the best of education, the individual who has experienced optimal psychological growth-the person functioning freely in all the fullness of his organismic potentialities; a person who is dependable in being realistic, self-enhancing, socialized, and appropriate in his behavior; a creative person, whose specific behaviors are not easily predictable; a person who is ever-changing, ever developing, always discovering himself and the newness in himself in each succeeding moment of time. Rogers also admits that the person he has described is a person who does not exist. He is the theoretical goal, the end-point of personal growth. We, the persons are moving in this direction from the best of experience in education and from the best experience in therapy, from the best of family and group relationships. But what we observe is the imperfect person moving toward this goal.[19]

The organismic growth in the concept of the therapeutic experience brought with it not only a wider field of functioning for the client, but a broadening of the concept of the therapist, functioning in the climate of the person-centered approach, and compatible with the metaphor of companion on a journey- "a more experienced and mature companion, but quite different from an expert or a doctor who prescribes a remedy, or a wise person who knows the solution" and will bring the patient around to it in due time, or at least will reveal in the process the causes of the illness or dilemma.

We are seeing the experienced therapist, a fallible human being, psychologically and emotionally mature, secure enough within him/herself to be real and open in the relationship, trustful enough of self to enter into the world of the other and move about comfortably in it without losing him/herself, and holding her/himself in sufficient respect, not only to accept the client with non-possessive, non-judgmental, caring, but to communicate that attitude accurately to the client.

We are seeing the therapist as a facilitator of change, of growth, of learning, of self-empowerment for the other. We are seeing the facilitator who can work with individuals or groups in an academic institution, an agency, a business organization, a center of conflict, in less depth and intensity over a shorter period of time but with many of the same results as a therapist working with a client, one-to-one in a traditional setting. As the therapist is a companion on the journey of a client, so the teacher is a companion to the student, and the group facilitator is a companion to each member of a group on the journey of self-discovery and growth. Other members of the intensive group, in turn, become companions in the search, and in the experience of hundreds of participants, the experience is even more

powerful in its own way than psychotherapy alone, and also can lead to long-lasting changes.

In retrospect, an excerpt from Rogers[20] was prophetic: "Doubtless there are other ways in which this loneliness can be allayed. I have simply tried to present one way, the encounter group or the intensive group experience, in which we seem to be creating a means of putting real individuals in touch with other real individuals. It is, I believe, one of our most successful modern inventions for dealing with the feeling of unreality, of impersonality, and of distance and separation that exists in so many people in our culture. What the future of this trend will be I do not know. It may be taken over by faddists and manipulators. It may be superseded by something even more effective. At the present time it is the best instrument I know for healing the loneliness that prevails in so many human beings. It holds forth the hope that isolation need not be the keynote of our individual lives".

Out of that moment came the term beyond elitism. "*We must move beyond*." Moving beyond elitism means moving beyond the hierarchical selectivity of the one-to-one-only, prescribed, long term, therapist/analyst dominated treatment pattern for patients. It means first moving away from the medical model, labels and diagnoses, prognoses, plans of treatment, quick dependence on drugs, and a "the doctor knows best" attitude, and moving toward trust in the potential of the human organism for growth and positive change, for self-direction, for assuming responsibility for its own growth and actions. It means laying aside the illusion that a psychotherapist, no matter how well-trained or well-grounded in a system, no matter how well-intentioned, can be the expert on another person's life and experience or destiny. It means stepping down from the dais. Moving beyond elitism means that I acknowledge the efficacy of even a large group of participants interacting in a climate conducive to emotional and psychological growth-facilitated, not dominated by experienced persons-to bring about growthful change in individuals who take the opportunity to empower themselves. It means that I accept many avenues by which a client or group participant can enter a growth-promoting or therapeutic relationship, and thereafter can find other ways of continuing the growthful change alone, in other groups or in a client-therapist relationship. It means that I trust that the process, once set in motion will continue even if I (the therapist or facilitator) am not there to make it happen or direct its course-or, what may be more wrenching to accept, because I am not there to direct or make it happen.

From Acceptance to love to Unconditional positive regard in the Therapeutic relationship: Response to a human need

Unconditional positive regard, one of the three conditions clearly stated as necessary and sufficient for constructive personality change to take place[21], has been selected for examination here because it illustrates the process of evolution of the concept from its use in 1951 to the present. As we trace the evolving meaning of the term, we become involved in the search for an exact word that would best communicate the essence of the concept. This purposeful search for meaning, for testing of hypotheses by research and experience, and a relentless striving for clarity and communication have marked the work of Rogers from the beginning. A second and by far more compelling reason for focusing on this one of the three conditions is stated clearly by Rogers:

As the awareness of the self emerges, the individual develops a need for positive regard. This need is universal in human beings, and in the individual, is pervasive and persistent. Whether it is an inherent or learned need is irrelevant to the theory.[21]

One hypothesis is that the client moves from the experiencing of himself as an unworthy, unacceptable, and unlovable person to the realization that he is accepted, respected, and loved, in this limited relationship with the therapist. "Loved" has here perhaps its deepest and most general meaning-that of being deeply understood and deeply accepted. The term nondirective was being used at the Counseling Center of the University of Chicago at the time that this hypothesis involving acceptance, respect and love was being formulated. Another way of "framing" the same hypothesis is that as the client experiences acceptance on the part of the therapist, he is able to accept and experience the same attitude toward himself and, as he thus begins to accept, respect, like, and love himself, he is capable of experiencing these attitudes toward others.

Oliver H. Bown, a member of the staff, felt strongly that the term love, easily misunderstood though it may be, is the most useful term "to describe a basic ingredient for the therapeutic relationship".[22] Bown supports his position as follows:

"It seems to me (Bown) that we can love a person only to the extent that we are not threatened by him. Thus, if a person is hostile towards me and I can see nothing in him at the moment except the hostility, I am quite sure that I will react in a defensive way to the hostility. If, on the other hand, I can see this hostility as an understandable component of the person's defense against feeling the need for closeness to people, I can then react with love toward this person, who also wants love, but who at the moment must pretend not to. Similarly, and somewhat more important to me in my experience, I feel that positive feeling expressed by the client toward us can be a very real source of threat, provided again that this positive expression, in whatever form it may take, is not clearly related to these same basic motivations mentioned above[22]".

Bown[22] suggests, "acceptance, caring, prizing" without the unconditional positive regard falls short of conveying the essential of unconditionality. There are in our society, in education, in business, in family and intimate relationships, including the relationship between client and therapist, varying levels of acceptance, prizing, caring-but usually with conditions attached. The parent implies, "I love you when you're good."; the teacher, "I accept you when you work hard and get a good grade."; the doctor to the patient in the hospital, "I like you or care more for you when you do as you're told."; the corporation says, "Follow the line and the boss will like you. You'll succeed."

Where and how many times in a lifetime does one know that she or he is unconditionally loved and accepted, valued, held in positive nonjudgmental regard? How many persons from the start have someone or ones significant in their lives who say verbally or nonverbally, "I love you. I respect your personhood even when I disagree or don't like what you do. I know you can't always 'do things right' anymore than I can. Sometimes I'll be angry and sometimes I can't be with you but I'll try to listen to you and understand and I don't stop caring for you or write you off because you do something I don't like." In such an experience, a relationship with another significant human being is great power for healing.

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Original Article

Psychiatric Morbidity Among Hypothyroid Patients – A Hospital Based Study

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Abstract

Intoduction: *The connection between hypothyroidism and depression is well known, and almost every psychiatrist will test patient's level of thyroid hormones before prescribing antidepressant medications, believing that even mild cases of low thyroid function can cause major depression. Patients with hypothyroidism present with high psychiatric morbidity such as depression which is prevalent in 28-50% among hypothyroid patients and anxiety disorder occurs in between 30-40% of the patients.* ***Material and method:*** *A total of 100 diagnosed patients of hypothyroidism were assessed on HRDS and HARS attending the thyroid clinic. They were assessed on socio-demographic profile, duration of illness, type of hypothyroidism and then the data was analyzed on different domains.* ***Results:*** *63 % of the patients of hypothyroidism had co-morbid depression . Females were showing high percentage of depression while mild anxiety was more in males (69.23%) as compared to females (47.29%). Moderate to severe levels of depression and anxiety was found more in females as compared to males. The severity level of depression was more in sub clinical hypothyroidism group than the clinical group irrespective of sex ($p<0.01$).*

Key words: *Hypothyroidism, Anxiety, Depression*

Introduction

The connection between hypothyroidism and depression is well known, and almost every psychiatrist will test patient's levels of thyroid hormones before prescribing antidepressant medications, believing that even mild cases of low thyroid function can cause major depression.[1,2] For the physicians and Endocrinologists the main focus in the treatment of hypothyroidism is hormonal therapy and they fail to consider psychiatric aspects in such patients.

Studies have found that significantly higher number of patients with unipolar depression have subnormal T3 and T4 levels and a corresponding increase in thyroid stimulating hormone (TSH) levels compared with healthy controls.[3,4] Another study found that 20.5 % subjects of major depressive disorder have hypothyroidism.[5]

Both hypothyroidism and depression appear to have a common basis in terms of physiological chemistry. Specifically, the same portion of red blood cells that takes up the thyroid hormone T3 also takes up the amino acid L-tryptophan, which is critical in depression. Low levels of L-tryptophan are associated with depression, and low levels of T3 are associated with hypothyroidism, so it is the common starting point on the cellular level that explains why the two conditions often exist together and adding thyroid hormones to a treatment regimen for depression often helps to alleviate the condition.

Psychiatric presentations are often the first sign

of hypothyroidism, occurring as the initial symptoms in approximately 2% to 12% of reported cases, with organic mental deficits being the most frequently reported initial symptoms. Anxiety and progressive mental slowing associated with diminished recent memory, speech deficits and diminished learning ability are the characteristic initial progression of symptoms. Patients may become delusional, paranoid and may complain of auditory or visual hallucinations.[6,7]

Anxiety disorders occur in between 30% and 40% of patients developing acute hypothyroidism.[8-10] Popkin[11,12] in addressing the issue of endocrine disorders presenting with anxiety, suggests that anxiety states frequently occurs in thyroid disorders including hypothyroidism, hyper thyroidism and thyroditis.

Patients with subclinical hypothyroidism are also reported to show increased anxiety and irritability, slowed information processing speed, reduced efficiency in executive functions and poor learning and decreased vitality and activity when compared to normal subjects.[13,14]

Psychiatric patients with subclinical hypothyroidism especially those with incomplete responses to psychotropic therapy should usually be treated with thyroid hormone, even if thyroxine and TSH concentrations are normal.[15]

Aims and Objectives

To assess the prevalence of psychiatry morbidity among hypothyroid patients by using standardized rating scales for depression and anxiety.

Material and Methods

A total of 100 diagnosed patients of hypothyroidism were assessed on HDRS and HARS attending the thyroid clinic. They were assessed on socio- demographic profile, duration of illness, type of hypothyroidism (sub clinical or clinical) and then the data was analysed on different domains.

Inclusion Criteria

- Patients were above the age of 20 years at the time of study.
- Patients of both the sexes were taken for study.
- Patients should be a known case of hypothyroidism.
- Patient not suffering from any depression/ anxiety before the diagnosis of hypothyroidism.

Exclusion criteria

- Associated drug and alcohol dependence.
- Presence of any serious organic illness.
- Any other major psychiatric illness like schizophrenia, mental retardation.
- Patients with severe cognitive impairment.
- Patient already on any psychotropic drug or any past history of psychiatric disorder.

Results And Discussion

Table 1 shows the demographic profile of the patients where 74% of the females were diagnosed a case of hypothyroidism in comparison to males (26%).

Table 1- Socio-demographic profile of the patients

Age (in years)	Male	Female
< 30	04	10
31-50	18	50
> 50	04	14
Educational Level		
Upto Matric	12	35
Matric- Graduation	08	25
> Graduation	06	14
> Graduation	—	—

This finding supports the findings of Redmond[16] which concludes that hypothyroidism is 10 times more common in females than in males, and also more in the older age than younger age groups.

Table 2 : Shows that about 63% of the patients of hypothyroidism had co- morbid depression which supports the findings reported by other researchers where depression reported in 28 to 50 % of the sample.[17]

Females were showing high percentage of depression, and the severity level was also higher in the females. Moderate to severe level of depression was found to be more in females (49.29%) as compared to males in which it was (30.77%).

Table 3: Shows that the common symptoms found in patients with hypothyroidism are depressed mood, anxiety gastrointestinal, somatic and genital symptoms. Genital symptoms were usually reported by the males (50%) as compare to females

Table 2- Hamilton Depression Rating Scale

	Male N= 26	Female N = 74	Total
< 08 (No depression)	12 (46.15%)	25 (33.78%)	(37%)
8-13 (Mild depression)	06 (22.08%)	12 (16.22%)	(18%)
14-18 (Moderate depression)	04 (15.38%)	10 (13.51%)	(14%)
19-22 (Severe depression)	03 (11.54%)	15 (20.27%)	(18%)
23 and above (Very severe depression)	01 (03.85%)	12 (16.22%)	(13%)

Table 3. Shows comparative distribution of positive rating on symptom checklist of HDRS between males and females

	Males (N=26)	Females (N=74)
Depressed Mood	18(75%)	40 (54.05%)
Suicide	5(19.23%)	30 (40.54%)
Insomnia	10(38.46%)	25 (33.78%)
Anxiety	20 (83.33%)	45 (54.05%)
Somatic Symptoms Gastro intestinal	10 (38.46%)	40 (54.05%)
Somatic Symptoms general	10 (38.46%)	45 (60.81%)
Genital Symptoms	13(50%)	16 (21.62%)
Loss of weight	5 (19.23%)	10 (13.51%)

(21.62%).

Carani et al[18] where in hypothyroid men, the prevalence of hypoactive sexual desire, delayed ejaculation and erectile dysfunction was 64% and pre mature ejaculation was in 7.1% of the cases.

Table 4 : Mild anxiety was more in males (69.23%) as compared to females (47.29%) whereas moderate to severe anxiety level were high in females (52.27%) as compared to males (30.76%). This is in consistent with the findings of many researchers where anxiety disorder reported in 30- 40% of patients developing acute hypothyroidism[8-10].

Table 5: Shows that on symptom checklist of HARS , anxious mood is mostly reported by females (81.08%) as compared to males (38.46%). Somatic symptoms are mostly reported by females (56.75%)as compared to males (30.76%) which is consistent with the findings of Haugg TT et.al, 2004 (mean number of symptoms women/men : 3.8/2.9).

Table 6 and 7 shows that the severity level of depression was more in sub clinical hypothyroidism

Table 4- Hamilton Anxiety Rating Scale

	Male N = 26	Female N= 74	Total
< 17 (Mild)	18 (69.23%)	35 (47.29%)	53%
18-24 (Moderate)	6 (23.07%)	30 (40.54%)	36%
25 (Severe)	02 (07.69%)	09 (12.16%)	11%

Table 5. Shows comparative distribution of positive rating on symptom checklist of HARS between males and females

	Males (N=26)	Females (N=74)
Anxious Mood	10 (38.46%)	60 (81.08%)
Tension	08 (30.76%)	45 (59.21%)
Insomnia	06 (26.08%)	30 (40.54%)
Depressed Mood	10 (38.46%)	40 (54.05%)
Somatic (Sensory)	08 (30.76%)	42 (56.75%)
G I Symptoms	12 (46.15%)	42 (56.75%)
Genitourinary Symptoms	15 (57.69%)	12 (16.21%)
Autonomic Symptoms	08 (30.76%)	38 (51.35%)

group than the clinical group irrespective of sex ($p<0.01$). similar findings were reported by Valeria et al[19].

Table 6. Severity of Depresson in Different Categories of Hypothoiroidism in Males (N=26)

Type of hypothyroidism / HDRS	Sub clinical (N=8)	Clinical (N=18)
Mean HDRS	20.8	16.5
Standard Deviation	2.63	2.16

t= 4.392; df= 24; p<0.01 (Highly Significant)

Table 7. Severity of Depresson in Different Categories of Hypothoiroidism in Females

Type of hypothyroidism / HDRS	Sub clinical (N=48)	Clinical (N=26)
Mean HDRS	21.4	18.25
Standard Deviation	2.22	2.13

t= 5.899; df= 72; p<0.01 (Highly Significant)

Conclusion

There is no doubt that thyroid hormone plays a major role in the regulation of mood, cognition and

behavior and thyroid dysfunction leads to variety of psychiatric manifestations such as anxiety, depression, diminished recent memory and learning ability. So patient presenting with such symptoms should have both the psychiatric and endocrine function evaluation to decrease the morbidity.

Limitations

The present study has assessed only anxiety and depression but the other psychiatric aspects should be explored. Other is that correlation between duration of hypothyroidism and severity of depression should be considered.

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Original Article

Mood Disorder Symptoms in first episode Schizophrenia : A descriptive study

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Abstract

Background: *Depressive features are commonly seen in most patients with first episode schizophrenia. The authors assessed the presence and frequency of depressive and manic as well as positive and negative schizophrenia symptoms, in a group of patients with first episode schizophrenia who were not in a major depressive episode or diagnosed with schizoaffective disorder.* ***Method:*** *72 outpatients with first episode schizophrenia between the ages 25 and 60 years were studied. Depressive symptoms were rated primarily with the Hamilton Depression Rating Scale. Standardized instruments were also used to measure positive and negative symptoms and manic features.* ***Results:*** *Depressive symptoms were more frequent and severe in first episode schizophrenic patients. Psychomotor agitation, decreased interest in day to day activities, decreased sleep and aggressiveness correlated across all groups.* ***Conclusions:*** *Depressive symptoms are common in patients with schizophrenia.They may be an independent, core component of the disorder or, alternatively, may be a product of severe psychotic symptoms.*

Key words: *First episode schizophrenia, Depressive symptoms, Positive and negative symptoms.*

Introduction

Various studies have noted that the same symptom constellation may have different meaning when they appear in different psychiatric disorders. Studies have found that depressive symptoms in patients with schizophrenia may be secondary to negative symptoms, medications, and drug induced movement disorders.[1] Some studies have reported that in patients with chronic schizophrenia and even first-episode schizophrenia, depressive symptoms may be a core component of various stages of this illness.[2,3] Various studies have put the frequency of depressive episodes as well as symptomatology in patients with first episode schizophrenia to range from 10% to 95%.[4,5] Differences in sample, duration of illness and assessment methods contribute to the variability of these estimates.[6] Depression during the acute phase of first episode schizophrenia may be a good prognostic factor[7] but several studies have suggested that depression in chronic schizophrenia is associated with a greater risk of suicide[8] and relapse.[9]

High rates of depressive symptoms have been associated with positive symptoms in schizophrenia along with physical limitations that interfere with activities, diminished social networks and low income.[10] In the present study, our primary aim was to assess the frequency and the degree of depressive symptoms in a clinical group of patients with first episode schizophrenia who were not diagnosed with concurrent major depression or schizoaffective disorder. We also tried to look at the symptom profile which was more common in this group.

Methodology

All subjects gave written informed consent to participate in this study conducted at a tertiary centre in Mumbai. The DSM-IV criteria[11] was used to diagnose schizophrenia determined on the basis of a clinical interview and evaluation by a psychiatrist. There were 72 subjects in the study that met the criteria for first episode schizophrenia. Socio-demographic information and symptom ratings were obtained by trained clinical raters. Severity of depressive symptoms was rated with the Hamilton Depression Rating Scale,[12] whereas positive and negative symptoms were rated with the Scale for the Assessment of Positive Symptoms (SAPS)[13-14] and the Scale for the Assessment of Negative Symptoms (SANS).[15-16] Manic symptoms were assessed using the Young's Mania Rating Scale (YMRS).[17] Descriptive statistics and percentages were used to describe the data.

Results

On assessing the symptoms on the SAPS of all cases of first episode schizophrenia (N = 72), it was seen that 61(85%) had persecutory delusions while 53(74%) showed both delusions of reference and bizarre behaviour. 51(71%) demonstrated aggressiveness in some form (Table 1). On assessing negative symptoms, 60(83%) showed affective blunting, 74% showed affective non responsibility, 79% were inactive during the mental status examination and 85% expressed an inability to feel intimate and close in a relationship or in general (Table 2). Psychomotor agitation (71%) and decreased need for sleep (28%) were the only manic scale symptoms that were shown a majority of cases. Irritability along with aggressive thought were correlated on the Young's Mania rating scale as they were on the SAPS (Table 3). Decreased interest in

Table 1. Scale for Assessment of Positive Symptoms

Symptoms	SCZ (N = 72)
Hallucination	36 (50%)
Auditory hallucination	33 (46%)
Voices commenting	03 (04%)
Voices conversing	12 (17%)
Somatic/tactile hallucination	01 (01%)
Olfactory hallucination	00 (0%)
Visual hallucination	04 (06%)
Delusion	61 (85%)
Persecutory delusion	61 (85%)
Delusion of jealousy	18 (25%)
Delusion of guilt/sin	02 (03%)
Grandiose delusion	17 (24%)
Religious delusion	03 (04%)
Somatic delusion	05 (07%)
Delusion of reference	53 (74%)
Delusion of being controlled	07 (10%)
Delusion of mind reading	02 (03%)
Thought broadcasting	04 (06%)
Thought insertion	02 (02.77%)
Thought withdrawal	00 (0%)
Bizarre behavior	53 (74%)
Clothing and appearance	32 (44%)
Social and sexual behavior	25 (35%)
Aggression and agitated behavior	51 (71%)
Repetitive and stereotyped behavior	1 (01%)
Positive formal thought disorder	15 (21%)
Derailment	02 (03%)
Tangentiality	04 (06%)
Incoherence	04 (06%)
Illogicality	08 (11%)
Circumstantiality	06 (09%)
Pressure of speech	15 (21%)
Distractible Speech	07 (10%)
Clanging	01 (01%)

Table 2. Scale For Assessment of Negative Symptoms

Symptoms	SCZ (N = 72)
Affective flattening/Blunting	60 (83%)
Unchanged facial expression	43 (60%)
Decreased spontaneous movements	41 (57%)
Paucity of expressive gesture	51 (71%)
Poor eye contact	47 (65%)
Affective non responsibility	53 (74%)
Lack of vocal inflection	48 (67%)
Inappropriate affect	10 (14%)
Alogia	46 (64%)
Poverty of speech	44 (61%)
Poverty of content	44 (61%)
Blocking	00 (0%)
Increased latency	41 (57%)
Avolition - apathy	67 (93%)
Grooming and hygiene	34 (47%)
Impersistence at work	67 (93%)
Physical anergia	57 (79%)
Anhedonia – asociality	65 (90%)
Recreational interest and activity	63 (88%)
Sexual activity	21 (29%)
Ability to feel intimacy and closeness	61 (85%)
Relationship	61 (85%)
Attention	60 (83%)
Social inactiveness	59 (82%)
Inactive during MSE	57 (79%)

Table 3. Symptoms on Young Mania Rating Scale

Symptoms	SCZ (N= 72)
Elated mood	14 (19%)
Increased motor activity	14 (19%)
Sexual interest	10 (14%)
Sleep	65 (90 %)
Irritability	65 (90 %)
Speech	17 (24%)
Language and thought disorder	15 (18%)
Content	61 (85%)
Disruptive – aggressive behavior	51 (71%)
Appearance	32 (44%)
Insight	67 (93%)

day to day activities (90%), decreased sleep (90%), issues related to insight and psychomotor agitation were the significant symptoms on the HDRS (Table 4).

Table 4. Symptoms on Hamilton Rating Scale for Depression

Symptoms	SCZ (N = 72)
Depressed mood	13 (18%)
Feeling of guilt	02 (03%)
Suicide	04 (05%)
Insomnia early	47 (65%)
Insomnia middle	37 (51%)
Insomnia late	25 (35%)
Work and activities	67 (93%)
Retardation	31 (43%)
Agitation	51 (71%)
Anxiety psychic	15 (21%)
Anxiety somatic	06 (08%)
Somatic symptoms – gastrointestinal	06 (08%)
Somatic symptoms – general	08 (11%)
Genital symptoms	05 (07%)
Hypochondriosis	04 (06%)
Loss of weight	14 (19%)
Insight	67 (93%)
Diurnal variation	00 (0%)
Depersonalisation /derealisation	01 (01%)
Paranoid symptoms	61 (85%)
Obsessional and compulsive symptoms	03 (04%)

Discussion

Depressive symptoms seemed to be a part and parcel of first episode schizophrenia, even in a cohort specifically defined so as not to be in a major depressive episode or to have schizoaffective disorder. Depressive symptoms were frequent and did not appear to be simply a by-product of age, neuroleptics, family history, negative symptoms or movement disorder but rather appeared as a part of the psychotic process. This could be the result of various symptom correlates seen as a part of various psychiatric disorders. Multiple neurobiological correlates and the same neurotransmitters involved in multiple psychiatric disorders could be another cause for the symptom overlap. This study had several strengths. First, the research group included only first episode schizophrenic patients largely overlooked in previous studies. Second, psychometrically sound instruments were used. Although the Hamilton depression scale was not designed as a measure of depressive symptoms for schizophrenic patients, it has been used in numerous studies to measure severity of depressive symptoms in schizophrenic patients, and factor analytic studies have found discrete factors reliable and valid for this population.[18]

There also are several limitations to the study. Although 72 subjects represent a relatively large group for first episode schizophrenia, a larger group would have allowed us to look at other clinically important subtypes (such as late-onset versus early-onset first episode schizophrenia and paranoid versus non-paranoid patients). We also did not include a control group. Finally, the study was cross-sectional; a longitudinal design would permit one to learn more about the meaning, course, and consequences of depressive symptoms in these patients. The frequency with which depressive or manic symptoms are routinely seen treated in clinical practice previously has been pointed out.[19] The study is similar to other work done in the past. The spectrum of depressive symptoms seen in schizophrenic patients was broad ranging from lack of interest to decreased sleep and agitation.

The clinical importance of depressive symptoms in schizophrenia by several studies is that subsyndromal depression symptoms in psychotic patients are associated with considerable social dysfunction and disability,[20] suicide attempts[21] and risk of later major depressive episodes.[22] Our results are not consistent with what one might have expected on the basis of epidemiological studies of depression in schizophrenic individuals living in the community[23] or some studies of younger individuals with schizophrenia that show men with a more severe illness than women.[24] Perhaps a larger group might have revealed more significant findings in terms of

various variables. A published study of younger individuals with schizophrenia found a significant association between depressive symptoms and family history of unipolar affective illness.[25] Our inability to find a significant relationship between family history and current symptoms of depression supports the notion that depression may be a core manifestation of schizophrenia[26], rather than simply a by-product of a familial risk of comorbidity.

Thus, depression may be a concomitant response to psychotic symptoms — a by-product and yet a common feature of schizophrenia, or it could be a relatively independent, albeit over-lapping, aspect of schizophrenia. An important next step would be to extend these findings to the entire age range of patients with schizophrenia, to assess the course and consequences of symptoms of depression over suitable follow-up periods with larger groups, and to measure the frequency and severity of depressive symptoms in patients with low, moderate, and high levels of psychotic symptoms across various cohorts and correlate the data.

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Original Article

Oral health of Non-Institutionalized psychiatric patients: A Dentist Perception

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Abstract

Background: *Patients with compromised mental health are more vulnerable to dental neglect and poor oral health. There is not as much of knowledge regarding dental ailments and maladies in this population in spite of possible risk factors.* ***Material & Methods:*** *The study was conducted in Outpatient psychiatric and dental department of a tertiary care government run GTB Hospital, Delhi. The purpose of this study was to evaluate the oral health status of non institutionalized 150 psychiatric patients and 30 healthy patients matched for age and gender as a control group. These patients were examined using the simplified WHO oral health assessment form and in addition, were questioned with respect to their oral hygiene habits also. Dental caries, oral hygiene status and other oral health findings like bruxism, attrition, xerostomia, missing and filled teeth were assessed and recorded.* ***Results and Conclusion****: Oral health status of the non institutionalized psychiatric patients was relatively poor when compared with the control group. Most of the psychiatric patients had poor oral hygiene habits and paid least attention to the dental treatment needs. Oral health has a significant impact on general health, self esteem, and quality of life but it is often has a low priority in the context of mental health. Further synchronized endeavors amongst medical, dental, and social care segments need to be initiated for addressing the oral health needs of this group of patients.*

Keywords: *Oral health, Non-Institutionalized, Psychiatric patients, Xerostomia, Dental Caries.*

Introduction

Patients with compromised mental health are more vulnerable to dental neglect and poor oral health.[1] There is not as much of knowledge regarding dental ailments and maladies in this population in spite of possible risk factors comprising of -diet and psychotropic drug side effects e.g. xerostomia. Population surveys[2-6] have also established that people with persistent mental health illnesses make less repeated scheduled appointments to the dentist and account for a larger number of neglected teeth than the general population.

Oral health assessment and treatment needs of the non-institutionalized psychiatric patients have obtained little response and there is still scarcity of information when assessed with the institutiona-lized patients.[2,5] Reduced salivary flow is commonly noted in patients secondary to the anti depressant drugs intake which may lead to glandular hypo function resulting in xerostomia, a significant causative factor of mucosal dryness and dental caries. Further, lack of motivation for maintaining good oral hygiene, dietary influences and accumulation of bacterial plaque are cumulative factors for causing two most common dental diseases i.e. caries and periodontal disease seen in psychiatric patients.[2,3,7-11]

The aim of the present study was to determine

the oral health status using the simplified WHO oral health assessment form (2010) and to evaluate dental treatment needs in non institutionalized patients suffering from chronic psychiatric illness.

Material and Methods

The study was conducted in Outpatient psychiatric and dental department of a tertiary care Guru Teg Bahadur Hospital, Delhi. The study was carried out as a part of psychiatric recall visits and patients with confirmed diagnosis of chronic psychiatric illness (schizophrenia and mood disorders) classified as per International Statistical Classification of Diseases – 10th edition[12] and consent was obtained from patients and caretakers before screening.

The study comprised of two groups i.e.

- *Group I* — Study group consisting of 150 chronic psychiatric patients (with schizophrenia or mood disorder)
- *Group II* — Control group consisting of 30 patients without a psychiatric disorder.

Oral examination was carried out on consenting subjects using mouth mirror, probe and explorer by two trained dental surgeons. Oral health indices were used to assess dental caries, oral hygiene status and other oral health parameters e.g. tooth wear, xerostomia, halitosis, oral hygiene habits and previous visits to dentist were also assessed.

Statistics

Descriptive statistics was used to arrive at the results of the study.

Results

The present study consisted of 150 patients. The mean age of patients was 33.83 years. Data on medical variables showed that 50-55% subjects attending the psychiatry OPD were suffering from mood disorders and were in the 2nd and 3rd decade while, nearly 35-40 % of the patients were suffering from neurotic disorders. This was followed by 5-10% patients who had schizophrenia. 21 % patients were taking psychoactive substance (most commonly tobacco followed by alcohol) regularly.

Xerostomia

The prevalence of xerostomia was as high as 99.3% in overall population (Table 1). Majority of population i.e. 65.2% between ages 21-40 years suffered from xerostomia. (Table 2)

Dental Caries

There was no significant difference between the sexes. It affected 69% (table 1) of the total subjects. When compared for age groups, the highest prevalence was found in the age group 21-40 years affecting 66% of the population. (Table 2)

Community Periodontal Index (CPI)

CPI was high in both the genders i.e. 94.6% (table 1) with greater periodontal involvement in 57.1 % males compared to 42.9 % females. The greater periodontal involvement was seen in age group 41-60 years i.e. 42.9%, signifying more need to visit to Dentist for maintaining oral hygiene. 59.7% of population presented with halitosis. Only 0.2% people resorted to flossing and only 22.8% of population did rinsing after meals.

Missing teeth

The overall prevalence of missing teeth in the study group was 21%, involving 52.2% of females (Table 1) and 47.8% of males. The highest prevalence of missing teeth was reported in the age group 21-40 years i.e. 60.2% where as it was only 17.7% for the age group below 20 years. (Table 2)

Tooth Wear & Bruxism

Tooth Wear was the most prevalent finding with 100% of population showing the same. There was

Table-1: Oral health status of psychiatric patients: Distribution and P-value of various dental parameters

Parameter	Females %	Males %	Overall percentage	P value
Missing Teeth	52.2%	47.8%	21%	.618
Dental Caries	48.9%	51.1%	69.1%	.533
Oral Hygeine Status	63.6%	36.4%	93.9%	.285
CPI	42.9%	57.1%	94.6%	.982
Oral Lesion	51.4%	48.6%	50%	.869
Tooth Wear	54.4%	45.6%	100%	.260
Xerostomia	56.5%	43.5%	99.3%	.397
Halitosis	52.5%	47.5%	59.7%	.295
Brushing	37.5%	62.5%	94.6%	.382
Flossing	47.9%	52.1%	.02%	.115
Rinsing	46.6%	53.4%	22.8%	.179
Previous Visit to Dentist	50%	50%	81.2%	1

no significant difference between males and females (Table 1).

Previous Visit to Dentist

Total of 57.1% population in the age group of 21-40 years had visited dentist before. Only 10.7% of the population between 41-60 years had visited dentist ever before (Table 2). 94.6% required oral prophylaxis.

Similary, Hede[16] assessed the dental health status among the homebound psychiatric patients and found that 63%of the total patients were in great need for dental treatment.

Shah et al[17] found that only 26.7% of the psychiatric patients in a group of 133 home bound patients had healthy gingiva in the age group of 21-50 years which further worsened with age.

The results of our study showed that psychiatric patients have plethora of dental problems, many of them requiring complex dental treatment and regular recall visits. The psychiatric patients were not visiting their dentist for routine checkup except in case of dental emergencies. Further, it was observed that awareness regarding oral health and motivation for practicing good oral hygiene habits was minimal in these patients.

Table 2:Distribution of patients according to Age

Age	Missing Teeth	Dental Caries	Oral Hygeine Status	CPI	Oral Lesion	Xero-stomia	Halitosis	Brushing	Flossing	Rinsing	Previ-ous Visit to Dentist (Never Visited)
<20	17.7%	17.0%	9.1%	42.9%	19.4%	26.1%	8.0%	14.2%	33.3%	9.1%	28.6%
21-40	60.2	66.0%	54.5%	14.3%	46.7%	65.2%	64.8%	63.8%	33.3%	63.6%	57.1%
41-60	19.5%	14.9%	18.2%	42.9%	18.1%	8.7%	21.6%	17.7%	33.3%	24.2%	10.7%
61-80	19.5%	2.1%	18.2%	.0%	2.8%	0%	5.7%	4.3%	.0%	3.0%	3.6%

Mean age for Females 33.72; Mean Age for Males 33.93; Overall Mean Age 33.83

Discussion

This study reported the dental findings of the psychiatric patients diagnosed by a senior psychiatrist (accordance to ICD-10). General characteristics of study subjects in our study showed that mean age of our study subjects was 33.83 years. The most common psychiatric diagnosis in non-institutionalized patients was mood disorders.

In the present study, we found significant amount of reduced salivary rates, halitosis, dental caries and bruxism. These results are similar to Javonic et al.[13] Also, we found greater periodontal involvement in the older age group, affecting more males than females. The reason could be attributed to smoking, tobacco chewing and low motivation to visit dentist and to practice good oral hygiene habits.

Further, the study revealed 99.3% of the patients were having xerostomia contributing to significant increase in the incidence of dental caries. Kumar et al[14] reported prevalence rate of caries as 32.2% with mean DMFT and DMFS scores of 0.92 ± 1.8 and 2.54 ± 5.0 respectively; these results are almost consistent with the results of our study. As the age increased, the oral health status in terms of OHI-S worsened, our findings are very much comparable with the findings of other studies.[2,15,16]

Conclusion

Oral health has a significant impact on general health, self esteem, and quality of life but it is often has a low priority in the context of mental health. Nonetheless, it habitually has a truncated significance in the framework of psychological health and further, in certain psychiatric diseases, the priority may be non-existent. Thus, psychiatric patients form a high risk group for dental diseases.

However, prevention of oral diseases should be the main objective because patients with advanced mental illness are often anxious and uncooperative in the dental clinics. Therefore, further synchronized endeavors amongst medical, dental, and social care segments need to be initiated for addressing —the oral health needs of this group of patients.

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Original Article

Knowledge, Attitude, Perception and Belief (K.A.P.B.) of patients' relatives towards mental illness: A cross-sectional study

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Abstract

Background: *Social science research contributes to development and evaluation of strategies to combat stigma by improving knowledge of and attitude towards mental illness. Study of attitudes occupies a central place in social-psychological research and there is need for research on effective approaches to stigma reduction so that various mental health programmes could be formulated and organised which can help in rehabilitation of patients as well.* ***Aims:*** *This study aimed at assessing the knowledge, attitude, perception and belief of patients' relatives about the causes, manifestations and treatment of mental illness.* ***Settings and Design:*** *Cross-sectional study design was used which was carried out in psychiatry department of a tertiary care teaching hospital.* ***Methods and Material:*** *Relatives of patients selected using serial sampling procedure were administered the scale, "Opinion about Mental Illness".* ***Statistical analysis used:*** *The chi-square (x^2) test.* ***Results:*** *Relatives of patients are better informed about treatment than to nature and aetiology of mental illness. Attitudes towards authoritarianism, social restrictiveness and interpersonal aetiology were negative while those towards benevolence and mental hygiene were positive.* ***Conclusions:*** *Educational programmes for the relatives of patients by developing psychoeducational intervention and sensitisation campaigns are needed.*

Keywords: *Knowledge, Attitude, Perception, Belief, Relatives, Mental illness.*

Introduction

Adverse attitudes to mental illness are found in all societies in the world.[1] The belief that mental illness is incurable or self-inflicted can also be damaging, leading to patients not being referred for appropriate mental health care.[1] Research on attitudes towards the mentally ill is necessary to ensure quality of life for persons with mental illness in the community.[2] Little is known about the knowledge and attitudes of rural community towards mental illness in India.[2] Research on mental health literacy in India is limited despite increasing growing evidence of mental illness among general population.[3]

Stigma

Stigma is defined as a sign of disgrace or discredit, which sets a person apart from others. The stigma of mental illness, although more often related to context than to a person's appearance, remains a powerful negative attribute in all social relations.[4]

The difference between a normal and a stigmatised person was a question of perspective, not reality.[5] Stigma (like beauty) is in the eye of the beholder, and a body of evidence supports the concept of stereotypes of mental illness.[6-8]

On a systemic level, stigma as a social phenomenon has a strong influence on the policies

that govern the nature of, access to and funding for treatment and support, eligibility for social assistance or the right to refuse treatment. At the level of the community, stigma may affect how organisations including social service agencies, employers, health care providers, or schools respond to individuals with a mental illness and to their families. At the individual level, stigma prohibits people from seeking the treatment they need, creates profound changes in identity and changes the way in which they are perceived by others.[9]

When people understand that mental disorders are not the result of moral failings or limited will power, but are legitimate illnesses that are responsive to specific treatments, much of the negative stereotyping may dissipate.[10] Fresh approaches to disseminate research information and to counter stigma need to be developed and evaluated.[9] Social science research has much to contribute to the development and evaluation of anti-stigma programmes.[11]

Long-term strategies are needed to combat stigma by disengaging mental illness from associated fears and anxieties, and by improving knowledge and attitudes. Two important ways of doing this are improvement in "mental health literacy" and stopping the constant reinforcement of stigma by the media.[12] Further, there is need for research on effective approaches to stigma reduction through public education.[9]

Mental health literacy

Health literacy has been defined as "the ability to gain access to, understand and use information in ways which promote and maintain good health". 'Mental health literacy' is defined as "knowledge and beliefs about mental disorders which aid their recognition, management or prevention". Mental health literacy consists of several components, including: (a) the ability to recognize specific disorders or different types of psychological distress; (b) knowledge and beliefs about risk factors and causes; (c) knowledge and beliefs about self-help interventions; (d) knowledge and beliefs about professional help available; (e) attitudes which facilitate recognition and appropriate help-seeking; and (f) knowledge of how to seek mental health information.[13]

Family

The role of family in psychiatry is of paramount importance for family is the universal, primary social unit and provides off-spring with both, his/her biological and cultural endowment. Stigma affects not only people with mental illness, but their families as well.[11] Family and friends may endure a stigma by association, the so-called "courtesy stigma".[5] It is important to underline that the challenge is as much about compelling people to change their attitudes towards and/or be more understanding of those who struggle with a mental illness as it is to move them to recognize and acknowledge their own mental health problems, and those of their families, friends and employees.[9]

Knowledge, attitude, perception and belief (K.A.P.B.)

The study of attitudes has occupied and continues to occupy a central place in social-psychological research. Knowledge of attitudes and their functioning is of interest both theoretically and practically. No theory of social behaviour can be complete without incorporation of attitude functioning, and it is doubtful that complex social behaviour can be predicted without a knowledge of attitude. To study attitudes requires that they be measured.[15]

Keeping in view these important aspects of public opinion about mental illness, this study on "knowledge, attitude, perception and belief (K.A.P.B.) of patients' relatives towards mental illness" seems meaningful so that various mental health programmes could be formulated and organised. This will also help in proper rehabilitation of the patient.

Methods

Aim

This study aimed at assessing the K.A.P.B. of patients' relatives about the causes, manifestations and treatment of mental illness.

Sample

The sample consisted of 100 relatives of patients. The study was done on one adult family member (related by blood/marriage) living with and accompanying the patient.

Inclusion criteria:

1. Relatives with age above 18 years.
2. Key relatives having a relationship of parent/sibling/spouse/off-spring with the patient.
3. Relatives are the primary caregivers identified as the family member who provides the most support and/or assistance.
4. Informed consent is given.

Exclusion criteria:

1. Relatives of patients having primarily substance related disorders.
2. Family members who could not speak English or Assamese.
3. Family members who refused to participate.

Mode of selection:

Subjects were selected using serial sampling procedure i.e. one relative each of continuously admitted patients, fulfilling the inclusion criteria.

Time of study

The study was carried out from December 2005 to July 2006.

Tools

1. Each subject was administered the scale, "Opinion about Mental Illness" constructed by Cohen and Struening[16] quoted from Shaw *et al*.[17] This scale consists of 51 statements relating to various causes, methods of treatment, legal and social (rehabilitation) aspects of mental illness. The validity and reliability of the scale were established.[16,18] Agrawal[19] and Kurian[20] used it in Indian population. The scale was translated into Assamese by the authors.
2. Psychiatric diagnoses were carried out according to the criteria of the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10): Clinical descriptions and diagnostic guidelines for mental and behavioural disorders.[21]
3. Sociodemographic data were collected using a standard proforma for the study.

There are five divisions of the scale, namely authoritarianism, benevolence, mental hygiene, social restrictiveness, and interpersonal aetiology. Authoritarianism included the following statements: (a) nervous breakdowns usually result when people work too hard, (b) it is easy to recognise someone who once had a serious mental illness, (c) when a person has a problem or a worry, it is best not to think about it, but keep busy with more pleasant things, (d) there is something about mental patients that makes it easy to tell them from normal people, (e) people would not become mentally ill if they avoided bad thoughts, (f) people with mental illness should never be treated in the same hospital as people with physical illness, (g) mental illness is usually caused by some disease of the nervous system, (h) college professors are more likely to become mentally ill than are business men, (i) sometimes mental illness is punishment for bad deeds, (j) one of the main causes of mental illness is a lack of moral strength or will power.

Benevolence included the following statements: (a) mental illness is an illness like any other, (b) even though patients in mental hospitals behave in funny ways, it is wrong to laugh about them, (c) patients in mental hospitals are in many ways like children, (d) more tax money should be spent in the care and treatment of people with severe mental illness, (e) anyone who tries hard to better himself deserves the respect of others, (f) people who have been patients in a mental hospital will never be their old selves again, (g) many mental patients are capable of skilled labour, even though in some ways they are very disturbed mentally, (h) to become a patient in a mental hospital is to become a failure in life, (i) if a patient in a mental hospital attacks someone, he should be punished so he doesn't do it again, (j) every mental hospital should be surrounded by a high fence and guards, (k) the law should allow a woman to divorce her husband as soon as he has been confined in a mental hospital with a severe mental illness, (l) regardless of how you look at it, patients with severe mental illness are no longer really human, (m) our mental hospitals should be organised in a way that makes the patient feel as much as possible like he is living at home, (n) there is little that can be done for patients in a mental hospital except to see that they are comfortable and well fed.

Mental hygiene included the following

statements: (a) most patients in mental hospitals are not dangerous, (b) most patients are willing to work, (c) if our hospitals had enough well trained doctors, nurses, and aides, many of the patients would get well enough to live outside the hospital, (d) our mental hospitals seem more like prisons than like places where mentally ill people can be cared for, (e) the best way to handle patients in mental hospitals is to keep them behind locked doors, (f) the patients of mental hospitals should be allowed more privacy, (g) people (both veterans and non-veterans) who are unable to work because of mental illness should receive money for living expenses, (h) many people who have never been patients in a mental hospital are more mentally ill than many hospitalised mental patients, (i) many mental patients would remain in the hospital until they were well, even if the doors were unlocked.

Social restrictiveness included the following statements: (a) although patients discharged from mental hospitals may seem alright, they should not be allowed to marry, (b) people who are mentally ill let their emotions control them: normal people think things out, (c) people who were once patients in mental hospitals are no more dangerous than the average citizen, (d) the small children of patients in mental hospitals should not be allowed to visit them, (e) a woman would be foolish to marry a man who has had a severe mental illness, even though he seems fully recovered, (f) anyone who is in a hospital for a mental illness should not be allowed to vote, (g) most women who were once patients in a mental hospital could be trusted as baby sitters, (h) most patients in mental hospitals don't care how they look, (i) although some mental patients seem alright, it is dangerous to forget for a moment that they are mentally ill, (j) all patients in mental hospitals should be prevented from having children by a painless operation.

Interpersonal aetiology included the following statements: (a) if parents loved their children more, there would be less mental illness, (b) although they usually aren't aware of it, many people become mentally ill to avoid the difficult problems of everyday life, (c) people who are successful in their work seldom become mentally ill, (d) mental patients come from homes where the parents took little interest in their children, (e) if the children of the mentally ill parents were raised by normal parents, they would probably not become mentally ill, (f) the mental illness of many people is caused by the separation or divorce of their parents during childhood, (g) if the children of normal parents were raised by mentally ill parents, they would probably become mentally ill.

Interview procedure

Aim of the study was explained to each subject and proper instructions were given. The statements were read out before each of them individually. Subjects used to choose any one of the following, "agree", "disagree" and "nut sure" to express views for each statement.

Analysis of data

A cross-sectional study design was used. Depending on the nature of the question, the statements were divided into two broad areas, namely 'area A' concerning "causes and nature of mental illness" (include statement numbers 1-9, 15-17, 20, 24, 25, 30, 32, 35, 39, 40, 42-45, 48 and 51) and 'area B' related to "humane treatment for the mentally ill and optimism regarding the outcome and need for expanding services for mentally ill" (include statement numbers 12, 13, 18, 21-23, 26-28, 31, 33, 34, 36, 38, 46, 47 and 49). The division into 'area A' and 'area B' is in accordance to Agrawal. [19]

Statement number 19 was ambiguous in its meaning and hence omitted at the time of analysis.

Further, statement numbers implicating "authoritarianism" were 1, 6, 9, 11, 16, 21, 39, 43, 46 and 48; that of "benevolence" were 2, 12, 17, 18, 22, 26, 27, 32, 34, 36, 37, 40, 47 and 49; indicating "mental hygiene" were 3, 13, 23, 28, 31, 33, 38, 44 and 50; of "social restrictiveness" were 4, 7, 8, 14, 24, 29, 41, 42, 45 and 51; as well as for "interpersonal aetiology" were 5, 10, 15, 20, 25, 30 and 35. The division of authoritarianism, benevolence, mental hygiene, social restrictiveness, and interpersonal aetiology were present originally in the scale.[16]

Three categories were employed to measure the degree of information, according to Agrawal[19]:

Very well informed – 80% correct answers or above.

Adequately informed – 50-79% correct answers.

Poorly informed – Below 50% correct answers.

Cross-tabulations were done systematically between all questions and sociodemographic variables, and the chi-square test was applied.

Results

The present study reveals that the degree of information concerning mental illness among the relatives of patients admitted in the hospital was fairly adequate; however, when their degree of information concerning to nature and aetiology of mental illness was considered, it was found that they lack significantly in their information (1% very well informed, 66% adequately informed and 33% poorly informed); but, their knowledge about humane treatment for the mentally ill and optimism regarding the outcome and need for expanding services for mentally ill (including rehabilitation) was better than the former (14% very well informed, 83% adequately informed and 3% poorly informed) [chi-square: 38.206, degrees of freedom: 2, the p value is <0.0001 (statistically significant)] (table 1, Figure 1).

Table-1: Degree of information in area A and area B

	Very well informed	Adequately informed	Poorly informed
Area A	1	66	33
Area B	14	83	3

Chi-square: 38.206, degrees of freedom: 2, the p value is <0.0001 (statistically significant).

Attitude towards authoritarianism was negative (3% subjects were very well informed, 33% were adequately informed and 64% poorly informed); attitude towards benevolence was positive (19% subjects were very well informed, 80% were adequately informed and 1% poorly informed); attitude towards mental hygiene was positive (19% subjects were very well informed, 67% were adequately informed and 14% poorly informed); attitude towards social restrictiveness was negative (9% subjects were very well informed, 44% were adequately informed and 47% poorly informed); and attitude towards interpersonal aetiology was negative (9% subjects were very well informed, 37% were adequately informed and 54% poorly informed) [chi-square: 130.07, degrees of freedom: 8, the p value is <0.0001 (statistically significant)] (Table 2, Figure 2).

Table-2: Degree of information in authoritarianism, benevolence, mental hygiene, social restrictiveness and interpersonal aetiology

	Very well informed	Adequately informed	Poorly informed
Authoritarianism	3	33	64
Benevolence	19	80	1
Mental hygiene	19	67	14
Social restrictiveness	9	44	47
Interpersonal aetiology	9	37	54

Chi-square: 130.07, degrees of freedom: 8, the p value is <0.0001 (statistically significant)

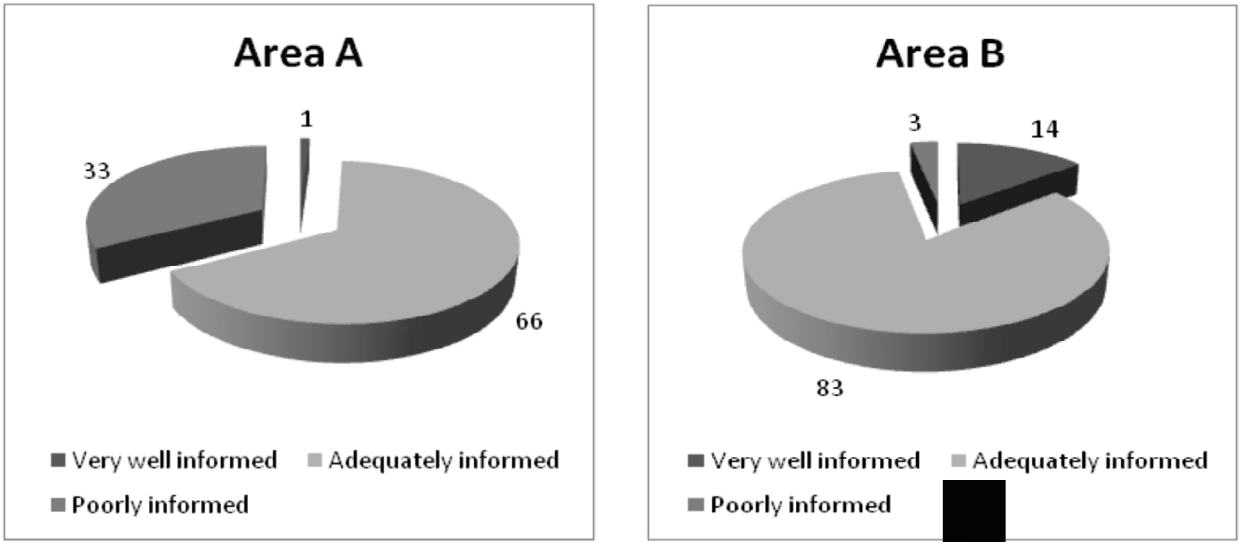

Figure 1 Degree of information in area A and area B

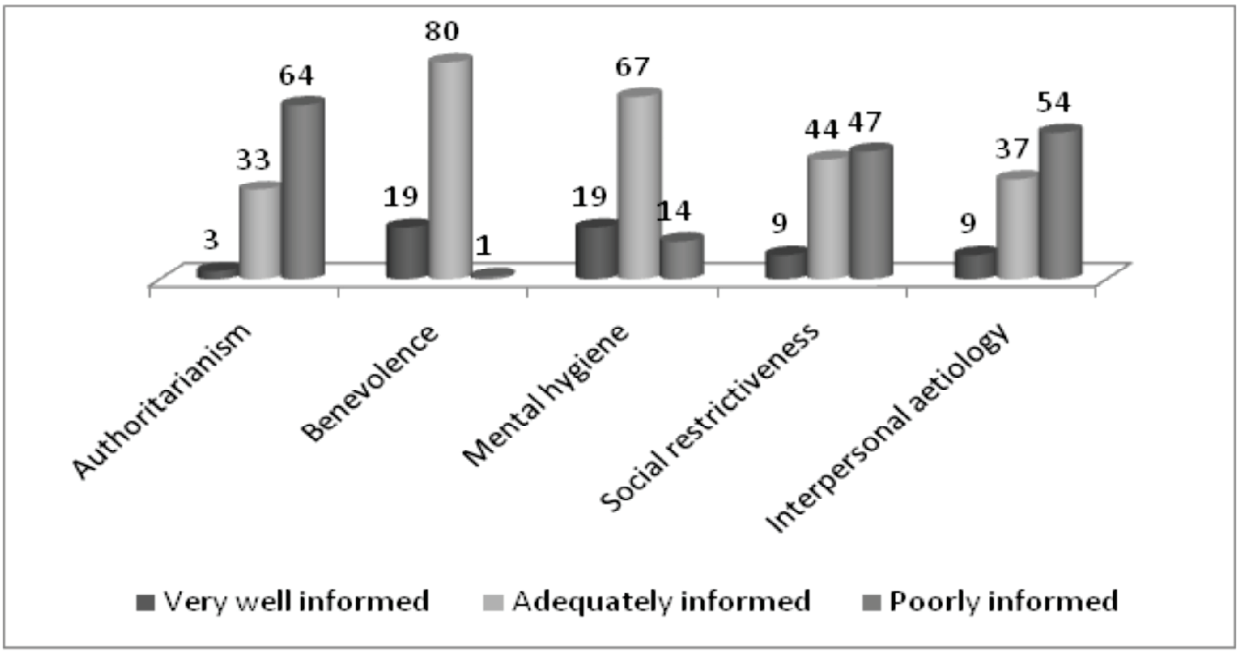

Figure-2: Degree of information in authoritarianism, benevolence, mental hygiene, social restrictiveness and interpersonal aetiology

Discussion

'Area A' is concerning "causes and nature of mental illness" and 'area B' is related to "humane treatment for the mentally ill and optimism regarding the outcome and need for expanding services for mentally ill (including rehabilitation)". Present study showed that knowledge in 'area A' was poor while the same in 'area B' was adequate. Moreover, subjects exhibited negative attitude towards authoritarianism, social restrictiveness and interpersonal aetiology. However, positive attitude was exhibited towards benevolence and mental hygiene.

This finding could be because of the fact that relatives of the patients are more concerned about the treatment and outcome of their wards rather than knowing about the aetiology and pathology. In addition, they are also exposed more to various modalities of the treatment in such institutions.

This is in confirmation with the findings of Agrawal.[19] Most families (76%) reported having no knowledge about the illness.[22] Poor knowledge of causation was common.[23]

Stip et al.[24] found that 70% of the people surveyed feel that they had inaccurate information regarding mentally ill people; this was alluded to in the report by the respondents identifying "lack of will" as the main cause of mental illness. Negative attitudes to mental illness may be fuelled by notions of causation that suggest that affected people are in some way responsible for their illness, and by fear.[23]

Craft[25] found that 72 percent agree that "virtually anyone can become mentally ill". There were favourable responses on statements regarding value of life, family life, decision-making ability, and the management and care of mental illness.[26]

Craft[25] found that most South Carolinians, 87%, do not believe in locking up people with mental illness. Many relatives experience considerable distress, but the evidence does not suggest that they avoided frequent contact with the patient as a consequence.[27]

Magliano et al[28] showed that relatives' pessimistic opinion on social restrictions imposed by schizophrenia were found to be associated with high levels of disability and high numbers of hospital admissions in the patient and old age in the relative. Lay respondents' opinions on patients' civil rights and social competence tended to be more similar to those expressed by professionals than to those reported by relatives.[29]

Findings revealed, among others, the widespread belief in "supernatural" causation of

mental illness in patients/relatives.[30] Biological causes were relatively rarely mentioned – mostly in response to suggestions of the interviewer.[31]

In contrast to results of the present study, Angermeyer et al.[32] found that relatives would usually look to biological factors when searching for the cause of schizophrenia. Stuart et al.[33] found that of those able to identify a cause of schizophrenia (two-thirds), most identified a biological cause, usually a brain disease.

