DELHI PSYCHIATRY JOURNAL

(Official Publication of Delhi Psychiatric Society)

Vol. 27 No. 1 April, 2024

Delhi Psychiatry Journal is Published by **Dr. M.S. Bhatia** on behalf of **Delhi Psychiatric Society (Regd.),** Hamdard Institute of Medical Sciences, Block-B, 1st Floor, Guru Ravidas Marg, New Delhi-110062 (India). **Layout Designing and Printing** by Rajan Nair, 5-C, Pocket B-7, Mayur Vihar Phase-III, Delhi-110096. **Correspondence to:** D-1, Naraina Vihar, New Delhi-110028 (India).

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Editorial

Navigating the Evidence: Pharmacological Management in Child Mental Health

Aparna Goyal,[1] M.S. Bhatia[2]
[1]Department of Psychiatry, IHBAS, Delhi-110095
[2]Department of Psychiatry, HIMSR & HAHC Hospital, Hamdard Nagar, Delhi-110062

Introduction

Mental health and its management has been a well debated topic over the years, where researchers have raised questions regarding the role and effectiveness of pharmacotherapy in treating mental illnesses. Over the years, sufficient evidence has been created for role of pharmacotherapy in mental illness dating since the time of Charak Samhita. However, the need for caution is more so in the situation where a young life is to be addressed. Over the last two decades, the use of psychotropics in youth has increased by 50% – 200% over the past 20 years, depending on the cohort.[1-3] United States of America reportedly being one of the leaders in high prevalence of use. Though there is an increase due to varying guidelines, perspectives, diagnostic criteria and health care systems, this is not uniform worldwide. It is unfortunate that the accessibility and acceptance of a biological psychiatric treatment model have contributed to poorly evidenced treatments becoming popular for pediatric mental health services. McLaren et al in its editorial expressed his concern not only at the alarming use of psychotropics in the youth but also the off label untested use of psychotropics in non-psychotic conditions.[4] Banaschewski et al. even challenged the notion of ADHD only being a biological construct and impressed upon the psychosocial role in the illness too.[5] A study by Comer et al reported an increase in co-prescription of antipsychotics and ADHD medications or antidepressants.[1] As per study by Olfson et al, national trends in antipsychotic prescription were compared between adults and youth and it was reported that even though medical visits were higher in adults, prescription rates of antispychotics were same or even higher in youth.[2] This brought on an urgent controversy between the pros and cons of the use of medicines in youth, its benefits, relevance, need, risks and interactions.

Medications - a boon

The debate on evidence-based pharmacological management in child mental health is a complex and multifaceted issue. Over the years, a debate over non-pharmacological management versus pharmacotherapy have been brought on in child mental health. On one side adequate scientific validation is available and the evidence-based pharmacological treatments are supported by rigorous scientific research and clinical trials. These studies demonstrate that certain medications can be effective in treating specific mental health disorders in children, such as ADHD, depression, and anxiety. These pharmacological treatments have shown significant improvement in symptoms, leading to better functioning in daily life, enhanced academic performance, and improved social interactions. Professional organizations, such as the American Academy of Pediatrics (AAP) and the National Institute for Health and Care Excellence (NICE), provide guidelines that recommend pharmacological treatments for certain conditions based on evidence. These guidelines are developed from extensive research and expert consensus. With more scientific advances and evolution of pharmacogenomics, it has become even more possible to provide personalized treatment approaches to all. It potentially increases the effectiveness and minimizing side effects by tailoring medications to individual genetic profiles. Pharmacological management can be part of a

comprehensive treatment plan that includes psychotherapy, behavioral interventions, and support for families, leading to better overall outcomes.

Medications - a bane

But with this integrated advanced personalized approach, lot of reservations have also been quoted. Children are particularly sensitive to medications, and long-term effects are not always well understood. Critics argue that the potential side effects, such as weight gain, sleep disturbances, and emotional blunting, may outweigh the benefits, especially if the medication is used long-term. There is concern that pharmacological treatments may be over-prescribed, with some practitioners relying too heavily on medication rather than exploring other treatment options like therapy, lifestyle changes, or environmental adjustments. Evidence suggests that while some medications are effective for some children, they may not work for others, and the response to medication can be unpredictable. This variability can lead to trial and error in prescribing, which may not always be in the best interest of the child. Although evidence supports the use of certain medications, it may be limited in scope. For example, there might be less robust evidence for the use of newer medications or for specific subgroups of children, leading to uncertainty about their effectiveness and safety. It is also reported that parents are seldom not aware of the risks and at times may not be involved in decision making specially in a patriarchal system of medicine. The Utah Psychotropic Oversight Program (UPOP) by Monson et al analyzed 8523 under 18 years children over four years and concluded that in the oversight program prescription rates decreased over time. Programs like UPOP can influence the prescribing practices without raising the need for higher levels of care.[6]

Indian Structure

India still lags behind in the arena of mental health care and well being, let alone targeting the youth. No doubt, public awareness and evolution is there in field of mental health, however, stigma, societal attitude, man power deficits, accessibility and availability of adequate resources amounts to poor understanding towards mental well being in a youth. India has also an enmeshed traditional systems including Ayurveda, Homeopathy, Unani, Sidha and to some extent faith healers apart from Allopathy, which makes it difficult to understand the prescribing patterns for a youth. Research needs to focus on such avenues so as to plan accordingly for the future of the country.

Recommendations

Possible solutions to this debacle can be

1. To weigh the risks and benefits not just short term but long term too.
2. Non-pharmacological or psychosocial interventions should be equally considered. A stepped care approach[7] in children can provide superior treatment to otherwise only medications.
3. Parents to be informed and involved in decision making and care
4. Polypharmacy and combination medications preferably be avoided.
5. Evidence-based guidelines and good research knowledge to be applied before prescribing and deprescribing

Conclusion

The debate around evidence-based pharmacological management in child mental health revolves around balancing the benefits of proven treatments with the potential risks and limitations. Advocates emphasize the importance of scientific evidence and the potential for improved quality of life, while critics highlight concerns about side effects, over-reliance on medications, and the need for more comprehensive evidence. Ultimately, the best approach often involves a personalized treatment plan that considers the specific needs and circumstances of each child, integrating both pharmacological and non-pharmacological strategies.

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Review Article

Psychopharmacological approach in Bipolar disorder during Pregnancy and Lactation

Sargun Arora, Ajeet Sidana
Department of Psychiatry, Government Medical College & Hospital (GMCH), Sector- 32, Chandigarh (UT)-160030
Contact: Ajeet Sidana, E-mail: ajeetsidana@hotmail.com

Introduction

Formerly known as 'manic-depressive' illness, bipolar disorder involves alternating episodes of depression and mania/hypomania, causing a change in person's mood, energy and ability to function. As per National Mental Health Survey (NMHS) 2016, the total weighted prevalence of bipolar affective disorder (BPAD) was 0.3% (95% confidence interval [CI]: 0.29–0.31) in the current (cross-sectional) period and 0.5% (95% CI: 0.49–0.51) in the lifetime.[1] In women, it presents with unique challenges and considerations as compared to men, especially during pregnancy and post-partum period. The degree of symptoms in women with bipolar illness may be correlated with changes in oestrogen levels, which could lead to increased exacerbations at the time of menopause and postpartum period.[2] Clinical knowledge, practical judgement, and a thorough grasp of the psychiatric problem are essential for the effective treatment of bipolar disorder during pregnancy.

Effect of pregnancy on bipolar disorder and vice-versa

Limited research exists on the impact of bipolar disorder on the progression of pregnancy, since retrospective studies are coloured by recall bias and prospective studies don't usually focus on the course but rather on the effect of medications during pregnancy and lactation. It was seen in one of the studies that frequency and recurrence of mood symptoms was lesser in pregnancy, than otherwise, in those with lithium responsive bipolar type 1disorder.[3] In a study conducted by Rusner et al. in 2015, bipolar disorder had shown to have a detrimental effect on the health of both mother and the infant, with adverse effects more likely to be in the form of gestational hypertension and antepartum haemorrhage. Also, higher rates were found for induction of labor and caesarean section along with elevated mood symptoms in the post-partum period.[4] In a systemic review and meta-analysis, done in 2016, it was found out that women with bipolar disorder have an overall risk of postpartum relapse at 37%, while those with a history of postpartum psychosis have a slightly lower risk at 31%.[5] The pathophysiology behind this is not completely understood and different researchers have come up with different hypothesis to explain the increased risk of relapse of bipolar affective disorder in post-partum period. As per some of the studies, post-pregnancy, there are problems in the hypothalamic-pituitary axis, and this coupled with sudden withdrawal of steroid hormones during this time, can be one of the reasons for exacerbation of mood symptoms, regardless of the diagnosis.[6] There is also an increased risk of post-partum bipolar depression. It differs from the other forms of depressive episodes in terms of some atypical depressive symptoms like diurnal mood variation, hypersomnia, mixed features, behavioural agitation, elevated mood in response to antidepressants and hypomanic symptoms during pregnancy and post-delivery. Since a lot of times, hypomanic symptoms are usually missed, unless specifically asked for, Bipolar type II disorder in post-partum period is often misdiagnosed as unipolar depression, leading to inadequate treatment and exacerbation of mood symptoms after initiation of antidepressants. This problem is further compounded due to the lack of proper screening, monitoring and diagnostic tools, specifically designed to identify the disease during pregnancy

and post-partum period.[7]

Physiological changes during pregnancy and post-partum period which can affect management

Management of bipolar illness during pregnancy and lactation comes with a number of therapeutic challenges. It further becomes difficult since the pharmacokinetic properties of a lot of drugs gets altered due to the physiological changes happening in pregnancy and in the immediate post-partum period. For a clinician, it is important to know the implications of these changes, so that adjustment of the dosages of various drugs can be done accordingly, to maintain the same pre-pregnancy therapeutic levels in the blood stream. A lot of physiological factors like fluid status, glomerular filtration, protein concentration and it's binding, drug metabolising enzymes, gastric motility and pH etc undergo notably changes in the pregnancy, compared to pre-pregnancy states, with a potential to affect pharmacokinetics of different drugs, which further has the potential to affect drug efficacy and toxicity.[8] With respect to post-partum period, physiological changes aim at restoring the parameters to pre-pregnancy levels and hence, there is a massive shift of fluids from extravascular to intravascular compartments. Apart from that, the glomerular filtration rate also returns back to baseline pre-pregnancy levels, along with decreased hepatic enzyme activity.[9] Due to these changes, adjusting the dose (decreasing it post-pregnancy) is vital, along with plasma monitoring to ascertain the therapeutic dose. Because of the age on onset of bipolar illness, women are exposed to the risk of episode throughout their reproductive life. Research indicates that women diagnosed with bipolar disorder face a significant likelihood of experiencing symptom relapse both during pregnancy and in the initial postpartum phase. During pregnancy, the risk of symptom relapse is estimated to exceed 50%, with a recurrence risk approximately 2.3 times higher following the cessation of mood stabilizers.[10]

General treatment guidelines for use of psychotropics in pregnancy

Pregnant women are excluded from a number of clinical trials due to ethical concerns, hence there is dearth of adequate human data on the effect of psychotropics in pregnancy in general, let alone their effect and concentrations in different trimesters. Most of our data and further treatment guidelines come from sparse case series and case reports and retrospective studies. In a female of reproductive age group, especially in a country like India, it is important to always keep a possibility of pregnancy, since a lot of pregnancies go unplanned. As per the latest NICE guidelines, it is always best to discuss methods of contraception and their plans of pregnancy in women of child bearing age group with known history of mental illness. It is important to discuss with them about different implications of pregnancy and childbirth on their mental health and how will it affect their course of illness. It is also important to discuss the risk versus benefit ratio of use of various psychotropics during this time. Valproate should not be offered to women in reproductive age group and should be avoided in young girls whose treatment is expected to continue till their child-bearing years. It is best to do proper documentation of all the treatment related decisions made during this time and get consultation liasoning from department of obstetrics and gynaecology.[11] As per CANMAT 2018 guidelines, pre-conception counselling should be provided to all women planning pregnancy, at least 3 months in prior and can be provided as soon as possible to those who are already pregnant. Pregnant women with bipolar disorder are frequently overweight, have a higher incidence of tobacco use during pregnancy, exhibit lower-quality dietary habits, and have a higher prevalence of substance abuse issues, including drug and alcohol misuse during pregnancy.[12] These are some of the risk factors which are modifiable, and if proper preconception counselling is provided, will have a positive impact on both the mother and the child. The decision on the type of management should be a collaborative one and if it is decided to stop pharmacotherapy before pregnancy, the decision should be on case-to-case basis and only after proper risk-benefit analysis. If pharmacotherapy is needed, it is best to keep a single drug and its lowest possible dose. It has also been seen, that in women with history of major depressive disorder, childbirth can trigger a hypomanic/manic episode. Hence, cautious use of antidepressants is warranted at this time and proper family history of bipolar disorders should be taken before initiating treatment. As per the

guidelines, as pregnancy progresses and drug handling is altered, dose changes and adjustments also become a necessity.[13] Also, for example, there are certain psychotropics which can affect ovulation and can decrease fertility by increasing serum prolactin levels. Antipsychotics like risperidone have this propensity and may need to be discontinued or switched to another antipsychotic in order to increase the likelihood of conception.[14]

Trimester-wise considerations while choosing a pharmacological agent

As such, in pregnancy, no psychotropic has been cleared for use by the US FDA food and drug administration. Earlier, when considering pharmacotherapy in pregnant females, only risk at the time of organogenesis, that is, during first trimester, was considered significant and discussed. However, over time, it has been seen that intrauterine exposure to psychotropics during second and third trimester can lead to obstetric complications in the form of preterm delivery, low birth weight and lower apgar scores and postnatal complications in the form of neonatal toxicity and behavioral teratogenicity. Hence, caution needs to be exercised while prescribing pharmacotherapy during the later trimesters.[15] During the first trimester, drug exposure has the maximum effect on the outcome of the pregnancy and hence it is more precarious than other trimesters. Even though, second generation antipsychotics are usually preferred over the first-generation ones, because of safer side-effect profile, due to decades of use of first-generation antipsychotics in pregnancy and their known relative safety, they are usually more preferable.[16] If not adequately addressed, the problem can resurface, as it was seen in one of the studies that in nearly half of the patients, recurrence occurred in first trimester for those who abruptly discontinued the medications, with median time being 2 weeks compared to 22 weeks with gradual tapering off of the medications.[17] As per CANMAT 2018 guidelines, rather than initiating pharmacological treatment, preference should be given to psychosocial strategies, however, this should be based on individual risk factors and is usually not recommended in women with increased risk of relapse.[13] As per NICE guidelines, if a woman is on psychotropics and discovers that she is pregnant, it is advisable to confirm the pregnancy as soon as possible, and once confirmed, psychoeducate her regarding the potential risk to the growing foetus and the need for regular monitoring and screening of growing foetus for any abnormalities, even after switching or stopping medications.[11] During the second trimester, it is important to identify the metabolic side effects of various psychotropics, especially second generation antipsychotics, since they can induce gestational diabetes mellitus in a pregnant female and hence can lead to a number of neonatal complications later. Regular blood pressure and blood glucose monitoring should be done for early detection and hence, timely intervention for such preventable side effects.[16] As pregnancy progresses, a lot of changes happen, especially during the late second trimester and towards the start of first trimester, like increased plasma volume and increased hepatic and renal clearance. All of these changes necessitate an increased dosage of drugs during this time. For instance, the dosage of tricyclic antidepressants (TCAs) must be increased to about 1.6 times the usual dose needed when patients are not pregnant in order to maintain serum levels within the therapeutic range, particularly during the third trimester.[18] It has been seen that, postpartum period is a time of increased risk of relapse, due to various hormonal and psychological changes, along with significant sleep deprivation in the mother, which is an independent risk factor for relapse.[19]As per CANMAT 2018 guidelines, quetiapine is a the preferred molecule for postpartum bipolar depression and for post partum mania, good evidence has been seen for the use of lithium, antipsychotics and benzodiazepines. In cases of acute episode, it is important to follow hierarchy of a no postpartum episode, but special considerations for breastfeeding should be made, wherever applicable. Also, it is important to maintain caution while using antidepressants since they can inadvertently induce manic switch.[13] In one of the studies, it has been seen that, patients who maintained monotherapy with lithium showed a higher rate of sustained remission compared to those who maintained monotherapy with antipsychotics.[20] While using mood stabilizers during breast-feeding, the clinician should keep the dosages to minimum effective dose and monitor the infant for baseline behavior, sleep, feeding and alertness. It is important to psychoeducate the parents regarding side effects of various medications and how to look for early

warning signs in the newborn. Consultation liasoning should be done with department of paediatrics to ensure adequate neonatal monitoring and normal development.[21]

Effect of different classes of mood stabilisers on pregnancy and lactation

1. *Lithium*

Lithium is one of those drugs whose potential risk to the growing foetus has been over-estimated in the past. As per the initial studies, lithium use in first trimester was considered to be highly teratogenic, with risk of Ebstein's anomaly being around 400 times higher than baseline rates. However, overtime, it has been recognised as one of the safest mood stabilisers to be used during pregnancy. In a recent metanalysis calculating the risk of Ebstein's anomaly with first trimester lithium use to be around 10-20 times higher than the general population, with absolute risk being even smaller than this (0.5%-0.1%).[22] However, these figures do not mean that monitoring is not needed whenever a woman is on lithium during pregnancy. As per CANMAT guidelines, if a woman is on lithium during the first trimester, it is imperative to closely monitor the pregnancy with appropriate screening tests like fetal ultrasound.[13] Once lithium is initiated, high resolution ultrasound and echocardiography at 16-18 weeks of gestation is warranted to rule out cardiac anomalies in the fetus. As per NICE guidelines, it is best to avoid lithium during the first trimester, and wherever possible, antipsychotics should be preferred over lithium in women who are planning pregnancy or have become pregnant. If the woman has been maintaining well with a lower risk of relapse, as per the guidelines, lithium can be gradually tapered off within a span of 4 weeks. If pharmacotherapy needs to be continued, lithium can be safely cross-tapered with an antipsychotic or it can be stopped and restarted in the second trimester. However, all these decisions need to be taken in collaboration with the mother and she needs to be psychoeducated about the potential risk versus benefit ratio of continuing/discontinuing lithium. If a woman is on lithium during pregnancy, NICE recommends regular monitoring, with plasma levels of lithium to be checked every 4 weeks. This frequency increases to weekly, starting from 36 weeks of gestation. With lithium, maintaining an adequate fluid balance is essential and as per latest guidelines, it can be safely stopped at the time of labor.[11]

2. Anticonvulsants

With respect to anticonvulsants, all of the guidelines, unanimously, contradict their use in females of child bearing age group, as the risk of teratogenicity is high with first trimester exposure to these drugs. Pre-natal exposure is associated with congenital anomalies in the form of atrial septal defect, hypospadias, craniosyntosis, radial ray and limb defects, pulmonary atresia, cleft palate etc.[23] It is recommended that women who are using valproate and are in their reproductive years, should use effective methods of contraception and adequate attempts should be made to shift to another alternative treatment, prior to contraception.[24] With respect to use of valproate, one should not just be cautious of the structural teratogenicity but also behavioral teratogenicity in the form of increased risk of Autism spectrum disorders, in children with in-utero exposure to valproic acid. It has been seen that this risk is also significant, if the woman was on valproate even pre-conceptionally.[25]

3. Atypical Antipsychotics

Due to their decades of availability, data on the safety and efficacy of first-generation antipsychotics, for their use during pregnancy and lactation is much wider as compared to the newer, atypical antipsychotics. It has been seen that these drugs have the propensity to raise prolactin levels and hence, serum levels of prolactin should be done before initiating treatment with this class of drugs. NICE guidelines recommend using a prolactin sparing antipsychotic, in case the levels raise. It also recommends to continue the antipsychotics in pregnancy if the woman is not maintaining well or is at an increased risk of relapse. However, one needs to be cautious of metabolic side effect profile of these drugs and the possible neonatal adverse effects that it can pose. Proper advice needs to be given regarding diet and monitoring of excessive weight gain and screening needs to be done to rule out gestational diabetes mellitus.[11] With respect to individual antipsychotic, it has been seen that quetiapine has the least placental passage, probably because of its large molecule size and is even safe in lactation.[26] Amongst all the

atypical antipsychotics, only clozapine has been put in category B of US FDA classification. Due to scarcity of studies, the data on its safety during lactation is limited, but it is usually avoided, due to increased risk of agranulocytosis and neonatal seizures in the newborn.

4. Antidepressants

These class of drugs which includes tricyclic antidepressants, SSRIs and SNRIs should be used cautiously both during pregnancy and especially in the postpartum period because of their risk of inducing manic switch due to underlying undiagnosed bipolar disorder. TCAs, when used near term, can lead to neonatal withdrawal symptoms in the form of post neonatal adaptation syndrome (PNAS) and clomipramine can even cause neonatal withdrawal seizures. Amongst TCAs, due to their least anticholinergic effect, desipramine and nortriptyline are usually preferred.[27] With respect to SSRIs, most of the studies have been reassuring for their first trimester use, however, some studies suggest small increases in rates of spontaneous abortions with first trimester use of SSRIs and SNRIs. Unlike previous studies, current studies report little to no correlation of SSRIs with increased risk of persistent pulmonary hypertension of the newborn (PPHN).[28] PNAS can also occur with the exposure of these drugs and most have been reported after use of paroxetine, fluoxetine and venlafaxine.[29]

5. Benzodiazepines

All of them readily diffuse across placenta and are excreted in breast milk. They are usually avoided during pregnancy due to high risk of fetal malformations during 2nd to 8th week of pregnancy. NICE guidelines recommend that they should not be prescribed in pregnancy or lactation, except for short term management of severe anxiety and agitation, with recommendation of using a benzodiazepine with shorter half-life for rapid tranquilisation. Clonazepam due to its myriad of side effects in the form of congenital malformations and neonatal adverse effects is best avoided and lorazepam is generally considered safe in lactation.[30]

6. Newer pharmacological agents

Research for finding newer and safer psychotropics has been limited by relatively lesser well controlled studies and with most of our data being from sparse case series and reports. Data suggests good safety and efficacy for the use of lurasidone, however, monitoring of serum concentrations is important as it can affect the disease outcome.[31] Lamotrigine has also been increasingly used and needs dose monitoring.

Management of acute episodes

As per the study in The Lancet by Cipriani et al., antipsychotic medications were found to be notably more effective than mood stabilizers in treating acute mania with haloperidol showing the highest level of antimanic effectiveness on integrated assessments.[32] CANMAT guidelines recommend starting a mood stabiliser at lowest possible dose for women who are not on any prophylactic therapy and if they already are on it, compliance needs to be checked. Lithium or an atypical antipsychotic can be used to manage acute episodes. For women, not responding to any of these, electroconvulsive therapy is a good alternative, especially during the first trimester.

Special considerations during lactation and postpartum period

Before considering management options, a thorough risk-benefit analysis needs to be done. The benefits of breastfeeding from a physiological and psychological standpoint, the mother's preferences, the possible risks of exposing the baby to medication, and the situation in which a seriously ill mother would prefer to forgo treatment over stopping breastfeeding must all be taken into account. It is important to assess the infant's baseline behaviour in terms of sleep, feeding and alertness and psychoeducate the family members about warning signs in the neonate. Consultation liasioning should be done with department of paediatrics and dose should be titrated to minimum effective dose.[21] While considering the choice of psychotropic, one must be familiar with the concept of relative infant dose (RID), that is, the amount of drug an infant receives via breastfeeding compared to infant's weight. For example, amongst atypical antipsychotics, quetiapine has the least RID (0.09-0.1%) and hence, is considered safe in breastfeeding.[33] This concept of relative infant dose can help make better informed decisions about whether to continue or discontinue breastfeeding while a

female is on a particular psychotropic. It is best to schedule medications right after breastfeeding. This way, impact of psychotropic medications on fetus can be reduced.

Conclusion

To conclude, it is essential to have a multidisciplinary team including psychiatrists, obstetricians, neonatologists and social workers to provide holistic care to the patients. Due to ethical concerns, the data is limited, but as per the available data, antipsychotics appear to be a safer and preferred management option. Quetiapine appears to be safe both during pregnancy and lactation and has also been approved for bipolar depression. In females with mild episode and lower risk of relapse, psychotherapy can be offered as a primary management option. In those maintaining well, medications can be tapered down and stopped during first trimester and then restarted again. For acute episodes, short term usage of benzodiazepines at lowest possible dose is warranted. It is important to keep the physiological changes in mind and alter the dose accordingly. Serum level monitoring is routinely down for some drugs like lithium and monitoring for levels of antipsychotics in the blood is an area of emerging interest. Efforts are underway within regulatory bodies to enhance current practices regarding drug labelling and information. Future pregnancy labels are expected to cover aspects such as clinical considerations, summarized risk evaluation, and the data supporting these assessments. All these endeavours will help clinicians make better informed decisions about the appropriate management options in each individual patient.

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Review Article

Bupropion Hydrochloride and Dextromethorphan Hydrobromide in Depression

Bushra Zahoor, Dimple Gupta, Sandeep Sekhon, Nimmi A. Jose, M.S. Bhatia
Department of Psychiatry, HIMSR & HAHC Hospital, New Delhi-110070
Contact: Bushra Zahoor, E-mail: Bushra.zhr07@gmail.com

Introduction

Major depressive disorder affects an estimated 280 million people worldwide and is a leading cause of disability.[1] Despite numerous pharmacologic options, treatment failure with monotherapy is common,[2] and therapeutic response times are typically slow, taking weeks to months.[3] Given the significant functional impairment and risk of suicide[1] associated with untreated depression, there is a pressing need for effective, rapid-acting oral antidepressants that can achieve remission.[2]

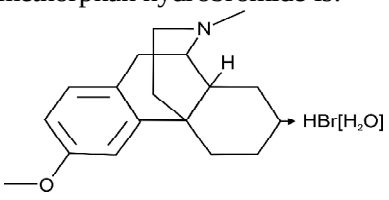
Current guidelines for managing non-psychotic depression recommend monotherapy with antidepressants that modulate the monoamine neurotransmitter system, such as selective serotonin reuptake inhibitors (SSRIs), serotonin norepinephrine reuptake inhibitors (SNRIs), or atypical antidepressants like bupropion and mirtazapine, as first-line treatments.[4,5] While these monotherapies do outperform placebos,[6] nearly two-thirds of patients do not achieve remission after initial treatment with an SSRI, and a similar proportion fail to remit after switching to another antidepressant. For those who do reach remission, response times are often delayed, and patients may experience adverse side effects during this period,[7] underscoring the need for new treatment options that are effective, fast-acting, and well-tolerated.

Combination therapies using agents from different antidepressant classes have proven to be effective alternatives to monotherapy for acute depression.[8,9] Several second-generation antipsychotics, such as aripiprazole,[10] brexpiprazole,[11] cariprazine,[12] olanzapine (combined with fluoxetine),[13] and quetiapine extended release,[14] have been approved by the FDA for adjunctive treatment of depression. However, these are associated with extrapyramidal symptoms and metabolic syndrome. Intranasal esketamine,[15] a rapid-acting non-competitive N-methyl D-aspartate (NMDA) receptor antagonist, has also been FDA approved for adjunctive treatment of treatment-resistant depression and depressive symptoms in adults with major depressive disorder and suicidality. Despite its efficacy, it requires strict monitoring due to side effects and abuse potential, and it is not available in an oral formulation.

In 2022, the FDA approved a fixed-dose, extended-release combination of dextromethorphan and bupropion (Auvelity) for the treatment of major depressive disorder in adults. This combination tablet, which acts as an NMDA receptor antagonist and a sigma-1 receptor agonist, offers a novel rapid-acting oral treatment option for depression.[16]

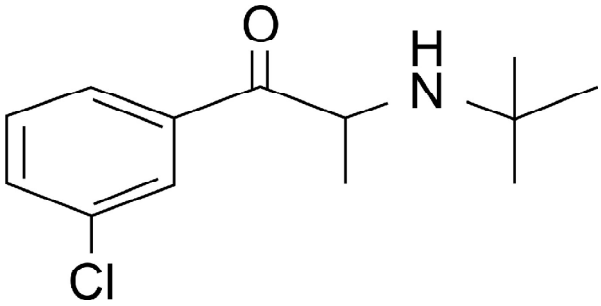
Structure

Dextromethorphan hydrobromide The Dextromethorphan hydrobromide is an uncompetitive NMDA receptor antagonist and sigma-1 receptor agonist, and bupropion hydrochloride is an aminoketone and CYP450 2D6 Inhibitor. The chemical name of dextromethorphan hydrobromide is morphinan, 3- methoxy-17- methyl, (9α, 13α, 14α), hydrobromide monohydrate. Dextromethorphan hydrobromide has the empirical formula $C_{18}H_{25}NO$ $HBrH_2O$ and a molecular weight of 370.33 (271.4 dextromethorphan base). Structural formula of Dextromethorphan hydrobromide is:



Bupropion Hydrochloride

The chemical name of bupropion hydrochloride is: (±)-1- (3-chlorophenyl)-2-[(1,1- dimethylethyl) amino]-1propanonehydrochloride. Bupropion hydrochloride has the empirical formula $C_{13}H_{18}ClN$ OHCl and a molecular weight of 276.2 (239.74 bupropion base). The structural formula is:



Mechanism of action

Dextromethorphan Hydrobromide: Dextromethorphan is an uncompetitive antagonist of the NMDA receptor (an ionotropic glutamate receptor) and a sigma-1 receptor agonist. The mechanism of dextromethorphan in the treatment of MDD is unclear.

Bupropion Hydrochloride

The mechanism of action of bupropion in the treatment of MDD is unclear; however, it may be related to noradrenergic and/or dopaminergic mechanisms. Bupropion increases plasma levels of dextromethorphan by competitively inhibiting cytochrome P4502D6, which catalyzes a major biotransformation pathway for dextromethorphan. Bupropion is a relatively weak inhibitor of the neuronal reuptake of norepinephrine and dopamine and does not inhibit monoamine oxidase or the reuptake of serotonin.

Pharmacokinetics

When bupropion is co-administered with dextromethorphan, the pharmacokinetics of dextromethorphan at steady state are non-linear, exhibiting greater than proportional increase in exposure (AUC and Cmax) with varying doses of dextromethorphan (60-120 mg; 0.67–1.33 times the maximum recommended dose of dextromethorphan/ bupropion) and less than dose-proportional changes with varying doses of bupropion (150–300 mg; 0.71–1.43 times the maximum recommended dose of dextromethorphan/bupropion). Steady state plasma concentrations for both drugs are achieved within 8 days of repeated administration of dextromethorphan/bupropion. The Cmax of dextromethorphan and bupropion is reached at a median of 3 hours and 2 hours, respectively, after administration. Taking dextromethorphan/bupropion with food has minimal impact on the exposure (Cmax and A_{UC} 12) of both drugs, allowing the combination to be taken with or without food.

Dextromethorphan is approximately 60–70% bound to plasma proteins, while bupropion is 84% plasma protein bound.

Dextromethorphan is primarily metabolized by CYP2D6 into dextrorphan. Bupropion undergoes extensive metabolism and produces three active metabolites (hydroxybupropion, threohydroxy bupropion, and erythrohydroxy bupropion). The mean half-lives ($t_{1/2}$) of dextromethorphan and bupropion after 8 days of administration are 22 hours and 15 hours, respectively. Kidney impairment and CYP2D6 poor metabolizer status increase exposure to dextromethorphan/bupropion, necessitating dosage reductions for patients with moderate kidney impairment and CYP2D6 poor metabolizers. However, dosage adjustments are not required for patients with mild or moderate hepatic impairment (Child-Pugh A and Child-Pugh B).

Pharmacodynamics

At the maximum recommended dose, Dextromethorphan Hydrobromide and Bupropion Hydrochloride does not prolong the QT interval to any clinically relevant extent.

Dosage

Each tablet of dextromethorphan/bupropion contains 45mg dextromethorphan hydrobromide (equivalent to 32.98 mg dextromethorphan base) in an immediate-release formulation and 105 mg bupropion hydrochloride (equivalent to 91.14 mg bupropion base) in an extended-release formulation. The recommended starting dosage is one tablet once daily in the morning with or without food. After 3 days, the dosage should be increased to one tablet twice daily, taken at least 8 h apart (maximum dosage; should not exceed two doses within the same day)

Dosing in adults and special populations[18]

Patient population	Initial dosing	Maintenance dosing
Adults	45/105 mg daily × 3 days	45/105 mg twice dailya
Hepatic impairment		
Child Pugh A–B	45/105mg daily × 3 days	45/105 mg twice
Child Pugh C	Not recommended, no data studied	
Renal impairment		
eGFR 30–59	45/105 mg daily	45/105 mg daily
eGFR <30	Not recommended, no data studied	
Poor CYP2D6 metabolizers	45/105 mg daily	45/105 mg daily
Concomitant strong CYP2D6 inhibitor use	45/105 mg daily	45/105 mg daily

eGFR, estimated glomerular filtration rate.

aDosing should be at least 8 hours apart. Do not exceed 2 tablets in 24 hours.

Major Side-effects

Most common adverse reactions (≥ 5% and more than twice as frequently as placebo): dizziness, headache, diarrhoea, somnolence, dry mouth, sexual dysfunction, and hyperhidrosis.

- Seizure: Risk is dose-related. Discontinue if seizure occurs.
- Increased Blood Pressure and Hypertension: Dextromethorphan and bupropion can increase blood pressure and cause hypertension. Assess blood pressure before initiating treatment and monitor periodically during treatment.
- Activation of Mania or Hypomania: Screen patients for bipolar disorder.
- Psychosis and Other Neuropsychiatric Reactions: Instruct patients to contact a healthcare provider if such reactions occur.
- Angle-Closure Glaucoma: Angle-closure glaucoma has occurred in patients with untreated anatomically narrow angles treated with antidepressants.
- Dizziness: Dextromethorphan and bupropion may cause dizziness. Take precautions to reduce falls and use caution when operating machinery.
- Serotonin Syndrome: Use of Dextromethorphan and bupropion with selective serotonin reuptake inhibitors (SSRIs) or tricyclic antidepressants increases the risk. Discontinue if occurs.
- Embryo-fetal Toxicity: May cause fetal harm. Advise pregnant females of the potential risk to a fetus. Discontinue treatment in pregnant females and use alternative treatment for females who are planning to become pregnant.

Clinical Studies

Study 1: GEMINI Trial-Efficacy and Safety of Dextromethorphan-Bupropion in Patients with Major Depressive Disorder[17]

The GEMINI phase 3 clinical trial was a randomized, double-blind, placebo-controlled, multicenter study that enrolled 327 adult patients with moderate-to-severe major depressive disorder (MDD), defined by a Montgomery–Åsberg Depression Rating Scale (MADRS) score of 25 or higher. Over 6 weeks, patients received either a placebo or a combination of dextromethorphan and bupropion. The primary efficacy endpoint was the change in MADRS total score after 6 weeks. Secondary endpoints included MADRS changes at weeks 1 and 2, clinical remission (MADRS scored ≤10), clinical response (≥50% reduction in MADRS score from baseline), clinician- and patient-rated global assessments, Quick Inventory of Depressive Symptomatology-Self-Rated, Sheehan Disability Scale, and quality of life measures. The study concluded that treatment with dextromethorphan-bupropion (AXS-05) significantly improved depressive symptoms compared to placebo, starting from week 1, and was

generally well tolerated.

Study 2: ASCEND trial-Effect of Dextromethorphan- Bupropion in Major Depressive Disorder[18]

The ASCEND phase 2 clinical trial, a randomized, double-blind, active-controlled, multicenter study, enrolled 80 adult patients with moderate-to-severe major depressive disorder (MADRS scoree ≥25). Participants received either dextromethorphan with bupropion or bupropion alone, twice daily for 6 weeks. The primary endpoint was the overall treatment effecton the MADRS score (average change from baseline for weeks 1–6). Secondary end points included clinical response, remission, CGI-I score, and QIDS-SR score. In this phase 2 trial, the combination of dextromethorphan and bupropion (DXM/BUP) rapidly reduced depressive symptoms as early as week 1, with statistically significant improvements over bupropion alone by week 2 an date very subsequent time point. There was rapid and sustained improvement in remission rate, response rate, CGI-I, and CGI-S scores. DXM/BUP was well-tolerated and did not cause weight gain or increased sexual dysfunction.

Study 3: COMET trial-Sustained Efficacy with Long term Treatment with Dextromethorphan-Bupropion[16]

The COMET phase 3 clinical trial, a non-randomized, open-label, multicenter study, included 611 adult patients with moderate-to-severe major depressive disorder (MADRS score ≥25). Participants were given dextromethorphan with bupropion twice daily for 12 months, including both new enrollees and those who completed a previous DXM/BUP study.

Efficacy was measured using the Montgomery Åsberg Depression Rating Scale (MADRS), clinical response (≥50% reduction in MADRS score), clinical remission (≤10 MADRS score), Clinical Global Impression of Improvement (CGI-I), and Sheehan Disability Scale (SDS). DXM/BUP led to rapid and significant reductions in depressive symptoms and improved f unctioning, with effects sustained over 12 months. Clinical response and remission rates on MADRS and functional response on SDS were notable. The treatment was well tolerated, with dizziness, nausea, and headache being the most common adverse events.

Study 4: COMETSI Trial–Rapid Reduction in Suicidal Ideation in Patients Treated with Dextromethorphan-Bupropion[16]

The COMETSI sub-study assessed the efficacy of Dextromethorphan-bupropion treatment in patients with major depressive disorder (MDD) and suicidal ideation. Patients received twice-daily doses of 45 mg dextromethorphan and 105 mg bupropion over up to 12 months. Results indicated significant reductions in suicidal ideation, improvements in depressive symptoms, and enhanced functioning. The study concluded that Dextromethorphan-bupropion effectively reduced suicidal ideation and improved overall outcomes in MDD patients with suicidal thoughts.

Study 5: COMET TRD Trial-Sustained Effects of Dextromethorphan-Bupropion in Treatment Resistant Depression patients[16]

The COMET TRD sub-study, part of the COMET Phase 3 trial, examined the long-term effectiveness and safety of Dextromethorphan-bupropion in patients with treatment-resistant depression (TRD). Participants, including those previously treated with Dextromethorphan-bupropion and new enrollees, received the combination therapy twice daily for up to 12 months. Results showed significant and lasting improvements in depressive symptoms and functional outcomes, with high rates of clinical response and remission on the MADRS scale.

Dextromethorphan-bupropion was generally well tolerated, with common adverse events including dizziness, nausea, and headache.

Study 6: EVOLVE Trial-Improvement in Anxiety Symptoms in Depressed Patients Treated with Dextromethorphan Bupropion in long-term study[19]

The EVOLVE trial was an open-label study conducted in the US to evaluate the effects of Dextromethorphan-bupropion in patients with major depressive disorder (MDD). Participants received treatment twice daily for up to 15 months. Eligible patients had a DSM-5 diagnosis of MDD, a MADRS score of ≥25, and had been treated with atleast one prior antidepressant in their current

depressive episode. A total of 186 patients were enrolled, including 146 directly enrolled and 35 rollover patients from a prior study (MERIT). Efficacy outcomes were assessed using MADRS, Hamilton Anxiety Rating Scale (HAM-A), and Sheehan Disability Scale (SDS). Dextromethorphan-bupropion treatment in the EVOLVE trial led to rapid reduction in anxiety symptoms among patients with MDD. Significant response and remission from anxiety symptoms were observed as early as one week after starting treatment. Long-term use of Dextromethorphan-bupropion was well tolerated by the patients in the study.

Study 7: A Phase III, Indian Clinical Trial Randomized, Double Blind, Active Controlled, Comparative Study to Evaluate the Efficacy, Safety and Tolerability of FDC of Dextromethorphanplus Bupropion ER Tablets versus Bupropion Hydrochloride SR Tablets in Adult Patients with Major Depressive Disorder (MDD).[16]

In a phase III clinical study conducted in India, 212 patients were randomized to receive either a fixed-dose combination (FDC) of Dextromethorphan Hydrobromide 45 mg + Bupropion Hydrochloride 105 mg.

Extended-Release Tablets or Bupropion Hydrochloride Sustained Release Tablets 150 mg for 42 days. The study aimed to evaluate the efficacy of the FDC compared to bupropion alone using MADRS total score change from baseline at week 6 as the primary end point. Secondary end points included clinical response, remission rates, and various other measures of depressive symptoms and quality of life.

The results indicated that the FDC demonstrated statistically significant antidepressant efficacy on the primary and most secondary endpoints. It was well-tolerated, and adverse effects such as weight gain or increased sexual dysfunction were not reported.

Conclusion

Dextromethorphan/bupropion is an orally administered, rapidly-acting, NMDA receptor antagonist with multimodal activity. The formulation achieves pharmacologic synergy by simultaneously targeting monoamines, NMDA receptors, and sigma-1 receptors, resulting in more rapid and robust decreases in depression rating scale scores than bupropion treatment alone. With its similar pharmacological properties to ketamine, dextromethorphan/bupropion represents a promising new agent for treating depression.

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Original Article

Subjective Caregiving Burden and Anxiety among Primary Family Caregivers of the Patients with Intellectual Disability: A Cross-Sectional Study

Kehksha,[1] Mohammed Reyazuddin,[2] Arshi Khanam[3]
[1]Department of Psychology, Tika Ram Girls' Degree, College, Raja Mahendra Pratap Singh State University, Aligarh, India;
[2] Department of Psychiatry, Jawahar Lal Nehru Medical College & Hospital, Aligarh Muslim University, Aligarh, India;
[3]Department of Psychiatry, All India Institute of Medical Sciences, Patna, India
Contact: Kehksha, E-mail: kehksha931@rediffmail.com

ABSTRACT

Background: *Providing care to the patients living with intellectual disability (ID) is a very stressful situation to the caregivers. These caregivers often report psychophysiological problems; specifically, subjective caregiving burden (SCB) and anxiety caused by ID. Many factors like severity of ID, sociodemographic characteristics of the patients and caregivers as well significantly contribute in developing subjective caregiving burden and anxiety among these caregivers.* ***Objectives****: We aimed to examine subjective caregiving burden (SCB) and anxiety among primary family caregivers of patients with intellectual disability (ID), and to find out association of sociodemographic characteristics of patients and caregivers with SCB and anxiety.* ***Methods****:190 consecutive family caregivers of patients with ID were taken from a tertiary care center by purposive sampling method. Severity of ID among patients was determined by Binet-Kamat scale of intelligence, and the Vineland social maturity scale. Semi-structured sociodemographic and clinical data sheet, Zarit burden interview, and Hamilton anxiety rating scale were administered on caregivers to examine the level of SCB and anxiety.* ***Results:*** *42% family caregivers had mild to moderate SCB, and 43% caregivers had mild to moderate anxiety. Severity of ID was significantly associated with SCB ($\chi^2 = 17.363$, $p = .000 < 0.05$), and anxiety ($\chi^2 = 43.634$, $p = .000 < 0.05$). Caregiver's relationship with the patients, patient's gender, and their age group were significantly associated with SCB and anxiety. Caregivers of patients with profound ID had significantly higher SCB ($M = 58.11$, $F = 5.39$, $p = .001 < 0.05$) and anxiety ($M = 25.56$, $F = 8.56$, $p = .000 < 0.05$) than other caregivers.* ***Conclusion:*** *Family caregivers of the patients with ID suffer from high SCB and anxiety which is significantly associated with the severity of disability and sociodemographic characteristics of patients and caregivers as well. Early identification and effective psycho-pharmacological treatment for SCB and anxiety should be given to the caregivers to prevent further psychiatric disorders.*

Keywords: *Intellectual disability; anxiety; subjective burden; caregivers; family.*

Introduction

Intellectual disability (ID) is a neurodevelopmental disorder characterized by deficit in intellectual and adaptive functioning of conceptual, social, and practical domains of life with the onset before 18 years of age or during the developmental period. Prevalence rate of ID has been estimated 2% in India for the last 60 years.[2] Patients with ID have impaired cognitive functioning and non-adaptive behavior. They have limited social skills and are less capable to use their past experiences in executing global functions. Consequently, they are completely

dependent on caregivers for meeting their basic psychophysical needs. These caregivers are often unpaid family members who provide care to chronically ill patients living with intellectual or functional disabilities.[3] Providing care to these patients demands a wide range of time and energy regardless of the available sources. Such situation produces burden and other psychological morbidities among caregivers.[4] Providing prolonged care to patients with ID causes caregivers depressive disorder, anxiety disorders, insomnia, psychotic disorders, and alcohol use disorder.[5] These psychological morbidities become severe if caregivers deal with disturbed family functions, low self-efficacy, and negative perceived health.[6] Some other factors like unemployment, low educational status, poor physical health, financial strain, and co-habitation with the patient also contribute to increase caregiving burden.[7,8] Besides this, gender of the caregivers and their relationship with the patient play a pivotal role in determining the occurrence of psychological morbidities among caregivers. In Asian countries like India, caregiving is supposed to be the responsibility of mothers of patients with ID or it is upon female members of the families. These female members are already occupied with other household duties that consume their enough time and energy. Imposing caregiving on these female members not only creates a paucity of time for meeting their ends but also produces burden and anxiety. This might be one of the reasons that several studies reported higher anxiety, depression, stress, and subjective burden among female caregivers in comparison to male caregivers.[9-11] Not only caregivers but other family members also feel burden of disease caused by providing partial care to the patients with ID.[12] Even caring for these patients at home does not reduce caregiving burden and does not provide any satisfaction to the caregivers.[13] Consequently, caregivers of patients with ID always remain under strain and gradually develop various psychological morbidities. Whenever caregiver of a patient with ID approaches health care center, primary focus of the health care providers is rather upon the patient. However, studies have already reported higher prevalence of psychological problems among these caregivers.[14,15] Therefore, this study was conducted to examine the level of SCB and anxiety among primary family caregivers of patients with ID. Researchers also aimed to find out the association of sociodemographic characteristics of patients and caregivers with SCB and anxiety.

Methods

Participants

This is a cross-sectional study conducted on 190 consecutive primary family caregivers of the patients diagnosed with ID, and who came to the psychiatry outpatient department of a tertiary care hospital in India for assessment, treatment, and certification of the patients for availing government facilities specifically provided to these patients. Inclusion/ exclusion criteria for the present study were set into two separate sections; one for the patients with ID and other for the caregivers. Those patients were taken in the study whose age ranged between 5-55 years, diagnosed with intellectual disability criteria as per DSM-5, and whose severity of ID was determined by Binet-Kamat scale of intelligence.[16] and the Vineland social maturity scale.[17] Patients attending any special school/ program, or suffering from any visual/ auditory/ speech impairment were excluded from the study. Patients spending any amount of time at half-day care, hospitals, or any daycare center were also excluded from the study. On the other hand, caregivers who had biological relationship with the patients living with ID, spent most of their time in caregiving, and not receiving any psychotherapeutic treatment for any psychological morbidity were taken in the study. Caregivers suffering from any psychiatric and/or medical illness were excluded from the study. First, all the patients were assessed by the psychiatrist for ruling out ID. Then, Binet-Kamat scale of intelligence, and the Vineland social maturity scale were administered on the patients to assess the severity of ID. After that, semi-structured sociodemographic and clinical data sheet was administered onall the caregivers for fulfilling inclusion-exclusion criteria. Meeting inclusion criteria led researchers to further administration of Zarit Burden Interview (ZBI) and Hamilton anxiety rating scale (HAM-A) on the caregivers.

Instruments

Semi-structured sociodemographic and clinical data sheet

This data sheet was constructed by researchers in order to collect sociodemographic and clinical

details of patients and their caregivers. In this datasheet, sociodemographic characteristics of the caregivers were collected along with the history of the prenatal, perinatal, and postnatal development of patients with ID.

Zarit burden interview (ZBI)

This is a 22 itemed scale used for the assessment of subjective caregiving burden. Responses on this scale are rated on 5 point Likert scale ranging from never (0) to nearly always (4) with the possible score range between 0-88. Higher score indicates higher level of caregiving burden, but score ≥16 has been suggested as the cutoff point by the original author of this scale.[18] However, in this study score range between 21-40 was considered as "mild to moderate burden", 41-60 for "moderate to severe burden", and score range between 61-88 was taken as the "severe burden" of caregiving. Cronbach's alpha value for this scale was found .92 while convergent validity ranged from .46 to .58.[19]

Hamilton anxiety rating scale (HAM-A)

This scale consists of 14 items assessing anxiety of clinical and non-clinical population. Score on this scale ranges from 0 to 4 where 0 stands for "no symptom of anxiety" and 4 indicates "severe level of anxiety". On this scale, scores <17 indicate mild anxiety, score range between 18-24 indicates "mild to moderate anxiety", and scores higher than 25 indicate "moderate to severe anxiety".[20] Coefficient alpha of HAM-A ranges from .41 to .89 while average reliability of this scale was found .79.[21]

Statistical analysis

Descriptive statistics like mean, mode, SD, and range were administered on the data to find out frequency of sociodemographic characteristics of the sample, severity of ID, severity of SCB, and anxiety. Chi-square test with Yate's continuity correction was applied to the data to find out the association of sociodemographic characteristics with SCB, anxiety, and severity of ID. Multivariate analysis of variance along with partial eta squared was used to find out the difference among caregivers on SCB and anxiety in terms of severity of ID along with the proportion of variance in the given parameters respectively. All the obtained data were analyzed by IBM SPSS Statistics 20.

Results

Majority of the caregivers were females (171, 90%), aged between 42-51 years, mothers of the patients (171, 90%), and married (174, 91.6%). On the other hand, majority of the patients were male (133, 70%), aged between 4-14 years & 15-25 years equally (85, 44.74%), and were suffering from mild ID (83, 43.7%). Psychiatric comorbidity (i.e., seizure disorder, substance use disorders, cerebral palsy, and psychosis) was present among 7.89% patients, and physical comorbidity was found in 1.05% patients (Table 1).

Table-1: Sociodemographic profile of caregivers (n=190) and patients with intellectual disability (n=190)

Characteristics of Caregivers		**N (%)**
Gender	Female	171 (90%)
	Male	19 (10%)
Age	22-31 years	34 (17.89%)
	32-41 years	55 (28.94 %)
	42-51 years	71 (37.37%)
	>51 years	30 (15.79%)
Relationship with patient	Mother	171 (90%)
	Father	10 (5.3%)
	Siblings	9 (4.7%)
Marital status	Married	174 (91.6%)
	Widow/widower	16 (8.4%)
Characteristics of patients with ID		
Gender	Female	57 (30%)
	Male	133 (70%)
Age	4-14 years	85 (44.74%)
	15-25 years	85 (44.74%)
	>25 years	20 (10.53%)
Severity of ID	Mild	83 (43.7%)
	Moderate	68 (35.8%)
	Severe	30 (15.8%)
	Profound	9 (4.7%)
Comorbidity	Normal	173 (91.05%)
	Psychiatric comorbidity	15 (7.89%)
	Physical comorbidity	2 (1.05%)

42% caregivers reported mild to moderate SCB, 37% caregivers reported moderate to severe SCB, and 21% caregivers reported severe SCB (Figure 1). On anxiety parameter, 43% caregivers had mild to moderate anxiety, 33% caregivers had mild anxiety, and 24% had moderate to severe anxiety (Figure 2).

Findings demonstrated that severe SCB was more prevalent among males (5, 26.3%), aged above

51 years (15, 50%), fathers (5, 50%), and widow/widowers (9, 56.2%). Age, relationship with patient (p = .001 <0.05), and marital status (p = .000 <0.05) are significantly associated with SCB except gender of the caregivers (p = .740 >0.05) (Table 2).

26.3%), and widow/widowers (4, 25%). Results indicated that anxiety was significantly correlated with age of the caregivers (p = .002 <0.05), and relationship with the patients (p = .026 <0.05) (Table 4).

Moderate to severe anxiety was also reported

Table-2: Correlation of sociodemographic characteristics of caregivers with subjective caregiving burden (SCB)

Variable		Subjective caregiving burden			χ^2	p
		Mild to moderate	Moderate to severe	Severe		
Gender	Female	71 (41.5%)	65 (38%)	35 (20.5%)	.589	.740
	Male	8 (42.1%)	6 (31.6%)	5 (26.3%)		
Age	22-31 years	22 (64.7%)	8 (23.5%)	4 (11.8%)	46.939	.000
	32-41 years	34 (61.8%)	20 (36.4%)	1 (1.8%)		
	42-51 years	18 (25.4%)	33 (46.5%)	20 (28.2%)		
	>51 years	5 (16.7%)	10 (33.3%)	15 (50%)		
Relationship with the patient	Mother	71 (41.5%)	65 (38%)	35 (20.5%)	16.506	.001
	Father	0 (0%)	5 (50%)	5 (50%)		
	Sibling	8 (88.9%)	1 (11.1%)	0 (0%)		
Marital status	Married	78 (44.8%)	65 (37.4%)	31 (17.8%)	14.485	.000
	Widow/widower	1 (6.2%)	6 (37.5%)	9 (56.2%)		

*Significant at the 0.05 level (p<0.05)

Table-3: Correlation of sociodemographic characteristics of patients with subjective caregiving burden

Variable		Subjective caregiving burden			χ^2	p
		Mild to moderate	Moderate to severe	Severe		
Age	4-14 years	56 (65.9%)	26 (30.6%)	3 (3.5%)	55.378	.000
	15-25 years	15 (17.6%)	40 (47.1%)	30 (35.3%)		
	>25 years	7 (36.8%)	5 (26.3%)	7 (36.8%)		
Gender	Male	63 (47.4%)	44 (33.1%)	26 (19.5%)	6. 229	.043
	Female	16 (28.1%)	27 (47.4%)	14 (24.6%)		
Severity of ID	Profound	1 (11.1%)	4 (44.4%)	4 (44.4%)	17.363	.000
	Severe	5 (16.7%)	16 (53.3%)	9 (30%)		
	Moderate	29 (42.6%)	26 (38.2%)	13 (19.1%)		
	Mild	44 (53%)	25 (30.1%)	14 (16.9%)		

*Significant at the 0.05 level (p<0.05)

Caregivers of patients aged above 25 years (7, 36.8%), female (14, 24.6%), and patients with profound ID (4, 44.4%) reported severe SCB than other caregivers. Correlational value obtained by chi-square test indicated that SCB is significantly correlated with age group of the patients (p = .000 <0.05), gender of the patient (p = .043 <0.05), and with severity of ID (p = .000 <0.05) (Table 3).

Moderate to severe level of anxiety was comparatively more prevalent among females (45, 26.3%), who aged above 51 years (10, 33.3%), mothers (45, majorly among the caregivers of patients aged above 25 years (6, 31.6%), female patients (30, 52.6%), and patients with profound ID (8, 88.9%). Results indicated that anxiety was significantly correlated with age (p = .000 <0.05), gender (p = .000 <0.05), and with the severity of ID (p = .000 <0.05) of the patients (Table 5).

There was a significant difference among all the caregivers of the patients suffering from profound, severe, moderate, and mild ID on SCB and anxiety, wilk's Λ =.862, F (6, 370) = 4.754, p =

Table-4: Correlation of sociodemographic characteristics of caregivers with anxiety

Variable		Anxiety level			χ^2	p
		Mild	Mild to moderate	Moderate to severe		
Gender	Female	54 (31.6%)	72 (42.1%)	45 (26.3%)	4.860	.087
	Male	9 (47.4%)	9 (47.4%)	1 (5.3%)		
Age	22-31 years	19 (55.9%)	7 (20.6%)	8 (23.5%)	20.045	.002
	32-41 years	23 (41.8%)	24 (43.6%)	8 (14.5%)		
	42-51 years	16 (22.5%)	35 (49.3%)	20 (28.2%)		
	>51 years	5 (16.7%)	15 (50%)	10 (33.3%)		
Relationship with the patient	Mother	54 (31.6%)	72 (42.1%)	45 (26.3%)	9.981	.026
	Father	2 (20%)	7 (70%)	1 (10%)		
	Sibling	7 (77.8%)	2 (22.2%)	0 (0%)		
Marital status	Married	60 (34.5%)	72 (41.4%)	42 (24.1%)	1.833	.400
	Widow/widower	3 (18.8%)	9 (56.2%)	4 (25%)		

*Significant at the 0.05 level ($p<0.05$)

Table-5: Correlation of sociodemographic characteristics of patients with anxiety among caregivers

Variable		Anxiety			χ^2	p
		Mild	Mild to moderate	Moderate to severe		
Age	4-14 years	41 (48.2%)	28 (32.9%)	16 (18.8%)	19.492	.000
	15-25 years	15 (17.6%)	46 (54.1%)	24 (28.2%)		
	>25 years	7 (36.8%)	6 (31.6%)	6 (31.6%)		
Gender	Male	57 (42.9%)	60 (45.1%)	16 (12%)	40.386	.000
	Female	6 (10.5%)	21 (36.8%)	30 (52.6%)		
Severity of ID	Profound	0 (90%)	1 (11.1%)	8 (88.9%)	43.634	.000
	Severe	3 (10%)	11 (36.7%)	16 (53.3%)		
	Moderate	20 (29.4%)	37 (54.4%)	11 (16.2%)		
	Mild	40 (48.2%)	32 (38.6%)	11 (13.3%)		

*Significant at the 0.05 level ($p<0.05$)

Table-6: Multivariate analysis of variance for finding the difference in anxiety and subjective caregiver burden based on severity of intellectual disability

Variable	Severity	Mean	SD	F	*p*	η^2
Subjective caregiving burden	Profound	58.11	11.92	5.390	.001	.080
	Severe	50.6	13.71			
	Moderate	44.6	14.17			
	Mild	42.04	14.61			
Anxiety	Profound	25.56	3.00	8.564	.000	.121
	Severe	22.4	5.49			
	Moderate	19.25	5.89			
	Mild	17.84	5.64			

.00, partial $\eta^2 = .072$. A separate ANOVA was conducted for each dependent variable and evaluated at alpha level of .05. There was a significant difference in SCB with medium effect size among the caregivers providing care to the patients with different levels of ID F (3, 186) = 5.390, $p = .001$, partial $\eta^2 = .080$ with higher burden among caregivers of patients with profound ID ($M = 58.11$) followed by the caregivers caring patients with severe ($M = 50.6$), moderate ($M = 44.6$), and mild ID ($M = 42.04$). Significant difference was also found in anxiety with medium effect size among the caregivers

providing care to the patients with different severity levels of ID F (3, 186) = 8.564, p = .000, partial η^2 = .121 with higher anxiety among caregivers of the patients with profound ID (M = 25.56) followed by patients with severe (M = 22.4), moderate (M = 19.25), and mild ID (M = 17.84) (Table 6).

Discussion

Major findings of the present study demonstrated that most of the patients with ID were males and between 4-25 years of age. Higher prevalence of ID among males is due to the x-linked chromosome that makes them more likely to suffer from ID than females.[22] Findings of a previous study also reported that ID is more prevalent among males than females in both adult and children/ adolescent population.[23] Higher prevalence of ID among this age group might be due to the lack of awareness of ID among parents, which resulted in late diagnosis of this disorder. In the present study, 7.89% patients were presented with psychiatric comorbidities like seizure disorder, substance use disorders, cerebral palsy, and psychosis. Contrary to the present study, findings from one metanalysis indicated that relative risk of mental disorders among children and adolescents with ID ranges from 2.8 to 4.5.[24] while approximately 40% of the older adult patients with ID suffer from psychiatric comorbidity.[25] Most of the patients in this study were suffering from mild ID and very few patients were diagnosed with profound ID. This finding is obvious because in the population with ID, distribution of mild, moderate, severe, and profound ID constitutes 85%, 10%, 3-4%, and 1-2% respectively.[26] Other studies also reported that in the whole sample, a high number of patients suffer from mild ID.[27,28]

Majority of the caregivers in the present study was females, mothers of the patients, and married. Gender of caregiver, relationship with patient, and marital status are the reflection of cultural effects on the sociodemographic characteristics of the caregivers. Previous studies conducted on Indian population also reported that most of the caregivers of the patients with ID are females, mothers, married, and Hindus.[29-31]

In the present study, majority of the caregivers reported mild to moderate SCB and mild to moderate anxiety. Caregivers of the patients with profound ID reported significantly higher SCB and anxiety than other caregivers. Caring for the patients suffering from ID produce depression or anxiety, or both depression and anxiety among caregivers, which is the outcome of physical and emotional stress, lack of familial support, and magnitude of severity of illness.[32] Severity of illness also causes financial and psychological strain among caregivers.[33] Stigmatized attitude of the parents and society against ID and the patients suffering from this, along with coping strategies of the parents increase their level of stress.[34] Poor sleep quality, lack of social support, and problematic behavior of children substantially add to the caregiving burden and anxiety among caregivers.[35] These caregivers always feel embarrassment, shame, and guilt due to the illness of their children which gradually worsens their psychological health.[36]

SCB and anxiety were significantly associated with caregivers' age, relationship with the patients, and marital status. SCB and anxiety both were also significantly associated with patients' age, gender, and with the severity of ID. All the mental and physical functions of humans verily depend on age. As people grow older, their global capacity of performing day to day work starts diminishing gradually. Providing care to the child with ID with growing age become more tiresome for such parents. Previous studies also demonstrated that age is a significant predictor of burden, stress, and depression among older parents of children with ID.[37-39] However, one study conducted in Indian context showed negative association between parents' age and burden.[40]

Findings also reported that fathers had higher burden while mothers had higher anxiety in comparison to their spouses. Significant association has been found between relationship, SCB and anxiety. Previous studies revealed that gender of the caregivers, their relationship with the patients, and patients' gender and age group contribute to SCB and anxiety among caregivers.[41,42] Similar to the findings of present study, Khamis also found that patient's age, caregiver's age, relationship with the patient, unemployment, mothers' education, and severity of ID are significantly associated with psychiatric symptoms, personal growth, and parental stress among caregiving parents of children with ID.[43]

Marital status of an individual provides way

more satisfaction to spouses because they share household duties and burdens of the family equally. Absence of any partner can produce various psychosocial issues after the death of any of the partners.[44,45] In such situation, providing care to a child with ID without partner and trying to make the family's ends meet become remarkably stressful for widow/widowers.[38,39] As children with ID grow up, their behavioral dysfunction also increases in the same way. Besides it, behavioral dysfunction in a grown-up child is usually unaccepted by general folks. Parents of such patients tend to have negative perception of their children and often take them as a challenge to their capabilities and resources.[46] Previous studies inference that challenging behavior of the child causes severe negative effects on the family members.[47] This might be one of the reasons that in this study patients' age was significantly linked with SCB and anxiety.

If gender of the patient is female, stress and burden increase to a great extent among caregiving parents.[36] Caregiving parents of a female patient always remain apprehended, because they need to provide extra care, safety, and security to the female patient as compared to male patient due to the gender based crimes against such females. It might be one of the reasons that caregivers of female patients report higher SCB and anxiety.[49] Perception of subjective burden increases when caregivers provide care to patients with severe disability. For instance; one study demonstrated that severity of burden, anxiety, and depression are comparatively higher among those caregivers who provide care to patients with profound ID.[50]

Limitations

First relatively small sample size raises the concerns of the study's generalizability. The experiences and challenges of caregivers may vary widely, which requires a larger and more diverse sample of caregivers to ensure the applicability of the results. Additionally, the study's reliance over self-reporting measures produces response bias among caregivers. caregivers may overrate or underrate their symptoms of anxiety and burden due to social desirability of responses and memory inaccuracies. This bias may compromise the accuracy of data collected and the validity of the results. Furthermore, present study is a cross sectional design which does not reflect the causality of the symptoms of anxiety and burden over a period of time. Caregiving experiences are dynamic and can evolve over months or years. A longitudinal approach would provide a more comprehensive understanding of how anxiety and caregiving burden fluctuate and interact over time. Future researchers should address all these limitations and provide more comprehensive understanding of this important context of caregiving.

Conclusion

Most of the patients were males, from adolescent & adult age group, and suffering from mild ID. Caregivers of ID patients were prominently married, females, housewives, and mothers of the patients. SCB and anxiety was way prevalent among all the caregivers providing care to the patients with ID but caregivers of the patients with profound ID reported significantly higher SCB and anxiety in comparison to the other caregivers. Both SCB and anxiety among caregivers were significantly associated with patient's age group & gender, severity of ID, and their relationship with the patients. SCB was significantly associated with marital status and religion of caregivers. Findings of the present study will further provide researchers an insight to explore the reason why caregivers with certain characteristics are prone to SCB and anxiety. Clinicians should focus on the caregivers along with the patients with ID to enhance their global well-being. This study will help researchers and mental health professionals to address psychological problems among caregivers at an earlier stage and to further manage them effectively at an initial stage.

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Original Article

A Study Exploring the Pathways to various Consultation for Acute and Transient Psychosis

Gourav Sanmotra ,Tushar Jagawat, Ritu Meena, Pankaj Tandon, Preet Kamal, Savita Jagawat, Yashika Gupta
Department of Psychiatry & Clinical Psychology, NIMSR, Jaipur, Rajasthan
Contact:Tushar Jagawat, Email: tusharjagawat@yahoo.com

ABSTRACT

Background: *Acute and Transient Psychotic disorders with favourable outcomes are acknowledged both in the ICD-10 (World health organization 1992) and as separate entities from Schizophrenia and affective psychosis. There are few studies in India in relation to pathway for psychiatric consultation for acute psychosis.* ***Aims and Objectives****: To study the Socio-demography, Clinical characteristics and Pathway to psychiatric consultation for Acute and Transient Psychotic disorder.* ***Materials and Methods:*** *70 subjects of age 18 years and above attending psychiatry OPD/ IPD and emergency were assessed.The socio-demographic and clinical profile were analyzed. The various routes of pathways before opting for psychiatric consultation were noted.* ***Results:*** *Out of 70 participants, majority (n=40, 57%) were females, in 20-40 years of age (n=37, 52%) from rural areas (n=56, 80%). Family history was present (n=20, 28%). Majority had precipitating factors (n=42, 60%), out of which life events was predominant (n=24, 34.2%), Financial factors (n=4, 5.7%) and substance factors (n=6, 8.5%). In the first consultation sought by patients, the majority (n=38,54%) of individuals turned to faith healers as their initial point of contact. Local practitioners were consulted by 22.8% of patients, 14% of individuals approached psychiatric services for their first consultation. Duration of first psychiatric consultation (n=44, 62.8%)took treatment within 7 to 15 days after onset. Brought to psychiatric centres by relatives (n=31, 44.2%) followed by neighbours/ friends (n=26, 37.1%) . The first symptom observed was disturbed sleep (n=19,27.1%) followed by Irrelevant talk (n=10, 14%).* ***Conclusion:*** *Findings underscore the relevance of triggers like life events, financial issues, and substance use. Family history's role, though variable, is noteworthy. It also out factors the various pathways of first point of contact before seeking psychiatric consultation and timely intervention's importance is evident from the duration of untreated illness findings.*

Keywords*: Acute and Transient Psychosis, Pathways to Psychiatric consultation.*

Introduction

Acute and transient psychotic disorders (ATPD) are defined as a heterogeneous group of disorders, with acute onset of psychotic symptoms such as delusions, hallucinations, perceptual disturbances and by the severe disruption of ordinary behaviour. The key features are an acute onset (within 2 weeks), presence of typical syndromes which are described as rapidly changing, polymorphic states and typical schizophrenic symptoms, evidence for associated acute stress in a substantial number of cases and complete recovery in most cases within 2-3 months.

The landscape of psychiatric classifications has witnessed significant transformations over time, with key milestones like the introduction of the ICD-10 marking pivotal moments in the field.[1] One such transformative shift involved the conceptualization of acute psychosis, which initially found itself within the broader category of schizophrenia. This narrative, however, underwent a paradigm shift, through

observations made in the Indian context by scholars like Wig and Singh.[2] Their work revealed the existence of acute psychosis and also highlighted that it is as a distinct clinical entity characterized by sudden onset, vivid symptomatology and a promising prognosis.The scientific journey is enriched by empirical evidence, exemplified by Susser and Wanderling's seminal work in 1994.[3] Their research highlighted the distinct epidemiological attributes of acute brief psychoses, coupled with a favourable long-term trajectory. This body of evidence solidifies ATPD's position as a unique clinical entity and reinforces its distinct classification within the ICD-10. This evolution of ATPD's recognition, particularly within the Indian context, is remarkable. Transitioning from an ambiguous classification within the shadow of schizophrenia, it has emerged as an independent disorder with its own defining characteristics. The hallmark features of sudden onset and brief duration, backed by compelling epidemiological evidence, establish ATPD's distinct identity in the realm of psychiatric disorders. Understanding the period between the onset of first psychotic symptoms and treatment initiation, known as the duration of untreated psychosis, is a critical concern for the mental health system. Longer duration of untreated psychosis are linked to negative outcomes and functional impairments. Efforts to comprehend this phenomenon have focused on identifying delays along the care pathway, which is shaped by various factors, including help-seeking behaviours, family influence, stigma, knowledge about mental health care, and interactions with healthcare providers.

Materials and Methods

- The study was conducted in psychiatry OPD/IPD and emergency department at tertiary care hospital. The study was conducted after permission from Scientific and Institutional Ethics Committee. It is a cross sectional study which included sample size of 70 participants in the age group of 18-60 years. A voluntary written informed consent was taken for participation from all subjects/relatives/informant after explaining the purpose and design of the study.
- To study the socio-demographic variables and its clinical correlation with the symptoms.
- The diagnosis of Acute and transient psychotic disorder in accordance to ICD-10[1]
- Various precipitating stress, factors, family history and duration of delay in psychiatric consultation were noted.
- The pathways of seeking help were analysed along with major symptoms for consultation were assessed via Encounter form.

Table-1: Sociodemographic variables (n=70)

Variables		N=70	%
Sex	Male	30	42.8
	Female	40	57.1
Age	<20	3	4.2
	20-40	37	52.8
	>40	30	42.8
Religion	Hindu	48	68.5
	Muslim	20	28.5
	Other	02	2.8
Domicile	Rural	56	80
	Urban	14	20
Education	Illiterate	36	51.4
	Primary	17	24.2
	Middle to higher secondary school	11	15.7
	Graduate and Post graduate	06	8.57
Occupation	Unemployed	48	68.5
	Skilled worker/ Semiskilled worker	11	15.7
	Professional	03	4.2
Marital status	Married	58	82.8
	Unmarried	12	17.1
Family type	Nuclear	32	45.7
	Extended Nuclear	12	17.1
	Joint	26	37.1

Table 1 shows the sociodemographic distribution of participants. Maximum number of patients (n=40, 57%) were females, among religions most of them belonged to Hindu religion (n=48, 68.5%), most of them were illiterate (n=36, 51%), married (n=58, 82%), unemployed (n=48, 68%), belonging to nuclear family (n=32, 45%), from rural background (n=56, 80%).

Table 2 Family history was present in 20 Patients (28%). In terms of precipitating stress, 42 participants experienced such stressors, while 28 reported the absence of such trigger. Among precipitating factors, Life events stood out as the most common (34.2%), followed by substance

Table-2: Precipitating factors, family history & duration of illness

Variable	N=70	(%)
Precipitating stress		
Present	42	60.0
Absent	28	40.0
Medical Factors	08	11.4
Life events	24	34.2
Financial Factors	04	5.7
Substance Factors	06	8.5
Family history-Present	20	28.5
Absent	50	71.4
Duration of First Psychiatric consultation (days)		
1-7	10	14.2
8-15	44	62.8
16-30	16	22.8

Table 3: Pathways to seek help for psychiatric symptoms

S.No.	Response		N = 70	%
1.	First consultation/Opinion	Native/faith healer	38	54
		Local practitioner	16	22.8
		General hospital	6	8.0
		Psychiatric service	10	14.0
2.	Who initiated for first contact	Patients himself	13	18.5
		Relatives	31	44.2
		Neighbours/Friends	26	37.1
3.	Distance from the Psychiatric services	0-10 kms	14	20.0
		11- 30 kms	22	31.4
		31 kms and above	34	48.5
4.	What symptoms caused decision to seek care	Irrelevant talk	14	20
		Excessive talk	2	2.8
		Disturbed Sleep	19	27.1
		Restlessness	7	10.0
		Fearfulness	12	17.1
		Disorganised behaviour	20	28.5
		Abusive behaviour	14	20.0
		Aggressive/Violent/Hostile behaviour	16	22.8
		Wandering behaviour	15	21.4
		Muttering to self	17	24.2
		Suspiciousness	14	20.0
		Suicidal attempt	3	4.2

related (8.5%) and financial factors (5.7%). 10 (14.2%) patients seeking Psychiatric treatment within a week of symptom onset, 44 (62.8%) took treatment within 7 to 15 days, and 16 (22.8%) patients opted Psychiatric treatment within 16-30 days.

Table 3

- The table shows the first consultation or opinion sought by patients, the majority (54%) of individuals turned to faith healers, Local practitioners were consulted by 22.8% of patients, while 8% sought help from general hospitals. 14% of individuals approached psychiatric services for their first consultation.
- The findings showed that 44% of participants were prompted by relatives, while 37% reached by neighbours or friends. Furthermore, 18.5% of individuals took the initiative themselves to seek assistance for their distress.

Among the reported symptoms, 28.5% of patients were influenced by disorganized behavior, 27.1% were driven by disturbed sleep, and 24.2% considered muttering to oneself as a key factor. Other symptoms that influenced the decision to seek care included aggressive or hostile behavior (22.8%), wandering behavior (21.4%), and irrelevant talk (20%). Notably, some participants reported symptoms of fearfulness (17.1%) as factors prompting them to seek help. A smaller percentage (4%) mentioned having made a suicidal attempt as a symptom driving their decision.

Table 4 shows the first symptom observed in the patients by the relatives are disturbed sleep - 27.1%, Irrelevant and excessive talk 14.2%, Aggressive behaviour-10% followed by Hallucinatory behaviour, fearfulness, suspiciousness and self harm

Table-4: First symptoms observed in the patients

Sr.No.	Symptoms	N = 70	
		N	%
1.	Excessive/Irrelevant talk	10	14.2
2.	Hallucinatory behaviour	06	8.5
3.	Disturbed Sleep	19	27.1
4.	Restlessness	4	5.7
5.	Fearfulness	5	7.1
6.	Disorganised behaviour	4	5.7
7.	Abusive behaviour	4	5.7
8.	Aggressive/Violent/ Hostile behaviour	7	10
9.	Wandering behaviour	4	5.7
10.	Muttering to self	3	4.2
11.	Suspiciousness	2	2.8
12.	Seif harm behaviour	2	2.8

behaviour, 2.8%.

Discussion

Our study had a cross sectional design and patients were included by purposive sampling technique. A total of 70 patients were included for the study. In our study the maximum patients age were range from 20-40 years which was similar to other studies,[4-10] with majority of them were females which was similar to previous studies[4,5,11] and different from some studies[6-8] in which males were predominant. In terms of Religion majorities were Hindus (78%) which was similar to previous Indian studies.[4-7]

Furthermore in our study, 51% were illite-rate to which was different from Khinchi et al[6] and Singh et al.[4] 82% were married which was similar to Mehta et al,[9] 68% were unemployed similar to other studies[6,10] and 45% were living in a nuclear family which was similar to some previous studies.[5,6,12]

Majority were residing in rural areas. This was also reported in others studies[6,8-10] but different from Singh et al.[4] Thus indicating that psychosis is common in low socio-economic families.