It has rightly been pointed out that the beliefs in supernatural causes of mental disorders are more widely held and traditional sources of help, such as spiritual healers, are preferred over medical advice for a range of mental health problems in developing countries.[34] About 72% of the tribals believed that insanity is the only cause of crime and pilgrimage can cure mental illnesses.[31]

Weisman et al.[36] found that most participants accepted the patients as having a legitimate illness. It seems that after psychiatric training, students expressed less authoritarian views and their opinion toward social integration of mentally ill persons was found to be more positive.[37]

Brewin[38] found that reductions in criticism were not related to attributional change, but reductions in hostility were associated with shifts towards more universal and uncontrollable attributions. Rural tribals believe that persons who have once been insane should never be entrusted with a responsible job.[35]

Magliano et al.[39] showed that relatives seemed to believe that mental hospitals are not appropriate places in which to treat their loved ones, even though most of them felt that they were alone in coping with the burden of their ill relative's disease. 72.2% of the sample held the opinion that people with schizophrenia are dangerous and 91.8% believed that these patients could not take responsibility for their own lives.[40]

Most of the relatives agreed that people who have schizophrenia should be allowed to vote, but they appeared to have a stricter attitude toward affective rights, such as the patient's right to get married and have children, and tended to perceive their loved ones as being subject to social discrimination.[39] More than 80% allowed the mentally ill client to attend social gatherings or visit public places. While two thirds did not advocate marriage as a cure for mental illness, 25% objected to marrying family members of a mentally ill person for fear of social stigma.[41]

Angermeyer et al.[42] found that while patients, like professional experts, endorsed a multifactorial aetiological concept, they clearly favoured psychosocial explanations over biological ones. The major changes were an increase in belief in genetic causes of depression and schizophrenia, increases in beliefs about problems from childhood and the death of someone close as causes of depression, and a decrease in the belief that "weakness of character" is a cause of schizophrenia.[43]

Vijayalakshmi and Math[3] aimed to investigate gender differences in mental health literacy of family caregivers of persons with mental illness. A cross-sectional descriptive survey was carried out among 161 randomly selected caregivers of persons with mental illness at outpatient department of a tertiary care centre. Data were collected through face-to-face interview using a structured questionnaire. This study supports the previous research that gender differences exist in mental health literacy of persons with mental illness. Men were less literate than women regarding knowledge of people with mental illness as well women hold better positive attitudes towards persons with mental illness. Hence, gender differences need to be considered in developing mental health literacy programmes for family caregivers of mentally ill and general population.

Vijayalakshmi et al.[2] assessed the attitudes of rural population towards persons with mental illness. It was a cross sectional descriptive study carried out in a rural community. A consecutive (n=102) sample was recruited by house-to-house survey. Findings revealed that participants held more stigmatising attitudes towards person with mental illness. It also revealed that community was more authoritarian (32.3 ±3.18) and socially restrictive views (31.9 ±3.25). However, they also held relatively more benevolent (29.1±3.51) and tolerant attitudes (31.8 ±2.69) towards community based mental health ideology. The findings of this study revealed that negative attitudes towards persons with mental illness are widespread and may impair their social reintegration into the community. Hence, there is an urgent need to develop strategies to enlighten the public regarding nature of mental illness to foster acceptance of people with mental illness by the rural community.

Aims of the study carried out by Kumar et al.[1] were (1) to assess the attitude towards mental illness of key informant of patients and general population, and (2) to compare the two groups in respect to attitude towards mental illness. Sample based on purposive sampling technique, consisting of 200 subjects (100 key informants and 100 from general population) within age range of 25-55 years had been taken. Sample of key informants was taken from Ranchi Institute of Neuro-Psychiatry & Allied Sciences (RINPAS) outpatients' department whereas the sample of general population was taken from Kanke area or within the radius of 5 K.M. from RINPAS, Kanke. Significant difference was found in the area of nature, cause, after effect and community mental health ideology between both groups. There was no significant difference in the area of treatment and stigma. The findings of this study suggested that there was growing awareness about mental illness even in general population and the people were being more receptive of the mentally ill people.

Limitations

A major limitation of the study was that like any other questionnaire based survey, the findings may reflect the 'social desirability bias' rather than actual practice. The fact that family members were not assessed for the presence of psychopathology was a limitation of this study.[44] We only studied patients in public hospital. The sample was not representative of all families of mentally ill patients. Lack of a control group limited from making comment on whether contact with patient influences K.A.P.B. Nonavailability of data about previous hospitalisations or psychiatric treatment hindered assessing role of prior medical care in K.A.P.B.

In conclusion, a key suggestion is for educational interventions aiming specific target groups, with prior identification of their attitudes. Family is a target group, the starting point for all target groups and at every level is education.[4] The results of this study underlined the need for educational programmes for the relatives of patients.[40]

Research suggests that one of the most effective ways to positively affect attitudes is to deliver relevant messages that will resonate with target audiences, encourage the public to recognise, acknowledge and disclose their own problems or those of family members, and provide information that will help the audience to access help.[9] The findings of this study may be useful for developing psychoeducational intervention and sensitisation campaigns.[39]

Media coverage of these interventions will be essential to disseminate positive mental health messages, while challenging current misinterpretations.[4] Further programmes must be developed which reach out to diverse communities and tailored to their specific needs.[9] Only these concerted efforts will eventually dispel the stigma associated with mental illness.[45]

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APPENDIX
Opinions about Mental Illness

1. Nervous breakdowns usually result when people work too hard. – Agree / Disagree / Not sure.
2. Mental illness is an illness like any other. – Agree / Disagree / Not sure.
3. Most patients in mental hospitals are not dangerous. – Agree / Disagree / Not sure.
4. Although patients discharged from mental hospitals may seem all right, they should not be allowed to marry. – Agree / Disagree / Not sure.
5. If parents loved their children more, there would be less mental illness. – Agree / Disagree / Not sure.
6. It is easy to recognise someone who once had a serious mental illness. – Agree / Disagree / Not sure.
7. People who are mentally ill let their emotions control them: normal people think things out. – Agree / Disagree / Not sure.
8. People who were once patients in mental hospitals are no more dangerous than the average citizen. – Agree / Disagree / Not sure.
9. When a person has a problem or a worry, it is best not to think about it, but keep busy with more pleasant things. – Agree / Disagree / Not sure.
10. Although they usually aren't aware of it, many people become mentally ill to avoid the difficult problems of everyday life. – Agree / Disagree / Not sure.
11. There is something about mental patients that makes it easy to tell them from normal people. – Agree / Disagree / Not sure.
12. Even though patients in mental hospitals behave in funny ways, it is wrong to laugh about them. – Agree / Disagree / Not sure.
13. Most mental patients are willing to work. – Agree / Disagree / Not sure.
14. The small children of patients in mental hospitals should not be allowed to visit them. – Agree / Disagree / Not sure.
15. People who are successful in their work seldom become mentally ill. – Agree / Disagree / Not sure.
16. People would not become mentally ill if they avoided bad thoughts. – Agree / Disagree / Not sure.
17. Patients in mental hospitals are in many ways like children. – Agree / Disagree / Not sure.
18. More tax money should be spent in the care and treatment of people with severe mental illness. – Agree / Disagree / Not sure.
19. A heart patient has just one thing wrong with him, while a mentally ill person is completely different from other patients. – Agree / Disagree / Not sure.
20. Mental patients come from homes where the parents took little interest in their children. – Agree / Disagree / Not sure.
21. People with mental illness should never be treated in the same hospital as people with physical illness. – Agree / Disagree / Not sure.
22. Anyone who tries hard to better himself deserves the respect of others. – Agree / Disagree / Not sure.
23. If our hospitals had enough well trained doctors, nurses, and aides, many of the patients would get well enough to live outside the hospital. – Agree / Disagree / Not sure.
24. A woman would be foolish to marry a man who has had a severe mental illness, even though he seems fully recovered. – Agree / Disagree / Not sure.
25. If the children of mentally ill parents were raised by normal parents, they would probably not become mentally ill. – Agree / Disagree / Not sure.
26. People who have been patients in a mental hospital will never be their old selves again. – Agree / Disagree / Not sure.
27. Many mental patients are capable of skilled labour, even though in some ways they are very disturbed mentally. – Agree / Disagree / Not sure.
28. Our mental hospitals seem more like prisons than like places where mentally ill people can be cared for. – Agree / Disagree / Not sure.
29. Anyone who is in a hospital for a mental illness should not be allowed to vote. – Agree / Disagree / Not sure.
30. The mental illness of many people is caused by

the separation or divorce of their parents during childhood. – Agree / Disagree / Not sure.

31. The best way to handle patients in mental hospitals is to keep them behind locked doors. – Agree / Disagree / Not sure.
32. To become a patient in a mental hospital is to become a failure in life. – Agree / Disagree / Not sure.
33. The patients of mental hospitals should be allowed more privacy. – Agree / Disagree / Not sure.
34. If a patient in a mental hospital attacks someone, he should be punished so he doesn't do it again. – Agree / Disagree / Not sure.
35. If the children of normal parents were raised by mentally ill parents, they would probably become mentally ill. – Agree / Disagree / Not sure.
36. Every mental hospital should be surrounded by a high fence and guards. – Agree / Disagree / Not sure.
37. The law should allow a woman to divorce her husband as soon as he has been confined in a mental hospital with a severe mental illness. – Agree / Disagree / Not sure.
38. People (both veterans and non-veterans) who are unable to work because of mental illness should receive money for living expenses. – Agree / Disagree / Not sure.
39. Mental illness is usually caused by some disease of the nervous system. – Agree / Disagree / Not sure.
40. Regardless of how you look at it, patients with severe mental illness are no longer really human. – Agree / Disagree / Not sure.
41. Most women who were once patients in a mental hospital could be trusted as baby sitters. – Agree / Disagree / Not sure.
42. Most patients in mental hospitals don't care how they look. – Agree / Disagree / Not sure.
43. College professors are more likely to become mentally ill than are business men. – Agree / Disagree / Not sure.
44. Many people who have never been patients in a mental hospital are more mentally ill than many hospitalised mental patients. – Agree / Disagree / Not sure.
45. Although some mental patients seem all right, it is dangerous to forget for a moment that they are mentally ill. – Agree / Disagree / Not sure.
46. Sometimes mental illness is punishment for bad deeds. – Agree / Disagree / Not sure.
47. Our mental hospitals should be organised in a way that makes the patient feel as much as possible like he is living at home. – Agree / Disagree / Not sure.
48. One of the main causes of mental illness is a lack of moral strength or will power. – Agree / Disagree / Not sure.
49. There is little that can be done for patients in a mental hospital except to see that they are comfortable and well fed. – Agree / Disagree / Not sure.
50. Many mental patients would remain in the hospital until they were well, even if the doors were unlocked. – Agree / Disagree / Not sure.
51. All patients in mental hospitals should be prevented from having children by a painless operation. – Agree / Disagree / Not sure.

Original Article

Prevalence of Mild Cognitive Impairment among Hospital Patients aged 65 and above

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Abstract

Background: *As per WHO estimates, the population of elderly is expected to increase several folds in the coming years and hence increase in the age related problems like the cognitive decline. However only few population based studies have investigated prevalence of Mild Cognitive Impairment in Indian population.* ***Material & Methods:*** *The present study is designed to know the extent of problem of MCI among elderly patients attending the general hospital. A crossectional study was done in elderly attending various clinics in Rajindra hospital, Patiala. Only the patients aged 65 and above who gave informed consent were included in the study. They were assessed on their sociodemographic profile and screening for cognitive functions was done using Saint Louis University Mental Status Examination. The cutoff points for SLUMS for MCI were 20-26 for those educated high school and above and 20-24 for those educated less than high school.* ***Results:*** *The prevalence for MCI based on SLUMS score was found out to be 31.53%. MCI was significantly associated with education and type of living arrangement ($p \leq 0.05$). The study also highlights the fact that as the age advances, there is shift from MCI towards Dementia.*

Keywords: *Elderly, Mild cognitive impairment, Memory*

Introduction

Life expectancy is increasing as a result of advances in medical science and the availability of better healthcare services; the proportion of elderly persons in the general population is therefore rising.[1,2] With the increasing number of older adults, there is a growing interest in improving quality of life in old age.

The concept of MCI has evolved considerably over the years. The first attempt to characterize cognitive changes dates back to 1962, when Kral[3] used the term "benign senescent forgetfulness" to describe early memory concerns with aging. It was followed by a National Institute of Mental Health workgroup in 1986 that proposed the term "age-associated memory impairment" (AAMI) to refer to memory changes that occurred as a result of normal aging process. Then The International Psycho- geriatric Association coined the term "age-associated cognitive decline".[4] The term "cognitive impairment-no dementia"(CIND) was coined by the Canadian Study of Health and Aging to describe the presence of nondemented cognitive impairment regardless of the underlying process (neurologic, psychiatric, medical).[5]

The term MCI (mild cognitive impairment) was introduced in 1988 by Reisberg and colleagues to describe individuals with a Global Deterioration Scale (GDS) rating of 3.[6] Another classification has used the Clinical Dementia Rating Scale (CDR) to identify individuals with CDR 0.5 stage of "questionable dementia.[7,8] The criteria proposed by the Mayo Clinic Alzheimer's Disease Research Center (MCADRC criteria) are: Memory complaint,

preferably qualified by an informant; objective memory impairment for age and education; preserved general cognitive function; intact activities of daily living; not demented.[9] MCI has been proposed as transition between normal, age associated cognitive change and early dementia.[10]

Petersen et al proposed three different MCI subtypes (Amnestic; Multiple-Domain; and Single Non-Memory Domain MCI) to describe possible subtypes of cognitive impairment and their possible differing etiologies and outcomes.[11] There is an evolving consensus that the patient with MCI is neither normal nor demented and has subjective cognitive complaints and objective evidence of cognitive deficits.

Although majority of cases of dementia are attributable to Alzheimer's disease, it is important to recognize that MCI does not predict the outcome of Alzheimer's disease alone. Rather MCI is the precursor to several types of dementia, including neurodegenerative dementia (e.g. Alzheimer's disease, Parkinson disease, Frontotemporal dementia, dementia with Lewy bodies); vascular dementia and dementia caused by neoplasm's, normal pressure hydrocephalus, metabolic factors and psychiatric factors.[12]

The need of the hour is that persons with MCI should be picked up early to prevent progression to full blown dementia. The present study was designed to know the extent of MCI among the elderly patients attending a general hospital. Early detection of MCI is important as prompt treatment can delay the progression of MCI to dementia.[11]

Materials and methods

After getting permission from the ethical committee, this descriptive, cross-sectional study was carried out on elderly patients aged 65 and above attending the various clinics in Rajindra Hospital, Patiala. The patients were selected through convenient sampling method from the following 4 different OPDs (25 patients each were taken from Psychiatry, Medicine, Neurology and Surgery). The patients who gave informed consent were enrolled in the study. The patients with Delirium, Schizophrenia, Mental Retardation, and Bipolar Disorder were excluded. The patients with severe hearing and visual defects were also excluded from the study. Detailed history was taken and the physical/mental status examination done. The patient's identification data including sociodemographic profile were recorded. The Saint Louis University Mental Status Examination (SLUMS) was applied on all patients to screen for mild cognitive impairment. The subjects picked up MCI on SLUMS were further evaluated.

The screening instrument used in this study was the validated education adjusted scale. The SLUMS has a sensitivity of 98% and a specificity of 100% when adjustments are made for education level. It measures aspects of cognition that include orientation, short-term memory, calculations, delayed recall ,digit span (attention), verbal fluency, executive function, visuospatial function, and recall of items from a story.

Results

Ninety two (92) out of 100 patients fulfilled the inclusion criteria and were enrolled in the study. The patients were divided into three groups depending upon the results of the screening method. Twenty nine patients (31.53%) had MCI, 37 (40.21%) had dementia and rest fall into the category of Normal cognition (n=26, 28.26%). Table 1 shows the sociodemographic profile of MCI patients. Mostly patients of MCI fall in the age group of 65-69

Table-1: Socio-demographic profile of MCI subjects

Variable	MCI	
Age group	**Number of subjects (n)**	**Percentage**
65–69	13	14.13%
70–74	10	10.87%
75–79	3	3.26%
80–84	2	2.17%
85 and above	1	1.09%
Gender		
Males	20	21.74%
Females	9	9.78%
Marital Status		
Married	24	26.09%
Unmarried/divorcee/ Widower	5	5.43%
Education		
Less than high school	18	19.57%
High school and above	11	11.96%
Living Arrangement		
Living with family	25	27.17%
Living alone	4	4.35%

followed by age group of 70-74 and thereafter there is subsequent decrease in number of subjects with age. Majority of subjects were males. There were 21.74 % (n=20) males and 9.78% females (n=9). Mostly they were married and living with their family. Most of the subjects in the MCI group had education less than high school (19.57 %) and 11.96% of the subjects who had MCI had education level of high school and above.

Table 2 shows that the only significant relationship among the sociodemographic factors was between MCI and educational level and type of living arrangement.($p<0.05$)

age advances. The X^2 gives a statistically significant value ($X^2=11.39$, $p<0.01$).

Table 4 shows the distribution of various subtypes of MCI. In this study most prevalent subtype was Amnestic MCI (multiple domain) followed by Non- amnestic MCI and least common was pure Amnestic MCI (single domain).

Discussion

The prevalence of MCI among the elderly patients attending the hospital was found out to be 31.53%. The present results support the previous hospital based study done in Deutschland where

Table-2: The comparison between various socio-demographic variables in MCI subjects

Variable	Comparison Group	Group 1		Group 2		
		Mean1	SD1	Mean2	SD2	t-test
Age Group	65-75 v/s 76 & Above	22.04	1.86	23.75	2.63	1.62
Gender	Males v/s females	22.55	2.19	22.11	1.83	0.52
Marital Status	Married v/s Widowed/Unmarried/Divorced	22.33	2.12	22.80	1.92	0.45
Education	Less Than High School v/s High School and above	21.50	1.38	24.00	2.05	**3.93****
Living Arrangement	Living With Family v/s Living Alone	25.16	2.08	23.00	2.16	**3.32****

**$p \leq 0.01$

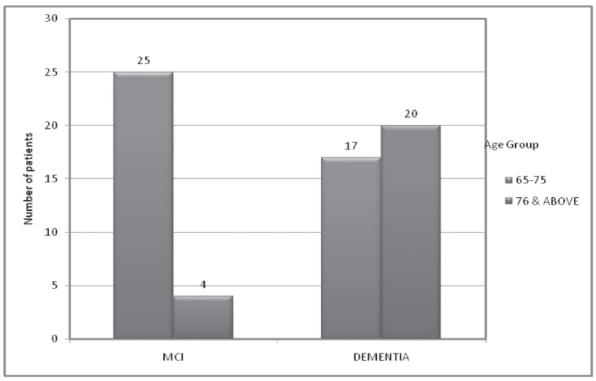
Table-3: The comparison of MCI and Dementia in different age groups

Age Group	MCI (number of subjects)	Dementia (number of subjects)	χ^2	p-value
65-75	25	17	**11.39****	0.01**
76& Above	4	20		

Table-4: Distribution of subjects into various subtypes of MCI

Type of MCI	N	%age
Amnestic MCI(single domain)	1	1.09%
Amnestic MCI (multiple domain)	24	26.09%
Non Amnestic MCI	4	4.35%
Total	29	31.53%

The chi square test was used for comparing the MCI and Dementia in different age groups (Table 3). The results showed that MCI patients fall maximum in the age group of 65-75 and decrease thereafter. Whereas the number of patients of Dementia are lesser in 65-75 and increase as the



prevalence was 36.1%.[13] However it was lesser than another clinic based Malaysian study where prevalence found out was 64.7%.[14] The possible variation in results could be due to the fact that the above mentioned study included the subjects in the age group of 60 and above but the subjects in the present study were in the age group 65 and above.

The socio-demographic factors significantly associated with MCI were the elderly patients' level of education and the type of living arrangement. the results showed significant association ($p \leq 0.05$) between lower level of education and MCI (as shown in Table) which was consistent with previous

studies which showed that low level of education tended to be associated with more cognitive impairment.[14-16] There was statistically significant difference in the scores of MCI group compared with normal in the same education group. Even after standardizing for education (education adjusted scale used), the statistically significant difference lies between the scores of MCI subjects who had education less than high school compared with the group with education of high school and above ($p \leq 0.01$) thereby suggesting that there is more cognitive impairment in the MCI group with lesser education as they scored lesser. The Mayo clinic study by Peterson et al[15] in Olmsted county supported that MCI prevalence decreased with increasing number of years of education. The clinic based Malaysian study also found out the patients with low educational achievement had a high likelihood of having MCI and those with a higher level of education were more likely to be 'normal' ($p < 0.05$). The population based longitudinal research by Tervo et al[16]. studied various risk factors for MCI and found out that high education is a protective factor for MCI.

Although majority of the MCI patients lived with their families but still there was statistically significant difference in the scores achieved by the subjects living with families compared to subjects living alone (shown in Table 2) thereby indicating that persons who live alone tend to have higher cognitive impairment ($p \leq 0.01$).This is in contrast with the Malaysian study which states that there is no association between living arrangement and cognitive impairment[14]. The possible reason could be deprivation of environmental stimuli and lack of family support.

Among the various subtypes, the Amnestic MCI was prevalent than the Non Amnestic MCI. Out of various subtypes, Amnestic MCI-multiple domain (26.09%) was the most prevalent followed by Non Amnestic MCI (4.35%) and least common being the pure Amnestic MCI-single domain (1.09%). This is in agreement with the previous study. This is being supported by the study done in Kolkata showing the prevalence of Amnestic MCI -multiple domain to be highest (8.85%).[17]

Limitations

Despite the study being a unique effort in this part of the country to find out the prevalence of MCI among the hospital subjects, it is not short of limitations. The sample size was small compared to previous hospital based research due to the limitations imposed by the time frame of the study. The present study is not reflective of MCI among the elderly in the community as this study was conducted among elderly patients in a tertiary level teaching hospital and patients were selected by convenient sampling method. The SLUMS is screening instrument and not a diagnostic test for MCI which needs to be further evaluated. The non availability of higher test techniques like fMRI, SPECT, biomarker and genetic marker study for MCI was a limitation in our hospital setting.

Conclusions

The study shows that the prevalence of mild cognitive impairment among elderly attending the hospital is high and might represent important health burden because subjects with MCI are at greater risk of developing Dementia as the age increases. A significant association was found between patients' level of education with MCI whereby patients with lower educational level tend to have a higher mild cognitive impairment. Also persons living alone tend to have higher cognitive impairment than in those living with their family.

Acknowledgement

The authors would like to thank all the participants, the doctors and attending staff of the different clinics for their cooperation and support towards this research project.

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Original Article

Self-concept, Stigma and Quality of Life in Chronic Schizophrenia and Chronic Skin Disorders: A Comparative Study

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Abstract

Background: *Often course and outcome of schizophrenia and various forms of chronic skin diseases are influenced by psychosocial factors. Factor like psychosocial stress has often been seen to exacerbate the illness. Chronic schizophrenia and chronic skin diseases generally have a major impact on patient's daily activities, emotional state and social relationships. Factors like one's own assessment or evaluation about his/her self-especially his/her self efficacy or esteem, self-image or abilities.* ***Aim and objective:*** *To assess the self-concept, stigma and quality of life in patients with chronic schizophrenia as compared to chronic skin disease patients.* ***Methods:*** *This study was a cross-sectional hospital based study and it was carried out at the Central Institute of Psychiatry, Ranchi, Jharkhand. For the purpose of study 30 patients having the diagnosis of schizophrenia and 30 patients of chronic skin diseases were selected purposively. For data collection Socio-Demographic and Clinical Data Sheet, Self-Concept-Questionnaire Stigma Interview Schedule and WHO-QOL-100 were used.* ***Results:*** *Significant differences were seen between the patients of chronic schizophrenia and chronic skin disease in the 'social', 'moral' domains of Self Concept Questionnaire, perceived stigma as well as two domains of quality of life measuring instrument (WHOQOL-100), e.g. 'psychological' and 'independence'. The details related to findings of the study will be shared at the time of presentation.* ***Conclusion:*** *Schizophrenia patients were found to have lower level of self-concept, and quality of life as well as higher level of stigma than the patients with chronic skin diseases.[1-3]*

Key words: *Self-concept, Stigma, Quality of life, Schizophrenia*

Introduction

Psychosocial factors are as important as biological or somatic factors in the onset as well as persistence of some chronic physical disorders like skin disorders. It is not that only core psychiatric disorders like schizophrenia will be affected by the psychosocial factors but skin disorders may also have some relationship with those factors. Many patients of chronic schizophrenia and chronic skin disease are influenced by psychosocial factors. They are associated with both psychosocial stress, which exacerbates the condition, and psychological and body image problems. Chronic schizophrenia and chronic skin diseases generally have a major impact on patient's daily activities, emotional state and social relationships. Factors like one's own assessment or evaluation about his/her self- especially his/her self efficacy or esteem, self-image or abilities.[1-3] Due to illness these areas are gravely affected and the patients start to devalue them while comparing them with others.[4,5] Root causes may be these people begin keeping themselves away from others or due to misconceptions society puts a label to them as 'ill

persons', 'weak', or 'incapacitated', so on. Both the affected persons and the society starts developing stigma about the illness. Here patients tend to develop self-stigmatizing behaviour and try to keep away from the main flow of the society, whereas society stigmatizes them by either pouring unwarranted sympathy to them or segregating them from others.[6-8] Patients having illnesses like schizophrenia and chronic dermato-logical disorders are at risk of experiencing these antagonisms from the society. In recent times it has been explored that some physical disorders do have psychosocial components, which may affect the course, prognosis and the management of these illnesses. Psychosocial factors like self concept, stigma and quality of life have become quite prominent in some chronic skin ailments like Psoriasis, Vitilgo and Leprosy.

Aim of the Study

The present study was designed to assess the self-concept, stigma and quality of life in patients with chronic schizophrenia as compared to chronic skin disease patients.

Materials and Methods

This study was a cross-sectional hospital based one and carried out at the Central Institute of Psychiatry, Ranchi, Jharkhand. For the purpose of study 30 patients having the diagnosis of schizophrenia and 30 patients of chronic skin diseases (e.g., Vitilgo, Psoriasis, Eczema and Non-Leprotomous diseases) as per dermatology consultant at CIP dermatology clinic were selected purposively as per the inclusion and exclusion criterion of the study. After selection of the samples instruments like Socio-Demographic and Clinical Data Sheet, Self-Concept-Questionnaire, Stigma Interview Schedule and WHO-QOL-100[3] were used for data collection. For measuring psychopathology of the schizophrenic patients Scale for Assessment of Positive Symptoms (SAPS) and Scale for Assessment of Negative Symptoms (SANS) were used.[9,10]

Self Concept Questionnaire (SCQ)[11] was applied for assessing the self-concept of the patients of schizophrenia and chronic skin disease. The test-retest reliability of this scale is found to be high. In various dimensions the test-retest reliability ranges from 0.67 to 0.88. Though there are many scales available for assessing self concept, most of these scales are translated from other languages, so there could be some discrepancies between the original scale and the translated versions. This scale has been standardized over the Indian population. This scale provides six separate dimensions of self concept (Physical, Social, Intellectual, Moral, Educational and Temperamental self concept). It also gives a total self-concept score. This scale requires 20 minutes for application.

The Stigma Interview Schedule Scale (Wahl, 1999)[12] was applied for assessing the stigma of the patients of schizophrenia and chronic skin disease. This scale was developed by Otto F. Wahl to measure the subjective version of stigma. Items of this scale were standardized on 1,301 mental health consumers in USA. This scale has two broad sections i.e., 'stigma section' and 'discrimination section'. This scale includes nine questions about people's interpersonal experiences as "consumers." Questions addressed such topics as specific treatment by others (e.g., being shunned or rejected), negative things seen or heard about illness (e.g., in mass media), and fears and behaviours related to disclosure of status as consumers. Respondents were asked to rate the extent of each experience on a 5-point scale from "never" to "very often". But in present study, only the 'stigma section' was applied to measure the level of subjective stigma of the samples of either group.

The WHO Quality of Life Scale (WHOQOL-100) was applied for assessing quality of life in schizophrenia and chronic skin disease. Today this scale is available in 40 languages worldwide. This scale gives emphasis on the subjective evaluation of the respondent's health, living conditions and functioning, rather than on their objective life conditions. Evaluation is based on the situation during the two weeks prior to assessment. In China, Li et al,[13] used the WHO Quality of Life Questionnaire (WHOQOL-100) on 460 patients with chronic disease (including hypertension, schizophrenia, stroke, end-stage renal disease, head and neck cancer and breast cancer) and these authors compared the General Quality of Life Inventory (GQOLI) with WHO Quality of Life Questionnaire (WHOQOL-100). They found that the domains of the WHOQOL-100 were strongly correlated with the physical, psychological, social and environmental

dimensions of the GQOLI. Comparisons between patients with physical and mental disorders and with a healthy control group have been reported in the context of the Danish WHOQOL validation study. The test-retest relia-bility of the various dimensions of scale ranges from 0.87 to 0.95.

Written as well as explained informed consent was taken from the samples of either group before starting data collection. SANS and SAPS 10 were administered upon schizophrenia patients for the purpose of evaluating their present illness status. Patients who had obtained the score >2 were not included in the study. After filling up of the socio-demographic and clinical data sheet other instruments like Self -Concept Questionnaire, Stigma Interview Schedule and WHOQOL-100 were applied on them. Diagnosis of the each group was made by Consultant psychiatrist and dermatologist of the institute as per ICD-10.

Statistical measures like descriptive statistics, Chi-Square Test, Independent Sample T Test and Pearson Correlation-Coefficient were used to analyse and interpret the collected data. Statistical software of Statistical Package for Social Sciences (Windows Version 13) was used in this study.

Results

Table-1: Socio-demographic characteristics of chronic schizophrenia and chronic skin disease patients

Variables		Chronic Schizophrenia Patients (N=30) Mean ± SD /n (%)	Chronic Skin Disease Patients (N=30) Mean ± SD /n (%)	t/χ^2	df	P
Age		35.70 ± 10.00	34.30 ± 11.05	.514	58	.609
Education		10.36 ± 3.91	10.06 ± 4.37	.280	58	.780
Sex	Male	28 (92.4)	17(56.1)	10.75	1	.002**
	Female	2(7.6)	13(53.9)			
Marital Status	Married	16(52.8)	24(79.2)	5.6	2	.061
	Unmarried	12(39.6)	6(20.8)			
	Others	2(7.6)	0(0.0)			
Religion	Hindu	25(82.5)	21(69.3)	2.04	2	.361
	Muslim	5(17.5)	8(26.4)			
	Others	0(0.0)	1(3.3)			
Family Type	Joint	16(52.8)	12(39.6)	1.071	1	.438
	Nuclear	14(47.2)	18(60.4)			
Ethnicity	Tribal	0	1(3.7)	1.017	1	1.00
	Non-tribal	30(100.0)	29(96.7)			
Domicile	Rural	14(46.2)	7(23.1)	3.90	2	.142
	Semi-Urban	9(29.7)	15(50.0)			
	Urban	7(23.1)	8(26.9)			
Occupation	Employed	20(67.0)	17(56.1)	8.56	2	.014*
	Unemployed	9(29.7)	4(13.2)			
	Housewife	1(3.3)	9(29.7)			

* Significant at the 0.05 level (2-tailed), ** Significant at the 0.01 level (2-tailed)

Table-2: Socio-demographic Clinical Data characteristics of chronic schizophrenia and chronic skin disease patients

Variables		Chronic Schizophrenia Patients (N=30) Mean ± SD /n (%)	Chronic Skin Disease Patients (N=30) Mean ± SD /n (%)	t/χ^2	df	P
Duration of Illness		4.30±1.96	3.13±1.52	2.56	58	.013*
Family History of Medical Illness	Present	6(19.8)	12(39.6)	2.85	1	.091
	Absent	24(80.2)	18(60.4)			
Family History of psychiatry Illness	Present	11(36.3)	2(6.6)	7.95	1	.010*
	Absent	19(63.7)	28(93.4)			
Medicine Adherence	Good	5(16.5)	2(6.6)			
	Medium	20(67.0)	17(56.1)	3.77	2	.151
	Poor	5(16.5)	11(36.3)			

* Significant at the 0.05 level (2-tailed)

Table-3: Comparison of self-concept in patient with chronic schizophrenia and chronic skin disease:

Variables	Chronic Schizophrenia Patients (N=30) Mean ± SD /n (%)	Chronic Skin Disease Patients (N=30) Mean ± SD /n (%)	t/χ^2	df	P
Self-physical	21.97 ± 3.47	23.40 ± 3.05	-1.7	58	.094
Self-social	22.43 ± 4.20	25.63 ± 3.12	-3.35	58	.001**
Self-temperamental	23.47 ± 3.45	24.27 ± 2.78	-.99	58	.327
Self-moral	27.00 ± 2.60	28.97 ± 2.66	-2.9	58	.005**
Self-intellectual	20.43 ± 3.53	21.80 ± 2.27	-1.79	58	.080

** Significant at the 0.01 level (2-tailed)

Table-4: Comparison of stigma in patient with chronic schizophrenia and chronic skin disease

Variables	Chronic Schizophrenia Patients (N=30) Mean ± SD /n (%)	Chronic Skin Disease Patients (N=30) Mean ± SD /n (%)	t/χ^2	df	P
Stigma	27.83 ± 3.13	24.87 ± 3.58	3.417	58	.001*

** Significant at the 0.01 level (2-tailed)

Table-5: Comparison of quality of life in patient with chronic schizophrenia and chronic skin disease

Variables	Chronic Schizophrenia Patients (N=30) Mean ± SD /n (%)	Chronic Skin Disease Patients (N=30) Mean ± SD /n (%)	t/χ^2	df	P
QOL-physical	12.13 ± .61	12.43 ± .75	-1.692	58	.096
QOL-psychological	11.80 ± 1.68	12.62 ± 1.40	-2.051	58	.045*
Independence	10.48 ± 1.06	11.53 ± 1.23	-3.545	58	.001**
Social relationship	11.87 ± 1.13	11.61 ± 1.22	.881	58	.382
Environmental	11.68 ± 1.12	11.25 ± 1.02	1.574	58	.121
Spiritual	12.47 ± 2.52	13.23 ± 2.69	-1.141	58	.259

* Significant at the 0.05 level (2-tailed), ** Significant at the 0.01 level (2-tailed)

Table-6: Correlation between stigma and self- concept in chronic schizophrenia and chronic skin disease patients

Variables	Stigma(r) Schizophrenia	Stigma(r) Skin disease
Self-physical	-.613**	-.589**
Self-social	-.629**	-.834**
Self-temperamental	-.679**	-.562**
Self-moral	-.589**	-.137
Self-intellectual	-.458*	-.578**

* Correlation is Significant at the0.05 level (2-tailed), ** Correlation Significant at the 0.01 level (2-tailed)

Table-7: Correlation between stigma and quality of life in chronic schizophrenia and chronic skin disease patients

Variables	Stigma(r) Schizophrenia	Stigma (r) skin disease
Qol –physical	-.217	-.552**
Qol-psychological	-.431*	-.668**
Qol-independence	-.483**	-.478**
Qol-social relationship	-.224	-.331
Qol-environmental	-.437*	-.239
Qol-spiritual	-.384*	-.520**

* Correlation is Significant at the0.05 level (2-tailed), ** Correlation Significant at the 0.01 level (2-tailed)

Discussion

In the past, studies on self-concept, stigma and quality of life in schizophrenia and chronic skin disease have been conducted, but no study included all these three areas i.e., self concept, stigma and quality of life as noted by Samuel et al.Staring et al found that Stigma moderates the associations of insight with depressed mood, low self-esteem and low quality of life in patients with schizophrenia spectrum disorders.[14,15]

In the present study purposive sampling was used to select 30 patients of chronic schizophrenia group and 30 patients of chronic skin disease group. In this study there were two study groups; schizophrenia in remission and chronic skin disease.

It was hypothesized that self-concept, stigma and quality of life would not be different between these groups. But in this study we observed that self-concept and quality of life were lower in schizophrenia patient than that of chronic skin disease patients. We also observed that the level of stigma was higher among the schizophrenia patients than the chronic skin disease patients.

The study was conducted on adequately matched 30 chronic schizophrenia patients and 30 chronic skin disease patients. The mean ages of the patients with schizophrenia and chronic skin disease were 35.70 ± 10.00 and 34.30 ± 11.05 years, respectively. In both the groups males were higher in number. There was a significant difference in both groups ($p < 0.01$). But in, both the groups there was a different distribution of males and females. In the schizophrenia group (92.4%) patients were males and rest of them were females, while in the chronic skin disease group there were (56.1%) males and (43.9%) females. Thus, if we consider both the groups separately; then in the group of Chronic Skin disease alone the proportion of females was higher than the group of chronic schizophrenia.

The difference of occupational status in both the study group is statistically significant ($p<0.05$). In the group of chronic skin disease the majority of females were house wives. Thereby the proportion of employment was found higher in the schizophrenia group.

In terms of marital status the number of unmarried persons was higher in chronic schizophrenia group (39.6%) as compared to (20.8%) in chronic skin disease group. However, this finding was not significant. It is unequivocally proved that patients with severe mental illness like schizophrenia have lower rate of marriage than patients with other illness. In this sense this finding of present study was consistent with the above observation made by many researchers regarding

the marital status of patients with schizophrenia. The mean duration of illness was found to be significantly higher in case of schizophrenic patients as compared to chronic skin disease ($p < 0.05$).[16,17]

Comparison on self-concept, stigma and quality of life in the patient with chronic schizophrenia and chronic skin disease:

In this study the self-concept of chronic schizophrenia patients was lower than chronic skin disease. This was found for all domains of self-concept (physical, social, temperamental, moral and intellectual self-concept). On comparing the mean scores of self-concept social and self-concept moral, significant differences were found.

Findings indicate that individuals with chronic schizophrenia perceived themselves negatively. Their view about their body, health, physical appearance and strength were negative. This is similar to earlier findings. Weckowicz and Karidi et al, found that patients with chronic schizophrenia have lower global self-esteem than other physical illness.[18,19]

On Stigma Interview Schedule Scale it was found that there was a significant difference in stigma scores between schizophrenia and chronic skin disease ($p < 0.01$). Patient with schizophrenia had higher scores on stigma scale. Similarly Wait and Harding[20] found that stigma was more in patients with chronic mental illness than chronic physical illness. People in the community remain largely uninformed about the nature and course of schizophrenia. Even when in complete remission, in the absence of psychotic symptoms, patients with schizophrenia may find themselves being shunned or derided. Undeniably, there are patients with schizophrenia who remain chronically ill and disabled. However, there are also those whose symptoms remit, leaving them with minimal impairment, but who find themselves subjected to social discrimination and rejection. Lai et al[14] compared and found that stigma was more in patient with schizophrenia than other chronic physical illness though the sample of their study was not divided equally into experimental group and control group. Secondly, in the course of the survey, stigma questionnaire used in the study did not cover subtle ramifications of measurement of stigma which were taken into account in this study.

On comparing WHOQOL-100 on both groups, quality of life of schizophrenia patients was found to be poorer than that in chronic skin disease. The level of psychological functioning and indepen-dence was found lower in chronic schizophrenia group than chronic skin disease group with statistically significance level ($p < 0.05$) and ($p < 0.01$) respectively; this finding is in line with study of Li et al[13] where they showed the lower level of psychological functioning and independence in chronic schizophrenia than in chronic physical illness like hypertension.

Our results indicate that chronic schizophrenia is associated with poor quality of life and negative self-concept when there was high stigma in this group. Our results show that perceived stigma can be regarded as a moderator of the detrimental associations of mental illness in patients with psychotic disorders. According to Samuel et al the severity of psychopathology not only directly leads to poor QOL, but also indirectly influences self-evaluation of side-effects. In chronic schizophrenia, poor quality of life increased reporting of side-effects, resulting in cumulative dysfunction in daily life for patients. Dixon, Atkinson et al found that poor quality of life was associated with chronic mental illness as compared with chronic physical illness like diabetes.[22,23]

The schizophrenia group's reporting of quality of life could have been influenced by the effects of emotional withdrawal, affective blunting, and poor insight and clinical characteristics of the patient population. Thus, higher satisfaction ratings of life quality may have been due to a minimization of the impact of illness-related dysfunction in social, psychological, and physical domains. Maria Ginieri et al[24] studied three groups of participants; a group with psychiatric disorders (n = 124), a group with physical illnesses (n = 234) and a control group with health participants (n = 67).The study showed significantly lower scores of overall quality of life in mentally ill participants as compared to physically ill participants, as seen in our study.

Relationship of stigma and self–concept in chronic schizophrenia and chronic skin disease:

There was significant negative correlation between stigma and self concept. In schizophrenia stigma is perceived as high. There was significant negative correlation found between stigma with all sub domains of self-concept (physical, social, temperamental, moral and intellectual). This result

is in line with the study of Farina et al.[25] Further, other studies have reported that self-stigma, impacts people's lives and results in lowered self-esteem as noted by Wahl.[12] Link, found that Stigma also acts as a major barrier to social roles in the work place or in personal relationships.

Relationship of stigma and quality of life in chronic schizophrenia and chronic skin disease:

Present study indicates significant negative correlation of stigma with quality of life and its other sub domains (physical, psychological, indepen-dence and spiritual). It might be because of existing stigmatizing beliefs about the patients, despite their engagement in occupational functioning and compliance with medication. These results are supported by Williams[26] who found that highly perceived stigma caused poor quality of life in schizophrenia. They concluded that there is an urgent need for interventions sensitive to the effects of perceived stigma, gender, and medical conditions to improve the QOL and mental health of leprosy patients.

Previous studies have shown that quality of life is very low for those who experience high levels of stigma. While the majority of the literature suggests that impact of stigma would be higher for the emotional domains of health-related quality of life.[27-29]

Summary and Conclusion

The findings can be summarized as:

- There were significant differences regarding sex and occupation between chronic schizophrenia and chronic skin diseases.
- There were significant differences in the domains of duration of illness and family history of psychiatric illness between both the groups.
- Self-concept revealed significant differences in the social and moral domains between chronic schizophrenia and chronic skin disease.
- Mean scores of stigma was significantly higher in the chronic schizophrenia group as compared to the chronic skin disease group.
- Quality of life revealed significant differences in the domains of psychological and social relationship. Mean scores was significantly higher in the chronic skin disease in comparison to the chronic chizophrenia group.
- There was significant negative correlation between stigma and physical, social, temperamental, moral and intellectual domains of self concept in chronic schizophrenia group.
- There was significant negative correlation between stigma and physical, social, temperamental, and intellectual domains of self-concept in chronic skin disease group.
- In chronic schizophrenia group, significant negative correlation was found between stigma and psychological, independence, environmental and spiritual domains of quality of life.
- In chronic skin disease group significant negative correlation was found between stigma and physical, psychological, independence, and spiritual domains of quality of life.

Conclusion

Based on our results we can conclude that there was a significant difference in the self-concept of patients with chronic schizophrenia and chronic skin disease. There was significant difference in perceived stigma in patients with chronic schizophrenia and chronic skin disease. There was significant difference in the quality of life in patients with chronic schizophrenia and chronic skin disease.

Limitations of this study:

- This study had a cross sectional design hence patients were assessed only once.
- The index study was conducted only on outpatients.
- The sample used for the present study may not represent the entire population from which it has been drawn because of the small size and heterogeneity of various variables.
- Both sexes were not equally represented.

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Original Article

Effectiveness of Cognitive Oriented Sex Group Therapy in Treatment of Dhat Syndrome

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Abstract

Objective: *Dhat Syndrome is a disorder prevalent in Indian males who present with loss of vigour, other somatic complaints, depressive and anxiety features, to the extent of being hypochondrical concerns, caused by loss of semen. The aim of the study was to determine the effectiveness of Cognitive Oriented Sex Therapy in treatment of Dhat Syndrome"* ***Method:*** *Thirty four healthy males attending psychiatry outpatient department (OPD), with complaints of loss of semen in urine or as nocturnal emission (wet dreams), were assessed for depression using Hamilton Rating Scale for Depression (HAM-D) and for anxiety using Hamilton Rating Scale for Anxiety (HAM-A). A model of short term cognitive focussed sex therapy was prepared. Patients were given 45 minutes sessions of the therapy every week in a group setting for four weeks. During their weekly visits they were evaluated using HAM-D and HAM-A. Four patients dropped out during the group therapy.* ***Results:*** *There was a significant decrease in HAM-D and HAM-A scores following 4 weekly sessions (one per week) of cognitive focussed sex therapy; which was statistically significant.* ***Conclusion:*** *The results showed a significant improvement in depression and anxiety following cognitive oriented sex therapy and hence its usefulness in the management of dhat syndrome and the sexual knowledge and awareness also improved.*

Key Words: *Dhat Syndrome, Cognitive oriented sex therapy, Group sex therapy*

Introduction

Human sexuality is often colored by myths, pertaining to a particular culture often leading to disordered sexual functioning, and subsequent psychological and somatic distress.

Dhat Syndrome is one such disorder prevalent in Indian subcontinent, suffered by males who are convinced about the loss of vigor, and intense weakness, along with symptoms of depression and anxiety, and other hypochondriacal concerns caused by loss of semen by way of nocturnal emission (wet dreams) or straining during micturition or defaecation.[1] These patients may also present with or without psychosexual dysfunction.[2,3]

Seminal fluid is considered an elixir of life both in the physical and in the mystical sense. Its preservation guarantees health, longevity, and supernatural powers.[4] Often this belief is deeply ingrained much as to challenge the treating psychiatrist in terms of formulation of an effective treatment plan. Many authors have concluded with the suggestion that Dhat syndrome may indeed be a culturally influenced somatoform disorder.[2,5]

Fatigue is a common symptom in Dhat syndrome.[6] Disorders with fatigue as the main symptom are often grouped together as functional somatic disorder.[7] Although it is an established fact that mild symptoms of anxiety and depression is a

part of this syndromal entity, at times these symptoms may be of a magnitude which demands a discreet diagnostic label of major depressive disorder and/or generalized anxiety disorder**.** In Dhat syndrome, "the depressive phenomenology may often meet the DSM – IV diagnostic criteria for depression and responds to selective serotonin reuptake inhibitors along with regular counselling".[8] However with all its nosological vagaries, it may be worth an argument that Dhat Syndrome in all its entirety fits itself to be called a Hypochondriacal disorder, and that it derives its origin on the basis of strong cultural beliefs, it may be addressed to as a "Culture Bound Hypochondriacal Disorder".

Understanding the conceptual origin of Dhat syndrome, in itself makes us believe that its roots are deep seated into the cultural beliefs established over decades of folklore passed down over generation coupled by the tenets of Ayurvedic teachings.[9]

Treatment of Dhat Syndrome is generally aimed at controlling the symptoms of associated depression and anxiety by way of psychopharma-cological means, which also can control the primary concern of semen loss. In its true sense however semen loss is a normal physiological process of a healthy male's sexual apparatus. Educating the patient on these lines may thus be a logical way to deal with such patients,[10] however such efforts have been faced with the problem of the patient dropping out of the therapy sometimes even as early as after the first consultation itself.

This research aims to present the effectiveness of sex oriented cognitive therapy in a group setting, with an initial individual session which is also cognitively oriented.

Methods

Subjects for the study were selected from outdoor patient department of Psychiatry, D Y Patil Medical College and Hospital, Navi Mumbai (India).Ethical Clearance from the local ethical committee was taken prior to the study being undertaken.

In the study duration of one year consecutive males diagnosed with Dhat syndrome were included in the study. A non-random sampling method was used. All patients attending the outpatient department at the study centre who fulfil the inclusion criteria were taken into the study. For the ease of administration the total number of patients was divided into three groups consecutively from 1 to 10; 11 – 20 and 21 – 30. All the sessions for three groups were undertaken by the same therapist.

Patients with the diagnosis of Substance use disorder, Schizophrenia, Delusional disorder and other psychotic conditions were considered as an exclusion criterion.

An informed consent was obtained from those who were willing to participate in the study. A total of 34 patients were enrolled out of which 4 dropped out during the course of study and hence the remaining 30 were considered for this study. All patients were well versed with the English and the local language.An open ended clinical performa was used, such that it could elucidate the symptoms elaborately along with mythical beliefs of the patients. The questions were appropriately translated by the investigators, such that the patient understood them and expressed themselves elaborately.

These patients were assessed clinically for Major Depressive Disorder, Anxiety Disorders and Hypochondriasis as per the DSM-IV-TR diagnostic criteria. Tools used were: (i) Hamilton Rating Scale for Depression (HAM-D),[11] and (ii) Hamilton Rating Scale for Anxiety (HAM-A).[12] HAM-D and HAM-A, was administered to each patient individually on a weekly basis. Same investigator administered the tests, so as to mitigate the rater bias.For the ease of administration the total number of patients was divided into three groups consecutively from 1 to 10; 11 – 20 and 21 – 30. All the sessions for three groups were undertaken by the same therapist on all sessions.

An initial individual session of psycho-education (sex education) was done over 30 to 45 minutes. Subsequently 1 session per week was administered by the same therapist in a group setting, using a psychoeducative cognitive model. Weekly sessions were undertaken over four weeks; each session was of about 45 minutes duration.

Results

In line with the selection criteria 34 patients with primary complains of passing white discharge in urine or in form of wet dreams were assessed.

DAY 1:
• Patient Interviewed Based on an Open ended questionaire
• Diagnosis formulated, Patient inducted in a group of 10

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WEEK 1:
• Initiation and introduction of group of 10 patients.
• Problem highlighted and discussed HAM-A and HAM-D scores noted
• Group given cognitive oriented sex therapy.

⇩

WEEK 2:
• Discussion of symptomatology
• Reappraisal of last session
• HAM-A and HAM-D scores noted
• Group given cognitive oriented sex therapy

⇩

WEEK 3:
• Discussion of Symptomatology
• Reappraisal of last session
• Cognitive errors delineated
• HAM-A and HAM-D scores noted
• Group given cognitive oriented sex therapy

⇩

WEEK 4:
• Discussion of Symptomatology
• Reappraisal of last session
• Reinforcement of Therapy
• HAM-A and HAM-D scores noted
• Group given cognitive oriented sex therapy

During the course of the assessment none of the patients met all the DSM-IV-TR diagnostic criteria of Depression, Anxiety or Hypochondriasis. These patients were given the designed questionnaire to understand their misconceptions about their symptoms.Out of the 34 male patients 25 patients were in the age group of 15-25 years; 8 patients were in the age group of 25-35 years; 1 patient was

Table 1A: Descriptive statistics for variables of anxiety

weeks	number	mean	std. deviation	std.error mean
1ST	30	15.5667	3.3701	0.6153
2ND	30	9.8000	2.3547	0.4299
3RD	30	5.7667	1.4782	0.2699
4TH	30	3.5000	1.2247	0.2236

Table 1B: Descriptive statistics for variables of depression

Weeks	Number	Mean	Std. Deviation	Std. Error Mean
1ST	30	12.0000	1.8004	0.3287
2ND	30	9.0000	1.8383	0.3356
3RD	30	5.5333	1.2794	0.2336
4TH	30	3.4333	0.8976	0.1639

Table 2A: Independent t-test and 95% Confidence limits for variables of anxiety one - sample test

Weeks	t	df	Sig. (2 tailed)	Mean Difference	95% Confidence interval of the difference	
					Lower	Upper
1ST	25.300	29	0.001	15.5667	14.3083	16.8251
2ND	22.795	29	0.000	9.8000	8.9207	10.6793
3RD	21.368	29	0.001	5.7667	5.2147	6.3186
4TH	15.652	29	0.001	3.5000	3.0427	3.9573

Table 2b: independent t- test and 95%confidence limits for variables of depression

Weeks	t	df	Sig. (2 tailed)	Mean Difference	95% Confidence interval of the difference	
					Lower	Upper
1ST	36.507	29	0.001	12.0000	11.3277	12.6723
2ND	26.816	29	0.001	9.0000	8.3136	9.6864
3RD	23.689	29	0.000	5.5333	5.0556	6.0111
4TH	20.950	29	0.001	3.4333	3.0982	3.7685

Table 3a: Analysis of variants (ANOVA) for variables of anxiety

Variables	d.f	F-Value	P-Value	Result
1ST Week - 2ND Week	29	34.328	0.001	Significant
1ST Week - 3RD Week	29	58.230	0.001	Significant
1ST Week - 4TH Week	29	45.794	0.000	Significant
2ND Week - 3RD Week	29	42.284	0.002	Significant
2ND Week - 4TH Week	29	48.337	0.001	Significant
2ND Week - 4TH Week	29	48.337	0.001	Significant
2ND Week - 4TH Week	29	48.337	0.001	Significant
3RD Week - 4TH Week	29	34.497	0.001	Significant

d.f=degrees of freedom
P<.0.05=Significant; Tabulated Value=2.78

Table 3b: Analysis of variants (ANOVA) for variables of depression

Variables	d.f	F-Value	P-Value	Result
1ST Week - 2ND Week	29	35.275	0.001	Significant
1ST Week - 3RD Week	29	42.577	0.001	Significant
1ST Week - 4TH Week	29	53.253	0.000	Significant
2ND Week - 3RD Week	29	41.433	0.002	Significant
2ND Week - 4TH Week	29	36.754	0.001	Significant
3RD Week - 4TH Week	29	31.979	0.002	Significant

d.f=degrees of freedom
P<.0.05=Significant; Tabulated Value=2.78

in age group of 35-45 years. Of the 34 patients 10 were married and 24 patients were unmarried. All the patients belonged to rural background.

Of the thirty four patients recruited for study four dropped out during the course of the therapy. The results in the 30 patients included in the study are shown in Table 1 to Table 3. There was a significant decrease in the scores of HAM – A and HAM – D following the administration of the cognitive oriented sex therapy over a period of 4 weeks as seen in tables 1, 2, 3.

Discussion

At the outset we try to understand the hypothesized symptom formation of Dhat syndrome in terms of a Psych-socio-somatic dynamics. The entity of Dhat syndrome is explained as a functional somatic symptom conglomeration by incorporating societal and cultural factors along the lines of a socio-somatic model.[13] The basic cognitive formulation to explain this is based on somato-sensory amplification, misattribution and abnormal illness behaviour. In Indian culture an open discussion about sexual issues is a taboo, leading to it becoming a focus of psychological pre-occupation; stress predisposes such individuals to amplification of somatic symptoms and such health anxiety may focus their attention on physiological changes such as turbidity of urine and tiredness. In such a scenario they misattribute these physio-logical changes to loss of semen in the light of widely prevalent health beliefs. These beliefs may then be confirmed by friends and other lay sources.[14]

Our study reveals that patients improved significantly as far as the symptoms of anxiety and depression were concerned even though the primary symptom of passing white discharge in urine or wet dreams were either decreased or if present did not cause any significant distress. The frequency of wet dreams were not measured but only the scores of HAM – A and HAM – D were taken.

Although sex education has been tried with varied results, an approach which is cognitive based is seldom looked into. A model of cognitive behavior therapy with a short term focused approach can come a long way in treating such patients as evidenced through our study. It may be argued that a cultural based distortion in the cognitive schemas is often hard to rectify as often faced by the treating clinician. However with empathetic communication and taking into consideration the lack of sex education along with the cultural belief systems the patients are often receptive to a psychotherapeutic treatment modality.There were few limitations of the study. There was no control group and the study sample was small.

Conclusion

The results showed a significant improvement in depression and anxiety following cognitive focussed sex therapy and hence its usefulness in the management of dhat syndrome and the sexual knowledge and awareness also improved. It is important that there was a need to establish the effectiveness of non pharmacological modalities of treatment of mild anxiety and depression associated with Dhat syndrome.

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Original Article

Substance use Pattern and types of Crime Committed by Juveniles attending the Out Patients Department in a Tertiary Care Hospital in Northern India

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Abstract

Background: *In the recent times there has been a sharp rise in the crimes committed by juveniles in the National Capital. Most of these crimes are committed by school dropout adolescents. There is paucity of published data in this regard.* ***Aims and Objectives:*** *To study the socio-demographic, clinical profile, substance use pattern and types of crime committed by juveniles attending the out patients department in a tertiary care hospital.* ***Methodology:*** *The medical records of all the patients who were referred by the courts between June 2009 and June 2011 were included in the study and analyzed for socio-demographic and clinical details. The pattern of substance use and types of crimes committed by them were also studied. The data was summarized using descriptive statistics.* ***Results:*** *Most of the patients assessed in the OPD were males (n=60, 97%).The most common diagnosis was Tobacco Dependence (n=20, 32.2%) followed by Alcohol dependence and Conduct Disorder (n=6, 9.6%), Cannabis use disorder and Volatile Substance Use disorder followed next (n=4, 6.4%). The commonest Crime committed was Theft. Almost 25% (n=15) of the patients were charged for Homicide and 4 patients were charged for Kidnapping (6.4%).* ***Conclusion:*** *The findings suggest a wide range of substance use disorders and myriad types of crimes committed by the juvenile patients sent by juvenile Courts. Commonest substance use disorder was Tobacco dependence and the commonest crime committed was Theft.* ***Implications:*** *To plan out juvenile correctional facilities within the juvenile homes considering the high likelihood of them having psychiatric disorders including substance use disorders.*

Keywords: *Juveniles, substance abuse, crimes*

Introduction

In the recent times there has been a sharp rise in the crimes committed by juveniles in the National Capital. Most of these crimes are committed by school dropout adolescents as per police records. Over 20,000 juveniles are implicated and apprehended in various crimes across the country.[1] The society perceives these juvenile convicts with vice and anger without ever realizing that the crimes they commit may be a vociferous call for help and psychological support given the adverse backgrounds these adolescents come from. At the same time it is the moral and ethical duty of the society at large to provide the best standards of care and correction and provide the juvenile convicts a fair chance for reintegration into the society with dignity and honor. From a sociological perspective, Juvenile crime and its recidivism have its roots in poverty, dysfunctional homes, school dropout and substance use. Most studies conducted in Juvenile

homes report 60-90% of juveniles with behavioral disorders and substance use in the inmates[2,3]. Changing policy and legal perspectives all over the World and in India does recognize juvenile offenders as persons in need of help in the form of specialized psychosocial services for psychological problems and drug use. The current study is a retrospective out-patient department chart review of the clinical profile, substance use pattern and crimes committed by juveniles referred by the Juvenile Courts over a period of two years with the attempt to suggest rectification measures at various levels.

Methodology

The medical records of all the patients who were referred by the courts between June 2009 and June 2011 were included in the study and analyzed for socio-demographic and clinical details. The pattern of substance use and types of crimes committed by them were also studied. The detail of the socio-demographic and clinical profile and the substance use pattern was obtained from the semi-structured performa used for assessment in the OPD. The final diagnosis was made based on the available information and clinical examination. The data for crimes committed was recorded from the official Court document available in the case record file of each of the patients. A total of 62 juveniles attended the OPD from June 2009 to June 2011. The Data was summarized using descriptive statistics.

Results

Socio-Demographic and Clinical Profile

Most of the patients assessed in the OPD were males (n=60, 97%). 51 (82%) belonged to Hindu religion and 11(18%) were Muslims (Table 1). More than 45% of the patients were educated upto class 10th and dropped school subsequently. The details of Residence and Family Structure of most patients (N=44, 70.9%) was not available and were living on streets. This was primarily because of absence of family members and subsequent homelessness, refusal of the juvenile to divulge details about family or lack of information from the Caretakers of the Juvenile Justice Homes.

Diagnosis and Substance use Disorder

Of the 62 patients, 19 patients i.e. 30% had no diagnosable psychiatric disorder (Table 2). Amongst the 43 remaining patients, the commonest diagnosis was Tobacco Use disorder (n=20, 32.2%) followed by Alcohol Use disorder (n=6, 9.6%) and Conduct Disorder (n=6, 9.6%). Cannabis use disorder and Volatile Substance Use disorder followed next (n=4, 6.4%). One patient each had Opioid dependence, Poly-substance dependence and one patient presented with a depressive episode. Thirty six (58%) patients had substance use disorder.

Table 1: Socio-demographic Profile of patients

S.N.	Variables		N(%)
1.	Sex	Male	60 (96.7%)
		Female	2(3.3%)
2.	Religion	Hindu	51(82.2%)
		Muslim	11(17.7%)
3.	Residence	Urban	18(29%)
		Not Known	44(70.9%)
4.	Education	Illiterate	1(1.6%)
		Primary	12(19.3%)
		Matric	28(45.16%)
		Not Known	21(33.8%)
5.	Family Structure	Nuclear	16(25.8%)
		Separated/Divorced	2(3.2%)
		Not Known	44(70.9%)

Table 2: Clinical Profile of patients

S.N.	ICD-10 Diagnosis	Disorder	N (%)
1	F10	Mental and Behavioral disorders due to use of Alcohol	6 (9.6%)
2	F11	Mental and Behavioral disorders due to use of Opioids	1 (1.6%)
3	F12	Mental and Behavioral disorders due to use of Cannabanoids	4 (6.4%)
4	F17	Mental and Behavioral disorders due to use of Tobacco	20 (32.2%)
5	F18	Mental and Behavioral disorders due to Volatile Solvents	4(6.4%)
6	F19	Mental and Behavioral disorders due to multiple drug use and use of psychoactive substances	1 (1.6%)
7	F32	Depressive Disorder	1(1.6%)
8	F91	Conduct Disorder	6(9.6%)
9		No Psychiatric Diagnosis	19 (30%)

All patients with Tobacco use disorder (n=20, 32.2%) had dependence pattern. Fifty percent of the patients with alcohol use had harmful use pattern (n=3, 4.8%) and only one patient had dependence pattern (n=1, 1.6%). Fifty percent of the patients with Cannabis Use had dependence pattern (n=2, 3.2%). All patients with Volatile Substance use disorder had dependence pattern (n=4, 6.4%). The main volatile substance of abuse was Toluene. Opioid dependence and poly-substance dependence was reported in one patient each (Table 3).

Table 3: Substance use Pattern of patients

S.N.	Disorder	Pattern	N(%)
1	Mental and Behavioral disorders due to use of Alcohol	Use Harmful use Dependence	2 (3.2%) 3 (4.8%) 1 (1.6%)
2	Mental and Behavioral disorders due to use of Opioids	Dependence	1 (1.6%)
3	Mental and Behavioral disorders due to use of Cannabanoids	Use Dependence	2 (3.2%) 2 (3.2%)
4	Mental and Behavioral disorders due to use of Tobacco	Dependence	20 (32.2%)
5	Mental and Behavioral disorders due to Volatile Solvents	Dependence	4 (64.5%)
6	Mental and Behavioral disorders due to multiple drug use and use of psychoactive substances	Dependence	1(1.6%)

Criminal Record

The Crimes committed by Juveniles encompassed the gamut of sections in the Criminal Penal Code. Murder, Hurt/Grievous injury, Theft, Kidnapping, Rape, Forgery, setting Fire were the crimes committed (Table 4). Of the 62 patients, 18 patients (29%) were charged under one section only, 20 patients (32.2%) under 2 sections, 10 patients (16.1%) under 3 sections and 4 patients (6.4%) under 4 different sections of the Criminal Penal Code. The data for 10 patients (16.1%) was missing. The commonest Crime committed was Theft. Approximately 42% patients (n=26) were booked for theft. 30% (n=19) were charged with dishonestly receiving stolen property. Almost 25% (n=15) of the patients were charged for homicide. 11% (n=7) patients were booked for Robbery. 6.4% (n=4) patients were charged for kidnapping 3% (n=2) were charged with causing hurt, grievous hurt and the same number with attempt to rape. One patient was booked for wrongful restraint and one for cheating and impersonation. Table 2 enlists the various sections of Criminal Penal Code under which the 62 juveniles were booked.

Table 4: Types of Crime Committed by Juvenile Patients

S.N.	Section (CrPC)	Crime committed	N(%)
1	Sc 302	Punishment for Murder	5 (8%)
2	Sc 307	Attempt to Murder	5 (8%)
3	Sc 308	Culpable Homicide	5 (8%)
4	Sc 323	Punishment for Voluntarily causing Hurt	2 (3.2%)
5	Sc 326	Voluntarily causing Grievous hurt by dangerous weapons or means	2 (3.2%)
6	Sc 341	Punishment for Wrongful Restraint	1 (1.6%)
7	Sc 356	Punishment for Theft	14 (22.5%)
8	Sc 363	Punishment for Kidnapping	1 (1.6%)
9	Sc 364	Kidnapping or Abducting in order to Murder	1 (1.6%)
10	Sc 366	Kidnapping, Abducting or Inducing a Woman to Compel her Marriage	2 (3.2%)
11	Sc 376	Punishment for Rape	2 (3.2%)
12	Sc 380	Theft in Dwelling House	10 (16.1%)
13	Sc 382	Theft after preparation made for causing death, hurt or restraint in order to the committing of the Theft	2 (3.2%)
14	Sc 392	Punishment for Robbery	2 (3.2%)
15	Sc 393	Attempt to Commit Robbery	2 (3.2%)
16	Sc 397	Robbery or Dacoity with attempt to cause death or Grievous injury	2 (3.2%)
17	Sc 398	Attempt to Commit Robbery or Dacoity when armed with a deadly weapon	1 (1.6%)
18	Sc 411	Dishonesty receiving stolen Property	19
19	Sc 419	Punishment for cheating by Personation	1 (1.6%)
20	Sc 420	Cheating and Dishonesty inducing delivery of Property	1 (1.6%)
21	Sc 436	Mischief by Fire or Explosive substance with the intent to destroy house	3 (4.8%)
22	Sc 457	Lurking House	6 (9.6%)
23	Sc 468	Forgery for purpose of Cheating	1 (1.6%)
24	Sc 471	Using as genuine a Forged Document	2 (3.2%)

Discussion

In the current study juvenile under trials were mostly males with only 2 female adolescent under trials. Although incidence of crime in juveniles is higher in males but there is a rising trend amongst female adolescents coming in conflict with law. Almost half of the juveniles were secondary school drop outs. More than 70% of the juveniles were homeless and living on the streets. Our findings are in accord with previous studies documenting a vicious cycle between lack of education and early school dropout, homelessness with substance use and juvenile crime.[1,2,5]

The commonest substance of abuse was Tobacco followed by Alcohol. Our results are in accord with most studies done in the past on juveniles in observation homes.[3] Like most studies in the past on juveniles and street children the pattern of substance use was difficult to establish for most as the information was primarily given by the juvenile who could have under reported because of social stigma and its perceived legal consequences. In 30% of the juveniles (n=19) there was no psychiatric disorder. This could either be because of concealing information related to any behavioral problem or substance use to escape the arms of law or because of chance involvement at the scene of crime. No correlation was drawn with substance use and the severity of crime for this reason as the data -would not be a veridical reflection of the high rates of crimes committed by juveniles. Previous studies have reported Theft as the commonest crime committed by juveniles[3]. Theft is perhaps analogous to 'Gateway drugs' as far as crime amongst juveniles is concerned followed by other serious crimes and so, intensive psychosocial intervention at the earliest stages of crime will go a long way in helping juvenile convicts. As noted in our study, parents fearing the clutches of Law for their ward may not divulge details of their child's behavioral problems failing to realize that early intervention would be helpful. It is these juveniles committing petty crimes such as theft, which later grow and evolve into becoming criminals.

Conclusion and Implications

There is an urgent need to study various factors leading to Juvenile delinquency so that appropriate models of care for their management in hospital setup and possibly within the juvenile home, can be developed. This in turn will help plan out juvenile correctional facilities within the juvenile homes considering the high likelihood of them having psychiatric disorders including substance use disorders.

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Original Article

Depression in Schizophrenia: Correlation with Psychopathology and Level of Functioning

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Abstract

***Background**: There has been ample research to show that depression is common in patients with schizophrenia. However, the characteristics and determinants of depressive symptoms in schizophrenic patients remain unclear. This study was done to assess the presence and severity of depressive symptoms in schizophrenic outpatients and to study the correlates.* ***Method**: The sample consisted of 50 patients with schizophrenia. Beck Depression Inventory (BDI) was used to measure depressive symptoms. Positive and Negative Syndrome Scale (PANSS), Clinical Global Impressions (CGI) scale and Global Assessment of Functioning (GAF) scale were administered to measure the psychopathology and the level of functioning.* ***Results**: Depressive symptoms were present in 78% patients with schizophrenia. There was no significant difference between the depressed patients and those not depressed with respect to age, gender, education or income. No significant correlation was found between BDI scores and PANSS, CGI and GAF scores.* ***Conclusions**: As no significant relationship was observed between depression and psychopathology or illness severity, it appears that probably depression is common in schizophrenia and could be independent of psychopathology and severity of illness. There is a need for more research on the determinants of depression in schizophrenia so that evidence-based preventive and treatment strategies can be devised.*

***Key words:** Schizophrenia, Depression, Psychopathology, Level of functioning, BDI, PANSS, CGI, GAF*

Introduction

Schizophrenia is chronic illness characterized by symptoms like delusions, hallucinations, disorganized behavior, negative symptoms, and socio-occupational dysfunction[1].

Recent findings are suggestive that depression is common in schizophrenia and is has a distinct course which compromise functional well-being and has poor prognosis[2].

Risk of developing depressive symptoms is more in patients with schizophrenia than the general population[3] and depression is prevalent even in stable schizophrenic patients[4]. The estimates of the frequency of depressive episodes in patients with schizophrenia range from 20% to 80%[5].

Depressive symptoms in schizophrenia significantly increase the distress and burden of the illness[6]. It has been shown that schizophrenic patient having concurrent depressive symptoms have poorer long-term functional outcome and therefore, identifying and treating depression in schizophrenia is of prime importance for recovery[7].

Depressive symptoms in schizophrenics may either fully satisfy the diagnostic criteria of a major depressive episode or may be sub-threshold and may be an independent component of the disorder or, alternatively, may be a consequence of severe psychotic symptoms[5]. Research on the relationship

between depressive symptoms and illness characteristics of schizophrenia has shown mixed results. Some authors have reported that depressive symptoms correlate with the severity of negative[8,9] and positive symptoms[10-12], while others have found no correlation between them[13].

We conducted this study to assess the presence and severity of depressive symptoms in outpatients schizophrenics to find out if there was a correlation with psychopathology and level of functioning.

Material and Methods

The study was conducted at Psychiatric OPD of NIMS Medical College attached hospital, Jaipur, India. Patients fulfilling the specified inclusion and exclusion criteria were included in the study.

Study Group

It consisted of 50 consecutive clinically stable schizophrenic patients attending the Psychiatric OPD. The following were the inclusion and exclusion criteria.

Inclusion criteria

All the patients in the age group 18 to 65 years who were diagnosed of schizophrenia according to I.C.D.–10 criteria ,clinically stable and taking regular psychiatric treatment.

Exclusion criteria

Patients with significant medical or neurological illness, schizoaffective disorder and pharmacological or otherwise anti depressive treatment, mental retardation, patient meeting criteria for drug dependence and addiction, history of indoor admission in past six months prior to assessment and duration of illness less than two year.

Study Design (Operational Procedure)

Patients fulfilling the inclusion criteria were taken up for the study. These cases were enrolled after taking informed consent from them to be included in the study. After detailed history and mental status evaluation a diagnosis of schizophrenia was confirmed by two senior consultant psychiatrist of the hospital according to ICD-10 criteria. Then the patients were evaluated clinically and complete physical examination was done.

The patient's socio-demographic data and illness history were recorded. Following this, psychopathology, illness severity and level of functioning were assessed using appropriate rating scales. Later on, these patients were subjected to Beck depression inventory (BDI)[3] to assess the presence and severity of depression.

Instruments of study

1. **Self-designed semi-structured performa was used. It included the following:**
   (a) Identification data sheet.
   (b) Socio-demographic data: age, sex, marital status, domicile, family size, family type, family income, education, occupation and other relevant information.
   (c) The details of present psychiatric illness.
2. **Positive and Negative Syndrome Scale (PANSS):** The PANSS includes 30 items on three sub scales, seven items covering positive symptoms (e.g. hallucinations and delusions), seven covering negative symptoms (e.g. blunted effect) and 16 covering general psychopathology (e.g. guilt, uncooperativeness). Each item is scored one to a seven point. Reliability for each scale is fairly high, with excellent internal consistency and inter rater reliability. Validity is also good based on correlation with other symptom severity measures and factor analytic validation of the subscales.[14]
3. **Global Assessment of Functioning (GAF):** It is a 100-point single item scale with value ranging from 1 to 100 representing the hypothetically sickest person to the healthiest. The scale is divided into 10 equal 10-point intervals with the 81 to 90 and 91-100 intervals for individuals who exhibit superior functioning. The 71 to 80 intervals are for person with minimal psychopathology. Most patients in outpatient setting will receive ratings between 31 and 70, and most inpatients between 1 and 40.[15]
4. **Clinical Global Impression (CGI):** This is a three-item scale, which measures overall severity of illness. If repeated, it can evaluate response to treatment. The CGI is rated by the patient's physician or a trained rater who evaluate severity of illness, clinical progress, and therapeutic efficacy[16].

For the present study we have used its first item "Severity of illness" to assess patients illness

severity

5. **Beck Depression Inventory (BDI):** The Beck Depression Inventory created by Dr. Aaron T. Beck in 1987, is a 21-question multiple-choice self-report inventory and is one of the most widely used instruments for measuring the severity of depression. The standard cut-offs are as follows : 0–9: indicates minimal depression, 10–18: indicates mild depression, 19–29: indicates moderate depression and 30–63: indicates severe depression[17].

Statistical analysis: All the data represent Mean ± S.D values for clinical variables [Table 2]. The relationships between BDI scores and demographic / clinical variables (PANSS, CGI and GAF) were studied using t-test, ANOVA and Pearson's correlation coefficient in SPSS 17 version. *P* values less than 0.05 were considered as statistically significant

Results

Table-1 and 2 shows socio-demographic and clinical characteristics of the study group. Patient group consisted of 35 males and 15 females with a mean age of 38.6 years, 48% of them were working, 56% were from urban background, 68% were married and 62% were living in Joint family. Majority of patients (72%) had an onset before the age of 30 years. In the study group 36% of patient had total duration of illness of more than 10 years, 28% of patient had duration of illness in range of 5-10 years and remaining 36% had duration of illness below 5 years.

Scores on the PANSS scale ranged from 7 to 29 on the positive scale (M=13.70, S.D. = 5.32), 8 to 29 on the negative scale (M=14.56, S.D. = 6.27) and 18 to 54 on the general psychopathology scale (M = 29.74, S.D. = 7.17) (Table – 2). The GAF mean score was 65.56, in our study, while the mean illness severity score as assessed from CGI- scale was 3.02 (S.D= 1.39) . Scores on the BDI scale ranged from 3 to 39. The mean score for depressive symptoms in this group of patients was 16.32 ± 7.95 points. Table–2

In our study, 78 % of patients were experiencing depressive symptoms. Majority of patients were suffering from mild depression (44%) followed by moderate depression (30%) and 4 % with severe depression. Only 11 patients out of 50 (22%) reported no depressive symptoms. Table-3, Figure 1.

There was no significant difference between the depressed patients and those not depressed with respect to age, gender, education or income. No significant correlation was found between BDI

Table - 1. Distribution of Sociodemographic and clinical profile of study group

Variable	Schizophrenia n = 50, f %)
Age in (years) – mean (SD)	38.62 (10.75)
Sex	
Male	35 (70)
Female	15 (30)
Marital status	
Married	34 (68)
Unmarried (Single)	10 (20)
Separated, widower and widower, divorced	6 (12)
Domicile	
Rural	22 (44)
Urban	28 (56)
Education	
Illiterate	11
Below Higher secondary	15
Higher secondary and above	24
Occupation	
Working	24 (48)
Nonworking	13 (26)
Housewives	13 (26)
Family Income in Rupees / month	
< 3000	25 (50)
3000-10000	16 (32)
> 100001	9 (18)
Family type	
Joint	31 (62)
Nuclear	19 (38)
Type of medication	
Atypical	14 (28)
Typical	30 (60)
Both	6 (12)

Table - 2. Clinical characteristic of study group

Variable	Schizophrenia n = 50, mean (SD)
PANSS	
Positive	13.70 (5.30)
Negative	14.56 (6.27)
General Psychopathology	29.74 (7.17)
GAF	65.56 (17.76)
CGI	3.02 (1.39)
BDI	16.32 (7.95)

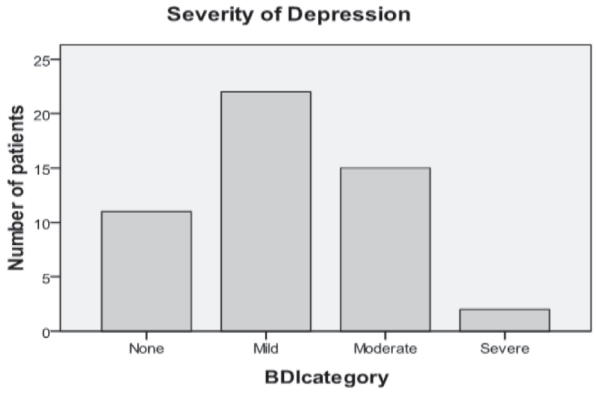

Figure 1. Severity of Depression in Schizophrenic outpatients.

Table -3 Severity of Depression (BDI Category)

Severity of Depression	Schizophrenia n = 50, (f %)
None	11 (22)
Mild	22 (44)
Moderate	15 (30)
Severe	2 (4)

Table – 4. Relationship between Sociodemographic and clinical factors with Depression in Schizophrenic patients (n = 50)

Variable	Depression (BDI)
Age	r = .196 (sig = .173)
Sex	t = -1.390, (sig- .171)
Domicile	t = 1.131 (sig = .264)
Family type	t = -.032 (sig = .974)
Occupation	f = .826 (sig= .444)
Marital status	f = 1.435 (sig = .248)
Income	f = 1.028 (sig = .366)
Onset of Illness	f = 1.596 (sig = .124)
Duration of Illness	f = 1.525 (sig = .148)
Type of Medicine	f = 1.388 (sig = .207)

Pearson product moment (r). t test (t), ANOVA (f); *P < .05, ** P < .01

Table–5. Correlation between Psychopathology, Level of functioning and Depression in Schizophrenic patients (n = 50)

Variable (PANSS)	Depression (BDI)
Positive	r = 0 .039 (sig = .786)
Negative	r = 0 .026 (sig = .856)
General Psychopathology	r = 0 .189 (sig = .266)
GAF	r = -0.148 (sig = .304)
CGI Severity	r = 0 .186 (sig = .195)

Pearson product moment (r). *P < .05, ** P < .01

scores and PANSS, CGI and GAF scores. Table 4, Table 5.

Discussion

Many authors have looked at the prevalence of depression in schizophrenia and have found highly variable rates, ranging from 6% to 80%. Some of this variation can be attributed to the differences in methodology. A previous study from India reported that out of 95 patients with schizophrenia, 87% had depressive symptoms but only 32% reported having a depressed mood.[8] Since the choice of the instrument used to measure depression can have a bearing on the results, we used the Beck Depression Inventory which has been shown to be a valid measure for identifying depressive symptoms in stable chronic schizophrenia.[18] We found that 78% patients had depressive symptoms out of which 34% had moderate to severe depression. These results are in accordance with previous studies which have found that depression is common in schizophrenia.[7,13,19–24]

Female gender is known to predispose to common mental disorders like depression, however in resonance with the findings of few other studies,[5,25] no sex differences were observed with regard to the presentation of depression in our study group. We also found no significant differences in age, education level and income between the depressed patients and those not depressed.

Relationship between depression and other symptoms of schizophrenia appear to differ during different stages of illness.[26] Various studies have reported mixed results regarding relationship between depression and psychopathology symptoms in Schizophrenics. Some authors reported that depressive symptoms correlate with either negative symptoms[8,9] or positive symptoms,[5,10-12,27] while other studies reported that depression correlate with both positive and negative symptoms[26] or exist as an independent, core component of the disorder.[5,13,28,29]

In this study, we found no such correlation between depressive symptoms measured by BDI scores and psychopathology measured by PANSS scores in our sample also suggesting that depression probably is an independent component of schizophrenia, rather than a by-product of psychotic symptoms. This was also supported by the absence

of a correlation of BDI scores with illness severity measured by CGI scores and level of functioning measured by GAF scores, similar to the finding of one study according to which depression cannot be explained by severe illness or poor functioning.[29]

In previous studies of Depression in schizophrenia, a reported confounding factor was the possible effect of concomitant treatments.[2] However in a review article on drug treatment for schizophrenia, its authors concluded that depressive symptoms decrease (rather than increase) in severity with effective neuroleptic treatment.[30] Several other controlled-study evidence also tends to refute the fact that neuroleptic medication are responsible for the development of depressive states in schizophrenia[29]. In fact, various studies have reported that neuroleptics do not appear to induce depressive disorder in acute or chronic schizophrenic patients.[31,32]

One important limitation of our study was that we did not take into consideration the antipsychotic drug induced extra-pyramidal side effects, which are known to be associated with depression.[29] However, since we found no significant correlation between the type of antipsychotic used and depressive symptoms, we believe that the treatment taken by patients would not have confounded our results.

Depressive episodes are a common and potentially severe occurrence in schizophrenia but are poorly recognized by psychiatrists.[33] People with schizophrenia and concurrent depressive symptoms have poorer long-term functional outcomes compared with the non-depressed.[7] It has been found that the occurrence of depression in schizophrenia has often been associated with impaired functioning, personal suffering, higher rates of relapse, and even suicide.[29] With so much at stake, it is important that depressive symptoms in patients with schizophrenia should not be missed, but thoroughly evaluated and adequately treated.

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Original Article

A Clinical Study of Subsyndromal Anxiety Symptoms in Children and Adolescents from a North Indian Child and Adolescent Clinic

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Abstract

Background & objectives: *Though anxiety disorders are one of the most common group of psychiatric disorders among children and adolescents, the frequency of subsyndromal forms of anxiety — in children and adolescents is unknown — and it is often under-diagnosed. No study is yet done in India on subsymdromal anxiety symptoms in children and adolescents. Therefore, this study was — planned with the aim to identify subsymdromal anxiety -symptoms in children and adolescents and elicit their phenomenology and assess their impairment.* ***Methods****: Patients between age group 6 to 16 years attending child and adolescent psychiatry clinic were screened by Screen for child anxiety related emotional disorders (SCARED) scale. Those patients having anxiety symptoms, but not fulfilling criteria for any anxiety disorder, their phenomenology was assessed by SCARED scale. Impairment due to anxiety symptoms was assessed by Children's global assessment scale (C-GAS).* ***Results:*** *Out of 1465 screened patients 21(1.43%) patients had anxiety symptoms. Mean age of patients with anxiety symptoms was 11.4 ± 1.8 years. Majority of patients were girls 15(71%). 4(19.05%) patients had family history of psychiatric disorders. Mean score of anxiety symptoms on SCARED scale was 18.24 ± 2.51. Most of the subjects had some difficulty in functioning (Mean C-GAS score -650 ± 3.78).* ***Interpretation & conclusion:*** *The significant prevalence of sub-syndromal anxiety symptoms, the significant psychosocial impairment associated with it and the possible chronicity of its course should make the subsyndromal anxiety symptoms a matter for serious consideration by both clinicians and researchers.*

Key words*: Anxiety, Subsyndromal, Children, Adolescents.*

Introduction

The specification of diagnostic algorithms and cut off scores for different psychiatric disorders has led to the new controversy as to whether the sub-threshold/subsyndromal symptoms are a clinical problem at all, or separate phenomenon in their own right, or a minor form of major psychiatric disorder.[1] Though anxiety disorders are one of the most common group of psychiatric disorders among children and adolescents, the frequency of subsyndromal forms of anxiety in children and adolescents is unknown. Unfortunately, these are often significantly underreported and under-diagnosed and difficult to recognize. Great Smoky Mountains Study[2] showed that subsyndromal anxiety symptoms in children and adolescents quadrupled the likelihood of developing anxiety disorders in the future. This study also revealed that children and adolescents with subsyndromal forms of anxiety were twice as likely to have impaired functioning

compared to those with no symptoms. Typically, the best predictor of later psychopathology, including anxiety disorders, is earlier psychopathology.[2] If symptoms of anxiety in children and adolescents are predictive of disorders and impairment, then treatment of — subsyndromal forms of anxiety in children and adolescents may be one of the few options for reducing the chronicity of anxiety disorder. No study is done in India on subsymdromal anxiety symptoms in children and adolescents. Therefore this study was planned in a psychiatry outpatient setting of a university department of psychiatry in northern India with the aim to identify subsymdromal anxiety symptoms in children and adolescents and elicit their phenomenology and impairment.

Material and Method

This cross-sectional, clinic based study was carried out at Department of Psychiatry, King Goerge's medical university, Lucknow, India from August 2010 to July 2011. The study was approved by the institutional ethics committee. Inclusion criteria were, (i) non psychotic patients between 6 to 16 years of age, (ii) availability of at least one reliable informant who may be a parent or guardian of the subject, (iii) informed consent of the parent or guardian of the patient. Exclusion criteria included, (i) patients with psychotic illness, (ii) patient with a severe physical disorder or condition requiring priority medical-management, (iii) Parents or guardians not willing to give informed consent, (iv) non-availability of a reliable informant, (v) mental age < 6 years.

All patients attending child and adolescent psychiatry OPD were screened for selection criteria. IQ assessment of the patients were done by the clinical psychologists by using Raven's Progressive Matrices[3]. All selected patients were screened—by screen for child anxiety related emotional disorders (SCARED) scale.[4] Those patients having anxiety symptoms, but not fulfilling criteria for any anxiety disorder their phenomenology was assessed by screen for child anxiety related emotional disorders (SCARED) scale. Impairment due to anxiety symptoms was assessed by children's global assessment scale (C-GAS).[5]

Results

A total of 1465 persons were screened. Out of which 21(1.43%) patients had anxiety symptoms. Mean age of patients with anxiety symptoms was 11.4 ± 1.8 years. Majority 15(71%) patients were in childhood (6-13 years) age group. 19(90%) had the onset of symptoms before 13 years of age. Majority of patients were girls 15(72%). Majority of patients belong to urban area 15(71%). Eighteen (86%) patients were students. Mean IQ of subjects was 92.24 ± 1.82. 4(19.05%) patients had family history of psychiatric disorders. Out of which 2 (50%) patients had family history of generalized anxiety disorder and 1(25%) each patients had family history of major depressive disorder and bipolar affective disorders. Mean score of anxiety symptoms on scared was 18.24 ± 2.51.

Table 1. Phenomenology of Anxiety Symptoms

Anxiety Symptoms	N (21)	%
Feeling of nervousness	14	67%
Frightened for no reason	8	38%
Worry about sleeping alone	7	33%
Afraid to be alone in the house	7	33%
Stomach ache at school	11	53%
Worry about going to school	10	48%
Nightmares that something is happening to me	5	24%
Worry about sleeping alone	6	29%
Headache when at school	8	38%
Do not like to be away from home	7	33%
Shy with unknown people	8	38%
Heart beats first when frightened	5	24%
Nervous with unknown people	12	58%
Difficult to breath when frightened	4	19%

*Not mutually exclusive

Most of the subjects had some difficulty in functioning (Mean C-GAS score - 65 ± 3.78).

Discussion

In our study out of 1465 screened patients 21 (1.43%) patients had anxiety symptoms not fulfilling criteria for any anxiety disorder. Though no Indian data is available regarding frequency of subsyndromal anxiety symptoms in children and adolescents, but while comparing with the frequency of anxiety disorders in children and adolescents from the epidemiological study conducted by Indian Council of Medical Research[6]

at two centers Bangalore (3.93%) and Lucknow (2.32%), it can be concluded that subsyndromal anxiety symptoms are significantly present in Indian children and adolescents population. This study is a clinic based study. So the frequency of sub-syndromal anxiety symptoms in children and adolescents may be more in community setting.

Though anxiety disorders in children and adolescents are associated with significant impairment in functioning[7] but in our study most of the subjects of subsyndromal anxiety symptoms also had some difficulty in functioning (Mean C-GAS score - 65 ± 3.78). This finding is supported by Great Smoky Mountains Study[2] on subsyndomal anxiety symptoms which revealed that children and adolescents only with symptoms of anxiety were twice as likely to have impaired functioning compared to those with no symptoms. Thus, in this study, subsyndromal anxiety symptoms were associated with children and adolescents' impaired ability to function well at home, at school, and with peers.

Great Smoky Mountains Study[2] on sub-syndromal anxiety symptoms also revealed that children and adolescents with anxiety disorders show clinical symptoms of anxiety even at times when they would not meet formal diagnostic criteria of any anxiety disorders. This study also showed that subsyndromal anxiety symptoms quadrupled the likelihood that children and adolescents without a previous history of anxiety disorders would develop anxiety disorders in future.

The significant prevalence of subsyndromal anxiety symptoms, the significant psychosocial impairment associated with it and the possible chronicity of its course should make the sub-syndromal anxiety symptoms a matter for serious consideration by both clinicians and researchers. Thus, there is a need to give research-based recognition to subsyndromal anxiety symptoms along with their associated repercussions and a comprehensive strategy including guidelines for addressing this matter from the management point of view.

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Original Article

Perceived Social Support and Stigma in the Adult with Epilepsy and Normal Control

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Abstract

Background: *In this study, we assessed the Perceived Social Support and Stigma in the adults with epilepsy as compared to normal control.* ***Materials and methods****: The study was cross-sectional hospital based study. Purposive sampling was used, in which included 60 adults, among which 30adults were diagnosed with epilepsy and 30 normal persons as control group. Socio-Demographic & Clinical Data Sheet, Social Support Questionnaire and Stigma Scale of Epilepsy_were used for the assessment. The Statistical Package for Social Sciences (SPPS) 16.0 for windows was used for statistical analysis.* ***Results and conclusion****: majority of patients was male, belonging to joint family and was from rural background. There was significant difference found in monthly income between patients with epilepsy and normal control. There were no significant differences of social support and stigma of epilepsy in the both group.*

Keywords: *Epilepsy, Social support,Stigma*

Introduction

Epilepsy is a chronic condition of the brain which is characterized by recurring seizures, which is the body's reaction to sudden excessive electrical discharges in the brain. Epilepsy has been around for centuries and is one of the oldest conditions of the human race. The earliest references date back to B.C. in Mesopotamia, where auras, generalized convulsions and other aspects of "the falling disease" were recounted very accurately. Seizures were thought to be caused by evil spirits or demons that had invaded a person's body. Ancient physicians challenged the superstition and recognized a seizure as a dysfunction of the brain. Nonetheless, attitudes from the past have left misconceptions which still persist today and people with epilepsy continue to deal with stigma. Stigma comes in many forms. It can be people who still believe myths about epilepsy or are fearful about how to respond to a seizure. Stigma can cause unhappiness and isolation for the individual[1]. It has found that adults who developed epilepsy during childhood often have social problems, even if they no longer experience seizures.[2]

It is estimated[3] that there are 55, 00,000 persons with epilepsy in India, 20, 00,000 in USA and 3,00,000 in UK. Three to five per cent of the populations have a seizure sometime in their life and half to one per cent of the population have *'active epilepsy'*. The incidence of epilepsy ranges from 40 to 70 per 100,000 in most developed countries and from 100 to 190 per 100,000 in developing countries. In most countries worldwide, the prevalence of active epilepsy ranges from 4 to 10 per thousand populations. Higher prevalence rates ranging from 14 to 57 per thousand have been reported from some African and South American countries.[4-7] In India the incidence rate of epilepsy is roughly 49.3 per 100,000 and the number of new persons with

epilepsy in India each year would be close to half a million.[8]

For years people with epilepsy have been stigmatized and discriminated by society. This attitude has been persisting for long time because of the presence of inadequate and erroneous knowledge about epilepsy in society and presence of some culturally defined notion about epilepsy and its patients. Even in modern societies, many persons with epilepsy have been put in institutions, kept segregated from others and often socially rejected. Attitude surveys since 1950 have shown a steady improvement in developed countries, but much improvement is still needed in developing countries. In case of epilepsy, stigma arises from the perception of difference from the norm, and is particularly powerful when the difference is not understood. This arises from the uniquely human need for predictability, resulting from people's ability to think abstractly, and thus to surmise the future. Prediction relies on learning from past experience plus inference from perceived causality. Most people have no experience of epilepsy and do not know what causes it. Thus they feel unable to predict what impact it will have on their future. This phenomenon in accordance with rustic terms can be referred as 'fear of unknown' as most people do have a sense of apprehension about epilepsy, its aetiopathogenesis and dos and don'ts to patients. So lesser knowledge about this illness evokes fear to people. Fear of the unknown evokes negative feelings in people, which people try to reduce by rejecting its cause - i.e. the people with epilepsy. Other causes of stigmatization of the people with epilepsy may be: (1) Socio-cultural conditioning that normal is beautiful, and beautiful is good; (2) Stress in childhood on the importance of health; (3) Feelings of guilt for being fit and well; (4) Belief that any disability is a punishment for sin; (5) Not knowing how to interact with a people with epilepsy; (6) Repulsion by the ugly; (7) Realization that the body may not be defensible; (8) Perception of the people with epilepsy as a marginal group, outside society; (9) Disability as a reminder of human mortality; (10) Socially inappropriate behaviour by the people with epilepsy, often arising from social isolation; (11) Degree of severity and visibility of the epilepsy; and (12) Demographic and personality factors which influence the likelihood of stigma.[9]

In epilepsy, the stigma has been considered to be one of the most important factors that have a negative influence on the people with epilepsy and their family. Patient with epilepsy, more frequently attributed stress at work and inadequate Despite commendable achievements in the field of neuropharmacology as well as introductions of newer antiepileptic medications still common people reckon epilepsy to be a disorder with contagiousness, or having some demonical influences or problems lie on the suffered so on. Still people could not get out from the misconcep-tions and prejudices related to epilepsy. This way people who have epilepsy do not get the social acceptance and image what others do and these peoples' social connectivity and repertoire tend to become limited and indeed restricted. Epilepsy is a common neurological condition, frequently associated with psychosocial difficulties. Prejudice and discrimination often have a greater impact on the daily lives of people with epilepsy than seizures. Previously done studies on epilepsy suggest that the stigma of epilepsy is one of the most important negative influences on the subjective well-being or life satisfaction of people with epilepsy. Epilepsy is known as a stigmatizing condition, as people with epilepsy sometimes cannot conform to usual social norms as a result of the unpredictable nature of seizures; consequently, society may fear dealing with someone who is having an epileptic seizure. From the patient's point of view, the diagnosis of epilepsy challenges self-perception, and causes concern about ability to function in normal social roles, such as getting a job or having a family. The perception of epilepsy is often negative, which impairs the patient's quality of life and reinforces the stigma. Stigma associated with epilepsy has been related to poor psychosocial health outcomes, such as low self-esteem, worry, negative feelings about life, and depression.[10-12] The concept of stigma emerged as an important factor related to subjective well-being as well as development of positive self-esteem in persons with epilepsy. To develop both individual and broad social interventions to decrease the impact of stigma, it is important to understand how people of all ages with epilepsy experience and cope with this problem in their lives.

The relationship between social support and health has received a great deal of research attention in health psychology and behavioral medicine in

recent days.[13,14] Social support is an important factor in immune, endocrine, psychological and cardio-vascular functioning; recovery from illness and injury; and health maintenance. Social support may benefit health by buffering stress, influencing affective states, and/or changing behaviors.[15] The direct effects may occur with hormonal and neuroendocrine influences on the immune system, and indirect effects may occur through lifestyle and health behaviors[16] or other aspects of social and psychological functioning (e.g., depression and anxiety)[17]. Social support also influences the ability to adjust to and live with illness.[18,19] Social support has a major role to play in the long term course and outcome of any chronic illness and epilepsy is not exception of that. Social support from the key caregivers and environment or community can play a very positive role by enabling the chronic disorder affected people to have abilities to deal with the stress very effectively or have the sense of mastery to overpower the stress or eliminate the negative consequences of stress. Positive functional social network can work as the 'buffering agent' against stressors for people with chronic disorder like epilepsy. This way social support can uplift the quality of life and subjective –well-being of people as well as enable people to develop and use positive forms of coping and problem-solving strategies.[20]

There is lack of study on social support and stigma of the adults with epilepsy. Because those disorders have also chronic courses and often require prolong treatment for alleviation of symptoms. So such kind of effort is required to have a clear picture about the stigma or subjective well being of these adults with epilepsy.

Aims

The aim of present study was to study perceived social support and stigma in the adults with epilepsy as compared to normal control.

Objectives

- To examine perceived social support of the adults with epilepsy and normal control.
- To examine perceived stigma of the adults with epilepsy and normal control.

Hypotheses

- There will be no significant difference in perceived social support between adults with epilepsy and normal control.
- There will be no significant difference in perceived stigma between adults with epilepsy and normal control.

Methodology

Place of the Study

Out Patient Department of Epilepsy Clinic of the Central Institute of Psychiatry, Ranchi.

Study Design

The present study was cross- sectional hospital based and single contact study. The study assessed the subjective well-being, dysfunction, perceived social support and perceived stigma in the adults with epilepsy as compared to normal control.

Sampling: Purposive sampling was used.

Sample Size: The present study included 60 adults, among which 30 adults were diagnosed with epilepsy and 30 normal persons as control group.

Inclusion Criteria of Patients

- Patients diagnosed with epilepsy as per International League against epilepsy (ILAE,1989)
- Both sexes (male & female)
- Age more than 18 years
- Those who gave written informed consent for the participation in study

Exclusion Criteria of Patients

- History of any chronic physical illnesses, mental illness, organic brain syndromes, and substance abuse/dependence.
- Comorbid significant psychiatric illness.
- Mental retardation.
- History of learning disorder, conduct disorder, ADHD.
- Age less than 18 years

Inclusion criteria for normal controls

- No history of any chronic physical illnesses, mental illness, organic brain syndromes, and substance abuse/depen-dence
- Age more than 18 years
- Age, Sex and education Appropriately

matched with the patients' group
- Those who gave written informed consent for the participation in study

Exclusion criteria for normal controls

- Having significant physical illness, substance addiction (except moderate use of tobacco and caffeine)
- Having significant psychiatric illness
- Not willing to participate in the study
- Age less than 18 years

Assessments Tools

- A socio demographic and clinical data sheet was made for the study to obtain the socio-demographic and clinical information from the participants of the study.
- Social Support Questionnaire (SSQ) by Nehra and Kulhara [21]
- Stigma Scale of Epilepsy (SSE) by Fernandes et al[22]

Brief Description of Tools

- ***Socio-Demographic and Clinical Data Sheet***
  A socio demographic data sheet was developed for this study which included variables like age, sex, education, income, occupation etc. for both groups, and illness-related parameters (illness-duration, number of. episodes, frequency of seizures).
- ***Social Support Questionnaire (SSQ) (Nehra and Kulhara, 1987)***
  This Indian adaptation of the Social Support Scale of Pollack and Harris measures perceived social support. It has 18 items rated on a 4-point (4-1) scale. Item number 2, 4, 8, 9, 11, 12,18 are positively worded and score as such but item number 1, 3, 5, 6, 7, 10, 13, 14, 15, 16 and 17 are negative item and have to scored in the reverse order (1-4). The total item indicates the amount of social support perceived by the individual. Higher scores indicate more perceived social support. [21]
- ***Stigma Scale of Epilepsy (SSE)***
- This scale was developed by Fernandes et al, (2007).[22] This scale identifies the perception of epilepsy stigma by a subject. This scale contains 24 items, each with a 4-point scale. Individuals are asked to indicate the most suitable answer for each item from the following options: 1 = not at all, 2 = a little, 3 = a lot, 4 = totally. Higher scores indicate more level of stigma.

SSE score calculation

$$\text{SSE} - \text{Score} = \frac{\text{Sum of all answered items - number of answered items}}{\text{Maximum score possible - minimum score possible}} \times 100$$

Maximum score possible = 4 × number of answered questions

Minimum score possible = number of answered items

The scores of two items (1 and 4 g) were inverted when the scores were added.

Procedure

The patients were selected based on inclusion and exclusion criterion through purposive sampling from epilepsy clinic of Central Institute of Psychiatry, Kanke, Ranchi. Normal controls were selected from adjoining area of Kanke Block. Informed consent was taken from all the participants after providing detailed information about the study. After filling of the socio-demographic data sheet, social support questionnaire, and stigma scale for epilepsy were administered on the patients with epilepsy and normal control individuals.

Statistical Analysis

The Statistical Package for Social Sciences (SPPS) 16.0 for windows was used for statistical analysis. Both descriptive and inferential statistics were used for analyzing data. Chi squire test was used for comparing categorical variables and t-test was used for comparing continuous variables. Similarly Pearson's and Spearman's Correlations were used to find out the relationship between variables and scales.

Results

Table 1 shows the comparison of two groups, i.e. experimental group (persons with epilepsy) and normal controls in relation to socio-demographic

parameters. Except in income these two groups did not have any significant difference on any other socio-demographic parameters. In income epilepsy patients group has significantly lower level of monthly family income than the normal control group. In this table both continuous variables and categorical variables have been shown; in case of continuous variables T-test was used for group comparison, whereas in categorical variables Chi-square test was used.

Table 2 shows the obtained scores of stigma and social support. The measuring instruments used in the present study is, i.e. Stigma Scale of Epilepsy and Social Support questionnaire. These two groups had no significant difference in any of the scores of Stigma Scale of Epilepsy and Social Support questionnaire.

Table-1: Comparison of Socio-demographic variables of Experimental (Epilepsy) and Control Group (Normal)

Variables		Group N=60			
		Epilepsy (*n* = 30) Mean ± S.D.	Normal (*n* = 30) Mean ± S.D.	t/÷2 (*df*=58)	*P*
Age (In years)		28.30 ± 9.62	30.27 ± 8.71	-.829	.410 (NS)
Monthly Income (In Rs.)		5533.33 ± 3269.32	7900.00 ± 3959.32	-2.524	.014*
Sex	Male	22(73.3%)	24 (80.0%)	.373	.761(NS)
	Female	8(26.7%)	6 (20.0%)	(*df*=1)	
Occupation	Unemployed	1(3.3%)	—	6.526	.163(NS)
	Student	15(50.0%)	10 (33.3%)	(*df*=4)	
	Service	1(3.3%)	1 (3.3%)		
	Self-employed	8(26.6%)	17 (56.6%)		
	Housewife	5(16.8%)	2 (6.6%)		
Education	Up to Class-V	5(16.8%)	—	5.798	.122(NS)
	Class-VI-XII	18(60.0%)	20 (66.6%)	(*df*=3)	
	Graduation	5(16.8%)	8 (26.6)		
	Post –Graduation	2(6.4 %%)	2 (6.6%)		
Domicile	Rural	21(70.0%)	27 (90.0%)	3.750	.104(NS)
	Urban	9(30.0%)	3 (10.0%)	(*df*=1)	
Religion	Hindu	21(70.0%)	25 (83.3%)	1.191	.755(NS)
	Muslim	4(13.3%)	2 (6.6%)	(*df*=3)	
	Christian	2(6.7%)	1(3.3%)		
	Others	3(10.0%)	2(6.6%)		
Marital Status	Married	12(40.0%)	20 (66.6%)	4.815	.090(NS)
	Single	17(58.6%)	10 (33.3%)	(*df*=2)	
	Divorced	1(3.4%)	—		
Family Type	Joint	28(93.3%)	23 (77.6%)	3.268	.145(NS)
	Nuclear	2(6.7%)	7 (23.3%)	(*df*=1)	

* Significant at the 0.05 level. NS= Not significant

Table-2: Comparison of perceived stigma and social support of Experimental (Epilepsy) and Control Group (Normal)

Variable(scale of stigma & social support questionnaire)	GroupN = 60		t value (*df* = 58)	*P*
	Epilepsy (*n* = 30) Mean ± S.D.	Normal (*n* = 30) Mean ± S.D.		
Stigma Scale of Epilepsy	51.17 ± 10.91	46.80 ± 8.49	1.736	.088 (NS)
Social Support Questionnaire	48.33 ± 6.48	46.23 ± 5.50	1.353	.181(NS)

NS= Not significant

Discussion

Present study was a cross sectional comparative study on the Perceived social support and perceived stigma between adults with epilepsy and normal controls. The study sample comprised of 60 individuals (30 epilepsy patients and 30 normal individuals). The researcher of the present study could not find any similar study which has seen social support and stigma together in a single sample, but many studies have conducted having different variables(like, subjective well being, dysfunction, social support ,stigma etc.) at one time in a larger sample.[23,24] A study done having 60 epileptic patients for investigating the relationship between subjective feeling of stigmatization and a tendency to conceal the presence of the disorder, but they didn't compare patient group with the normal controls.[25] In another study 40 epileptic patients were studied to examine how they face stigmatization at their respective work place. In that study researcher found that 21 samples out of total 40 samples reported that they did not disclose their disorder to their employer because of fear of being stigmatized by office administration and colleagues.[26] Though the sample size of the present study was comparatively less (N=60; Epilepsy patients (n)=30 and Normal Persons(n) =30]. These could be stated as the strengths of the present study. In the present study the epilepsy group consists of 73.3% males and 26.7% females and the control group consist of 80.0% males and 20.0% females. The average age of the epilepsy patients was 28.30 ± 9.62 (in years) and that of the normal controls was 30.27 ± 8.71 (in years). There was no significant difference found in age distribution between the two groups. In the present study significant difference was found in monthly income among patients with epilepsy and with normal control. It was found that the monthly income of epilepsy patients was around Rs. 5,500 and for normal controls it was approximately around Rs.8, 000. This might be due to the reason that half of the participants of epilepsy were students and many of them were found to be unemployed in comparison to normal controls.

In the present study did not show any significant difference in stigma between epilepsy patients and normal controls. Study done by Fernandes et al investigated stigma among patients with epilepsy by applying the same tool used in the present study, and found epilepsy as a stigmatizing condition, and various factors such as education, socio-economic class can influence the stigma level in epilepsy patients[12]. The present study failed to replicate similar finding, can be because of the lesser sample size (n=30) compared to the sample size of the study done by Fernandes et al (n=1850). It may also be that the number of educated patients were high in number thus less stigma.[12]

The present study did not show any significant difference in social support between epilepsy patients and normal controls. A recent population based study by Strine showed that patients with epilepsy rate their social support as poor.[27] This difference can be due to the difference in sample size and the difference in the tools used to assess social support.

People with epilepsy, more frequently attributed stress at work and inadequate nutrients in the body as causes of this disorder.[28] It has found that education about epilepsy is the most effective enhancer of self-esteem in people with epilepsy and that their social support networks may be restricted to family, neighbors, and health-care providers, who compensate for a lack in friends and work associates.[29] Early identification and appropriate treatment of psychological factors in the people with epilepsy can positively influence medical outcome and quality of life.[30]

Conclusion

It can be concluded that majority of patients was male, belonging to joint family and was from rural background. There was significant difference found in monthly income between patients with epilepsy and normal control. There were no significant differences of social support and stigma of epilepsy in the both group.

Limitations

1. This study was a cross sectional design hence patients were assessed only once.
2. The sample used for the present study may not represent the entire

Population from which it has been drawn because of the small sample size and heterogeneity in relation to various socio-demographic and cultural variables.

3. Both sexes were not equally represented

in the selected samples.

Future Directions

1. In future, a similar type of study can be conducted on a larger sample with a prospective design.
2. In future stratification can be done to ensure appropriate representation of people of all socio-economic class.
3. Both sexes should be equally represented in the study sample.
4. To get the actual representation of the epilepsy patient and normal control, sample should be collected from community.

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Original Article

Multi-Informant Reporting of Behavioural and Emotional Problems of School Students

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Abstract

Background: *Behavioural and emotional problems in school children are assessed using various methods with different informants. However level of agreement varies between different informants. The present study explores the nature of problems in school students as reported by father and teacher and their inter-rater agreement.* ***Method****: A sample of 100 students was randomly selected from class 6th to 10th (age group 10-16 years) from a public school in Kashmir valley. Behavioural and emotional problems of students were assessed separately by fathers and teachers using Strength and Difficulties Questionnaire (SDQ).* ***Results****: Results revealed that fathers rated 28% students falling in abnormal category as compared to 25% by teachers. Significant relation was found between teacher and parent rating on pro-social behavior. Significant association of informant ratings and gender was also observed. Inter-rater agreement was calculated using Kappa coefficient which explained poor agreement ranging from .06 to .32.* ***Conclusion****: The assumption that multi-informant report is better in assessing the psychiatric morbidity among school students rather than single informant is verified as agreement among multi informants was marginal in the sample of the present study.*

Key words: *Multi-informants, SDQ, Behavioural and Emotional problems, School children.*

Introduction

Behavioural and emotional problems are common in school students. Research evidences from India shows the prevalence of behavioural and emotional problems in the range of 13.7%[1] to 50%[2] in school students. The manifestation of such problems at an early age may create considerable distress for the child and the family and also have a signiûcant impact on the child's social, emotional and psychological development.[3] Hence objective identiûcation of mental health problems at early age becomes important so as to minimise the likelihood of persistence of psychological problems into adulthood.[4]

Methods of clinical assessment that researchers and practitioners use to assess behavioural and emotional problems of school students are many. This commonly involves interview with the child, family and school; completion of rating scales and questionnaires and behavioural observations in a diagnostic setting.[4] Questionnaires completed by both the parent and child serve as valuable adjuncts to the clinical interview and are being increasingly used as screening tool in the assessment process[5]. However there is no single measure or method of assessing psychopathology in children that could provide a definitive or "gold standard" to gauge which school students are experiencing a given set of problems or disorders.[6] The lack of such a standard stems, in part, from the need to incorporate information from multiple informants like parents, teachers, and friends. Hence, multi-informant approach to the evaluation of psychological problems in students is considered as best-practice approach.[7]

However, Informant discrepancies have been found in virtually every method of clinical

assessment. In a meta-analysis of 119 studies, Achenbach, McConaughy, and Howell[8] identified what has come to be one of the most robust findings in clinical child research and different informants' (e.g., parents, children, teachers) ratings of social, emotional, or behaviour problems in children yielded discrepant results (e.g., r > 0.20). This finding has been replicated further in recent studies[9]. Moreover, discrepancies have also been found in the samples of informants encompassing diverse ethnic and cultural backgrounds[10]. The wide disparity in the reported prevalence rates of mental disorders in Indian studies have been attributed to methodological differences like varying modes of case ascertainment, sampling methods, instruments, the informants chosen across the studies, and unspecified clinical criteria for case ascertainment.[11] Inter informant disagreement may reflect reporter bias, measurement error, or variation in symptomology across settings.[12] Informants' reports often disagree in their perceived levels of a behavior and examining multiple reports in a single study often yielded inconsistent findings.[13]

Keeping in view the discrepancies among multi informants, the present study was designed to assess the association of informant's ratings of father and teachers with respect to behavioural and emotional problems in male and female school children of Kashmir valley.

Material and Methods

Sample: A sample of 100 students with equal number of males and females were randomly selected in the age range of 10-16 years. These students were from class 6th to 10th of two schools of District Shopian in Kashmir. Majority of children were middle born (47%) and 49% were from low economic background (<5000 Rs/month) and have spent at least six months in school. Fathers and teachers of these children were the main respondents.

Measure: A 25 item Parent and Teacher version of Strengths and Difficulties Questionnaire (SDQ) developed by Goodman (1997)[14] was the main tool of data collection. It can be administered to parents or teachers of 3– 16 year olds or directly to 11 – 16 year old children to screen emotional and behavioural difficulties. The 25 items are further divided into 5 subscales which measure emotional symptoms, conduct problems, hyperactivity, peer problems and prosocial behaviour; all but the last one are summed to generate a total difficulties scores. Each of the 25 items is rated as being rated as *Not true* (0), *Somewhat true* (1), or *Certainly true* (2), and each of the SDQ scale consists of five items with positive and negative valences, thus yielding scores between 0 and 10. The total difficulties score can range from 0 to 40.

Procedure: The process of data collection started by contacting the school principals, explaining the purpose of the study and removing their apprehensions, if any. In every class, students were selected randomly and their fathers were contacted in parental meetings. They were informed about the purpose and requested to give their consent and completing the Strength and Difficulties questionnaire (Parent version). Similarly a Teacher version was filled by the class teachers who have spent at least six months in the school.

Statistical Analysis: Percentage of students falling in normal, borderline and abnormal categories on SDQ, based on Goodman's cut off criteria (>16) was calculated. Chi square test was used to find the association of father and teachers reporting on various subscales of SDQ and with gender. Kappa coefficient was used to assess the inter-rater agreement. Data was analyzed using SPSS version 16.

Results

Table 1 shows that on SDQ fathers rated 50% students as normal, 22% as borderline and 28% as abnormal, whereas teachers rated 61% as normal, 14% as borderline and 25% as abnormal. Parents reported more students having emotional problems (49%), followed by conduct problems (38%) and peer problem (23%). However teachers reported high percentage of conduct problems (24%) followed by emotional (18%) and hyperactivity (14%) in school students. In total sample significant effect of rating by fathers and teachers was not found on any of the subscales of SDQ.