In our study 28% patients are having positive family history similar to Chandak & Gowda,[11] Mehta et al[9] and which differs from Jilani et al.[8] 60% were having precipitating stress as a major cause similar to Khinchi et al[6] and differs from Mehta et al.[9] Among these factors, life events was one of the reason behind aggravating psychosis, followed by substance and financial factors which was similar to Khinchi et al[6] and different from Mehta et al.[9] The duration of untreated psychosis were analysed with 14.2% patients seeking treatment within a week of symptom onset, Most of the pattients 62.8% consulted in 2nd week which different from Mehta et al,[9] which was a retrospective study on admitted patients and 22.8%, waiting for over 15 days before seeking medical attention. While seeking help for their first episode of psychosis majority of them had a first point of contact with faith healers 54% as reported in other studies.[4,5,7,8] This was followed by contact with local practitioners similar to Jilani et al[8] and Kumar and Kiran;[7] and visit to general hospitals 8%. Only 14% of the patients, consulted psychiatrist for first point of health which is different from Singh et al[4] where the sample was taken from institutes situated in the city. The distance from the psychiatric services are maximum for above 30 kms (48.5%) followed by 11-30 kms from the patients residence which may be because of lack of mental health services in rural areas.

The study also examined into who took the initiative to commence the first contact for seeking help. The findings showed that 44% of patients were prompted by relatives, while 37% reached by neighbours or friends which is different from the Kumar and Kiran[7] where patients himself initiated first contact, most of the studies have not taken this variable. Furthermore, 18.5% of individuals took the initiative themselves to seek assistance for their distress. In the various reported symptoms, 28% of participants were disturbed by disorganized behaviour, 27% were facing disturbed sleep which was similar to Chandak and Gowda[11] different from Kumar and Kiran[7] and 17.1% considered muttering to oneself as a key factor. Other symptoms that influenced the decision to seek care included aggressive or hostile behaviour (22.8%), wandering behaviour (21.4%), and irrelevant talk (22%) differentiating our study from Kumar and Kiran[7] in which decreased talk was the most common symptoms. Notably, some participants reported symptoms of fearfulness (17.1%) and abusive behaviour (20%) as factors prompting them to seek help.

A smaller percentage (4%) mentioned having made a suicidal attempt as a symptom driving their decision. The first symptom observed in the patients by the relatives are disturbed sleep -27.1%, Irrelevant and excessive talk 14.2%, Aggressive behaviour -

10% followed by Hallucinatory behaviour, fearfulness, suspiciousness and self harm behaviour, 2.8% which is different from the study by Kumar and Kiran[7] where fearfulness was the first symptom (13.5%), decreased talk -11.5%, followed by Disturbed sleep -8.3%, poor self hygiene, perceptual disturbances, unusual thoughts and disturbed work was 1%. Self harm behaviour was not found as first symptom in the study.

Limitations

Our study is limited by small sample size that might reduce the power leading to type II errors. Secondly our study used cross sectional study design that can raise the question about the generalisability of our results. Thirdly the duration of untreated psychosis above one month were not included. Fourthly our study only included those the patients who presented to the mental health professionals and finally no follow up of treated psychosis were done.

Conclusion

The distribution of patients' sociodemographic profiles reveals that the majority were females, belonged to the Hindu religion, considerable number had an illiterate background. Marriage rates were high (82%) and most patients were unemployed (68.5%) participants were from nuclear families. Family history also played a role, being present in a subset of cases. Among 70 patients, 60% experienced precipitating stress, while 40% did not. Notably, individuals had a family history of psychiatric disorders, whereas did not. 14.2% patients sought early treatment within a week of symptom onset, 62.8% delayed treatment for 7 to 15 days, and 22.8% waited for over 15 days before seeking medical help. In summary, this study delved into crucial aspects of help-seeking behaviours' and initial care for individuals confronting psychological distress. The study revealed that faith healers were the primary initial contact for the majority (54%), while local practitioners and general hospitals were sought by 22.8% and 8% of participants, respectively. Relatives (44%) and neighbours'/friends (37%) were instrumental in initiating contact, and a notable 18.5% took personal initiative. Various symptoms influenced care-seeking, with disorganized behaviour (28.5%), disturbed sleep (27.1%), and muttering (24.2%) being significant. Aggressive behaviour (22.8%), wandering (21.4%), and irrelevant talk (20%) also impacted decisions, alongside fearfulness (17.1%) and abusive behaviour (20%). A smaller proportion (4%) cited suicidal attempts as triggers. Overall, these findings provide essential insights into the initial care-seeking process for psychological distress, emphasizing the role of cultural factors, social networks, and diverse symptoms in shaping individuals' choices.

In summary, this study yields insights into sociodemographic characteristics, precipitating factors, and family history related to acute and transient psychotic disorder (ATPD). Findings underscore the relevance of triggers like life events, financial issues, and substance use. Family history's role, though variable, is noteworthy. It also out factors the various pathways of first point of contact before seeking psychiatric consultation and timely intervention's importance is evident from the duration of untreated illness findings.[12] This collective information deepens our understanding of ATPD's complexity, informing targeted management approaches.

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Original Article

Typology and Prevalence of Psychosis, Bipolar affective disorder and Depression; by study of inpatient documents of a psychiatry ward in a tertiary care hospital: A Retrospective study

Om Panda,[1] Priyadarshee Patra,[2] G. Madhusudhan[3]
[1,2]Department of Psychiatry, INHS Asvini
[3]Department of Psychiatry, Military Hospital, Jammu
Contact: Om Panda, E-mail: ompanda6172@gmail.com

ABSTRACT

Aim: *To study the (1) Typology and prevalence of Psychosis in tertiary care center. (2) Typology and prevalence of Bipolar Affective disorder in tertiary care center. (3) Typology and prevalence of depression in tertiary care center.* ***Methodology:*** *A cross-sectional retrospective file review was carried out for all inpatient documents, freshly or previously diagnosed with Psychosis, BPAD or depression according to ICD-10 criteria, in psychiatry ward, in tertiary care hospital from 15 Sep 2021 to 14 Sep 2022. Socio-demographic, clinical and relevant assessments data were extracted from electronically saved opinions of Psychiatrists. Ms-Excel sheet was used to extrapolate data gathered and subsequently was analyzed by SPSS. Prevalence of above-mentioned illnesses was calculated. Descriptive analysis was used for socio-demographic and clinical data.* ***Results****: 12 month prevalence of schizophrenia 27.5%, schizoaffective disorder 1.7%, delusional disorder 22.4%, acute and transient psychotic disorder 10.3%, psychotic disorder due to an organic condition 6.8% and substance induced psychotic disorder 3.4% of all Psychotic disorder was found. Bipolar affective disorder (BPAD) I constituted 46.8% of all bipolar cases in 12 month period whereas BPAD II constituted 53.2% of the same. About 17% of patients were diagnosed with depression and 12% had severe depressive features.* ***Conclusions:*** *Findings underscore the important public health significance of depression, BPAD and Psychosis among Psychiatry inpatients and the need for improvement in screening and treatment access in this population. Pharmacotherapy used also differs with type and severity of depression. Moreover, depression in bipolar affective disorders and unipolar depression varies in their management. Hence knowing prevalence and typology of depression helps in better management strategies and hence leads to effective outcomes.*

Keywords: *Typology, Psychosis, Bipolar affective disorder, Depression*

Introduction

The International Statistical classification of Diseases and related health problems 10th edition (ICD-10), in its classification of Mental and Behavioral disorders has laid down criteria and explanation for Psychiatry illnesses. Psychosis is a mental disorder in which ability to recognize reality, ability to communicate and relate to others are sufficiently impaired to interfere grossly with capacity to deal with reality. This includes schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, acute and transient psychotic disorder, psychotic disorder due to an organic condition and substance induced psychotic disorder. World-

wide prevalence rates remains constant (1-3%).[1] The bipolar affective disorders (BPAD) commonly includes BPAD I commonly referred to as manic depression, BPAD II and cyclothymia.[2,3] The lifetime prevalence estimates of bipolar I (BP I) disorder is 3.3, Bipolar II (BP II) of 1.1 with corresponding 12 month rates of 2.0 and 0.8, respectively.[1,4,5] Depression is quite prevalent and an estimated 5% of the adult world population has suffered from depression. It is estimated as high as 7% in US. Lifetime prevalence according to latest studies is 13.2% whereas 12-month prevalence is 5.3%.[6-8] ICD-10 divides it into mild, moderate, severe and recurrent, with or without Psychotic symptoms. As limited literature is present on Indian population, this retrospective study aims to assess prevalence and typology of above-mentioned disorders in past one year, to allocate human resources and develop specialized center in tertiary care setup

Methodology

A cross-sectional retrospective file review was carried out for all inpatient documents of Psychiatry ward in a tertiary care Industrial hospital. All the patients were part of a Industrial station located in western region of India.

Inclusion criteria

1. All freshly or previously diagnosed cases with relapse symptoms of Psychosis, BPAD or depression were included. (All diagnosis were in accordance with ICD-10 criteria).
2. The study included opinions given between 15 Sep 2021 and 14 Sep 2022.

Exclusion criteria

1. All opinions preceding or succeeding the dates were excluded.
2. Opinion of patients re-admitted for routine checkup was omitted.

Socio-demographic, clinical and relevant assessments data were extracted from electronically saved opinions given by Psychiatrists. Tabulation of extrapolated data gathered was done and subsequently analyzed. 12-month prevalence of above-mentioned illnesses was calculated. Descriptive analysis was used for socio-demographic and clinical data.

Results

A total of 656 patients, who fulfilled inclusion criteria and were discharged from Psychiatry ward of this tertiary care hospital, between 15 Sep 2021 to 14 Sep 2022 were selected. Out of these 214 patients (~33 %) were diagnosed with BPAD, Psychotic disorder or depression. Amongst them 02 were female (~1%) and rest male. 58 patients (8.8% of total) were diagnosed to have Psychosis, 47 patients were diagnosed with BPAD (7.1%) and 109 were diagnosed with depression (17%). [Table 1]

Table-1: Presence of BPAD, Psychosis and Depression

Disorder	Total number	Percentage
Psychosis	58	8.8
BPAD	47	7.1
Depression	109	17

Prevalence of different typology of psychosis

Of the total diagnosed patients with psychosis, 27.5 % patients (16 patients) had Schizophrenia and similar were diagnosed with Severe depressive disorder with Psychotic symptoms. 22.4% (13 patients) were given a diagnosis of persistent delusional disorder. Only 1.7% (01 patient) was diagnosed with schizoaffective disorder. [Table 2]

Prevalence of different typology of depression

17% were diagnosed with some depression. Out

Table-2: Distribution of various typology of Psychosis

Typology of Psychosis	Total number	Percentage (%)	% of total admissions
Schizophrenia	16	27.5	2.4
Schizoaffective	01	1.7	0.15
Persistent delusional disorder	13	22.4	1.9
Acute and transient psychotic disorder	06	10.3	0.9
Severe depressive episode with psychotic features	16	27.5	2.4
Other (unspecified and organic) psychosis	04	6.8	0.6
Substance induced psychosis	02	3.4	0.3

of these, maximum (77 patients, 70.6%) were diagnosed with moderate depressive disorder. Recurrent depressive disorder, was diagnosed in 08 patients (7.4%). Dysthymia was diagnosed in 04 patients (3.6%) and mixed anxiety and depressive disorder in 05 (4.5%). [Table 3]

Prevalence of different typology of BPAD

Prevalence for both BPAD 1 and BPAD 2 was almost equal with 25 patients (53.19%) diagnosed with BPAD 2 [Table 4].

Table-3: Distribution of various typology of Depression

Typology of Depression	Total number	Percentage (%)	% of total admissions
Mild depressive episode	02	1.8	0.3
Moderate depressive episode	77	70.6	11.7
Severe depressive episode without Psychotic features	13	11.9	1.9
Recurrent depressive disorder	08	7.3	1.2
Dysthymia	04	3.6	0.6
Mixed anxiety and depressive disorder	05	4.5	0.7

Table-4: Distribution of various typology of BPAD

Typology of BPAD	Total number	Percentage (%)
BPAD 1	22	46.80
BPAD 2	25	53.19
Cyclothymia	00	0.00

Discussion

Socio-demographic profile of cohort revealed predominance of male population (99.7%), with 70% of them below the age of 40 years. Predominance of younger cohort could be attributed to improved and accessible health facilities in Industrial station and regular check-ups in Industrial set-up. It also revealed almost 3/4th of the cohort being referred from organizational institutions, either under routine check-ups or urgent referral, establishing the higher standards of medical care being provided. These led to decreased severity of illnesses. Majority of the patients were educated between 10th and 12th standard. The total number of Psychosis cases was

Table-5: Socio-demographic profile of cases

Domain	Sub-domain	Number	%
Gender	Male	654	99.7
	Female	02	0.3
Age (yrs)	20-30	167	25.4
	31-40	292	44.5
	41-50	169	25.7
	>50	28	4.4
Education	<10th std	190	28.9
	10-12th std	414	63.1
	Graduate	49	7.4
	Post-graduate	03	0.4
Referral	Organized medical evaluation	356	54.2
	Urgent referrals	132	20.2
	On leave, but reported to organizational hospital	111	17
	Follow up post treatment outside organization	57	8.6

8.8%, which is comparable to other studies.[2,9,10] The diagnosis of delusional dis-order was made for 1.9 % of patients which is com-parable to previous studies stating 1-2%.[2,9] The rates of schizoaffective disorders are also comparable to previous data.[2,11,12] As compared to previous studies, schizophrenia has been diagnosed for only 2.4% of total patients with psychosis, as against 10%.[2,9,11] The reason for the same could be because of younger age of admitted patients in this tertiary care hospital. Almost 70% of patients were below age of 40 years. It can also be attributed to the fact that all these patients came from the same cohort which worked under one organization. Most of the patients under-went regular medical review and were under constant medical supervision. Robust medical cover may have prevented the conversion of psychosis to full blown schizophrenia, which could have led to reduced representation of schizophrenia. Also since psychotic depression was subsumed under psychosis, it could be a contributing factor for reduction in schizophrenia representation. The percentage of diagnosed BPAD cases was 7.1%, which was comparable with lifetime prevalence rates of available studies.[4,5,13,14] The prevalence rates of BPAD I and II were almost similar, comparable to earlier studies.[3,15,16] Depression was diagnosed in ~17% of the patients, which is slightly less as compared to previous data.[6-8] Being in an industrial set-up with regular community

awareness programs and availability of wellness centers and peer oriented mentor system, could have led to fewer admissions for depression. Also, prevalence of depression is almost two fold in women as compared to men. Due to service exigencies, there is a predominance of men in the study sample, leading to reduced percentage of patients with depression as compared to other studies which includes women as well. Most studies[7,17-20] have been done on major depressive disorder and data could not be found on mild, moderate or severe depressive disorder, however our results revealed moderate depressive episodes were maximum in number (77, 70.6%). Industrial medical echelons are very pro-active, not only in providing treatment, but also in prevention and awareness strategies. These could have led to earlier admissions in individuals developing symptoms of depression, hence stalling progression to severe depressive episodes.

Limitations

Our study was a cross-sectional descriptive study which comes with it an inherent set of limitations. In addition, it was of relatively short duration, and being a hospital-based study, it would be unfair to try and generalize out findings in the community.

Conclusion

This study has revealed lower cases of schizophrenia and depression as compared to previous conducted studies amongst psychiatry inpatients. This could be a reflection of successful community outreach program on awareness on mental health. It also establishes the equity in prevalence of BPAD I and II and need for more research on the same. Pharmacotherapy used also differs with type and severity of depression. Moreover, depression in bipolar affective disorders and unipolar depression varies in their management. Hence knowing prevalence and typology of depression helps in better management strategies and hence leads to effective outcomes.

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Original Article

Cognitive and Psychosocial Functioning in Patients with Schizophrenia in Remission – A Comparative Study

Shivangi Mehta, Navkiran Sooch Mahajan, Ranjive Mahajan
Department of Psychiatry, Dayanand Medical College and Hospital, Ludhiana
Contact: Shivangi Mehta, E-mail: shivangi02@gmail.com

ABSTRACT

Background: *Schizophrenia is known to be a neurodegenerative disorder with progressive deteriorating course. The definition of remission and recovery are difficult to conceptualize leading to concept of functional remission.* ***Methodology****: After approval from research and ethics committee of a tertiary care hospital of Northern India, the study was conducted at outpatient department of Psychiatry. Thirty patients (age 18-60 years) diagnosed with Schizophrenia as per ICD-10 Diagnostic Criteria for Research, found to be in remission since 1 year using PANSS scale (following the Revised Schizophrenia Working Group criteria for remission) were assessed on PGI Memory and Cognitive functioning scale and Dysfunction Analysis Questionnaire .* ***Results and Conclusion****: The deficits in cognition persisted in attention, working memory and executive functioning and corelated with psychopathology and dysfunction at one year of remission. Hence, the cognitive deficits are trait marker of Schizophrenia and persist despite symptomatic remission.*

Keywords: *Schizophrenia, Remission, Cognitive deficits, Trait marker, Executive functioning.*

Introduction

Schizophrenia, since the time of Kraepelin, has been considered to be a progressive deteriorating illness.[1] The recent literature world-wide points towards the better outcomes in people with Schizophrenia. The definition of outcome in the form of remission and recovery have been exigent. To define functionality for a lifelong disorder is a rather difficult task. Despite the introduction of effective pharmacological treatments and evidence-based psychosocial interventions, this pervasive disease leads to a significant residual morbidity occurring through a process of behavioural deterioration.[2] As a result, fewer than one in seven people affected are considered to meet criteria for recovery.[3]

Since recovery seemed nearly implausible to attain in Schizophrenia , the scientists proposed the term 'Functional remission'.[4] The definition of functional remission is still difficult to consolidate. The known outcome of symptomatic remission with antipsychotics doesn't encompass the concept of functional recovery in its entirety. Even if full functional recovery is attained, its maintenance even after symptoms have remitted seems quite rare.[5] Profound impairments persist despite symptomatic remission and these are generally evident in the domains of occupation, social relationships, family responsibilities and cognition, thus hampering the independent living in persons with Schizophrenia and adding to the caregiver burden.

The concept of remission in schizophrenia, as proposed by the Remission in Schizophrenia Working Group (RSWG),[6] is defined as: A state in which patients have experienced an improvement in symptoms to the extent that any remaining symptoms are of such low intensity that they no longer interfere

significantly with behaviour and are below the threshold typically utilized in justifying a diagnosis of Schizophrenia. The RSWG further outline the remission under two components — symptom severity and time — the symptom severity from established scales including the Positive and Negative Syndrome Scale (PANSS), the RSWG provides a score of ≤ 3 points corresponding to mild severity or less on the eight items of the PANSS-8: P1 (delusions), P2 (conceptual disorganization), P3 (hallucinatory behavior), N1 (blunted affect), N4 (social withdrawal), N6 (lack of spontaneity), G5 (mannerisms and posturing), and G9 (unusual thought content). Those items are assumed to map onto three dimensions of psychopathology: psychoticism (P1, P3, and G9), disorganization (P2 and G5), and negative symptoms (N1, N4, and N6). And for the component of time, the criteria for symptom severity need to be met for at least 6 months as per RSWG. Since their publication, the RSWG have been widely employed to define *remission* in psychotic disorders. Recent empirical research has consistently demonstrated that although psychopathological symptoms disrupt patients lives, deficits in cognitive functioning have the strongest influence on their overall level of independent functioning.

Cognitive functioning and impairments in activities of daily living are two major concerns in persons suffering from Schizophrenia. The integration of pharmacological and psychosocial interventions has made it possible to redefine outcome measures integrating clinical and psychosocial parameters, indicating that therapeutic efforts should be oriented towards achieving symptomatic remission and improving psychosocial functioning.[7] Cognitive deficits in various domains have been consistently replicated in patients with Schizophrenia.

Persons suffering from schizophrenia frequently have significant difficulties in everyday functioning. It has been estimated that as many as two-thirds of schizophrenia patients are unable to accomplish psychosocial roles, even when psychotic symptoms are in remission. Psychosocial remission is a new concept that has been recently introduced and recommended for measuring impairments — a core feature of schizophrenia. Psychosocial impairments are expressed in daily living skills (e.g., personal hygiene, interest in daily life, family relations), and in various occupational, social, and community settings.[7] These impairments might contribute to disability.

Functioning and symptomatic remission can be considered two relevant outcome measures leading to functional remission. Functioning is another important contributor to functional remission since it is related to personal, social, and occupational role functioning. However, the assessment of functioning is a complicated issue, since there is no consensus on evaluating functioning in schizophrenia and it is unclear what constitutes "appropriate functioning".[8]

Most research evaluating the relationship between cognitive dysfunction and disability stem from developed countries and very few from developing countries. The present study aims to compare the cognitive functions in patients with schizophrenia currently in remission with that of normal age matched controls and to determine if there is a relationship between cognitive functioning and social dysfunction.

Methodology

After approval from research and ethics committee of the institute, the study was conducted at outpatient department of Psychiatry, of a tertiary care hospital in northern India. Thirty patients (age 18-60 years) diagnosed with Schizophrenia as per ICD-10 Diagnostic Criteria for Research[9] and found to be in remission using PANSS scale[10] (following the Revised Schizophrenia Working Group criteria for remission[6]), no psychiatric hospitalization in the past one year, no change in antipsychotic medication or ECT or cognitive training in the last 6 months, no history of severe and unstable medical disease, Intellectual Disability and Pervasive developmental disorders and cognitively able to give informed consent were recruited for the study. Thirty age matched subjects were taken from the general population with no psychiatric impairment as per ICD-10 Diagnostic Criteria for Research, fulfilling the inclusion and exclusion criteria, were taken up for the study.

Procedure

Collection of the socio-demographic details from 30 patients of Schizophrenia and 30 controls, was done. Thereafter , Cognitive Functioning Scale[11] and PGI Memory Scale[12] were applied to assess the

cognitive functioning. Then, Dysfunction Analysis Questionnaire[13] – Hindi version was applied to assess the social disability in both the groups. Finally, the results from both the groups were compared and assessed using appropriate statistical analysis as specified below.

All subjects who were included in the study were assessed once using the specified tools that took about 1 hour. Assessments were performed in a fixed order in a quiet room by SM. At the subject's request, a short break was permitted halfway through the assessment. Patients were not allowed to smoke or consume stimulant drinks during the assessment. The last dose of medication was taken at least 6 h before the testing.

Tools

Socio-Demographic Performa

To assess the age, sex, educational status, occupational status, type of occupation, marital status, type of family and time since remission, a predesigned format was used.

Cognitive Functioning Scale[11]

The Cognitive Functioning Scale is designed to provide a brief assessment of cognitive functioning in patients with Schizophrenia as revealed in their day to day functioning. It is organized into two sections. The first section assesses the functioning using a series of fifteen items specifically tailored to sample functioning related to seven distinct cognitive domains-Working memory, Attention and vigilance, Verbal learning and memory, Spatial learning and memory, Reasoning and problem solving, Speed of processing, Social cognition. For each of these items, a number of probing questions are suggested that attempt to clarify functioning in these areas and are graded on a score continuum of 1 to 7 graded as per dysfunction. The second section contains three global questions (Global severity of cognitive impairment, Global impression of change in cognitive functioning, Global assessment of cognitive function). The ratings should represent functioning in the past month.

PGI Memory Scale[12]

PGI memory is used to determine memory function in patients. It determines Remote memory, Recent memory, Mental balance, Attention and Concentration, Delayed recall, Immediate recall, Verbal retention of similar Pairs, Verbal retention of dissimilar Pairs, Visual retention, and Recognition. PGI memory scale was found to have a correlation of .71 with Boston memory scale and .85 with Wechsler memory scale.

Dysfunction Analysis Questionnaire[13]

Dysfunction Analysis Questionnaire(D.A.Q.) was developed to fulfil the needs of comparison of subjects belonging to different strata and to compare the individual himself with his pre-morbid level with regard to psychological functioning in the five domains of life-Vocational (V), Social (S), Personal (P), Familial (F) and Cognitive functions (C). Higher score indicates greater dysfunction in each area. Scores on each of the five areas or the average of the five areas suggest no disability if it is up to 40; mild disability if between 41-60; moderate disability if between 61-80; and severe disability if between 81-100.

Statistical Analysis

Data entry and cleaning was done using MS Excel software. The final data was analyzed using SPSS software. The categorical data was expressed in frequencies and percentages, and the continuous data was expressed in mean with standard deviation. ANOVA and t-tests were used. The Pearson correlation test was used to assess the correlation between the quantitative variables except education level for which Spearman correlation analysis was done. All inferential statistics were carried out at a confidence level of 95% with a p-value less than 0.05 being significant.

Ethical issues

Patients were enrolled as per protocol approval from Institutional Ethical Committee after obtaining informed written consent from all subjects and no extra cost was inferred to the individual taken up for the study.

Results

The study was conducted on a total of 60 subjects

Subject characteristics

Equal proportion of males and females were found in both the groups. The mean age of each

group was 29.47 ± 11 years but the years of education and number of married participants was higher in the control group. The mean duration of illness was 11.3 ± 5.8 years, ranging from 2 to 27 years. A total of 33.33% of the patients were unemployed as compared to 13.33% of the controls. Details of medication are shown in supplementary Table 1. A total of 86.66% of the patient group were on an atypical antipsychotics.

Table-1: Sociodemographic Profile

Characteristic	Cases (n=30)	Controls (n=30)	p-Value
Mean Age ± SD (years)	29.47 ± 11	29.47± 11	
M:F	1:1	1:1	
Educational Status			0.028
Illiterate	2	0	
<12 years of	17	8	
Education	11	22	
>12 years of Education			
Marital Status			0.27
Married	8	13	
Unmarried	19	17	
Widowed	1	0	
Divorced	2	0	
Type of Family			0.204
Nuclear (% age)	25 (83.3%)	28 (93.3%)	
Joint	5 (16.7%)	2 (6.67%)	
Occupation			
Farmer	4 (13.34%)	0 (0%)	
Service	3 (10%)	9 (30%)	
Shopkeeper	7 (23.3%)	1 (3.33%)	
Ex-Service	1 (3.3%)	0 (0%)	
Student	5 (16.67%)	16 (53.34%)	
Unemployed	10 (33.33%)	4 (13.3%)	0.008

Cognitive functioning

Results are reported according to the domains of cognition tested (Table 2). Overall, the mean values obtained for total scores were higher in controls (87.70 ± 6.36) than the cases (75.67 ± 9.62) showing increased time taken by cases to complete the test and the difference was statistically significant. Patients with schizophrenia scored less well than controls on all tests of cognition, except recent memory and retention for similar pairs where the difference in scores is not statistically significant. Similar findings were seen on Cognitive Functioning Scale, statistically significant better cognitive functioning is seen on all the domains in controls than schizophrenia patients despite remission (Table 3). Overall, the mean values obtained for total scores were higher in cases (42.57 ± 11.64) than the controls (28.43 ± 3.98) and the difference was statistically highly significant ($p<0.001$).

Dysfunction assessment

In Dysfunction Analysis Questionnaire[13] (Table 4) the comparison of the cases and the controls show greater dysfunction on all domains except Vocational Dysfunction wherein it is comparable to controls (35.47 ± 6.68 and 34.20 ± 3.80 respectively). Overall, the mean values obtained for total scores were higher in cases (215.8 ± 18.31) than the controls (171.33 ± 17.39) and the difference was statistically highly significant ($p < 0.001$).

Correlation of demographic details, illness variables and dysfunction with cognitive function

On PGI Memory Scale, age was found to be a

Table-2: Performance on PGI Memory Scale

	Cases(n=30)	Controls(n=30)	t-test	p- value
Rem. Mem	5.73 ± 0.69	5.97 ± 0.18	1.787	0.079
Rec. Mem	4.50 ± 0.68	4.93 ± 0.25	3.261	0.002*
Men. Bal	6.57 ± 2.14	7.90 ± 1.03	3.070	0.003*
Att. & Conc.	9.27 ± 1.64	11.3 ± 2.38	3.914	<0.001*
Del. Rec.	8.27 ± 1.23	9.43 ± 0.82	4.327	<0.001*
Imm. Rec.	9.27 ± 1.74	10.60 ± 1.25	3.409	0.001*
Ret. F.S.P.	4.70 ± 0.59	4.87 ± 0.43	1.238	0.221
Ret. F.D.P.	7.97 ± 3.09	10.67 ± 3.19	3.331	0.002*
Vis. Ret	10.67 ± 2.32	12.43 ± 0.94	3.862	<0.001*
Recog.	9.23 ± 1.10	9.80 ± 0.41	2.637	0.011*
Total Score	75.67 ± 9.62	87.70 ± 6.36	5.717	<0.001*

*- Significant; n – Number

significant correlator of duration of illness but negatively with delayed recall and retention for dissimilar pairs – components of cognition. Education level corelated with all domains of cognition except recent memory and recognition. Duration of illness corelated negatively with delayed recall. Time since remission did not corelate with any cognitive domain. (Supplementary table-2) On Cognitive Functioning Scale, the age corelated with Duration of illness and Spatial and learning memory. Educational level corelated negatively with all domains except GACF. (Supplementary table-3) On DAQ, age corelated with duration of illness and Family dysfunction whereas education level corelated negatively with family dysfunction and positively with total dysfunction. Duration of illness and time since remission did not show any corelation with DAQ. (Supplementary table-4).

The corelation of Positive symptom, Negative symptom, General psychopathology and Total score on PANSS is significant across almost all domains of PGI Memory Scale. (Table-5) Further, on PGI Memory scale, the social dysfunction on DAQ did not corelate with any domain of cognition. Vocational dysfunction corelated negatively with retention for similar pairs. Personal dysfunction corelated negatively with mental balance, delayed recall, retention for similar and dissimilar pairs and visual recognition. The Family dysfunction corelated negatively with Remote memory and Visual recognition. The

Table-3: Performance on Cognitive Functioning Scale

	Cases(n=30)	Controls(n=30)	t-test	p- value
Working Memory	6.20 ± 2.51	3.47 ± 1.28	5.313	<0.001*
Attention/Vigilance.	7.47 ± 2.45	5.20 ± 1.27	4.505	<0.001*
Vis. Learn & Memory	5.83 ± 1.80	4.50 ± 1.51	3.112	0.003*
Spat. Lear & Memory	5.67 ± 1.95	4.00 ± 1.20	3.979	<0.001*
Reas & Prob.Sol.	8.87 ± 2.64	5.53 ± 1.38	6.134	<0.001*
Speed of Proc.	3.27 ± 1.02	2.10 ± 0.31	6.030	<0.001*
Social Cogn.	5.30 ± 1.64	3.77 ± 1.16	4.169	<0.001*
Total Score	42.57 ± 11.64	28.43 ± 3.98	6.290	<0.001*
GSCI	4.27 ± 1.20	1.40 ± 0.56	11.832	<0.001*
GICCF	2.73 ± 0.52	3.30 ± 0.47	4.441	<0.001*
GACF	52.27 ± 13.79	80.13 ± 4.59	10.500	<0.001*

n– Number * Significant

Table-4 Dysfunction Analysis Questionnaire Scores

	Cases (n=30)	Controls (n=30)	t-test	p-value
Social Dysfunction	46.60 ± 8.69	33.13 ± 3.31	7.927	<0.001*
Vocational Dysfunction	35.47 ± 6.68	34.20 ± 3.80	.902	0.371
PersonalDysfunction	45.07 ± 4.66	34.60 ± 3.24	10.096	<0.001*
Family Dysfunction	41.67 ± 5.17	33.73 ± 5.35	5.838	<0.001*
Cognitive Dysfunction	47.00 ± 5.98	34.07 ± 3.54	10.189	<0.001*
Total Score	215.8 ± 18.31	171.33 ± 17.39	9.463	<0.001*

n – Number; * Significant

Table-5: Correlation of domains on PGI Memory scale and PANSS

	Rem Mem	Rec Mem	MB	AC	DR	IR	RSP	RDP	VR	REC	Total
Positive	-0.239	-0.181	0.344*	0.334*	-0.430*	-0.353*	-0.332*	-0.317*	-0.475*	-0.350*	-0.4978
Negative	-0.378*	-0.439*	-0.535*	-0.411*	-0.486*	-0.365*	-0.252	-0.413*	-0.686*	-0.383*	-0.697*
Gen	-0.350*	-0.3378	-0.469*	-0.565*	-0.458*	-0.382*	-0.213	-0.464*	-0.634*	-0.244	-0.712*
Total	-0.3888	-0.391*	-0.544*	-0.545*	-0.552*	-0.449*	-0.303*	-0.496*	-0.739*	-0.381*	-0.787*

* Statistically significant. TSR- time since remission Rem Mem- Remote Memory, Rec mem- Recent Memory, MB- Mental Balance, DR- Delayed recall, IR-Immediate recall, RSP- Retention for similar pairs, RDP- Retention for dissimilar pairs, VR- Visual retention, REC-Recognition

Table-6: Correlation among domains of PGI battery and DAQ

	Rem Mem	Rec Mem	MB	AC	DR	IR	RSP	RDP	VR	REC	Total
SA	-0.076	-0.285	0.040	-0.191	-0.177	-0.257	0.089	-0.307	-0.222	0.028	-0.289
VA	0.102	0.045	-0.098	0.026	-0.083	0.025	-0.388*	0.089	-0.163	-0.235	-0.046
PA	-0.144	-0.174	-0.408*	-0.318	-0.364*	-0.266	-0.402*	-0.577*	-0.437*	-0.157	-0.595*
FA	-0.373*	-0.127	-0.287	-0.306	-0.202	-0.028	-0.503*	-0.216	-0.703*	-0.324	-0.464*
CA	-0.133	-0.329*	-0.352*	-0.612*	-0.469*	-0.523*	-0.184	-0.457*	-0.288	0.193	-0.580*
Total	-0.184	-0.306	-0.317	-0.449*	-0.417*	-0.360	-0.404*	-0.471*	-0.569*	-0.141	-0.626*

* Statistically significant. SA- Social, VA- Vocational, PA- Personal, FA- Family, CA- Cognitive; Rem Mem- Remote memory, Rec mem- Recent Memory, MB- Mental Balance, DR- Delayed recall, IR-Immediate recall, RSP- Retention for similar pairs, RDP- Retention for dissimilar pairs, Visual retention, REC-Recognition

Supplementary Table-1: Pattern of use of antipsychotic agent

Name of Antipsychotic	Males (n=15)		Females (n=15)	
	N	%	N	%
Typical Antipsychotic				
Haloperidol	1	6.67	1	6.67
Haloperidol + Chlorpromazine	1	6.67	0	0
Triflouperazine	0	0	1	6.67
Atypical Antipsychotic				
Olanzapine	4	26.66	0	0
Risperidone	3	20	2	13.33
Quetiapine	1	6.66	2	13.33
Aripiprazole	0	0	1	6.67
Amisulpiride	0	0	0	0
Risperidone + Olanzapine	3	20	3	20
Quetiapine + Amisulpiride	1	6.67	2	13.33
Paliperidone + Levosulpiride	1	6.67	0	0
Risperidone + Quetiapine	0	0	1	6.67
Amisulpiride + Olanzapine	0	0	2	13.33
Total	**13**	**86.66**	**13**	**86.66**

N – Number; % - Percentage.

Supplementary Table-2: Correlation among variables - PGI battery components and clinical variables (Age, Educational status, Duration of illness and Time since remission)

	Age	Edu Level	Duration of illness	TSR	REM Mem	Rec REM	MB	AC	DR	IR	RSP	RDP	VR	REC	Total
Age	1	0.047	0.680*	-0.147	0.039	-0.183	-0.038	-0.176	-0.268*	0.123	-0.154	-0.296*	-0.234	-0.124	-0.186
Edu level		1	-0.012	-0.015	0.309*	0.214	0.434*	0.553*	0.307*	0.330*	0.341*	0.327*	0.579*	0.225	0.555*
Duration of illness			1	0.092	0.040	0.329	-0.253	-0.106	-0.440*	-0.188	-0.059	-0.171	-0.232	-0.304	-0.284
TSR				1	0.129	-0.079	-0.144	0.056	-0.013	0.030	0.169	-0.272	-0.221	0.001	-0.269
REM Mem					1	0.323*	0.331*	0.097	0.294*	0.028	0.192	-0.040	0.133	0.288*	0.232
REC Mem						1	0.254	0.363*	0.371*	0.369*	0.252	0.237	0.191	-0.043	0.428*
MB							1	0.450*	0.555*	0.320*	0.307*	0.404*	0.533*	0.354*	0.716*
AC								1	0.573*	0.448*	0.353*	0.611*	0.510*	0.117	0.787*
DR									1	0.393*	0.409*	0.402*	0.448*	0.223	0.709*
IR										1	0.180	0.340*	0.362*	0.024	0.571*
RSP											1	0.277*	0.331*	0.175	0.448*
RDP												1	0.470*	0.144	0.780*
VR													1	0.266*	0.756*
REC														1	0.342*
Total															1

* Statistically significant, TSR - Time Since Remission, Rem Mem - Remote memory, Rec mem - Recent Memory, MB - Mental Balance, DR - Delayed recall, IR - Immediate recall, RSP - Retention for similar pairs, RDP - Retention for dissimilar pairs, Visual retention, REC - Recognition.