Table 2 shows that both father and teacher reported conduct problems to be more in male students as compared to other problems. On chi square test, there was found significant association in the reporting of father and teacher on prosocial behaviour, χ 2(4) = 11.29, p < .01, and on total

Table 1: Percentage and χ^2 value of students falling under Normal, Borderline and Abnormal categories for various Behavioural and Emotional Problems as reported by informants (N=100)

Subscales of SDQ	Informants	Normal(%)	Borderline(%)	Abnormal(%)	χ^2 (p-value)
Emotional Problem	Father	34	17	49	5.92
	Teacher	68	14	18	(.19)
Conduct Problem	Father	44	18	38	2.64
	Teacher	68	8	24	(.65)
Hyperactivity	Father	91	4	5	1.01
	Teacher	78	8	14	(1.0)
Peer Problem	Father	51	26	23	4.53
	Teacher	75	12	13	(.32)
Pro-social Behaviour	Father	79	14	7	9.01
	Teacher	66	26	8	(.03)
Total Difficulties	Father	50	22	28	6.09
	Teacher	61	14	25	(.18)

Table 2: Percentage and χ^2 value of male students falling under Normal, Borderline and Abnormal categories for various Behavioural and Emotional Problems as reported by informants (N=50)

Subscales of SDQ	Informants	Male			
		Normal (%)	Borderline (%)	Abnormal (%)	χ^2 (p-value)
Emotional problem	Father	32.7	24.5	42.9	4.65
	Teacher	81.6	4.1	14.3	(.22)
Conduct problem	Father	34.7	18.4	46.9	2.19
	Teacher	75.5	6.1	18.4	(.77)
Hyperactivity	Father	83.7	6.1	10.2	1.52
	Teacher	87.8	4.1	8.2	(1.00)
Peer problem	Father	38.8	36.7	24.5	3.11
	Teacher	79.6	10.2	10.2	(.52)
Pro-social behaviour	Father	81.6	12.2	6.1	11.29
	Teacher	73.5	22.4	4.1	(.01)
Total difficulties	Father	36.7	28.6	34.7	10.22
	Teacher	73.5	10.2	16.3	(.01)

difficulties, χ 2(4) = 10.22, p < .01.

Table 3 shows that father reported emotional problems more in female students than other problems while as teacher reported conduct problems more in female students than other problems. On chi square test, there was insignificant association in the reporting of father and teacher on various sub problems of female students on SDQ.

Table 4 and 5 shows that chi square test was used to determine association between SDQ subscales and gender for both father and teacher version separately. The results showed that there was significant association between emotional problems rated by teachers and female gender (χ^2 (2) = 10.11, p = .006) as well as for total difficulties (χ^2 (2) = 6.33, p =.04). Likewise, in parent reported SDQ, significant association was found with male gender in hyperactivity (χ^2 (2) = 10.61, p =.01), on peer problems (χ^2 (2) = 7.17, p =.03), as well as on total difficulties (χ^2 (2) = 6.81, p =.03). All other associations were found to be statistically insignificant.

Overall proportion of agreement as well as chance corrected Kappa coefficient was computed to estimate the agreement between father and teacher reporting (see Table 6). Overall proportion of agreement was best found in prosocial behaviour (p_o= .87) and least found in emotional problems (p_o=.49).

Kappa coefficients range from 0 to 1, with 1 indicating perfect agreement. In general, values less than 0.20 indicate poor agreement, values between 0.20-0.40 indicate fair agreement, values between 0.41 and 0.60 indicate moderate agreement, values

Table 3: Percentage and χ^2 value of female students falling under Normal, Borderline and Abnormal categories for various Behavioural and Emotional Problems as reported by informants (N=50)

Subscales of SDQ	Informants	Male			
		Normal (%)	Borderline (%)	Abnormal (%)	χ^2 (p-value)
Emotional problem	Father	35.3	9.8	54.9	6.98
	Teacher	54.9	23.5	21.6	(.10)
Conduct problem	Father	52.9	17.6	29.4	1.16
	Teacher	60.8	9.8	29.4	(.93)
Hyperactivity	Father	98.0	2.0	0.0	1.26
	Teacher	68.6	11.8	19.6	(1.00)
Peer problem	Father	62.7	15.7	21.6	7.63
	Teacher	70.6	13.7	15.7	(.06)
Pro-social behaviour	Father	76.5	15.7	21.6	5.04
	Teacher	58.8	29.4	11.8	(.22)
Total difficulties	Father	62.7	15.7	21.6	2.82
	Teacher	49.0	17.6	33.3	(.62)

Table 4: Percentage and χ^2 value of male and female students falling under Normal, Borderline and Abnormal categories for various Behavioural and Emotional Problems as reported by father (N=100)

Subscales of SDQ	Gender	Male			
		Normal (%)	Borderline (%)	Abnormal (%)	χ^2 (p-value)
Emotional Problems	Male	80	6	14	10.11
	Female	56	22	22	(.006)
Conduct Problems	Male	76	6	18	2.49
	Female	60	10	30	(.28)
Hyperactivity	Male	88	4	4	5.35
	Female	68	12	20	(.07)
Peer Problem	Male	80	10	10	1.11
	Female	70	14	16	(.57)
Prosocial Behaviour	Male	74	22	4	3.12
	Female	58	30	12	(.21)
Total Difficulties	Male	74	10	16	6.33
	Female	48	18	34	(.04)

Table 5: Percentage and χ^2 value of male and female students falling under Normal, Borderline and Abnormal categories for various Behavioural and Emotional Problems as reported by teacher (N=100)

Subscales of SDQ	Gender	Male			
		Normal (%)	Borderline (%)	Abnormal (%)	χ^2 (p-value)
Emotional Problems	Male	34	24	42	3.96
	Female	34	10	56	(.14)
Conduct Problems	Male	36	18	46	3.92
	Female	52	18	30	(.14)
Hyperactivity	Male	78	12	10	10.61
	Female	98	2	0	(.01)
Peer Problem	Male	40	36	24	7.17
	Female	62	16	22	(.03)
Prosocial Behaviour	Male	82	12	6	0.40
	Female	80	16	8	(.82)
Total Difficulties	Male	38	28	34	6.81
	Female	62	16	22	(.03)

Table 6: Proportion of Agreement, Kappa and p-values for father – teacher rating on scales of SDQ (N=100)

Subscales of SDQ	Proportion of Agreement	Kappa coefficient	p-value
Emotional Problems	.49	.14	.50
Conduct Problems	.54	.09	.18
Hyperactivity	.81	.10	.11
Peer Problems	.72	.18	.20
Pro-social Behaviour	.87	.32	.003
Total Difficulties	.55	.06	.40

between 0.61 and 0.80 indicate good agreement and those above 0.81 indicate very good agreement.[15] According to Altman's criteria, Table 4 shows that there is poor agreement between father and teacher reporting on various subscales of SDQ except that of prosocial behaviour in which it was found fair (k =.22, $p < .01$)

Discussion

Overall findings suggest a difference in reporting of potential cases by different informants. Fathers tend to report problems more prevalent among students than teachers. Similar findings were found in Cury and Golfeto (2003)[16] study in which parents rated high scorings (18.7%) as compared to teachers report (8.25%) for the total difficulties. It could also be due to parental stress in Kashmir which is found to have positive relation between parent reported level of child stress and informant discrepancies on rating of child internalising and externalising problems[17] as well as parental sensitivity to various aspects of child's behaviour[18]. When comparing on the basis of gender, parents report problems more prevalent in boys while as teacher reported more in girls. Overall father reported more male students with total difficulties as compared to teacher who reported more female students with total difficulties. In Kashmir, parents show less control on boys than girls. Low parental acceptance of the child is related to greater parent-teacher discrepencies on child externalising and internalising problems[17] While assessing the nature of problems among students as reported by different informants, it was found that conduct problem were more prevalent among all other problems when reported by teachers, although parents report that majority of children have emotional problems. Teachers high rating on conduct disorder and ADHD could be partly because teachers are more aware of such difficulties and more influenced by their knowledge of these difficulties when rating the children. School related problems appear more strongly associated with teacher than with parental ratings of emotional distress.[19] Children are more attached with parents and their emotional problems are more vivid to their parents.[20] Cury and Golfeto (2003)[16] also found emotional problems (30.8%) reported more than that was reported by teachers (1.83%). Similarly higher rates of internalising problems (12.2 percent) compared to externalising problems (4.9 percent), were found in other study[21]

The inter-rater agreement was found poor. Moderate to low correlation for parent –teacher rating was reported by Eric et al[22], as well as Crane et al[23] who reported parent–teacher agreement to be moderate (rs=.20–.28). Partly it can be due to changing circumstance and lack of consistency in behaviour of subjects. Rating could be affected by raters own biases or due to so-called 'halo effects'.[24] When parents reported the most symptoms, low parental educational level, low income and male gender of the child played an additional role.[25]

The study has some limitations as well. It comprised of small sample and was limited to only two schools which limits its chances of generalisation. Self reports could have been incorporated as well for better understanding.

In conclusion, the study gives us an idea about the ratings given by different informants and possible effects it could have upon the final results. The findings indicate that assessment may benefit from a shift to multiple informant reports, as different informants' ratings may contain more information than single informant. Hence the study highlights the need of multi-informant rating while assessing children and need for preventive and well as other secondary measures which need to be taken to save children form such problems.

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Original Article

Medication Errors in Dentistry — A Cross-Sectional Study

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Abstract

Background: *Prescription errors are quite frequent in routine dental practice but are hardly ever reported. These errors cause significant morbidity, mortality and health care costs to the society.* ***Aims & Objectives:*** *To analyze current prescription writing practices among dentists and to identify and quantify various types of medication errors in dental prescriptions.* ***Materials and Methods:*** *A cross-sectional study of prescription errors was carried out in the extra-mural dental prescriptions brought by the patients attending the Dental Outpatients Department of the M.M College of Dental Sciences & Research (M.M.C.D.S.R), Ambala, Haryana. The study period ranged from 1st September, 2013 to 1st December, 2013. Microsoft Excel was used for data analysis. The WHO guidelines for prescription writing were used as a standard while making an assessment of the results.* ***Results:*** *Most common groups of drugs prescribed by dental surgeons were NSAIDs, antimicrobials, antiseptics and multivitamins. NSAIDs- 86%; Antimicrobials- 85%; Antiseptics- 8.6%; Multivitamins- 12.3%. The average number of drugs prescribed per patient was 3 and the average number of antimicrobial prescribed per patient was 1. Most of the prescriptions analyzed, showed polypharmacy, illegible handwriting, lack of necessary details pertaining to the patients (address, weight), as well as those pertaining to the prescriber (signature, contact details, registration number) and necessary instructions for the patient regarding taking prescribed drugs and advice for follow up. In the extramural prescriptions analyzed, the most common error was prescription of "drug without dose" (30 %).* ***Conclusions:*** *Routine dental prescriptions exhibit large number of easily identifiable errors which are preventable. The need of the hour is to promote rational drug prescribing practices among dentists, encouraging them to detect and report prescription errors encountered by them. This approach will enrich our existing research base about this hitherto neglected domain of dental practice thereby helping us in developing and implementing effective strategies to combat this menace of prescription errors.*

Keywords: *Prescription error, Dentists, Cross-sectional study, Extra-mural prescriptions*

Introduction

The issue of medication prescribing errors received little attention until 1962, when Barker and McConnell in the United States of America (USA) first demonstrated that medication errors occur more frequently than suspected. They estimated a rate of 16 errors per 100 doses[1] and suggested that the apparent increasing rate of prescribing errors was proportionate to the increasing number of drugs that

were available. As the awareness about the potential implications of prescription errors grew, dedicated systems for reporting medication errors were set up in the USA and Europe.[2,3] Health care professionals who encounter actual or potential medication errors were encouraged to report them confidentially or anonymously if preferred. In 1995, a multidisciplinary group of 17 national organizations formed the National Coordinating Council for Medication Error Reporting and Prevention. The National Coordinating Council for Medication Error Reporting and Prevention defines a medication error as any pre-ventable event that may cause or lead to inappropriate medication use or patient harm, while the medication is in the control of the health-care professional, patient, or consumer.[3]

Prescription errors are reported to be quite frequent in clinical practice.[4] A survey from Italy had revealed that overall 23.9% of prescriptions were illegible and 29.9% of prescriptions were incomplete.[4] A similar survey from Rosa et al reported that on an average, 3.3 errors were observed in each prescription order form.[5] Most of the prescription errors were due to omissions of dosage, administration route, and length of treatment and may potentially cause harm to the elderly outpatients. Even advanced nations like UK had reported 15% of the prescription to be containing one or more errors in critical care units.[6]

Adverse drug events and prescription errors have received extensive study recently in a variety of clinical population, though compared to many other areas relatively little work has focused on this area in dentistry.

A comprehensive study from India reporting the lacunae in the prescription writing trends of dentists have been lacking. The present study was undertaken to understand the current prescription writing practices among dentists and to detect the common errors in them at a tertiary level dental care centre situated in Ambala, Haryana.

Aims and Objectives

1. To Analyze current prescription writing practices among Dentists
2. To identify and quantify various types of medication errors in Dental prescriptions.

Material and Methods

A cross-sectional study of prescription errors was carried out in the extra-mural Dental prescriptions brought by the patients attending the dental outpatient's department of the M.M. College of Dental Sciences and Research (M.M.C.D.S.R), Ambala, Haryana. The study period ranged from 1st September, 2013 to 1st December, 2013.

Data collection

The data obtained was entered into a semi structured proforma. The proforma had four parts. Important information regarding the patient, doctor, drug and the overall impression of the prescription were recorded in these four parts separately. Patient's details that were sought included the name, age, sex, weight and address. The prescriptions were further checked for the following details of the prescribing authority: name, qualification, complete address, phone/mobile number, signature and registration number of the doctor. The drug name, dose, frequency and route of administration, instructions and follow up advice to patient were recorded. The overall clarity, readability, presence of date was included in a separate section of the proforma. The details pertaining to the patients and the doctors were kept confidential. The WHO guideline[7] for prescription writing was taken as a standard while making an assessment of the results.

Operational definitions and classification system used in our study:

Prescription Error

For the purpose of the study, following operational definition of "prescription error" was used: "A clinically meaningful prescribing error occurs when, as a result of a prescribing decision or prescription writing process, there is an unintentional significant (1) reduction in the probability of treatment being timely and effective or (2) increase in the risk of harm when compared with gener-ally accepted practice."[8]

Classification of prescription errors[8,9,10,11]

The prescription errors can be categorized as follows:

1. ***Illegible prescription****:* any item of medical prescription in which information was illegible.
2. ***Prescription with duplicate item****:* one or more drugs prescribed more than once to

the same patient taking the dosage into account.

3. ***Drugs with the same indication:*** prescription of two or more drugs with the same indication keeping in view the respective mechanisms of action.
4. ***Wrong frequency:*** prescription of a drug with a frequency not consistent with literature.
5. ***Drug without administration route:*** prescription of a drug with no mention of administration route.
6. ***Drug without doses:*** prescription of a drug without a specified dose or dosage.
7. ***Dose higher than recommended:*** prescription of a drug at a dose higher than the one mentioned in literature.
8. ***Wrong drug:*** prescription of a wrong drug.
9. ***Wrong dose:*** prescription of a drug with inexistent dose, according to the literature.
10. ***Drug-drug Interactions:*** prescription with potential drug-drug interactions or incompatibilities.
11. ***Prescriber signature missing***
12. ***Abbreviated and non standard drug names***
13. ***Error prone abbreviations, symbols and dose designations***

The Drug Drug interactions were checked using Medscape drug interaction checker.[12]

Statistical Analysis

Microsoft Excel was used for data analysis. The WHO guideline[7] for prescription writing was used as a standard while making an assessment of the results.

Results

Out of the 100 patients, 61 were male patients. The age distribution showed that 25 patients were below the age of 18 years; 20 patients were in the 18-60 years age group and 55 patients were over 60 years of age.

Most common groups of drugs prescribed by dental surgeons were NSAIDs, antimicrobials, antiseptics and multivitamins. NSAIDs-86%; Antimicrobials-85%; Antiseptics-8.6%; Multivitamins-12.3%. The average number of drugs prescribed per patient was 3 and the average number of antimicrobial prescribed per patient was 1. Most of the prescriptions analyzed, showed polypharmacy, illegible handwriting, lack of necessary details pertaining to the patients (address, weight), as well as those pertaining to the prescriber (signature, contact details, registration number) and necessary instructions for the patient regarding taking prescribed drugs and advice for follow up. In the extramural prescriptions analyzed, the most common error was prescription of "drug without dose" (30 %).

Date of consultation was clearly written on 99%prescriptions. The name, age and sex of the patient were mentioned in majority of the prescriptions (Table 1). None of the prescriptions analyzed had mention of address and weight of the patient. The name of the prescribing doctor was found in 84 % of the prescriptions analyzed. Qualification of the prescribing authority was missing in more than half of the prescriptions, while details like address and telephone/mobile numbers were mentioned in only about one-fourth of the prescriptions (Table 1). Over 86 % of the prescriptions had signature of the prescribing authority, registration number was not mentioned in over three-fourth of the prescriptions. None of the prescriptions had mention of generic names of drugs. Details like strength, route of administration and duration of treatment was clearly mentioned in over 80 % of the prescriptions. The dose 'of the drug was clearly written in only 67 % of the prescriptions

Table 1. A representation of patient and prescriber/doctor information on the 100 prescriptions analyzed

	Yes n (%)	No n (%)
Patient		
Name	100 (100)	0 (0)
Age	90 (90)	10 (10)
Sex	95 (95)	5 (5)
Address	0 (0)	100 (100)
Weight	0 (0)	100 (100)
Prescriber/Doctor		
Name	84 (84)	16 (16)
Qualification	45 (45)	55 (55)
Address	25 (25)	75 (75)
Telephone/Mobile Number	15 (15)	85 (85)
Signature	86 (86)	14 (14)
Registration Number	25 (25)	75 (75)

Table 2. A representation of drug information on the 100 prescriptions analyzed

	Yes n (%)	No n (%)
Name		
Brand	100 (100)	0 (0)
Generic	0 (0)	100 (100)
Strength	80 (80)	20 (20)
Dose		
Clearly mentioned	70 (70)	30 (30)
Correct	48 (48)	52 (52)
Route of Administration	80 (80)	20 (20)
Duration	75 (75)	25 (25)
Instructions for Patients	43 (43)	57 (57)
Advice for follow up	10 (10)	90 (90)

while it was correct only in 45 % of total prescriptions. More than half of the prescriptions did not contain instructions for patients while the advice for follow up was missing in over 90 % the prescriptions (Table 2).

A total of 13 types of errors were detected in the 100 prescriptions analyzed and these are illustrated in the table 3 with their respective frequencies. A significant number of the prescriptions (27%) were written in illegible handwriting.

In the 100 extramural prescriptions analyzed, among the various categories of prescription errors identified, the most common error was prescription of "drug without dose" (35 %) followed closely by "the use of Error prone abbreviations, symbols and dose designations" (30%). The least common prescription error was the prescription of a "wrong drug" (3 %). (Table 3)

(Most of the prescriptions analyzed had more than more than one category of prescription errors and therefore the total number of identified prescription errors is more than the total number of prescriptions analyzed)

Table 3. Respective frequencies of various categories of prescription errors found in 100 prescriptions analyzed

Category of Prescription Error	Observed Frequency %
Illegible prescription	27
Prescription with duplicate item	16
Drugs with the same indication	15
Wrong frequency	4.5
Drug without administration route	20
Drug without doses	35
Dose higher than recommended	14
Wrong drug	3
Wrong dose	13
Drug-drug Interactions	4
Prescriber signature missing	8
Abbreviated and non standard drug names	8
Error prone abbreviations, symbols and dose designations	30

Discussion

This study was an attempt to find out the existing pattern of prescription writing among dentists and to identify as well as to quantify various types of prescription errors in them.

The concurrent use of multiple psychoactive medications in a single patient, i.e. polypharmacy was clearly evident in almost all of the prescriptions analyzed. It is considered to be an increasingly common and debatable contemporary practice in clinical practice. Concerns with polypharmacy include the possibility of cumulative toxicity[13] as well as adherence issues which emerge with increasing regimen complexity.[14] There is a need to develop evidence-based strategies for polypharmacy to avoid most of the above mentioned problems.[15] There was a relative lack of information about the patient and prescriber in the prescriptions analyzed. The date of writing the prescription was missing in 4 % of the prescriptions which was considerably lower than the rates of 56.1 % reported by Calligaris et al. [4] Illegible handwriting was identified in 22 % of the prescriptions which was comparatively higher than the rates suggested by studies conducted in the US (10%)[16] and the UK (15%).[17] Among reasons offered by doctors for poor handwriting, heavy workload was the most common.[18] All the prescriptions analyzed had the name of the patient, a finding similar to that of Irshaid et al[19] while the mention of the age (90 %) and sex (95 %) of the patient in our study was considerably higher than that in the above mentioned study. Details of address and weight of the patients was absent in all the prescriptions. Information about address is important to decide when patient is to be called for follow up. Weight of the patient determines the actual quantity of the drug per dose and hence it must be mentioned in the prescription.

As regards the information regarding the prescriber, plenty of deficiencies were identified. 18

% of prescriptions did not bear the name while more than 50% did not mention the qualification. Address details of the doctor were not available in three-fourth of the prescriptions while 85%. did not have any mention of the telephone/mobile number of the doctor. Signature of the prescribing authority was missing in 8 % of the prescriptions; only one-fourth of the prescriptions mentioned the registration number of the prescribing person. The lacunae in providing these details can be considered a violation of WHO guidelines on prescription writing.[7] The study from Saudi Arabia revealed 16.7% of prescriptions deficient in the prescriber's name and 18.1% deficient in the prescriber signature.[19] These findings are distinctly in contrast with the results of our study. Doctor's complete address details are important as the patient/caregiver may need to contact him/her during an emergency. Not mentioning qualification of the prescriber raises doubts about his/her credibility. Generic name of the drugs was not mentioned in any of the prescriptions analysed, a finding consistent with glaringly uncommon rates of mention of generic names in prescriptions in the studies by Irshaid et al[19] and Pandey et al.[20] In our study, less than half of the prescriptions had mention of instructions for the patients, while advice for follow up was missing in most of them (90 %).In the study by Irshaid et al [19] almost 90 % of the prescriptions had partial instructions for the patient. Prescription of drug without dose was observed to be the most common prescription error with a frequency of 35%. In our study, use of abbreviated and non standard drug names (8%) and error prone abbreviations, symbols and dose designations (30%) appeared as one of the major categories of prescription error. These findings are in contrast to the results of Sapkota et al[21] who in their study on prescription error in elderly found considerably rates of 11.76% for use of non-standard drug names and 0.65 % for use of error-prone abbreviations, symbols and dose designations. The least common prescription error was the prescription of a wrong drug (2%), a finding similar that reported by Pote et al.[22]

Our study had a few limitations. The design of our study was cross-sectional; the sample for the study was not homogenous; and the sample size was small. However, despite these limitations, the study has provided valuable insights into the existing prescription pattern among dentists and the glaringly high prevalence of identifiable prescription errors which are essentially preventable.

Conclusions and Future Directions

The drug prescription pattern observed in our study suggests the need to promote rational drug prescribing among clinicians in general and dentists in particular. A high number of prescription errors were found. Whilst many of these were minor and unlikely to have had serious consequences, some were of potentially great significance and may represent only the tip of iceberg. Prescription errors are a source of considerable mortality, morbidity, and health-care costs in the world today but the important thing is that they can be prevented. Educational intervention programs and computer aided prescription order entry can substantially contribute in the lowering of such errors. Prospective observational studies with robust methodology are needed to more accurately determine the frequency of prescription errors in dentistry.

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Original Article

Vitamin D Deficiency among Psychiatric Outpatients

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Abstract

Vitamin D is not only known for building up strong bones but also keeping healthy mind. Many of the recent researches have shown correlation of the psychiatric illness with the Vitamin D deficiency. 25-hydroxyvitamin D (25[OH]D) is the principal vitamin D metabolite measured to determine vitamin D status in the body. Although previous researches have found out the Vitamin D levels in specific psychiatric disorders but the presentstudy is a unique effort and preliminary kind of study in this regard. It examined the prevalence of Vitamin D levels in a varied group of psychiatric out-patients.

Keywords: *Vitamin D deficiency, depression, somatoform*

Introduction

Vitamin D is well-known for maintaining calcium homeostasis and healthybones. The inadequate levels of vitamin D have been associated predominantlywith bone disorders of defective mineralization such as rickets, osteomalacia, and osteoporosis.[1]

The research has also shown the correlation of low levels of Vitamin D with the metabolic disorders. An epidemiologic study found that adults with 25-hydroxyvitamin D (25[OH]D) levels < 21 ng/mL had an increased risk of hypertension, diabetes, obesity, and dyslipidemia.[2]

The recent findings that most body tissues and cells including the brain have vitamin D receptors, has provided new insights into the function of this vitamin.[3]

The role of Vitamin D in psychiatric illnesses is suggested by specific expression of vitamin D receptors in the cingulate cortex, thalamus, cerebellum, amygdala, and hippocampus.[4] Most of these regions also express 1α-hydroxylase enzymes capable of metabolizing 25(OH)D to 1,25(OH)$2D_3$, which suggests that vitamin D may have an autocrine or paracrine function in brain.[5] Vitamin D also regulates expression of tyrosine hydroxylase which is the rate-limiting enzyme in the biosynthesis of dopamine, norepinephrine, and epinephrine.[6] Vitamin D promotes neuronal survival by inhibiting oxidative pathways in the brain through inhibition of inducible nitric oxide synthase[7] (reducing free radical formation) and upregulation of γ-glutamyl transpeptidase[8] (increasing antioxidant production).

The lack of exposure to sunlight and dietary deficiency of Vitamin Dare common in patients developing psychiatric disorders.[9] Dark skin, use of sun blocks, certain religious customs such as purdahsystem also make a person vulnerable to develop Vitamin D deficiency.[10]

Low levels of vitamin D are associated with depression, cognitive dysfunctionand seasonal affective disorder. Evidence also suggests a potential link between vitamin D deficiency and psychotic disorders. It is not clear whether vitamin D deficiency is a cause or effect of depression. Limited research suggests vitamin D supplementation might have a role in treating depression and Seasonal Affective Disorder. The recent data also suggest

high prevalence of Vitamin D among patients with Bipolar Disorder. The supplements of Vitamin D have proven to be beneficial in the above psychiatric disorders.

Materials and Methods

After taking approval from ethical committee of the institute,the present study was conducted in patients attending Psychiatry OPD at Guru TegBahadur Hospital, Delhi.It is a crossectional study involving a sample of 112patients done over a period of year 2013 where every patient had detailed Psychiatric evaluation. The patients were diagnosed with psychiatric illness as per ICD-10. After taking consent from the patients, all patients were screened for Vitamin D levels. The patients were tested for serum 25(OH)D levels using radioimmunoassay. Vitamin D levels are expressed as ng/mL or nmol/L; the conversion factor from ng/mL to nmol/L is 2.496.The patients were classified into three categories having different levels of vitamin D as described underneath.

Deficient < 25 nmol/L
Insufficeint 25-50 nmol/L
Normal > 50 nmol/L.

Inclusion criteria

1. All patients attending Psychiatry OPD in the age group of 18- 60 years who consented to participate in the study.

Exclusion criteria

1. Patients with chronic medical illness.
2. Patients who refuse to give consent.
3. Patients who refuse to undergo laboratory test for Vitamin D.

Results

Out of 120 cases, 112 were enrolled in the study following inclusion/exclusion criteria.64 (57.14%) males and 48 (42.85%) females participated in the study. According to ICD-10 diagnosis 22 (19.64%) patients had somatoform disorder out of which 20 (17.85%) had deficient levels and 2 (1.78%) had insufficient levels of Vitamin D. 16 patients (14.28%) had a diagnosis of Depressive disorder out of which most of the patients (N = 14, 12.5%) had deficient levels and 1 (0.89%) had insufficient level and only 1 (0.89%) had normal Vitamin D. Out of 10 (8.9%) patients with Bipolar affective Disorder, 8 (7.14%) had deficient levels and 3 (2.68%) fell into the insufficient Vitamin D level category. 13 patients (11.60%) of Schizophrenia and psychotic spectrum disorder were enrolled for Vit D. Among them 10 (8.92%) patients were found to have deficiency and 3 (2.68%) had insufficient levels of VitD.The 20 (17.85%) patients of Generalized anxiety disorder were screened and 15 (13.39%) reported deficiency and 4 (3.57%) had insufficient levels. But only 1 (0.89%) patient of GAD reported to have normal value of Vitamin D. All the 4 (3.57%) patients of Phobic disorder had deficiency of Vitamin D. When 8 (7.14%) patients of the OCD were subject to testing, 6 (5.35%) had deficient and 2 (1.78%) had insufficient levels of Vitamin D. out of the 3 (2.68%) patients with dissociative disorder 2 (1.78%) had deficiency and 1 (0.89%) had insufficient levels of Vitamin D. Even the patients with the sleep disorders (N=4, 3.57%) had deficient (N=3, 2.68%) and insufficient levels (N=1, 0.89%). Not only this the patients with the alcohol dependence too reported with low levels of Vitamin D. 12 (10.71%)

Table showing total 112 cases (Males-64; females-48) evaluated for Vitamin D deficiency

Psychiatric Disorder	N (%)	Deficient (%)	Insufficient (%)	Normal (%)
Schizophrenia	13 (11.60%)	10 (8.92%)	3 (2.68%)	—
Bipolar Disorder	10 (8.9%)	8 (7.14%)	2 (1.78%)	—
Unipolar Depression	16 (14.28%)	14 (12.5%)	1 (0.89%)	1 (0.89%)
GAD	20 (17.85%)	15 (13.39%)	4 (3.57%)	1 (0.89%)
Phobic Disorder	4 (3.57%)	4 (3.57%)	—	—
OCD	8 (7.14%)	6 (5.35%)	2 (1.78%)	—
Somatoform Disorder	22 (19.64%)	20 (17.85%)	2 (1.78%)	—
Sleep disorder	4 (3.57%)	3 (2.68%)	1 (0.89%)	—
Dissociative disorder	3 (2.68%)	2 (1.78%)	1 (0.89%)	—
Alcohol dependence	12 (10.71%)	10 (8.92%)	2 (1.78%)	—

alcoholicdependent patients had deficiency reporting in 10 (8.92%) patients and 2 (1.78%) had insufficient levels of 25 OH(D).

Discussion

In our study the majority of the patients of somatoform followed by GAD, depressive disorder and then psychotic patients and bipolar affective disorders reportedhigh prevalence of low levels of Vitamin D. This is in accordance with the previous population based researches done sporadically in specific psychiatricdisorders showing the deficiency of Vitamin D.

The Third National Health and Nutrition Examination Survey studied in a sample of 7,970 non-institutionalized U.S. residents aged 15 to 39 that individuals with serum vitamin D d"50 nmol/L are at a significantly higher risk of developing depression than those with vitamin D e"75 nmol/L.[11] The study done by Mayo clinic has shown correlation of depression with deficient levels of Vitamin D.[12] Wilkins et al found out in a group of elderly subjects the mean Vitamin D levels to be 18.58 ng/mL, with 58% subjects with levels below 20 ng/mL reporting frank deficiency. It was thus concluded that, low vitamin D was strongly associated with the presence of mood disorder (oddsratio 11.7, 95% CI2.0 - 66.9).[13] Gloth et al randomized the subjects with seasonal affective disorder. Seven subjects received phototherapy and 8 were given 100,000 IU of vitamin D. It was found out that the subjects givenVitamin D showed improvement rather than those who were subject to phototherapy.[14]

A research in Norway has also shown consistently low levels of 25OH(D) among patients presenting with psychotic symptoms.[15] A study of 8,411 Swedish women also revealed that low vitamin D levels were associated with psychotic symptoms.[16]

One of the recent researches have been done in Waikato region in New Zealand. Among the 19 participants with severe deficiency of Vitamin D (< 25 nM), 13 had a diagnosis of schizophrenia. This equates to 34% of participants with that diagnosis, compared to 9.4% of other participants (n = 102, p = 0.003). Taken together, disorders in the schizophrenia spectrum (schizophrenia and schizoaffective disorder) were over-represented among those with Vitamin D deficiency and had markedly reduced average levels (n = 49, mean = 36.4 nM, one-sample t = 6.58, p < 0.001) compared to the other diagnostic groups, none of which showed a mean difference from the deficiency threshold of 50 nM.[17]

A research done in Italy concluded that persons with severe vitamin D deficiency (< 25 nmol/L) had a higher risk of substantial cognitive decline than those with sufficient levels (≥ 75 nmol/L) as is evident on scores of Mini mental status examination.[18,19]

Conclusion

The prevalence of Vitamin D is high among the psychiatric patients. The patients with vitamin D deficiency often present with vague symptoms which includes tiredness and general aches and pains. Majority of the patients belonged to the group of somatoform disorder who often present with the vague body aches. But even the people without any symptoms were found to have Vitamin D deficiency. They need to be addressed and be given Vitamin D supplements.

Limitations

The sampling done was non-randomized because of which the finding of the study can only be representative of the study population and cannot be extrapolated to the community at large. Longitudinal studies involving larger samples selected by systematic sampling methods would be needed to estimate true prevalence of Vitamin D levels in these patients.

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Psychomicrobiology

Depression and Inflammatory Markers: The Clinical Correlation

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Introduction

Depression

Major Depressive Disorder (MDD) (also known as clinical depression, major depression) is a mental disorder characterized by a pervasive and persistent low mood that is accompanied by low self-esteem and by a loss of interest or pleasure in normally enjoyable activities. Major depressive disorder is a disabling condition that adversely affects a person's family, work or school life, sleeping and eating habits, and general health. In the United States, around 3.4% of people with major depression commit suicide, and up to 60% of people who commit suicide had depression or another mood disorder[1].

The diagnosis of major depressive disorder is based on the patient's self-reported experiences, behavior reported by relatives or friends, and a mental status examination. There is no laboratory test for major depression, although physicians generally request tests for physical conditions that may cause similar symptoms. The most common time of onset is between the ages of 20 and 30 years, with a later peak between 30 and 40 years[2].

Typically, patients are treated with antidepressant medication and, in many cases, also receive psychotherapy or counseling, although the effectiveness of medication for mild or moderate cases is questionable[3]. The course of the disorder varies widely, from one episode lasting weeks to a lifelong disorder with recurrent major depressive episodes. Depressed individuals have shorter life expectancies than those without depression, in part because of greater susceptibility to medical illnesses and suicide.

Symptoms and Signs

A person having a major depressive episode usually exhibits a very low mood, which pervades all aspects of life, and an inability to experience pleasure in activities that were formerly enjoyed. Depressed people may be preoccupied with, or ruminate over, thoughts and feelings of worthlessness, inappropriate guilt or regret, helplessness, hopelessness, and self-hatred. In severe cases, there may be symptoms of psychosis such as delusions or, less commonly, hallucinations. Other symptoms of depression include poor concentration and forgetfulness (especially in those with melancholic or psychotic features), withdrawal from social situations and activities, reduced sex drive, and thoughts of death or suicide. Insomnia affects at least 80% of depressed people but hypersomnia can also occur. Some antidepressants (e.g. SSRIs) may also cause insomnia. Major depression significantly affects a person's family and personal relationships, work or school life, sleeping and eating habits, and general health[4]. Its impact on functioning and well-being has been compared to that of chronic medical conditions such as diabetes[5].

A depressed person may report multiple physical symptoms such as fatigue, headaches, or gastrointestinal problems. Physical complaints are the most common presenting problems in developing countries. Appetite often decreases, with resulting weight loss, although increased appetite and weight gain occasionally occur. There may be agitation or lethargy. Older depressed people may have cognitive symptoms, such as forgetfulness, and a more noticeable slowing of movements[5]. Depression often coexists with physical disorders

common among the elderly, such as stroke, other cardiovascular diseases, Parkinson's disease, and chronic obstructive pulmonary disease. Depressed children often have an irritable mood[6] and show varying symptoms depending on age and situation. Most children show a decline in academic performance.

Etiology

The biological, psychological, and social factors play a role in causing depression. It occurs when a preexisting vulnerability, or diathesis, is activated by stressful life events.

Depression may be directly caused by damage to the cerebellum as is seen in cerebellar – cognitive affective syndrome[5]. These interactive models have gained empirical support. The variation among the serotonin transporter (5-HTT) gene affects the chances that people who have dealt with very stressful life events will go on to experience depression. To be specific, depression may follow such events, but seems more likely to appear in people with one or two short alleles of the 5-HTT gene. The heritability of depression is around 40% for women and 30% for men. The evolutionary psychologists have proposed that the genetic basis for depression lies deep in the history of naturally selected adaptations. A substance-induced mood disorder resembling major depression has been causally linked to long-term drug use or drug abuse, or to withdrawal from certain sedative and hypnotic drugs.

Most antidepressant medications increase the levels of one or more of the monoamines — the neurotransmitters serotonin, norepinephrine and dopamine in the synaptic cleft between neurons in the brain. Some medications affect the monoamine receptors directly.

Serotonin is hypothesized to regulate other neurotransmitter systems; decreased serotonin activity may allow these systems to act in unusual and erratic ways According to this "permissive hypothesis", depression arises when low serotonin levels promote low levels of norepinephrine, another monoamine neurotransmitter. Some anti-depressants enhance the levels of norepinephrine directly, whereas others raise the levels of dopamine, a third monoamine neurotransmitter. These observations gave rise to the monoamine hypothesis of depression. In its contemporary formulation, the monoamine hypothesis postulates that a deficiency of certain neurotransmitters is responsible for the corresponding features of depression: Norepinephrine may be related to alertness and energy as well as anxiety, attention, and interest in life; lack of serotonin to anxiety, obsessions, and compulsions; and dopamine to attention, motivation, pleasure, and reward, as well as interest in life The proponents of this theory recommend the choice of an antidepressant with mechanism of action that impacts the most prominent symptoms. Anxious and irritable patients should be treated with SSRIs or norepinephrine reuptake inhibitors, and those experiencing a loss of energy and enjoyment of life with norepinephrine- and dopamine-enhancing drugs.

Inflammation and Depression

The relationship between the immune system and the brain was not protested until 1985, when a group of scientists discovered evidence that immune responses are suppressed in depressed patients[6] However, a number of immune components, such as inflammatory biomarkers, are in fact heightened in subgroup of depressed patients[7]. Inflammatory processes can be triggered by negative cognitions or their consequences, such as stress, violence, or deprivation. Thus, negative cognitions can cause inflammation that can, in turn, lead to depression[8].

Proposed Theories

It is known that MDD involves both immune suppression which is indicated by a reduction in natural killer cell cytotoxicity and lymphocyte proliferation as well as immune activation which is indicated by an increase in inflammatory mediators[9]. The current hypothesis is that excess inflammation is thought to play a role in the pathophysiology of MDD, however, the mechanism by which this occurs is unclear. In addition to this, it is not known whether inflammation causes depression or vice verse. A role of cytokines in depression is supported by the phenomenological similarities between the symptoms of cytokine-induced sickness behaviour and depression such as behavioral inhibition, anorexia, weight loss, anhedonia, psychosomatic symptoms, anxiety and neurocognitive symptoms[10]. The initial acute immune response in sickness behaviour is thought to be beneficial to the individual

since it enables them to cope better with illness or infection However, in order to establish this, the inflammatory mechanisms involved must first be explored. Inflammation occurs as a result of an imbalance between pro-inflammatory mediators such as cytokines, chemokines and acute phase proteins and anti-inflammatory mediators, the former being in excess.

Depression and Immune System

The innate immune system is thought to play an integral role in depression and it determines the type of adaptive immune response involving T helper (Th) 1 or Th2 lymphocytes. In Th1 response, macrophages release pro-inflammatory cytokines such as interferon gamma (INF-ã), tumour necrosis factor alpha (TNF-á), interleukin (IL)-1 and 2. This occurs in response to the recognition of pathogens, tissue damage or destruction via cell surface Toll-like receptors (TLRs)[11]. There is currently no conclusive evidence for a role of TLRs in MDD in humans, even though they are thought to be the gatekeepers of innate immune activation. Although there is no evidence for a role of infectious agents (such as the Borna virus) in MDD, lipopoly-saccharides (LPS) and endotoxin have been shown to induce symptoms of MDD through peripheral immune activation[12]. TLRs activate nuclear factor–êB (NF-êB) which induces an innate inflammatory response involving the release of pro-inflammatory cytokines such as IL-1, IL-2, IL-6 and TNF-á[11]. Once activated, microglia may remain activated for several months and release pro-inflammatory cytokines which could explain why patients who suffer from chronic physical illnesses often develop MDD. These cytokines provide a communication between the peripheral and central immune system by at least four different mechanisms in MDD Cytokines access the brain via leaky regions in BBB; active transport across the BBB; a neural pathway involving afferent nerve fibers such as the sensory vagus which relay information through the nucleus tractus solitarius; a humoral pathway, involving volume diffusion from circumventricular organs across the BBB[12]. The BBB may even be impaired in MDD, allowing the extravasation of leukocytes and upregulation of intracellular adhesion molecules

Neurotransmitter and Inflammation

Once inside the brain, cytokines influence the pathways involved in the pathophysiology of depression such as metabolic alterations of neurotransmitters including serotonin (5-HT), dopamine (DA), noradrenaline (NA). Tryptophan is an amino acid precursor of 5-HT and melatonin In the Th1 response microglia and macrophages secrete IL-1, IFN-ã and TNF-á which induce indoleamine 2,3- dioxygenase (IDO) expression favoring conversion of tryptophan into quinolinic acid, a N-Methyl-D-aspartic acid (NMDA) agonist, and kynurenic acid, NMDA antagonist. This reduces tryptophan availability for 5- HT and melatonin synthesis. Confirming the presence of activated microglia in depression is the synthesis of quinoloic acid, since they are the only cells in the central nervous system (CNS) which express all the enzymes required for its synthesis, whereas multiple cell types express IDO[13]. As a result, there is reduced availability of 5-HT which is implicated in the pathogenesis of MDD. Quinoloic acid is a NMDA agonist and therefore its accumulation may contribute to excitotoxicity as well as calcium-mediated cell death in MDD[14.] Although paroxetine has shown efficacy to treat INF-á-induced depresssion, kynurenine levels remain unaltered suggesting that increases in synaptic 5-HT by paroxetine compensate for the IDO favoring the kynurenic acid pathway DA is another monoamine which is implicated in MDD, particularly with the symptoms of sickness behaviour and anhedonia. There is a reduction in DA in the basal ganglia in MDD and this may contribute to the blunted reaction to positive reinforces and abnormal response to negative feedback observed in depressed patients. As with 5-HT described previously, proinflammatory cytokines influence dopamine synthesis and reuptake. Kynurenine is converted into kynurenic acid, an NMDA antagonist, which antagonizes á7 nicotinic acetylcholine (ACh) receptors and can reduce striatal dopamine release. Dopamine reuptake is increased through phosphorylation of the dopamine transporter (DAT) by mitogen-activated protein kinase kinase (MAPKK)[7]. NA is also thought to be involved in

the pathogenesis of depression however the role of inflammation in the reduction of NA has not yet been established in humans with MDD.

Inflammation and Endocrine system

Inflammatory cytokines have been found to activate the HPA axis and increase in corticotropin-releasing hormone (CRH) and glucocorticoid activity in depressed patients. The HPA axis is involved in regulation of cortisol levels in response to stress. In response to psychological stress in cortical brain regions and prostaglandin E2 (PGE2) release from activated microglia, the hypothalamus releases CRH and adrenocorticotropic hormone (ACTH) CRH stimulates the pituitary gland to release corticotrophin which induces the release of cortisol from the zona fasciculata of adrenal gland into the plasma[15]. Overactivity of the HPA axis in MDD causes hypercortisolemia which may either result in decreased expression of the glucocorticoid receptor (GR) or decrease functionality of the GR, both ultimately resulting in glucocorticoid resistance. Through these mechanisms, there is evidence for a role of proinflammatory cytokines such as TNF-á in glucocorticoid resistance. The peripheral immune cells no longer respond to cortisol due to glucocorticoid resistance and the negative regulation on the immune response is lost which may increase inflammation. Glucocorticoid mechanisms have been recently linked to neurogenesis and it is thought that GR activation is necessary for its modulation by antidepressants[16].

Inflammation and Neurotoxicity

A number of studies have implicated neurotoxicity and a loss of neuroplasticity in MDD and evidence suggests inflammation may contribute this. Neuroplasticity involves the Th2 response which involves antibody production and the release of anti-inflammatory cytokines such as IL-4, IL-5, and IL-10 which inhibit the Th1 response. These activate neuroprotective microglia which result in increased expression of brain-derived neurotrophic factor (BDNF), a growth factor involved in neurogenesis. An imbalance between the Th1 and Th2 response has been found in some patients with MDD and this could impair neuroplasticity. Pro-inflammatory mediators activate microglia in the brain and this leads to the release neurotoxic substances in such as reactive oxygen species (ROS), positive feedback to the peripheral Th1 immune arm to increase the release of IL-2, IFN-ã and IL-1. Neurotoxic substances are released in excess of neurotrophic factors and therefore, a loss of plasticity and an increase in excitotoxicity is observed in patients with MDD[17]. Activated microglia cause oxidative stress and nitric oxide production which increases inflammation. IL-1â activates nitric oxide production and this result in glutamate release and consequent excessive NMDA agonism. Successful treatment with antidepressants inhibit IFN-ã-induced microglial production of IL-6 and nitric oxide and this reduces depressive symptoms in MDD .Over the past few years, it has also been demonstrated that excessive NMDA agonism by quinoloic acid may be implicated in MDD through excitotoxicity and calcium-mediated neuronal death[18]. Exposure to IL-1â and TNF-á enhances excitotoxicity through NMDA and AMPA receptor agonism in hippocampal neurons. IL-1â is thought to act through R1 receptors via phosphorylation of the NR2B subunit. This results in upregulation of the NMDA receptor. TNF-á induces the production of AMPA receptors which lack GluR2 subunit. This is thought to predispose the neuron to further excitotoxicity and cell death. Treatment with a non-competitive NMDA antagonist, ketamine, has been shown to have depressogenic effects with immediate onset as well as inhibit TNF-á synthesis in microglia after treatment with endotoxin[19]. Excessive NMDA receptor agonism also leads to glutamate release and further microglial activation and release of proinflammatory cytokines. IL-1â also activates nitric oxide production which also results in glutamate release although glutamate is known to cause neurotoxicity in excess; it has not been unequivocally demonstrated in MDD. Glutamate increases endotoxin activation of microglia and direct activation of microglia, and its effects can be inhibited by memantine Excess synaptic glutamate is usually taken up by astrocytes via excitatory amino acid transporters (EAATs) to prevent excitotoxicity in neurons, however, IL-1â released from activated microglia impairs astroglial removal of synaptic glutamate which is corrected by riluzole, an antidepressant There is a correlation between the activation of microglia and loss of EAAT1 or EAAT2 in astrocytes in multiple sclerosis patients with focal

loss and cortical lesions, resulting in excitotoxicity.

MDD and Levels of Pro-inflammatory Mediators

In a third of patients with MDD, the most replicated alterations to serum and cerebrospinal fluid (CSF) concentrations of inflammatory markers include an elevation of IL-6, TNF-á, and C reactive protein compared to non-depressed patients This occurs even in the absence of physical illness or infection and therefore the increase in inflammatory markers can only be accounted for by depression. In a meta-analysis of 22 cross-sectional studies, the standard mean difference (SMD) of sIL-2R, TNF-á, and IL-6 (all studies) in patients with MDD were significantly higher than those of healthy controls with a p value of less than 0.05.

DSM was used to elicit depressive symptoms of MDD in included studies and heterogeneity was evaluated by the Cochran Q test in both serum and plasma studies and this casts doubt on its role in NMDA agonism. There was no significant increase in IL-1â (-0.525) oxide release in MDD. Different methods of measuring the proinflammatory markers were used in the included studies and some subjects had a dual diagnosis of depression and substance misuse which may have affected the results. This study indicated an association between infla-mmatory markers and depression and no causal relationship has been found since the severity of depression was not included in the analysis [20].

Clinical Studies

A 22 study meta-analysis on antidepressant treatment supports a role of inflammation in MDD by indicating a reduction in IL-1 and IL-6 as well as depressive symptoms in response to antidepressant treatment. This response was specific for serotonin-selective reuptake inhibitors (SSRIs) which is the most common first-line treatment for MDD suggesting that antidepressants block the effect of inflammatory cytokines and relieve depressive symptoms [21]. However, other studies have failed to find a correlation between infla-mmation and the severity of depressive symptoms In addition to this, some studies have found incongruent and opposite correlations for different pro-inflammatory markers as well as failure to identify an association between inflammation and the pathogenesis of depression[22]. In addition to this, other studies show that this association has been attenuated when factors such as body mass index, personality or gender have been included in the analyses[22].

A longitudinal study for MDD was performed on a targeted population consisting of children. Interviews were carried out to elicit depressive symptoms rather than self-report and CRP levels in blood spots were measured to identify the degree of inflammation in each individual. This study demonstrated depression was associated with later levels of CRP from measurements in blood spots and therefore depression promotes inflammation. In addition to this, an elevation in CRP from a young age may increase the risk of late life recurrent depressive episodes, or even increase the risk of treatment-resistant depression. The level of inflammation promoted by depression in this case may also increase the risk of ailments such as cardiovascular disease (CVD), type-2 diabetes mellitus (T2DM) associated with increased age[23]. Therefore, inflammation is not primary in MDD and inflammatory processes may take place after there is reduced immunocompetence in depressed patients. A post-mortem study indicated that microgliosis was present in the brains of both patients with schizophrenia and depression who had committed suicide, but not in patients from the same diagnostic groups who died from other causes [24].

Only few other studies have investigated inflammatory changes associated with suicidal behavior. An initial study reported elevated concentrations of soluble IL-2 receptor (sIL-2R) in the blood of suicide attempters[25]. The current study reported high levels of IL-6 in the cerebrospinal fluid (CSF) of suicidal patients The study found increased IL-6 and TNF-á as well as decreased IL-2 concentrations in the plasma of suicide attempters, compared to non-suicidal depressed patients and healthy controls. Although there was a trend towards higher MADRS scores in depressive, non-suicidal patients than in the suicide attempters, a clinical relevance of this is unlikely since both scores represent a depression of moderate severity. The study thus suggests that inflammatory changes may not be present in all depressed patients, but may be confined to suicidal states. Further studies are warranted to analyze additional subgroups of depressive patients with respect to inflammation.

Depression and Other Clinical Diseases

MDD occurs at a 5-10 times higher rate in patients with a physical illness, suggesting that immune dysregulation may be a central feature common to depression and to its co-morbidity. These diseases can be classified as either infla-mmatory involving autoimmunity or immune activation, or non-inflammatory[25]. Autoimmune diseases which occur with MDD include rheumatoid arthritis (RA) whereby depression worsens prognosis and disability. In addition to this, MDD is associated with diseases with 'low' grade inflammation such as cancers, coronary artery disease, stroke and epilepsy. Even though these diseases are not traditionally associated with inflammation, it occurs secondary to initial ischemia. In patients with coronary heart disease, the proportions of participants with levels of IL-6, CRP and fibrinogen were 22-24% in those without depressive symptoms at any interview, 23-28% in those with depressive symptoms at one interview and 30-35% in patients with depressive symptoms at two or more interviews in a 5 year follow-up[26]. Despite a significant association of pro-inflammatory mediators and depressive symptoms in coronary heart disease, this study showed that there was no effect of inflammation on depressive symptoms after adjustment for age, race, gender, education, history of diabetes or myocardial infarction, congestive heart failure, use of aspirin and the baseline levels of inflammatory markers. In addition to this, a Patient Health Questionnaire was used to elicit nine depressive symptoms and this is not a reliable method for diagnosis of MDD. Therefore, this suggests that inflammation in medical illnesses does not cause MDD even though co-morbidity of MDD is associated with excess peripheral inflammation in diseases such as multiple sclerosis, psoriasis, Crohn's disease, human immunodeficiency disease.

Immunotherapy and Depression

Immunotherapy involves the systemic injection of cytokines such as INF-á and IL-2 in the treatment of infectious diseases such as hepatitis C and cancer. Immunotherapy is a potent inducer of pro-inflammatory cytokines, especially IL-6, and INF-á treatment is strongly associated with the onset of depressive symptoms. It induces neurovegetative symptoms associated with sickness behaviour within 1-8 weeks of therapy suggesting inflammation has an initial beneficial role [26]. This can be an early indication of depression which usually occurs 1-3 months after treatment whereby changes in mood and cognition occur after 8 weeks. INF-á induced depression is associated with changes in 5-HT metabolism via IL-6 which induces IDO and increases the plasma levels of kynurenine, alterations in CRH function which occurs as a result of a heightened response of ACTH and cortisol . In a study of 30 patients who received 3 months of 3MU INF-á treatment for chronic active Rating Scale (MADRS) scores for insomnia, sadness, irritability, loss of appetite and asthenia compared to baseline scores . This is simply a correlation between depressive symptoms and INF-á treatment and no casual relationship has been established. Positron emission tomography suggests INF-á disrupts metabolic activity in the basal ganglia affecting both psychomotor speed and anhedonia in patients with MDD INF-á may also alter processing within the dorsal anterior cingulate cortex which has increased activity to negative events in patients with MDD compared to controls, a possible mechanism of inducing depressive symptoms.

Conclusion

The role of inflammation in MDD has been of growing interest over the past two decades; however, there are inconsistencies in evidence. Cytokines and other pro-inflammatory mediators, including IL-6, IL- 1â, and TNF-á, interact with almost every pathophysiologic area involved in mood regulation such as neurotransmitter metabolism, neuroendocrine function, anterior cingulate cortex activity, basal ganglia, and synaptic plasticity. However, MDD is not an inflammatory disorder since it is neither necessary, nor sufficient, to be the sole cause of MDD and it only occurs in a subgroup of patients. These individuals may be predisposed to develop MDD coupled with inflammation as suggested by studies on genetic variations in serotonin transporter gene or those genes affecting T-cell function[27]. In addition to this, there is also a polymorphism in the promoter region of IDO 1 (rs9657182) which predicts the development of moderate or severe depression in Caucasian individuals undergoing INF-á therapy for Hepatitis C [28]. It has also been proposed that childhood

adversity may predispose individuals to develop depression with inflammation. Therefore, inflammation may contribute to some cases of depression acting simply as a trigger to a cascade of events which ultimately leads to depression or as an underlying physiological response as a result of a heightened sensitivity of the immune system to stress[29]. Inflammation may even occur secondary to the reduced immunocompetence observed in patients with MDD, rendering them more vulnerable to invasion and proliferation of pathogens. Difficulties with studying this topic are heterogeneity of depressed patients as they may have differences in the immune profiles as well as in symptom profiles or demographic features. It is difficult to establish which factor occurs first in middle-aged or elderly subjects since the chances of being free of an episode of depression or inflammation significantly decrease with age and a large population is required to have sufficient power. The relationship between depression and inflammatory-driven illnesses is bidirectional and is driven by several biological processes, including immune dysregulation. In this context, inflammation may precipitate a depressive episode in a vulnerable individual with a medical illness. However, inflammation may also act as a perpetuating factor and prevent recovery from depression in these predisposed individuals [30].

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Psychophysiotherapy

Enhancing Mental Wellbeing and Health of Postmenopausal Women

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Menopause marks a major life transition for women, an end to the childbearing years and the cessation of the menstrual cycle. Many premenopausal women have concerns that they will experience mental instability, sudden signs of aging and diminution of sexuality at this time[1]. Menopause is an adaptation process during which women go through a new biological state. This process is accompanied by many biological and psychosocial changes[2]. These symptoms are a common reason for seeking medical attention during this phase of life. More recently, women have been seeking alternative therapies for menopausal symptoms, including increasing or maintaining a high level of physical activity[3]. Exercises have shown to improve mental health by reducing anxiety, depression and negative mood and by improving self esteem and cognitive function. Exercise has also been found to alleviate symptoms such as low self esteem and social withdrawl[4].

Effects of Menopause

Menopause brings in a whole lot of changes in the body of women and in most of them leads to troublesome symptoms namely hot flashes, sleep disturbances, weight gain, fatigue, aches and pains, altered cognitive functions, genitourinary problems like vaginal dryness, irritation, incontinence, urinary tract infections and weakness of connective tissue supporting the pelvic viscera, skin , a decrease in libido, sexual dysfunction, an increase in the risk of cardiovascular diseases, bone loss, and somatic and vasomotor symptoms, Depressed mood and sleep disorders[2,5]. Apart from these physical symptoms, there are a series of various psychological as well as emotional symptoms as well.

Emotional and Psychological Effects of Menopause

Menopause is linked to certain psychological and behavioral changes. Thinking, speech, short- and long-term memory, spatial and time changes are all relevant changes that many women may experience. Common emotional and psychological symptoms include[6,7]

1. *Mood swings:* Abrupt hormone changes alter the equilibrium in neurotransmitter systems in the brain and the woman experiences mood swings and behavioral changes.
2. *Fatigue:* Fatigue, mostly mental, is experienced by many menopausal women. It is typically the result of mental stress due to unwanted physical changes a woman undergoes during this phase. Mental fatigue manifests in the form of decreased attention span, problems with concentration, and physical exhaustion.
3. *Low libido:* Reduced sexual desire, poor arousal and orgasm and impaired sexual satisfaction are the most common sexual complaints in the context of menopausal women.
4. Irritability, forgetfulness, and memory loss
5. Lack of motivation, excessive worry, anxiety, diminished energy, problems in

concentration and feeling of low self-esteem.

6. *Depression and anxiety:* Depression may hit during the peri-menopause phase up to menopause. Although most theories link it directly to the fluctuating hormones, mood swings are also thought to lead to depression and anxiety. Many believe that the vasomotor symptoms such as hot flushes and night sweat 'provoke' physical changes such as insomnia that further affects mood stability. This is known as the domino theory. These mood swings cause depression over time.

All these short and medium-term effects influence the health related quality of life of these women adversely by the way of Depression and Anxiety.

Various Reasons for psychological changes [6]

1. Many women notice a change in their emotions and feelings during midlife for various reasons (i.e., changes in self-concept, body image, stress, relationship issues, cultural reasons, etc.).
2. Different stresses around the time of menopause may be attributed to psychological changes. Frequently, women in their midlife face many challenging stresses including divorce, having grown children leave the house, concerns about aging and widowing, and caring for older parents.
3. Changes in ovarian hormone levels (declining estrogen and progesterone levels) may also explain the emotional inconsistencies women face at this time. Some experts believe the loss in estrogen

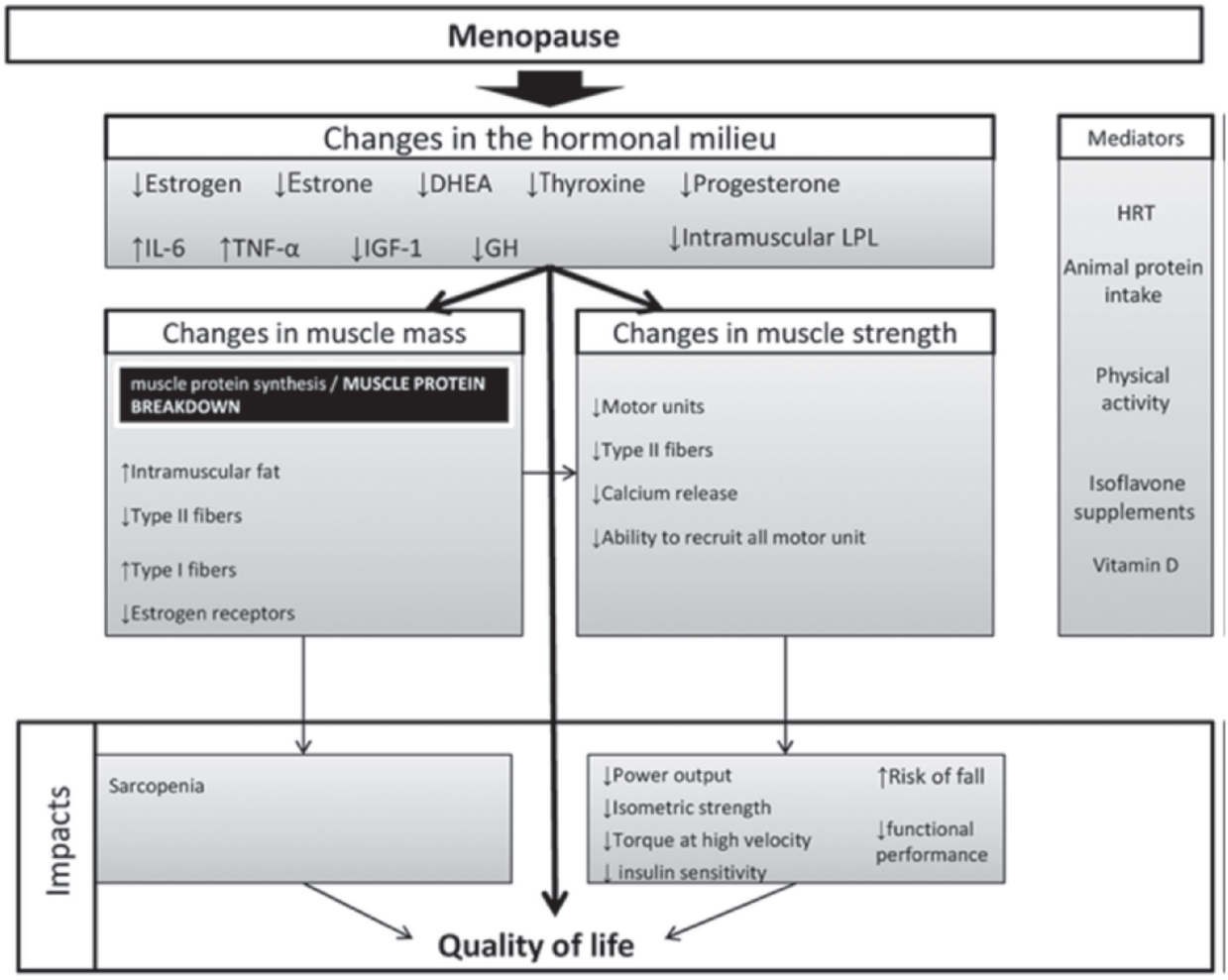

Table: How Menopause affects the Quality of Life[12]

in relation to glucose levels may affect cognition. Female reproductive hormones do exert some effect on the brain neurotransmitter systems, particularly the serotonin and gamma amino butyric acid systems. Though these complex interactions presently have limited clinical application, it is important to realize that hormones are psychoactive, so it is possible that fluctuating hormone levels may cause women to experience subtle mood and cognitive changes[1].

4. Sleep deprivation can also cause emotional and behavioral changes. Sleep changes can occur for various reasons including disruption caused by hot flashes and hormonal changes.

Exercise

Nelson et al[3] found that high levels of physical activity were related to lower levels of perceived stress, and levels of anxiety, stress, and depression were significantly lower among physically active, postmenopausal women. These results suggest that maintaining or increasing physical activity during the menopausal transitional period and postmenopause may assist in reducing a variety of psychological symptoms, including anxiety, stress, and depression[3]. Similarly, Mutrie conducted a meta-analysis and concluded that physical activity is associated with decreased risk of developing clinical depression.[8]

The exercise program for postmenopausal women should include the endurance exercise (aerobic), strength exercise and balance exercise; it should aim for two hours and 30 minutes of moderate aerobic activity each week. Every woman should be aware of her target heart rate range and should track the intensity of exercise employing the talk test. Other deep breathing, yoga and stretching exercises can help to manage the stress of life and menopause-related symptoms. Exercises for women with osteoporosis should not include high impact aerobics or activities in which a fall is likely[5].

Benefits of Exercise

1. Exercise increases the cardio-respiratory function.
2. Exercise can help create a calorie deficit and minimize midlife weight gain.
3. It increases the bone mass. Strength training and impact activities (like walking or running) can help to offset the decline of bone mineral density and prevent osteoporosis[5].
4. It improves strength, endurance, quality of life parameters[9]
5. It is proven to help reduce stress and improve the mood, HRQOL and psychosocial factors (depression, stress and social support)[10]

Mechanics of exercise

It is hypothesized that endorphin concentrations in the hypothalamus decrease as estrogen production declines, enhancing the release of norepinephrine and serotonin. Exercise may have a similar effect to HRT (Hormone Replacement Therapy) in the amelioration of vasomotor symptoms by increasing the presence of hypothalamic and peripheral endorphin production. Researchers have shown that physically active individuals have higher basal levels of endorphins than those who are inactive.[11]

Exercise can also serve as a source of distraction or a 'time out' strategy from daily worries and depressing thoughts. Exercise influences mental health outcomes such as depression through enhancement of self-esteem. People who are depressed often feel they lack control and feelings of mastery in their lives. Exercise may provide women with an opportunity to experience a sense of achievement, efficacy and self-determination during a time of change. Exercise may be a form of behavioral activation, which is typically an important component of some effective psychotherapy interventions for depression.[11,12]

Exercise - A Stepwise Approach

Lee et al concluded that women who were depressed had more menopausal symptoms than women who were not depressed, and women who exercised regularly were less depressed and less symptomatic than women who did not exercise[13].

An exercise program for menopausal women that includes both aerobic and resistance training may prevent or relieve problems such as cardiovascular disease, obesity, muscle weakness, osteoporosis, and depression.[14]

Steps

Step 1: Stretch, walk on a treadmill for five minutes or go for a brisk walk to get ready for exercise. As owing to age, the body becomes less flexible, it is important to warm up the body before a work out.

Step 2: Engage in aerobic activity that elevates the heart rate and burns fat. Whether it is a dance class, aerobics class, going for a run or a bike ride, signing up for kickboxing or taking time on an elliptical machine, each helps to benefit the large muscle groups and helps the cardiovascular function.

Step 3: Lift weights, use resistance bands or try body weight strength training in order to keep the bones strong. Menopause is a common time for women to experience a loss of bone density or osteoporosis. They have to aid in keeping their bones strong by keeping the muscles strong. Strength training also can help to revive up the decreasing metabolism and help in burning the fat, even while resting, to avoid the dreaded menopausal weight gain.

Step 4: Foster better flexibility by trying workouts that cause the stretching of muscles, such as yoga and Pilates. This can promote better muscle function. The woman must take time for yoga and meditating each night to reduce some of the anxiety that also is a common symptom of menopause.

Step 5: Cool down at the end of a workout by walking for a few minutes and stretching to relieve any pain as a side effect from a particularly grueling workout. This gives the body a chance to relax and promotes regular breathing and a slowing of the heart rate as one finishes exercising for a healthy end to this menopausal friendly workout.

Contraindications for exercise

- Recent electrocardiogram changes or recent myocardial infarction
- Uncontrolled arrhythmia
- Unstable angina
- Third degree heart block
- Acute progressive heart failure
- Elevated blood pressure
- Cardiomyopathy
- Valvular heart disease
- Complex ventricular ectopy

Conclusion

Many women experience detriments in mental health during the menopausal transition. Vasomotor symptoms are less common among physically active postmenopausal women than among sedentary women.

Even a moderate exercise schedule can not only keep the weight in check, but it also lowers the risk of stress, anxiety, and depression. Exercise works by improving muscle mass, strength, balance, and coordination. Regular exercises can improve cognitive function, enhance mood and daytime alertness and promote nocturnal sleepiness. Thus, it can enhance the quality of life.

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Psychophysiotherapy

Rehabilitation for Alcohol abuse

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Introduction

Alcohol abuse is a leading cause of morbidity and mortality world-wide.[1] Alcohol use disorders are a major public health concern[2]. Both chronic and acute alcohol consumption have the potential to impair health and well-being. Alcohol affects virtually every organ and tissue in the body, with multi-factorial actions on cellular and molecular functions[3].Alcohol consumption has a deleterious effect on various systems within the body. It affects many aspects of metabolism, neural function, cardiovascular physiology, psychology, behavior, thermoregulation and skeletal muscles.[4]

Skeletal muscles and bones: Alcohol misuse is associated with skeletal myopathy, reduced muscle mass, strength and function, muscle pain and a loss of proprioception.[4,5] Chronic myopathy is characterized by progressive weakness in proximal muscles. Affected individuals develop fatigue with difficulty in climbing stairs, walking, and rising from squatted and seated positions, frequent falls, difficulties in gait, proximal weakness and muscle cramps.[1,6] Chronic ethanol abuse alters whole body protein metabolism as it compromise the homeostatic utilization of amino acids which would otherwise incorporate into muscle protein.[6] The rate of fracture among alcoholics was found to be four times higher than non-abusers. Incidences of osteopenia or osteoporosis were also found more in alcoholics.[7] Previously studies have observed that alcohol exposure inhibits formation of the external callus tissue and the endochondral ossification process thus it increases risk of developing nonunion and delayed union[7].The lack of normal fracture callus formation in both intoxicated patients and rodents suggests that alcohol inhibits bone fracture repair at an early stage in the healing process.[3,7]

Nervous system - Alcohol is a well-known depressant and thus acts to reduce central nervous system excitability and cerebral activity.[4] Alcohol abuse may leads to blackouts, withdrawal symptoms, brain damage, stroke, and nerve and muscle damage.[8] Excessive use of alcohol results in physiological and cognitive changes. Polyneuropathy is the most common neurologic complication in alcoholism.[9,10] In the early stages of alcoholic neuropathy, patients complain of severe pain in the extremities, which may be described as burning or 'like tearing flesh off the bones' and is characterized by spontaneous burning pain, hyperalgesia and allodynia.[11] Patients report paresthesias, pain, and weakness, especially in the feet[10]. Clinical features of alcoholic peripheral neuropathy include abnormalities in sensory, motor, autonomic and gait functions. Alcohol related neuropathy is associated with several risk factors, such as malnutrition and thiamine deficiency[11]. Brain function may be chronically impaired with alcohol abuse and can lead to memory impairment, loss of higher brain functions such as judgment, abstract thinking, reaction time, visual search, recognition, memory and accuracy of fine motor skills and language and disturbances of balance and coordination.[4,9] A cerebellar syndrome occurs in some patients with chronic alcohol use. It is characterized by gait ataxia with lesser degrees of limb ataxia affecting the legs more than the arms[10].

Exercise and work performance: Alcohol

abuse may leads to impaired work performance and decision making, decreased work productivity, increased risk of accidents.[8]

Alcohol abuse is associated with detrimental effect on other systems of body like arrhythmias, cardiomyopathy and hypertension in cardiovascular system and esophagitis, gastritis, impaired healing of peptic ulcers, diarrhea and malabsorption, acute and chronic pancreatic problems in gastro intestinal system.[8]

Fetal alcohol syndrome occurs in one third of infants born to women with chronicalcoholism. Fetal alcohol syndrome may result in mental retardation, congenital facial malformations, gene-ralized hypotonicity, cerebral palsy, hemiparesis or hemiplegia, variety of organ, musculoskeletal, neurologic, developmental differences, Neural tube defects including lumbosacral myelomeningocele and anencephaly. Alcohol related birth defects include orthopedic abnormalities like congenital hip dislocation, radio-ulnar synostosis, elbow flexion contractures, cervical spine fusion, club foot, and scoliosis.[12]

Cross-sectional studies have often revealed a strikingly high prevalence of depressive disorders in persons with alcohol use disorders.[13]

Treatment for Alcohol abuse

Treatment for patients with alcohol abuse may include education on the physical, emotional, and mental aspects of addiction and recovery, stress management skills, improved coping skills and relaxation training along with medications.[9,14] Lifestyle-enhancing factors such as exercise and fitness may play an important role in the prevention and treatment of addictive disorders.[2]

Medical management

Medication management for alcohol use disorder includes benzodiazepines to decrease tremors and to maintain blood pressure and heart rate, in addition to medications for other symptoms (e.g., diarrhea or muscle aches).[5,14] Benfotiamine, Methylcobalamine, Vitamin E, Alpha-lipoic acid are used for treatment of peripheral neuropathy. Tricyclic antidepressants (TCAs) are often the first line drugs to alleviate neuropathic pain symptoms.[11] A narcotic antagonist such as naltrexone that diminishes the effects of alcohol, can be used to help some individuals remain abstinent.[9,14]

Psychological management

Alcohol misuse often co-exists with common mental disorders so it is vital to maintain good working protocols with the mental health team.[8] Specific psychosocial treatment is required to treat substance abuse. Cognitive behavior therapy can also be used to treat depressed alcoholics.[14,15] Motivational enhancement techniques can be used for patients with substance abuse.[8] Therefore, prevention of depressive disorders in alcohol-dependent persons has the potential to enhance mental health care.[13]

Physical therapy treatment

Physical therapy along with medical and psychological management plays an important role in treatment of various alcohol abuse induced polyneuropathies, myopathies and alcohol related birth defects like cerebral palsy and various contractures.Physiotherapy helps individuals with myopathy or neuropathy to manage their symptoms and improve their ability with everyday tasks.

- A structured exercise program to improve balance, coordination, flexibility and endurance is required to treat myopathies and neuropathies.
- Physiotherapy treatment program may involve a structured exercise program to increase muscle strength and endurance, Active and passive movements to reduce muscle cramps and prevent muscle wasting, strengthening exercises, exercises to improve stability, gait and coordination. Stretching exercises to lengthen tight muscles and ease muscle spasms.
- Comprehensive physical therapy for patients with alcoholic neuropathy includes gait and balance training, possibly with an assistive device for safety, Range of motion (ROM) exercises and stretching, particularly for the gastrocnemius-soleus muscle, to prevent contracture and maintain normal gait mechanics, Strength training of weakened muscles.
- Physical therapists can play an important rolein the management of balance dysfunction. Examples of physical therapy

interventions include lower extremity strengthening exercises, guided practice of integrating internal and external sensory input, education on sensation loss andfall risk, instruction on home modifications and assistive devices to minimize balance dysfunction.[16] When balance is improved, risks of tips and falls are decreased. Lower-leg strengthening and balance exercises have been proven to improve tandem and one leg stance time and functional reach in older adults.[17]

- Exercise is proven to be cost-effective, flexible and accessible; many forms of exercises (e.g., running, walking, and swimming) may be conducted independently, either at home or outdoors. Also exercise has minimal side effects compared to pharmacological treatment.[2] Exercise and increased physical activity may help reduce cravings, negative changes in mood, and withdrawal symptoms as an aid to cessation. Exercise may reduce risk for relapse by reducing depressive symptoms as they help to alleviate sleep disturbances and improve cognitive functioning.[14] Healthy lifestyle changes may contribute to long-term maintenance of recovery in patients with alcohol abuse.[2]
- Advise about equipment for mobility if required i.e. walking aids, orthosis for dropped foot or feet, wheelchairs.
- Hydrotherapy treatment relaxes, increase circulation and strengthens muscle. It also reduces pain and enables a person with a myopathy to maximize their mobility within the water.
- Electrotherapy modalities can also be used to treat painful neuropathies. It was observed that high-frequency electrical stimulationand high-frequency TENS can be used as effective pain-relieving modalities for painful neuropathies.[18] Functional Electrical Stimulation can also be used to treat muscle weakness in the legs or dropped feet. Electrical stimulation can also be used to treat neuropraxias caused by compression of any nerve.
- Physiotherapy is an important part of managing alcohol related birth disorders like cerebral palsy. Depending on the specific needs of the child, a variety of techniques, approaches, and theories may be incorporated for treatment. These approaches may include neurodevelopmental treatment, behavioral shaping, developmental training and sensory integration.[19]

Occupational therapy treatment

Occupational therapy also can be an important component of the rehabilitation process in individuals with alcoholic neuropathy it helps individual in developing communication skills, identifying and matching personal skills and work habits to the workplace, Training in performance of activities of daily living (ADL), with adaptive equipment if necessary, and learning how non-alcohol-related participation in leisure activities contributes to overall health and well-being.[9,14]

Others

Nutritional advice is also required for treatment of neuropathies. Various expressive therapies like art, music, or dance therapy, and relaxation techniques may be helpful to decrease stress levels that can increase risk of relapse.[9,14]

Conclusion

Treatment of the alcoholic, to be successful, must be multidisciplinary. It includes medical management, counselling, psychotherapy, nutri-tional guidance, physical therapy, occupational therapy and other recreational activities. Education and participation of the families concurrently in the rehabilitation program can enhance the results.

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Commentary

The effect of Spiritual Music on Health in Different Religions

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Introduction

History of using of sound in treating illnesses goes back to the distant past.[1] The central thesis of this article is very simple: What is the effect of Spiritual music on health in different religions. In fact, in some ways, mental health professionals have moved away from effective lifestyle interventions. Psychiatrists in particular are being pressured to offer less psychotherapy and prescribe more drugs[2] but it is not a useful therapy especially when the life style of patients is not healthy. As a result, patients suffer from over attention to physical therapy[3]. In this position, some psychologists think incorporating spirituality into therapeutic environments could bring new treatment options into the limelight. Today the Power of Spirituality in therapy could not be neglected. In the other hand, Music and music therapy are used in a variety of clinical settings and this kind of therapy is so useful, too. Music is not just another way to entertain yourself. In fact music can have surprising effects on our health; it depends on the kind of music. Playing and listening to music benefits both mental and physical health. Traditional psychoanalysis has many advantages, yet there are very versatile treatment, such as art therapy, hypnosis, color therapy and music therapy. Human has always used music instruments to express his thoughts and feelings. For years, man has tried to make use of music for various occasions such treatment, healing and relaxation[4]. From the era of Avesina, there has been much philosophical and discussion about the benefits of art and healing, but less empirical research exists in the literature[5]. Music is the most accessible and most researched medium of art and healing, and there has been a principal emphasis on the soothing capacity of music and its ability to offset overly technological approaches to care[6].The question here is that does spiritual music affects on our health?

Review of literature

Recent studies have pointed to the beneficial effects of music therapy[7], although its use is still controversial[8] Some kind of music has positive effects and some negatives. Research has shown that non-verbalmusic and listening to soft musicand even Quran effects on relaxation[9,10] vice versa music video, harsh music, and violent music creates aggressive thoughts, feelings and behaviors.[11,12] Thoma and et al confirm that music listening has been suggested to beneficially impact health via stress-reducing effects.[13,14] Aldridge and beck in different research showed a widely researched phenomenon is the use of music in the control of chronic cancer pain.[15] Music therapy has positive effects on cancer and reductions in pain[16] and increases in immunity, decreases in anxiety, and reductions in psychological and physical symptoms[17] in several clinical studies examining the effects of music and music therapy on healing and wellness, music has been found to be a form of relaxation and anxiety reduction.[18-20] Nazemian confirms Linguistic and musical structures of Qur'an are associated with verbal content.[21] Zakerikish and et alissues that music can cause the balance in human body.[22] Khalafbeygi and et al research is about the effect of music in schizophrenia which is a serious mental illness which is associated with movement disordersand executive dysfunction. Some type of executive impairments could be treated by training.

This Clinical trial 56 patients who were members of Iranian Association of Patients with Schizophrenia, randomly divided into two groups. The intervention group took part in 24 music therapy sessions. There was no significant difference between groups in Strop (P > 0.05), however after course, scores has increased in intervention group. Tower of London results were not significantly different (P > 0.05), although intervention group showed less speed but more points. The results of Purede Peg Board were not significantly different (P > 0.05). In Wisconsin Card Sorting Test, intervention group had better function (P < 0.05). Therefore they conclude Music can enhance executive function scores, but factors like larger sample size and follow up periods should be considered.[23] Gromko (2005) studied those precedent who received themusic instruction showed significantly greater gains in phonemic awareness when compared to the control group.[24] McIntosh and colleagues have evaluated the positive effect of auditory stimulation on Parkinson's Disease Activity.[25] Attigh and et al suggest that musical voice activities can accelerate the improvement in balance function of autistic children with low performance.[26] Shayesteh and khosropanah studies the effects of music on human morality with respect to historical , ethical and educational use, it is an undeniable. View of Ethics embracing secular music as a tool for education, business spirit and virtues of humanity is good. Although, logically, the relationship between music and ethics is not deniable .In the view of Islamic morality, the value of spiritual music is ignored by more attention dancing music. This article aims is to investigate the effects of music on mood, and results is that music affects the taste of humans and repeated behavior.[27] Gholami and etalreveals in his study on the impact of two methods of music therapy and relaxation on the aggression in high school students that the results of the analysis showed that combined relaxation and music therapy is effective on aggression of male students. The aggression mean scores of the experimental group was significantly reduced as compared to the control group (P < 0.001) and concludes that the use of combined relaxation and music therapy is effective on reducing aggression in male students[28].Many religious and spiritual traditions and practices can be called disembodied through their ignoring of vital facets of the whole person and rendering them illegitimate as reliable sources of spiritual insight in their own right.[29] In fact, systematic reviews of the research literature reveal more than 300 studies that document a positive association between religion and physical health-effects that have been found over time and across socio demographic groups[30,31]. Rabei taste music therapy and conclude positive effects[32]. Heidariand Shahbaziconcludeslistening to Quran is more effective in reducing stress than listening to music. Impact of listening to religious sound verified surgery, anesthesia and vital signs of stability[33,34]. Masoumy and Ganju compare the Effects of religious sound (Holly Quran) and Music Sound on Student's Text Anxiety. They result the mean of test anxiety in post test in the religious listener group and the music group was respectively (35/27± 3/ 41) and (43/67±6/50). This differences statistically were significant (P < 0/05). The comparison between the two groups shows that mean test anxiety in Quranic group significant decreased and the differences was spatiality significant (t=6.26, p < 0.05). They conclude Listening to the both religious sound and music sound before test is effective to decrease test anxiety,[35] but the results show that Quran sound is more effective.[36] There are just some studies which are concentrated on negative effects of music for example Masoudi and Radad studies listening to music is one of the preventive factors for students froms tudying[37]. In different religions there are various theories towards music for instance the Post Christian West has often dismissed notions of spirituality as superstitious in line with scientific, objective rationality or to be avoided in the interests of religioustolerance. The notion of spirituality as part of self-actualization led people to regard the musical experience as the last remaining place for the spiritual in Western society.[38,39]

Conclusion

Spirituality with different mechanism lends to health. People who spend more time with mild and meaningful sound are more in balance than others. Research shows that music is applicable for reducing stress, but not all of religions suggest people to use it. Scientist says music is impact in health but some kind of music has bad effects and some are good, therefore it is good for people to listen to music and choose the best kind. Reviewing of studies shows

that just listening to spiritual music especially listen to Holy Quran and religious music in different beliefs is so useful for health and it shows although subject of music is so ancient but the study in this context is too few and it is essential for scientist to do more research on religious music and the effects on health.

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Commentary

DSM 5: Changes and Controversies

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The first diagnostic and Statistical manual of American Psychiatric Association (DSM) first appeared in 1952 and current edition, DSM-5, is the sixth revision. There are various significant changes in the classification system now. DSM-5 is using Arabic numeral in its name, instead of roman number and multiaxial system is removed.[1]

Since its introduction in May 2013, DSM 5 is widely debated all over the world. It is argued that DSM 5 increase the sensitivity of psychiatric diagnosis and reduce its specificity by lowering the thresholds for diagnosis and introducing new mental disorders with low diagnostic validity and reliability.[2-4]

Here we outline the important differences in DSM- 5 from text revision of DSM IV (DSM IV TR)[1,5,6] and controversies faced currently in the different areas.

Neuro Developmental Disorder

Table 1: Changes in Criteria for Neurodevelopmental disorders in DSM-5

1.	**Intellectual disability**	☆ It was termed Mental Retardation and criteria now emphasize need of assessment of both cognitive capacity and adaptive functioning.
2.	**Communication disorders**	☆ Language disorders (combines DSM IV expressive and mixed receptive and expressive language disorders) ☆ Speech sound disorder(known as phonological disorder in DSM IV TR) ☆ Childhood onset fluency disorder(a new name for stuttering)and ☆ A new identity social (pragmatic)communication disorder**.**
3.	**Autism spectrum disorder (ASD)**	☆ In this new entity DSM- 5 considers Autistic disorders, Asperger's disorder, childhood disintegrative disorder, pervasive developmental disorder not otherwise specified as single entity with varied levels of severity in two domains of social interaction deficit and restricted repetitive behaviors (RRBs)
4.	**Attention deficit /Hyperactivity disorder (ADHD)**	☆ It was earlier included in the chapter of diagnoses usually first made in infancy, childhood or adolescence. ☆ Examples have been added, cross situational requirement has been strengthened ☆ Onset criterion changed to presence of several symptoms before age 12. ☆ Subtypes has been replaced with presentation specifier ☆ A co morbid diagnosis with ASD is now allowed ☆ Symptom threshold changed to cut off of 5 symptoms for adults and instead of 6 for younger persons.
5.	**Specific Learning disorder**	☆ It combines DSM IV TR reading disorder, mathematics disorder, disorder of written expression and learning disorder not otherwise specified. ☆ Coded specifiers for the deficit type in each area are included
6.	**Motor disorders**	☆ It includes developmental co ordination disorder, stereotypic movement disorder, Tourette's disorder, persistent (chronic) motor or vocal tic disorder, provisional tic disorder, other specified and unspecified tic disorders

Controversies: loosening the criteria would facilitate diagnosis of ADHD in adults eager to get stimulant drugs for performance enhancement and recreation.[2]

Schizophrenia Spectrum and other Psychotic Disorder

Table 2: Changes in criteria for Schizophrenia spectrum and other psychotic disorders in DSM-5

Schizophrenia	☆	In criterion A special attribution of bizarre delusion and Schneiderian first rank auditory hallucination has been eliminated due to poor specificity and reliability.
	☆	Now it is specified that at least one of the two symptoms in criterion A must be delusions, hallucination and disorganized speech.
	☆	DSM IV subtypes are now eliminated
Schizoaffective	☆	Now based on the conceptual and psychometric grounds, it is required that a major mood episode must be present for majority of the disorder's duration after criterion A has been met, making it a longitudinal diagnosis instead of cross sectional.
Delusional Disorder	☆	Criterion A does not require delusion to be non bizarre and a specifier for bizarre is now added**.**
	☆	New exclusion criteria for obsessive compulsive disorder (OCD) and body dysmorphic disorders has been added**.**
	☆	Delusional disorders are not separated from shared delusional disorder.
Catatonia	☆	Same criteria are used to diagnose in all context and all require three symptoms from 12 to be met.
	☆	It can be diagnosed as specifier for depressive, bipolar and psychotic disorders**.**
Other specified schizophrenia spectrum and other psychotic disorder	☆	In DSM-5, Attenuated psychosis syndrome, Delusional symptoms in partner of delusional disorder are included here.

Controversies: Loosening the criteria and inclusion of prodromal syndromes create about some of the inherent risks in prodromal research as medication exposure, stigma among false positives, effect of information on the patients, families and institutions.[7]

Bipolar and Related Disorders

Table 3: Changes in criteria for bipolar and related disorders in DSM-5

Bipolar disorders	☆	Criterion A for manic and hypomanic episodes now emphasizes on changes in activity and energy as well as mood.
	☆	Bipolar I, mixed episode now does not requires presence of full criteria for mania and major depressive episode (MDE). Instead a new specifier "with mixed features", has been added.
Other specified bipolar and related disorder	☆	Categorize those with past history of MDE and meeting criteria for hypomania except duration criterion or having too few symptoms of hypomania to meet criteria for bipolar II disorders but duration is sufficient at 4 or more days.
	☆	Anxiety distress specifier has been added

Depressive Disorders

DSM 5 contains several new categories: Disruptive mood dysregulation, which is to prevent over diagnosis of bipolar disorders in children up to age 18 years, who exhibit persistent irritability and frequent episodes of extreme behavioral dyscontrol and premenstrual dysphoric disorders.

Table 4: Changes in criteria for Depressive disorders in DSM-5

Major depressive episode	☆	Specifier "with mixed features" has been added
	☆	Bereavement exclusion is omitted**.**
Persistent depressive disorder	☆	Includes chronic MDE and dysthymia

Controversies: Now it is too easy to diagnose MDE in people experiencing normal grief. It is also suggested that instead of omitting 'bereavement exclusion' DSM 5 should have considered extending a similar exclusion for sadness in situations of severe life stress.[8]

Anxiety Disorders

The requirement for individual above 18 years of age, that they should recognize their anxiety as excessive or unreasonable has been deleted in agoraphobia, specific phobia and social phobia. Instead anxiety must be out of proportion to the actual danger or threat after taking cultural contextual factors into account. The 6 months duration which was for individuals less than 18 years of age, has been extended to all ages.

Table 5: Changes in criteria for Anxiety disorders in DSM-5

Panic attacks	☆ DSM IV terminology for different types has been replaced with unexpected and expected panic attacks.
	☆ .Panic attacks can now be listed as specifier applicable to all DSM 5 disorders.
Panic disorders and agoraphobia	☆ Panic disorder and agoraphobia are unliked in DSM 5. The co occurrence of panic disorder and agoraphobia are now coded as two diagnosis
	☆ Endorsement of fears from two or more diagnosis is required.
	☆ Criteria for agoraphobia are extended to be consistent with other anxiety disorders.
Specific phobia	☆ Different types are now referred to as specifiers.
Social anxiety disorder	☆ "Generalized" specifier has been replaced with a "performance only" specifier.
Separation Anxiety disorders	☆ Earlier included in disorders first diagnosed in infancy, childhood, or adolescence.
	☆ No specification for the age of onset as 18 years or before.
	☆ Duration criteria for 6 months or more, for adults has been added.
Selective mutism	☆ Earlier included in disorders first diagnosed in infancy, childhood, or adolescence.

Obsessive Compulsive and Related Disorder

- ☆ This is a new chapter in DSM 5 and includes disorders related to one another in terms of diagnostic validity and clinical utility. New disorders include hoarding disorder, excoriation (skin-picking) disorder, substance-/medication-induced obsessive-compulsive and related disorder, and obsessive-compulsive and related disorder due to another medical condition.
- ☆ Specifier "with poor insight" for level of insight has been more refined .
- ☆ The "tic related" specifier has been added, reflecting that this co morbidity may have important clinical implication.
- ☆ Body dysmorphic disorder has a diagnostic criterion based on prevalence, describing repetitive behaviors or mental acts in response to preoccupation with perceived defects or flaws in physical appearance has been added. A "with muscle dysmorphia" specifier is also added.
- ☆ Hoarding disorder, earlier listed as one of the possible symptoms of obsessive compulsive personality disorders, is now a separate diagnosis.
- ☆ Trichotillomania is now known as Trichotillomania (hair pulling disorder) and has been placed here from DSM IV impulse control disorder not elsewhere classified.

Dissociative Disorders

- ☆ Derealization is included in the name and symptom structure of earlier depersonalization disorder and is now called as depersonalization/derealization disorder.
- ☆ Dissociative fugue is now a specifier of dissociative amnesia and not a separate diagnosis.
- ☆ Dissociative identity disorder criterion A has been expanded to include certain possession forms phenomena and functional neurological symptoms. Symptoms may be self reported or observed and recall gaps can occur for everyday events and not just for experience.

Somatic Symptom and Related Disorders

- ☆ In DSM 5 somatoform disorders are now referred to as somatic symptom and related disorders.
- ☆ The DSM 5 reduces the number of these disorders and subcategories to avoid problematic overlap.
- ☆ Diagnosis of somatization disorders, hypochondriasis, pain disorders, and undifferentiated somatoform disorder has been removed.

Controversies: It is argued that the new diagnosis are understudied and overinclusive and going to mislabel many medically ill patients also as mentally ill.[2]

Feeding and Eating Disorders

It includes several diagnosis from the DSM IV chapter "Disorders usually first diagnosed in infancy, childhood or adolescence" as pica and rumination disorder, elimination disorder and feeding disorder which is renamed as Avoidant/ restrictive food intake disorder.

- ☆ The rapid eye movement sleep behavior disorder and restless legs syndrome are now independent disorder to reduce the use of DSM IV diagnosis of "not otherwise specified."

Sexual Dysfunction

- ☆ DSM 5 considers the research that sexual response is not always a linear uniform process and distinctions between certain phases (eg. desire and arousal) are artificial.
- ☆ In DSM-5, gender-specific sexual dysfunctions have been added, and, for females, sexual desire and arousal disorders have been combined into one disorder: female sexual interest/arousal disorder.
- ☆ All of the sexual dysfunction (except substance/ medication induced) now require a minimum duration of 6 months.
- ☆ DSM IV categories of vaginismus and dyspareunia are combined as Genito-pelvic pain/ penetration disorder in DSM 5 and sexual aversion disorder has been removed.
- ☆ Sexual dysfunction due to a general medical condition and the subtype due to psychological versus combined factors have been deleted.
- ☆ The following associated features are described in the text: partner factors, relationship factors, individual vulnerability factors, cultural or religious factors, and medical factors.

Table 6: Changes in criteria for Feeding and Eating Disorders in DSM5

Pica and rumination	☆ Diagnosis can be made for individuals of any age.
Avoidant/restrictive food intake disorder	☆ Criteria are significantly expanded to include any age group
Anorexia nervosa	☆ The requirement for amenorrhea has been eliminated.
	☆ Guidance regarding how to judge that individual is at or below a significantly low weight is now given in text.
	☆ Criterion B is expanded to include persistent behavior that interferes with weight gain along with overt fear of weight gain.
Bulimia Nervosa	☆ Reduction in minimum average frequency required for binge eating and inappropriate compensatory behavior from twice to once weekly.
Binge eating disorder	☆ Minimum average frequency of binge eating has been changed from at least twice weekly for 6 months to at least once weekly over the last three months.

Controversies: The reduction in the frequency required for diagnosing binge eating would include unnecessary false positive diagnosis.[2]

Sleep-Wake Disorders

- ☆ Sleep disorders related to another mental disorder and sleep disorder related to a general medical condition have been removed from DSM 5.
- ☆ The diagnosis of primary insomnia is renamed insomnia disorder to avoid differentiation of primary and secondary insomnia.
- ☆ DSM 5 also distinguishes narcolepsy from other forms of hypersomnolence, which is now known to be associated with hypocretin deficiency.
- ☆ The pediatric and developmental criteria and text are integrated throught the classification.
- ☆ Breathing related sleep disorders are divided into three relatively distinct disorders: obstructive sleep apnea hypopnea, central sleep apnea, and sleep related hypoventilation.
- ☆ The subtypes of circadian rhythm Sleep-wake disorders expanded to include advanced sleep phase syndrome, irregular sleep wake type, and non 24 hour sleep wake type, whereas jet lag type has been removed.

Gender Dysphoria

- ☆ Gender dysphoria is a new diagnostic class in DSM-5 and reflects a change in conceptualization of the disorder's defining features by

emphasizing the phenomenon of "gender incongruence" rather than cross-gender identification, as was the case in DSM-IV gender identity disorder.

- ☆ Separate criteria sets are provided for children, adolescents and adults and the previous Criterion A and B (cross-gender identification and aversion toward one's gender) are merged.
- ☆ The term "the other sex" is replaced by "some alternative gender." Gender instead of sex is used system-atically because the concept "sex" is considered inadequate.
- ☆ In the child criteria, "strong desire to be of the other gender" replaces the previous "repeatedly stated desire" and Criterion A1("a strong desire to be of the other gender or an insistence that he or she is the other gender . . .) is now necessary (but not sufficient).
- ☆ The subtyping on the basis of sexual orientation and a posttransition specifier has been added.

Disruptive, Impulse-Control and Conduct Disorders

- ☆ This is a new chapter which brings together disorders that were previously included in the chapter "Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence" (i.e., oppositional defiant disorder; conduct disorder; and disruptive be-havior disorder not otherwise specified) and the chapter "Impulse-Control Disorders Not Otherwise Specified".

Substance Related and Addictive disorder

- ☆ This chapter has been expanded to include gambling disorder.
- ☆ DSM- 5 does not separate the diagnosis of substance abuse and dependence and disorders are divided into two groups: substance use disorders(SUD) and substance induced disorders.
- ☆ The DSM IV recurrent legal problems criterion has been deleted and a new criterion for craving has been added.
- ☆ The threshold for substance use disorder diagnosis in DSM 5 is set at two or more criteria.
- ☆ Cannabis and caffeine withdrawal are new addition in DSM 5.
- ☆ The criteria in DSM 5 that are from DSM IV abuse are new for tobacco in DSM 5.
- ☆ Severity of the substance use disorder is based on number of criteria endorsed.
- ☆ The DSM IV specifier for a physiological subtype and diagnosis of polysubstance abuse has been eliminated.
- ☆ A new specifier include "in a controlled environment" and on "maintenance therapy."

Controversies: There is a expert debate going on DSM Diagnosis of substance use disorders and it is argued that the DSM 5 does not articulate any specific model of how SUD are related to SUD symptoms and the resulting checklist is undimensional. DSM 5 has also opened the door to fad diagnosis of internet, sex, shopping and other untested addictions by adding gambling disorders as first example of the concept of behavioral addictions.[9]

Table7: Changes in the criteria for Disruptive, impulse-control and conduct disorders in DSM-5

Oppositional defiant disorder	☆ Symptoms are divided into 3 categories to reflect both emotional and behavioral symptomatology.
	☆ Exclusion criteria for conduct disorder has been removed
	☆ Note on guidance on frequency needed for a behavior to be considered symptomatic has been added
	☆ Severity rating has been added to the criteria
Conduct Disorder	☆ Descriptive feature specifier for those with limited prosocial emotion.
Intermittent Explosive Disorder	☆ Verbal aggression and non destructive aggression also included.
	☆ Now provides more Specific criteria for frequency.
	☆ A minimum age of 6 years is now required.
	☆ The relationship with other disorder like ADHD is now more clarified.

Neuro Cognitive Disorders

- ☆ The criteria for delirium have been updated and clarified.
- ☆ The DSM IV diagnosis of dementia and amnestic disorder are subsumed under new

entity called as major neuro cognitive disorders (NCD).

- ☆ A new disorder named mild NCD permits the diagnosis for less disabling conditions that require focus.
- ☆ DSM 5 provides an updated list of neuro cognitive domains necessary for establishing the presence of NCD

Personality Disorders

- ☆ In the general criteria for personality disorders, in section III, a revised personality functioning criterion (Criterion A) has been developed.
- ☆ Moderate level of impairment in personality functioning is now required for diagnosis and based on greater impairment and presence of pathological traits, a diagnosis of personality disorder—trait specified replaces personality disorder not otherwise specified.

Controversies: DSM 5 task was to create a dimensional system for diagnosing personality disorder but it failed badly and the result is too complicated for clinical practice.[2]

Paraphilic Disorders

- ☆ In DSM 5 there is addition of the course specifier "in a controlled environment" and "in remission" to the diagnostic criteria sets for all paraphilic disorders.
- ☆ In DSM 5 there is distinction between paraphilias and paraphilic disorders. A paraphilic disorder is a paraphilia that is currently causing distress or impairment to the individual.
- ☆ Individuals who meet both criteria A(specifies the qualitative nature of paraphilia) and B(specifies the negative consequences), would now be diagnosed as having paraphilic disorder.

Controversies: DSM -5 does not resolve the foreseen consequences of rewording criteria and disruptive nature of changes made earlier. It still requires criterion B i.e. the negative consequences of paraphilia to be there to call it a paraphilic disorder.[10]

Conclusion

The research cited the limitations of categorical diagnosis, including excessive co morbidity, excessive use of "not otherwise specified" category and boundary problems and it was thought that dimensional assessment would reduce these problems but there are a lot of controversies and concern about the potential complexity in the diagnosis and additional burden placed on mental health care providers by the dimensional approach of DSM-5. The inclusion of subthreshold and premorbid disorders as formal diagnosis creates lots of false positive diagnosis and unnecessary treatment and difficulty getting insurance for these disorders.[11] Now there is a need to consider these issues in the forthcoming classificatory systems and balance these unresolved and foreseen problems.

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Newer Development

Pica as a Culture Bound Syndrome

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Introduction

Pica is defined as the persistant eating of non-nutritive, non-food items over a period of at least 1 month. This eating is inappropriate to the developmental level of the individual and is not a part of culturally sanctioned practice.[1] It is not a disease or disorder but a behavior that results from the interaction of biological, environmental and psychological factors.[2] The term pica comes from the Latin for "magpie" a bird known for its indiscriminate and unusual eating habits.[2,3] Some of the most commonly described types of pica are eating earth, soil or clay (geophagia), ice (pagophagia) and starch (amylophagia) etc. Infants and toddlers are typically excluded from this diagnosis since mouthing objects is a normal developmental behavior at that age. The persons withhabit of pica typically eat plaster, paper, paint, cloth, hair, insects, animal droppings, sand, pebbles, and dirt. Some even eat ashes/burnt matches, cigarette buts, soap etc.[2] Ice-eating (Pagophagia) has also been reported as a variant of Pica.[4] Among all the varieties, geophagia (mudeating) is the most common pattern of pica practiced across the world.[5]

Epidemiology

The exact prevalence of Pica is often under reported as many people may be embarrassed to admit to these unusual eating habits, and may hide it from their physicians. Although Pica is seen in all age groups and both genders but more prevalent among the children and females. In children age group of infants is vulnerable. Pica is considered to be non pathological up to 2 years of age because they are in habit of exploring things while mouthing and teething. Pica is also commonly observed among children with the developmental disabilities (mental retardation, autism etc).[6-8]

A prospective studydone on African American women reported 38 percent prevalence rateof pica[9]. Often females who are pregnant have pica. It is particularly found in those living in poverty, tropical areas and the tribal areas. In India pica is more prevalent among rural population than urban settings. A recent survey by Obstetrics-Gynecology Clinic at Duke Medical Center in North Carolina shows that about one quarter of patients were clay-eaters.

Etiology

Though exact etiology of pica is not known but there arecertain proposed hypothesis. Organic, psychodynamic, socioeconomic, and cultural factors have been implicated in the etiology of pica. Most widely accepted theory points towards the nutritional deficiency to be the cause of Pica. Research has reported high prevalence of pica in individuals with documented iron deficiency.[2,7,10] Studies have shown that pica cravings in individuals with iron deficiency stop once iron supplements are given to them. Deficiency of calcium, zinc and many of B Complex vitamins is usual among persons with Pica.[2,11] The geophagia also has medicinal value in being used as an antidiarrhoeal agent due to its proposed tendency to absorb dietary toxins.[7]

The traumatic events are associated with pica. Common events that could signal pica include parental separation, broken families, parental neglect, lack of parent-child interaction, and child abuse. In certain people, pica may be a form of compulsive behavior in OCD.[8,9,12-16,] The persons with developmental disabilities have pica due to their inability to discriminate food and non-food items.[6,8] Even the persons with Psychosis sometimes eat non edible material.

Ingestion of paint is most commonly seen among children belonging to low socioeconomic status and is associated with lack of supervision. In the southern United States in the 1800s, geophagia was a common

practice among the slave population.[17] People who diet may attempt to ease hunger by eating nonfood substances to get a feeling of fullness. Many patients with pica say that they just enjoy the taste, texture, or smell of the item they are eating.Pica is a cultural feature of certain religious rituals, folk medicine, and magical beliefs.[17] Starch eating in pregnancy particularly treats morning sickness. Pica is followed as a cultural practice across the African population to maintain fertility.[18] In some myths many men believed that eating clay increased sexual power.

The association of pica, iron deficiency and a number of pathophysiologic states with the decreased Dopamine levels has raised the possibility of a correlation between diminished dopaminergic neurotransmission and expression and maintenance of pica.[19]

Culture bound issues

Although pica is related to certain nutritional deficiencies but it is a part of cultural practice in certain parts of the world. Cultural pica is a common practice followed in various communities and cultures across the world. Cultural pica differs from pica in children and mentally retarded people in being restricted to certain items only like mud, chalk etc.

Some historical aspects pointing towards the causative explanations of pica include (a) retention of impure blood due to cessation of menses during pregnancy, (b) delayed development of sexual organs (chlorosis), (c) digestive disorders, (d) iron deficiency, (e) cosmetic reasons, such as to achieve pale skin, (f) sexual frustration, and (g) tight-corseting.[20]

In many rural parts of India, pregnant females consume mud, clay, ash, lime, charcoal and brick in response to cravings.Nonfood substances are believed to have positive health or spiritual effects.In North India, cravings for pica substances are used as a means to predictthe sex of an unborn child. If a woman craves ash, peoplebelieve she will have a girl, whereas dust cravings indicate that she is pregnant with a boy.[21] Children practice geophagy because of the proximity to the soil play. In India, Pica for mud eating is culturally accepted in families in western Uttar Pradesh. The whole of the family members follow Pica in form of mud eating.

It is also prevalent among the African females specially "Chagga" women where they relate soil eating to the fertility and reproduction. The Chagga is one of the African ethnic groups in Tanzania.The women's identity is closely related to the productivity of land. It is a form of learned behavior. The women revealed that they had experience "Tamaa" (desire) in some pregnancies and not in others. They also express specific preferences for different forms of soil, obtained from specific places and preferred to be eaten either "dry" or "wet". The practice of geophagy is culturally controlled in this part of the world. Women who break the cultural norms related to this practice are negatively regarded. Thechagga community believes in the magical artefacts related to male and female sexual organs. After the child birth, the husband and wife are not allowed to have sexual intercourse for three months to ensure that semen does not pollute the mother's milk. And during that period the practice of geophagy helps to perpetuate life and maintain productivity. The experience and satisfaction from eating soil is compared to the satisfaction from the sexual intercourse. The mother figure is highly regardedand worshipped in the Chagga society for carrying on act of reproduction. When interviewed, some of chagga women also replied that eating mud increases the blood which is needed during pregnancy.[22]

Kaolin (white clay or chalk eating) ingestion has been found out to be a form of pica in central Georgia Piedmont area.In Georgia it appears to have culturally transmitted and not associated with other psychopathology.[23] Those who began eating chalk during pregnancy were introduced to this behavior by their mothers. In the 1950s and 1960s, the practice was so popular that clay-filled lunch bags were sold at Alabama bus stops as snacks for travelers. Magnesium carbonate blocks are sold and advertised as edible products within certain regions of the United States and Mexico. Simpsonfound that magnesium carbonate was one of the most popular non-food items to ingest, and that some women even believed that consuming chalk would be beneficial for the fetus.[24] Nelson et al found that magnesium carbonate was the main component of the antacids most frequently taken by pregnant women.[24] In Africa Kaolin is eaten for suppressing hunger or for feelings of pleasure. It is also used as an antidiarrhoeal agent. Kaolin is naturally occurring clay used in manufacture of ceramics, as a coating

or filler for paper or textiles.

Some tribes in Peru and Bolivia eat clay to overcome the health risks associated with toxins found in their staple diet, potatoes.[26] In certain parts of Haiti clay mud is turned into pancakes and cookies and sold as eatable items in poorer population. One recent study analyzed samples of African soil that was sold for geophagic purposes in various parts of Africa, Europe, and the U.S. of particular concern to the researchers were the microbe and lead levels. Small levels of mercury and cadmium were also present.[27]

Differential diagnosis

Ingestion of nonnutritive, nonfood substances may also be observed in individuals with autism, schizophrenia, or certain physical disorders (e.g. Kleine-Levin syndrome). Anorexia nervosa (AN) is an eating disorder characterized by a weight loss of at least 15% of expected body weight, with dreaded fear of weight gain, relentless dietary habits that prevent weight gain, and a disturbance in the way in which body weight and shape are experienced.[28] Bulimia is characterized by recurrent episodes of binge eating with a sense of lack of control over eating during the episode (e.g. a feeling that one cannot stop eating) with self induced vomiting as a compensatory behaviour. Avoiding/ restrictive behaviour is manifested by persistent failure to meet appropriate nutritional needs associated with significant loss of weight.[1]

Health hazards

Pica may have many adverse consequences on body. These patients are susceptible to electrolyte and metabolic disorders, lead and mercury poisoning, parasitic infections, intestinal obstruction/perforation and ultimately may land up with medical or surgical emergency[2]. The mud or clay is often contaminated by animal or human faeces and may contain parasite eggs like Ascaris. Lead toxicity is the most dangerous complication that can be faced with Pica.[2,29] In certain cases dental abnormalities like tooth abrasion and surface tooth loss may be evident.[30] Pica foods do not generally possess any calorie values. The babies born to mothers who practice pica during pregnancy may be low birth weight, premature, bornwith physical abnormalities, and even death is reported among such newborns.[31]

Treatment

Pica remits spontaneously in majority of the cases. Treatment plan must focus on the factors contributing towards pica and address the underlying psychological issues.[2] There is no specific treatment for pica. Any nutritional deficiencies also need to be addressed and corrected. The medical emergencies like lead poisoning or surgical emergency like intestinal obstruction has to be treated accordingly.[17] However recent research suggests that the drugs enhancing Dopaminergic activity may prove to be beneficial. SSRIs have been shown to be effective.[4,14,16] One of the case reports has shown the benefit after using Olanzapine in a patient of Pica.[32] The comorbid psychiatric illness and underlying psychological issues need to be addressed and managed. It is also recommended to expand the supplementation programs like mandatory flour fortification to enhance population-wide iron supply and safer pregnancies. The use of brief physical restraints and aversive conditioning can be helpful in infants and toddlers.[2,28] As Pica is acquired in cultural settings, affected people may be given cognitive behavioral therapy.

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Drug Review

Vortioxetine: A New FDA Approved Antidepressant

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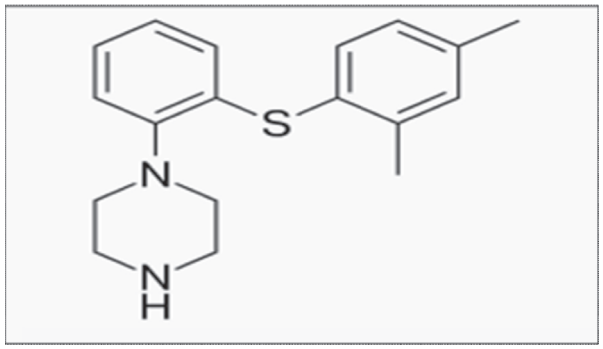
Introduction

Vortioxetine is a new antidepressant that has been approved for the treatment of major depressive disorder (MDD) by the US Food and Drug Administration (FDA) on 30 September 2013.[1,2] Though it is a serotonin reuptake inhibitor, it has got multimodal action on different subtypes of serotonin receptors. Its chemical structure is as follows:

The chemical name of vortioxetine is 1-[2-(2,4-



Dimethyl-phenylsulfanyl)-phenyl]piperazine].

Pharmacodynamics

Vortioxetine has action on multiple serotonin receptor subtypes. It acts as an antagonist on 5-HT3A and 5-HT7, a partial agonist on 5-HT1B receptor and as an agonist on 5-HT1A receptor. It also inhibits reuptake of serotonin by blocking serotonin transporter.[3] Due to its multimodal neurotransmitter enhancer profile, it has been suggested that it might need lesser receptor occupancy rate for clinical effectiveness than other selective serotonin reuptake inhibitors (SSRIs) and selective norepinephrine reuptake inhibitors (SNRIs).[1,2] Vortioxetine has high affinity for serotonin receptor, but not for Dopamine or Nor-epinephrine receptors.[4] Studies have also suggested that serotonergic modulation of glutamate neuro-transmission through 5-HT1A, 5-HT1B, 5-HT3, and 5-HT7 receptors could be responsible for few clinical effects like improvement in cognitive dysfunction[5]. In a clinical study, it was found that in contrast to fluoxetine, Vortioxetine was effective in suppressing the neuronal firing without saturating serotonin transporter (SERT) occupancy and producing faster recovery of serotonergic neuronal firing.[6]

Pharmacokinetics

Vortioxetine has linear pharmacokinetics for the dose range of 2.5–60 mg and exposure is proportionate to the dose.[4,7] After oral administration maximum concentration in plasma is reached in 7-11 hours. The plasma half-life is approximately 66 hours and it takes around two weeks to achieve steady state plasma levels. The oral bioavailability is around 75%.[4] It is 98% protein bound in plasma[4]. Vortioxetine is primarily metabolized by CYP2D6 to pharmacologically inactive compounds.[4,8] The poor vortioxetine metabolizers have about twice the drug plasma concentration in comparison to extensive metabolizers.[4] The maximum recommended dose of vortioxetine is 10 mg/day in known CYP2D6 poor metabolizers. Reduction in the dose of vortioxetine is required by one half when patients are receiving a CYP2D6 strong inhibitor (e.g., bupropion, fluoxetine, paroxetine, or quinidine) concomitantly.It is recommended to increase the dose of vortioxetine when a strong CYP inducer (e.g., rifampin, carbamazepine, or phenytoin) is co-administered for greater than 14 days. The maximum recommended dose should not exceed three times the original dose. The dose of

vortioxetine should be reduced to the original recommended dose within 14 days, when the inducer is discontinued.[4]

Efficacy In Clinical Trials

The efficacy of vortioxetine has been established in both elderly and non elderly patients by short term (6-8 weeks) placebo controlled trials.[9-13] Most of the studies have used changes in either Montgomery–Åsberg Depression Rating Scale (MADRS) score as primary outcome measure or have used Hamilton Depression Rating Scale (HDRS) to assess changes in depressive symptoms[9-13].Patients with treatment resistant depression or comorbid psychiatric and medical disorders were excluded. In all the clinical trials, Vortioxetine was used at dosages of 1, 2.5, 5, 10, 15 and 20 mg/day; all administered once daily.[9-13] Five of the studies included an active control group for assay sensitivity,[9-13] either venlafaxine extended-release 225 mg/day,[9] or duloxetine 60 mg/day.[9,10,12,13]

Five studies found vortioxetine to be effective as an antidepressant at doses of 1 mg/day [14], 5 mg/day,[9, 14] 10 mg/day,[9, 14] 15 mg/day,[12] and 20 mg/day[12,13,15] respectively.