Supplementary Table-3: Correlation among variables – Cognitive Functioning Scale components and clinical variables (Age, Educational status, Duration of illness and Time since remission)

	Age	Edu Level	Duration of illness	TSR	WM	AV	VLM	SPM	RPS	SP	SC	GSCI	GIC	GACF	Total
Age	1	0.047	0.680*	-0.147	0.183	0.192	0.202	0.258*	0.102	0.149	0.138	0.214	0.134	-0.071	-0.175
Education level		1	-0.012	-0.015	-0.470*	-0.338*	-0.262*	-0.288*	-0.493*	-0.378*	-0.297*	-0.455*	-0.360*	0.166	0.432*
Duration of illness			1	0.092	0.243	0.305	0.234	0.315	0.235	0.261	0.131	0.300	0.378*	0.125	-0.394
TSR				1	-0.036	0.096	-0.146	-0.146	0.015	-0.015	0.198	-0.004	-0.003	0.314	-0.069
WM					1	0.719*	0.652*	0.610*	0.667*	0.801*	0.661*	0.883*	0.775*	-0.370*	-0.819*
AV						1	0.615*	0.700*	0.665*	0.688*	0.677*	0.878*	0.761*	-0.364*	-0.783*
VLM							1	0.587*	0.445*	0.606*	0.425*	0.734*	0.575*	-0.440*	-0.630*
SPM								1	0.634*	0.639*	0.443*	0.798*	0.681*	-0.327*	-0.702*
RPS									1	0.666*	0.698*	0.851*	0.791*	-0.404*	-0.817*
SP										1	0.624*	0.840*	0.789*	-0.368*	-0.815*
SC											1	0.778*	0.712*	-0.228*	-0.727*
GSCI												1	0.878*	-0.437*	-0.919*
GIC													1	-0.432*	-0.962*
GACF														1	0.435*
Total															1

* Statistically significant. TSR - Time since remission, WM - Working memory, AV - Attention/Vigilance, VLM - Verbal and learning memory, SPM - Spatial learning and memory, RPS - Reasoning and problem solving, SP - Speed of processing, SC - Social cognition, GSCI - Global severity of cognitive impairment, GIC - Global Impression of Change, GACF - Global assessment of cognitive functioning.

Supplementary Table-4: Correlation among variables - DAQ components and clinical variables (Age, Educational status, Duration of illness and Time since remission)

	Age	Education level	Duration of illness	TSR	SA	VA	PA	FA	CA	Total
Age	1	0.047	0.680*	-0.147	0.086	0.134	0.201	0.392*	-0.098	0.220
Education level		1	-0.012	-0.015	-0.116	-0.264	-0.195	-0.452*	-0.220	0.432*
Duration of illness			1	0.092	0.103	0.099	0.088	0.042	0.047	0.134
TSR				1	0.030	0.020	-0.160	-0.079	-0.056	-0.060
SA					1	-0.213	0.484*	0.167	0.574*	0.755*
VA						1	-0.300	0.437*	-0.304	0.212
PA							1	0.261	0.628*	0.654*
FA								1	0.105*	0.622*
CA									1	0.678*
Total										1

* Statistically significant. TSR- Time since remission, SA- Social, VA- Vocational, PA- Personal, FA- Family, CA- Cognitive

Supplementary Table-5: Correlation among variables (CFS and DAQ)

	WM	AV	VLM	SPM	RPS	SP	SC	GSCI	GIC	GACF	Total
SA	0.424*	0.434*	0.407*	0.284	0.301	0.567*	0.315	0.460*	0.380	-0.161	-0.368*
VA	0.163	-0.031	0.021	-0.183	-0.196	-0.019	-0.092	-0.064	-0.179	-0.003	0.165
PA	0.423*	0.511*	0.506*	0.465*	0.568*	0.492*	0.488*	0.610*	0.588*	-0.163	-0.594*
FA	0.579*	0.449*	0.452*	0.398*	0.305	0.267	0.264	0.487*	0.348	-0.188	-0.314
CA	0.340	0.575*	0.502*	0.549*	0.442*	0.500*	0.565*	0.589*	0.556*	-0.133	-0.517*
Total	0.643*	0.640*	0.622*	0.478*	0.447*	0.626*	0.499*	0.681*	0.545*	-0.216	-0.523*

*Statistically significant, SA - Social, VA - Vocational, PA - Personal, FA - Family, CA - Cognitive; WM - Working memory, AV - Attention/Vigilance, VLM - Verbal and learning memory, SPM - Spatial learning and memory, RPS - Reasoning and problem solving, SP - Speed of processing, SC - Social cognition, GSCI - Global severity of cognitive impairment, GIC - Global Impression of Change, GACF - Global assessment of cognitive functioning.

cognitive dysfunction corelated negatively with recent memory, mental balance, attention and concentration, delayed recall, immediate recall and retention for dissimilar pairs. (Table 6)

Discussion

The present study assessed the cognitive functions in patients of schizophrenia currently in remission and compared it with healthy controls.

This study also attempted to study the relationship between cognitive functions and disability in various domains in these patients. They were all matched for age and gender.

Sociodemographic factors

In the present study the mean age of the persons with Schizophrenia was 29.47 ± 11.78 with a range of 18 to 60 years with equal preponderance in both males and females (15 each) similar to Hafner et al[14] and Giordano et al[15] who found the lifetime risk for schizophrenia was equal for males and females.

In the present study 63.33% persons with Schizophrenia belonged to unmarried category. This can be either the cause or the effect of the illness as supported by the study by Ponnudurai et al[16] and Thara[17] who found that the percentage of schizophrenia patients who get married was much lower than normal individuals or those with other psychiatric disorder. The lower percentage of schizophrenia patients who get married can be attributed to poor pre-morbid adjustment impairing the development of relationships and social and occupational disability arising due to the illness. In the present study, there were 2 cases who were divorced as compared to none in control group which is supported by Thara[17] who reported higher rate of divorce among patients than in the general population.

In the present study, the distribution of the two groups with respect to education status and gender shows maximum subjects belonging to the > 12 years of education in control group i.e. 22 (73.33%) while in the schizophrenia group, 14 (46.67%) belonged to the category of < 12 years of education also supported by the meta-analysis by Dickson et al.[18] This can be attributed to the fact that the existence of this disease – its onset, ongoing course or remission affects the ongoing education and impairs the working capacity of the individual.

In the present study, the patients and the controls were sub grouped as per occupation (Table 5). A total of 33.33 percent of the cases versus 13.33 percent controls were unemployed, a difference that was statistically significant (Chi-square = 15.548, p = 0.008*). Overall, we found almost two-thirds of the Schizophrenia group to be employed. This is in conformity with the landmark Madras longitudinal study by Thara[19] which found that more than 75 percent of patients with Schizophrenia remained employed at the end of 20 years. In another study from India by Khare et al[20] more than 60 percent were employed and maintained same job for 10 years. While the western literature shows poorer employment after Schizophrenia diagnosis.[21]

In the present study, it is evident that both among males and females, maximum patients were on atypical antipsychotics during the period of remission because of the lower incidence of side-effects of atypical antipsychotics. This is supported by Sabe et al[22] in their meta-analysis.

Cognitive domain

In our study, we found that schizophrenia group took significantly longer time than controls to complete the tests of PGI Memory Scale and Cognitive Functioning Scale. We also found that the patients with schizophrenia scored less well than controls on all tests of cognition, except Remote Memory and Retention for Similar Pairs and fared worse on visual retention subtest of PGI Memory Scale and the Cognitive Functioning Scale ($p < 0.001$*) where the difference was statistically non-significant. This is supported by many studies by Green et al[23] and Krishandas et al[24] who also reported that schizophrenia is accompanied by impairments in several domains of cognitive functioning.

The most distinct cognitive impairments in schizophrenia are found within the domains of attention, processing speed, executive functioning, and different memory functions (episodic memory, working memory, verbal memory, visual memory. Torgalsbøen et al[25] showed that attention was the only cognitive domain that predicted remission status after six months. Cognitive domains related to long-term remission status were executive functions, working memory, and premorbid functioning.[26]

We found in our study that there was statistically significant corelation of age with cognitive functioning and duration of illness (on both PGI Memory scale and Cognitive Functioning Scale). This is in contrast to Krishandas et al[24] and Heaton et al[27] who did not find any relationship between age, duration of illness and cognitive functioning. This contrasts with Srinivasan et al[28] who reported a relationship between age, duration of illness and neurocognitive function with the older schizophrenia patients (>65) showing a progressive decline in cognitive functioning as compared to younger

schizophrenia patients. Similarly Fucetola[29] reported an age-related decline across most neuropsychological functions in patients with schizophrenia.

Educational level is corelated with most of the domains of cognitive functioning on both the scales especially-Remote memory, mental balance, attention and concentration, delayed recall, immediate recall, retention for similar and dissimilar pairs and visual retention on PGI Memory Scale and all domains on Cognitive Functioning Scale. Duration of illness did not in itself correlate with most domains except delayed recall.

The aetiology of cognitive deficits in schizophrenia is quite diverse – ranging from genetic and neurotransmitter on one hand to the environmental factors on the other. Both twin and GWAS indicate common genetic pathways in schizophrenia and cognition.[30] Dopamine signalling in the cortex leads to normal attention, working memory, and executive function. Hence, impairment in dopamine signalling results in working memory and executive functioning deficits similar to the current study. The disruption of cortico-cerebellar-thalamocortical circuitry with variable involvement of neurotransmitters like dopamine, acetylcholine, glutamatergic and GABAergic systems accounts for the pathophysiology.[31] Volumetric MRI studies have elicited hippocampus and amygdala volume loss while FDG PET studies have elicited frontal hypometabolism.[32]

The complexity in the aetiopathogenesis of cognitive deficits in Schizophrenia is further reflected in the failure of treatment modalities as Pro-dopaminergic drugs and varenicline or acetylcholinesterase inhibitors despite strong association between cholinergic signalling and cognition in schizophrenia.[33]

Social Dysfunction

The dysfunction assessment on various domains-Social, Vocational Personal, Familial and Cognitive showed that the dysfunction was statistically significant in the patients with schizophrenia in remission in all domains except the vocational domain where despite being low, it was comparable to the control group.

Despite being in remission, there was no corelation of dysfunction scores with type of family, age group, type of occupation or duration of illness. The dysfunction scores were corelated with the married participants and was found to be lower in patients of schizophrenia who were married. This is similar to Gupta et al[34] The Indian population has family and marriage as strong proponents of social support unlike the western world thus leading to better outcomes even in neurodegenerative disorders as Schizophrenia.

In our study, the corelation analysis between domains of PGI memory scale and DAQ and between Cognitive Functioning scale and DAQ showed statistically significant corelation on Personal, familial and cognitive domains of DAQ and cognition. Measures of functioning are more strongly correlated with measures of cognition in individuals with schizophrenia than in healthy controls.[33,35] Similarly, Barch[36] documented that disturbances in social and occupational functioning in individuals with schizophrenia may be more influenced by the severity of cognitive deficits. Villalta et al[37] found that negative symptoms were the major source of disability and were associated with cognitive dysfunctioning. Addington et al[38] in their study concluded that cognition is significantly associated with social functioning and thus, social outcomes remain poor for many individuals with schizophrenia. This wide variation in the findings can be explained on the basis of the fact that most of the studies disfavouring our results have been carried out in the developed countries. But in the developing countries like ours, the absence of access to social security and public funds in the form of disability allowance might have contributed to the pressure to earn for a living and hence the high employment rate. Employment might offer the patient a form of practical, social and functional rehabilitation. The possibility of finding a job in the unorganized sector as street vendors and manual labourers make it easy for people to earn a living and remain functional from an employment point of view.

Conclusion

This study suggests that persistent cognitive deficits are seen in patients with Schizophrenia under remission at one year when compared to normal controls further embalming that the cognitive deficits in the form of attention and executive function deficits are trait marker of Schizophrenia and persist despite symptomatic remission. The aetiopathogenesis of cognitive deficits is complex and thus the

concept of functioning in Schizophrenia is a conundrum requiring lots of research to reach a workable solution.

Limitations of the study

The study entails a number of limitations in the form of small sample size leading to non-generalisability of the results. We did not test the premorbid intelligence (and hence the cognitive reserve) of the patients, although any history suggestive of a developmental delay was taken as the exclusion criteria. All patients in our study were on medications, including typical and atypical antipsychotics known to cause cognitive deficits. Although previous studies on first episode drug naïve patients have confirmed that the deficits are independent of medication, the possibility of the medications worsening the cognitive functioning cannot be ruled out.

Acknowledgements

Dr. Rohit Sharma and Dr. Sahil Mehta for compilation of the manuscript, statistical analysis, and critical review for formulation of the manuscript.

Author Contributions

Dr. SM-Conceptualization, data collection and analyses, writing the manuscript, review, and editing. Dr. NSM: Conceptualization, review and editing of manuscript. Dr. RM: Conceptualization, editing and review of the manuscript. All the authors confirm that all of them have contributed to the conception of design; analysis, interpretation of data; drafting of the article; critically revisiting the article for important intellectual inputs; and approval of the final version. Manuscript has been read and approved by all the authors.

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Original Article

Prevalence of Depression and Anxiety in Post Covid-19 Patients

Supeen Yadav, Tushar Jagawat, Ritu Meena, Pankaj Tandon, Preetkamal, Savita Jagawat, Neelu Yadav
Department of Psychiatry & Clinical Psychology NIMS & R, Jaipur, Rajasthan
Contact: Tushar Jagawat, Email: tusharjagawat@yahoo.com

ABSTRACT

***Background**: According to literature depression and anxiety are the most common psychiatric disorder in patients post covid-19 survivors **AIM:** To estimate the prevalence of Depression and Anxiety in Post Covid-19 patients. **Material & Methods:** Thirty-two patients who were diagnosed with COVID-19 and presented in NIMS & R OPD/IPD were evaluated. A psychiatric evaluation was done on a structured proforma containing sociodemographic details. The diagnosis was made according to ICD-10 criteria. A general health questionnaire (GHQ), Hamilton anxiety rating scale, and Hamilton rating scale for depression were applied. **Result:** The study revealed that psychiatric comorbidity was seen in a total of 19 patients (59.3) Out of which 13 (40.6%) fulfilled the criteria of depression and 6 (18.7%) fulfilled the criteria of anxiety. **Conclusion**: The prevalence of depression and anxiety is high in patients, post COVID-19. The severity of these disorders is significantly associated with the severity of COVID-19. Hence, thorough evaluation and management of these disorders in post-COVID-19 patients can help in improving the overall outcome.*

Keywords: Covid-19, Depression, Anxiety.

Introduction

The Coronavirus Disease 2019 (COVID-19) was first reported in Wuhan, China in 2019 and then it spread like wildfire engulfing the whole world. COVID-19 is generally characterized by symptoms such as fever, chills, cough, nausea, coryza, sore throat, myalgia, and breathing difficulty of varying proportions[1]. The COVID-19 pandemic worldwide represents a dangerous and potentially traumatic event and it can be regarded as a mental health catastrophe. Evidence from previous outbreaks such as the severe acute respiratory syndrome (SARS) has demonstrated the impact on mental health conditions. Lee et al[2] found that SARS survivors had higher stress during the outbreak and the stress level persisted a year after infection. They were also found to have had higher anxiety, depression, and posttraumatic symptoms. Multiple causative factors have been attributed to explaining psychiatric manifestations of coronavirus infection; like a direct neuronal invasion by coronavirus, dysregulated immunological response, abnormal activation of the hypothalamic-pituitary-adrenal axis, cerebrovascular hypoxia, and metabolic derangements. Coronavirus infection can affect the brain resulting in some neurological and psychiatric symptoms including headache, dizziness, depression, anxiety, psychotic symptoms, PTSD, anosmia, altered sensorium, impaired consciousness, confusion and/or delirium. Additionally, patients suffering from COVID-19 can develop psychiatric morbidity because of the stigma associated with the disease, social isolation, concern and uncertainty about the outcome of this infection, and limited information about the pandemic. Psychological distress and depression may have an adverse effect on a patient's immune system an adverse effect on patient's immune system response. The new realities of working from home, temporary unemployment, and children's home-schooling can positively impact the public.

Long COVID or post-COVID-19 condition has been referred to as the persistence of physical and mental symptoms beyond three months of COVID-19 infection. Some studies have explored the development of mental health symptoms in different time intervals after diagnosis of COVID-19 and the extent to how demographic profiles of COVID-19 survivors predict long-term mental health. A review by Renaud-Charest et al[3] revealed that depression was commonly seen in individuals as part of long COVID. Bourmistrova et al[4] in a review on the long-term effects of COVID-19 on mental health noted that the prevalence of anxiety, depression, and sleep disturbance was comparable to the general population suggesting that the symptoms at the longer term could be attributed to the indirect effect of COVID-19 psychosocial factors.

Material & Methods

After approval from the Ethics Committee, this observational cross-sectional study was carried out in the department of psychiatry of a tertiary care hospital attached to a medical college. We included thirty-two patients whose age were more than 18 years and COVID–19 survivors (confirmed COVID-19 infection either admitted in hospital or kept in isolation and taking treatment at home).

The patients suffering from pastor present significant medical/surgical/neurological illness, Patients with chronic medical conditions before COVID-19 infection such as chronic obstructive pulmonary disease, bronchial asthma, and pulmonary tuberculosis, Survivors who had a history of previous psychiatric illnesses, pre-existing psychological symptoms, and those who refused to participate in the study were excluded from the study.

All of the participants were assessed on OPD/ IPD basis. The socio-demographic data was obtained on a specially designed semi-structured proforma consisting of socio-demographic, and clinical profiles. Following that General Health Questionnaire (GHQ) was administered. The diagnosis was made according to the ICD 10 criteria. The severity of psychiatric morbidity was assessed based on the Hamilton Anxiety Rating Scale, and Hamilton Rating Scale for Depression.

Assessment tools

General Health Questionnaire (GHQ)[5]. — The General Health Questionnaire (GHQ) is a self-administered screening questionnaire, designed for use in consulting settings aimed at detecting individuals with a diagnosable psychiatric disorder. The 12-Item General Health Questionnaire (GHQ-12) is the most extensively used screening instrument for common mental disorders, in addition to being a more general measure of psychiatric well-being. 12 items, each one assessing the severity of a mental problem over the past few weeks using a 4-point Likert-type scale (from 0 to 3). High scores indicate worse health. In a review of 17 published research studies on the GHQ-12, Goldberg, and colleagues found that the most common cutoff score was 2/3 (a score of 2 or less indicating the absence of a mental disorder and a score of 3 or greater indicating the presence of disorder).

Hamilton depression rating scale (HAM-D)[6]. — This scale is used to measure a patient's level of depression and its severity. A score of 0–7 is generally accepted to be within the normal range (or in clinical remission), while a score of 20 or higher (indicating at least moderate severity). The following cut-offs were used for HAM-D score interpretation: no depression (0-7); mild depression; (8-16); moderate depression (17-23) and severe depression (>24).

Hamilton Anxiety Rating Scale (HAM-A)[7]. — The scale is utilized to determine a patient's anxiety level and symptom profile, as well as to measure its severity. It is a 5-point Likert-type scale comprising a total of 14 questions about both somatic and mental symptoms. Each item is scored on a scale of 0 to 4, with a total score of 0-56. The following cut-offs were used for interpreting HAM-A scores: 0-7 = no/minimal anxiety; 8-14 = mild anxiety; 15-23 = moderate anxiety and 24 or greater = severe anxiety.

Results

The socio-demographic characteristics of the 32 patients are presented in Table 1.

Eighteen (56.2%) were in age group 40-59, sixteen (50%) were illiterate, majority 65.62% (n=21) were Hindu, seventeen (53.1%) were employed, majority 50% (n=16) have middle socioeconomic status, twenty (62.5%) were from nuclear family.

Pie chart 2 shows that out of 32 participants, 13 (40.6%) had depression, 6 (18.7%) had anxiety

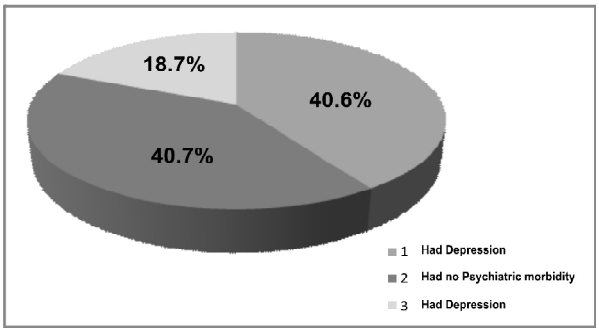
and 13 (40.6%) had no psychiatric co-morbidity.

As shown in Table 3 out of 13 diagnosed depression patients six (46.1%) had moderate depression, four (30%) had mild depression and three (23.07%) had severe depression.

As visible in Table 4 out of 6 diagnosed anxiety patients three (50%) had moderate anxiety, two (33.33%) had mild anxiety and one (16.66%) had severe anxiety.

Table-1: Socio-demographic variables

Variables		No. of Patients (n=32) (%)
Age	21-39	4 (12.5%)
	40-59	18 (56.2%)
	60-89	10 (31.2)
Education	Illiterate	16 (50%)
	Primary	6 (18.75%)
	Secondary	2 (9.3%)
	Graduate & above	8 (25%)
Religion	Hindu	21 (65.62%)
	Muslim	11 (34.37%)
	Christian	0 –
	Others	0 –
Occupation	Homemaker	09 (28.12%)
	Student	0 –
	Employed	17 (53.1%)
	Unemployed	6 (18.75%)
Socioeconomic status	Upper	4 (12.5%)
	Middle	16 (50%)
	Lower	12 (37.5%)
	Urban	09 (28.1%)
	Rural	23 (71.87)
Family	Nuclear	20 (62.5%)
	Joint	12 (37.5%)



PIE CHART : Showing psychiatric morbidity

Table-3: Severity of depression according to Hamilton Depression Rating Scale (HDRS)

Depression	
Mild	4 (30%)
Moderate	6 (46.1%)
Severe	3 (23.07%)

Table-4: Severity of anxiety according to Hamilton Anxiety Rating Scale (HAM-A)

Anxiety	
Mild	2 (33.33%)
Moderate	3 (50%)
Severe	1 (16.66%)

Discussion

In this cross-sectional study of patients diagnosed with COVID-19 infection, out of 32 patients, 19 patients have psychiatric comorbidity post-COVID-19. The majority were found to have depression and anxiety.

Our findings confirmed the high prevalence of mental health ailments like depression, and anxiety, following SARS CoV-2 infection in the long term. 13 (40.6%) of the patients had a depressive disorder. This is consistent with other studies like Pathak et al[8] who found depression in 37.50%, in our study majority 6 (46.1%) had moderate depression followed by 4 (30%) had mild depression, and 3 (23.07%) had severe depression. Which is in contrast to the findings by Pathak et al[8] in which majority had mild depression (47%).

In this study 6 (18.7%) had anxiety, and majority had mild to moderate anxiety, which is in resonance with the study. The prevalence of psychiatric morbidity has ranged from 10% to 48% in various studies. According to Pathak et al[8] reported that 37.50% had depression. Gaur et al[9] also reported that depression was the most common diagnosis observed in 44.44% of patients followed by anxiety (34.72%). The present study also depicted similar findings in terms of the prevalence of depression and anxiety. The COVID-19 infection has been known to have far-reaching ramifications on the brain and cause plethora of psychological symptoms in an individual. Viral infections trigger dysregulated inflammatory response and as a result, a number of proinflammatory mediators such as Tumor Necrosis Factor-alpha (TNF-alpha), interleukin-6 (IL-6), and interleukin beta cytokines are released. Noradrenaline is also known to play an important role in regulating depression like symptoms such as fatigue, aches, pains, and loss of appetite. Extant literature indicates that pro-inflammatory cytokines moderate serotonin levels, hypothalamic–pituitary–adrenal (HPA) axis self-regulation, microglial cells, and neuroplasticity, leading to negative regulation of brain function. In another study by Devi et al[10-12],

the prevalence of depression and anxiety among patients post-COVID-19 was 21.9% and 11.9%, respectively.

Long COVID symptoms may last for weeks or months following initial recovery from SARS-CoV-2 infection. The long-term health risks of COVID-19 include fatigue and adverse mental health outcomes such as anxiety and depression. Lastly, social, economic and spatial factors may have a significant effect on the development and persistence of depressive symptoms among individuals with post-COVID-19 syndrome. In addition to the fear of the virus, the pandemic has led to job and housing uncertainty, school closures, deaths of closed ones, and social isolation. long-term ramifications of infection, social stigma, and fear of reinfection might perpetuate the development of anxiety and depression in patients. Effective screening and robust consultation liaison services are the need of the hour for the holistic management of such patients. This will not only help in the recovery of the patients but will also help in improving the overall quality of life of such patients.

Conclusion

To conclude, our study has revealed a high prevalence of comorbid depression and a modest prevalence of anxiety in post-COVID-19 survivors. Patients suffering from COVID-19 should be thoroughly evaluated and adequately managed for comorbid anxiety and depressive symptoms for a better outcome. As further research advances, a holistic approach that combines medical and psychological interventions will be essential in enhancing individuals' overall quality of life in Post-COVID-19 phase.

Limitations

Our study is subjected to several limitations. First of all, the study included a small number of participants restricting statistical power and most of the participants were from rural background. A large sample size would have provided a better understanding of the relationship between variables studied in the study. Secondly, participants were chosen through a convenient, non-probabilistic sampling method, which may not adequately represent the entire population. Lastly, being a cross-sectional observational study, any causal relationship cannot be established between the variables studied.

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Original Article

Neurocognitive functions of individuals with schizophrenia and their siblings

N. Suresh Kumar,[1] S. Raja Kumari,[2] V. Geethaanjali,[3] Aarzoo[4]
[1,4]Department of Psychiatry, Madurai Medical College, Madurai
[2]Department of Psychology, Government Arts College, Coimbatore-12
[3]Department of Psychiatry, Government Medical College and Hospital, Chandigarh

ABSTRACT

***Background**: Schizophrenia is a severe, chronic and potentially disabling thought disorder associated with deterioration in social and occupational functioning. The liability to schizophrenia is highly heritable, as shown through the pattern in the first degree relative. Cognitive deficits in schizophrenia have been found to correlate with other symptom domains in schizophrenia and are linked with functional outcome. **Objectives**: The current study therefore aimed to draw profile of neuropsychological performance of individuals with schizophrenia and their unaffected siblings in comparison to HC. **Methods**: This cross comparative study using purposive sampling with sample size of 120, 40 in each of three groups namely; individuals diagnosed with schizophrenia (SCZ), sibling of patients diagnosed with schizophrenia (SIB), and healthy controls (HC). The assessment measures used included tests of attention, verbalfluency, working memory, executive functions, and verbal memory. **Results**: Individuals with schizophrenia performed poorly than unaffected siblings and HC while siblings performed superior than individuals with schizophrenia but inferior to HC. **Conclusion**: Findings of the study could be helpful in developing preventive measures, psychological management and cognitive retraining for the unaffected siblings.*

***Keywords**: Schizophrenia, unaffected siblings, cognitive functions, cognitive deficits.*

Introduction

Schizophrenia is a severe, chronic and potentially disabling thought disorder associated with deterioration in social and occupational functioning.[1,2] The patients display positive, negative, disorganized and cognitive symptoms.[3] The preva-lence varies between regions, lifetime prevalence of schizophrenia spectrum disorders in Indian population is estimatedat 1.41%; with a current prevalence of 0.42%.[2,4] One-year and lifetime prevalence of schizophrenia pooled globally was estimated to be 0.34% and 0.55%, respectively.[5] According to the Global Burden of Diseases (GBD) study of 1990–2017, the prevalence of schizophrenia in India is estimated to be 0.3%, accounting 10% of the total disability-adjusted life years (DALYs) due to mental morbidity.[6] The liability to schizophrenia is highly heritable, as shown through the pattern in the first degree relative (FDR).[7] However, the robust susceptibility genes for schizophrenia have remained elusive but neurobiological traits identified with its inheritable vulnerability include endophenotypes. These are the measurable phenotypes that lie intermediate between genetic infrastructures and clinical presentation. An endophenotypes could be a result of any neurobiological measure than can be related to the underlying molecular genetics of the illness.[8] The endophenotypes vulnerability measures had higher rates of association in relative of probands than general population and co-segregation within families.[7] Schizophrenia risk in FDRs having 1 or 2 probands with schizophrenia was investigated by conducting a systematic review of cohort and case-control studies published between 1977 and 2018; the reported estimates for schizophrenia risk were odd ratio (OR) = 7.69 (95% CI 5.11–11.56) for

FDRs of one proband with schizophrenia compared to healthy control probands, increasing to OR = 11.11 (95% CI = 1.45–85.02) for FDRs with two probands with schizophrenia.[9] A meta-analysis of twin studies estimated heritability at 81% and family studies revealed that if one parent is diagnosed, the risk of each offspring is 10–15%, while both parents having schizophrenia increased the risk to 35–46%.[10,11] The siblings of individuals with schizophrenia in comparison to the general population reported an 8 to10 fold higher risk of developing the disorder.[7]

On the other hand, the studies of neuropsychological performance of unaffected siblings individuals with schizophrenia have produced contradictory results.[12-15] A meta-analysis of cognitive performance in first degree adult relatives of individuals with schizophrenia established that cognitive findings contributed to the in consistency among studies.[15]

Cognitive deficits in schizophrenia have been found to correlate with other symptom domains in schizophrenia and are linked with functional outcome.[16] The unaffected siblings of patients with schizophrenia share partially the genes of their affected siblings. However, these siblings are unaffected by the illness yet they exhibit smaller degree of cognitive impairments similar to patients.[15] In unaffected siblings of patients with schizophrenia deficits have been observed on tests of executive functioning,working memory, episodic memory and psychomotor speed.[17-19] This cognitive impairment in unaffected siblings have recently been used as probable intermediate phenotypes in genetic studies of schizophrenia.[7,17,20]

Cognition is the sum total of mental processes that makes individual acquires knowledge and keeps the individual aware of one's surroundings and thus enables the individual to arrive at appropriate judgments about understanding cognition provides an insight into complex pattern of human behavior.[21,22] Hence, it becomes very important for functioning appropriately in society. The process of cognition is complex and it involves various processes. Selection of in formation from the environment, modification of the information, making appropriate associations among them are also involved in cognitive process.[23] The processed information can be stored in the brain and may be retrieved and reanalyzed later.

Cognitive deficits are recognized as enduring and persistent features of schizophrenia and are related with different level of disease process.[24] The current study therefore aimed to draw profile of neuropsychological performance of individuals with schizophrenia and their unaffected siblings in comparison to HC. This will help the clinicians to develop strategies to enhance cognitive functions in affected individuals leading to superior treatment outcome.

Materials and Methods

Research design and setting

The present study investigated and compared the profile of cognitive functions in patients diagnosed with Schizophrenia (F20) as per ICD 10 Classification of Mental and Behavioral Disorder: Clinical Descriptions and Diagnostic Guidelines (ICD10 CDDG),[25] their unaffected siblings, and unrelated healthy controls. The study was cross-sectional and carried out at out-patient department of Psychiatry of a tertiary care medical college located in urban southern India. Sample was recruited using non-probability (purposive) sampling,[26] and sample comprised of three groups; namely patients diagnosed with schizophrenia (SCZ), sibling of patient diagnosed with schizophrenia (SIB), and healthy controls (HC). Each group had 40 participants and HC were matched with SIB group on age, sex, and education.

Participants

Those diagnosed with schizophrenia (F20) according to the ICD10 Diagnostic criteria, aged between 20 to 40 years, having minimum 8 years of formal education, those displaying right-handedness were included. Those with any co-morbid psychiatric illness currently or in the past, any known disability currently or in the past, history of head injury with any documented cognitive sequel or with loss of consciousness or any neurological diseases, having undergone brain surgery currently or in the past, any unstable medical illness impairing cognitive function including cardiovascular or liver diseases, or unstable surgical condition, any clinical evidence of mental retardation were excluded.

The criteria for SIB sample was characterized 20 to 40 years of age, having minimum 8 years of formal education, having no psychiatric morbidity,

no clinical evidence of intellectual disability, no unstable medical, surgical, neurological condition, and no known disability.

The HC sample was characterized by 20-40 years of age, with minimum 8 years of formal education years, no psychiatric morbidity, no clinical evidence of intellectual disability, no unstable medical, surgical, neurological condition, no known disability and having score of <2 on General Health Questionnaire (GHQ-12).[27]

Assessment measures

Socio demographic and clinical profile sheet: Sociodemographic details including age, sex, education, occupation, income and marital status etc. was recorded. Also, details of clinical conditions such as onset, duration and type of illness, family history of illness and information on comorbidities was sought.

General Health Questionnaire (GHQ-12):This study used the GHQ-12 proposed by Goldberg has good reliability (.88) and validity (.79).[27] This questionnaire includes 12 items and has a four-point Likert scale, with each item ranging from 0 to 3. A total score < 3 indicates good health.[28]

Digit span test (DST): This is a test of attention/ short-term memory involving strings/series of digits (numbers) of varying length.[2] It consists of 6 items each for forward and reverse assessments, respectively. Further, each item has two trials.[29]

Trail making test (TMT-A) is also a sub test from NIMHANS Neuropsychology Battery 2004.[29] The TMT consists of two parts, A and B. Part A consists of one sample test and one task. The numbers are randomly printed on the sample worksheet. The subject is required to join consecutive numbers in order by drawing connecting lines. The worksheet consists of numbers 1 to 25. The time taken to join consecutive numbers is taken as the subject's score.

Animal naming test (ANT): This test is a measure of category fluency which is another form of verbal fluency.[29] In this, it is the content of the words rather than the phonetic similarity of the words that is regulated. The subject generates words which belong to a particular category. Subject is asked to generate the names of as many animals as possible in one minute. The subject is asked to exclude the names of fishes, birds and snakes. The total number of new words generated form the scores. The current study used maximum score for statistical analysis.

Controlled Oral Word Association Test (COWA FAS) is also a sub test from NIMHANS Neuropsychology Battery 2004.[29] COWA FAS was developed by Benton and Hamsher in 1989 as a measure of phonemic fluency. Each participant is instructed to speak as many words as he can starting with the consonant presented to him in span of one minute one consonant at a time. The mean of acceptable new words from three trials form the score. The current study used Hindi consonants (Ka, Pa, and Ma), and it took around 5 minutes to complete.

Letter number sequencing (LNS): Letter-number sequencing is a subtest of the WAIS (Wechsler Adult Intelligence Scale).[30] It is a test that measures an individual's short-term memory skills in being able to process and re-sequence information. It's an assessment of verbal working memory requires a person to mentally arrange a random sequence of letters and numbers after hearing it. He should state the numbers in ascending order and the letters in alphabetical order.

Spatial span test (SST): Its subtest from the Wechsler Memory Scale – Third Edition (WMS-III)[31] to assess visual working memory and has internal reliability co-efficient of 0.79. It consists of a board with ten spatially distributed cubes mounted on top of it. In the Spatial Span Forward test, the examiner taps a series of blocks on a three-dimensional board in a specified sequence. The participant must tab to the same blocks in the same order. In the Spatial Span Backward test the participant has to tab the blocks in the reverse order of which the examiner presented them.

Stroop Test (ST): It's a subtest of NIMHANS Neuropsychological battery and measures response inhibition, a test of executive function. In this test, the colour names 'blue', 'green,' 'red,' and 'yellow' are printed in capital letters on paper. The words are printed in 16 rows and 11 columns. It takes 15-20 minutes for administration with reliability of 0.78 and validity of 0.79.[29]

Wisconsin Card Sorting Test (WCST) is a sub test from NIMHANS Neuropsychology Battery 2004.[29] WCST was developed by Heaton in 1981. It is a measure of set shifting ability; however, it is also said to be the test of executive function. It

includes four stimulus cards and 128 response cards. There are different geometrical figures in different colors. The subject has to place the response cards one by one against the stimulus card by using a certain rule of matching. The examiner gives feedback on whether the rule used by the subject is correct or wrong, according to which he has to change his rule of matching the cards; however, it is not told to the subject. The authors used four parameters of measuring score for the current study, namely total number of errors, perseverative errors, non perseverative errors, and number of trials to complete the six categories. There are other scoring categories included in WCST. It takes around 30 minutes to complete.

Rey's Auditory Verbal Learning Test: RAVLT is a measure of verbal learning and memory (with immediate and delayed recall). This test consisted of a list of 15 words presented five times with an immediate recall after each of the five trials. A delayed recall was measured after a delay of 30 min filled with other non-verbal tests.[29]

Procedure

The research proposal was approved by IEC (6087/E4/3/2011). The clinical sample was referred to the clinical psychologist, first author, after assessment by two psychiatrist to confirm the clinical diagnosis of F20 and to rule out comorbidities. The informed consent was sought. Those consented, their sociodemographic and clinical details were recorded. Those who met the criteria were recruited to the study and assessment was carried out while those excluded were referred back to the treating clinician and note was put in file. The participants consent was also sought to contact their sibling telephonically except a few who were accompanied by their sibling were directly asked to participate in the research. Telephonic call was attempted in the presence of the index participant to seek consent and availability for participation. In addition, siblings of those who were excluded from the clinical sample were also contacted for recruitment. Once forty participants were recruited in SIB group then HC sample was approached. The participants in HC were matched on age,education and sex with SIB group. The participants in all the three groups were administered tests in the same sequence and same time of the day (between 9 am to 12 noon). They were matched on right-handedness. It took around 130-150 minutes (including break time) per participant to collect the data. Each assessment session had a break of 10 minutes after completion of Stroop test. The report of performance for each participant was put in the file within 72 hours of completion of assessment for benefits in management. The data were then analyzed for interpretation of findings.

Statistical analysis

Descriptive statistics was used to compute sociodemographic variables and raw scores of neurocognitive assessments. Further, analysis of variance was used compare the mean scores of the three groups.

Results

Table 1 presents the sociodemographic of the participants in all three groups namely; SCZ, SIB and HC.