Two trials found vortioxetine as an effective antidepressant at dosages of 20 mg/day, but not for 10 mg/day[15] or 15 mg/day.[13] In one trial both vortioxetine and active control failed to separate from placebo.[10] One trial was unequivocally negative for vortioxetine as the active control (duloxetine) did separate from placebo.[11] Two other trials failed to demonstrate statistical superiority for vortioxetine over placebo on the primary outcome measure, but the absence of an active control for assay sensitivity makes it impossible to differentiate these studies as being failed vs. negative.[16,17] The antidepressant effect of vortioxetine was observed to start only after 2 weeks and reached maximum at or after 6 weeks.[4] Also vortioxetine was found to be superior over placebo as measured by improvement in the Sheehan Disability Scale (SDS) functional improvement total score.[12,15] A double blinded study on elderly with MDD compared vortioxetine with placebo and an active control group on duloxetine (60 mg/day) found vortioxetine to have statistically significant greater improvement in reducing depressive symptoms in comparison to placebo.[18] One placebo controlled trial supported use of vortioxetine as maintenance treatment with superiority of vortioxetine over placebo on the primary outcome measure of time to relapse.[19] No difference had been observed in treatment effectiveness on the basis of age, gender or race.[4]

Bidzan et al. in 2012 found that Vortioxetine is effective in the treatment of generalized anxiety disorder at a dose of 5 mg/day with minimal side effects and significant reduction in anxiety in Hamilton's Anxiety rating scale.[20] Rothschild et al in 2012 had conducted a similar study (vortioxetine 5mg/day for 8weeks with placebo control) in US population on patients suffering from generalized anxiety disorder and found insignificant clinical improvement.[21]

It is expected that Vortioxetine is likely to improve the cognitive function due to its properties of 5-HT3, 5-HT7 receptor antagonism and 5-HT1A receptor agonism.[22] In animal (rat) models, vortioxetine is increasing the extracellular level of acetylcholine and histamine, thereby improving the episodic memory.[22] Study on animal models revealed that, vortioxetine also increases proliferation, survival and maturation of granule cells at the dentate gyrus of hippocampus, improving the memory function.[23]

Dosage and Administration

The recommended oral dose is 10 mg orally, once a day which can be increased to 20 mg/day as tolerated and higher dosages have demonstrated better treatment effect in trials. In patients who are unable to tolerate higher dosages, 5 mg/day can be used. No effect of food on absorption has been found.[4]

The abrupt stoppage of vortioxetine can cause discontinuation symptoms in the form of headache and muscle tension and if on higher dosages like 15 mg or 20 mg, it is recommended to gradually taper the dose to 10 mg for one week before full discontinuation.[4]

Side Effects

Vortioxetine was generally well tolerated in clinical trials in patients with major depressive disorder (MDD).[4] The most commonly observed adverse reactions in patients suffering from MDD treated with vortioxetine in 6 to 8 week placebo-controlled studies (incidence e"5% and at least twice the rate of placebo) were nausea, constipation and

vomiting.The incidence of spontaneously reported side effects related to sexual function was low and similar for vortioxetine as for placebo[9,14,18]. Vortioxetine had no significant effect on body weight as measured by the mean change from baseline in the placebo-controlled studies[4]. Vortioxetine lacks deleterious cardiac side effect like prolongation of QTc Interval so can be safely used in patients with cardiac conduction abnormalities.[4]

Contraindications

Vortioxetine is contraindicated if there is known hypersensitivity reaction to it. The use of vortioxetine is to be avoided within 14 days of stoppage of monoamine oxidase inhibitors (MAOIs) and co-administration with linezolid or intravenous methylene blue.[4]

Warnings and Precautions

Vortioxetine is a Pregnancy Category C medication.[4] In certain special circumstances vortioxetine, is to be used cautiously and the risks associated with use of vortioxetine are as below[4]:

- Suicidal thoughts and behaviors in adolescents and young adults
- Serotonin syndrome: Especially when co administered with other serotonergic agents
- Abnormal bleeding: Especially when administered with aspirin, NSAIDS and anti-coagulants
- Manic/hypomanic switch
- Discontinuation syndrome
- Hyponatremia
- Vortioxetine had undergone its phase II and III trial in 73 study locations of different countries out of which five study centers were in India.[24] It is yet to be launched in India.

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Drug Review

Guanfacine Extended release for ADHD

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Introduction

Guanfacine extended release (Intuniv) is a new non-stimulant drug approved for treatment of ADHD in the age group of 6-17 years. It was approved by the FDA in September 2009. Chemically it is *N*-amidino-2-(2,6-dichlorophenyl) acetamide monohydrochloride. The molecular formula is $C_9H_9C_{l2}N_3O \bullet HCl$.

The drug has demonstrated efficacy for inattentive and hyperactive/impulsive symptom domains in 2 large placebo controlled clinical trials lasting 8 and 9 weeks.[1,2] Guanfacine has important advantages over stimulants. It is just as effective and does not suppress appetite.

Mechanism of Action

Deficits in PFC (Pre Frontal Cortex) function lead to poor impulse control, distractibility, hyper-activity, forgetfulness, and poor organization and planning. There is general agreement that ADHD involves weakened PFC function and medications that treat ADHD ameliorate PFC deficits Guanfacine ER is indicated for treatment of ADHD as monotherapy and as adjuvant to CNS stimulants. Being a selective α_{2A} receptor agonist, it helps to improve attention span as the receptors are concentrated heavily in the prefrontal cortex.[3]

As an antihypertensive agent, Guanafacine IR stimulates α_{2A} - adrenergic receptors, thereby reducing sympathetic nerve impulses from the vasomotor center to the heart and blood vessels resulting in decrease in peripheral vascular resistance and a reduced heart rate.[4]

Pharmacokinetics

Guanfacine has an oral bioavailability of 80%. High-fat meals increases the absorption of the drug—Cmax increases by 75%. The drug is approximately 70% bound to plasma proteins, and it is widely distributed throughout the body. The time taken to reach peak plasma concentration is 5 hours.

Fifty percent of the drug is metabolized in the liver, primarily to the glucuronide and sulfate of 3-hydroxyguanfacine, oxidized mercapturic acid derivatives, and other minor metabolites. Guanfacine HCl is metabolized primarily by cytochrome P-450 (CYP) 3A4. The elimination half-life is 17 hours and it is eliminated mainly via kidney.[5]

Clinical results

Guanfacine XR reduced both inattentive and hyperactive/impulsive symptoms in 2 phase III randomized, placebo-controlled trials. In the first trial, 345 children aged 6 to 17 received placebo or GXR, 2 mg, 3 mg, or 4 mg once daily for 8 weeks. In the second study, 324 children aged 6 to 17 received placebo or GXR, 1 mg, 2 mg, 3 mg, or 4 mg, once daily for 9 weeks; the 1-mg dose was given only to patients weighing < 50 kg (<110 lbs). GXR use was associated with significantly lower ADHD Rating Scale-IV score compared with placebo (-16.7 vs -8.9) in 8 weeks trial and (-19.6 vs -12.2) in 9 weeks trial.[6]

Indications

Guanfacine extended release is indicated for the treatment of ADHD in the age group of 6-17 years.

Dosage and administration

Guanfacine is available in four strengths: 1 mg 2 mg, 3 mg, and 4 mg.The initial dose is 1mg , and then increase by 1mg each week to reach a target dose of 3 mg or 4 mg. Recommended daily dose is 0.05 to 0.12 mg/kg once daily. The patient should be adivised to swallow GXR whole because crushing, chewing, or otherwise breaking the tablet's enteric coating will markedly enhance guanfacine release. While discontinuing the drug, taper the dose

in decrements of no more than 1 mg every 3 to 7 days.[7]

In hepatic or renal Impairment the dose reduction may be required in patients with clinically significant impairment of hepatic or renal function.

Adverse Reactions

The most common adverse reactions include somnolence, sedation, fatigue, abdominal pain, dizziness, hypotension, dry mouth, irritability, nausea, decreased appetite, and constipation. Many of these adverse reactions are dose-related, particularly sedation, abdominal pain, dizziness, and hypotension. Caution should be taken to prevent dehydration while on the drug to avoid hypo-tension.[8]

Precautions

The drug should not be used if patient has hypersensitivity to it or to any of the active ingredients.

Hypotension, bradycardia, and syncope: Use the drug with caution in patients at risk for hypotension, bradycardia, heart block, or syncope (e.g., those taking antihypertensives). Monitor the heart rate and blood pressure while patient is on the drug.Avoid patients becoming dehydrated or overheated.

Sedation and somnolence occurs commonly with Guanfacine. One must take caution to prevent additional CNS Supression with use of other sedatives like alcohal. Caution should be taken by patients while operating heavy equipment or driving while they are using the drug.[7]

Drug interactions

Caution should be used when guanfacine ER is given with other CYP 3A4 inhibitors such as ketoconazole. Patients should be monitored for hypotension, bradycardia, and sedation. Dose needs to be increased if used concomitantly with CYP3A4 inducers like Rifampicin.

Patients should be monitored for potential CNS side effects when guanfacine ER is given with valproic acid because of increased concentrations of valproic acid with coadministration. Sedation and somnolence may be heightened if guanfacine ER is taken with other CNS suppressants like alcohol, sedatives, hypnotics, benzodiazepines, barbiturates, or antipsychotic agents.[7]

Conclusion

The CNS stimulants form the first line treatment in ADHD. The use of non-stimulants needs to be evaluated on long term basis.

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Drug Review

Current Update on Evidence based Literature of Tofisopam

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Introduction

Tofisopam was first approved over 35 years ago and currently marketed in more than 15 countries and recently being launched in India. It has a benign safety profile and a broad spectrum of clinical activity. It is a Benzodiazepine (BZDs) derivative but differs from the classical BZDs by a variation in the position of nitrogen on the chemical structure which in case of Tofisopam is 2, 3- vicinal, which reflects in the difference in pharmacokinetics and clinical effects from other tranquilizers in use. Although Tofisopam is not approved for sale in the United States or Canada, but the D-enantiomer (Dextofisopam) of Tofisopam is undergoing a phase 2 clinical trials for the treatment of irritable bowel syndrome.[1,2]

Chemical structure: $C_{22}H_{26}N_2O_4$

IUPAC NAME: 1-(3, 4-dimethoxyphenyl)-5-ethyl-7, 8-dimethoxy-4-methyl-5*H*-2, 3-benzodiazepine.[3]

Material and Methods

Literature search was done using electronic data base on 'Google scholar' and 'pub med' using "Tofisopam "as a key word. Hand search using all the standard psychiatry, pharmacology, and medicine textbooks was conducted and all the relevant journals (last five years) related to psychiatry, neurology and allied sciences were searched. Around 104 articles related to Tofisopam were found on 'PubMed'.

Mechanism of Action

Tofisopam is a racemic 2, 3-benzodiazepine in contrast to the traditional 1, 4-benzodiazepines. It is possibly due to this difference that its pharmacology is complex. Tofisopam does not bind to the benzodiazepine binding site of GABA receptors.[4] Tofisopam has anxiolytic action but is devoid of anticonvulsant, sedative, amnestic, and muscle relaxant activities. It enhances the effects of barbiturates and ethanol only in higher doses. After repeated administration, tolerance to the drug was not observed.[5]

Tofisopam has no sedative effect and no effect on attention; it could prove to be an ideal drug of choice for day time anxiolytic use.

Animal experiments on Swiss albino mice have revealed that Tofisopam raises the c GMP and c AMP levels in brain by selectively inhibiting the PDE-4A1 (0.42 ìM) followed by PDE-10A1 (0.92 ìM), PDE-3 (1.98 ìM) and PDE-2A3 (2.11 ìM).[4] It has shown significant improvement in animal models of psychosis with special focus on negative symptoms.

Pharmacokinetics

The pharmacokinetics and metabolism of Tofisopam were studied in animals (mice, rat, rabbit, dog, and monkey) and humans. The pharmacokinetics profile of the compound can be described by a two-compartment open model, where the absorption and distribution phase were found to be rapid (t max 0.5-1.0 hour in rats and 1.0-1.5 hours

in humans). The unchanged Tofisopam and 14C-total radioactivity were eliminated from human plasma with a biological half-life (t beta 1/2) of 2.7-3.5 hours and 15-21 hours, respectively, which show a slower elimination of the metabolites).[6]

Tofisopam was administered in human volunteers in the dose of 100 mg and 200 mg twice daily for one week with oral capsules. Both the plasma and urine metabolites of Tofisopam in clinical samples were eliminated by glucoroni-dation.[7] In vitro studies of metabolism of Tofisopam found that the major metabolite of R-Tofisopam was catalysed by CYP2C9 and the metabolite of S-Tofisopam was catalyzed by CYP3A4.[7]

Animal studies

Animal models of negative symptoms of psychosis:

Tofisopam is also found to be active in animal model of negative symptom of psychosis.[4] One of the negative symptoms of schizophrenia is avolition. Prolonged immobility of animal is an indicator of avolition. Avolition is quantified with the help of forced swim test in animals. One study has established the animal model of negative symptom of psychosis using the NMDA-receptor antagonist Dizocilpine. The results of this study have shown that Tofisopam can be helpful in amelioration of negative symptoms of psychosis induced with the help of Dizocilpine. This effect of tofisopam is believed to be due to combination of facilitation of the D1-receptor-dominated prefrontal pathways and inhibition of the D2-receptor-dominated pathways.[4]

Indications

As anxiolytic and tranquilizer

Tofisopam is equi-efficacious to classical BDZs like Diazepam, Oxazepam, Nitrazepam and Lorazepam to treat different anxiety states. Significant reduction in somatic and psychic anxiety has been demonstrated in both males and females.

Some of the earlier studies on this drug date back to 1970's, in which the authors found the mild tranquilizer effect of Tofisopam. A double blind study done by Goldberg and Finnerty was conducted with a sample of 57 patients suffering from anxiety and depression. Raskin and Covi depression rating scale score was 13, Hamilton anxiety scale score >18. The subjects were divided into either the treatment group; or the control group, and were categorized based on the severity of symptoms prior to beginning the treatment and evaluations were done at 1, 2 and 4 weeks. All patients were at least moderately ill. Twenty one percent of the patients receiving Tofisopam and 10 % of those receiving placebo reported side effects. It was found to be especially effective in somatic difficulties.[8]

A comparative open label study[9] of Tofisopam and Lorazepam was conducted with a sample of 32 in anxiety disorders and the results of Tofisopam (50-100mg thrice daily) and Lorazepam (0.75mg to 1.5 mg twice daily) were evaluated over 1, 2, 4 weeks using Hamilton Anxiety Rating Scale (HAM-A). The results showed Tofisopam to be anti-anxiety drug of identical potency as lorazepam. Tolerability of Tofisopam was superior to Lorazepam.[9] Filip et al reported improvement in anxiety symptoms with Tofisopam when compared to a placebo in a small sample size (N=50), and short duration of follow up (4 weeks) in the dose of 150-300 mg/day with minimal side effects.[10]

Molcan et al 1981 conducted a study for 3 weeks on (30) patients with anxious depressive syndrome of non-psychotic origin in the dose range of 100-300mg/day. The authors concluded that Tofisopam shows significant anxiolytic effect with moderate psychoenergetic effects along with no impairment of attention or somnolence and was well tolerated with no adverse effects. Method of evaluation was based on clinical findings and Beck's scale.[11]

Varady et al.1975 had done an open, labelled prospective study on 325 outpatients of anxiety associated with various psychiatric disorders for variable period of time and utilized self rating scale of Derogatis, physicians Szobor's symptom rating scale. The effect of Tofisopam was seen as early as 1 week, maximum response by week 3 in a dose of 150-300mg/day. It was found to be an effective psychorelaxant in geriatric patients. The patients also showed increased sociability and reported no drowsiness and tiredness with no major effects on concentration and driving skills. Patients with compulsive neurosis having neuroleptic treatment failure showed the best results. Authors concluded that the drug Tofisopam may be considered as psychovegetative stabilizer or harmonizing tranquilizer agent which may be used without hazard

because of the lack of unwanted and toxic effects.[12]

Pellow and File (1986) reviewed the behavioural and biochemical profile of Tofisopam that differs considerably in its effect and mechanism of action from the classical Benzodiazepines. In man Tofisopam appears to possess anxiolytic activity without appreciable sedative and muscle relaxant side effects; in animals, however, Tofisopam totally lacks anxiolytic and anticonvul-sant properties in tests sensitive to the effects of 1, 4-benzo-diazepines. Tofisopam also has mixed dopamine agonist and antagonist-like properties in several in vivo and in vitro tests in animals. The possible relevance of the latter effects to the unusual behavioural profile of Tofisopam are discussed, and its effects compared with those of Buspirone, a novel anxiolytic that has similar activity at benzodiazepine and dopamine systems. It is proposed that these two drugs may represent a novel class of compounds that reduce anxiety by increasing the ability of patients to cope with daily tasks, rather than classical anxiolytics, that reduce anxiety by tranquilization.[13]

Effect on concentration and psychomotor skills

The effect of Tofisopam on concentration and driving skills was studied in a cross over double blind trial by Gerevich et al.1975 involving 61 young lorry drivers aged between 19-25 years. No accidents or collisions were reported after consuming Tofisopam in the morning and evening at a dose of 50mg. there were no driving mistakes observed by the driver's escort, which were liable to cause any harm to the Lorries. Also, 12 persons reported decrease in nervous tension which is very commonly found in lorry drivers.[14]

Tofisopam's effect on psychomotor skill and memory was studied by Seppala et al.,1980 in a double blind, cross over design in healthy male volunteers (N=12) and compared with diazepam and alcohol. Subjects were trained in co-ordination test, choice reaction test, Flicker fusion test, Maddox wing, attention test, time anticipation test, and memory and learning tasks. Tofisopam failed to impair performance both as a single dose (100 mg) and after repeated doses (100 + 50 + 50 + 100 mg). The subjects felt more fatigue, dizziness, calmness, and passiveness after diazepam as compared to Tofisopam. Impairment of memory function was not seen with Tofisopam, moreover, it also failed to enhance the ethanol induced impairment. There was no impaired hand eye co-ordination, and the psychomotor side effects are significantly less than diazepam.[15]

As a preanaesthetic medication

Tofisopam was found to be better than Nitrazepam as pre-anaesthetic medication with significant reduction in excitement and apprehension, and in controlling day time sedation. This was concluded in a double-blind, randomized study, involving 47 patients who received Tofisopam 100mg night before and 100mg on the morning of operation; 49 patients received Nitrazepam 5mg and 50 patients received placebo.[16]

As PDE-4 inhibitor

As PDE-4 inhibitor - Isomers of Tofisopam are currently being investigated as PDE-4 inhibitors via modulation of cAMP.[17]

In functional gastrointestinal disease

Osipenko et al, 2000 in a trial recruited (n =59) patients with functional gastrointestinal, emotional and vegetative disorders who failed standard therapy. Tofisopam showed anxiety improvement (Spilberg-Khanin scale) and vegetative dysfunction (Vein scale) reduction in 90% of the test group more significantly than the control group. Intestinal passage in patients with irritable colon syndrome also improved with Tofisopam.[18]

Symptoms of vertigo and tinnitus

30 patients with vertigo, dizziness and/or tinnitus were studied by Kiyota et al.1991. They were given oral Tofisopam 150mg daily (50 mg three times a day), and the severity of their complaints was recorded before and two or four weeks after the start of treatment. Tofisopam was effective in 63.2% of the patients with vertigo and/or dizziness, and in 66.7% of the patients with tinnitus.[19]

Psychic disorders accompanying muscular complaints

The efficacy of Tofisopam in the treatment of psychic disorder accompanying myasthenia gravis has also been studied. 47 patients were treated with Tofisopam two groups (39 patients in myasthenia gravis and 8 patients in myopathy group). Tofisopam

was given in dose of 150 – 300mg/day for 21 days on average. Overall, 89.36 % of patients showed very good and moderately good improvement.[20]

Immunomodulating effects

An in-vitro study by Kalashnikov et al, 2002 has shown that Tofisopam has some immuno-modulating effects. This study used cell cultures of peripheral blood mononuclear cells (PBMC) isolated from heparinized peripheral blood of healthy subjects. The results of this study have shown that Tofisopam increased PBMC proliferation and IL-2 production in low and moderate doses, but both were suppressed in high doses. The results also indicate that same dose of Tofisopam could show different effects on IL-2 production in different individuals. This study also shows that tofisopam in all doses increases dexamethasone-induced suppression of lymphocyte proliferation and decreases TNF-α production. These immuno-modulating effects are attributed to the action of tofisopam on peripheral-type BDZ receptors (PBR) to some extent. These receptors have been found to be present on thymocytes, macrophages, neutrophils, leukocytes and lymphocytes. The results of this study also emphasize on the need of administration of tofisopam in individually selected doses and courses and cautious prescription of this drug in immuno-compromised persons.[21]

Effect on dysrhythmias

In patients of Paroxysmal supraventricular tachycardia (PSVT), Tofisopam administration is associated with improved heart rate variability (HRV) and decreased frequency of PSVT. Tofisopam helps in correction of imbalance of the sympathetic and vagus nervous system in these patients.[22]

Alcohol withdrawal syndrome: In a clinical study,Tofisopam is found to be having same potency with less sedative effect in management of alcohol withdrawl syndrome as compared to benzodiazpines like diazepam .This study consists of two groups of 17 and 12 patients getting Tofisopam and diazepam respectively. The average age of group was 38 years (varies between 25 to 59 years). The patients were examined for severity of withdrawl syndrome after 12 hours,2 days and 4 days after hospital admission.One patient on Tofisopam showed increased irritability and 4 patients on diazepam showed increased sedative effects.[23]

In menopausal symptoms,Gout

Levotofisopam is also under investigation for the treatment of menopausal symptoms and gout.[24]

Dosage and Side-Effects

Usual adult dosage of Tofisopam is 50-300mg daily,frequency of administration is 1-3 times a day.[25]

Several side effects of this drug reported are minor. They include nausea, epigastric pain, itching, rashes. Extreme liveliness, hyperactivity, aggressiveness which usually disappear on dose reduction. Sleep disturbance can be avoided by taking the last dose earlier(before 5:00 pm).[25]

Physical or psychological dependence has not been observed with the use of Tofisopam.[25] In therapeutic doses, it has not been shown to potentiate the effects of alcohol.[16]

Tofisopam is found to be devoid of genotoxic and mutagenic activities in bacterial strains under experimental conditions.[26]

Tofisopam is found to be devoid of teratogenic potential in humans but the amount of information is limited for different Congenital Abnormalities.[27]

Other side effects may be dryness of mouth ,blurring of vision, dizziness, syncope ,excitement and confusion.[8]

Contraindications

A patient with known hypersensitivity to benzodiazepines is not a candidate for therapy with Tofisopam. It is also avoided in pregnancy because of potential harm to the fetus. Psychosis and psychomotor agitation, aggressiveness or severe depression are other contraindications for the use of Tofisopam. Its safety profile has not been clearly evaluated in pediatric patients. It has to be used with caution in case of renal or hepatic impairment.[25]

Conclusions

Tofisopam is a safe and effective anxiolytic drug. However, it is a new drug and needs to be studied in different study populations for longer duration and large sample size specially in Indian patients.

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Forensic Psychiatry

The Mental Health Care Bill : Changes and Appraisal

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"All human beings are born free and equal in dignity and rights"

— *The Universal Declaration of Human Rights*

Introduction

Mental, neurological, and substance use disorders are common in all regions of the world, affecting every community and age group across all income countries. More than 450 million people suffer from mental disorders worldwide[1]. There is a substantial gap between the burden caused by mental disorders and the resources available to prevent and treat them in low income countries. It is estimated that four out of five people with serious mental disorders living in low and middle income countries do not receive mental health services that they need.[2] As per the Mental Health Atlas 2011 by World Health Organization (WHO), only 36% of people living in low income countries are covered by mental health legislation.[2] Outpatient mental health facilities are 58 times more prevalent in high income compared with low income countries. User/consumer organizations are present in 83% of high income countries in comparison to 49% of low income countries.[2] Dedicated mental health legislation can help to legally reinforce the goals of policies and plans in accordance with international human rights and practice standards and bridge this mental health gap.[2]

Violation of human rights in mental health is a growing concern especially in the developing nations[3]. People with mental disabilities experience a wide range of human right violations. Many of them do not have access to the basic human rights such as shelter, food and clothing. They face stigmatization and discrimination in the fields of employment, education and housing. Many are denied the right to vote, marry and have children. As a result of which the sufferers of mental illness, live in poverty and isolation which greatly hampers their chances of procuring treatment, recovering from their illness and integrating into society. The mentally ill are often the most violated and least legally protected groups in a society. A climate that respects and protects basic civil, political, socio-economic and cultural rights is fundamental to mental health promotion. Without the security and freedom provided by these rights, it is very difficult to maintain a high level of mental health. Thus the importance for effective mental health legislation for protecting the rights of people with mental disorders is paramount.

The current paper will endeavour to throw light on the current status of Mental Health Act in India, the ongoing changes proposed in the new Mental Health Care Bill, its merits and demerits and the implications these changes are prognosticated to have on the lives of the mentally ill of the country.

The Rights of the Patients with Mental Illness: The Driving Force behind change in Mental Health Legislation

Right from the inception of mental health legislation, it is the pursuit of protection of the rights of the mentally ill and disabled that has been the impetus for change and amendments of laws regarding the mentally ill. The care for the mentally ill has undergone drastic changes in India since the

days of its inception under the European influence in the 1700s, when asylums and institutions were set up with segregation and institutionalization as the main modalities of treatment. The initial laws regarding the mentally ill, the Indian Lunatic Asylum Act, 1858 and Indian Lunacy Act (ILA), 1912 were primarily concerned with custodial aspects involving the mentally ill. The act was mainly concerned with the segregation of the mentally ill for protection of the society, and implementation of the rights of the mentally ill was grossly neglected. India was a signatory to the "Universal Declaration of Human Rights' adopted by the UN General Assembly in 1948. This set the wheels in motion for change. This led the Indian Psychiatric Society (IPS) to submit a draft Mental Health Bill in 1950. However it was only after a long and protracted course that the Mental Health Act (MHA) was finally enacted in 1987. In light of the global rights emergency in mental health, the UN Convention on the Rights of Persons with Disabilities (UNCRPD) came into force in 2008. The Convention sets out to promote human rights of persons with disabilities including, civil and political rights, the right to live in the community, participation and inclusion, education, health, employment and social protection. The convention marks a paradigm shift in views regarding persons with disability. The new paradigm is based on the presumption of legal capacity, equality, and dignity, and it acknowledges that societal barriers and prejudices are themselves disabling[4]. After India ratified the UNCRPD, revision of legislation relating to persons with disabilities to bring them in consonance with the UNCRPD became obligatory for the government. The process of amendment in of the MHA, 1987 and the PWD Act, 1995 was initiated by the Government of India in 2010. It is in pursuance of this that the Mental Health Act 1987 ("MHA") is sought to be replaced by the new Mental Health Care Bill 2012. The new Mental Health Care Bill has been cleared by the Union Cabinet on June 14, 2013, and once approved by the Parliament, will repeal the Mental Health Act 1987.

The Indian Mental Health Act (1987)

The current Indian law in place dealing with mental ill persons is the Mental Health Act, 1987. The Mental Health Act was drafted by the parliament in 1987 but it came into effect in all the states and union territories of India in April 1993. The act was defined as "An act to consolidate and amend the law relating to the treatment and care of mentally ill persons, to make better provision with respect to their property and affairs and for matters connected therewith or incidental thereto." The Indian Mental Health Act, 1987 is divided into 10 chapters and consists of 98 sections.

The terminologies used in the Indian Lunacy Act, 1912 were changed in the Mental Health Act, 1987 as follows: the term 'psychiatric hospital' replaced 'nursing home asylum', 'mentally ill person' replaced 'lunatic' and 'mentally ill prisoner' replaced the term 'criminal Lunatic'[5].

The main aims of the Mental Health Act are to establish governing bodies at the central and state levels for licensing and supervision of psychiatric hospitals and nursing homes, to establish such institutions and to protect the patients and the society from the dangerous manifestations of psychiatric illnesses. The Mental Health Act made admitting a mentally ill person to a general nursing home an offence. The Mental Health Act also laid down guidelines for the procedure of admission and discharge of mentally ill, to safeguard the rights of the detained individuals, to make sure that no one was detained unnecessarily and to provide for the maintenance of poor mentally ill prisoners undergoing treatment in these hospitals.

The Mental Health Act, 1987 had several merits in comparison to its predecessors. It removed the offensive terminologies used and attempted to somewhat restore the dignity of people suffering from mental illnesses. It made provisions for establishment of licensing authorities for setting up and keeping a check on the working of mental health institutes across the country. Procedures for admission and discharge of psychiatric patients from these institutes were simplified and out-patient care was started which helped in better delivery of the health services to the masses. Provisions were made for bearing of the expenses by the relatives or the government for the treatment and also for the appointment of guardians for maintenance of person and property of the mentally ill[6].

Shortcomings Of Mental Health Act, 1987

The current Mental Health Act has failed to

satisfactorily protect the rights of individuals who it seeks to protect. Incidents of abuse and violation of the mentally ill have been making the headlines sporadically if not often. Instances of illegal detention and institutionalization of women, sub-standard facilities and services at government psychiatric hospitals, unlawful use of reception orders to detain a family member or spouse and ill treatment of the mentally ill across many institutions in the country have been reported[7].

The incident in Erawady when a fire that broke out in an asylum in Erwady village (Tamil Nadu) killed 28 people with mental illnesses who were chained to stone pillars shockingly highlighted the plight of the mentally ill in our country. It is unclear as to whether rehabilitation centres, half way homes etc, especially in the private sector, where persons with mental illness are admitted come under the definition of 'psychiatric hospital' in the Mental Health Act. General hospitals and centres are not included in the definition of 'psychiatric hospital' in the Mental Health Act. The act also does not refer to nonmedical institutions where persons with mental illness are housed. The exclusion of general hospitals and exclusion of any other facilities from the Act does not ensure the rights of the mentally ill in these institutions. It is important that all institutions, medical or nonmedical, government or private, where persons with mental illness are admitted to or reside at, come under the Act.

On the other hand, this restrictive definition of 'psychiatric hospital' lays emphasis on the establishment of new hospitals and institutions which is not practically implementable in a developing country like India. The Mental Health Act's failure to recognise the private sector for provision of mental health services is a major drawback. The rigorous licensing and registration procedures hinder the setting up of services for the mentally ill. In view of the mental health gap in our country, it appears imprudent not to utilise all the available resources at hand. Often the mentally ill are denied admission in general hospitals even when they suffer from physical illnesses. Inclusion of general hospitals and centres would not only bring about less expenditure in establishing treatment facilities, but would also help in achieving comprehensive health care of the mentally ill individuals.

The care and management of mentally ill prisoners also leaves a lot to be desired. Statistics suggest that 1.4% to 1.7% of all patients in psychiatric hospitals in India are mentally ill prisoners[8]. There is a need for separate wards and arrangements for the mentally ill prisoners and the prisoners sent for psychiatric evaluation to psychiatric hospitals. However, in most psychiatric units, no special arrangements exist for the care of mentally ill prisoners. They are admitted in general wards along with other patients, thus raising important issues of safety. Mentally ill prisoners are returned to prisons once they are deemed to be asymptomatic, in order to serve out their sentences. There are no procedures in place to systematically asses and manage the risk of violence that these mentally ill prisoners pose to themselves or others[9]. The follow up care of those prisoners with mental illness also remains sadly lacking.

The Mental Health Act provides for the formation of central and state regulating bodies for supervising psychiatric hospitals and nursing-homes. However it does not state explicitly who would constitute these regulating bodies. As a result, most of these bodies have mainly consisted of bureaucrats and have not included mental health professionals. A mental health professional with knowledge and insight into mental illnesses and the specific needs of the mentally ill is in a better position to supervise the state of hospitals and nursing-homes.

The National Mental Health Program (NMHP) via the District Mental Health Program envisioned a decentralised community based approach to the problem of the mental health gap. It aims at the adequate provision of services to promote early detection and treatment of mental illness in the community itself with both OPD and indoor treatment and appropriate follow up measures. However the mental health care legislation in place, the MHA, with its stringent licensing protocol and focus on legal issues is not in keeping with the goals of the NMHP.

The Mental Health Care Bill: Significant Changes And Implications

Amendments in the existing mental health legislation have given birth to the new Mental Health Care Bill, which is longer than the existing MHA having 16 Chapters and 137 clauses.[10,11] Discussion of all the changes in the new bill would be

cumbersome and voluminous. Outlined below are the significant changes in the new bill and its implications.

- One of the significant contributions of the Mental Health Care Bill is the definition of *"mental illness"* as "a disorder of mood, thought, perception, orientation and memory which causes significant distress to a person or impairs a person's behaviour, judgment and ability to recognize reality or impairs that person's ability to meet the demands of daily life and includes mental conditions associated with the abuse of alcohol and drugs, but does not include mental retardation". The definition of 'mental illness' is a major improvement from the one given by the Mental Health act which defined 'a mentally ill person' merely as a person who is in need of treatment by reason of any mental disorder other than mental retardation'. The bill also lays down criteria for determination of mental illness.
- The Bill introduces a provision known as *"Advance Directive"* addressing the controversial area of the right to consent of a mentally ill person. The 2012 Bill provides every person with a right to make an advance directive, empowering him/her to decide how he/she should and should not be cared and treated for a mental illness in the future. This ensures every person's right and capacity to decide what treatment or care he/she must be subjected to. The Bill also lays down an elaborate procedure for registration and revocation of advance directives as well as situations in which such directives can be overridden. The concerned person is also empowered to "nominate" an individual, who will take decisions for him or her in the advent of any mental illness.
- Under the proposed new law, there is provision for involuntary admissions known as *"supported admissions"* in specific circumstances when the mentally ill person is in imminent danger of causing harm to herself or to others, for which appeals can be made to the Mental Health Review Commission. These involuntary admissions can only be for thirty days at a time.
- It is provided in the Bill that any person, who is not a minor, and who considers himself/herself to have a mental illness can get himself/herself admitted and be discharged on request. However, a mental health professional may prevent discharge when the patient is at risk of causing bodily harm to self or others. This authority vested on the treating psychiatrist needs to be used scrupulously to prevent potential abuse.
- The Bill also states that in certain specified emergency situations, treatment can be initiated by any registered medical practitioner with the consent of nominated representative, at any health establishment or in the community. But the treatment under this section will be limited to 72 hours, and ECT and medical treatment not directly related to the emergency shall not be provided under this section. This provision would be greatly beneficial to those with mental illness as it ensures access to emergency treatment from their nearest service provider.
- The Bill states that person with mental illness shall be treated equal to physical illness, and stipulates that public and private insurance providers "shall make provisions for medical insurance for treatment of mental illness on the same basis as is available for treatment of physical illness". If implemented this can usher in great benefits for persons suffering from mental illness.
- The Bill brings about a rights-based protection of mentally ill persons. The Bill caters to the requirements of the UNCRPD by providing all persons the right to access mental health care. It also provides a range of services for persons with mental illness including shelter homes, supported accommodation, community based rehabilitation; the right to community living, the right to live with dignity, protection against cruel, degrading and inhuman treatment, the right to equality and non-discrimination, the right to information, confidentiality and access to medical records; right to personal

communication, legal aid and the right to make complaints about deficiencies in provision of services in addition to other similar legal remedies.

- It is also important to note that in this new Bill, psychiatric patients will have the right to seek complete information about their treatment, including all records, even in case of involuntary admissions. If the treating psychiatrist feels the patient is not fit at the time of admission or during treatment to take this information, it is the primary responsibility of the medical officer or psychiatrist in charge of the person's care to ensure that the full information is provided promptly when the individual is in a position to receive it.
- The responsibility of planning, designing, implementing programs for promotion of mental health and prevention of mental illness, creating awareness about mental illnesses, reducing stigma, sensitizing government officials including police officers, implementing public health programs to reduce suicide and other such programs has been placed on the appropriate government. Insufficient awareness, advocacy and sensitization about mental illness are serious issues that hamper a mentally ill person's rehabilitation and it is encouraging to see these issues being addressed in this bill.
- The Bill seeks to create various new bodies and completely overhaul the existing mental healthcare system in the country. It provides for the establishment of the Central and State Mental Health Authori-ties, which would be responsible for the registration and oversight of mental health establishments by laying down minimum standards and a monitoring mechanism to ensure statutory compliance. The Bill also sets up the Mental Health Review Commi-ssion (MHRC) and state-wise Mental Health Review Boards (MHRB). The MHRC and MHRB are equipped with several administrative and adjudicatory functions and will form the first level of interaction of any person with mental illness or his/her representative with the mental healthcare system for violation of any of his or her rights. This is in comparison to the MHA which gave an unbridled power to the Magistrate in the mental health system. With the introduction of these new bodies, a person with mental illness can directly approach a forum for protection of his/her rights.
- The bill states that proof of a person's current or past admission to a mental health establishment or proof of a person's current or past treatment for mental illness shall not by itself be ground for granting divorce, adding that if during judicial proceedings, any current proof of mental illness is raised, the matter will have to be referred to the State Mental Health Review Commission for further scrutiny.
- The bill bans electric-convulsive therapy without anaesthesia (direct electric-convulsive therapy) and restricts psychosurgery. This would mean that centres in which direct electro convulsive therapy would have to stop or delay the delivery of electro-convulsive therapy until the facilities for delivering electroconvulsive therapy with anaesthesia are in place.
- A welcome provision in the Bill is the requirement that medical research on mentally ill persons can be done only with their free and informed consent (after provision of adequate information about the risks, benefits, alternatives in understandable language) or with the consent of the State authority, where the person is not capable of giving consent.
- Another important change that new Bill brings forth is that it decriminalises suicide by stating that a person who has attempted suicide shall be examined by a psychiatrist before a criminal investigation is initiated. If the doctor concludes that the act was prompted by mental illness, then "no complaint, investigation or prosecution shall be entertained against the person who attempted to commit suicide, notwithstanding anything contained in the Indian Penal Code."

Critical Appraisal of the Mental Health Care Bill, 2013

The Mental Health Care Bill in spite of its progressive nature and improvements over the MHA does have its fair share of criticisms.

- The Bill seeks to completely change the existing mental health care system in India. It provides for the creation of various new bodies such as the Mental Health Review Commissions and Central and State Mental Health Care Authorities. The bill thus places a huge infrastructural, economic and human resource burden on State and Central Governments. The implementation of these ambitious changes a comprehen-sive plan of action. A complementary National Mental Health Policy to convert the goals of this piece of legislation into workable measures is the only way the changes in the Bill can be implemented.
- In a country like India, where a majority of patients are from a rural background, a paternalistic doctor-patient relationship still prevails. There are also multiple logistic difficulties in the implementation of mental health care given the scarcity of mental health professionals. Giving sole liberty to the patient to choose the treatment modality and decision for admission and refusal to a particular modality of treatment are steps to ensure patient rights. However, taking into consideration the lack of insight and judgement that characterise certain psychotic illnesses, many of these patients often refuse admission or treatment when acutely ill and until they regain their insight into the illness. Thus, the new Bill will possibly increase the burden on the caregiver and the mental health professional when trying to ensure that those with mental illness are properly treated.
- Stringent restrictions on essential modalities of treatment like ECT may further increase the stigma associated with this often lifesaving treatment. Further-more, most of the general hospital psychia-tric units are still running without proper anaesthesiology back up and the future use of ECT will be definitely restricted due to legal as well as resource constraints.
- The provisions in the Bill relating to the rights to community care and independent living do not appear adequate. The bill states that the Government shall provide community-based living such as half-way homes, group homes. However details as to how Governments should go about providing these services, budgets that need to be allocated and time frames within which these changes should be imple-mented are lacking.
- It took nearly 45 years to bring the mental health act in to force after its inception and it is still not implemented in the country to its full strength till now despite attempts to do so following several critical appraisals. The changes in the current mental health legislation are in accordance with the UNCRPD. However, merely framing a mental health law in the light of these lofty concepts is unlikely to do much to meet the needs of mentally ill in a resource poor country like India. The commitment of the government to develop adequate infra-structure and provide mental health resources in accordance with the new laws will be paramount in the inception of the changes that the mental health bill proposes to bring about. Otherwise, the Mental Health Care Bill will remain a piece of legislation only on paper for long[11].

Conclusion

The Mental Health Care Bill 2013 makes significant strides over the MHA bringing about protection and empowerment of persons with mental illness. It is essential that the changes in the Mental Health Act and the National Mental Health Programme should go hand in hand with each other in order to ensure optimum provision of the health services to those who need it.

Humane laws, better induction of resources, emphasis on ethics and respect for human rights are the need of the hour, and these are the issues which the current mental health care bill attempts to address. This is indeed a welcome change. However it is essential for further discussion and

debates to overcome perceived shortcomings before the final bill is put in place. Effective implementation will require a complete change in the system currently in place and will involve substantial inputs of manpower and finances. These possible hurdles need to be crossed via proper administration and planning.

Acknowledgement – We heartily acknowledge the initiative, encouragement and guidance of Late Prof. J.K. Trivedi, M.D., F.R.C. Psych (U.K.) F.A.P.A. (U.S.A.), F.A.M.S. (India), Department of Psychiatry, K.G. Medical University, Lucknow, India.

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Forensic Psychiatry

The Narcotic Drugs and Psychotropic Substances (Amendment) Bill, 2011 Bill No. 78 of 2011

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Introduction

The Narcotic Drugs and Psychotropic Substances Act, 1985 (Act 61 of 1985) was enacted consolidating and amending the provisions for the control and regulation of operations relating to narcotic drugs and psychotropic substances under the Opium Act, 1857, Opium Act, 1878 and the Dangerous Drugs Act, 1930[1]. This Act regulates[2]:

1. The procedure for preparation and approval of schedules of narcotic drugs and psychotropic substances.
2. The procedure for handling narcotic drugs and psychotropic substances and precursors thereof.
3. The procedure for inspection and identification of narcotic drugs, psychotropic substances and precursors, the procedure for issue of permits required for the handling of narcotic drugs, psychotropic substances and precursors, and supervision over the implementation of such procedure.
4. The procedure regarding information and reporting on narcotic drugs, psychotropic substances and Precursors.
5. The procedure for prevention of the spread of drug addiction, and treatment and rehabilitation of drugaddicts.

This Act was amended once in 1989 and subsequently in 2001[1]. However during the implementation of this act certain shortcomings were noticed like[1]:

1. Confusion related to the quantities of seized drugs and differentiating between drug traffickers who traffic in significant quantities and addicts who commit less serious offences. Earlier only the pure drug content was being taken cognizance of but the need was felt to take the entire quantity of the drug seized in a case for determining the quantum of punishment.
2. The need was felt to expand the scope of provisions for tracing and seizing of illegally acquired properties pursuant to drug trafficking activity, so that it becomes more difficult for drug traffickers to enjoy the fruits of drug trafficking activity.
3. The need was felt to introduce an alternate method of obtaining alkaloids of opium through production of concentrate of poppy straw instead of production and processing of opium, in the country.

With these shortcomings, a need was felt for further amendments. The Narcotic Drugs and Psychotropic Substances (Amendment) Bill, 2011 was introduced in the Lok Sabha on September 8, 2011 by the Minister of Finance, Shri Pranab Mukherjee. The Bill was referred to the Standing Committee on Finance (Chairperson: Shri Yashwant Sinha) on September 13, 2011. The committee then submitted its report on 21st March 2012. The bill was subsequently passed by Lok Sabha on 20th February 2014 and by Rajya Sabha on 21st February 2014[3]. The main amendments that have been done in this bill are shown in the following table[4]:

Original Provisions of NDPS Act 1985	Amendments done in the current bill
Insertion of clause (4a) in Section 2 clause (4)	(4a) "Central Government factories" means factories owned by the Central Government or factories owned by any company in which the Central Government holds at least fifty-one per cent. of the paid-up share capital
Substitution of clause (7a) In Section 2 dealing with definition of '**commercial quantity**'	(7a) "commercial quantity", in relation to a narcotic drug, psychotropic substance or any preparation of such drug or such substance, means any quantity of such drug, substance or preparation of such drug or substance greater than the quantity specified, in terms of the pure drug content or otherwise, by the Central Government by notification in the Official Gazette
Substitution of clause (13a) In Section 2 dealing with of '**small quantity**'	(13a) "small quantity", in relation to a narcotic drug,definition psychotropic substance or any preparation of such drug or such substance, means any quantity of such drug, substance or preparation of such drug or substance lesser than the quantity specified, in terms of the pure drug content or otherwise, by the Central Government by notification in the Official Gazette
Insertion of sub-clause (iiia) after sub-clause (iii) In section 9 of the original bill **(dealing with power of central government to permit, control and regulate)**	(iiia) the possession, transport, import inter-State, export inter-State, warehousing, sale, purchase, consumption and use of poppy straw produced from plants from which no juice has been extracted through lancing
Insertion of following words after the words 'poppy straw' In section 10 of the original bill, in sub-section (1), in clause (a), in sub-clause (i)	"except poppy straw produced from plants from which no juice has been extracted through lancing"
Substitution of section 27**(dealing with punishment for consumption of any narcotic drug or psychotropic substance)**	Whoever consumes any narcotic drug or psychotropic substance in contravention of any provision of this Act or any rule or order made there under shall be punishable with imprisonment for a term which may extend to six months or with fine which may extend to ten thousand rupees or with both
Insertion of new section 27 B After section 27 A **(dealing with punishment for consumption of any narcotic drug or psychotropic substance)**	"27B. Whoever contravenes the provision of section 8A shall be punishable with rigorous imprisonment for a term which shall not be less than three years butwhich may extend to ten years and shall also be liable to fine."
Amendments in Section 31 **(enhanced punishment for offences after previous conviction)**	• In Sub-section 1 for the words ""**one-half of the maximum term**" and "**one-half of the maximum amount**", following words shall be respectively substituted "**one and one half times of the maximum term**" and "**one and one half times of the maximum amount**" • In Sub-section 2 for the words ""**one-half of the minimum term**" and "**one-half of the minimum amount**", following words shall be respectively substituted "**one and one half times of the minimum term**" and "**one and one half times of the minimum amount**"
Amendments in section 52 A**(Disposal of seized narcotic drugs and psychotropic substances)**	• Sub-section 1 shall be substituted by the following sub-section, namely- o The Central Government may, having regard to the hazardous nature,vulnerability to theft, substitution, constraint of proper storage space or any other relevant consideration, in respect of any narcotic drugs, psychotropic substances, controlled substances or conveyances, by notification in the Official Gazette, specify such narcotic drugs, psychotropic substances, controlled substances or conveyance or class of

	narcotic drugs, class of psychotropic substances, class of controlled substances or conveyances, which shall, as soon as may be after their seizure, be disposed of by such officer and in such manner as that Government may, from time to time, determine after following the procedure hereinafter specified." • In sub-section 2 o for the words "narcotic drug or psychotropic substance" and "narcotic drugs or psychotropic substances", wherever they occur, the words "narcotic drugs, psychotropic substances, controlled substances or conveyances" shall be substituted o in clause (b), for the words "such drugs or substances", the words "such drugs, substances or conveyances" shall be substituted • In sub-section 4 o for the words "narcotic drug or psychotropic substance" and "narcotic drugs or psychotropic substances", the words "narcotic drugs, psychotropic substances, controlled substances or conveyances" shall be substituted
Insertion of new section 57 A after section 57	"57A. Whenever any officer notified under section 53 makes an arrest or seizureunder this Act, and the provisions of Chapter VA apply to any person involved in thecase of such arrest or seizure, the officer shall make a report of the illegally acquired properties of such person to the jurisdictional competent authority within one hundredand eighty days of the arrest or seizure."
Change in Heading of Chapter VA	For the heading "FORFEITURE OF PROPERTY DERIVED FROM, OR USED IN ILLICIT TRAFFIC", the heading "FORFEITURE OF ILLEGALLY ACQUIRED PROPERTY" shall be substituted
Amendment of Section 68 B **(Definitions)**	• In clause (g), sub-clause (i) o for the words "of this Act; or", the words "of this Act or the equivalent value of such property; or" shall be substituted • In clause (g), sub-clause (ii) o For the words "such property,", the words "such property or the equivalent value of such property; or" shall be substituted • Insertion of sub-clause (iii) after sub-clause (ii) o any property acquired by such person, whether before or after the commencement of the Narcotic Drugs and Psychotropic Substances (Amendment) Act, 2011, wholly or partly out of or by means of any income, earnings or assets the source of which cannot be proved, or the equivalent value of such property" Substitution of original clause (h) by new clause (h) o "property" means any property or assets of every description, whether corporeal or incorporeal, movable or immovable, tangible or intangible, wherever located and includes deeds and instruments evidencing title to, or interest in, such property or assets
Amendment of section 68 D **(Competent authorities)**	in sub-section (1) o for the words "any Collector of Customs or Collector of Central Excise", the words "any Commissioner of Customs or Commissioner of Central Excise" shall be substituted

Amendment of section 68 H	**Explanation** inserted at the end o For the removal of doubts, it is hereby declared that in a case where the provisions of section 68J are applicable, no notice under this section shall be invalid merely on the ground that it fails to mention the evidence relied upon or it fails to establish a direct nexus between the property sought to be forfeited and any activity in contravention of the provisions of this Act
Amendment of section 68 O **(dealing with appeals)**	Adding the following provisio in sub-section (4) o "Provided further that if the office of the Chairman is vacant by reason of his death, resignation or otherwise, or if the Chairman is unable to discharge his duties owing to absence, illness or any other cause, the Central Government may, by order, nominate any member to act as the Chairman until a new Chairman is appointed and assumes charge or, as the case may be, resumes his duties
Amendment of section 71(**Power of Government to establish centers for identification, treatment, etc., of addicts and for supply of narcotic drugs and psychotropicsubstances**)	Insertion of word 'management' after the words 'identification, treatment' in sub-section (1)

Thus the main features of the new bill are:

1. Addition of new definitions like "central government factories', amending definitions like 'commercial quantity' and 'small quantity'.
2. Broadening the definition of 'illegally acquired property' to include not just property derived from income out of an illegal act under this law but also the equivalent value of such property. It also includes any property acquired out of earnings whose source cannot be proved.
3. It Stipulates that any person who consumes drugs in contravention of this Act shall be penalized with imprisonment for upto six months or a fine uptoRs 10,000 or both.
4. The Act prohibits any person from possessing or transferring any property or concealing the nature and location of property derived from an offence under the Act or any other corresponding law in another country. The Bill provides for penalizing such an offence with rigorous imprisonment between 3 to 10 years and a fine
5. The Bill enhances the penalty for offences committed after previous convictions to one and one-half times the term he can be convicted for in terms of time and amount of fine.
6. Transfer of power to regulate "poppy straw concentrate" from the State to the Central Government.
7. The Bill states that drugs, psychotropic substances or controlled substances shall be disposed off in the manner to be prescribed.
8. As per the bill whenever an officer (notified either by the central or state government) makes an arrest or seizes goods under this Act, he shall report to the jurisdictional competent authority within 180 days of the arrest or seizure.
9. The competent authorities under this Act include the Collector of Customs or Central Excise. The Bill changes this to Commissioner of Customs or Central Excise.
10. The Act provides for an Appellate Tribunal headed by a Chairman. The Bill adds that in case the Chairman's position is vacant due to absence, resignation or death, the central government can nominate any member to act as the Chairman till he resumes his duties or a new Chairman is appointed.

Positive Aspects of the Bill

1. The Bill is an attempt to overcome the shortcomings of the original NDPS Bill. Definitions have been modified and

explanations and clarifications have been introduced to make the bill as clear and as unambiguous as possible.

2. Restrictions on the use of narcotic drugs like morphine for medicinal purposes, especially for pain management, are set to be eased[5].
3. The licensing power has been transferred to a single authority, the state food and drug administration, and the power to regulate and frame rules will be vested with the Drug Controller General of India. Thus procedural delays are likely to be reduced[5].
4. An acknowledgement of the need for treatment and management of patients dependent on substances of abuse by laying emphasis on the same in the bill.

Shortcomings and Concerns over the Bill

1. It is being said that the definitions of 'commercial quantity' and 'small quantity' continue to be vague even in the amendment. It is being recommended that these terms should be clearly spelt out[6].
2. Concerns are being raised over the reduction in punishment for consumption of morphine, cocaine and heroine. The parliamentary committee recommended that t prescribing deterrent punishment is important and the punishment for the consumption of morphine, cocaine, and heroine should not be reduced. Instead, the punishment for trafficking in small quantities should be increased to one year[6].
3. The parliamentary committee recommended that the seizure of property of the person arrested by the notified officer should be made within 90 days of the arrest instead of 180 days as in the current bill. Concerns were raised that 180 days is a long period and would allow manipulation/ distortion of facts about the seized property in question[6].
4. It was recommended that by the stakeholders that mandatory death penalty as provisioned in the bill in crimes involving large quantity of drugs be changed to 'may be punishable with death'[6].
5. Several stakeholders have suggested that the Act should be amended to enable compounding of unintended or procedural lapses and to address undue harassment of legally authorized manufacturers, dealers, and traders during investigations.

Conclusion

To conclude, it can be said that there is a need for a comprehensive and broad based legislation for control over the narcotic and psychotropic substances. While the amendment bill has ade some important changes over the original NDPS Bill, important shortcomings still remain. A lot needs to be done before this bill truly addresses the changing needs of the nation in this important area.

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Case Report

Managing Trichotillomania with Trichobezoar

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Introduction

Trichotillomania derives its meaning from the Greek: trich-(hair), till (en) (to pull), and mania ("an abnormal love for a specific object, place, or action")[1] and is classified as an impulse control disorder by DSM-IV. It is described as a symptom complex of compulsive urge to pull out one's own hair leading to noticeable hair loss, increasing sense of tension immediately preceding or when resisting hair pulling, and pleasure, gratification or relief when pulling out the hair. These symptoms of trichotillomania must cause clinically significant distress or impaired social or occupational functioning. Though the prevalence is reported to be less than 1 percent, it is often chronic and difficult to treat. The peak age of onset is usually 9 to 13 years and it may be triggered by stressful life events.[2] The prevalence of trichotillomania is reported to be far more in females than males. The most common site of hair pulling is the scalp, although eyelashes, eyebrows, the pubic region, or other parts of the body may also be involved.[3]

Trichotillomania is often complicated by trichobezoar, resulting from trichophagia that consists of ingesting the pulled hairs. It may present as gastrointestinal tract obstruction with nausea and vomiting, nutritional deficiencies like hypochromic anemia, vitamin B_{12} deficiency, weight loss, an abdominal mass or pain in abdomen, ulcerations and perforation.[3-6] No formal algorithm for management of trichotillomania has been formulated. Associated medical complications, misconceptions and ignorance about the disorder make treatment more difficult. Further, lack of co-ordination and clarity of management options between different treating departments delay the clinical care pathway for the patient resulting in associated complications. We are reporting a case of trichobezoar in a 9 year old female child with trichotillomania who was referred from the dermatology department, the challenges and difficulties encountered in the management.

Case Report

Patient A, a 9 year old unmarried Hindu girl, resident of a nuclear family of middle socio-economic status presented with complaints of 2 years duration characterized by compulsive pulling of hair from front and center of scalp and ingesting them. She initially consulted a pediatrician after 6 months who advised treatment from a dermatologist and the child was diagnosed with alopecia areata due to presence of isolated patchy hair loss and concealment of history by mother due to attached stigma. There was no improvement in symptoms over the next 6 months on treatment. Due to complain of severe pain abdomen, she was referred to a surgeon where detailed investigations revealed trichobezoar and the child had to be operated (Image 1). After recovery, she was referred to the psychiatry department, was diagnosed with trichotillomania and started on Cap. Fluoxetine 20 mg (Selective Serotonin Reuptake Inhibitor) which was raised to 40 mg after 1 month of inadequate response. An aggressive treatment plan comprising of pharma-cological and psychotherapeutic interventions was initiated. Psycho-education of family regarding the illness was started to improve compliance and build trust in

Image 1 – Post operative abdominal scar

treatment. Antipsychotic augmentation (Risperidone 2 mg) of fluoxetine was initiated after 12 wks. of treatment with inadequate response on The Massachusetts General Hospital (MGH) Hairpulling Scale.[7] Psychotherapeutic approaches (social skill training, supportive psychotherapy, 12 sessions each of art therapy and play therapy) were also instituted alongside. The child was successfully managed and was followed up for a period of 1 year. No recurrence occurred. The challenges encountered in the management were frequent breaks in compliance and follow up due to various psychosocial stressors (lack of social support, father's death, maladjustment with stepsister from mother's previous marriage, financial constraints), ignorance about psychiatric illnesses, rigid socio-cultural beliefs and intermittent consultation and treatment with skin and child specialists and faith healers.

Discussion

Hair-pulling behaviors lie along a continuum. They may range from a relatively benign form that produces no significant cosmetic, physical and emotional distress, to a more serious form that is often disfiguring and leads to great personal suffering. In severe form, it can lead to formation of trichobezoar resulting from the ingestion of pulled hairs. The prevalence of trichotillomania is probably underestimated because of the secretiveness due to attached stigma as well as under-recognition by medical professionals. The incidence of trichobezoar in trichotillomania is unclear, although attempts have been made. Christenson and Mansueto[8] did not report any case of trichobezoar in their series of 186 patients with trichotillomania. Bhatia et al.[9] reported presence of bezoars in 37.5% of 24 young patients with trichotillomania (25% trichobezoar, 12.5% tricho-phytobezoar). Given that endoscopic removal of trichobezoar is possible at times, an early diagnosis is advantageous. However, this does not usually happens as the associated medical complications, misconceptions and ignorance about the disorder make diagnosis and treatment more difficult. Further, lack of co-ordination and clarity of management options between different treating departments delay the clinical care pathway for the patient resulting in associated complications.

Given the limited available clinical research evidence, no formal treatment algorithm for trichotillomania can be formulated, and it is important to make patients aware of this; the patient's choice is likely to be paramount in treatment selection. Pharmacotherapy in form of SSRIs (Selective Serotonin Reuptake Inhibitor) or clomipramine is effective and should be initiated[10] Dual treatment, using pharmacotherapy and psychotherapy (Habit Reversal Training) is more effective than monotherapy, at least in patients who do not respond to an SSRI in the first instance.[11] Other types of behavior therapy like art therapy, play therapy, social skills training are also effective in management of trichotillomania and should be initiated along with drug treatment. Various psychosocial factors which precipitate and perpetuate the pathology of trichotillomania should be taken into consideration, addressed adequately and dealt with through psycho-education and public awareness campaigns.

In summary, the importance and severity of the medical complications of trichotillomania should not be underestimated. These can be life threatening but are preventable if the disorder is diagnosed early in its course. Awareness of the disorder along with greater inter-departmental collaboration would help the cause. Although no guidelines on treatment of trichotillomania are formulated, pharmacotherapy as well as psychotherapy should be instituted early in the course.

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Case Report

Effectiveness of the EMDR Therapy on Specific Phobia in Young Children

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Introduction

Eye Movement Desensitization and Reprocessing (EMDR) (Shapiro, 2001) is a comprehensive, integrative psychotherapeutic approach. It contains elements of a number of effective psychotherapies in structured protocols that are designed to maximize treatment effects. These include psychodynamic, cognitive behavioural, interpersonal, experiential, and body-centred therapies (Shapiro, 2002). EMDR psychotherapy is an information processing therapy and uses an eight phase approach to address the experiential contributors of a wide range of pathologies. It attends to the past experiences that have set the groundwork for pathology, the current situations that trigger dysfunctional emotions, beliefs and sensations, and the positive experience needed to enhance future adaptive behaviours and mental health. During treatment, various procedures and protocols are used to address the entire clinical picture. One of the procedural elements is "maintain dual awareness using bilateral stimulation" using bilateral eye movements, tones or taps. During the reprocessing phases the client attends momentarily to past memories, present triggers, or anticipated future experiences while simultaneously focusing on a set of external stimulus. During that time, clients generally experience the emergence of insight, changes in memories, or new associations. The clinician assists the client to focus on appropriate material before initiation of each subsequent set. In our experience EMDR has proved helpful in many childhood psychological and psychiatric disorders (Gupta & Choudhary, 2012, 2013). When applied to children, EMDR can often be used in adjunction with some innovative Cognitive Based therapeutic techniques to elicit better results (Rachamim et al, 2009).

Aim

A case conceptualization highlights sudden onset specific phobia and its treatment through EMDR in children.

Case Conceptualization

A 9 year old, school going boy from Agra, without any preceding history of neurological or psychological disorder experienced a sudden onset of specific phobia in October 2013. Four days prior to coming in for consultation, he developed a severe fear towards a song that was sung by a peer on stage at school. On hearing the song, his instant reaction was breaking into tears and remembering his mother. After that incident, he continued to miss her even though she would be around him at all times. He also refused to go to school for the next few days till his parents decided to bring him to the clinic for consultation.

Session 1

Phase 1: Patient history and treatment planning

The patient was a young, well turned out boy and did not look very apprehensive about being in the clinic. Since he was young, the case history was given by the parents in his presence. He seemed comfortable and safe around his parents. The patient had an elder sister, who was 20 years of age, but wasn't present during the session.

The case history revealed that, at the age of 8 months, the patient's mother injured herself and had to stay away from him for a period of 3 to 4 months where she could not play with him or spend much time with him. According to his mother, his childhood fears mainly related to ghosts and the whistle of the pressure cooker. Academically, he did well in school and his parents never received any complaints regarding his behaviour.

On asking the child why his parents have brought him to the clinic, he replied by saying that he was scared ever since he heard 'the song' and because he does not want to go to school. He was further asked what parts in the song disturbed him the most. The patient appeared to be afraid of specific stanza's in the song and those lines made him remember his mother even more. Further, the child was asked whether he wanted this fear to go away and he answered by saying; "I want the song to get out of my head."

Phase 2: Preparation Phase

Being an outstation patient, the child and his family were only in the city for two days. Therefore Phase 2 of the EMDR protocol was conducted on the same day. The Preparation Phase began by installing the 'safe place' as a protocol of the EMDR therapy. A safe place or a calm place refers to any experience, place, situation that relieves the patient. This place along with its specific associated features (sounds, smell, and visual imagery) is imagined by the client and strengthened using bilateral stimulation. It can be used any time during the session where the therapist feels it is required to calm the client down. Before beginning the same, the patients' father was asked to be seated outside and only his mother along with the patient continued to be in the room. The child was asked what he liked doing the most that gave him happiness and joy. He answered by saying that he enjoyed playing cricket and he feels safest in his house. Therefore, he chose his 'home' as the calm/ safe place. Utilizing the language uniqueand comfortableto the child, the mechanics for Bilateral Stimulation (BLS) was practiced. The child preferred tapping to eye movement. He was asked to think about his calm/safe place and say the word 'home' aloud. On asking him how he felt, the child replied by saying that he felt good and comfortable.

Thereafter, the container exercise was introduced to the patient. Containers are used to assist children to close incomplete sessions and as a source for affect management, especially between sessions. The patient was asked to imagine a box of his preferred shape, dimensions, material and colour. At each step he was asked to describe the box and was told the use of the box.

Phase 3 and 4: Assessment & Desensitization Phase

Since there weren't adequate days with the patient for therapy, phase 3 was incorporated in the same session as well. The target memory was chosen as the song since that was the clients triggering traumatic event. Subjective Unit of Disturbance Scale (SUDS) was rated as 9 on a scale of 0 to 10. On identifying where he felt maximum discomfort in his body, the patient replied by saying that he felt uneasy in his chest. On being asked to think about the song along with Bilateral Stimulation (BLS), which in his case was tapping, the child felt extremely 'scared'. After a few more sets of BLS, the particular song was played aloud, to which he immediately reacted with tears and more anxiety. The song was stopped thereafter and the child was asked to think about his calm/safe place (his 'home'). Sets of BLS continued and holding onto the feeling that absorbed the client when he thought of the song. Eye movement stimulation replaced tapping after seeking his consent if he was comfortable with the change. The reason for the shift from tapping to eye movement was because he appeared to be dissociating. He was also asked to open his eyes during the stimulation to sustain his attention which was getting diverted.

The child reported positive feelings as sets of BLS went on. He continued to say that he was feeling good. His positivity was further reiterated by telling him that he has control over his mind and which thoughts enter it. This sense of power that was induced into him strengthened more as the therapy went on. EMDR with children has to be innovatively changed according to each child and the situation, specifically in cases of phobias (Jongh.et.al, 1999). On that note, the child was asked to write the line that disturbs him most in the song, on the whiteboard. As BLS took place, he gradually kept erasing the words on the whiteboard. He felt

better each time he did so. Finally he was asked to report SUD once again. This time it went down from 9 to 1 on the scale. After a few more sets of BLS, the child was asked if he could sing the song aloud. He expressed distress and did not want to sing it. After restoring the calm/safe place to stabilize the child, BLS continued along with assuring the child that the he himself has left the song back in the clinic by erasing it on the board and therefore will not take it along with him. Thereafter, the patient spoke out the lyrics of the song that disturbed him most.

Before ending the session, the container exercise was conducted by asking the child to imagine that he locked in all of his thoughts and fears in the box (that he had earlier created) and left the box in the clinic itself.

The session lasted about an hour and ended on a positive response from the child which was that he would resume going to school from the coming Monday. The patients' mother who was seated in the room all along and his father who was called in at the end were both asked to make the patient enjoy the rest of the day in the city and not bring up anything that related to the fear. As a part of the day's home task they were told to behave as normally as any other usual day with the patient.

Session 2

The successive session was held on the next day itself as the family was departing for Agra in the evening. In order to slowly prepare the child for reprocessing, the session began with the child singing his favourite song, after which he was asked to shut his eyes and think about the song he sang. He reported SUD of 0 out of 10. Thereafter, the he was asked if he could sing the song he feared. After initial hesitation, he sang the first line thrice. This was being recorded so that he could be shown the recordings later. After a few rounds of BLS, he was made to hear his own recording. He noticed that he was not getting anxious when he heard himself.As sets of BLS continued, the child felt better and reported a SUD of 0. Although he wasn't ready to hear the original track at that moment, he did say that he will go to school from Monday and that he felt better. The metaphor about the remote control and television were used in the patients' case where he was told that his thoughts were in his hands just like the remote control of the television is in the hands of the user. When a channel gets disinteresting the user changes it by using the remote control. Just the same way, the patient has the power to control his thoughts and fears.

The 45 minute long session ended on the above positive note with the container exercise as closure, where he was asked to take the empty imaginary box back to his house in Agra. His home task entailed that he would put all his fears and worries in the box and locks it as and when he felt the need to. At the same time, the patient should also empty the box when his fears and thoughts would go away in order to make room for new thoughts.

The child's parents were emailed the recordings of the song so that they could show him the video to slowly take away the little fear that remained.The sessions were recorded because it was observed that the child responded well to visual assurance (Blackboard erasing of words) and as he was from Agra and would not be able to come in for a follow up. According to the telephonic conversation with the parents of the child, he resumed school from Monday and has been going regularly along with no reports of fear ever since.

Conclusion

The above case substantiates research that now recommends EMDR as a treatment for specific phobias and problems other than PTSD (Marquis, 1991; Shapiro, 1995; Renssen, 1999). The three pronged approach was followed in the case study as well and by the end of the second session, the child was slowly getting desensitized to the stimuli (song) and also getting prepared for future confrontation. However, the breakthrough occurred when the child himself, was ready to go to school from the next day. Ost (1991), conducted research that highlighted how a single treatment session of 2.5 hours is successful in about 90% patients with specific phobias. EMDR works extremely well on children and a Meta-analysis by Rodenburg.et.al, (2009) reiterates the same. There are many advantages of using EMDR therapy with children, adolescents and young people who might be suffering from traumatic experience and phobias.The effectiveness of EMDR can be ascertained after a few sessions itself (rapid onset of improvement).Also, the EMDR is quite a simple

task for the child. Since the changes occur automatically, and in a non volitional manner, it makes it comparatively easier to use with children (child friendly), as compared to other therapies.But at the same time using EMDR with children requires constant innovation and creativity in technique that suits the child and situation well.

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Case Report

A Case of Tuberous Sclerosis with Normal Intelligence: An unusual presentation

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Introduction

Tuberous sclerosis (TS), the name is composed of the Latin word "tuber" (swelling) and the Greek word "skleros" (hard), refers to the pathological finding of thick, firm and pale gyri, called "tubers," in the brains of patients on autopsy. These tubers were first described by Désiré-Magloire Bourneville in 1880.[1]

TS is a rare multi-system autosomal inherited disorder that causes non-malignant tumors to grow in the brain and on other vital organs such as the kidneys, heart, eyes, lungs, and skin. TS is caused by a mutation of either of two genes, TSC1 and TSC2, which code for the proteins hamartin and tuberin respectively. These proteins act in a complex as growth suppressors by inhibiting the activation of a master, evolutionarily conserved kinase called mTOR. Loss of regulation of mTOR occurs in cells lacking either hamartin or tuberin, and this leads to abnormal differentiation and development, and to the generation of enlarged cells.[2]

TS have an approximate incidence of one in ten thousand to fifty thousand. The clinical triad of papular facial nevus, seizures and mental retardation is found in less than half of the patient. Thus the radiological hallmarks of this neurocutaneous syndrome are universally accepted as sufficient for diagnosis.[3]

The prognosis for individuals with TS is highly variable and depends on the severity of symptoms. Those individuals with mild symptoms usually do well and have a normal life expectancy. Individuals who are severely affected can suffer from severe mental retardation and persistent epilepsy.[2]

The physical manifestations of tuberous sclerosis are due to the formation of hamartia (malformed tissue such as the cortical tubers), hamartomas (benign growths such as facial angiofibroma and subependymal nodules) and, very rarely, cancerous hamartoblastomas. The effect of these on the brain leads to neurological symptoms such as seizures, mental retardation, developmental delay, behavioral problems, skin abnormalities, lungs and kidney disease. Symptoms also include trouble in school and concentration problems.[4]

About 50% of people with TS have learning difficulties ranging from mild to significant,[4] and studies have reported that between 25% and 61% of affected individuals meet the diagnostic criteria for autism, with an even higher proportion showing features of a broader pervasive developmental disorder.[5] A 2008 study reported self-injurious behavior in 10% of people with TS.[6] Lower IQ is associated with more brain involvement on MRI.

Because of its phenotypic variability, the diagnosis of tuberous sclerosis complex can be difficult in individuals with subtle findings.[7] There have been few reports and data showing that patients with TS can still have no intellectual impairment.[8,9]

Case Report

A 25 year old female; resident of urban area, a teacher of mathematics teaching tenth standard; presented to the Department of Psychiatry, with the complaint of involuntary movements involving all the four limbs since last two days (last episode was about 10 hours ago).

History of presenting illness: As informed by the brother of the patient she has been having such type of involuntary movements since the age of 12 years. Though diagnosed as epilepsy by a local doctor but has never been on anti-epileptic medication. Currently, she presented with two days history of generalized tonic- clonic seizures, each lasting for 2-3 minutes. The seizures were associated with urinary incontinence, frothing, uprolling of eyeballs, tongue bite and loss of consciousness lasting for a period of about 20 – 25 minutes. Such episodes were occurring repeatedly during the above mentioned period (average of 2-3 episodes per month). No preceding history of fever, cough, diarrhea or fall.

Past history was not significant.

Birth and developmental history: Born out of non-consanguineous marriage; full term normal delivery at hospital. Vaccinations were done as per the schedule.

Milestones: Gross motor, fine motor, social and emotional and language milestones were normal.

Family history: Not contributory

General physical examination and systemic examination was normal.

Dermatological findings: No angiofibroma over the face or shagreen patch seen.

Dental Examination: Dental hypoplasia with pitting enamel.

Investigations

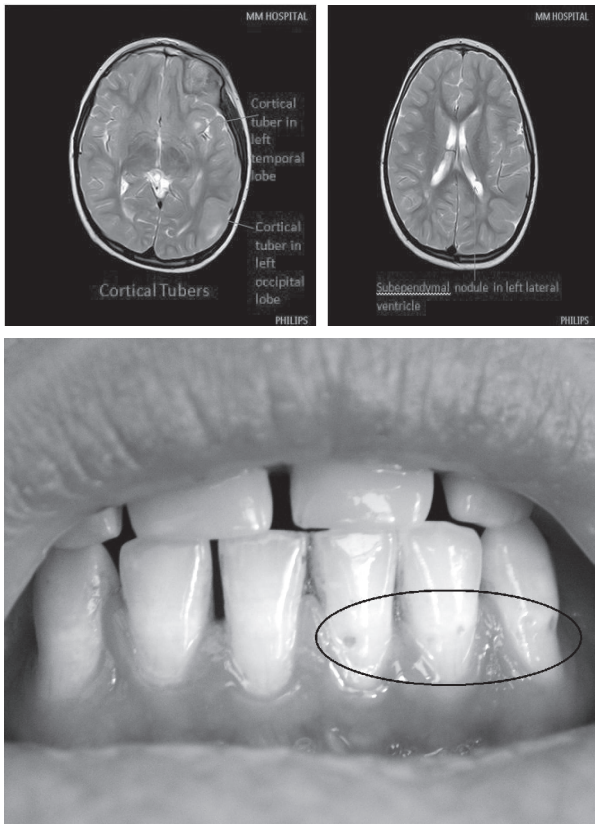
Haemogram; urine routine examination; routine chemistry, serum tests including prolactin, phosphorus, and magnesium, were all within normal limits.

Ultrasound abdomen; Chest X-ray; Skull X-ray and Fundoscopy were normal.

2D ECHO revealed normal findings with LVEF 62%.

EEG: Generalized slow waves observed throughout the EEG recording (also during photic stimulation and hyperventilation). EEG record was suggestive of inter-ictal period of generalized seizure disorder.

MRI Brain: There is evidence of multiple small calcified subependymal nodules along bilateral lateral ventricles which appear slightly iso to hyperintense on T1W and hypointense to grey matter on T2W and show blooming on GRE. Largest of them measures 5 mm. Multiple focal areas of altered signal



intensity appearing hypointense on T1W and hyperintense on T2W/ FLAIR images is seen involving the cortex and subcortical white matter in bilateral cerebral hemispheres suggestive of cortical tubers. Small focus of blooming is seen within one of them in left posterior parietal region.

IQ assessment was not done as the patient was a graduate and currently a teacher of mathematics at a high school.

Diagnosis: Tuberous Sclerosis (In view of Tubers and subependymal nodules on MRI; Seizures; Dental Hypoplasia with pitting enamel).

Treatment: Pharmacotherapy in the form tab. Levetiracetam 250 mg twice daily and tab. Clonazepam 0.25 mg twice daily was started. Levetiracetam was gradually increased to 1250 mg per day. She is currently maintaining seizures free for more than 6 months after the initial presentation.

Discussion

We presented the case of an adult woman with normal IQ having syndrome generally seen in children and is associated with mental retardation. In childhood it is easy to pick up the characteristic clinical features.

TS is an important genetic disorder; recognition of which is delayed in a minority until later childhood or adolescence when the manifestations are predominantly dermatological. The adult presentation in our patient was exceptional in not presenting until adulthood with any other features except the seizures. The mild and less clinical features in our patient show that there can be a variation in the presentation of tuberous sclerosis. Thus, it's an unusual presentation in adulthood with certain clinical and radiological signs suggesting of the disorder.[10]

Dental enamel pitting is observed in up to 100% of the patients with TS. Dental pits can also be observed in the general population, but at lower frequency and with fewer lesions than in TS. [11]

Conclusion

TS must be included in the differentials of children presenting with seizures, developmental delay and mental retardation. This case also provides a striking illustration of the extraordinary variation in expression of this disorder both with respect to age at presentation and severity of disease.

Recommendations for family screening

- Investigate family members when indicated, including family history.
- Clinical examination including examina-tion of skin with UV light and fundoscopy.
- Brain CT or MRI.
- The offer of genetic counseling.

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Case Report

Gilles de la Tourette's Syndrome: Spectrum of Illness in Family

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Introduction

Gilles de la Tourette's syndrome (TS) is a childhood onset neuropsychiatric disorder characterized by presence of concurrent vocal and motor tics and associated echo and coprophenomenon over a period of 12 months[1]. It is associated with other co morbid disorders like Obsessive compulsive disorder (OCD), hyperkinetic disorder (ADHD), anxiety disorders and substance use disorders. The disorder has definite heritability but the exact pattern of inheritance is not known. Functional MRI studies have implicated caudate nucleus and prefrontal cortex in the motor abnormalities and executive dysfunction seen in patients with Tourette's syndrome[2]. Illustrated below are two interesting cases with spectrum of illness in their families

Case 1

Mr. A 31 years, married, male studied up to 12th, a businessman by profession presented with history of isolated episodes of vocalization of abusive language since 15 years of age, in an otherwise well mannered child. His complaints began from 15 years of age when he was in 10th class. There were isolated episodes of use of abusive language. These episodes would occur de novo and preceded by an intense inner urge to abuse which would decrease only after vocalization. He used to develop intense anxiety if tried to suppress the vocalization. There was gradual increase in frequency of these episodes resulting in significant socio-academic dysfunction. Over the next 2-3 years he developed gradually progressive repetitive behavior in which he had to repeat each action twice, else would develop intense anxiety and the fear that something untoward is going to happen. These ideas were intrusive and he was unable to suppress them. He also had history of abnormal movement of the angle of mouth as if 'half smile', since 8 years of age. These movements were involuntary, non- rhythmic and increased on attempts to suppress. Similar movements were noted in eyelids, fingers and toes at variable times. In childhood history, he was an inattentive and hyperactive child with frequently disrupting the class and unable to complete his class work and homework. He was also a diagnosed case of hypothyroidism on thyroxin (25mcg) supplement.

Regarding family history (Fig. 1), father is 54 years of age, asymptomatic. Mother is 53 years of age and reported similar episodes of use of abusive language and expletives preceded by an intense urge, till 30 years of age. Younger sister 29 years of age has coprolalia and obsessive concern with symmetry and orderliness.

He was diagnosed as having TS with OCD predominantly mixed type and possibility of ADHD in childhood. He was treated with Paroxetine with gradual increase in dosage to 37.5mg. He responded well with 50% reduction in YBOCS score over a period of 8 weeks. Vocalizations decreased but were still present.

Case 2

Mr. S, 28 years, married, marine engineer presented with abrupt onset progressive complaints of abnormal movement at the shoulder joint since 8 years of age, repetitive production of abnormal sounds as if grunting or swallowing since 12 years of age and obsessive concern with numbers and checking and counting compulsions since 18 years of age. There was also history of isolated episodes

of vocalization of abusive words and obscene gestures since 11 years of age.

In family history (Fig. 2), father is a retired naval officer and is asymptomatic. Mother 52 years of age, a diagnosed case of OCD predominantly mixed with obsession regarding contamination and compulsion of cleaning and washing. Elder sister, 31 years of age, also has similar complaints and is on treatment for OCD.

Regarding personal history, birth and developmental history was normal. He was excellent in academics and sports. Recently married, he had asked for his Psychiatrist to be present during the marriage from the fear of urge to abuse during marriage.

He was diagnosed as having TS with OCD predominantly mixed. He was treated with haloperidol 1.5mg in divided dosages. He was also given fluoxetine 20mg/ day and gradually titrated over 8 weeks to 80mg/day. As assessed on Yale Global Tic Severity scale, there was 40% improvement.

Discussion

Initially considered rare, TS is known to affect 4-6 in every 1000 children.[3] Clinical picture can be described as a ladder with increasing complexity at each higher level e.g. pure TS to full blown TS and TS plus.[4]

Both patients had childhood onset, involuntary, repetitive and non-rhythmic movements that involved discrete muscle groups; called tics. Initially both developed motor tics, then 2-3 years later developed vocal tics and in late adolescence developed OCD.

An extensive literature search by Bloch MH and Leckman[5] also shared similar observations. It concluded that motor tics; simple (e.g. eye blinking) or complex (e.g. facial grimaces) begins around 4-6 years of age. Vocal tics which can be simple (e.g. grunting) or complex (e.g. coprolalia) usually develop around year or two after motor tics. Tics are suppressible but at the cost of increased inner restlessness and anxiety. Severity of tics tends to wax and wane. Symptoms change from one tic to another. Tics reach their worst ever around 10-12 years of age and improve during adolescence[5]. Similar progression of tics was seen in both cases.

Both the patients described above were diagnosed as having co-morbid OCD. OCD is seen in 1/3rd to ½ of patients with TS.[6] OCD with co-morbid TS tend to have greater rates of symmetry obsessions and counting, repeating, ordering and arranging compulsion.[10] OC symptoms in patients with co-morbid tics follow course similar to tics.[7]

First case was also diagnosed as having co-morbid ADHD. ADHD is seen in more than half of patients with TS. Symptoms of ADHD tend to precede the onset of tics. The inner sense of restlessness and state of hyper arousal seen in patients with TS is worsened in presence of ADHD, resulting in significant behavioural and academic problems than tics alone.[8]

Both cases have positive family history of psychiatric illness. Mothers in both the families have OCD. Even the female siblings have OCD. Genetic relationship between TS, OCD and ADHD though evident has not been clearly elucidated. A review done by Rourke et al concluded TS to be a heterogenous disorder with complex inheritance pattern. Though no causative gene was identified, largest wide linkage was observed with Chromosome 2p 23.2.[9] Heritability analysis done by Grados AM and Mathews CA that included 952 individuals from 222 TS affected sib-pair families; demonstrated high heritability of OCD and ADHD in TS families with significant correlation between TS and OCD and OCD and ADHD.[10] It further observed, offspring were more likely to have complex phenotypes than were parents. Mothers were more likely to have OCD alone and fathers were more likely to have ADHD alone. Parental diagnosis of full blown TS showed a stronger association indicating greater genetic loading.

To conclude presence of co-morbidity in both patients further decreased the functional outcome, especially in first case that had full blown TS. Greater association of OCD and TS can be seen in both the families. It is further to be seen whether OCD can be explained as an alternative phenotypical expression of TS susceptibility genes.

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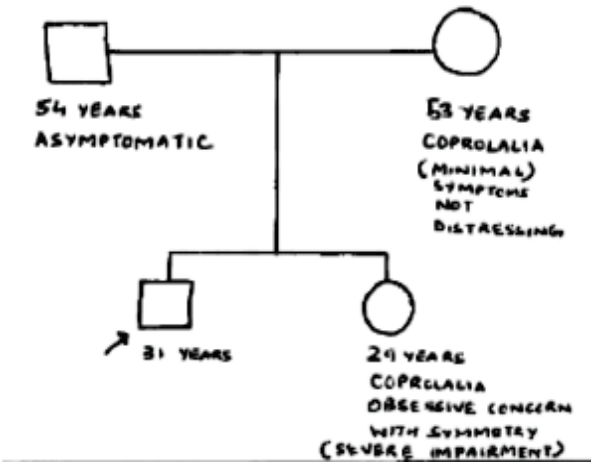

Figure 1 shows the pattern of illness in family of Patient Mr. A

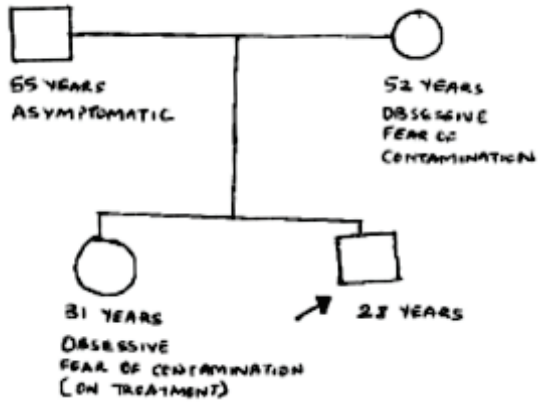

Figure 2 shows the pattern of illness in family of Patient Mr S.

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Case Report

Trichotillomania, Self Injurious Behaviour: A Case Study of Mental Retarded Child

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Introduction

Trichotillomania (TTM), a disorder characterized by repetitive pulling out of one's hair, has recently been identified as more common, more debilitating, and more complex with regard to structure and phenomenology then previously assumed[1-6]. According to 10th Revision of the International Classification of Diseases and Related Health Problems (ICD-10), trichotillomania is a disorder characterized by noticeable hair loss due to a recurrent failure to resist impulses to pull out hairs, usually preceded by mounting tension and followed by a sense of relief.[7] The first describer of the disease in history is the French dermatologist Francois Hallopeau, who coined the term "trichotillomania" (Greek for "hair-pulling madness"). Self-Injurious Behaviour is commonly found in development disabilities and mostly common forms of these behaviors include: head-banging, hand biting, hitting by hand and excessive scratching. Mental retardation is defined as significantly sub average general intellectual functioning associated with significant deficit or impairment in adaptive functioning, which manifests during the developmental period (before 18 years of age). In this definition, general intellectual functioning means, the result of a standardized intelligence test(s); significantly sub average means two standard deviations below the mean (usually an IQ of below 70); while adaptive behavior is the person's ability to meet the responsibilities of social, personal, occupational and interpersonal areas of life according to his or her age and sociocultural and educational background.

Case History

In our patient - a seven year old boy brought by his father with the chief complaints of pulling out hairs from head since 8-9 months, usually saying please tie my hands beyond back, head banging and hand biting and excessive scratching. His IQ was 57 which indicated mild moderate mental retardation in addition to self injurious behavior. The hair pulling was occurring throughout the day and did not have any precipitating events or factors. Pulling hair was evident in head. He was born after a full term normal delivery. Birth cry was present and his birth weight was 3.3 kg but there was delay in speech development. He had a history of fever (103ºF) at the age of one year for which he was admitted in the intensive care and was diagnosed as having viral encephalitis. No family history of psychiatric illness was found.

Behavioral Observation

He is a young male child who looks like his stated age. He was neatly and appropriately dressed. Attention could be aroused and sustained for the required period of testing without significant difficulty. Rapport could be established with him. He maintained proper eye contact and was active during testing session.

Management

Before going to management part, Malin's Intelligence Scale for Indian Children (MISIC) was used to check his Intelligence Quotient (IQ) and Vineland Social Maturity Scale (VSMS) was used to check his Social Quotient (SQ). His IQ was found to be 58 and SQ was 54. After that family members were psycho educated.

Functional Analysis

In order to obtain a detailed description of trichotillomania, self injurious behavior and the relationship between the behavior and his physical and social environment was collected. Which included, what happened before, during and after the behavior? Who was present? When did it happen? Where did it happen? For all this we find that family and school should be contacted and then session with both were planned.

Self-Injurious Behaviour

It was targeted first and functional analysis was done. The focus of functional analysis was first on specific behaviour and a step by step process was used. Like salient characteristic of the self-injurious behaviour was recorded in terms of frequency, duration and severity. Physical environment was understood, which included: the setting (e.g., classroom, playground), Sounds (e.g., another child screaming, Singing by other children), Lighting (natural light, bright light by lamp). Through functional analysis, we found that there were different forms of self-injuries. But no one of them appeared like attention seeking behaviour. Differential reinforcement technique was used and good results were found. Differential reinforcement refers to the process in which "desirable behaviours are reinforced and undesirable behaviours are not reinforced". After 12 sessions and hard work of teachers he showed 70% improvement, which was also reported by parents and teachers. Feedback was given to all those who were working with child.

Behavioural treatment of trichotillomania

Assessment was done first; it was done with the help of observation, parental reports, teachers report. He was finally diagnosis to be suffering from Trichotillomania. Than following steps were taken for the assessment and treatment of trichotillomania.

Functional Analysis

It is step which led us know about what factors cause a person to develop trichotillomania. In this step as we move on to get new perspectives on hair pulling and new skills for gaining control over this problem. A detailed discussion of such an analysis was done. Following factors were assessed in planning for the treatment: Antecedents to pulling (which includes cues that trigger the urge to pull and discriminative stimuli that facilitate pulling), the actual behaviours involved in the pulling and the consequences of pulling that either maintain or terminate pulling episodes. Above detail confirmed (a) settings where pulling takes place (b) visual or tactile sensation, such as the color and texture of hairs (c) time (d) activity. We also focused on discriminative stimuli (SDs) set the stage for pulling to occur and indicate that reinforcement is forthcoming. External SDs includes environments free of potential observers (e.g., bathroom, bedroom) and the presence of pulling implements (e.g., tweezers, mirrors). Internal SDs includes the urge itself, postural cues, such as "free" hands near the hair, and thoughts that facilitate pulling, such as "I deserve to pull or I will only pull a few". Emotional state was also assessed in which we try to understand what happen before, during, and following a pulling episode and how many hairs were pulled. The second phase of assessment involves the organization of information derived from the functional analysis into a system that points the way to specific treatment strategies. It can be observed in the example above that elements critical to the maintenance of the individual's hair pulling involve cues and feedback from both the external environment and experiences internal to the individual. These involve a number of interrelated but relatively independent response systems or modalities.

Management Techniques Used

A lot of information take from functional analysis made our work easy and from the functional analysis, it was clear that our patient shows this behaviour when he has no work or he is free. It was also found that he do it mostly in house. So, all this information was shared with parents and teachers. All teachers dealing with the child were informed and trained about the therapy. All long goals were divided in short goals and family was told to concentrate differential reinforcement technique.

Behavioural monitoring diary and activity schedule was also found were effective. In between he was given training of muscular relaxation, He was taught to make a clenched fist with the hand he uses to pull hairs, to bend the arm at the elbow as 90 degree and to press the arm and hand firmly

against his side at waist level. In last few sessions, treatment strategies were monitored for effectiveness and were modified as needed. Review of the rationale and types of replacement or competing behaviours that would help decrease his symptoms was constantly done. The patient showed significant improvement from session 8 onward. The therapy sessions were terminated with mutual consent between the therapist and the patient after 22 sessions. The patient remained on regular follow ups thereafter.

Discussion

After 22 sessions he showed improvement, but the important thing is that it would have not been possible with the cooperation of family members and teachers. Using functional analyses is an important step because it clarifies at a lot of symptoms. The treatment model used in this paper has really effected in reducing symptoms. But the therapist must decide in consultation with parents and teachers, which therapeutic techniques to employ, and how to apply.

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Case Report

Primary Disgust Obsessions

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Introduction

Obsessive Compulsive Disorder (OCD) is a condition characterised by repeated intrusive and inappropriate thoughts called obsessions and repetitive acts to counteract the same called as compulsions. The most common of these are dirt and contamination or doubt obsessions where the compulsions involve cleaning, washing or checking.[1] Very often however disgust may be implicated in the management and genesis of OCD. The evidence of the relationship between disgust and obsessive thoughts have been suggested across many studies.[2] Fronto-striatal region metabolic, blood flow and neuropsychological abnormalities have been noted in cases of OCD and this circuitry has been implicated in the emotion of disgust.[3-4] Studies also document that obsessions related to dirt, contamination and negativity may stem from disgust as a primary thought in a cognitive behavioural model for OCD.[5] We present here a case of a patient with OCD with primary disgust and negative thoughts in the absence of other common obsessions and compulsions.

Case Report

A 25 year old unmarried Muslimfemale who was a post-graduatepresented to the out-patient department of psychiatry with chief complaints of same disgusting thoughts coming to her mind out of the blue and suddenly. She was apparently alright 2 months prior to presentation when without any apparent stressor she suddenly started getting feelings of disgust accompanied by thoughts in her mind saying,'I will not give you Gold and money'. These thoughts would come while she was praying and in such a way that she was speaking these words to God and even while reading the Quran, she would feel disgust and these words would pop into her mind between sentences that she was reading. She would experience disgust almost simultaneously but she did not feel they were blasphemous. She claims that even when she was walking on road and in case she saw a person who had worn gold ornaments or was handling money, she would experience disgust and the above described words immediately come in her mind. She hadinsight that these were baseless and useless thoughts and tried not to think of it but they would come on their own.

She was very distressed with these thoughts and started feeling guilty about the same. To reduce the guilt, she started with the compulsive behavior of visiting multiple mosques daily and praying excessively to ask for forgiveness from God but yet her distress did not decrease. She also began to feel depressed and mild depressive features were present. There was no significant past medical or surgical history. No family history of any psychiatric illness was present. This was the first instant that she experienced OCD symptoms.She was started on Fluoxetine 20mg per day which was increased to 60mg per day in increments of 20mg per week over the next 2 weeks. She was later also started on Clomipramine 25mg thrice a day and the total daily dose of Clomipramine was increased from 75mg per day to 125 mg per day over 2 weeks. She and her family were called for psychoeducation sessions about OCD and she was started on behavior therapy involving thought stopping techniques. She was 50-60% better in a period of 3 weeks and has been following up regularly in our facility. She was not assessed on any formal scale and this was her clinical impression.

Discussion

There are far reaching clinical implications for the relationship between disgust and OCD features. Most patients report that disgust is more troublesome than anxiety and very often OCD patients may prioritize disgust reduction over anxiety reduction.[6] Very often OCD symptoms may not stem from anxiety as a primary emotion but may come up from the nature of disgust a symptom or thought produces and may result in guilt obsessions and compulsions.[7] Some researchers have proposed a need to subtype OCD on the basis of anxiety related OCD and disgust related OCD and the need to identify subtypes of obsessions and compulsions that may stem from these basic emotions.[8] The present case is a rare case of primary disgust related obsessions though further studies across diverse clinical populations are needed to identify and typify this rare form of obsession.

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Case Report

Migration and Schizophrenia

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Introduction

Interest in migration as a putative risk factor for schizophrenia has grown in recent years, generated partly by alarmingly high incidence rates obtained for persons of African Caribbean background in the United Kingdom.[1,2] Similarly, high incidence rates of schizophrenia have also been found for persons of Surinamese, Dutch Antillean, and Moroccan background in the Netherlands.[3] An increased risk for developing schizophrenia has recently been found for all migrants in Denmark, particularly those from Australia, Africa, and Greenland.[4] Such findings are yet to be adequately explained. A meta-analysis of incidence rates from 18 studies in a number of countries found an overall weighted relative risk (RR) for schizophrenia of 2.9 (95% CI 2.5–3.4) in first and second-generation migrants compared with non-migrants. The authors suggested that these findings cannot solely be explained by selection.[5] The exact mechanism remains obscure, although the focus remains on various psychosocial issues. Migration may add to the overall risk of schizophrenia, if not causing it by itself. We report the role of migration in the context of development of schizophrenia and subsequent management issues in an NRI lady of Indian origin who migrated to USA.

Case Report

Mrs. X, a 40 year old graduate Indian Hindu female migrated to USA in 1995 after her marriage to an NRI. The patient developed schizophrenia in 1997, insidious in onset, continuous and progressive characterized by delusion of reference and persecution, delusion of infidelity against husband, 2nd and 3rd person auditory hallucinations, disturbed sleep, disorganized behavior, violence, aggression and socio-occupational decline. There was no history of organicity or genetic predisposition. She was on treatment from U.S.A. for the last 10 years and had received many anti-psychotics, both oral and injectable with variable response. There had been 3 hospitalizations in the U.S.A. during this time. The patient had poor social support, had been detained in legal institutions and mental hospital multiple times in USA, did not receive appropriate and timely treatment, had consistent and frequent breaks in compliance, was neglected by her husband and relatives and had no full time caregiver. She was left in India by her husband in 2011 with her mother, who subsequently flew back to USA without seeking or taking any responsibility for her psychiatric care. She was presently staying in Delhi with her mother since May, 2011.

Psychiatric help was sought by her mother at the psychiatry department of a tertiary care general hospital in New Delhi as the patient was violent and could not be further managed at home by her as she was the only caregiver. The patient was evaluated in detail with regards to psycho-social and pharmacological perspective. At admission, the patient was conscious but uncooperative and did not allow to talk to her. She was treated appropriately with pharmacological (T. Haloperidol 30 mg oral and injectable depot haloperidol, due to history of poor compliance) and non- pharmacological (Family intervention, Psychoeducation, Crisis intervention, Relaxation training, Activity scheduling) interventions. The patient gradually improved during her 28 days stay in the hospital and was discharged with advice of consistent follow ups. On further follow ups, detailed interviews with the patient

revealed the many Psychosocial stressors that she experienced - Change in lifestyle, lack of social support, burden of all household responsibilities, child birth and rearing, perceived social and religious discrimination.

Discussion

The case highlights the role of migration in development and progress of schizophrenia in a lady of Indian origin migrated to the U.S.A, without any apparent genetic influence and substance use. It pertains to a change in the psycho-social environment, from a socially, culturally and religiously supportive environment to a more liberal, independent and unsupportive one. Change in lifestyle, lack of social support, burden of all household responsibilities, child birth and rearing, perceived social and religious discrimination add up to the existing levels of social stress and predispose to psychiatric illnesses.

A number of substantive explanations have been proposed to account for the repeated finding of elevated schizophrenia incidence in many migrant and minority ethnic groups: (a) selective migration, (b) genetic, (c) neurodevelopmental, (d) substance use, and (e) (psycho)social. The recent concept of 'Socio-developmental' model which focuses on the role of social issues superimposed on genetics and neurodevelopmental insults might be important (6). Recent research suggests that repeated exposure to social adversity can link to psychosis through the generation of cognitive biases and affective states that predispose to symptom formation, eg, repeated exposure to threat may link to paranoia and formation of persecutory delusions through increased threat anticipation, anxiety, and a consequent tendency to jump to conclusions.[7,8] Treatment issues like inadequate psychiatric care in another country, lack of records, financial problems, abandonment by relatives in country of origin without resources also need due consideration.

Current research proposes that schizophrenia is caused by a genetic vulnerability coupled with environmental and psychosocial stressors, the so-called diathesis-stress model. Family studies suggest that people have varying levels of inherited genetic vulnerability. At the same time, the development of schizophrenia also depends on the amount and types of stresses the person experiences over time. Migration may serve as a stressor, adding on to the genetic vulnerability. However, this was not true in this case. Further epidemiological and descriptive studies are needed to establish the role of migration in development of schizophrenia as well as treatment issues in patients who have migrated to other countries.

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Case Report

Cannabis Induced Anaphylaxis

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Introduction

In Indian literature the earliest reference to the word "bhang" occurs in the Atharva Veda, which, according to western scholars, dates as far back as 2000 to 1400 B.C[1]. According to a UN report cannabis remains the most widely used illicit substance globally. It was estimated that in 2010, 2.6-5% of the adult population used cannabis.[2] Key ingredient in cannabisis THC (tetrahydro-cannabinol), whose amount determines the drug's strength. Cannabis's effect lasts for 2-3 hours if smoked and 24 hrs if ingested. Three types of Cannabis products, often called charas, bhang, or ganja are abused throughout our country. Anaphylaxis is a severe, whole-body allergic reaction to a chemical that has become an allergen. After being exposed to an allergen, the person's immune system becomes sensitized to it. Anaphylaxis happens quickly after the exposure, is severe, and involves the whole body. Tissues in different parts of the body release histamine and other substances. This causes the airways to tighten and leads to other symptomsoften within seconds or minutes, including abdominal pain, abnormal (high-pitched) breathing sounds, anxiety, chest discomfort or tightness, cough, difficulty breathing, difficulty swallowing, dizziness or light-headed-ness, itchiness, nasal congestion, palpitations, skin redness, slurred speech, swelling of the face, eyes, or tongue, wheezing and also sometimes uncon-sciousness. Signs includearrhythmia, tachycardia, pulmonary edema, hypotension, mental confusion, cyanosis, pallor, angioedemain the throat that may be severe enough to block the airway, swelling of the eyes or face, weakness and wheezing. Importantly, anaphylaxis is a clinical diagnosis. No specific lab investigations are required to diagnose anaphylaxis.[3,4]

In India, where the popularity of cannabis has still a stronghold; reporting and discussion of life threatening adverse reaction like anaphylaxis becomes extremely important.

Case Report

A patient, 18-year-old man, presented to medical emergency with abrupt onset of symptoms including upper respiratory tract involvement in form of difficulty in breathing and speaking, nasal congestion, hoarseness of voice, itching sensation in the eyes and nose and sensation of swelling and tightness in the throat and chest. Patient became extremely distressed due to above symptoms and was rushed to the hospital. His symptoms began within 5-10 minutes of smoking 3-4 bidis of cannabis.There were no associated features of any rash, bladder involvement, seizures, mental confusion and weakness. No h/o of any other poisonous substance, any previous h/o similar episode and also patient had no past history of cannabis use, although history of occasional tobacco smoking was present. On examination patient was conscious, cooperative, well oriented to time place and person. Glascow Coma Scale score was 14 (E4M5V5). Vital examination revealed pulse 100mg/min, BP 110/70 mmHg, respiratory rate 30/min with accessory muscles of respiration working, temperature 99 F. Patient's pupils were dilated, sluggishly reactive to light. Further, physical examination revealed non-pruritic and non-pitting angioedema of the tongue and lips; alsoodema over eyelids was observed which led to difficulty in opening the eyes completely in the patient. Patient appeared extremely restless. Poisoning due to dhatura, opioids and other substance of use was also ruled out by thorough physical and biochemical testings. As the symptoms and signs were found to be suggestive of anaphylaxis, immediate and aggressive supportive management of the same was initiated in the patient including

vital monitoring, regular cardiac monitoring, antihistamines and high dose corticosteroids, in medical ICU setting. On the basis of the strong evidence of the temporal relation between the onset of anaphylaxis and cannabis consumption and thorough clinical examination, diagnosis of cannabis induced anaphylaxis was made. Patient started showing improvement within few hours of initiating treatment.

Discussion

Cases of marijuana allergy are rare in the medical literature, but they may be more common in real life. Humans are often allergic to pollens and other chemical compounds in weeds.Reactions range from the annoying (runny noses) to dangerous (anaphylactic shock). Delaying the diagnosis or misdiagnosis of anaphylaxis can be fatal. In this case report, smoking of cannabis was documented as a trigger factor for abrupt and severe allergic reaction; anaphylaxis, which could have been fatal in an 18 year old male who was experimenting with cannabis for the first time. The reaction wasvery rapid in onset, occurring within minutes, and wasmoderate in severity. He was managed on the same lines under ICU setting and showed complete improvement within 2 days of starting treatment and was further advisednot to consume cannabis in any form. By avoiding the same, he didnot develop any reaction in 2consecutive months of follow up. Till date, no case report of cannabis induced anaphylaxis has been reportedpreviously as far as the authors have searched the literature, though cases of allergic reaction have been reported. Some cannabis users on online marijuana communities have reported allergic reaction to various forms of cannabis.[5] Yet, it is still not clearly established. Importantly, locally made cannabis "bidis" may contain manycontaminants which may be the trigger anaphylactic responses in many. Out of the scanty literature about the allergic reaction to cannabis a frequently cited report describes an essentially negative pathological and toxicological study of a 23-year-old man found dead in the presence of marihuana, and hashish. A cannabinoid was detected in his and authors pondered at the anaphylactic reaction as one of the possible cause of sudden death in the man. Several case reports noted acute severe, physiological disturbances and acute collapse (shock, chills and fever) subsequent to intravenous injection of suspensions of marihuana[7]. They also wondered at the possibility that these symptoms could have been possibly due to an allergic reaction to injected foreign plant material.[6,8]

These cases potentiate the fact that cannabis per se or the various impurities present in the cannabis products may precipitate severe allergic reaction or anaphylaxis in the patient. The widespread use of cannabis,in spite, of the ban under NDPS act, for its medicinal, recreational and cultural value, it becomes extremely crucial that awareness about the possibility of a fatal reaction like anaphylaxis because of its use is reported and looked for as well in future.

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Case Report

Socio-cultural Difficulties Faced by a Family of Interior Village Community of India while Managing a Violent Psychiatric Patient at Home: Analysis of a case

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Introduction

Most mental health facilities in India actually function as passive recipients of patients. They become operational only where coping mechanisms in the community fail. It is essential that the role of all mental health institutions in India become more active with the knowledge of socio-cultural mechanisms involved, not only in the development of mental illness, but also in the more important issue of maintaining mental health.[9-12] Researchers of the mental health field have very little knowledge and understanding about the local coping mechanism, coping resources as it exist and operate especially in the village community of India. With almost 75% of the Indian population still living in the rural areas and all sorts of inadequate facilities namely poverty, poor education, poor housing, drinking water and health care facilities including very few number of mental health professionals (viz. one psychiatrists per million people) only reinforce the local beliefs, shape the help seeking behaviour in alleviating distress of the people.[1,5,6,10,11] Most of the rural people approach the faith and religious leaders for treatment of physical, mental and emotional problems. These may include the shaman, sufies, priests of some of some special deities guarding against evil powers.[2,7,11] In India during the last few years at least four important research studies documented well the need of understanding and involving the socio-cultural mechanism of the community to develop a successful, effective model for community mental health care.[1,2,3,9,12,15] The present study is a timely effort which thoroughly studied a case where a family of rural based interior village community, were seeking help for acute psychiatric problem with one of their family member.

Method

This study aims to enumerate the underlying factors and situations behind time lag in reaching out for treatment to a tertiary psychiatric hospital by a poor family of a village in managing a violent patient at home. Well-informed consent was taken from the father of the patient (during psychosocial assessment of hospital based in-house treatment) to share their personal distressful experiences to the scientific educational, research forum to enrich existing knowledge of the socio-cultural barriers of community mental health field. The study analyses the socio-cultural difficulties faced by the family in caring for their psychiatrically ill relative, hailing from a poverty stricken rural based interior village community of Muzaffarnagar district of Uttar Pradesh, who was ultimately brought to IHBAS, Delhi for treatment as a last resort.

The socio-cultural problems experienced by the family were basically identified as stigmatization, lack of community support and participation due to utter ignorance, poverty and misconception about causes of mental health problems. Community based rehabilitation of this case remained unanswered since the parents of the patient lost their lives untimely due to his symptomatic homicidal behaviour.

The Case

Md. S, a 17 year old Muslim boy, was hailing from a interior rural village community

Muzaffarnager district of the state Uttar Pradesh, staying along with his parents and three siblings. He was one of the major earning member of the family, working with his father in the brick making factory as well as in the agricultural land as and when there was work, until he got high fever with convulsions. He had high fever continuously for seven days, since had no relief in the condition, his parents were taken to a local ayurveda? doctor as was the usual practice in that village to approach him for any problem. Md S's fever subsided in a few days but he did not get back to his 'normal' life. He started to show some abnormal behaviour like disorientation, confusion, inability to recognise known persons, irrelevant talk, talking to self, occasional head banging and incontinence.

Various indigenous treatments were sought in which the experts said 'something unnatural' had happened with the person which was possibly due to some bad deeds committed in the past. As the person's condition deteriorated, his poor father with help of the neighbours was taken the patent to the local 'primary health care' centre a distance place for better treatment, however, in absence of any specialized doctor, the physician prescribed and put him on some tranquilizer medication although referred to district level hospital or a private practitioner for better treatment.

However, the father had so far spent all his savings on his son's treatment couldn't gave priority to provide treatment in the district level hospital. After lose of major earnings from one of the significant member, the family was already in a financially collapsed condition. He then kept the son 'home bound' in tied up condition, as he was becoming unmanageable and some people in the neighbourhood advised him not to let the boy out as he was possessed by an evil spirit. Patient's condition worsened with increase in aggression and violent behaviour and one day, when he was being taken for bath, he suddenly and unexpectedly attacked the mother with a boulder, poked the eyes of the father with a sharp stick who rushed to save his wife, ran out and stoned to death his pet. The neighbours and others in the village then managed to catch him, beat him, tied him up and brought him back to his home.

While both parents suffered severe crisis of being badly injured, being unable to go to work and earn money for their daily bread, the doctors of primary health care centre referred the mother for immediate surgical intervention and the father for immediate medical emergency. The incidence left father in totally helpless condition and the expenses incurred on the mother were paid off by taking a huge loan from a money lender charging very high interest, but the mother could not be saved, she died within three months. These stressors were compounded when the villagers 'ostracized' the entire family, by completely cutting off any interaction and communication, any kind of support to the family and warned the family to stay away from the village, failing which, they would be compelled to get the boy arrested and send him to police lock up for their own safety. The village panchayat and other village level leaders remained ignorant since they had lack of clear knowledge to address the problem and also having strong belief in evil spirit, black magic etc.

Thus the father even with his ill health again tried to survive, to manage household by earning of daily wages as restarted work in the brick making factory while had no time and choice to take care, give city based hospital treatment of the psychiatrically ill son. A further crisis happened when the patient threw the infant sister from atop the first floor killing her instantly. Due to all this, the father became helpless and though grieving, had the entire onus of caring for the patient.

At last the community people, village level leaders acknowledged and understood the possible danger of keeping the patient in the community so they took the boy in tied up condition to nearest police station, handed over him to keep in locked up in jail for safety of the community people. However, the police refused to provide any such help against the boy as he was psychiatrically ill and send them back to seek help from village pradhan. The village pradhan then took the pain to discuss the matter with possible places and higher authorities, referred the patient to Delhi, Institute of Human Behaviour & Allied Science (IHBAS) for treatment. The village pradhan and other community people then contributed money to arrange the travel fare as well as other expenditure, some of them also accompanied father to brought the patient to Delhi based hospital.

At IHBAS patient was admitted and necessary

evaluation and the treatment was initiated. It was found that the patient had been suffering with organic psychosis condition, and he was kept in strict vigilance. With appropriate medication some improvement found in his symptomatic condition but did not reach to manageable functioning condition to stay at home. However, within six months of patient's admission, his father also died for his old untreated injuries and ill health condition. Community based rehabilitation of this case remained unanswered since the significant family members (i.e. parents) of the person lost their lives untimely due to his symptomatic homicidal behaviour.

Discussion

The reality is very tough for the village level poverty stricken people as hunger comes first before health care and related issues. The patient's father and the majority of the villagers in the village, do not possess any agricultural land, but used to work in local factories or work as daily wage earners in the land owned by other higher caste people of the village. At time of any health problems they often have to rely upon the indigenous doctors not only out of their believe system but also for easy accessibility, affordability of such treatment. In order to access semi urban, urban based good health care facilities, they have to spend good amount of money as a travel cost, which remains difficult to manage from their little earning. In support of this, previous studies revealed that the traditional belief shows the prevalent illiteracy and the poverty which is evident by the money spent and the time devoted to treatment.[1,3,5,6,10,11] The present study reflects the truth that the local faith healers often reinforce the distress, discomfort and sense of stigma while dealing with unresolved critical aspects of health, mental health or emotional problems but due to their poor understanding, lack of scientific knowledge. Under influence of these faith healers the village people often ostracize the whole family. Such findings also reported by in the previous studies[7,5,6,14] that the local religious leaders, faith healers take a leading role in alleviating pain, distress yet often increase sense of stigma with prevailing misconceptions of misdeed of past life, effects of evil spirit, effects of dissatisfied demon etc.

Instead of showing alertness in such situation the local government, non government bodies, local leaders expressed casual indifferent attitude, do not give due attention and try to super impose the responsibility to other department or even exclusively to the sufferers family or even upon the "spirit of demon", "sprit of evil" or upon almighty. The panchayate raj is a very successful and positive aspect of socio cultural and political scenario of India. However, the role of Gram Pradhan is questionable since they do not have enough awareness and knowledge in handling such critical situation beyond their stereotype, traditional mind set.

In this case the time lag between the identification of symptoms by the local doctors and consultation from a mental health professional at city based hospital was high, which delayed the initiation of psychiatric treatment of the patient. Often the physician of primary health centre or even health workers identify & recognize the "criticality" of the "case" or situation but various limitations of the local community people also do not allow them to extend helping hand beyond their capacity to move the family and patient to a better treatment centre. The present study strongly emphasizes the view that early intervention at community level is indeed needed which can help to reduce this time-lag in initiating psychiatric treatment which can lead to save the lives of the family members from unfortunate accidental homicidal acts by the patient.

The problems or difficulties faced by the family were tough enough and challenging. It does not specify a single pathway of barriers faced by the family while searching help for managing their violent psychiatrically ill son at home. Rather it is a societal structure in which multiple negative pathways experienced by the family during their constant efforts of adjustment and help seeking process in providing treatment for their son's psychiatric illness. The difficulties were coming in a broad spectrum, where the total family unit was struggling to have its existence in managing everyday earning to have food, facing everyday distress as stigmatized and ostracized by the local people, which also added fear of losing job, fear of becoming homeless as already indebted with huge loan, to bear the cost of minimal level of treatment of the severely injured significant family member of the family, to bear the pain of accidental death of children by the

homicidal act of patient, and also the painful feelings of lost of 'normalcy' in their eldest son who was once major earning member of the family. A financially collapsed, compromised, stigmatized, helpless, hopeless condition were evident and invariable phenomena congruent with the prevailing socio-cultural situation of such village community of India, through which the family had undergone. An early and timely local level supportive participation and scientific intervention by the community leaders could have been made a positive stroke to show a ray of hope for the family.

Suggestions

The study also reflects the need to assert 'socio-cultural model', in implementation of health, mental health programs, which is indeed required to meet the social needs, challenges of the rural people of India. A shared scientific realistic information based approach, services through various awareness generating programs, training programs among different stakeholders (both government and non-government i.e. panchayate pradhan, village leaders, local religious, educational institutions, local faith healers, health workers) at the village community level may bring a positive change in the community mental health aspects of such interior village community of India.

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Case Report

Psychosis in SLE

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Introduction

Systemic lupus erythematosus (SLE) is the prototype of systemic autoimmune diseases. Damage to tissues and cells results from pathogenic auto antibodies and immune complexes.[1]

Neuropsychiatric manifestation of SLE (NPSLE) is one of the major and most damaging presentations. It comprises a wide range of neurological syndromes affecting the central, peripheral and autonomic nervous systems, as well as psychiatric syndromes. In view of the diverse clinical manifestation of NPSLE, the American College of Rheumatology research committee devised a nomenclature which gives case definitions for 19 neuropsychiatric syndromes in SLE.[2]

Neuropsychiatric manifestations are present in two-thirds of the patients with SLE.[3] No single clinical, laboratory, neuropsychological, and imaging test can be used to differentiate neuropsychiatric systemic lupus erythematosus (NPSLE) from non-NPSLE patients with similar neuropsychiatric manifestations.[4]

Non-SLE causes such as complications of lupus therapy, infection of the central nervous system (CNS), metabolic dysfunction and drug intoxication must be excluded before being ascribed to the underlying immunopathogenic mechanisms[5]. Here we describe one case of psychosis which presented as a diagnostic dilemma.