Table-1: Sociodemographic details of the participants in three groups (N=120)

Variable		Mean (SD)		
		SCZ	SIB	HC
Age		31.28 (5.21)	2.70 (5.08)	29.08 (4.49)
Sex	Male	19 (47.5%)	22(55%)	23 (56.1%)
	Female	21 (52.5%)	18(45%)	17 (41.5%)
Education level	8^{th}-10^{th}	18(45%)	19 (47.5%)	19 (47.5%)
	11^{th}-12^{th}	12(30%)	8(20%)	8(20%)
	Above 12^{th}	10(25%)	13 (32.5	13 (32.5%)
Locality	Rural	25 (62.5%)	26(65%)	26(65%)
	Urban	15 (37.5%)	14(35%)	14(35%)
Family type	Nuclear	32(80%)	33 (82.5%)	33 (82.5%)
	Joint	8(20%)	7 (17.5%)	7 (17.5%)

SCZ – individuals with schizophrenia, SIB – unaffected sibling of individuals with schizophrenia, HC – healthy controls

Table-2: Differences in mean scores on tests of attention, verbalfluency, and workingmemory of the participants in three groups (N=120)

Domain	Measures		Mean ± SD SCZ	SIB	HC	F	Sig
Attention	DST	Forward	5.90 ± 0.90	8.45 ± 2.47	9.50 ± 1.94	38.61	.01**
		Backward	4.73 ± 1.13	7.20 ± 2.35	8.08 ± 1.87	35.22	.01**
	TMT	A	80 ± 18.18	50.70 ± 8.88	46.53 ± 7.85	101.78	.01**
		B	177.38 ± 37.93	95.50 ± 13.53	80.05 ± 12.04	185.85	.01**
Verbal Fluency		ANT	10.50 ± 2.80	12.53 ± 1.96	15.23 ± 1.72	46.06	.01**
	COWA	Ka	7.33 ± 1.72	9.35 ± 1.90	12.48 ± 1.57	89.54	.01**
		Pa	6.08 ± 2.20	9.68 ± 2.03	12.23 ± 1.64	98.31	.01**
		Ma	5.56 ± 1.90	9.40 ± 2.49	11.28 ± 1.50	81.57	.01**
Working memory		LNS	8.13 ± 1.71	12.35 ± 2.94	14.57 ± 1.84	86.13	.01**
	SST	Forward	6.35 ± 1.48	8.48 ± 1.40	9.10 ± 1.15	45.73	.01**
		Backward	5.35 ± 1.59	6.38 ± 1.39	6.98 ± 1.12	14.14	.01**
		Total	11.70 ± 2.95	14.85 ± 2.69	16.08 ± 2.09	30.09	.00**

*p< 0.05, ** p< 0.01, *** p< 0.001SCZ – individuals with schizophrenia, SIB – unaffected sibling of individuals with schizophrenia, HC – healthy controls, DST – digit span test, TMT – trail making test, COWA – controlled word association test, ANT – animal naming test, LNS – letter number sequencing, SST – spatial span test,

Table 2 displayed the mean scores and their differences on tests of attention (DST, TMT), verbalfluency (ANT, COWA), and working memory (LNS, SST) of the participants in three groups; SCZ, SIB and HC.

Table 3 displayed the mean scores and their differences on tests of executive functions (Stroop, WCST) of the participants in three groups; SCZ, SIB and HC.

Table 4 displayed the mean scores and their differences on test of verbal memory (AVLT) of the participants in three groups; SCZ, SIB and HC.

Discussion

Schizophrenia is a clinical syndrome, with intensely disruptive psychopathology that includes disturbances in cognition, emotion, perception, thinking, and behavior. Despite the significant advances in understanding of the nature of the disorder, its cause and main brain mechanisms are yet to be fully revealed; therefore, schizophrenia remains a great challenge to psychiatry.[32] The aim of the present research was to assess the cognitive functions in patients with schizophrenia, their unaffected siblings and healthy controls. The results revealed that both

Table-3: Differences in mean scores on tests of executive functions of the participants in three groups (N=120)

Measures	of executive functions	Mean ± SD SCZ	SIB	HC	F	Sig.
Stroop	Word naming	56.37 ± 1.90	75.12 ± 1.90	76 ± 1.0	34.18	.01**
	Colour naming	40.60 ± 14.31	61.80 ± 10.47	68.88 ± 8.32	67.71	.01**
	Colour word naming	24.18 ± 11.10	43.72 ± 9.18	53.63 ± 9.81	88.67	.01**
WCST	Numberoftrials	125.53 ± 6.18	117.18 ± 18.04	108.58 ± 18.92	11.94	.01**
	Number of correct responses	60.03 ± 19.39	73.60 ± 11.20	74.38 ± 8.37	13.67	.01**
	Numberoferror	65.85 ± 22.30	43.63 ± 18.71	34.50 ± 14.47	29.54	.01**
	Perseverative responses	45.10 ± 27.24	29.15 ± 15.58	23.98 ± 12.98	12.62	.01**
	Perseverative error	36.78 ± 21.13	24.90 ± 13.12	20.25 ± 9.95	12.14	.01**
	Non-perseverative error	8.65 ± 6.92	4.20 ± 3.72	4.15 ± 4.45	9.83	.01**
	Conceptual level responses	45.60 ± 22.59	60.48 ± 15.40	65.85 ± 10.03	15.58	.01**
	Categories completed	2.30 ± 1.99	4.58 ± 1.39	5.45 ± .88	47.58	.01**
	Trials to Complete category	50.90 ± 41.99	20.35 ± 12.70	20.15 ± 14.03	17.72	.01**

*p< 0.05, ** p< 0.01, *** p< 0.001WCST - Wisconsin Card Sorting Test, SCZ – individuals with schizophrenia, SIB – unaffected sibling of individuals with schizophrenia, HC – healthy controls,

Table-4: Differences in mean scores on test of verbal memory (AVLT) of the participants in three groups (N=120)

	Mean ± SD				
AVLT	SCZ	SIB	HC	F	Sig.
Trial 1	5.65 ± 1.70	7.10 ± 1.52	7.43 ± 1.34	15.34	.01**
Trial 2	6.65 ± 1.89	8.08 ± 1.47	8.48 ± 1.32	14.76	.01**
Trial 3	7.80 ± 2.19	9.15 ± 1.48	9.60 ± 1.26	12.33	.01**
Trial 4	8.68 ± 2.54	10.15 ± 1.41	10.40 ± 1.39	10.08	.01**
Trial 5	9.63 ± 2.78	11.25 ± 1.32	11.85 ± 1.15	14.77	.01**
List B	5.80 ± 2.03	6.75 ± 1.37	7.03 ± 1.33	6.38	.01**
Immediate call	8.55 ± 2.81	9.40 ± 1.55	10.83 ± 1.30	13.24	.01**
Delayed recall	7.10 ± 2.67	7.63 ± 1.69	9.13 ± 1.51	10.83	.01**
Recognition	5.40 ± 2.25	8.73 ±1.62	10.93 ± 1.33	98.30	.01**

*p< 0.05, ** p< 0.01, *** p< 0.001AVLT - Auditory Verbal Learning Test, SCZ – individuals with schizophrenia, SIB – unaffected sibling of individuals with schizophrenia, HC – healthy controls

individuals with Schizophrenia (SCZ) and their unaffected siblings (SIB) performed significantly worse than HC on tasks of attention (DST, TMT), verbal fluency (COWA, ANT), working memory (LNS, SST), executive functions (Stroop, WCST), verbal memory (AVLT).

Attention

Attention is a central mechanism for control of information processing, activating and inhibiting processes and constituting a complex system that involves different networks in specific areas of the brain.[33] It was observed that SCZ and SIB samples took longer time to complete trails and had decreased digit span than HC sample. This implied that individuals with schizophrenia and their unaffected siblings had poor selective attention function. The study on tests of visual attention and psychomotor performance found that the unaffected relatives performed poorly when compared with controls but the differences did not reach statistical significance.[34] Attention and working memory deficits have been implicated in the pathophysiology of schizophrenia, their presence in first-degree relatives suggesting a substrate (probably genetically mediated) for future development of the disorder.[35] Another study found no significant between-group differences on TMT-A between schizophrenia and HC.[36]

Verbal Fluency

Verbal fluency is the ability to generate as many words as possible starting with a particular letter within particular time duration and the later refers to the ability to produce words within a particular meaning based category. The tasks used to evaluate verbal fluency were controlled word association test (COWA), animal naming test (ANT).

The individuals with schizophrenia and their unaffected siblings generated fewer words than controls, repetition and intrusions of few words were also observed. This poor performance may be mediated by deficits in memory and speed of processing.[37] Individuals with schizophrenia and their unaffected siblings are known to share neuropsychological deficit in verbal fluency.[13] Verbal Fluency deficits have been reported to remain stable across the clinical course of schizophrenia.[38] Semantic fluency function has been considered as a potential endophenotypes for the early diagnosis of schizophrenia in the Chinese population.[39] This cognitive deficit might be caused by familial predisposition to schizophrenia and verbal fluency could be a candidate endophenotypes for schizophrenia specifically.

Working memory

Baddeley (1992) regarded working memory as a short-term storage system that involves the manipulation of recently stored information.[40] The working memory is divided in to two types they are visual working memory and verbal working memory. Verbal WM can be measured by Letter Number Sequencing (LNS) requiring the participant to encode, rearrange, and verbally produce letters and numbers in a series. SST was used to assess visual WM by moving the blocks in series of positions.

Individuals with schizophrenia display impairment in maintenance as well as active manipulation

of information and tend to perform poorly when compared with HC.[41,42] Whereas siblings of the patients with schizophrenia demonstrated impairment in encoding the information and retrieving of information, but not in maintenance of information.[43] However, another study concluded no differences between unaffected siblings and healthy controls on working memory.[44]

The performance of SIB sample was intermediate between SCZ and HC samples on SST. This was similar to previous research findings.[45,46] Park (1995) found that non-psychotic first-degree relatives of patients with schizophrenia tend to show spatial working memory deficits.[47] He further in his work in 2002 reported that spatial working memory deficits may be a potentially useful addition to the endophenotypic characterization of family members in establishing a comprehensive genome wide linkage analysis of Palauan families. A review suggested that spatial working memory deficits exist prior to illness onset and may be more potent trait markers for psychosis than cognitively dense tasks such as verbal memory.[48]

Executive functions

Response inhibition is a cognitive process that permits the individual to inhibit their impulses habitual, natural or a dominant behavioral in order to select a more appropriate behavior that consistent with their goals. It is also known as inhibitory controls. The functional magnetic resonance image (fMRI) studies have consistently revealed hypoactivation of dorsolateral pre frontal cortex (DLPFC) and of anterior cingulate cortex (ACC) in patients with schizophrenia performing a Stroop task.[49,50] Similarly using another response inhibition task, it has been show that schizophrenia patients and their sibling have decreased ACC activation and altered connectivity between ACC & DLPFC. Genetic variation of the COMT gene has been shown to affect performance on the Stroop test.[51]

WCST performance activated a neural network in dorsolateral region involved in information processing.[52] Poor WCST performance that is well known in schizophrenia reflected prefrontal cortical dysfunction and poor WCST performance in the sibling group could implicate similar prefrontal dysfunction.[20,53] In young high-risk relatives of schizophrenia patients, Edinburgh High Risk Studies found deficits in executive function,[54,55] while another study displayed poor performance on WCST in relatives of patients having a family history of schizophrenia when compared to relatives of patient without a family history of schizophrenia.[56] Relatives of SZ patients show higher perseverative errors but relatively normal non-perseverative errors than controls, suggesting cognitive set shifting to be a vulnerability marker of the illnesses.

Auditory memory

In the present study, patients with schizophrenia and their unaffected sibling performed significantly poorer on auditory verbal memory test compared to HC. Functional imaging studies have reported alterations of functional inter-connection in siblings of the patients with schizophrenia, particularly in prefrontal cortex areas and para hippocampus.[57,58] Genetic polymorphisms studies have linked with verbal memory deficit in sibling and patients with schizophrenia.[59] Additionally, the verbal memory impairments of healthy relatives have been associated with impaired encoding and manipulation of information, reasonably increased rates of forgetting.[60,61]

Important areas of the brain involved in memory function are hippocampus, amygdala, and frontal cortex. As a result, finding memory deficits in patients helps ascertain the exact brain structures linked with schizophrenia. Diagnosis of memory dysfunction in first-degree relatives who are genetically related, not only helps to explain endophenotypes but also can somewhat establish the concerned brain structures in the patients relatives.[62] Additionally, hippocampus and amygdala of relatives of the patients with schizophrenia have abnormalities in terms of quantity. Selective impairment in verbal memory among relatives proposes that such deficit comprises a familial, probably genetic, risk factor for schizophrenia.[63]

To conclude, the individuals with schizophrenia performed poorly than unaffected siblings and HC while siblings performed superior than individuals with schizophrenia but inferior to HC. The study used a comprehensive battery of neurocognitive tests that can be useful to understand the extent of scores on the tests. However, study had limitations of not controlling or matching intelligence, and using non cognitive measures of assessment.

This study would be helpful in understanding the endophenotypes value of cognitive functions in patients with schizophrenia and their unaffected siblings. Findings of the study could be helpful in developing preventive measures, psychological management and cognitive retraining for the unaffected siblings. The present research finding can be also helpful for caregiver of schizophrenia patients and also for understand the cognitive functions of the illness. The future work can be supported by electrophysiological measures, and genetic analysis to enhance insights into structural brain changes and their impact on functioning.

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Original Article

Assessment of Key Elements of Peer support among persons with severe Mental Illness

Ajay Badoni,[1] Nimmi A. Jose,[2] Irish Sheikh,[3] Shikha Tyagi,[4] B.S. Chavan[5]
[1-3]Department of Psychiatry, HIMSR & HAHC Hospital, New Delhi and
[4,5]Department of Psychiatry, GMCH Sector-34, Chandigarh
Contact: Ajay Badoni, E-mail: badoniajay13@gmail.com

ABSTRACT

Introduction: *Severe mental illness (SMI) is a major public health concern. SMI affects not just the individual but also the individuals in the microsystem. SMI potentially affects all significant aspects of a person's life: everyday activities, work functioning, interpersonal interaction, social involvement, etc. Peer support is an innovative, cost-effective, and cost-saving approach that comprises sustenance or services provided to people with mental health problems by other people who have experienced mental health problems themselves.* ***Aim:*** *To study the key elements of peer support amongst persons with SMI.* ***Material and Methods:*** *This was exploratory and cross-sectional in nature. The study was conducted at Disability Assessment Rehabilitation and Triage (DART) services of Mental Health Institute, Sector 32-B, Chandigarh. The sample size comprised 20 patients having Severe Mental illness (SMI) enrolled at DART services. The snowball sampling technique was used for data collection. Participants who had the capacity and gave written informed consent were included in the study. The capacity assessment tool was used, FGD was conducted and the result was found.* ***Results:*** *A total of 20 patients participated in the study and after FGD the different themes and sub-themes were identified. The themes were understanding, learning, role model, ventilation, social support, motivation, and socialization.* ***Conclusion:*** *The study concludes that the findings regarding peer support and its advantages were similar to those reported from other countries as well. Peer support groups as a concept will be an important addition to the management of patients with SMI. These groups will also increase the value, efficacy, and range of rehabilitation services being provided for patients with SMI. Therefore, we should be applying the findings from our study and developing this concept further for use in our patients and clinical services.*

Keywords: *Severe mental illness, Peer support, Focused Group Discussion, Disability Assessment and rehabilitation triage, Mental health institute.*

Introduction

Severe mental illness (SMI) is a major public health concern. It affects not just an individual but also the individuals in the microsystem.[1] SMI affects all significant aspects of a person's life including everyday activities, work functioning, interpersonal interaction, social involvement, etc. NIMH, UK has defined Severe Mental Illness based on (a) ICD-10 diagnosis of mental disorder including the diagnosis of Schizophrenia and functional psychosis and Severe affective disorders. Schizophrenia and functional psychosis including the following ICD-10 codes: F20, F21, F22, F23, F24, F25, F28, F29; Severe affective disorders include the following ICD-10 codes: F30, F31, F32.2, F33.3 (b) Duration of service contact of 2 years or more. (c) Severe dysfunction is measured by a score lower or equal to 50 in the Global Assessment of Functioning (GAF).[3] As per the National Mental Survey, 1.9% of the population have had a severe mental disorder in their lifetime, and 0.8% of people currently have one.

Although the burden of mental illness is especially concentrated among those with SMI disabilities, it also has multidimensional, social, and

long-term effects on family members of people with severe mental illness, including grandparents, parents, siblings, offspring, and spouses as well.[4] All persons who attend to the needs of a person with severe mental experience stress and subjective burdens as well as objective burdens.[5,6] The level of burden is higher among female caregivers, because of ongoing gender role expectations, females were mostly considered to take on the duty of providing direct care in different roles such as mother, wage earner, household manager, and key provider of emotional supporter.[9]

Peer support is an innovative, cost-effective, and cost-saving approach that comprises sustenance or services provided to people with mental health problems by other people who have experienced mental health problems themselves.[10] Peer support is defined as "social-emotional support, frequently coupled with instrumental support, that is mutually offered or provided by persons having a mental health condition to others sharing a similar mental health condition to bring about a desire for social or personal change."[14] Improves self-efficacy of persons with severe mental illness and is regarded as an effective intervention.[15] It is a system of giving and receiving help founded on key principles of respect, shared responsibility, and mutual agreement of what is helpful.[16]

Recovery in the field of mental health is an informally raised concept that has gained increasing distinction in the delivery of mental health services to people with severe mental illness over the past years. It is defined as a deeply personal, unique process of changing one's attitudes, values, feelings, goals, skills, and roles. It has been found that peer support is more effective and as well as an adjunct to conventional mental health services. Personal benefits to consumers and peers, and substantial savings to systems.[19]

Peer Supporters have the unique ability to help people in recovery and one another based on shared affiliation and understanding of their experiences with mental illness and/or substance use disorders. They offer support, strength and provide hope to peers. This promotes personal growth, wellness, and recovery. Although peer support is accepted and recognized in the treatment of many conditions (e.g. addictions, cancer...), the stigma and stereotypes associated with mental illness have led people at an advanced stage of their recovery to refrain from contributing to the mental health system.[17]

Often referred to as self-help or mutual assistance, peer support has been used by people dealing with different types of social situations, mental difficulties, and health issues, including those with alcohol or drug problems, bereaved persons, and people living with physical illness or impairment. Peer support has a long history among people with psychiatric diagnosis.[24]

In national epidemiological survey it has been found that the estimated overall weighted current prevalence of 10.6% and lifetime prevalence of 13.7% for any mental morbidity (including alcohol and substance abuse). This epidemiological survey also estimated a treatment gap of 73.6% for severe mental illness and of 85.0% for common mental disorders as well. A huge treatment gap of 73.6% makes it pertinent to use peer support services for providing continued informal or formal support to persons with SMI which is a chronic condition.[26] Hence the study was planned to assess the components of peer support group that might play an important role in the recovery of SMI patients.

Review of Literature

Peer support is an emerging field in mental health in India. Mostly the researches have been done in western countries and the findings are positive ones. A review study was done by Davidson and his colleagues at Yale's University School of 7 Medicine in the year 1999 on peer support among individuals with severe mental illness. They identified three primary methods of peer support developed by and for the community and reviewed the current empirical evidence of efficacy, effectiveness, and application of each of these strategies in contributing to the recovery of clinically disabled individuals. The three types reviewed were (1) naturally occurring community support groups, (2) consumer-run services, and (3) the health and rehabilitative environments employing customers as providers.[27]

In a study conducted on peer-to-peer psychoeducation in persons with schizophrenia, a peer counsellor was engaged for taking counselling sessions. The peer counsellor referred to a person who had been living with Schizoaffective disorder over the last 20 years. The counselling was done for an hour once a week that lasted for about 20–30

minutes, included two to three participants in each session and considered the participant's suggestions, thoughts and questions related to the disease. The finding suggested that peer therapy in schizophrenia is possible, and the peer counsellor can effectively address the questions of the patients.

Aims and Objectives

To study the key elements of peer support amongst persons with SMI.

Materials and Methods

Design: Study design was exploratory and cross-sectional in nature.

Setting: Study was conducted at Disability Assessment Rehabilitation and Triage (DART) services of Mental Health Institute, Sector 32 B, Chandigarh.

Sample Size: Sample size was comprised of 20 patients having Severe Mental illness (SMI) enrolled at DART services. Snowball sampling technique was used for data collection. Participants who had capacity and gave written informed consent were included in the study. The following inclusion and exclusion criteria was used for selection of participants:

Inclusion Criteria

- Patients with the diagnosis of SMI.
- Age group between 21-60 years.
- Patients attending DART services since at least six months.
- For the study peer was defined as the person with lived experience of SMI.

Exclusion Criteria

- Patients suffering from major medical (tuberculosis, HIV & AIDS), surgical and neurological disorders (head injury, dementia, delirium or epilepsy) requiring admission or active intervention.
- Patients with substance dependence (except nicotine and caffeine).
- Patients lack in capacity.
- Suicidal, violent or unmanageable patients.
- Patients with intellectual and other developmental disabilities

Tools

1. ***Socio-demographic and Clinical Data Sheet: (Annexure 1)*** It was specially designed for the study to capture socio-demographic and clinical details such as name, age, sex, contact details, education, marital status, family size, age of onset, duration of illness, duration of treatment, number of hospitalization, treatment details side effects if any etc.
2. ***Capacity assessment tool: (Annexure 2)***[51] Capacity assessment tool approved by ministry of health and family welfare society of India was used for assessing the capacity of the person.[51]
3. ***Focused group discussion: (Annexure 3)*** A focus group discussion was used as a tool in many qualitative researches. This usually involves group interviews, in which a small group of approximately 8 to 12 individuals are involved in a loosely structured discussion of various topics of interest. The discussions are led by a moderator or interviewer.

Procedure

For the purpose of the study, 10 male and 10 female patients with the diagnosis of SMI (NIMH UK) at the time of entry to DART and continued to attend DART services for at least last six months and fulfilled inclusion and exclusion criteria were considered. The patients were assessed for capacity on the basis of capacity assessment tool given by Government of India criteria listed in Mental Health Care Act, 2017[52] and those having capacity were approached for informed consent. Patients who gave informed consent were considered for the study.

Further the participants were administered socio-demographic and clinical datasheet. In this study, two separate groups of male and female patients each having 10 patients were formed and for each group, 3 Focus Group Discussions. (FGDs) were conducted for studying the components of peer support among SMI pts. For the purpose of forming focus groups snow ball sampling was used where one key person (who has been oldest and regular amongst patients) was identified first. One male and one female were identified and were asked to identify their peers who in turn were asked to name 2-3 peers till 10 such members were identified. All the FGDs were conducted along with the supervisors for about 45 to 60 minutes. To begin with, the group was encouraged to discuss the points which were relevant

for them. The researcher ensured that the discussion was goal directed and each participant was encouraged to participate. Each FGD was audio recorded, transcribed and analyzed.

Statistical Analysis

Qualitative data analysis was done using the method of thematic analysis which goes beyond counting explicit words or phrases and, focuses on identifying and describing both implicit and explicit ideas within the data, that is, themes.[53]

It had involved the following six stages of data analysis.

- Familiarizing with data (transcription and re-reading)
- Generating initial codes
- Searching for themes
- Reviewing themes
- Defining and naming themes
- Producing the report and Model Building

Result

Table-1: Themes and Sub-themes identified in FGD done with Male and female patients

S. No.	Theme	Sub-themes	Frequency
1.	Understanding	Ease of talk Regarding illness due to similarproblem	7
		Non Judgmental	3
		Non discriminating	5
		Non-stigmatizing	4
2.	Learning	Conversation skills	5
		Good habits	7
		Cleanliness	6
		New skills	8
		Money management	3
		Coexisting	6
		Shopping	4
3.	Role model	Inspiration	5
		Guiding others	3
		Importance of time management	4
		Progress	5
		Confidence	2
4.	Ventilation	Share personal family matters	5
		Sharing problems	5
		Stress reduction	4
		Emotional support	6
		Sharing sorrows and happiness	6
5.	Social Support	Instrumental support	6
		Educational support	6
		Financial support	5
		Source of secondary support	6
6.	Motivation	To attend rehab daily	7
		To try and learn new things	7
		Hard work	3
7.	Socialization	Having tea/coffee together	5
		Visiting homes and meeting their families	4
		Spending leisure time with them & talkingTelephonically	7, 5

Table-2: Ranking and rating of themes across 6 FGDs

Rank order	Theme	Total
I	Learning	65
II	Social support	53
III	Ventilation	51
IV	Socialization	43
V	Role Model	36
VI	Understanding	33
VII	Motivation	32

Discussion

The aim of the present study was to study the key elements of peer support among persons with severe mental illness (SMI). A total of 20 patients with SMI enrolled in DART services for the past six months duration were interviewed in focus group discussion (FGD) method using a semi-structured interview schedule designed especially for the purpose of this study. The interviews were

subsequently transcribed and the data was analyzed qualitatively using thematic analysis.

Table 1 depicts the socio-demographic profile of the patients. There was equal gender distribution (50% were male and 50% female), predominantly single (70%), studied up to at least graduate/post graduate (50%), unemployed (60%), of Hindu religion (75%), with a family income range of 20,001 & above rupees/month (60%), of urban background (95%), and from Chandigarh (50%).

Table 2 depicts the clinical profile of the patients. They had a predominant diagnosis of Schizophrenia and related psychosis i.e. ICD-10 code of F20-F29 (80% suffering with Schizophrenia) with duration of illness being more than 120 months in 80% of sample. Duration of Pharmacological current/on-going treatment was more than 120 months in 47% (N=7) followed by 60-120 months (N=5; 33%). Duration of non-pharmacological current/on-going treatment was 24-60 months in 47% (N=7), followed by 60-120 months (N=4; 26%) and 12-24 months (N=4; 26%) equally.

The sample had 3 FGDs from 10 males, and 3 FGDs from 10 females. From the FGDs, sub-themes were generated for each gender. A total of 145 and 257 sub-themes were generated from male and female FGDs respectively. The common major themes identified for each gender were- (1) Understanding, (2) Learning (3) Role model, (4) Ventilation, (5) Social Support, (6) Motivation and (7) Socialization.

These themes are hereby discussed in detail in the light of the evidence from present study and available literature.

1) Understanding

A total of four different sub-themes were identified (ease of talking about illness, possibility of other discussions related to sports/ politics, non-discriminating, non-stigmatising) for males. Same numbers were identified for females, but qualitatively they were a little different (ease of talking about illness, non-discriminating, non-stigmatising, non-judgemental). The commonest sub-theme for both males and females was "ease of talking about their illness".

*"***ge ,d nwljs lsa chekjh vkj nokb;ksa dh ckr djrs gSA tc Hkh ejht eu esa ;k ifjokj ls tM+h dksbZ leL;k gks rks viuh nkLrksa dh lkFk lk>k djrh gS] ftlls ejk fny vkj fnekx gydk gks tkrk gSA***"*

[We talk to each other about the illness & medicines. When there is any problem in my mind & family related matters share with my friends, which makes my mind & heart lighter.]

*"***esa lger gSA D;ksafd oks ykx Hkh blh nkSj ls xqtj jgs gSa vkj mudh lkFk Hkh oSlh gh 0;ogkj fd;k x;k gSA doy oks gh le> ldrs gSa vkj dksbZ ughA tc e>s lkl Qyus dh leL;k gksrh gS] rks o e>s nok,¡ yus vkj LokLF; dk Ë;ku j[kus dh fy, dgrs gSA oks e>s ejht ifjokj dh ckjs esa iNrs gSa] lkFk dke djrs gSa vkj ejht lkFk ?kwers gSA***"*

[I agree. Because those people have also facing & going through same phase, they have been treated in the same way. Only they can understand & no one else. When I have a breathlessness problem, they ask me to take medicines & take care of the health. They ask me about my family, work together & roam with me.]

2) Learning

A total of seven different sub-themes were identified (good habits, co-existing, conversation skills, new skills, cleanliness, shopping, money management) for males. Same numbers were identified for females, but quantitatively they were different regarding the frequency (new skills, good habits, co-existing, cleanliness, conversation skills, shopping, money management). The commonest sub-theme for both males and females was "good habits" and "new skills" respectively.

*"***fe= ge lgh fn'kk nsrs gSA ftUnxh esa nkLr rks gksus gh pkfg,(vki fdlh Hkh le; mUl viuh leL;k ij ppk dj ldrs gS fd vkidk viuk dke] eukjatu] ikfjokfjd ekeys vkfn fo"k;ksa ij /;ku dsfUnzr djuk pkfg, vkj rukoeDr jguk pkfg,A thou esa ge gj ckrksa ls dN u dN lh[kus dks feyrk gSA ge mUl vPNs thou] LoPNrk vkj lQkbZ] le; dk egRo vkfn dh ckjs esa cgqr dN lh[kus dks feyrk gSA***"*

[Friends give us the right direction. There should be friends in life; you can discuss your problem with them at any time. They say that

you have to do your work, entertainment, family matters, etc. should focus on the subject & remain relaxed. In life, we get to learn something from every human being. We get to learn a lot from them about good livings, hygiene & cleanliness, importance of time, etc.]

3) Role Model

A total of six different sub-themes were identified (sharing sorrows and joy, inspiration, progress, importance of time management, guiding others, confidence) for males. Five different sub-themes were identified for females, but quantitatively they were different regarding the frequency (guiding others, confidence, moving ahead in life, progress, inspiration). The commonest sub-theme for both males and females was "sharing sorrows and joy" and "guiding others/confidence/moving ahead in life" respectively.

"mu l ge thou thu dh {kerk] dke dk le; ij djuk] thou e vkx c<r jguk] vkRefo'okl e of)] [k'kh vkfn lh[k ldr gA o ge fyQkQk cukuk] ?keuk] nljk dh enn djuk t l fofHké dk;k e Hkh enn oxjg dj ldr gA"

4) Ventilation

A total of four different sub-themes were identified (emotional support, sharing personal family matters, sharing problems, stress reduction) for males. Same numbers were identified for females, but qualitatively they were a little different (sharing sorrows and happiness, sharing personal family matters, sharing problems, stress reduction). The commonest sub-theme for males and females was "emotional support" and "sharing sorrows and happiness" respectively.

"mud lkFk ikfjokfjd ckr] [ky lcf/k leL;k, vkfn lk>k djd e> ldu feyrk gA ejk eu vkjke vkj [k'kh egl l djrk gA tc e vdyk gkrk g vkj dN lkprk g] rk o ejh leL;k lurk g vkj ,d ifjokj d :i e e> lykg nrk gA"

[I feel relaxed by sharing family matters, sports related problems, etc. with them. My mind feels relax & happy. When I am alone & think about something, they listen to my problem & advice me as a family.]

5) Social Support

A total of four different sub-themes were identified (instrumental support, source of secondary support, educational support, financial support) for males. Five different sub-themes were identified for females, but quantitatively they were different regarding the frequency (emotional support, source of secondary support, instrumental support, educational support, financial support). The commonest sub-theme for both males and females was "instrumental support/source of secondary support" and "emotional support/ source of secondary support" respectively.

"ejk nkLr ejk ijk leFku djr g vkj dgr g fd ,slh dkbZ chekjh ugh g ft ldk byk t u fd;k tk ld vkj ftl ijh rjg l Bhd u fd;k tk ldA o e> 'kkjhfjd vkj lkekftd :i l çkRlkfgr djr g rkfd ejk vkRefo'okl c< ldA ejk nkLr dbZ fo"k;k ij ckr djd] l[k&n[k lk>k djd] Hkfo"; vkj thou Lrjd ckr e] Qlcd ij rLohj lk>k djd vkj thou'kyh e cnyko ij ppk djd ejk vdykiu nj dju e lgk;d gA"

[My friends fully support me & say that there is no disease that can't be treated and can't be cured completely. They encourage me physically & socially so that my self-confidence may increases. My friends are helpful in alleviating my loneliness by talking on many topics, sharing happiness & sorrow, about future & living standard, sharing photos in facebook and discussing lifestyle changes.]

6) Motivation

A total of three different sub-themes were identified (to attend rehab daily, to try and learn new things, hard work) for males. Same numbers were identified for females, which were qualitatively a little different (to attend rehab daily, to try and learn new things, hard work). The commonest sub-theme for males and females was "to attend rehab daily/to try and learn new things/hard work" and "to attend rehab daily/to try and learn new things"

respectively.

"eSa igys cgqr cgl djrk Fkk yfdu vc ugha djrkA xLlk Hkh de gks x;k gS] igys esa flQZ viuh ckr esa gh lksprk Fkk yfdu nksLrksa dh vkuk dh ckn esjh lkp esa dkQh cnyko vk;k gSA vc] esa nksLrksa dh ckr esa Hkh lksprk gS] mUgsa vPNk lq>koksa feyrk gS vkj nwljksa dh enn djus dh dkf'k'k djrk gSA esa viuh my>u [kqn gh lqy>k ysrk gS] lkekftd xfrfof/k;ksa esa Hkh Hkkxhnkjh c<+xbZA"

[I used to argue a lot before but now I don't. Anger has also decreased. Earlier I used to think only about myself but after the advent of friends, my thinking has changed a lot. Now, I also think about friends, meet them well & try to help others. I resolve my own complication myself. Involvement in social activities will also enhance.]

7) Socialization

A total of three different sub-themes were identified (to attend rehab daily, to try and learn new things, hard work) for males. Same numbers were identified for females, which were qualitatively a little different (to attend rehab daily, to try and learn new things, hard work). The commonest sub-theme for males and females was "to attend rehab daily/to try and learn new things/hard work" and "to attend rehab daily/to try and learn new things" respectively.

"ejs nksLrksa us [kq'k;ksa vkj n[kksa Hkfo"; vkj thou Lrj dh ekeyksa dh ckr esa ckr djdk vkj lq>k djdk] Qksudc ij rLohj lq>k djdk] thou'kSyh esa cnyko tSls fo"k;ksa ij pPkk djdk ejs vdsyiu dk nj djus esa ejh enn dh gSA"

[From them we can learn the ability to live life, do the work on time, keep moving forward in life, increment in self-confidence, happiness, etc. They can also help us to do various works like making envelope, roaming, helping others, etc.]

Peer support is an emerging field in mental health in India. No single study has yet been done in the field of peer support among persons with severe mental illness in India.

The main themes identified in the study for both males and females (in descending order of frequency) were: (1) Learning, (2) Social Support, (3) Ventilation, (4) Socialization, (5) Role model, (6) Understanding, (7) Motivation.

Mostly the research has been done in western countries and findings are positive. Therefore, the comparison of findings in our study will be done in relation to literature from West. Also, the findings of our study are being discussed together as western literature is mixed comprising of reviews and original research.

Around 2 decades back, a review had shown that peer support among individuals with SMI helped in improving symptoms, promoted enhancing larger social networks and enhancing quality of life of the individual as well.[27] This is in keeping with Theme 2 in our study.

A study conducted on peer to peer psycho education in schizophrenia. They found that they can act as role models and help cope with the illness. Also, peer-to-peer groups have positive therapeutic outcomes, such as returning to work, enhancing self-esteem and gaining fresh insight into individual issues. This is in keeping with Theme 6 in our study.[53]

Castelein et al (2008)[31] conducted a randomized control trial in Amsterdam, Netherland on "Effectiveness of peer support groups in psychosis". They showed that peer support groups are a valuable tool for psychosis sufferers by improving their social network and facilitating and reinforcing shared relationships. This is in keeping with Themes 2 and 4 in our study.

A study was conducted on peer support among 10 inpatients in adult mental health setting in Montreal, Canada, which had 6 suffering with schizophrenia.[32] Interviews were transcribed and analyzed in verbatim form using a descriptive qualitative system. Supporting acts includes assisting with everyday living tasks, sharing material items, providing information and therapy, sharing a social life, and offering emotional support. Positive outcomes included improved mental health outcomes and quality of life. This is in keeping with Themes 1, 3 and 4 in our study.

Rogers et al did a systematic review study in the field of psychiatric rehabilitation. The study had examined 53 research studies to evaluate disability rigor and reported results for peer services on minimum research quality thresholds. This systematic analysis research showed the advantages of peer driven programs in engaging and sustaining indivi-

duals in programs but not in outcomes, and providing specific and distinguishing skills and perspectives that can be useful in attracting and keeping individuals in programs. This is in keeping with Themes 5 and 7 in our study.[34]

A systematic review study conducted on peer-based health interventions for people with serious mental illness[25] showed that peer workers offers support and motivation which is especially warm and empathetic as it is grounded in personal experience and they provide role models for rehabilitation to service users. This is in keeping with Themes 2, 5 and 7 in our study.

Summary and Conclusions

The present study aimed to study the key elements of peer support among persons with severe mental illness (SMI). A total of 20 patients with Severe Mental illness (SMI) who had undergone rehabilitation and had been placed through Disability Assessment and Rehabilitation Triage (DART) Services, Mental Health Institute (MHI) for the past six months. Participants who gave written informed consent and fulfilled the specified inclusion and exclusion criteria were inducted into the study. They were interviewed using focus group discussion (FGD) method by applying a semi-structured interview schedule designed especially for this study. The interviews were then transcribed and the data was analyzed qualitatively using thematic analysis.

The sample had 3 FGDs from 10 males, and 3 FGDs from 10 females. From the FGDs, sub- themes were generated for each gender. A total of 145 and 257 sub-themes were generated from male and female FGDs respectively. The commonest major themes identified for each gender were - (1) Understanding, (2) Learning (3) Role model, (4) Ventilation, (5) Social Support, (6) Motivation and (7) Socialization. On combining the sub-themes for both genders, the main themes that emerged (in descending order of frequency) were - (1) Learning, (2) Social Support, (3) Ventilation, (4) Socialization, (5) Role model, (6) Understanding, (7) Motivation.