Case report

Miss S. 36 year old, unmarried, graduate, unemployed, lady belonging to middle socio-economic status residing in Delhi presented in Psychiatry Out Patient Department with abrupt onset illness of 10 days duration characterized by irrelevant talks, disorganized behavior in terms of rubbing stools over body and urinating in bed, disrobing in front of family members. She was muttering to herself and could hear voices which were clear, and not under her control. The voices were discussing her among themselves and also commanding her. Patient could also see people who were talking to her which others could not see. Patient was aggressive and violent and could not be controlled by family members and was admitted in the psychiatry ward.

On detailed history it was revealed that they had consulted a private psychiatrist immediately after start of illness, was diagnosed as acute psychosis and started on olanzapine 10mg, increased to 20 mg within a week but patient's symptoms didn't show any improvement. Also that three years back patient developed oral ulcers, hair loss and joint pain and was diagnosed to be suffering from Systemic Lupus Erythematosus (Anti Nuclear Antibody-positive, Anti ds DNA-positive). She was prescribed Hydroxychloroquin 200 mg twice a day but took it for a week and experienced abdominal discomfort and stopped the medications on her own. Joint pain and oral ulcers have been present on and off.

There is also family history of psychosis in her elder sister who is currently asymptomatic and on treatment. There is also history of psychosis in her step sister (daughter from mother's first marriage). She is currently residing in a long term care facility in and no other information is available about her.

On general physical examination there was pallor, oral ulcers, enlarged bilateral axillary and cervical lymph nodes that were firm, non-tender, mobile, hepatomegaly 3 cm below costal margin non-tender regular margins and smooth surface, splenomegaly 2 cm below costal margin non-tender. Neurological examination was unremarkable. Mental status examination revealed inappropriate affect, loosening of association, delusion of

grandiosity, auditory (third person) and visual hallucinations and insight was absent.

Investigations

Haemoglobin-8.9 g/dl, Peripheral smear-RBC's Microcytic Hypochromic with mild anisopoikilocytosis. Total WBC- 4,000/cumm, Differential Leukocyte Count P-47, L-42, E-3, M-8. MRI (Brain)-no significant intracranial abnormality. Liver function and kidney function, electrolyte, urine examination were within normal limits, FNAC of left axillary lymph node revealed reactive lymphadenitis, Anti Nuclear Antibody-1.9 (positive>1.2), Anti RNP Antibodies-99.66 U/ml (positive>9) Anti ds DNA Antibody-64.31 IU/ml (positive >55).

Treatment

Patient was continued on 20 mg of olanzapine per day. Initially patient required injectable haloperidol (5mg) and phenargan (25mg) once a day for 2 days to control her aggressive behavior. She was also started on oral iron and folic acid. For lupus she was given intravenous methylprednisolone 1 gram for 3 days which was then shifted to oral dose of 50 mg per day. During the hospital stay of 4 weeks patient showed gradual improvement in psychotic symptoms and the general physical condition. After discharge she was followed up till next three months when she has no psychotic features and sign or symptom of SLE. Olanzapine was continued at 20 mg per day.

Discussion

The following case presents with diagnostic difficulty as the distinction between lupus psychosis and schizophrenia is difficult in the background of SLE, steroid intake and a strong family history of psychosis.

In view of the strong genetic loading of psychosis patient could be suffering from schizophrenia which was precipitated by neuro-inflammatory process of SLE. It has been suspected for many years that autoimmune mechanisms may play a role in the pathophysiology of schizophrenia, and studies over the last 10 years, have revealed that a subgroup of schizophrenia patients have several significant immunological abnormalities, including increased prevalence of autoimmune diseases and of antinuclear antibodies (ANA) and anticytoplasmic antibodies (ACA), and an association with HLA antigens which are also characteristic of SLE.[6]

Although the dose of steroid in this case was 20 mg per day and corticosteroid-induced psychiatric disease occurs in 10% of patients treated with prednisone 1 mg/kg or more and it manifests primarily as mood disorder (93%) rather than psychosis.[7]

The diagnosis of lupus psychosis at the onset of the disease has been described in one-third of the cases of a large series of SLE patients[8] and it has been observed that it usually occurs within the context of florid activity of the disease, associated mainly with cutaneous and haematological manifestations[9]. There is a paucity of data on the manifestations and long term outcome of lupus psychosis as well as treatment strategies and more research is needed in all these aspects.

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Case Report

Paranoid Schizophrenia with Plica Polonica

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Introduction

Matting of scalp hair is sometimes normal but irreversible matting is rare.[1] Minor degrees of matting is common in long hair, but the severe form of it involving almost all the scalp hair is much rarer. In situations of gross neglect, it has been referred to as plica polonica and in hysterical patients as plica neuropathica.[2, 3] Hair are irregularly twisted and irreversibly entangled to form a malodorous, encrusted and sticky moist mass.[4] The matting is sometimes compared to a process of felting which is well known in textile and wool industry which causes conglomeration of contiguous fibers when exposed to friction and compression in a liquid medium.[5]

It was first coined by Le Page in 1884 to describe a case of sudden onset entangled hair in a patient with hysteria. It was a prevalent condition in Poland in the 19th century. It is also known as 'Bird's nest hair.[6] It is also a common religious custom to raise a plica for wish fulfillment or to have a divine image among the public (e.g in sadhus known as "JaTaa" and "CaTai" which means "to twist or to wrap". Plica polonica though rarely reported is seen frequently in the Indian culture as the scalp hair has been a prime target of superstitious beliefs. There are multiple beliefs like clipping of Plica could result in death.[7]

Case History

A 32 year old divorced Hindu female was admitted to psychiatry ward with history of aggressive and suspicious behavior, socially withdrawn, refusal to eat and drink for 20 days now. On exploring history it was found that she had a long history of paranoid behavior since age of 22 years. History of delusion of paranoia and reference were present. She had history of auditory hallucinations (third person and commenting) in the past which she denied at present. As if hallucinatory behavior was present in the form of muttering and gesturing. Refusal to eat was because of paranoia itself. She belonged to a well educated family and she was teaching botany in a college as lecturer before she left it because of her illness. Marriage could not last 6 months as she filed divorce alleging infidelity against spouse. Her pre morbid personality was a reserved and introvert person with few friends. She was described by family a socially shy girl but sincere in studies and duties she assigned for. Her all laboratory investigations were within normal limits. She was diagnosed to be suffering from paranoid schizophrenia as per ICD-10(International Classification of Diseases) criterion. In ward, Rapport was very difficult to establish. She was made to take bath by staff forcibly and during that only she became aggressive when made to wash her hair. She was hiding it all under the scarf a big nest of hair. Family told that she in not taking proper bath for few months now and her hair started getting messed up. Patient started applying lots of oil and different creams, shampoo and conditioner many times a day on occasions to smooth her hair. She will occasionally take head massage vigorously and tried pins, small rods etc to straighten her hair (as few of pins and part of metal object were removed from hair nest). Dermatology consult was taken for same and she was advised head shave and explained cause of her hair problem. From psychiatry point of view, she was managed with clozapine 150mg per

day and clonazepam 1 mg per day in divided doses and 3 ECT's (Electro convulsive therapy). She was discharged in a satisfactory condition and had 3 follow up till now and maintaining well. After her consent, head was shaved hair are growing again nicely.

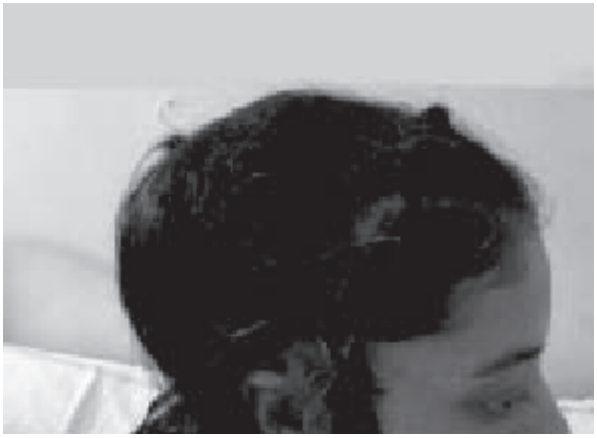

Figure-1

Figure-2

Figure-3

Discussion

Matting of scalp hair was formerly supposed to be a disease peculiar to Poles (hence named as plica polonica) and was usually the result of neglect, filth, the invasion of parasites (lice), and consequent exudative disorders of the scalp.[8] As a result, a confluent purulent mass of matted hair, lice, nits, crusts and purulent discharge formed. It has a peculiar, unpleasant odor and looks more like a "decaying bird's nest" than scalp hair.[9] The Polish custom of wearing tight fur caps and the superstitious belief that a lousy scalp was healthy no doubt contributed to the frequency of plica polonica in Poland in the 19th century.[10]

The exact mechanism for plica polonica is not well understood but according to evidence, psychological, physical and chemical factors may play a role in the damage to the hair shaft. The major factor in the pathogenesis of matting of hair is thought to be the physical phenomenon of felting, possibly enhanced by electrostatic attraction between hairs under some circumstances or by the formation of liquid crystal.[2] overly vigorous mechanical manipulation, especially of long, curly hair, may be responsible for the hair matting.[3] Kinky hairs, use of shampoos, febrile illness, and psychological disturbances are possible predisposing factors for the matting of scalp hair.[10,11] This phenomenon is also reported following irritant contact dermatitis of the scalp[12], with neglect of long hair resulting in scalp hair infestations & scalp pyoderms,[11] hysteria[4] and schizophrenia.[13] In our case patient had curly kinky hair and other predisposing factors like extensive oiling, shampoo, vigorous massage and her poor insight and poor self care contributed to plica polonica. Bogaty and Dunlap found that increased matting occurred with mechanical action, bleaching solution treatment and fine or dense hair[5]. Simpson described this in five of seven women with hysteria[14]. The scalp in these cases had a healthy appearance, free of offensive odor and parasites. Kumar et al described plica formation suddenly in a patient of schizophrenia.[4] Rare possibility in few cases could be poor sleep hygiene and unconscious habit of making whorls of hair which could mechanically damage the hair (sometimes associated with compulsive hair pulling, trichotillomania).

Since the condition usually occurs due to neglect of the hair, the emphasis of good hair care practices should be taught to all psychiatric patients in order to further decrease the prevalence of this

uncommon disorder. Prevention includes hair care measures like regular cleaning of hair with mild cleansers or shampoos, gentle oiling and combing to avoid entangling and regular hair trimming. Hair piling over vertex while washing and backcombing should be avoided. Manual separation using organic solvents are of limited benefit only in early cases. Also back combing and hair piling over vertex should be avoided.[11]In our case, plica polonica probably was due to poor hair care practices and thus we emphasized the importance of good hair care practices. In our case, all the mechanical and chemical solution for treatment of plica failed and patient was advised head shave. On follow up her hair were growing nicely and she was satisfied with response. She was taught hair hygiene by dermatologist.

It would be worthwhile to analyze the various psychosocial and physiological basis of this condition by doing further detailed studies.

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Case Report

Visual Realism in Autism

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Introduction

Autism is a pervasive developmental disorder generally presenting in children in the first 18 months of life with a triad of impairments in communication, social interaction, and imagination. Researches have been done to ascertain the representational drawing and transition from intellectual to visual realism in children with autism. Representational drawing emerges in children at the age of 20 months when scribbling occurs.It occurs through a series of stages: (i) intellectual realism- in which children draw what they know about an object; and (ii) visual realism- in which the children draw what is actually seen [1]. Striking realistic drawing talent has been reported in a small percentage of savants, those individuals diagnosed with autism who exhibit a disproportionate ability in one domain such as realistic drawing, music etc. Some savants use a strategy "construction by local progression" exhibited by savant E.C. for realistic drawings in which details are drawn first, and then moving on to an adjacent part before completing a part already begun. However, local processing strength exist alongside intact global processing in autism that,is they have "enhanced perceptual functioning [2]. This case report describes an autistic boy with realistic drawing talent.

Case Report

A.W., a right-handed male, age 13 years, 2 months presented with motor stereotypies rocking and headbanging, marked impairment in the use of eye-to-eye-gaze, gestures to regulate social interaction and repetitive use of language. Exploration of history revealed that A.W. was born full-term by the cesarean delivery. He weighed 3.3 kg at birth with good spontaneous cry and respiration. A.W developed jaundice after birth and home phototherapy was started. The jaundice resolved, but thereafter he did not return to breast feeding as well as he had before. In his infancy, he did not smiled, resisted being hugged, did frequent hand-flapping, and headbanging behaviors . During the first 6 months of life, A.W displayed difficulties in domain of social interaction including difficulty to maintain eye contact , and marked difficulties in oral motor coordination and muscle tone that fluctuated between hypotonia and hypertonia. He startled easily and was hypersensitive to touch. A.W was administered the Childhood Autism Rating Scale (CARS) to ascertain the severity of autism. He received a total score of 35.5 .He clearly met diagnostic criteria of mild autism based on CARS. He experienced expressive language delay but showed special talents in drawing.

Cognitive Assessment

A.W was administered Differential Ability Scales at the age of 3. He received a global cognitive ability score of 130, indicating superior to very superior cognitive abilities in understanding of spoken language and block building .

Representational Drawing tasks

A pre-test was administered initially to ascertain A.W.'s concept of view specificity in pictures. Successful performance on the pre-test was necessary for the presentation of representational drawing tasks. For the pre-test, he was shown a teddy and a doll and was asked to select the photograph showing their present position. A.W showed successful performance on these tasks and thus the representational drawing tasks involving a striped mug, teapot, cup-and-saucer, and cup-and-

Fig-1. Drawing of the mug.

Fig-2. Drawing of the teapot.

Fig-3. Drawing of the cup-and-saucer

Fig-4. Drawing of the cup-and-flowers

Figs. Representational Drawings of A.W.

flower tasks were presented with their occluded handles and A.W. was asked to draw them exactly showing their present position.

The tasks presentation and scoring of representational drawings were done as per Ford and Rees.[3] A.W. was able to produce realistic drawings for all the tasks as shown in figure 1 to 4. While drawing, A.W. used the 'construction by local progression' strategy used by the savant, E.C.[4] He did not sketch in the overall shape but rather drew part-by-part. Thus, for the mug (Figure 1), A.W. began at the top and worked his way down to the base.A.W's drawings were assessed for visual realism in terms of feature positioning. His drawings received a perfect score of 1.0 for attainment of visual realism by portraying stripes on the mug (Figure 1), saucer at the base of the cup in a partially occluded manner (Figure 3)and for depicting flowers (Figure 4). The drawing tasks were also assessed for visual realism in terms of fine details. A.W. received a score of 1 for depicting the rim of mugs (Figure 1), knob on the lid of the teapot, the hole in spout of the teapot and the elongated base of the teapot (Figure 2), and the ridged edge of the saucer (Figure 3).

Discussion

The current case report analyzed the representational drawings of A.W. an autistic child with noted strengths in drawing. A.W.'s drawings showed attainment of visual realism. He exhibited the same drawing strategy of 'construction by local progression' as used by E.C.[4] He moved onto an adjacent part before completing the part already begun.

Drawings made by non-savant children with autism have been reported to show similar levels of visual realism as those produced by mental age-matched children, with some qualitative differences to the topics drawn and the representational drawing process. Individuals with autism lack 'global processing' that is they have 'weak central coherence',[5] Some autistic savants exhibit drawing skills considerably advanced for their mental age.[6] One explanation of such phenomena is that they are impaired in their ability to conceptualize the world and thus it benefits their drawings. In contrast, the findings here, suggests that A.W.'s drawings were prone to an influence of meaning and were guided by genuine conceptualizations. For instance, A.W. showed omission errors for the teapot's pattern than mug's stripes as the teapot's representation was complex consisting of the handle, spout and lid as compared the mug's representation which was simple consisting of the handle of the mug.

In summary, A.W.'s drawings' revealed the existence of visual realism in children with autism. The findings have implications for improving our understanding of the capacity of autistic children for conceptualization as well as providing some important insights into their cognitive underpinnings through their drawings.

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Case Report

Glossodynia Responded to Opipramol

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Introduction

Glossodynia is a painful or burning sensation in tongue. It is also known as glossalgia, burning tongue, glossopyrosis, stomatopyrosis, sore tongue, burning mouth syndrome, burning mouth, orodynia or stomodynia.[1] It is characterized by burning pain or hot sensation which can be localized to lips or widespread in the mouth. It can be continuous or intermittent and may be associated with dryness, unpleasant taste or feelings of numbness, fatigue, altered sleep or depression.[2,3] It can increase with talking, stress or eating hot or spicy foods. It can decrease by sleep, rest, some foods or drinks and distraction. It affects 1 to 15 % population and is more common in women, especially in menopause group. Recent studies suggest that there is change in the way tongue transmits sensation to brain resulting in discomfort, pain or burning.[2,3] In ICD-9-CM, it is coded under K529.6[4] and in ICD-10-CM (forthcoming from October 2014), it will be coded as 14.6. The patient described developed persistent burning sensation over lips and tongue and responded to a new drug, Opipramol.

Case Report

We describe a case of 42-year-old housewife. She was living with husband and two children in urban area. She presented with a two months history of feeling of a burning sensation over lips and tongue. It was also associated with dull aching pain on eating or drinking hot things. This started about two months back and was intermittent. It had increased in severity over the period and had become continuous. She tried different analgesics, toothpaste and mouth washes but without any relief. Due to symptom, she had developed anxiety, sad mood and sleeplessness and was unable to do her household activities regularly. There was only one obvious stressor that her husband, a bank officer, had been transferred outside Delhi about three months back. There was no past or family history of psychiatric disorder, drug abuse, chronic physical illness or any allergy. There was no history of dental or surgical intervention. Dental and otorhino-laryngeal examination and laboratory investigations (including blood sugar, serum iron, vitamin $B_{12,}$ and folic acid levels) did not reveal any abnormality.

Detailed systemic examination including neurological examination and relevant hematological and radiological investigations including CT scan (head) did not reveal any abnormality. There no past history of any chronic psychiatric disorder, physical disease, or drug abuse. Family history was nothing contributory. Mental state examination revealed a middle-aged lady of endomorphic build. Psychomotor activity and speech were normal. There was no perceptual abnormality. Thinking revealed the preoccupation with complaint. Higher mental functions were normal.

She was psycho-educated about the problem and was convinced that it requires treatment with systemic psychotropic drugs. The patient was started on tablet gabapentin 300 mg/day and tab zolpidem 10 mg at night, if required. There was no improvement in 4 weeks. She was then started on tablet Opipramol 150 mg/day in divided doses. Tablet gabapentin were gradually withdrawn. The dose of Opipramol was gradually increased to 200 mg/day in four weeks. There was improvement in the burning sensation and complete remission in eight weeks. Her depressive and anxiety symptoms and sleeplessness also improved in four weeks and remitted in six weeks. On following her up at four months, she did not develop the symptom again.

Discussion

Glossodynia has been reported in local diseases (e.g. candidiasis, herpes, lichen planus) and systemic conditions (e.g. Diabetes mellitus, hypothyroidism), neuropathy (injury to chorda tympani), iron and vitamin B deficiencies, drugs (protease inhibitors, captopril), allergy, arsenic and mercury poisoning.[1,5-9] Opipramol is an antidepressant and is anxiolytic[2,7-10] and has been used in pain[11] and somatoform disorder[12] as in the present case. Menopause has been associated with glossodynia[1,2,5] but in the present case there were no menstrual disturbance. Some patients improve with the use of gabapentin[2,3] but in the present case there was no response. Opipramol is a sigma one receptor agonist (which has antidepressant and anxiolytic effect) and has antihistaminic and muscarinic acetylcholine antagonistic properties.[8,9,11] It improves quality of sleep.[12] Yoga, meditation and relaxation exercises have been found useful,[2,3,7] they were not used in the present case.

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Case Report

Amitriptyline Induced Hyponatremia

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Introduction

Hyponatremia is a recognised side effect of all classes of antidepressants. Studies have shown that different classes of antidepressants have different levels of risk of producing hyponatremia.[1,2] Selective serotonin reuptake inhibitors (SSRIs) have been shown to be more frequently associated with hyponatraemia than other antidepressants.[3,4] Among the SSRIs, paroxetine,[5,6] citalopram,[7] fluoxetine[8] and sertraline[9] are commonly reported to carry a high risk of hyponatraemia. Among other classes of antidepressants venlafaxine,[10] mirtazapine[11] are also associated with hyponatremia. Tricyclics are associated with risk of hyponatraemia less commonly as compared with other class of antidepressants. Antidepressant induced hyponatremia commonly occurs in old age people. Amitriptyline induced hyponatremia is rarely reported[12] and its occurrence at young age is itself more rare. We are reporting a case of amitriptyline induced hyponatremia in a 28 years old patient.

Case history

Mr. A., 28 year old male presented to the psychiatry OPD with complaints of lethargy, fatigue, loss of appetite, muscle cramps for last 4 days. He was hospitalized for proper evaluation and management. On physical examination he had a pulse rate of 84/min and BP of 114/78 mm of Hg. Examination of the abdomen and the respiratory and cardiovascular systems were within normal limits. CNS examination revealed no focal neurological deficit. There was no history of fever, headache, seizures or focal neurological deficit. Mental status examination revealed a conscious, cooperative patient with a depressed mood, anhedonia, impaired concentration, reduced self-confidence. Hb%, total leucocyte count, differential count, blood glucose level, and blood urea and creatinine were within the normal range. Her serum sodium was 108 mEq/l. Other electrolytes were within normal limits. Treatment history revealed he was taking tablet amitriptyline 50 mg/ day from last 10 days, prescribed by a local physician. On the basis of history and examination a diagnosis of moderate depressive episode (F32.1) with amitriptyline induced hyponatremia was established as per International Classification of Diseases-10th Edition criteria (ICD-10)[13]. He was managed by fluid restriction and 3% normal saline and the amitriptyline was withdrawn. The patient began showing improvement after 48 hours. Serial serum sodium measurements were done which were as follows: 112 mEq/l on day 2, 124 mEq/l on day 4 and 136 mEq/l on day 6. The patient was started on tablet mirtazapine 15 mg/ day and discharged from the hospital. On his first follow up visit after 1 month his depressive features improved significantly and no other symptoms were reported.

Discussion

In our patient strong temporal association between amitriptyline use and hyponatremia, a lack of an alternative explanation, and reversal of the process with discontinuation of the drug, suggests a causal relation between amitriptyline and hyponatremia. Amitriptyline-induced hyponatraemia, although relatively uncommon, is an important clinical problem with a serious outcome. Hyponatremia occurring as an adverse effect of an antidepressant is under-diagnosed because most of the cases are asymptomatic or patients may present with fatigue, weakness, and apathy, which may simulate worsening of depression. Mirtazapine might be

considered as an alternative treatment option of patients with depression who develop amitriptyline or SSRI induced hyponatremia[14]. Most cases of antidepressant-induced hyponatremia are reported in elderly people, which could be related to altered antidiuretic hormone regulation or action of the antidiuretic hormone on the kidneys[15]. But in this case hyponatremia is observed in a young man. So clinicians should be alert for antidepressant -induced hyponatremia, not only in the elderly patient, but also in the younger patients, particularly if the onset of symptoms has a close temporal association with the start of antidepressant treatment.

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Case Report

Sheehan's Syndrome with Psychosis: A Rare Case Presentation

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Introduction

Sheehan's syndrome is a state of postpartum hypopituitarism caused due to pituitary necrosis in cases of postpartum hemorrhage, secondary to excessive blood loss. It was first described in 1937 by H L Sheehan.[1] It is an not a rare cause of hypopituitarism in developed countries and Abs et al in his demographic study describes it as sixth most frequent cause of growth hormone deficiency, responsible for 3.1% cases.[2] In India Zargar et al estimated the prevalence of 3%, in women above 20 years of age.[3]

It can present in the postpartum period or after many months or years following the delivery. Gei Guardia et al estimated average time between the delivery and diagnosis of this syndrome to be 13 years.[4]

Most common initial symptoms of Sheehan's syndrome are agalactorrhea and/or amenorrhea. Endocrinologic manifestations of hypopituitarism reveal the deficiencies of specific hormones secreted from pituitary gland including hypoadrencortico-tropinemia, hypothyroidism and hypogonadism. Deficiency in corticotropin is characterized by a decrease in adrenal androgens and production of cortisol. Acute loss of adrenal function is a medical emergency and may lead to hypotension and death if not treated. Signs and symptoms of corticotropin deficiency include myalgias, arthralgias, fatigue, headache, weight loss, anorexia, nausea, vomiting, abdominal pain, altered mentation or altered consciousness, dry wrinkled skin, decreased axillary and pubic hair, anemia of chronic disease, and impaired gluconeogenesis. Gonadotropin deficiency cause amenorrhea, hot flushes and decreased libido. Growth hormone deficiency causes many vague symptoms including fatigue and decreased muscle mass and hypothyroidism causes tiredness, intolerance to cold, constipation, weight gain, hair loss, a slowed heart rate and thinking.[5, 6,7]

Haemotological abnormalities are commonly found and include normocytic normochromic anaemia, pancytopenia, and acquired factor VIII and von Willebrand factor deficiency.[8]

Factors proposed in the pathogenesis of Sheehan's syndrome are abnormalities of hypophyseal arteries (external compression, vascular spasm and thrombosis), autoimmunity, enlarged pituitary gland and small sella.[9]

Case Presentation

Case History

A 35 years, Hindu, married female, housewife by occupation, referred from obstetrics and gynecology department with complaints of irrelevant talk, suspiciousness towards family members, becoming abusive towards them, talking to self and aggressive behavior. She would repeatedly try to escape from home without any reason and would not do any household work or take care of children. She would not sleep and eat properly and neglected her personal hygiene.

Patient had full term vaginal delivery eleven years back at home with history of retained placenta and postpartum hemorrhage followed by failure to lactate after delivery. Gradually she had developed fatigue, lethargy, joint pains, anorexia, swelling on whole body and weight gain. She started having genitals and axillaries hair loss and developed hirsutism. She continued having amenorrhea since delivered. There was no past or family history of psychiatric illness and no history of substance abuse.

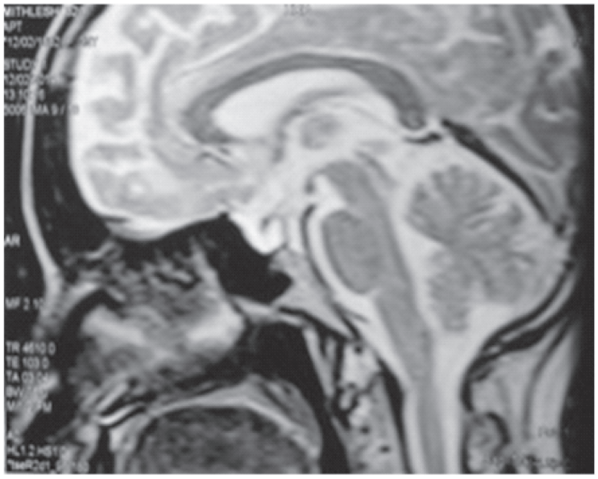
Examination

On examination patient was conscious, oriented. She was afebrile and her vital signs were normal. On general physical examination, she had anaemia, excessive facial hair, atrophied breasts and pedal edema. On neurological examination no other abnormality was found.

On mental status examination patient was unkempt and uncooperative. Her thought process revealed ideas of persecution and reference. Her attention and concentration were impaired and she had poor insight and judgment.

Lab Investigations and Neuroimaging

She was thoroughly investigated and her serial investigations were done in follow up which are presented in table 1.

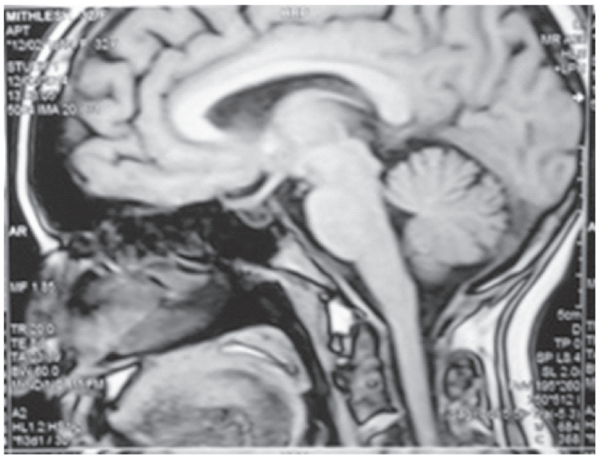


gland. Two years later Non Contrast T1 weighted magnetic resonance images show marked dimunition in size of pituitary gland and cerebrospinal fluid (CSF) signal intensity in the sella (empty sella).

Table 1 : Lab Investigations

Test	2/12/2010	26/7/2011	21/11/2012	Reference Range
T3(ng/dl)	25	135	0.43	60 - 181
T4 (ug/dl)	1.2	10.9	4.21	4.5 – 10.9
TSH(uIU/ml)	0.55	0.30	0.50	0.35- 5.50
Prolactin (ng/ml)	23.1	16.7	4.1	1.4- 24.3
LH(mIU/ml)	0.5	8.8	7.8	1.5 – 95.5
FSH(mIU/ml)	0.3	25.2	33.7	0.8-102.8
Haemoglobin(gm/dl)	6.5	8.0	11.4	12 - 16
Total leucocyte count(x 10^9/l)	5.6	7.6	7.8	3.8 – 11.0
Platelet count (x 10^9/l)	124	291	321	150 - 440
Urea(mg/dl)	34	14	13	20 - 40
Creatinine(mg/dl)	1.0	0.7	0.6	1-2
Total bilirubin(mg/dl)	0.5	0.3	0.3	<1
Total cholesterol(mg/dl)	240	210	190	200-239
Triglycerides(mg/dl)	321	312	240	40 - 200

Her initial Gadolonium enhanced T1 weighted Magnetic resonance imaging show central low signal intensity and peripheral enhancement in the pituitary



Management and Follow up

The patient was managed on steroid and thyroxine replacement therapy. She was started on 20 mg of prednisolone and 100 microgram of thyroxine daily. For psychotic symptoms she was put on amisulpride 200 mg daily. Her symptoms improved gradually and she became euthyroid in follow up and there has been no further episode of psychosis. Her medications were slowly reduced over years and now she is maintaining well on 100 mg of amisulpride and 75 microgram of thyroxine.

Discussion

This is case of Sheehan's syndrome presenting with psychosis which is a rare presentation. Her

symptoms of hormone deficiencies developed postpartum with history of significant obstetric complication indicating ischemic necrosis of pituitary. She had symptoms of hypothyroidism, cortisol and gonadotropin deficiency. Her laboratory investigations and MRI findings strongly support the diagnosis of pituitary dysfunction. She recovered with hormone replacement therapy and maintaining well on follow ups.

Sheehan's syndrome causes hypoadrenalism, which causes a decrease in cortisol. The cortisol decrease can then induce immunological alterations which can be responsible for thyroiditis.

Glucocorticoids are the main endogenous anti-inflammatory agents in vivo, interfering with every step of immune and inflammatory responses, and are commonly used in the treatment of autoimmune diseases.[10,11]

This case emphasizes the importance of clinical suspicion of Sheehan's syndrome and endocrionological work up in patients presenting with psychotic symptoms postpartum with significant history of obstetric complications.

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Case Report

Catatonia as first presentation of Chickenpox

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Introduction

It is not uncommon to find Catatonia in the Indian subcontinent as compared to the West[1]. Patients who frequently present with catatonic signs and symptoms are diagnosed with an underlying organic or psychiatric illness which usually resolves with treatment. Seldom does one come across a viral aetiology as a cause of catatonia. As far as the review show there are no reports of chicken pox presenting as catatonia and in our knowledge this is the first case of chicken pox presenting as catatonia. The case highlights the clinical presentation and management difficulties in an adolescent with catatonia diagnosed with chicken pox.

Case study

Mr. X a 15 year old adolescent male was brought to this tertiary care centre with complaints of fever, mutism, decreased oral intake and withdrawn behavior for past 2 weeks and rash on body for 1 week. Examination had an alert oriented male with pustular rash on face and trunk with crust, pyrexia, mutism, active negativitism, posturing and ambitendecy. He also had waxy flexibility, stereotypy and automatic obedience. There was no history of any substance use or ingestion of any medication in recent past. Rest past, family, and personal history was unremarkable. He was admitted in Child and Adolescent Ward and investigations were sent. He was diagnosed as a case of catatonia with chickenpox in consultation with a dermatologist. All blood serum parameters including complete blood count, liver, kidney and thyroid function tests, serum electrolytes, blood sugar, urine routine and microscopy and brain neuro-imaging came out to be within normal limits. An EEG was done which did not reveal any abnormality. A lumbar puncture was performed and CSF was sent for cytological study and chemistry which was unremarkable.

He was started on intravenous lorazepam 8 mg/day for catatonic signs and was managed conservatively for chickenpox rash which cleared within 2 weeks. Patient was monitored on Bush Francis Catatonia Rating Scale but patient did not show any significant progress while on lorazepam up to 16 mg/day over a period of 72 hours. He was subsequently started on Modified ECTs on alternate day basis. He received a total of 22 MECTs over a period of 2 months. Patient showed gradual improvement after initiation of MECTs and the symptoms resolved within a period of 2 months. After catatonia improved, the patient was interviewed and found to have psychotic symptoms of delusion of persecution and control. Patient was started on tab olanzapine and discharged. On follow up, he is maintaining well on 10 mg olanzapine / day.

Discussion

Catatonia is a common manifestation of a multitude of etiologies. There are many case reports[2] of CNS infections presenting as catatonia but extensive literature search had only 1 case report of chicken pox with catatonic sign catalepsy[3]. However no case of catatonia has been reported as a sequel of chicken pox per se. Pfister et al[4] in 1993 reported a case presenting with an acute encephalopathy manifesting with catatonia. They were able to isolate Borrelia burdorferi from CSF and patient recovered after a course of intravenous ceftriaxone therapy. Sherese et al[5] also described two cases of encephalitis and catatonia where encephalitis was complicated by catatonic features and showed a good response to use of MECT. They also concluded that intravenous lorazepam may not suffice in cases of catatonia nested in

encephalopathy. Shukla et al[6] also described a case of 20-year-old woman presenting with an acute illness consisting of fever, delirium, perceptual abnormalities, and catatonic state. After trials with antiviral medications, benzodiazepines, and atypical antipsychotic medications, she was treated with 6 sessions of electroconvulsive therapy with complete recovery and no complications. As per the available literature catatonia has been seen in various CNS infections which have included Herpes Simplex Virus, typhoid fever, HIV West Nile River virus etc.

Historically, Catatonia like features were also described in cases of encephalitis lethargica in past and even in recent times[7,8] although no viral agent was ever isolated from these cases.

The above mentioned case is first to our knowledge as no previous record of a case of chicken pox presenting as catatonia has been reported. Also, as per the available review it is well known that secondary catatonia responds slowly and poorly to benzodiazepine treatment. This fact was another similarity we could find with respect to secondary catatonia.

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Case Report

Naltrexone Induced Panic Attacks

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Introduction

Alcohol dependence has been conceptualized as a chronic relapsing medicalillness with relapses and remissions and a strong genetic component similar to diabetes type II and hypertension.[1] Risk for relapse isheightened because the neurobiological changes in brain pathways created by many years of alcohol use do not completely revert to normal after the detoxification process.[1] Chronic dependence on alcohol can result in periods of severe withdrawal syndromes marked by increased heart rate and blood pressure, anxiety, and withdrawal seizures and in severe cases delirium tremens and even death.[2] Medications for alcohol withdrawal syndromes include benzodiazepines that act on gamma-aminobutyric acid (GABA) at the GABA (A) receptors in the brain to stimulate release of GABA. GABA is a neurotransmitter that is responsible for decreasing activity throughout the nervous system and acts to gradually detoxify the patient from alcohol by reducing heart rate, blood pressure, sweating, and anxiety associated with alcohol withdrawal. Benzodiazepines can also improve treatment outcome but should only be used on a short-term basis. They should be avoided as a long-term strategy for controlling alcohol dependence because physical tolerance of these medications can occur rapidly and can result in dangerous interactions if patients using the medication relapse into alcohol use.[3]

Naltrexone is a good example of an anticraving medication for the long-term treatment of alcohol dependence. Naltrexone is a competitive opioid antagonist that presumably blocks the rewarding aspects of drinking by occupying opioid receptors. When Naltrexone is present in the brain, alcohol cannot stimulate the release of dopamine, thereby, reducing the intoxicating effect of alcohol. Naltrexone has been shown to reduce the frequency and intensity of drinking, to reduce the risk of relapse to heavy drinking, and to increase the percentage of days abstinent.[4] The majority of published controlled studies of Naltrexone show improved efficacy compared to placebo. However, one Veterans Administration study of Naltrexone among patients with more severe alcohol dependence showed no benefits from it.[5]

Though Naltrexone is well tolerated and is considered a good anticraving agent, this case is being reported to highlight the intolerable panic attacks due to Naltrexone.

The case

Mr. M, a 35 year old average built male educated up to M.B.A. working as executive finance officer in a multinational company, living in nuclear family of upper-middle socio-economic status, urban background was brought to adult psychiatry OPD, Department of Psychiatry, PGIMER, Dr R M L Hospital, New Delhi by his wife with total duration of alcohol intake of ten years.

Exploration of history revealed that the patient was introduced to alcohol by his college friends in 2004 at 25 years of age. He also wanted to have that experience and enjoyed it. Occasionally he used to take beer approximating to one bottle in his friend's company at about two to three months interval. Frequency of alcohol intake increased over a period of six months to about, taking one bottle of beer in one month. There was gradual increase in both the frequency and amount of alcohol over a period of next few years. Over period of two years i.e. in 2007, he used to take about two–three pegs of whisky as he did not feel the same effect as he

used to feel with one peg and he used to take at weekly interval as he felt strong desire to take it. On visiting any social gathering, he was keen on taking alcohol and used to search for it as soon as he reaches there. His consumption amount increased to about one quarter over three months and half bottle over six months. Since 2008, he started taking about one bottle of whisky nearly every day. Gradually, over a period of 10 years his tolerance to alcohol increased. He was taking approximately one bottle per day from last 5 years. He reported of intense desire to take alcohol in evening hours and occasional morning drinking started at the same time on holidays. There was history of multiple episodes of blackouts also present.

Over a period of time, his compulsion to take alcohol increased and he was not able to control his alcohol taking behavior. He started spending more time in activities to obtain alcohol. He stopped involving in pleasurable activities. He started avoiding family gatherings and would prefer taking alcohol alone. There was significant impairment in his office work due to frequent absenteeism. He was repeatedly being questioned in his office for his poor performance. He also stopped visiting his friends. There was significant decline in his social, occupational and personal life due to his alcohol taking behavior.

He was brought by his wife for alcohol de addiction. He tried to leave alcohol four days back but reported complaints of having disturbed sleep, restlessness, uneasiness, nausea, headache and tremors for about three-four days but he did not took any treatment for the same. He tried to quit alcohol nearly six months back, but he went to some marriage and took one peg of whisky. He resumed his pattern of alcohol intake in one-two months and was unable to control it. But his alcohol intake started to affect his family with repeated quarrels with his wife and frequent absenteeism from his work. Because of these repeated problems, he decided to quit alcohol and came along with his wife for seeking help.

No history of another substance use was reported. Past history, family history and personal history were noncontributory. Premorbid personality revealed impulsivity and frequent anger burst in the past. There have been significant relational problems in the family. Patient had very few social relations.

Mental status examination revealed normal psychomotor activity, average personal hygiene. Patient was conscious of himself and his surroundings and he was oriented to time, place, and person. His attention and concentration was aroused and sustained. His affect was euthymic throughout the interview. His thinking showed concern for his job and marital life and stage of change was contemplation and no perceptual abnormality could be detected. Patient's motivation was low.

His immediate, recent and remote memory was intact. Patient had average intelligence level as his general fund of knowledge was adequate and had average arithmetic ability. His abstract thinking was impaired. His judgment was intact. Patient had insight about his illness.

His complete blood count and renal function test was within normal limits. Liver function test was slightly deranged with level of SGOT and SGPT approximately three times the normal values. Ultrasonography of whole abdomen revealed stage one fatty change.

CIWA –Ar was used for assessment of withdrawal symptoms and his score was 14. He was in moderate withdrawal. Patient was detoxified with fixed dosing regimen of benzodiazepines over ten days with regular motivation enhancement therapy. After detoxification, with mutual discussion with patient, tablet Naltrexone 50 mg OD was started. On next day, patient reported of two episodes of severe panic attacks and Naltrexone was withheld and complete investigations were repeated with cardiac profile which came out to be within normal limits. Other medical causes for anxiety were ruled out as all repeat investigations were within normal limit. So Naltrexone was restarted and patient again developed panic attacks of nearly 5 minutes duration of about 5-6 episodes per day for next two –three days. Since patient was not able to tolerate Naltrexone, it was finally stopped and his panic attacks did not occurred further. Finally, patient was prescribed Acamprosate at appropriate doses. Patient did not report any panic attacks after the medication change. Patient was discharged after a hospitalization period of two weeks. Patient came for regular follow up for next 6 months and was abstinent throughout and did not report of any panic attacks.

Discussion

The average dose of Naltrexone is 50 mg daily. Naltrexone is usually well tolerated, and the most frequent side effects are mild nausea and headache. Though there are reports of mild anxiety associated with Naltrexone, there is no case report till now indicating needs to change the medicine due to panic attacks. In the indexed case, patient was not able to tolerated Naltrexone despite complete detoxification and absence of any medical illness, although there was no intolerance to other medicines. Though exact mechanism could not be explained for the precipitated panic attack but frequent interaction of opioid receptors and GABA receptors might be responsible, as panic attacks are mainly due to altered neurotransmitter response at GABA.[1] Though these side effects are rare, but presence of such side effects could lead to relapse in case of alcohol dependence.

It has been evident in one of the study with Naltrexone among patients with more severe alcohol dependence, which showed no benefits from it.[5] A major issue with Naltrexone in outpatient long-term treatment settings is the variability of adherence to it. But none of the studies have mentioned the reason for non adherence. Lack of compliance with the medication is strongly correlated with higher relapse rates.[6-8] Reason for noncompliance due to Naltrexone is still not explained in any of the studies.

The main reason for reporting the indexed case is to highlight the side effect related non compliance of Naltrexone, which can result in relapse of alcohol dependence. Thus, while managing a patient of alcohol dependence, Naltrexone related side effects should be kept in mind and these should be managed properly to prevent relapse.

Conclusion

Addiction is a chronic disorder that requires a long-term approach. In recent years, pharmacotherapies have been developed and now play an important role in the treatment of alcohol dependence at the levels of detoxification, initial recovery, and relapse prevention. Medication sadd to the benefits of psychosocial interventions and work synergistically in combination with behavioral therapies. Proper knowledge of side effect profile of anticraving agents will help in better management of alcohol dependence.

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Letter to Editor

Somatic delusional disorder at 106 years of age

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Persistent delusional disorder is characterized by the development of either a single delusion or a set of related delusions that are not bizarre and cannot be classified as organic, schizophrenic or affective. Individuals affected with somatic-type delusional disorder typically complain of infestation, deformity, personal ugliness, exaggerated sizes of body parts, foul body odour or halitosis. Delusional disorder is generally regarded as a disease starting in middle to late adult life.[1] The mean age of onset is 40 years and ranges from 18-90 years.[2] As per ICD-10 and DSM-IV-TR criteria, there is no consideration for age in establishing the diagnosis of delusional disorder.[3,4]

The emergence of psychotic symptoms for the first time in later life poses a diagnostic challenge for clinicians assessing and treating elderly persons with mental illness. In these situations clinicians are frequently confronted with comorbidity issues relating to cognitive deficits, affective symptomatology and physical illness. Most commonly, late- onset delusions and hallucinations are associated with dementing and affective disorders; however, in a small but substantial number of cases these symptoms arise de novo. Whether schizophrenia or delusional disorder can be diagnosed in these situations has been the subject of considerable debate. We present a case of persistent delusional disorder of somatic variant with onset at the age of 106 years.

Mrs. A., 106 -year-old Hindu married female presented to psychiatry outpatient department (OPD) with complaints of mud coming out of her both ears and spreading over her cheeks for last 6 months. Along with these symptoms there was decreased sleep. There had been no complaint of forgetfulness. There were no symptoms to suggest a mood disorder or delirium. She had no significant medical, surgical, or psychiatric illness in the past or any significant family history. She had no history of substance abuse . On mental status examination the patient was conscious, orientated , denied auditory or visual hallucinations or other types of delusions. She had unshakable belief that mud is coming from her both ears and spreading over her both cheeks. She had tactile hallucination of feeling mud over her both cheeks. She had good immediate, recent, and remote memory and had a good fund of general information and could correctly perform simple verbal calculations. She had no insight into her psychiatric illness.

Her hemogram, renal, hepatic and thyroid function tests, lipid profile, serum electrolytes, and routine urinary examination were within normal range. Her chest roentgenogram and electrocardiogram were normal, magnetic resonance imaging (MRI) of brain revealed generalized age-related cerebral atrophy and electroencephalogram (EEG) was a normal awake record. On Mini-Mental State Examination (MMSE), she scored 27/30, with points lost for not reading, writing, and copying the interlocking pentagons (as she was uneducated).

On the basis of history and examination a diagnosis of persistent delusional disorder (somatic variant) was established according to International Classification of Diseases-10th Edition criteria.[3] She was prescribedtablet risperidone 1mg per day which was titrated to 3mg per day along with tablet trihexyphenidyl 2 mg per day. On her next follow up visit after 1 month she had improved significantly and she was continued on the same medications.

In this patient, the diagnosis of dementia and delirium were excluded as there was no cognitive deterioration and she was alert and oriented to time, place, and person. She had no delusion which was diagnostic of schizophrenia and also she had no persistent hallucination in any modality except tactile hallucination which is related to the delusional theme.

Possibility of psychotic disorder due to a general medical condition can be ruled out as there was no evidence of medical conditions known to cause psychiatric illness from history, physical examination, and laboratory results. As there were no predominant affective symptoms at any point of time, diagnosis of mood disorder with psychotic features can also be ruled out.

Few cases of schizophrenia have been reported earlier with onset at an age of 100 years or more.[5,6] But no case of delusional disorder has been reported at an age of 100 years or more. So our case will encourage the treating psychiatrists to diagnose delusional disorder at extreme old age.

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Book Review

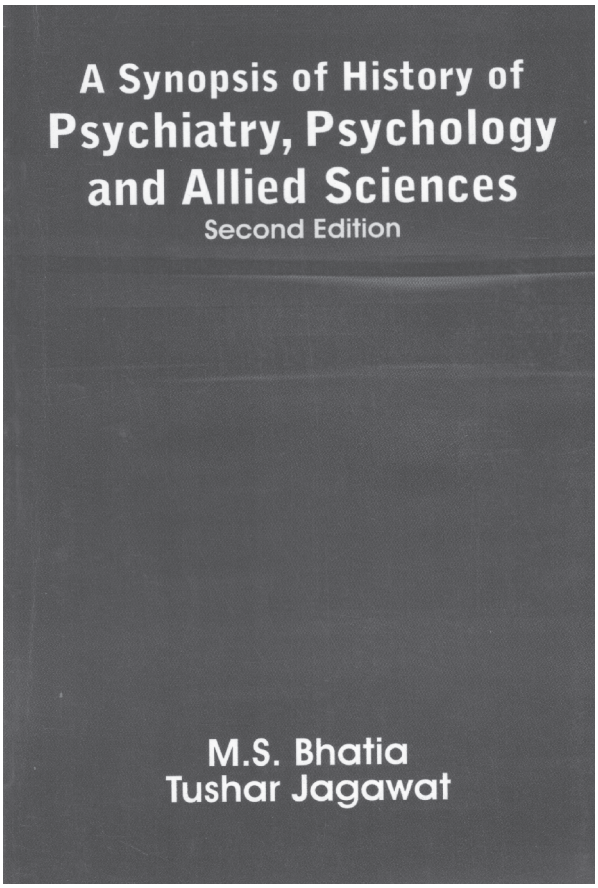
A Synopsis of History of Psychiatry, Psychology and Allied Sciences

Second Edition
Author: M.S. Bhatia, Tushar Jagawat
Publisher: CBS Publishers and Distributors Pvt. Ltd.
174 pages ISBN: 978-81-239-2239-3

The field of psychiatry, psychology and allied sciences related to mental health has undergone a sea change in the past few decades. The changes happening can be compared to an avalanche with each passing decade adding exponential information to an already existing knowledge base. From Freud's Influential theories of psychoanalysis to current emphasis on neuro-biological and genetic basis of psychiatric disorders, the rapid advancements make it extremely difficult for a student of the field of mental health to keep pace with them. This is where this book fills the felt gap.

This book is broadly divided into two sections. Section I is on history of World Psychiatry. It is further divided into 7 sub-sections dealing with specific areas like terms/concepts related to various psychiatric disorders, important figures in mental health, various psycho-social theories/therapies and their proponents, world famous personalities and their psychopathology among others. Section II deals with the history of Indian psychiatry which is further divided into 9 subsections dealing with important areas like listing of Institutes catering to the needs of people with mental illness, important Indian books on mental health, famous personalities in Indian Psychiatry and laws relating to Indian Psychiatry among others.

The authors' have made a laudable effort in compiling such a vast amount of information in such a concise manner and still have managed to keep the book to a reasonable size. As rightly pointed by Dr. (Col) Kirpal Singh in his foreword to this book "this book is functionally a dictionary except that the layout is not alphabetically arranged." Only the authors of the stature and experience of Dr. M.



S. Bhatia and Dr. Tushar Jagawat could have made this compilation possible. This book will be a valuable addition to the collection of every working person in the field of mental health.

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Interesting Articles

- Garety PA, Freeman D. The past and future of delusions research: from the inexplicable to the treatable. BJP November 2013; 203 : 327-333.
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Fourthcoming Events in Psychiatry

INTERNATIONAL

3rd April 2014, Medicine for Psychiatrists Conference, Auckland, New Zealand

4th April 2014, Spring Global Symposium for Psychology Professionals, Los Angeles, United States of America

4th April 2014, Psychology and Psychiatry Conference, Bangkok, Thailand

8th April 2014, 16th International Neuroscience Winter Conference, Sölden, Austria

10th April 2014, 4th International Music Therapy Students' Congress titled "Music therapy - BRINGING HOPE " Wroc? aw, Poland

17th April 2014, The 10th International Conference on Psychiatry "Psychiatric Models; Biological and Psychological perspectives", Jeddah, Saudi Arabia

24th April 2014, 8th Annual Risk and Recovery Forensic Conference, Hamilton, Canada

28th April 2014, International Society for Affective Disorders Congress, Berlin, Germany

1st May 2014, Maudsley Masterclass 2014: "Towards Recovery", London, United Kingdom

2nd May 2014, 12th Global Conference: Violence, Lisbon, Portugal

7th May 2014, EFCAP Congress 2014, Manchester, United Kingdom

10th May 2014, 5th Global Conference: Storytelling: Global Reflections on Narrative, Lisbon, Portugal

13th May 2014, Recent Advances in Neuropsychiatric, Psychological and Social Sciences, Athens, Greece

14th May 2014, Annual International Conference Cognitive - Social, and Behavioural Sciences (icCSBs 2014), Mersin, Turkey

21st May 2014, 17th EPA Section Epidemiology and Social Psychiatry Meeting - "Disease Burden and Service Delivery", Ulm/Neu-Ulm, Germany

6th June 2014, Cognitive Remediation in Psychiatry, New York, United States of America

12th June 2014, 3rd International Symposium on Controversies in Psychiatry :: Combined Treatments, Mexico City, Mexico

24th June 2014, International Congress of the Royal College of Psychiatrists, London, United Kingdom

25th June 2014, EXTENDED DEADLINE: Cognition and Action: The Jagiellonian-Rutgers Conference in Cognitive Science 2014 (CogSciJR14), Krakow, Poland

4th August 2014, 3rd International Conference and Exhibition on Addiction Research & Therapy, Chicago, United States of America

4th August 2014, 3rd International Conference and Exhibition on Addiction Research & Therapy, Chicago, United States of America

10th September 2014, 3rd European Conference on Mental Health, Tallinn, Estonia

11th September 2014, Memory: Forgetting and Creating, Gdansk, Poland

16th September 2014, 6th World Congress on Mental Health and Deafness, Belfast, United Kingdom

24th September 2014, 2nd Global Conference: Suicide, Self-harm and Assisted Dying, Oxford, United Kingdom

10th October 2014, 2014 FALL GLOBAL PSYCHOLOGY SYMPOSIUM, Los Angeles, United States of America

30th October 2014, WPA Thematic Conference on Intersectional Collaboration, 5th European Congress of INA & 2nd Interdisciplinary Congress on Psychiatry and Related Sciences, Athens, Greece

1st December 2014, CPSYC 2014 International Congress on Clinical and Counselling Psychology Antalya, Turkey

10th December 2014, 10th International Congress on Mental Dysfunction and Non-Motor Features of Parkinson's Disease & Related Disorders, Nice, France

12th December 2014, WPA Regional Congress, Hong Kong, Hong Kong, China

17th July 2015, 2015 Summer Global Nursing Symposium, Los Angeles, United States of America

16th July 2015, 22nd International Association for Child & Adolescent Psychiatry and Allied Professions, Calgary, Canada

Other useful links Websites: 1.

http://www.psychnet-uk.com/x_new_site/events_conferences/events.html

2. http://www.conference-service.com/conferences/psychiatry.html

3. http://www.conferencealerts.com/topic-listing?topic=Psychology

4. http://www.medical.theconferencewebsite.com/conferences/psychiatry

5. file:///C:/Users/dr.%20bhatia/Downloads/2014-professional-meetings-and-conferences.pdf

INDIAN EVENTS

ANCIPS 2015 – Hyderabad (contact: G. Prasad Rao)

IPS-NZ Midterm CME – Government Medical College Chandigarh, 11th May 2014

IPS-NZ Annual Conference- SKIMS Srinagar, 1-2 Nov 2014

IASP- XX Annual Conference Mysore, 21-23 November 2014

SCARF India Conference on Schizophrenia, Chennai, 21-24 August, 2014

ERRATA TO OCTOBER 2013 ISSUE

1. Article – **'Head Banging – A Behavioral manifestation of birth asphyxia related extensive brain damage'** was authored by SujitKar, Shwetank Bansal, Akhila Kumar Panda

2. Article – **"Doctor Shopping In Psychiatry"** was authored by Supriya Vaish, Sachin Sharma, (Brig.) S. Sudarsanan, Sandeep Choudhary

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Sample citations

According to our previous work,[1,3-8,19]
The Patient's were studied as follows.[3,4]

Sample References

- **Articles**
  1. Roest AM, Zuidersma M, de Jonge P. Myocardial infarction and generalised anxiety disorder : 10-year follow up. Br J Psychiatry 2012; 200 : 324–329.
  2. Bremner JD, Shearer KD, McCaffery PJ. Retinoic acid and affective disorders: The evidence for an association. J Clin Psychiatry 2012; 73 : 37–50.
- **Book**
  1. Stahl SM. The Prescriber's Guide (Stahl's Essential Psychopharmacology, 4th ed. Cambridge, U.K.: Cambridge University Press, 2011.
- **Chapter of a book**
  1. Blacker D. Psychiatric Rating Scales In: Sadock BJ, Sadock VA, editors. Kaplan and Sadock's Comprehensive Text Book of Psychiatry. Vol. I. Philadelphia: Lippincott Williams and Williams; 2000. pp 755-782.

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DELHI PSYCHIATRY JOURNAL

(Official Publication of Delhi Psychiatric Society)

Indexed in Index Medicus for South East Asia (IMSEAR) & Indian Science Abstracts

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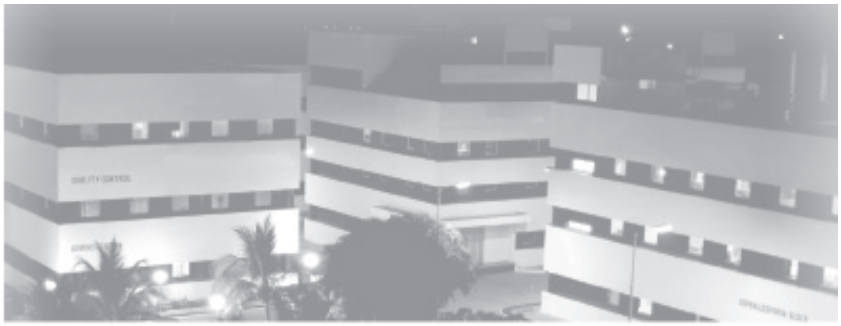

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