These findings regarding peer support and its advantages were similar to that reported from other countries as well. Also, people have talked about need for peer support groups and services from developing countries, like India. Peer support groups as a concept will be an important addition to the management of patients with SMI. These groups will also increase the value, efficacy and range of rehabilitation services being provided for patients with SMI. Therefore, we should be applying the findings from our study and developing this concept further for use in our patients and clinical services.

Strength and Limitations

Strengths of the Study

- Qualitative nature of the study allowed in depth exploration of the subject.
- First study on assessment of key elements of peer support among persons with severe mental illness from mental health rehabilitation center in India.
- Usage of sound methodology for studying the subject matter.

Limitations of the Study

- Small sample size of the study.
- As the study is qualitative in nature, chance of subjective bias is relatively high.
- As the study is qualitative, results can't be generalized on a larger population

Future Directions

- Findings of the study can serve as pilot for future studies in the field of peer support among persons with SMI.
- Results can help introduce formal peer support programmes as part of management regime.
- Need of carrying out quantitative studies on the topic.

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Psychomicrobiology

Brain Microbiota Axis in Alzheimer's disease: A New Insight

Akanksha Patel,[1] Anmol Singh Bhatia,[2] Nirmaljit Kaur,[3] M.S. Bhatia,[4] Shalini Malhotra[5]
[1,5]Department of Microbiology & [3]Medicine, ABVIMS & Dr. R.M.L. Hospital, New Delhi-110001 and
[4]Department of Psychiatry, HIMSR & HAHC Hospital, New Delhi-110062
Contact: Nirmaljit Kaur, E-mail: njkbhatia@yahoo.co.in

Introduction

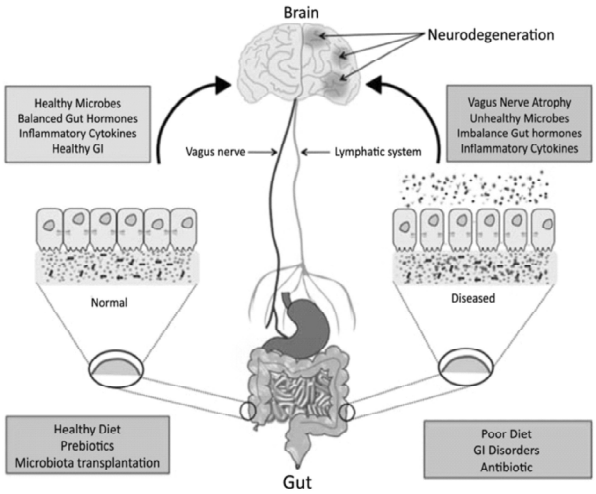
Alzheimer's disease (AD) is a progressive neurodegenerative disorder. It is characterized by the presence of extracellular amyloid-β (Aβ), peptides and neurofibrillary tangles (NFTs) in the brain hyperphosphorylated and aggregated tau being the major components. The etiology of Alzheimer's disease (AD) is multifactorial.[1]

AD is a complex multigenic disease. It is associated with mutations in three genes presenilin 1 (PSEN1), presenilin 2 (PSEN2) located on chromosome 14 and APP on chromosome 21. Mutation can be induced by Genetic and environmental factors. Genetic mutations are very complex to understand and various hypothetic causes has been attributed to it like oxidative stress, neuroinflammation and bacterial infection theory.[2] According to bacterial infection theory dysbiosis of microbiota has association with neuroinflammation and hence neurodegenerative disorders.[3]

The human microbiota is composed of trillions of microorganisms which interact intricately with approximately 20,000 human genes. This complex interplay between the host microbiota and our genetic makeup plays a critical role in health and disease susceptibility. A variety of bacterial flora is present in various parts of the body like skin, oral cavity, gut, nose and lungs etc.

Microorganisms in a healthy gut regulate digestive pH and collaboratively construct a protective barrier against infectious agents. An imbalance in gut microbiota can occur when normal friendly gut flora are replaced by toxigenic microbiomes. The toxic substances secreted by bacteria like endotoxins, lipopolysaccharide (LPS) and amyloids can increase the permeability of the intestinal epithelial barrier, leading to the production of proinflammatory cytokines and chemical substances that can cross the blood-brain barrier promoting a neuroinflammatory response and microglial activation in brain. This leads to formation of senile plaques and neurofibrillary tangles (NFTs).[1,4] Connection between brain and gut microbiomes is labelled as microbiota-gut-brain axis (MGBA) MGBA.

MGBA is connected through various pathways like immune, neuroendocrine, metabolic and neural pathway facilitating comprehensive coordination across physiological systems within the body.[5] Understanding gut microbiota–brain axis via vagus nerve has opened up the possibilities of developing novel treatments for neuropsychiatric disorders.[6]



Blood brain barrier (BBB) plays an important role in prevention of neuroinflammation by inhibiting entry of toxic substances. Normally, capillary endothelial cells in brain restrict the entry of toxic and other harmful substances into the brain by P-glycoprotein pathway (PGP) which is an ATP dependent

efflux pump. PGP efflux pump removes drugs and toxins out of the cells. Microbiome alteration in AD patients causes dysregulation of PGP leading to increased permeability of BBB. Another protective mechanism is formation of short chain fatty acid by the gut microbiota. Gut microbiota produce Short chain fatty acid (SCFA) like butyric acid by fermentation of dietary fiber. SCFA inhibits aggregation of beta amyloid proteins. In AD patients it has been seen that SCFA producing microbes are reduced and thus there is aggregation and accumulation of amyloid in brain.[7] Apart from toxins there are other substances which are produced by the gut microbiota, such as monoamines and amino acids that can travel through the lymphatic and vascular systems to reach central neurons potentially influencing their activity and affecting human behavior.[2]

The MGBA is influenced by microbiome inhabiting oral cavity which is the primary entry point to the human body. The oral cavity microbiota are diverse microbial community. These colonize two distinct regions: soft tissues of oral mucosa and hard surfaces of teeth. Interference in these microbiomes may contribute to development of autoimmune diseases apart from psychiatric and neurodegenerative disorders. Alteration in oral microbiota has been linked to neuroinflammation due to stimulation of immune cells of central nervous system (CNS) such as microglia. According to a study conducted in the year 2023, patients with chronic periodontitis have been found to have Porphyromonas gingivalis. P. gingivalis is a potential risk factor for the formation of amyloid-beta (Aβ) plaques, cognitive impairment and dementia including Alzheimer's disease.[6] Through everyday activities like brushing teeth oral microbiota can enter systemic circulation and may harm various organs and the central nervous system (CNS). From systemic circulation microbiome or its products can enter the brain if the BBB is compromised or becomes more permeable. Factors like systemic inflammation or aging can increase BBB permeability. There is direct neuroanatomical connection of Locus coeruleus in pons with periodontal free nerve endings and proprioceptors. Toxicants selectively enter Locus coeruleus and cause decreased production of noradrenaline triggering neurodegenerative Alzheimer's disease.[8]

Similarly, nasal microbiota brain axis has important role to play in neuroinflammation. Diverse community of microorganisms reside in nasal mucosa that contributes to maintenance of nasal mucosal health and overall immune system function. The nasal microbiota–brain axis is connected through various pathways like intracellular, paracellular and transcellular. **Intracellular pathway** provides a direct route from the nasal mucosa to the brain via nerves. From nasal mucosa axons of olfactory receptor neurons reach olfactory bulb in the brain. In p**aracellular pathway s**ubstances navigate through the intercellular spaces between the epithelial cells causing modulation of the immune system. **Transcellular Pathway** involves the passage of substances across epithelial cells. Advancing age significantly causes olfactory dysfunction, which could be an early indication with neurodegenerative disease. Other mode through which nasal microbiota can affect CNS is by production of neurotransmitters or metabolites capable of crossing the blood-brain barrier.[7]

Additionally, the lung microbiome through the lung-brain axis play a significant role in the development of central nervous system diseases and neuropsychiatric disorders.[9] Lung brain axis is not much explored for now. There are various potential pathways through which the lung microbiome could brain function. Microbes or their by-products can breach the capillary barrier to enter bloodstream, eventually reaching the brain through blood brain barrier. This route could potentially trigger immune responses thereby affecting neural functions. Alterations in lung microbiome could lead to immune modulation. Production of cytokines or other signaling molecules could impact brain function through systemic humoral and cell-mediated immunity.

Understanding these pathways of bidirectional communication between oral cavity, gut nose, lung and the brain is crucial to unravel the complex interplay between the microbiome and brain health.

The gut microbiota is the most explored among human microbiomes, others such as those associated with the skin, oral cavity and respiratory tract require further research to fully understand their significance.[7]

Conclusion

Alzheimer's being the old age disease has a "age-related dysbiosis" hypothesis which suggests that AD may develop as a consequence of immune

modulation caused by significant alterations in the gut microbiota composition as the individual ages. As age advances there is an observed increase in proteobacteria and a reduction in beneficial probiotics such as bifidobacteria, which releases neuroprotective molecules like short-chain fatty acids (SCFAs). These changes are critical because SCFAs have anti-inflammatory properties and play a role in maintaining the health of the gut-brain axis. The age-related dysbiosis hypothesis highlights the interconnectedness of the gut microbiota, systemic inflammation, and brain health. As the composition of the gut microbiota shifts to proteobacteria with age, it may exacerbate inflammatory processes both peripherally and within the central nervous system. This creates a conducive environment for the accumulation of amyloid-beta plaques and tau tangles hallmark features of AD.

Research on the gut microbiota–brain axis in neuropsychiatric disorders is advancing rapidly. Apart from AD, alterations in the composition and diversity of the gut microbiota have also been linked to various other psychiatric disorders including schizophrenia, major depressive disorder (MDD), bipolar disorder (BD), autism spectrum disorder (ASD), anxiety and even neurodegenerative disorders such as Parkinson's disease. Future research on the role of the host microbiota in neuropsychiatric disorders holds significant promise for uncovering the complex relationship between microbiota in various tissues and the brain.

Change in diet and habits can cause substantial shifts in the composition of core microbial communities in the gut. These shifts impacts overall wellbeing, influencing not only gastrointestinal health but also metabolic and neuropsychiatric health. Introduction of lifestyle modification like inclusion of prebiotics, probiotics, symbiotics in diet may help. Fecal microbiota transplantation and vagus nerve stimulation are novel therapeutic measures.[10,11]

Exploration of the microbiota's impact on neuropsychiatric disorders is a burgeoning field with profound implications. By understanding how the microbiota influences brain function and behavior, scientists may develop other targeted treatments that would modulate the microbiota to improve mental health outcomes. As our understanding deepens, it may pave the way for novel therapeutic interventions like fecal microbiota transplantation and vagus nerve stimulation. The mental health and overall well-being emphasizes the importance of the microbiota in maintaining a balanced and healthy life.[3,4]

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Psychophysiotherapy

Role of Psychophysiotherapy in Low Back Pain

Jaswinder Kaur,[1] Mansi Gupta,[2] Megha Masaun,[3] M.S. Bhatia[4]
[1-3]Department of Physiotherapy, Dr.RML Hospital & ABVIMS, New Delhi
[4]Department of Psychiatry, HIMSR & HAHC Hospital, New Delhi
Contact: Jaswinder Kaur, linktojk@yahoo.com

Introduction

Low back pain (LBP) is one of the most widely recognized conditions that debilitate people functional capacity in activities of daily living and at work, as well as their general wellbeing and quality of life. It represents an especially socio-economic problem due to the costs associated with repeated treatments, long-term absence from work and need for social support.[1] Patients with severe LBP usually present with high disability,[2] poor postural habits,[3] impaired proprioception, high number of comorbidities and high degree of functional limitation.[4] Chronic low back pain (CLBP) is the most common cause of disability worldwide, affecting about 12% to 30% of the adult population.[5] Chronic pain has a multidimensional nature and in addition to nociceptive and physiological aspects, it also includes aspects relating to the emotional and cognitive sphere.[6] Low back pain pathogenesis can also be diverse, including organic, non-specific etiology, and psychological causes.[7,8] Psychological factors play an important role in the experience of pain,[9,10] as patients with chronic low backpain who experience anxiety tend to exacerbate the painful sensation and increase illness behavior,[11] with catastrophizing pain.[12] Psychosocial factors (e.g., anxiety or maladaptive beliefs) negatively influence clinical outcomes in patients with LBP as they contribute to pain development, pain aggravation,[13] and pain chronification.[14] Individuals who suffer from low back pain may have a wide range of problems - physical, psychological, physiological and so on.[15] History and physical examination are enough to determine the cause of back pain in most cases. Early imaging in the adult population correlates with worse outcomes, as it tends to result in more invasive treatments that provide little benefit to patients.[16] However, the presence of concerning signs warrants diagnostic testing. In adults, back pain persisting longer than 6 weeks despite appropriate conservative manage-ment is also an indication for imaging.[17]

One of the most prevalent causes of LBP among patients is Lumbar prolapsed Intervertebral Disc (PIVD), affecting approximately 10% of the population leading to job-related disability and is also a leading contributor of absenteeism.[18] Prevalence is higher in men as compare to women and most of the individuals are between 30 and 50 years of age.[19] Obesity, smoking, sedentary lifestyle, and socio-economic conditions are associated risk factors.[20] PIVD may result in radiculopathy symptoms including tingling, numbness, weakness and neurological impairment of unilateral or bilateral lower extremity[21] Radiculopathy symptoms may arise from disc herniation, spinal stenosis or post-operative scarring and radiates down the leg in a dermatomal pattern and is often described worse than backpain. In approximately 90% of the cases of low backpain, radiculopathy is caused by herniated disc with associated nerve root compression but lumbar stenosis and less frequently tumors are the possible causes. Approximately 3-5% of the population are affected by chronic low back pain with radiculopathy where men and women are equally affected.10-25% develop symptoms that persists for more than 6 weeks.[22] Sciatica is the classic presenting symptom of lumbosacral radiculopathy, and patients describe this pain as sharp, dull aching, burning or throbbing pain in unilateral or bilateral lower extremity. Pain related to disc herniation is exacerbated by bending forward, sitting, coughing or sometimes walking. Characteristic feature of lumbar canal stenosis is worsening of pain during walking and relieved by bending forward. Paraesthesia is specific to pain radiation and this helps in identifying the level of involvement.[23]

Treatment

Lumbar PIVD results in significant disability, pain, and loss of productivity.[24] Therefore, an evidence-based treatment technique for the management of lumbar PIVD has immense clinical significance. Guidelines for management of low back pain recommend the non-pharmacological and non-invasive management including the provision of advice to stay active and the use of patient education and exercise therapy.[24] Guidelines regularly recommend the use of physical exercise for non-specific LBP. Conservative management including Medications, Physical therapy modalities, exercises are typically first-line treatments used for treatment and prevention of low back ache with sciatica. Physical therapy modalities provide relief from symptoms by promoting healing of the underlying causative factors and prevent recurrences. Medical management includes NSAIDs, acetaminophen, and in severe cases, opiates. Systemic steroids are often prescribed for acute low back pain with radiculopathy. Interventional techniques include epidural steroid injections and percutaneous disc decompression and IN refractory cases, surgical decompression and spinal fusion is performed. A study revealed that appropriate use of epidural injections to treat sciatica could significantly improve the pain score and functional disability score leading to a decrease in surgical rate.[25] A study evaluating the effect of non-steroidal anti-inflammatory drugs, or Cox-2 inhibitors reported that the drugs have a significant effect on acute radicular pain compared with placebo.[26] But other studies say that there are no positive effects on lumbar radicular pain.[27] Studies on the effect of acupuncture in people with acute lumbar radicular pain found a positive effect on the pain intensity and pain threshold.[28] Among patients with acute lumbar radiculopathy, oral steroids provides pain relief and improve function.[29] Physical therapy modalities including low frequency currents like Transcutaneous electrical stimulation, Medium Frequency Currents like Interferential therapy, and high frequency currents including Short wave Diathermy, Microwave Diathermy, Ultrasonic Therapy and Traction are commonly used in the treatment of lumbosacral pain with radiculopathy. Conservative Management is indicated in first 6-8 weeks and if complaints remain present for at least 6 weeks post conservative treatment, surgical treatment can be offered.[30]

In addition to this, there is significant evidence to prove that encouraging early movement in lower back pain through various exercises is one of the most important aspects of treatment of lumbar pain with radiculopathy. Spinal Mobilization Techniques including Maitland Vertebral Mobilizations, Mulligan's Movements with Mobilizations, Mckenzie approach of Mechanical Diagnosis and therapy and Manipulative physiotherapy consisting high velocity thrusts concentrates on promoting mobilization of the specific affected area thereby decreasing lumbosacral pain with radiculopathy. The McKenzie method of mechanical diagnosis and therapy (MDT) is designed to categorize patients into homogeneous subgroups (derangement, dysfunction, or postural syndrome) in order to direct treatment with specific exercises and postural advice.[31] Neural mobilization technique is one of the interventions which are used for treatment of low back radiculopathy. This aims to mobilize the peripheral neural tissue and structures surrounding them thus influencing the mechanical properties of peripheral nerve.[32]

Core stabilization exercises[33] emphasize on improving the strength and stability of the muscles which have been weakened due to the lower back pain. A series of properly structured exercises and stretching are usually carried out in context of the patient's individual condition and cause of the lower back pain. Since work-related hazards account for more than 65% of lower back problems, physiotherapists also concentrate on providing ergonomic advice and prescribing accurate ergonomic device, guiding the patient on using the appropriate infrastructure at work to avoid and cure lower back pain. Postural Care and Correction including guiding the patient about the correct postural habits and ways to maintain accurate posture to avoid lower back pain. Regular education concerning the causes, the mechanisms, the natural history, and prognosis of low back pain, and benefits of physical activity and exercise are delieverd. In addition, guidance and reassurance on self-management, guidance to stay active and avoid bed rest, guidance to return to normal activities, or referral for a group or an individual exercise program are also included.[33]

Treatment guidelines of low back pain, promote the avoidance of bed rest, and the continuation with activities as usual. The aim of physical treatments

is to improve function, and to prevent disability from getting worse. In chronic low back pain, exercise therapy has become a first-line treatment and should be routinely used.[33] Saragiotto et al showed that exercise is a moderately effective treatment for chronic LBP, especially motor control exercise. However, evidence shows that MCE (Motor Control Exercises) is not superior to other treatments. The exercise choice of patients with CLBP may depend on the preference of patients or therapists, the training of therapists, cost and safety.[34] Searle et al reviewed published reports and found that strength/ resistance training and coordination/stability training have better therapeutic effects in the treatment of CLBP compared with other types of exercise.[35] Kamper et al evaluated the long-term implications of multidisciplinary biopsychosocial recovery for patients with LBP. Multidisciplinary interventions were found to be more effective than general management and physiotherapy in reducing pain and disability for CLBP. For the impact of work, multidisciplinary recovery is more effective than physical therapy but not more effective than general management.

Since psychosocial factors are critical to the development of chronic, disabling LBP so chronic LBP should be approached by considering not only its morphological basis but also its biopsychosocial interventions.[37-39] Psychotherapeutic approach plays an important role in treatment outcomes in patients with chronic pain[40] which consists of building Patient-clinician relationship, Reassurance, Combined behavior therapy CBT, Acceptance and commitment therapy (ACT) and mindfulness, Encouragement of self-management.

Patient-clinician relationship, particularly rapport building, plays an important role in treatment outcomes in patients with chronic pain as it enhances placebo effect[41] Patient satisfaction is positively associated with affiliative behaviors, such as forward-leaning posture, smiling, nodding, and a relatively high-pitched vocal tone, and negatively associated with physician control.[42] Patient-centered support, including psychological support, promotion of patient's health literacy, and empowerment of patients to cooperate in finding the correct treatment is equally important Clinician's empathy and trustworthiness plays an important role to influence outcome in patients with chronic pain.[43,44]

In recent trend of behavioral medicine intervention, it has been found that CBT significantly improves disability and pain catastrophizing in patients with chronic pain after treatment and at follow-up.[45,46] As negative and catastrophic thoughts are highly correlated to pain complaints,[47] CBT focuses on restructuring the negative cognition of the patient into a realistic appraisal.

Acceptance and commitment therapy (ACT)[45] is used increasingly for treating chronic pain now a days. ACT focuses particularly on the concepts of acceptance, and mindfulness. Mindfulness has been associated with a small effect of improved pain symptoms compared with control treatment for chronic pain in a meta-analysis of 30 randomized controlled trials; however, there was substantial heterogeneity among these studies.[48]

Conclusion

Many chronic low back pains have both organic and psychological factors. People with chronic pain usually suffer from not only pain but also overlapping problems, such as depression, anxiety, sleep disorders, working with disabilities, drug overuse, and low quality of life). Thus, Physiotherapy Management along with biopsychosocial treatment, which can be substituted by a multidisciplinary approach, is an essential strategy for treating chronic low back pain.

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Psychophysiotherapy

Psychophysiotherapeutic Approach in Sleep disorders

Jaswinder Kaur,[1] Shweta Sharma,[2] Ashoo,[3] M.S. Bhatia[4]
[1-3]Department of Physiotherapy, Dr. RML Hospital & ABVIMS, New Delhi
[4]Department of Psychiatry, HIMSR & HAHC Hospital, New Delhi
Contact: Jaswinder Kaur, linktojk@yahoo.com

Introduction

Sleep is a natural and reversible state of relative inactivity and reduced responsiveness to external stimuli, accompanied by a loss of consciousness, occurring at regular intervals.[1] It occupies one-third of human life and it is essential for physical, emotional and cognitive wellbeing. It is essential for many brain processes including the consolidation of memories, alertness, processing speed and decision making.[2,3] Healthy regulation of these processes by sleep has a significant relationship with global functioning and scores of quality of life.[4] Sleep disorders are a group of conditions that affect the ability to sleep properly in a routine. Inadequate sleep leads to a plethora of problems associated with a wide range of endocrinal, metabolic, and immunological systems dysfunctions thus compromising the higher cortical functions, post-physical activity recovery, cognitive performance and mood.[5,6]

Types of Sleep Disorders

Sleep disorders are grouped into six major categories in the ICSD-3-TR:[7]

- Insomnia Disorders
- Sleep-Related Breathing Disorders
- Central Disorders of Hypersomnolence
- Circadian Rhythm Sleep-Wake Disorders
- Parasomnias
- Sleep-Related Movement Disorders

Insomnia Disorder

It is the expressed dissatisfaction with the quantity, quality or timing of sleep. Insomnia may present in different ways including, prolonged sleep latency, early or multiple awakenings during sleep or that the sleep is non restorative and do not feel refreshed in the morning.[8] The prevalence is estimated to be approximately 10% and is higher among women than among men (17.6% vs. 10.1%%, respectively).[9]

Sleep Related Breathing Disorders are divided into four sections: Obstructive sleep apnea (OSA), central sleep apnea (CSA) syndromes, sleep-related hypoventilation disorders, and sleep related hypoxemia disorder. Obstructive sleep apnea is characterized by partial upper airway obstruction (hypopneas) or repetitive episodes of cessation of breathing (apneas).[10] The obstructive sleep apnea is associated with an obstruction in the airway resulting in inadequate ventilation due to reduced blood oxygen saturation and increased breathing effort. Central sleep apnea is a disorder with unknown cause is characterized by recurrent episodes of cessation of breathing during sleep without associated ventilatory effort where as Central sleep apnea due to Cheyne-Stokes breathing pattern is characterized by recurrent apneas and/or hypopnea as alternating with prolonged hyperpnea in which tidal volume waxes and wanes in a crescendo–decrescendo pattern.[11]

Hypersomnia of Central Origin

The hypersomnia disorders are those in which the primary complaint is daytime sleepiness and the cause of the primary symptom is not attributable to disturbed nocturnal sleep or misaligned circadian rhythms. Central hypersomnia is usually divided into three main subtypes: narcolepsy type 1, narcolepsy type 2, and idiopathic hypersomnia (IH).[12]

Circadian Rhythm Sleep Disorders (CRSD)

The major feature of these disorder is a

persistent or recurrent misalignment between the patient's desired sleepiness. The wake episodes can occur at undesired time thus the patient may complain of insomnia or excessive Sleepiness. The prevalence of CRSD among the general population depends on the type and is reported to be between 0.13 to 0.17%.[13]

Parasomnias

It is an abnormal disruption of sleep like nightmares, sleep walking, sleep talking, bed-wetting, sleep apnea or nighttime seizures. It consists of abnormal sleep-related movements, behaviors, emotions, perceptions, dreaming and autonomic nervous system functioning. These arousals are common in children and can occur not only from nocturnal sleep but also from daytime naps.[14]

Sleep-Related Movement Disorders

The sleep-related movement disorders are characterized by relatively simple and stereotyped movements that disturb sleep such as periodic limb movement disorder and restless legs syndrome. Restless legs syndrome is characterized by the complaint of a irresistible strong urge to move the legs, often accompanied by painful symptoms.[15] Periodic limb movement disorder is an independent disorder of repetitive, highly stereotyped limb movements that occur during sleep.[16] Sleep related leg cramps are painful sensations that are associated with sudden intense muscle contractions, usually of the calves or small muscles of the feet.[17]

More specifically, sleep disruptions are associated with higher risk of diabetes, stroke, coronary heart disease and heart attack, obesity as well as mental disorders.[18]

Several studies have shown that sleep disorders (insufficient sleep, excessive amount of perceived sleep, abnormal movements during sleep) are common among the non-motor symptoms in patients with neurological disorders.[19]

Furthermore, physical therapy may be considered a powerful non pharmacological intervention for sleep disorders also in neurological disease with consequent positive effects on both motor and non-motor functions and with minimal side effects.[20]

Management

Pharmacological treatment: The FDA has approved an array of prescription medications for the treatment of insomnia, including Benzodiazepines (BZD) and non-BZD drugs, the melatonin agonist ramelteon, the sedating antidepressant doxepin, and the orexin receptor antagonist suvorexant.[21] Excessive daytime sleepiness is often associated with obstructive sleep apnea, Amphetamines may be used to induce daytime alertness. Restless legs syndrome and periodic limb movement disorder may be treated with dopamine agonists[22]

Psychological Interventions

Cognitive behavioral therapy (CBT-I): It is an evidence-based treatment of insomnia which includes a combination of behavioural and cognitive techniques in order to change maladaptive sleep habits, lower sleep-disrupting arousal and to alter sleep-related misconceptions and thought patterns. Studies have demonstrated that eight sessions or 2 months of CBT-I is equal to pharmacological treatment in the short-term and has longer lasting effects.[23,24] Studies investigating the effects of CBT-I on psychiatric populations are vitally important as insomnia is commonly comorbid with psychiatric diagnoses and the comorbidity has been associated with more severe psychopathology.[25] Dedicated CBT group treatment for insomnia improves sleep more than treating sleep as an adjunct to other mental health treatment. It includes education about sleep and components including relaxation training, stimulus control, sleep restriction and cognitive strategies.[26]

Interpersonal and Social Rhythm Therapy (IPSRT): There is a prevalence of comorbid mental conditions, such as sleep difficulties and irregular sleeping patterns in bipolar disorder patients.[27] Interpersonal components of therapy focuses on the resolution of existing interpersonal issues, such as unresolved sorrow, interpersonal disagreements, role shifts, and interpersonal isolation.[28] Social rhythms are regular patterns of everyday routines that can affect the circadian clock both directly and indirectly through light exposure. Social rhythm therapies (SRTs) encourage the adoption of consistent, daily routines of activity to aid in the restoration of circadian biological processes and to enhance mood.[29] The goal of IPSRT is to stabilize everyday rhythms including sleeping, waking up and mealtimes.[30] Study concluded that applying interpersonal and social rhythm therapy proved to

be effective in improving sleep and psychological adjustment among patients with bipolar disorders.[31]

Imagery: Imagery rehearsal therapy (IRT) have been shown to successfully reduce nightmare frequency, posttraumatic stress disorder severity and other mental health problems including depressive symptoms and increase sleep quality when treating nightmare disorder(ND).[32] It also helps to reduce sleep onset latency and the subjective severity of insomnia and to enhance sleep quality.[33]

Mindfulness: It is the self-regulation of attention along with the inhibition of elaborative processing.[34] It involves nonreactive, moment-to-moment monitoring of the content of experience. Studies suggested that mindfulness may improve sleep via reductions of both primary and secondary arousal. Primary arousal is the mental activity directly related to the inability to sleep such as beliefs about daytime consequences. Secondary arousal is the relationship with thoughts about sleep such as the tendency to create bias in the attention and perception of sleep related thoughts.[35] Studies demonstrated the efficacy of mindfulness in women experiencing night sweats and hot flashes symptoms in addition to improving subjective sleep quality, anxiety, perceived stress, and quality of life.[37] Studies shown that it increases sleep quality and self-esteem in adolescent psychia-tric outpatients while also reduces depression, anxiety and somatic distress.

Physiotherapy interventions

Role of physical therapist:

- Assess overall sleep health & screen for sleep disorders esp. Insomnia, OSA & RLS.
- Identify impairments related to sleep dysfunction
- Implement and progress therapeutic interventions
- Educate society and patients about healthy sleep behaviors and sleep hygiene.
- Monitor and, if indicated, manage sleep quality and quantity in patients to enhance physical therapy outcomes.
- Consider positioning to promote sleep quality.

Benefits of regular exercise in sleep disorders:[38]

- Rise in body temperature followed by compensatory drop after few hours makes it easier to fall asleep
- Acts like physical stressor to the body and Brain compensates by increasing deep sleep
- Increase flexibility and stretching of muscles leads to release the endorphins.
- Improves lung capacity which reduces the snoring thereby having sound sleep.
- Increased amount of Oxygen reaching to the blood improves the sleep quality.
- Balanced and toned skeletal muscles as well as respiratory muscles reduces Sleep apnea and Restless leg syndrome.
- Activates endocrine system and helps to maintain circadian rhythm and reduces hormonal imbalance.

Aerobic Exercises: Study has shown that graded exercises significantly reduced the severity of insomnia and fatigue in individuals diagnosed of inorganic insomnia. This includes 15 mins of warm-up and cool-down phases consisted of breathing exercise, stretching exercise of the neck, shoulder, upper limbs, the trunk and lower limbs musculature. The main exercise consisted of six minute walk, 5 mins stair climbing, mild jogging at a spot which lasted for duration of 4 minutes and ball throwing exercises at 30 meters away from the participant. Instructions on throwing and catching and holding of football were given.[39] The exercises were performed three times a week starting at 70% of the expected exercise heart rate of the participants and was progressed at 30% at every two weeks. Results of a meta-analysis suggest that physical therapy exercises could be a useful strategy for managing sleep disorders in neurorehabilitation.[40]

Resistance Exercise has been demonstrated to improve neuroplasticity thereby improving synaptic functioning of brain areas related to anxiety.[41] Resistive exercise is recommended for post menopausal women with sleep disturbance to improve estradiol level, insomnia, and sleep quality. It consisted of closed-chain exercises such as modified push-ups, forward, backward, and sideways lunges, calf lifts, bridging, marching, and chair sit-ups.[42] Progressive resistance exercise is an alternative modality that has also been shown to improve sleep quality and co morbidities associated with poor sleep like depression and cardiovascular disease.[43]

Respiratory Muscle Training: It aims to strengthen the pharyngeal, intercostals and diaphragmatic muscles, which can reduce the collapsibility

of the upper airway during sleep. Since OSA involves the collapse of the upper airways with inspiration during sleep, hence the practice of strengthening respiratory muscles through inspiratory muscle training (IMT) may reduce the number and/or severity of apneas by improving upper airway muscle tone.[44] Studies demonstrated that RMT have been suggested to lower the apnoea-hypopnea index (AHI) of the OSA patient population and improvement in functional capacity.[45] A systematic review and meta-analysis from 2020 showed that respiratory muscle training may be an adjunct therapy for the treatment of OSA.[46]

Positional Therapy: Sleeping in the lateral decubitus (right and left) and/or with elevation of the head of the bed by in 30º is recommended for subjects having difficulty in supine lying. Positional therapy is considered a simple and inexpensive technique and can be used alone or as an adjunct treatment with other methods.[47] Epidemiological studies pointed that this position has influence on the lymphatic system, as the action of gravity can interfere with cerebral blood flow and affect the elimination of waste products from the brain hence offers significant protection against cervical, scapular and arm pain and generates better sleep quality.[48]

Positive Airway Pressure (PAP) Therapy: PAP therapy may also be indicated for patients with obesity hypoventilation syndrome (OHS).[49] In obese individual's adipokines mediates impairments in respiratory mechanics and central respiratory drive leading to a shift in the equilibrium between CO_2 production and excretion. Studies suggests that PAP therapy prevents repetitive interruptions in nocturnal breath-ing which contribute to sleep hypercapnia and compensatory bicarbonate retention.[50] Positive airway pressure (PAP) therapy prevents the collapse of the pharyngeal airway to improve hypoxemia, hypercapnia and sleep fragmentation.[51] PAP therapy recommended as a treatment for children if the patient is ineligible for adenotonsillectomy surgery, or if OSA persists after surgery.

Transcranial Magnetic Stimulation (TMS): Repetitive Transcranial Magnetic Stimulation (rTMS) is a widely used non-invasive neuromodulatory technique. When applied in sleep medicine, the main hypothesis explaining its effects concerns the modulation of synaptic plasticity and the strength of connections between the brain areas involved in sleep disorders.[52] Findings of a RCT and meta analysis indicate that rTMS may improve sleep quality through increasing slow wave and rapid eye movement (REM) sleep thus a safe and effective option for insomnia.[53]

Life Style Modification: Use of sedatives medication, smoking and alcoholic beverages increase the awakening threshold and promote longer obstructive events. The patient should be advised to stop the consumption of these substances as it can cause edema, which increases airflow resistance, reduces airway caliber, and promotes dysfunction of the upper respiratory tract.[54] Regular physical exercise along with an active lifestyle improves overall health and prevents sleep related illnesses however exercise around bed time should be avoided due to the alertness that it produces.[55] Night time physical activities might inhibit the secretion of melatonin and cause an elevation in body temperature, both of which can interfere with the sleep mechanism and consequently lead to sleep onset delay.[56] Sleep hygiene guidelines also caution against engaging in high-intensity physical activities in the evening and suggests that it should be done 2 hours prior to sleep. Studies suggested that engaging in moderate-intensity exercise 2–4 hours before sleep may positively change sleep-related variables such as sleep delay and slow-wave sleep.[57]

Conclusion

Sleep disorders have a multifactorial origin and occur concomitantly with other clinical conditions, compromising patient adherence to appropriate treatment. Multidisciplinary approach is needed. Physical therapy professionals can play a significant role in this team, working both in the prevention and treatment of different sleep disorders. Exercising can be the most cost-effective health intervention and has been shown to be as effective as pharmacotherapy in improving sleep quality and duration.

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Newer Development

Popcorn Brain: The Digital Age's Emerging Psychosocial Challenge

Tushar Jagawat, Ritu Meena, Pankaj Tandon, Neelu Yadav, Preetkamal, Savita Jagawat, Yashika Gupta, Shubham Thadhani, Kashish Garg
Department of Psychiatry & Clinical Psychology, NIMSR, Jaipur, Rajasthan
Contact: Tushar Jagawat, Email: tusharjagawat@yahoo.com

Introduction

According to Dr. Daniel Glazer, a clinical psychologist, a Popcorn Brain refers to how focus and attention tend to quickly jump from one thing to another, akin to how corn kernels pop.

"Popcorn Brain," a term introduced by David Levy, a researcher at the University of Washington in 2011, describes a mental state characterized by scattered thoughts, fragmented attention, and a propensity for the mind to swiftly transition from one topic to another, akin to the rapid popping of popcorn kernels in a heated pot.[1] This is not considered a disorder or disease. Clinical neuropsychologist Jessica McCarthy describes the phenomenon as a mental state resulting from excessive screen time and overstimulation from the internet. It can also be considered as "Restless Hyperactive Mind".[2] Most of the information and observation available is in newspapers.[3-5] To the best our knowledge there is no research study on this emerging issue in Indian Literature.

Etiology

"Popcorn brain" or "Popcorn mind" is primarily caused by overuse of electronic social media and excessive multitasking. Constant engagement with social media leads to cognitive overload and fragmented attention. Additionally, multitasking strains cognitive resources, making it hard to focus on a single task. Together, these factors contribute to the development of "popcorn mind," impacting mental well-being and cognitive function.

Signs and Symptoms[1-5]

People experiencing "popcorn brain" or "popcorn mind" often struggle to focus on tasks or sustain a coherent train of thought, depicting instances of mental overload or cognitive disarray. This phenomenon predominantly affects youth and young adolescents.

The condition is primarily marked by diminished focus, heightened stress, and fatigue, making it difficult for individuals to maintain their usual levels of concentration and energy. Information overload exacerbates these issues, leading to poor attention and increased anxiety. It can also lead to insomnia, confusion and irritability. These symptoms collectively have an overall detrimental impact on relationships and quality of life, as individuals struggle to manage daily responsibilities and maintain social connections effectively.

Impact on Cognitive Function[1-3]

"Popcorn Brain" underscores the negative impact of excessive digital media consumption on cognitive function and emphasizes the importance of finding a balance in screen time to maintain mental well-being.

Management[1-5]

- Digital Fasting/Digital Dieting/Digital Detox helps to reduce stress, improve mental clarity, and boost productivity. It encourages reflection, creativity, and healthier engagement in non-digital activities. It is a beneficial practice to maintain a balanced and healthy lifestyle and foster good interpersonal relationships.
- Make it a habit to avoid your phone at least one hour before bedtime and one hour after waking up.
- Keep your phone away from your bed to

reduce the urge to check it frequently.

- Engage in Hobbies. Find hobbies that helps channelize your mind and make the most of your free time, whether it's reading, drawing, or playing a musical instrument along with outdoor activities.
- Take Breaks from Mindless Scrolling and watching excessive reels.
- Use screen time apps to set limits on your usage, helping to prevent overuse and mindless scrolling.
- Spend Time in Nature and with Family and friends along with family meals.
- Watching television is better than smartphones/laptops/tablets.
- Incorporate Physical Activities. Replace phone scrolling with physical activities that keep you active and mentally refreshed, such as walking, swimming, cycling, meditation, mindfulness, jogging, or yoga.
- If not relieved by above measures than contact mental health professionals like psychiatrist and clinical psychologist.

By adopting these strategies, you can effectively manage the impact of "popcorn brain" or "popcorn mind" and create a healthier balance with digital media.

Conclusion

To the best of our knowledge, there is limited literature on "popcorn brain," an emerging psychosocial issue characterized by scattered thoughts, excessive multitasking and cognitive overload due to excessive screen time. The rise of digital technology necessitates further studies to understand its causes, long-term effects, and effective interventions. Addressing this issue is crucial for promoting better mental health and well-being in our increasingly connected world.

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Newer Development

Ketogenic Diet and Mental Health

Bushra Zahoor, M.S. Bhatia, Dimple Gupta, Sandeep Sekhon, Nimmi A. Jose
Department of Psychiatry, HIMSR & HAHC Hospital, New Delhi-110070
Contact: Bushra Zahoor, E-mail: Bushra.zhr07@gmail.com

Introduction

Worldwide, an estimated 85 million people suffer from severe, persistent bipolar mood and psychotic illnesses,[1] and at least 280 million[2] are affected by depressive illness. Despite access to modern professional care, many do not experience significant improvement, with remission being rare. Nearly half of those receiving treatment for bipolar disorder continue to have recurrent mood episodes.[3] In Europe, approximately 19% of individuals with depression are considered "treatment-resistant,"[4] while globally, only 23% of individuals with schizophrenia respond well to antipsychotic medications,[5] often with trade-offs in quality and length of life. Metabolic issues such as hyperglycemia, hypertriglyceridemia, and weight gain are common in both bipolar disorder[6] and schizophrenia,[7] increasing the risk of obesity, type 2 diabetes, cardiovascular disease, and other chronic health conditions.

These challenges with psychopharmacological treatments underscore the critical need for new approaches to mental illness. One emerging intervention of interest is the ketogenic diet (KD), which restricts carbohydrates and promotes the production of ketone bodies through lipolysis. Ketone bodies serve as an alternative energy source for the brain, reducing its reliance on glucose.[8]

While research on KDs for psychiatric illnesses is still in its early stages, their use in neurological conditions like epilepsy has been well-established for over a century.[9] The robust evidence supporting KDs in neurological disorders suggests potential benefits for psychiatric conditions as well.[10] Epilepsy and bipolar disorder, for example, share common neurochemical pathways, supported clinically by the overlap in medications used to manage seizures and stabilize mood.[11]

The distinction between brain illnesses labeled as neurological and those deemed psychiatric is likely more rhetorical than biological. Both types of diseases originate in the same organ and share numerous biochemical similarities, such as neurotransmitter system dysregulation, neural network destabilization, neuroinflammation, excessive oxidative stress, impaired neuroplasticity, mitochondrial dysfunction, and disrupted cerebral glucose metabolism.[12-15]

The ketogenic diet (KD, also known as metabolic therapy) has demonstrated effectiveness in treating obesity, type 2 diabetes, and epilepsy. Recently, there has been growing interest in its potential therapeutic role in psychiatric disorders. A pilot study was conducted over four months to investigate the impact of a KD on individuals with schizophrenia or bipolar disorder who also had existing metabolic abnormalities. Twenty-three participants were enrolled in a single-arm trial. The results demonstrated improvements in metabolic health, with none of the participants meeting criteria for metabolic syndrome by the end of the study. Adherent individuals experienced significant reductions in weight (12%), BMI (12%), waist circumference (13%), and visceral adipose tissue (36%). Biomarker improvements observed included a 27% decrease in HOMA-IR and a 25% reduction in triglyceride levels. Interms of psychiatric measurements, participants with schizophrenia showed a 32% decrease in Brief Psychiatric Rating Scale-scores. Overall Clinical Global Impression (CGI) severity improved by an average of 31%, with 79% of participants starting with elevated symptoms showing atleast a1-point improvement on CGI.

Psychiatric outcomes across the cohort included increased life satisfaction (17%) and enhanced sleep quality (19%). This pilot trial highlighted the potential benefits of adjunctive ketogenic dietary treatment for individuals dealing with severe mental illness.[16]

Despite promising case reports of individuals with major depressive, bipolar, and psychotic illnesses benefiting from KDs, rigorous clinical trial evidence is lacking.

The biological plausibility that KDs may be of therapeutic benefit in various psychiatric disorders is strongly supported by the scientific literature as described below.

Depression

Inflammation is involved in all three conditions, with the most extensive research conducted on its role in depression. In many cases of clinical depression, inflammation contributes to its development and progression and is linked to poor responses to antidepressant medications.[17,18] The ketogenic diet (KD) has been demonstrated to reduce inflammation by affecting both central and peripheral immune regulatory pathways.[19-22] Additionally, the KD impacts several neurotransmitter systems related to depression, including the dopaminergic, serotonergic, glutamatergic, and GABAergic systems.[23]

Cox and colleagues reported that a 65-year-old female with type 2 diabetes and major depressive disorder showed significant improvement in depression and quality of life after 12 weeks on a KD, with substantial reductions in her medication and blood glucose levels.[24]

Bipolar Disorder and Schizophrenia

Individuals with bipolar disorder have a higher incidence of impaired glucose metabolism, even if they have not been treated with medication.[25] Research by Calkin[26] indicates that those with insulin resistance or type 2 diabetes are more prone to rapid mood cycling, less likely to respond to lithium, and tend to have a more severe disease progression. The dysregulation of glucose and insulin may affect mood by causing oxidative stress, which can impair mitochondrial function. Recent findings by Napolitano et al[27] suggest that the KD can increase brain levels of glutathione, a key antioxidant in combating oxidative stress. Campbell and Campbell[28] propose that the KD may alleviate bipolar symptoms by shifting the brain's primary fuel source from glucose to ketone bodies, thus bypassing mitochondrial defects and preventing further mitochondrial damage. This study found that 85% of participants with bipolar disorder reported positive mood effects from the KD, including mood stabilization and increased energy. Calkin et al[29] have suggested that insulin resistance can damage endothelial cells, compromising the blood-brain barrier (BBB) integrity in bipolar patients. This disruption of the BBB is also observed in major depression and schizophrenia.[30]

Hyperinsulinemia, insulin resistance, and impaired glucose metabolism are more prevalent in individuals experiencing first-episode psychosis who have not yet received treatment than in the general population[31]. Although this associational one does not establish a causal link between metabolic dysregulation and psychotic symptoms, there have been cases of acute hyperglycemia linked to transient psychotic symptoms in patients with type 1 and type 2 diabetes.[32] KDs also help rebalance neurotransmitter systems, stabilize neural networks, enhance neuroplasticity, and bridge the energy gap resulting from cerebral glucose hypometabolism associated with major depression, bipolar disorder, and schizophrenia.[33,34]

Emerging evidence indicates that the ketogenic diet (KD) may have positive effects on bipolar disorder and schizophrenia. Clinical studies and case reports suggest significant symptom improvements in mood stabilization, anxiety reduction, and psychotic symptom remission among patients following KD interventions. These improvements were observed across various psychiatric conditions, including bipolardisorder, major depressive disorder, and schizophrenia. For example, a cohort study reported symptom improvements and reduced medication dosages, while another study demonstrated the feasibility of the KD intervention in bipolar patients, including economic considerations.[35]

Mechanisms through which the KD may exert its effects include increased ATP availability, reduced oxidative stress, decreased inflammation, modulation of neurotransmitters (e.g., glutamate and GABA), improved mitochondrial function, and neuroprotective impacts. Animal and human studies have shown increased mitochondrial biogenesis and energy production, enhanced GABA biosynthesis, and modulation of ion concentrations, mimicking the

effects of mood stabilizers. Additionally, ketones have been shown to reduce neuroinflammation and oxidative stress, and β-hydroxybutyrate, a ketone body, has neuroprotective effects and antiinflammatory properties.[35]

The pathophysiology of bipolar disorder and schizophrenia involves impaired energy metabolism and oxidative stress, and the KD appears to address these aspects. However, more clinical trials are necessary to establish the KD's efficacy, identify the most responsive patient populations, and determine the optimal level of ketosis. Comprehensive studies, including randomized controlled trials and preclinical research, are needed to understand the metabolic mechanisms underlying the KD's antipsychotic effects.

Phelps et al. described two bipolar patients who significantly improved and discontinued their mood stabilizers after maintaining ketosis for 2 to 3 years.[36]

Kraft and Westman described a 70-year-old female with schizophrenia who experienced reduced hallucinations and paranoia, weight loss, and increased energy after 12 months on a KD. Additional case reports were excluded due to insufficient details.[37]

Alzheimer Disease

Three studies explored the effects of the ketogenic diet (KD) on Alzheimer disease. Morrill and Gibas[38] documented improvements in a 71-year-old female with mild Alzheimer's, noting enhanced cognitive scores after using a low-carbohydrate, high-fat KD and cognitive training. Taylor and colleagues conducted a 3-month study on 15 Alzheimer's patients, finding significant cognitive improvements which regressed after a 1-month washout period.[39] Lastly, Ota and colleagues' randomized controlled trial in 20 patients showed significant cognitive improvements with a KD formula containing medium-chain triglycerides, with diarrhea being the only side effect reported.[40]

Anorexia Nervosa

Scolnick and colleagues reported on a 29-year-old female with a long history of anorexia nervosa who improved after 3 months on a KD. Ketamine IV infusions further enhanced her recovery, resulting in stabilized weight and the elimination of obsessive thoughts and behaviors related to anorexia, without any side effects from the KD.[41]

Autism Spectrum Disorder

Five studies examined the KD's effects on autism spectrum disorder (ASD). Leeetal[42] found significant improvements in ASD symptoms in children on a modified KD. Evangeliou et al. observed notable improvements in CARS scores in children on a high-fat, low-carbdiet.[43]

Herbert et al. reported a significant reduction in ASD symptoms and an increase in IQ in a child on a gluten-free, casein-free KD.[44] Żarnowska et al. documented behavioral and intellectual improvements in a child on a KD.[45] El-Rashidy et al. found that both KD and gluten-free, casein-free diets improved ASD symptoms, with the KD showing superior results.[46]

Narcolepsy

Husain and colleagues studied the effects of a low-carbohydrate KD on narcolepsy symptoms in nine participants, noting an 18% reduction in symptom severity and decreased day time sleepiness after 8 weeks, with only mild, self-limiting side effects reported.[47]

Practical Considerations

Ketogenic diet protocols range widely, from the traditional "classic" KD used initially for treating refractory pediatric epilepsy (90% fat, 6% protein, 4% carbohydrate), to modified versions allowing higher protein intake,[49] including the modified Atkins diet.[48] Certain health conditions are considered absolute contraindications for initiating KDs in adults, including acute pancreatitis, nephrolithiasis, renal and liver failure, congestive heart failure, anorexia nervosa, and concurrent use of SGLT2 inhibitors.

A well-formulated KD can swiftly improve blood glucose, insulin, and blood pressure levels, which are generally beneficial in patients, particularly those with metabolic syndrome. However, careful medication management is crucial. Medications lowering blood glucose (like insulin, sulfonylureas, and meglitinides) and blood pressure (such as diuretics and ACE inhibitors) may need adjustment or discontinuation from the outset of KD initiation to mitigate the risks of hypoglycemia, hypotension, and hypovolemia.[50,51]

Research on potential interactions between ketogenic diets and psychotropic medications is

limited, except for anticonvulsants, which have shown minor adjustments in levels among epilepsy patients on a KD. Monitoring blood levels of anticonvulsants and other psychiatric medications, such as lithium, is advisable due to potential clinical implications.[52,53]

Effective transition to a KD in any population hinges on comprehensive dietary education and support. For patients with serious mental illness, including depression and psychosis, access to prepared meals and snacks is essential to overcome logistical barriers in adopting and adhering to the KD or any new diet. An intensive outpatient program could offer structured support for patients transitioning to a KD.

Conclusion

This review summarizes the current evidence supporting the use of the KD in treating various psychiatric disorders. Overall, the findings are positive, with all included studies reporting some degree of benefit in participants who adhered to the diet, regardless of their specific condition. However, a limitation of these studies is the small number of participants, which hinders the ability to generalize the findings. Before implementing dietary changes, it is important to carefully weigh the benefits and risks of the KD for each patient. Given the limited evidence, there is a clear need for larger, well-controlled trials to better define the therapeutic benefits of integrating the KD into psychiatric treatment protocols. While not within the scope of this review, future research exploring medical food approaches may also be valuable in the treatment of psychiatric disorders. Clinical pharmacists and healthcare providers may consider the KD as a potential adjunctive treatment option for patients with Alzheimer disease, anorexia nervosa, autism, bipolar disorder, major depressive disorder (MDD), narcolepsy, and schizophrenia. The KD has the potential to reduce the need for pharmacotherapy and mitigate unwanted medication side effects.

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Drug Review

Viloxazine in the Treatment of Attention Deficit Hyperactive Disorder: A Review

Kaviya K,[1] Rachna Gupta,[2] MS Bhatia[3]
[1,2]Department of Pharmacology, University College of Medical Sciences & GTB Hospital, University of Delhi, New Delhi, India
[3]Department of Psychiatry, HIMSR, Hamdard Nagar, New Delhi-110062
Contact: Rachna Gupta, E-mail: drrachna1@rediffmail.com

Introduction

Attention deficit hyperactivity disorder (ADHD) in children is a common neurodevelopmental disorder marked by persistent inattention and/or hyperactivity and impulsivity that are more disruptive than expected for a child's age.[1]

Approximately 366.3 million adults worldwide equating to 6.8%, are found to be suffering from symptomatic ADHD.[2] Although ADHD is frequently identified in childhood, it is a persistent condition that can continue in as many as 90% of individuals into adolescence and beyond. It can significantly affect an individual's social, occupational and academic function.[3,4]

ADHD is a complex disorder with its pathophysiology linked to genetic, neurological, and environmental factors, though the primary mechanisms remain unknown. Extensive evidence suggests that dysregulated dopamine and norepinephrine neurotransmission play a key role in the pathophysiology of ADHD.[5,6]

The American Academy of Paediatrics' current treatment guidelines for children and adolescents with ADHD recommend a combination of psychosocial/behavioural therapy and pharmacotherapy. Pharmacological management primarily involves two categories of drugs: CNS stimulants (methylphenidate and amphetamine) as the first-line treatment and non-stimulants (guanfacine, clonidine, and atomoxetine) as second-line options.[7] While CNS stimulants are effective, they cause notable side effects such as mood changes, appetite suppression, growth inhibition, drug abuse and dependency.[8] Non-stimulant medications are limited by their delayed onset of action, modest improvement in core ADHD symptoms, and mild effects on associated cognitive impairments.[9]

Recent developments in pharmacotherapy have introduced viloxazine as a new treatment option for ADHD, providing a non-stimulant alternative to traditional medications. Viloxazine Extended Release (ER) has been approved for children aged 6 years and older in 2021 and for adults in 2022 by the FDA for the treatment of ADHD.[10]

Viloxazine

The immediate-release formulation of Viloxazine was approved in 1974. It was marketed for over 25 years in the United Kingdom and various European Union nations for treating major depressive disorder in adults.[11] Studies have suggested that Viloxazine not only significantly improves depressive symptoms but may also alleviate symptoms of comorbid central nervous system conditions such as anxiety,[12] alcoholism,[13] and epilepsy.[14] However, due to its short elimination half-life of approximately 2.5 hours, the immediate-release formulation required multiple daily doses so it was discontinued in the early 2000s for business reasons unrelated to its efficacy or safety.[11] Recently Viloxazine, particularly in its ER formulation, has demonstrated efficacy in reducing symptoms of Attention-deficit/hyperactivity disorder (ADHD) in children and adolescents. Studies have shown that viloxazine effectively reduces ADHD symptoms such as in attention, hyperactivity/impulsivity, peer relation impairments, and challenges with school learning.[15] Additionally, viloxazine has been found to significantly reduce executive function deficits in individuals with ADHD, indicating its effectiveness in managing cognitive aspects of the disorder.[16]

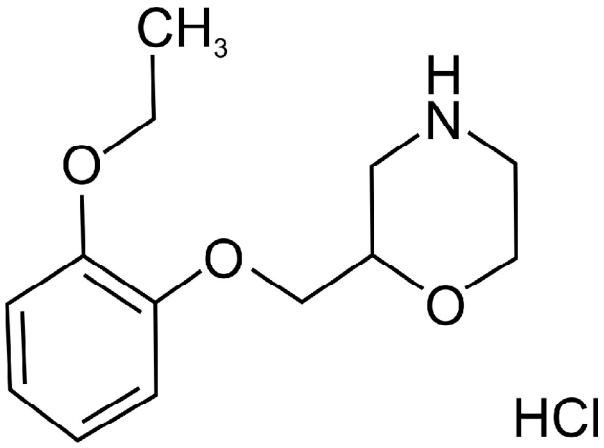

Fig. 1: The chemical structure of viloxazine

Mechanism of Action

Viloxazine is a serotonin-norepinephrine modulating agent (SNMA) with a unique mechanism of action compared to other ADHD and depression medications. It modulates serotonergic activity by acting as a selective 5-HT2B receptor antagonist and a 5-HT2C receptor agonist.[17] Additionally, it binds to norepinephrine transporter (NET), thereby blocking the reuptake of norepinephrine.[10]

Pharmacokinetics

Viloxazine's Cmax and AUC increase proportionally with doses ranging from 100 mg to 600 mg once daily, reaching steady-state within two days without accumulation.[10]

Absorption: The extended-release form of viloxazine has a relative bioavailability of about 88% compared to the immediate-release form. After a single 200 mg dose, the median time to peak plasma concentration (Tmax) is around 5 hours (range: 3 - 9 hours). When viloxazine is taken with a high-fat meal (800 to 1000 calories), its Cmax and AUC decrease by approximately 9% and 8%, respectively, and Tmax increases by about 2 hours. Sprinkling the capsule contents on apple sauce results in a reduction in Cmax and AUC by about 10% and 5%, respectively.[10]

Distribution: Viloxazine binds to human plasma proteins at a rate of 76-82% within a concentration range of 0.5 mcg/mL to 10 mcg/mL. Its mean half-life is 7.02 ± 4.74 hours[10].

Metabolism: Viloxazine is primarily metabolized by CYP2D6, UGT1A9, and UGT2B15, with 5-hydroxy-viloxazine glucuronide as the major plasma metabolite.[10]

Excretion: Viloxazine is mainly excreted through the kidneys. After administering radiolabelled viloxazine, 90% of the dose was recovered in the urine within the first 24 hours, while less than 1% was excreted in the faeces.[10]

Adverse Effects

Viloxazine is generally well-tolerated, some common adverse effects associated with its use include decreased appetite, mild sleepiness and headache. However, these side effects are typically mild and transient.[16,18]

Contraindications

Viloxazine should not be used in patients who are:

- Currently taking monoamine oxidase inhibitors (MAOIs), or within 14 days of stopping an MAOI, due to the increased risk of hypertensive crisis.[10]
- Taking drugs that are sensitive CYP1A2 substrates or have a narrow therapeutic range.[10]

Warnings and Precautions

- Rise of Blood pressure and Heart rate: Hence, heart rate and blood pressure should be assessed before starting treatment, after increasing the dosage, and periodically throughout the treatment.[10]
- Risk of Mania or Hypomania: Therefore, patients should be screened for bipolar disorder.[10]
- Somnolence and Fatigue: Therefore, patients should be advised to be cautious while driving or operating any hazardous machinery.[10]

Clinical Trials with Viloxazine in the Treatment of ADHD

A phase II clinical trial by Jhonson et al. 2019, investigated the effectiveness of viloxazine ER in treating ADHD in children aged 6-12 years old. This double-blind, placebo-controlled study assessed four fixed doses of viloxazine ER (100 mg, 200 mg, 300 mg, and 400 mg daily) administered over eight weeks. The primary outcome measure was the

change in ADHD symptoms as measured by the ADHD Rating Scale-IV (ADHD-RS-IV) compared to baseline scores. The study found that the average ADHD-RS-IV total score and subscale scores decreased from baseline at all time points for all treatment groups. All viloxazine ER doses except 100 mg/day showed statistically significant improvements in the primary outcome (ADHD-RS-IV score) and severity ratings (CGI-S score) compared to placebo.[18]

A study by Nasser et al. 2020, investigated the effectiveness of viloxazine ER in treating ADHD symptoms in school children. It was a phase III, randomized, double-blind, placebo-controlled trial lasting 6 weeks. The primary focus was on the change in ADHD-Rating Scale-5 (ADHD-RS-5) scores from baseline to the end of the study. A total of 477 children with similar characteristics were randomly assigned to receive viloxazine ER at 100 mg or 200 mg daily, or a placebo. The results showed significant improvement in ADHD-RS-5 scores for both viloxazine ER doses compared to placebo (p-values indicate high statistical significance). Additionally, improvements were observed in other measures: CGI-I score, Conners 3-PS Composite T-scores and WFIRS-P Total average scores. These findings suggested that viloxazine ER effectively improved ADHD symptoms in the study participants. Treatment side effects were mild and included headache, drowsiness, and decreased appetite.[19]

A Phase III study by Nasser et al. 2021, investigated the effectiveness and safety of SPN-812 (viloxazine extended-release) in treating ADHD symptoms in children aged 6-11 years. A total of 313 children were randomly assigned to receive a daily dose of either 200 mg or 400 mg SPN-812, or a placebo for 8 weeks. The results showed that both SPN-812 doses significantly improved ADHD symptoms and overall clinical impression compared to placebo. Additionally, the 200 mg dose improved parent-reported child behaviour, but the effect wasn't statistically significant for the 400 mg dose. No significant improvement in parent-reported functional impairment was observed in either group. Importantly, side effects were minimal, and less than 5% of children discontinued treatment due to side effects.[20]

In addition, another phase III trial by Nasser et al. 2021, assessed viloxazine extended-release (VLX-ER) for treating ADHD in adolescents (12-17 years old). A total of 310 participants received either 200 mg or 400 mg of VLX-ER daily, or a placebo for 6 weeks. The primary outcome was the change in ADHD symptoms (measured by ADHD Rating Scale-5) from baseline to the end of the study. Additional assessments included a doctor's evaluation of improvement and parent-reported changes in behaviour and functional impairment. Results showed:

- Both VLX-ER doses significantly improved ADHD symptoms compared to placebo, including inattention and hyperactivity/impulsivity.
- Greater improvement in adolescents taking VLX-ER compared to placebo.
- Parent-reported improvements in behaviour and function trended positive but weren't statistically significant compared to placebo.
- The most common side effects were drowsiness, headache, decreased appetite, nausea, and fatigue.
- Less than 5% of participants in each group stopped taking the medication due to side effects.

Overall VLX-ER treatment resulted in improvement in ADHD symptoms for adolescents and was well-tolerated.[16]

Further the team Nasser et al, investigated the effectiveness and safety of SPN-812 (viloxazine extended-release) for treating ADHD in adolescents. A total of 297 adolescents were randomly assigned to receive a daily dose of either 400 mg or 600 mg SPN-812, or a placebo for a set period. The main measure of success was the change in ADHD symptoms (measured by ADHD Rating Scale-5). While the 400 mg dose significantly improved ADHD symptoms compared to placebo, the 600 mg dose did not reach statistical significance, possibly due to an unexpected high response in the placebo group. Both doses however were well-tolerated with less than 5% of participants discontinuing treatment due to side effects.[21]

Nasser et al also examined viloxazine extended-release (viloxazine ER) for adults with ADHD. A total of 374 adults were randomly assigned to receive either a daily dose of viloxazine ER (adjustable between 200 mg and 600 mg) or a placebo for 6

Table-1 has summarized all the clinical trials mentioned above

Author	Study Design	Dose of viloxazine & Duration	Sample size (n)	Age Group	Primary Outcome	Secondary Outcome	Results
Johnson et al[18]	Phase II, double-blind, placebo-controlled	100 mg 200 mg 300 mg 400 mg OD dose *(8 weeks)	222	6-12 years	ADHD-RS-IV score	CGI-S, CGI-I, Safety assessments	Significant improvement in ADHD-RS-IV scores was seen at 200, 300, and 400 mg doses. CGI-S scores ($p < .05$) at all dose except 100 mg. significantly reduced ADHD symptoms and was well tolerated
Nasser et al[19]	Phase III, randomized, placebo-controlled, double-blind.	100 mg/day 200 mg/day *(6 weeks)	477	6-11 years	Change from baseline in ADHD-RS-5	CGI-I, Conners 3-PS Composite T-score, WFIRS-P Total average score	Significant improvement in ADHD-RS-5 CGI-I scale (P = 0.0020 for 100 mg/day, P < 0.0001 for 200 mg/day), Conners 3-PS Composite T-score (P = 0.0003 for 100 mg/day, P = 0.0002 for 200 mg/day), and WFIRS-P Total average score (P = 0.0019 for 100 mg/day, P = 0.0002 for 200 mg/day) compared to placebo.
Nasser et al[20]	Phase III, randomized, double-blind, placebo-controlled	200 mg/day, 400 mg/day *(8 weeks)	313	6-11 years	Change from baseline in ADHD-RS-5	CGI-I, Conners 3-PS Composite T-score, WFIRS-P Total average	Significant improvement in ADHD-RS-5 Total score at EOS for both 200 mg (P = 0.0038) and 400 mg (P = 0.0063) , CGI-I score for both 200 mg (P = 0.0028) and 400 mg (P = 0.0099), Conners 3-PS Composite T-score for 200 mg (P = 0.0064), but not 400 mg (P = 0.0917) vs. placebo. No significant difference in the WFIRS-P Total average score. Discontinuation rate due to AEs was <5%
Nasser et al.[21]	Phase III, randomized, double-blind, placebo-controlled	200 mg/day, 400 mg/day *(6 weeks)	310	12-17 years	Change from baseline in ADHD-RS	CGI-I, Conners 3-PS Composite T-score, WFIRS-P Total average score	Significant improvement in ADHD-RS-5 Total score at EOS for 200 mg (P = 0.0232) and 400 mg (P = 0.0091), significant improvement in Inattention (P = 0.0424 for 200 mg, P = 0.0390 for 400 mg) and Hyperactivity/ Impulsivity (P = 0.0069 for 200 mg, P = 0.0005 for 400 mg) subscale scores. Significant improvement in CGI-I score. No significant difference in Conners 3-PS Composite T-score and WFIRS-P Total average score. Common AEs included somnolence, headache, decreased appetite, nausea, and fatigue. Discontinuation rate due to AEs was <5%.

Author	Study Design	Dose of viloxazine & Duration	Sample size (n)	Age Group	Primary Outcome	Secondary Outcome	Results
Nasser et al[21]	Phase III, randomized, placebo-controlled, double-blind	400 mg/day, 600 mg/day *(6 weeks)	297	Adolescents (12-17 years)	Change from baseline in ADHD-RS-5	CGI-I, Conners 3-PS Composite T-score, WFIRS-P Total average score	Significant improvement in ADHD-RS-5 Total score at EOS for 400 mg (P = 0.0082) ; no significant difference for 600 mg (P = 0.0712). Significant improvements were observed in several secondary endpoints. Discontinuation rate due to AEs was <5%.
Nasser et al.[22]	Phase III, randomized, double-blind, placebo-controlled	Flexible dose day of 200–600 mg/ *(6 weeks)	374	18-65 years	Change from baseline in AISRS total score	Change from baseline in CGI-S, AISRS subscales, BRIEF-A, GAD-7, CGI-I	Significant reduction in AISRS total score for viloxazine (P = 0.0040). Significant reduction in CGI-S score (P = 0.0023. Significant improvements in AISRS Inattention (P = 0.0015) and Hyperactivity/Impulsivity (P = 0.0380) subscales, CGI-I (P = 0.0076), BRIEF-A Global Executive Composite (P = 0.0468) and Metacognition Index (P = 0.0100). AISRS 30% response rate is significantly higher for viloxazine ER (P = 0.0395). The discontinuation rate due to AEs was 9.0% for viloxazine ER vs. 4.9% for placebo.

weeks. The main outcome was improvement in ADHD symptoms as measured by a specific rating scale. Viloxazine ER significantly improved ADHD symptoms, including both inattention and hyperactivity/impulsivity, compared to placebo. Additionally, viloxazine ER led to better scores on measures of overall illness severity, executive function, and global functioning. Importantly, these improvements were observed as early as week 2. Viloxazine ER was well-tolerated with side effects like insomnia, fatigue, and nausea being the most common. Overall, this study suggests that viloxazine ER is an effective and well-tolerated treatment for ADHD in adults.[22]

Conclusion

Viloxazine represents a promising non-stimulant treatment for ADHD in both paediatric and adult populations, offering significant symptom relief with a favourable safety profile. Its unique mechanism of action, favourable pharmacokinetics, and demonstrated efficacy in clinical trials position it as an invaluable addition to the ADHD treatment armamentarium. As research continues to evolve, viloxazine may play an increasingly prominent role in the therapeutic landscape of ADHD, providing clinicians with a valuable tool for addressing this complex disorder. Continued research into viloxazine's long-term effects, optimal dosing strategies, and its impact on various ADHD subtypes is warranted.

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Forensic Psychiatry

The Rights of Persons with Disabilities Act, 2016

(Act No. 49 of 2016; 27th December)

An Act to give effect to the United Nations Convention on the Rights of Persons with Disabilities and for matters connected there with or incidental there to.

WHEREAS the United Nations General Assembly adopted its Convention on the Rights of Persons with Disabilities on the 13th day of December, 2006.

AND WHEREAS the aforesaid Convention laysdown the following principles for empowerment of persons with disabilities,—

(a) respect for inherent dignity, individual autonomy including the freedom to make one's own choices, and independence of persons;
(b) non-discrimination;
(c) full and effective participation and inclusion in society;
(d) respect for difference and acceptance of persons with disabilities as part of human diversity and humanity;
(e) equality of opportunity;
(f) accessibility;
(g) equality between men and women;
(h) respect for the evolving capacities of children with disabilities and respect for the right of children with disabilities to preserve their identities;

AND WHEREAS India is a signatory to the said Convention;

AND WHEREAS India ratified the said Convention on the 1st day of October, 2007;

AND WHEREAS it is considered necessary to implement the Convention aforesaid.

BE it enacted by Parliament in the Sixty-seventh Year of the Republic of India as follows:—

CHAPTER-I
PRELIMINARY

1. Short title and commencement —

(1) This Act may be called the Rights of Persons with Disabilities Act, 2016.

(*2*) It s hall come intof orce on such[1] date as the Central Government may, by notification in the Official Gazette, appoint.

2. Definitions —

In this Act, unless the context otherwise requires,—

(a) "appellate authority" means an authority notified under sub-section (*3*) of section 14 or sub-section (1) of section 53 or designated under sub-section (1) of section 59, as the case may be;
(b) "appropriateGovernment"means,—
  (i) in relation to the Central Government or any establishment wholly or sub-stantially financed by that Government, or a Cantonment Board constituted under the Cantonments Act, 2006 (41 of 2006), the Central Government;
  (ii) in relation to a State Government or any establishment, wholly or substantially financed by that Government, or any local authority, other than a Cantonment Board, the State Government.
(c) "barrier" means any factor including communicational, cultural, economic, environmental, institutional, political, social, attitudinal or structural factors which hampers the full and effective participation of persons with disabilities in society;
(d) "care-giver" means any person including parents and other family Members who with or without payment provides care, support or assistance to a person with disability;
(e) "certifying authority" means an authority designated under sub-section (1) of section 57;
(f) "communication" includes means and formats of communication, languages, display of text, Braille, tactile communication, signs, large print, accessible multimedia, written, audio, video, visual displays, sign language, plain-language, human-reader, augmentative and alternative modes and accessible information and communication technology;
(g) "competent authority" means an authority appointed under section 49;
(h) "discrimination" in relation to disability, means any distinction, exclusion, restriction on the basis of disability which is the purpose or effect of impairing or nullifying the recognition, enjoy mentor exercise on an equal basis with others of all human rights and fundamental freedoms in the political, economic, social, cultural, civil or any other field and includes all forms of discrimination and denial of reasonable accommodation;
(i) "establishment" includes a Government establishment and private establishment;
(j) "Fund"means the National Fund constituted under section 86;

(k) "Government establishment" means a corporation established by or under a Central Act or State Act or an authority or a body owned or controlled or aided by the Government or a localauthority or a Government company as defined in section 2 of the Companies Act, 2013 (18 of 2013) and includes a Department of the Government;

(l) "high support" means an intensive support, physical, psychological and otherwise, which may be required by a person with benchmark disability for daily activities, to take independent and informed decision to access facilities and participating in all area softlife including education, employment, family and community life and treatment and therapy;

(m) "inclusive education" means a system of education where in students with and without disability learn together and the system of teaching and learning is suitably adapted to meet the learning needs of different types of students with disabilities;

(n) "information and communication technology" includes all services and innovations relating to information and communication, including telecom services, web based services, electronic and print services, digital and virtual services;

(o) "institution" means an institution for the reception, care, protection, education, training, rehabilitation and any other activities for persons with disabilities;

(p) "local authority" means a Municipality or a Panchayat, as defined in clause (e) and clause (f) of article 243P of the Constitution; a Cantonment Board constituted under the Cantonments Act, 2006 (41 of 2006); and any other authority established under an Act of Parliament or a State Legislature to administer the civic affairs;

(q) "notification" means a notification published in the Official Gazette and the expression "notify" or "notified" shall be construed accordingly;

(r) "person with benchmark disability" means a person with not less than forty per cent of a specified disability where specified disability has not been defined in measurable terms and includes aperson with disability where specified disability has been defined in measurable terms, as certified by the certifying authority;

(s) "person with disability" means a person with long term physical, mental, intellectual or sensory impairment which, in interaction with barriers, hinders his full and effective participation in society equally with others;

(t) "person with disability having high support needs" means a person with benchmark disability certified under clause (a) of sub-section (2) of section 58 who needs high support;

(u) "private establishment" means a company, firm, cooperative or other society, associations, trust, agency, institution, organisation, union, factory or such other establishment as the appropriate Government may, by notification, specify;

(v) "public building" means a Government or private building, used or accessed by the public at large, including abuilding used for educational or vocational purposes, work place, commercial act ivities, public utilities, religious, cultural, leisure or recreational activities, medical or health services, law enforcement agencies, reformatories or judicial for as, railway stations or platforms, roadways bus stands or terminus, airports or waterways;

(w) "public facilities and services" includes all forms of delivery of services to the public at large, including housing, educational and vocational trainings, employment and career advancement, shopping or marketing, religious, cultural, leisure or recreational, medical, health and rehabilitation, banking, finance and insurance, communication, postal and information, access to justice, public utilities, transportation;

(x) "reasonable accommodation" means nece-ssary and appropriate modification and adjustments, without imposing a disproportionate or undue burden in a particular case, to ensure to persons with disabilities the enjoyment or exercise of rights equally with others;

(y) "registered organisation" means an association of persons with disabilities or a disabled person organisation, association of parents of persons with disabilities, association of persons with disabilities and family members, or a voluntary or non-governmental or charitable organisation or trust, society, or non-profit company working for the welfare of the persons with disabilities, duly registered under an Act of Parliament or a State Legislature;

(*za*) "rehabilitation" refers to a process aimed at enabling persons with disabilities to attain and maintain optimal, physical, sensory, intellectual, psychological environmental or social function levels;

(*zb*) "Special Employment Exchange" means any office or place established and maintained by the Government for the collection and furnishing of information, either by keeping of registers or otherwise, regarding —

(i) persons who seek to engage employees from amongst the persons with disabilities;

(ii) persons with benchmark disability who seek employment;

(iii) vacancies to which persons with benchmark disabilities seeking employment may be appointed;

(*zc*) "specified disability" means the disabilities as specified in the Schedule;

(*zd*) "transportation systems" includes road transport, rail transport, air transport, water transport, paratransit systems for the last mile connectivity, road and street infrastructure, etc;

(*ze*) "universal design" means the design of products, environments, programmes and services to be usable by all people to the greatest extent possible, without the need for adaptation or specialised design and shall apply to assistive devices including advanced technologies for particular group of persons with disabilities.

CHAPTER-II
RIGHTS AND ENTITLEMENTS

3. Equality and non-discrimination —

(1) The appropriate Government shall ensure that the persons with disabilities enjoy the right to equality, life with dignity and respect for his or her integrity equally with others.

(2) The appropriate Government shall take steps to utilise the capacity of persons with disabilities by providing appropriate environment.

(3) No person with disability shall be discriminated on the ground of disability, unless it is shown that the impugned act or omission is a proportionate means of achieving a legitimate aim.

(4) No person shall be deprived of his or her personal liberty only on the ground of disability.

(5) The appropriate Government shall take necessary steps to ensure reasonable accommodation for persons with disabilities.

4. Women and children with disabilities —

(1) The appropriate Government and the local authorities shall take measures to ensure that the women and children with disabilities enjoy their rights equally with others.

(2) The appropriate Government and local authorities shall ensure that all children with disabilities shall have right on an equal basis to freely express their views on all matters affecting them and provide the map propriate support keeping in view their age and disability."

5. Community life —

(1) The persons with disabilities shall have the right to live in the community.

(2) The appropriate Government shall endeavour that the persons with disabilities are,—

(a) not obliged to live in any particular living arrangement; and

(b) given access to arange of in-house, residential and other community support services, including personal assistance necessary to support living with due regard to age and gender.

6. Protection from cruelty and inhuman treatment —

(1) The appropriate Government shall take measures to protect persons with disabilities from being subjected to torture, cruel, inhuman or degrading treatment.

(2) No person with disability shall be a subject of any research without,—

(i) his or her free and informed consent obtained through accessible modes, means and formats of communication; and

(ii) prior permission of a Committee for Research on Disability constituted in the prescribed manner for the purpose by the appropriate Government in which not less than half of the Members shall themselves be either persons with disabilities or Members of the registered organisation as defined under clause(*z*) of section 2.

7. Protection from abuse, violence and exploitation —

(1) The appropriate Government shall take measures to protect persons with disabilities from all forms of abuse, violence and exploitation and to prevent the same, shall —

(a) take cognizance of incidents of abuse, violence and exploitation and provide legal remedies available against such incidents;

(b) take steps for avoiding such incidents and prescribe the procedure for its reporting;

(c) take steps to rescue, protect and rehabilitate victims of such incidents; and

(d) create awareness and make available information among the public.

(2) Any person or registered organisation who or which has reason to believe that an act of abuse, violence or exploitation has been, or is being, or is likely to be committed against any person with disability, may give information about it to the Executive Magistrate within the local limits of whose jurisdiction such incidents occur.

(3) The Executive Magistrate on receipt of such information, shall take immediate steps to stop orprevent its occurrence, as the case may be, or pass such order as he deems fit for the protection of such person with disability including an order—

(a) to rescue the victim of such act, authorising the police or any organisation working for persons with disabilities to provide for the safe custody or rehabilitation of such person, or both, as the case may be;

(b) for providing protective custody to the person with disability, if such person so desires;

(c) to provide maintenance to such person with disability.

(4) Any police officer who receives acomplaint or otherwise comes to know of abuse, violence or exploitation towards any person with disability shall inform the aggrieved person of —

(a) his or her right to apply for protection under sub-section (2) and the particulars of the

Executive Magistrate having jurisdiction to provide assistance;

(b) the particulars of the nearest organisation or institution working for the rehabilitation of persons with disabilities;

(c) the right to free legal aid; and

(d) the right to file acomplaint under the provisions of this Actor any other law dealing with such offence:

Provided that nothing in this section shall be construed in any manner as to relieve the police officer from his duty to proceed in accordance with law upon receipt of information as to the commission of acognizable offence.

(5) If the Executive Magistrate finds that the alleged act or behaviour constitutes an offence under the Indian Penal Code (45 of 1860), or under any other law for the time being in force, he may forward the complaint to that effect to the Judicial or Metropolitan Magistrate, as the case may be, having jurisdiction in the matter.

8. Protection and safety —

(1) The persons with disabilities shall have equal protection and safety insituations of risk, armed conflict, humanitarian emergencies and natural disasters.

(2) The National Disaster Management Authority and the State Disaster Management Authority shall take appropriate measures to ensure inclusion of persons with disabilities in its disaster management activities as defined under clause (e) of section 2 of the Disaster Management Act, 2005 (53 of 2005) for the safety and protection of persons with disabilities.

(3) The District Disaster Management Authority constituted under section 25 of the Disaster Management Act, 2005 (53 of 2005) shall maintain record of details of persons with disabilities in the district and take suitable measures to inform such persons of any situations of risk so as to enhance disaster preparedness.

(4) The authorities engaged in reconstruction activities subsequent to any situation of risk, armed conflict or natural disasters shall undertake such activities, in consultation with the concerned State Commissioner, in accordance with the accessibility requirements of persons with disabilities.

9. Home and family —

(1) No child with disability shall be separated from his or her parents on the ground of disability excepton an order of competent court, if required, in the best interest of the child.

(2) Where the parents are unable to take care of a child with disability, the competent court shall placesuch child with his or her near relations, and failing that within the community in a family setting or in exceptional cases in shelter home run by the appropriate Government or non-governmental organisation, as may be required.

10. Reproductive rights —

(1) The appropriate Government shall ensure that persons with disabilities have access to appropriate information regarding reproductive and family planning.

(2) No person with disability shall be subject to any medical procedure which leads to infertility without his or her free and informed consent.

11. Accessibility in voting —

The Election Commission of India and the State Election Commissions shall ensure that all polling stations are accessible to persons with disabilities and all materials related to the electoral process are easily understandable by and accessible to them.

12. Access to justice —

(1) The appropriate Government shall ensure that persons with disabilities are able to exercise the right to access any court, tribunal, authority, commission or any other body having judicial or quasi-judicial or investigative powers without discrimination on the basis of disability.

(2) The appropriate Government shall take steps to put in place suitable support measures for persons with disabilities specially those living outside family and those disabled requiring high support for exercising legal rights.

(3) The National Legal Services Authority and the State Legal Services Authorities constituted under the Legal Services Authorities Act, 1987 (39 of 1987) shall make provisions including reasonable accommodation to ensure that persons with disabilities have access to any scheme, programme, facility or service offered by them equally with others.

(4) The appropriate Government shall take steps to –

(a) ensure that all their public documents are in accessible formats;

(b) ensure that the filing departments, registry or any other office of records are supplied with necessary equipment to enable filing, storing and referring to the documents and evidence in accessible formats; and

(c) make available all necessary facilities and equipment to facilitate recording of testimonies, arguments or opinion given by persons with disabilities in their preferred language and means of communication.

13. Legal capacity —

(1) The appropriate Government shall ensure that the persons with disabilities have right, equally with others, to own or inherit property, movable or immovable, control their financial affairs and have access to bank loans, mortgages and other forms of financial credit.

(2) The appropriate Government shall ensure that the persons with disabilities enjoy legal capacity onan equal basis with others in all aspects of life and have the right to equal recognition every where as any other person before the law.

(3) When a conflict of interest arises between a person providing support and a person with disability in a particular financial, property or other economic transaction, then such supporting person shall abstain from providing support to the person with disability in that transaction:

Provided that there shall not be a presumption of conflict of interest just on the basis that the supporting person is related to the person with disability by blood, affinity or adoption.

(4) A person with disability may alter, modify or dismantle any support arrangement and seek the support of another:

Provided that such alteration, modification or dismantling shall be prospective in nature and shall notnullify any third party transaction entered into by the person with disability with the aforesaid support arrangement.

(5) Any person providing support to the person with disability shall not exercise undue influence and shall respect his or her autonomy, dignity and privacy.

14. Provision for guardianship —

(1) Notwithstanding anything contained in any other law for the time being in force, on and from the date of commencement of this Act, where a district court or any designated authority, as notified by the State Government, finds that a person with disability, who had been provided adequate and appropriate support but is unable to take legally binding decisions, may be provided further support of a limited guardian to take legally binding decisions on his behalf in consultation with such person, in such manner, as may be prescribed by the State Government:

Provided that the District Court or the designated authority, as the case may be, may grant total support to the person with disability requiring such support or where the limited guardianship is to be granted repeatedly, in which case, the decision regarding the support to be provided shall be reviewed by the Court or the designated authority, as the case may be, to determine the nature and manner of support to be provided.

Explanation — For the purposes of this sub-section, "limited guardianship" means a system of joint decision which operates on mutual understanding and trust between the guardian and the person with disability, which shall be limited to a specific period and for specific decision and situation and shall operate in accordance to the will of the person with disability.

(2) On and from the date of commencement of this Act, every guardian appointed under any provision of any other law for the time being in force, for a person with disability shall be deemed to function as a limited guardian.

(3) Any person with disability aggrieved by the decision of the designated authority appointing a legal guardian may prefer an appeal to such appellate authority, as may be notified by the State Government for the purpose.

15. Designation of authorities to support —

(1) The appropriate Government shall designate one or more authorities to mobilise the community and create social awareness to support persons with disabilities in exercise of their legal capacity.

(2) The authority designated under sub-section (1) shall take measures for setting up suitable support arrangements to exercise legal capacity by persons with disabilities living in institutions and those with high support needs and any other measures as may be required.

CHAPTER-III
EDUCATION

16. Duty of educational institutions —

The appropriate Government and the local authorities shall endeavour that all educational institutions funded or recognised by them provide inclusive education to the children with disabilities and towards that end shall

(i) admit them without discrimination and provide education and opportunities for sports and recreation activities equally with others;

(ii) make building, campus and various facilities accessible;

(iii) provide reasonable accommodation according to the individual's requirements;

(iv) provide necessary support individualised or otherwise in environments that maximise academic and social development consistent with the goal of full inclusion;

(v) ensure that the education to persons who are blind or deaf or both is imparted in the most appropriate languages and modes and means of communication;

(vi) detect specific learning disabilities in children at the earliest and take suitable pedagogical and other measures to overcome them;

(vii) monitor participation, progress interms of attainment levels and completion of education in respect of every student with disability;

(viii) provide transportation facilities to the children with disabilities and also the attendant of the children with disabilities having high support needs.

17. Specific measures to promote and facilitate inclusive education —

The appropriate Government and the local authorities shall take the following measures for the purpose

of section 16, namely:—

(a) to conduct survey of school going children in every five years for identifying children with disabilities, ascertaining their special needs and the extent to which these are being met:

Provided that the first survey shall be conducted within a period of two years from the date of commencement of this Act;

(b) to establish adequate number of teacher training institutions;

(c) to train and employ teachers, including teachers with disability who are qualified in sign language and Braille and also teachers who are trained in teaching children with intellectual disability;

(d) to train professionals and staff to support inclusive education at all levels of school education;

(e) to establish adequate number of resource centres to support educational institutions at all levels of school education;

(f) to promote the use of appropriate augmentative and alternative modes including means and formats of communication, Braille and sign language to supplement the use of one's own speech to fulfil the daily communication needs of persons with speech, communication or language disabilities and enables them to participate and contribute to their community and society;

(g) to provide books, other learning materials and appropriate assistive devices to students with benchmark disabilities free of cost upto the age of eighteen years;

(h) to provide scholarships in appropriate cases to students with benchmark disability;

(i) to make suitable modifications in the curriculum and examination system to meet the needs of students with disabilities such as extra time for completion of examination paper, facility of scribe or amanuensis, exemption from second and third language courses;

(j) to promote research to improve learning; and

(k) any other measures, as may be required.

18. Adult education —

The appropriate Government and the local authorities shall take measures to promote, protect and ensure participation of persons with disabilities in adult education and continuing education programmes equally with others.

CHAPTER-IV
SKILL DEVELOPMENT AND EMPLOYMENT

19. Vocational training and self-employment —

(1) The appropriate Government shall formulate schemes and programmes including provision of loans at concessional rates to facilitate and support employment of persons with disabilities especially for their voca-tional training and self-employment.

(2) The schemes and programmes referred to in sub-section (1) shall provide for —

(a) inclusion of person with disability in all main stream formal and non-formal vocational and skill training schemes and programmes;

(b) to ensure that a person with disability has adequates up port and facilities to avails pecific training;

(c) exclusive skill training programmes for persons with disabilities with active links with the market, for those with developmental, intellectual, multiple disabilities and autism;

(d) loans at concessional rates including that of microcredit;

(e) marketing the products made by persons with disabilities; and

(f) maintenance of disaggregated data on the progress made in the skill training and self-employment, including persons with disabilities.

20. Non-discrimination in employment —

(1) No Government establishment shall discriminate against any person with disability in any matter relating to employment:

Provided that the appropriate Government may, having regard to the type of work carried on in any establishment, by notification and subject to such conditions, if any, exempt any establishment from the provisions of this section.

(2) Every Government establishment shall provide reasonable accommodation and appropriate barrier free and conducive environment to employees with disability.

(3) No promotion shall be denied to a person merely on the ground of disability.

(4) No Government establishment shall dispense with or reduce in rank, an employee who acquires a disability during his or her service:

Provided that, if an employee after acquiring disability is not suitable for the post he was holding, shall be shifted to some other post with the same pay scale and service benefits:

Provided further that if it is not possible to adjust the employee against any post, he may be kept on a supernumerary post until a suitable post is available or he attains the age of superannuation, whichever is earlier.

(5) The appropriate Government may frame policies for posting and transfer of employees with disabilities.

21. Equal opportunity policy —

(1) Every establishment shall notify equal opportunity policy detailing measures proposed to be taken by it in pursuance of the provisions of this Chapter in the manner as may be prescribed by the Central Government.

(2) Every establishment shall register a copy of the said policy with the Chief Commissioner or the State Commissioner, as the case may be.

22. Maintenance of records —

(1) Every establishment shall maintain records of the persons with disabilities in relation to the matter of employment, facilities provided and other necessary information in compliance with the provisions of this Chapter in such form and manner as may be prescribed by the Central Government.

(2) Every employment exchange shall maintain records of persons with disabilities seeking employment.

(3) The records maintained under sub-section (1) shall be open to inspection at all reasonable hours by such persons as may be authorised in their behalf by the appropriate Government.

23. Appointment of Grievance Redressal Officer —

(1) Every Government establishment shall appoint a Grievance Redressal Officer for the purpose of section 19 and shall in form the Chief Commissioner or the State Commissioner, as the case may be, about the appointment of such officer.

(2) Any person aggrieved with the non-compliance of the provisions of section 20, may file acomplaint with the Grievance Redressal Officer, who shall investigate it and shall take up the matter with the establishment for corrective action.

(3) The Grievance Redressal Officer shall maintain a register of complaints in the manner as may be prescribed by the Central Government, and every complaint shall be inquired within two weeks of its registration.

(4) If the aggrieved person is not satisfied with the action taken on his or her complaint, he or she may approach the District-Level Committee on disability.

CHAPTER-V
SOCIAL SECURITY, HEALTH, REHABILITATION AND RECREATION

24. Social security —

(1) The appropriate Government shall within the limit of its economic capacity and development formulate necessary schemes and programmes to safeguard and promote the right of persons with disabilities for adequate standard of living to enable them to live independently or in the community:

Provided that the quantum of assistance to the persons with disabilities under such schemes and programmes shall beat least twenty-five percent higher than the similar schemes applicable too thers.

(2) The appropriate Government while devising these schemes and programmes shall give due consideration to the diversity of disability, gender, age, and socio-economic status.

(3) The schemes under sub-section (1) shall provide for, —

(a) community centres with good living conditions interms of safety, sanitation, healthcare and counselling;

(b) facilities for persons including children with disabilities who have no family or have been abandoned, or are without shelter or livelihood;

(c) support during natural or man-made disasters and in areas of conflict;

(d) support to women with disability for livelihood and for upbringing of their children;

(e) access to safe drinking water and appropriate and accessible sanitation facilities especially in urban slums and rural areas;

(f) provisions of aids and appliances, medicine and diagnostic services and corrective surgery free of cost to persons with disabilities with such income ceiling as may be notified;

(g) disability pension to persons with disabilities subject to such income ceiling as may be notified;

(h) unemployment allowance to persons with disabilities registered with Special Employment Exchange for more than two years and who could not be placed in any gainful occupation;

(i) caregiver allowance to persons with disabilities with high support needs;

(j) comprehensive insurance scheme for persons with disability, not covered under the Employees State Insurance Schemes, or any other statutory or Government-sponsored insurance schemes;

(k) any other matter which the appropriate Government may thinkfit.

25. Healthcare —

(1) The appropriate Government and the local authorities shall take necessary measures for the persons with disabilities to provide, —

(a) free healthcare in the vicinity specially in rural area subject to such family income as may be notified;

(b) barrier-free access in all parts of Government and private hospitals and other healthcare institutions and centres;

(c) priority in attendance and treatment.

(2) The appropriate Government and the local authorities shall take measures and make schemes or programmes to promote healthcare and prevent the occurrence of disabilities and for the said

purpose shall —

(a) undertake or cause to be undertaken surveys, investigations and research concerning the cause of occurrence of disabilities;
(b) promote various methods for preventing disabilities;
(c) screen all the children at least once in a year for the purpose of identifying "at-risk"cases;
(d) provide facilities for training to the staff at the primary health centres;
(e) sponsor or cause to be sponsored awareness campaigns and disseminate or cause to be disseminated information for general hygiene, health and sanitation;
(f) take measures for pre-natal, peri-natal and post-natal care of mother and child;
(g) educate the public through the pre-schools, schools, primary health centres, village level workers and *angan-wadi* workers;
(h) create awareness amongst the masses through television, radio and other mass-media on the causes of disabilities and the preventive measures to be adopted;
(i) healthcare during the time of natural disasters and other situations of risk;
(j) essential medical facilities for life saving emergency treatment and procedures; and
(k) sexual and reproductive health care especially for women with disability.

26. Insurance schemes —

The appropriate Government shall, by notification, make insurance schemes for their employees with disabilities.

27. Rehabilitation —

(1) The appropriate Government and the local authorities shall within their economic capacity and development, undertake or cause to be undertaken services and programmes of rehabilitation, particularly in the areas of health, education and employment for all persons with disabilities.
(2) For the purposes of sub-section (1), the appropriate Government and the local authorities maygrant financial assistance to non-Governmental Organisations.
(3) The appropriate Government and the local authorities, while formulating rehabilitation policies shall consult the non-Governmental Organisations working for the cause of persons with disabilities.

28. Research and development —

The appropriate Government shall initiate or cause to be initiated research and development through individuals and institutions on issues which shall enhance habilitation and rehabilitation and on such other issues which are necessary for the empowerment of persons with disabi-lities.

29. Culture and recreation —

The appropriate Government and the local authorities shall take measures to promote and protect the rights of all persons with disabilities to have a cultural life and to participate in recreational activities equally with others which include,—

(a) facilities, support and sponsorships to artists and writers with disability to pursue their interests and talents;
(b) establishment of a disability history museum which chronicles and interprets the historical experiences of persons with disabilities;
(c) making art accessible to persons with disabilities;
(d) promoting recreation centres, and other associational activities;
(e) facilitating participation in scouting, dancing, art classes, outdoor camps and adventure activities;
(f) redesigning courses in cultural and arts subjects to enable participation and access for persons with disabilities;
(g) developing technology, assistive devices and equipments to facilitate access and inclusion for persons with disabilities in recreational activities; and
(h) ensuring that persons with hearing impairment can have access to television programmes with sign language interpretation or subtitles.

30. Sporting activities —

(1) The appropriate Government shall take measures to ensure effective participation in sporting activities of the persons with disabilities.
(2) The sports authorities shall accord due recognition to the right of persons with disabilities to participate in sports and shall make due provisions for the inclusion of persons with disabilities in their schemes and programmes for the promotion and development of sporting talents.
(3) Without prejudice to the provisions contained in sub-sections (1) and (2), the appropriate Government and the sports authorities shall take measures to, —

(a) restructure courses and programmes to ensure access, inclusion and participation of persons with disabilities in all sporting activities;
(b) redesign and support infrastructure facilities of all sporting activities for persons with disabilities;
(c) develop technology to enhance potential, talent, capacity and ability in sporting activities of all persons with disabilities;
(d) provide multi-sensory essentials and features in all sporting activities to ensure effective participation of all persons with disabilities;
(e) allocate funds for development of state of art sport facilities for training of persons with disabilities;
(f) promote and organise disability specific sporting events for persons with disabilities and also facili-

tate awards to the winners and other participants of such sporting events.

CHAPTER-VI
SPECIAL PROVISIONS FOR PERSONS WITH BENCHMARK DISABILITES

31. Free education for children with benchmark disabilities —

(1) Notwithstanding anything contained in the Rights of Children to Free and Compulsory Education Act, 2009 (35 of 2009), every child with benchmark disability between the age of six to eighteen years shall have the right to free education in a neighbourhood school, or in a special school, of his choice.

(2) The appropriate Government and local authorities shall ensure that every child with benchmark disability has access to free education in an appropriate environment till he attains the age of eighteen years.

32. Reservation in higher educational institutions —

(1) All Government institutions of higher education and other higher education institutions receiving aid from the Government shall reserve not less than five per cent, seats for persons with benchmark disabilities.

(2) The persons with benchmark disabilities shall be given an upper age relaxation of five years for admission in institutions of higher education.

33. Identification of posts for reservation —

The appro-priate Government shall —

(i) identify posts in the establishments which can be held by respective category of persons with benchmark disabilities in respect of the vacancies reserved in accordance with the provisions of section 34;

(ii) constitute an expert committee with representation of persons with benchmark disabilities for identification of such posts; and

(iii) undertake periodic review of the identified posts at an interval not exceeding three years.

34. Reservation —

(1) Every appropriate Government shall appoint in every Government establishment, not less than four per cent of the total number of vacancies in the cadre strength in each group of posts meant to be filled with persons with benchmark disabilities of which, one per cent each shall be reserved for persons with benchmark disabilities under clauses (a), (b) and (c) and one per cent for persons with benchmark disabilities under clauses (d) and (e), namely:—

(a) blindness and low vision;

(b) deaf and hard of hearing;

(c) locomotor disability including cerebral palsy, leprosy cured, dwarfism, acid attack victims and muscular dystrophy;

(d) autism, intellectual disability, specific learning disability and mental illness;

(e) multiple disabilities from amongst persons under clauses (a) to (d) including deaf-blindness in the posts identified for each disabilities:

Provided that the reservation in promotion shall be in accordance with such instructions as are issued by the appropriate Government from time to time:

Provided further that the appropriate Government, in consultation with the Chief Commissioner or the State Commissioner, as the case may be, may, having regard to the type of work carried out in any Government establishment, by notification and subject to such conditions, if any, as may be specified in such notifications exempt any Government establishment from the provisions of this section.

(2) Where in any recruitment year any vacancy cannot be filled up due to non-availability of a suitable person with benchmark disability or for any other sufficient reasons, such vacancy shall be carried forward in the succeeding recruitment year and if in the succeeding recruitment year also suitable person with benchmark disability is not available, it may first be filled by interchange among the five categories and only when there is no person with disability available for the post in that year, the employer shall fill up the vacancy by appointment of a person, other than a person with disability:

Provided that if the nature of vacancies in an establishment is such that a given category of person cannot be employed, the vacancies may be interchanged among the five categories with the prior approval of the appropriate Government.

(3) The appropriate Government may, by notification, provide for such relaxation of upper age limit for employment of persons with benchmark disability, as it thinks fit.

35. Incentives to employers in private sector —

The appropriate Government and the local authorities shall, within the limit of their economic capacity and development, provide incentives to employer in private sector to ensure that at least five per cent. of their work force is composed of persons with benchmark disability.

36. Special employment exchange —

The appropriate Government may, by notification, require that from such date, the employer in every establishment shall furnish such information or return as may be prescribed by the Central Government in relation to vacancies appointed for persons with benchmark disability that have occurred or are about to occur in that establishment to such special employment exchange as may be notified by the Central

Government and the establish-ment shall there upon comply with such requisition.

37. Special schemes and development programmes —

The appropriate Government and the local authorities shall, by notification, make schemes in favour of persons with benchmark disabilities, to provide, —

(a) five per cent reservation in allotment of agricultural land and housing in all relevant schemes and development programmes, with appropriate priority to women with benchmark disabilities;

(b) five per cent reservation in all poverty alleviation and various developmental schemes with priority to women with benchmark disabilities;

(c) five per cent reservation in allotment of land on concessional rate, where such land is to be used for the purpose of promoting housing, shelter, setting up of occupation, business, enterprise, recreation centres and production centres.

CHAPTER-VII
SPECIAL PROVISIONS FOR PERSONS WITH DISABILITIES WITH HIGH SUPPORT NEEDS

38. Special provisions for persons with disabilities with high support —

(1) Any person with benchmark disability, who considers himself to be in need of high support, or any person or organisationon his or her behalf, may apply to an authority, to be notified by the appropriate Government, requesting to provide high support.

(2) On receipt of an application under sub-section (1), the authority shall refer it to an Assessment Board consisting of such Members as may be prescribed by the Central Government.

(3) The Assessment Board shall assess the case referred to it under sub-section (1) in such manner asmay be prescribed by the Central Government, and shall send a report to the authority certifying the need of high support and its nature.

(4) On receipt of a report under sub-section (3), the authority shall take steps to provide support inaccordance with the report and subject to relevant schemes and orders of the appropriate Government in this behalf.

CHAPTER-VIII
DUTIES AND RESPONSIBILITIES OF APPROPRIATE GOVERNMENTS

39. Awareness campaigns —

(1) The appropriate Government, in consultation with the Chief Commissioner or the State Commissioner, as the case may be, shall conduct, encourage, support orpromote awareness campaigns and sensitisation programmes to ensure that the rights of the persons with disabilities provided under this Act are protected.

(2) The programmes and campaigns specified under sub-section (1) shall also, —

(a) promote values of inclusion, tolerance, empathy and respect for diversity;

(b) advance recognition of the skills, merits and abilities of persons with disabilities and of their contributions to the workforce, labour market and professional fee;

(c) foster respect for the decisions made by persons with disabilities on all matters related to family life, relationships, bearing and raising children;

(d) provide orientation and sensitisation at the school, college, University and professional training level on the human condition of disability and the rights of persons with disabilities;

(e) provide orientation and sensitisation on disabling conditions and rights of persons with disabilities to employers, administrators and co-workers;

(f) ensure that the rights of persons with disabilities are included in the curriculum in Universities, colleges and schools.

40. Accessibility —

The Central Government shall, in consultation with the Chief Commissioner, formulate rules for persons with disabilities laying down the standards of accessibility for the physical environment, transportation, information and communications, including appropriate technologies and systems, and other facilities and services provided to the public in urban and rural areas.

41. Access to transport —

(1) The appropriate Government shall take suitable measures to provide, —

(a) facilities for persons with disabilities at bus stops, railway stations and airports conforming to the accessibility standards relating to parkings paces, toilets, ticketing counters and ticketing machines;

(b) access to all modes of transport that conform the design standards, including retrofitting old modes of transport, wherever technically feasible and safe for persons with disabilities, economically viable and without entailing major structural changes in design;

(c) accessible roads to address mobility necessary for persons with disabilities.

(2) The appropriate Government shall develop schemes programmes to promote the personal mobility of persons with disabilities at affordable cost to provide for,—

(a) incentives and concessions;

(b) retrofitting of vehicles; and

(c) personal mobility assistance.

42. Access to information and communication technology —

The appropriate Government shall take measures to ensure that,—

(i) all contents available in audio, print and electronic media are in accessible format;

(ii) persons with disabilities have access to electronic media by providing audio description, sign language interpretation and close captioning;

(iii) electronic goods and equipment which are meant for every day use are available in universal design.

43. Consumer goods —

The appropriate Government shall take measures to promote development, produc-tion and distribution of universally designed consumer products and accessories for general use for persons with disabilities.

44. Mandatory observance of accessibility norms —

(1) No establishment shall be granted permission to build any structure if the building plan does not adhere to the rules formulated by the Central Government under section 40.

(2) No establishment shall be issued a certificate of completion or allowed to take occupation of a building unless it has adhered to the rules formulated by the Central Government.

45. Time limit for making existing infrastructure and premises accessible and action for that purpose —

(1) All existing public buildings shall be made accessible in accordance with the rules formulated by the Central Government with in a period not exceeding five years from the date of notification of such rules:

Provided that the Central Government may grant extension of time to the States on a case to case basis for adherence to this provision depending on their state of preparedness and other related parameters.

(2) The appropriate Government and the local authorities shall formulate and publish an action plan based on prioritisation, for providing accessibility in all their buildings and spaces providing essential services such as all primary health centres, civil hospitals, schools, railwaystations and bus stops.

46. Time limit for accessibility by service providers —

The service providers whether Government or private shall provide services in accordance with the rules on accessibility formulated by the Central Government under section 40 with in a period of two years from the date of notification of such rules:

Provided that the Central Government in consultation with the Chief Commissioner may grant extension of time for providing certain category of services in accordance with the said rules.

47. Human resource development —

(1) Without prejudice to any function and power of Rehabilitation Council of India constituted under the Rehabilitation Council of India Act, 1992 (34 of 1992), the appro-priate Government shall endeavour to develop human resource for the purposes ofthis Act and to that end shall, —

(a) mandate training on disability rights in all courses for the training of Panchayati Raj Members, legislators, administrators, police officials, judges and lawyers;

(b) induct disability as a component for all education courses for schools, colleges and University teachers, doctors, nurses, paramedical personnel, social welfare officers, rural development officers, ashaworkers, *anganwadi* workers, engineers, architects, other professionals and community workers;

(c) initiate capacity building programmes including training in independent living and community relationships for families, members of community and other stake holders and care providers on caregiving and support;

(d) ensure independence training for persons with disabilities to build community relationships on mutual contribution and respect;

(e) conduct training programmes for sports teachers with focus on sports, games, adventure activities;

(f) any other capacity development measures as may be required.

(2) All Universities shall promote teaching and research in disability studies including establishment of study centres for such studies.

(3) In order to fulfil the obligation stated in subsection (1), the appropriate Government shall in every five years undertake a need based analysis and formulate plans for the recruitment, induction, sensitisation, orientation and training of suitable personnel to undertake the various responsibilities under this Act.

48. Social audit —

The appropriate Government shall undertake social audit of all general schemes and programmes involving the persons with disabilities to ensure that the scheme and programmes do not have an adverse impact upon the persons with disabilities and need the requirements and concerns of persons with disabilities.

CHAPTER-IX
REGISTRATION OF INSTITUTIONS FOR PERSONS WITH DISABILITIES AND GRANTS TO SUCH INSTITUTIONS

49. Competent authority —

The State Government shall appoint an authority as it deems fit to be a competent authority for the purposes of this Chapter.

50. Registration —

Save as otherwise provided under this Act, no person shall establish or maintain any insti-tution for persons with disabilities except in accordance with a certificate of registration issued in this behalf by the competent authority:

Provided that an institution for care of mentally ill persons, which holds a valid licence under section 8 of the Mental Health Act, 1987 (14 of 1987) or any other Act for the time being in force, shall not be required to be registered under this Act.

51. Application and grant of certificate of registration —

(1) Every application for a certificate of registration shall be made to the competent authority in such form and in such manner as may be prescribed by the State Government.

(2) On receipt of an application under sub-section (1), the competent authority shall make such enquiries a sit may deemfit and on being satisfied that the applicant has complied with the requirements of this Act and the rules made there under, it shall grant a certificate of registration to the applicant within a period of ninety days of receipt of application and if not satisfied, the competent authority shall, by order, refuse to grant the certificate applied for:

Provided that before making any order refusing to grant a certificate, the competent authority shall give the applicant a reasonable opportunity of being heard and every order of refusal to grant a certificate shall be communicated to the applicant in writing.

(3) No certificate of registration shall be granted under sub-section (2) unless the institution with respect to which an application has been made is in a position to provide such facilities and meet such standards as may be prescribed by the State Government.

(4) The certificate of registration granted under sub-section (2), —

(a) shall, unless revoked under section 52 remain in force for such period as may be prescribed by the State Government;

(b) may be renewed from time to time for a like period; and

(c) shall be in such form and shall be subject to such conditions as may be prescribed by the State Government.

(5) An application for renewal of a certificate of registration shall be made not less than sixty days before the expiry of the period of validity.

(6) A copy of the certificate of registration shall be displayed by the institution in a conspicuous place.

(7) Every application made under sub-section (1) or sub-section (*5*) shall be disposed of by the competent authority within such period as may be prescribed by the State Government.

52. Revocation of registration —

(1) The competent authority may, if it has reason to believe that the holder of a certificate of registration granted under sub-section (2) of section 51 has, —

(a) made a statement in relation to any application for the issue or renewal of the certificate which is in corrector false in material particulars; or

(b) committed or has caused to be committed any breach of rules or any conditions subject to which the certificate was granted, it may, after making such inquiry, a sit deemsfit, by order, revoke the certificate:

Provided that no such order shall be made until an opportunity is given to the holder of the certificate to showcause as to why the certificate of registration shall not be revoked.

(2) Where a certificate of registration in respect of an institution has been revoked under sub-section (1), such institution shall cease to function from the date of such revocation:

Provided that where an appeal lies under section 53 against the order of revocation, such institutions hall cease to function,—

(a) where no appeal has been preferred immediately on the expiry of the period prescribed for the filing of such appeal; or

(b) where such appeal has been preferred, but the order of revocation has been upheld, from the date of the order of appeal.

(3) On the revocation of a certificate of registration in respect of an institution, the competent authority may direct that any person with disability who is an inmate of such institution on the date of such revocation, shall be —

(a) restored to the custody of his or her parent, spouse or lawful guardian, as the case may be; or

(b) transferred to any other institution specified by the competent authority.

(4) Every institution which holds a certificate of registration which is revoked under this section shall, immediately after such revocation, surrender such certificate to the competent authority.

53. Appeal —

(1) Any person aggrieved by the order of the competent authority refusing to grant acertificate of registration or revoking a certificate of registration may, within such period as may be prescribed by the State Government, prefer an appeal to such appellate authority, as may be notified by the State Government against such refusal or revocation.

(2) The order of the appellate authority on such appeal shall be final.

54. Act not to apply to institutions established or maintained by Central or State Government —

Nothing contained in this Chapter shall apply to an institution for persons with disabilities established or maintained by the Central Government or a State Government.

55. Assistance to registered institutions —

The appro-priate Government may within the limits of their economic capacity and development, grant financial assistance to registered institutions to provide services and to implement the schemes and programmes in pursuance of the provisions of this Act.

CHAPTER-X
CERTIFICATION OF SPECIFIED DISABILITIES

56. Guidelines for assessment of specified disabilities

The Central Government shall notify guidelines for the purpose of assessing the extent of specified disability in a person.

57. Designation of certifying authorities —

(1) The appropriate Government shall designate persons, having requisite qualifications and experience, as certifying authorities, who shall be competent to issue the certificate of disability.

(2) The appropriate Government shall also notify the jurisdiction within which and the terms and conditions subject to which, the certifying authority shall perform its certification functions.

58. Procedure for certification —

(1) Any person with specified disability, may apply, in such manner as may be prescribed by the Central Government, to a certifying authority having jurisdiction, for issuing of a certificate of disability.

(2) On receipt of an application under sub-section (1), the certifying authority shall assess the disability of the concerned person in accordance with relevant guidelines notified under section 56, and shall, after such assessment, as the case may be,—

(a) issue a certificate of disability to such person, in such form as may be prescribed by the Central Government;

(b) inform him in writing that he has no specified disability.

(3) The certificate of disability issued under this sections hall be valid across the country.

59. Appeal against a decision of certifying authority

(1) Any person aggrieved with decision of the certifying authority, may appeal against such decision, within such time and in such manner as may be prescribed by the State Government, to such appellate authority as the State Government may designate for the purpose.

(2) On receipt of an appeal, the appellate authority shall decide the appeal in such manner as may be prescribed by the State Government.

CHAPTER-XI
CENTRAL AND STATE ADVISORY BOARDS ON DISABILITY AND DISTRICT LEVEL COMMITTEE

60. Constitution of Central Advisory Board on Disability —

(1) The Central Government shall, by notification, constitute a body to be known as the Central Advisory Board on Disability to exercise the powers conferred on, and to perform the functions assigned to it, under this Act.

(2) The Central Advisory Board shall consist of,—

(a) the Minister incharge of Department of Disability Affairs in the Central Government, Chairperson, ex-officio;

(b) the Minister of State incharge dealing with Department of Disability Affairs in the Ministry in the Central Government, Vice Chairperson, ex-officio;

(c) three Members of Parliament, of whom two shall be elected by Lok Sabha and one by the Rajya Sabha, Members, ex-officio;

(d) the Ministers in charge of Disability Affairs of all States and Administrators or Lieutenant Governors of the Union territories, Members, ex-officio;

(e) Secretaries to the Government of India incharge of the Ministries or Departments of Disability Affairs, Social Justice and Empowerment, School Education and Literacy, and Higher Education, Women and Child Development, Expenditure, Personnel and Training, Administrative Reforms and Public Grievances, Health and Family Welfare, Rural Development, Panchayati Raj, Industrial Policy and Promotion, Urban Development, Housing and Urban Poverty Alleviation, Science and Technology, Communications and Information Technology, Legal Affairs, Public Enterprises, Youth Affairs and Sports, Road Transport and Highways and Civil Aviation, Members, ex-officio;

(f) Secretary, National Institute of Transforming India (NITI) Aayog, Member, ex-officio;

(g) Chairperson, Rehabilitation Council of India, Member, ex-officio;

(h) Chairperson, National Trust for the Welfare of Persons with Autism, Cerebral Palsy, Mental Retardation and Multiple Disabilities, Member, ex-officio;

(i) Chairman-cum-Managing Director, National Handicapped Finance Development Corpora-

tion, Member, ex-officio;

(j) Chairman-cum-Managing Director, Artificial Limbs Manufacturing Corporation, Member, ex-officio;

(k) Chairman, Railway Board, Member, ex-officio;

(l) Director-General, Employment and Training, Ministry of Labour and Employment, Member, ex-officio;

(m) Director, National Council for Educational Research and Training, Member, ex-officio;

(n) Chairperson, National Council of Teacher Education, Member, ex-officio;

(o) Chairperson, University Grants Commission, Member, ex-officio;

(p) Chairperson, Medical Council of India, Member, ex-officio;

(q) Directors of the following Institutes:—

(i) National Institute for the Visually Handicapped, Dehradun;

(ii) National Institute for the Mentally Handicapped, Secunderabad;

(iii) Pandit Deen Dayal Upadhyay Institute for the Physically Handicapped, New Delhi;

(iv) Ali Yavar Jung National Institute for the Hearing Handicapped, Mumbai;

(v) National Institute for the Orthopaedically Handi-capped, Kolkata;

(vi) National Institute of Rehabilitation Training and Research, Cuttack;

(vii) National Institute for Empowerment of Persons with Multiple Disabilities, Chennai;

(viii) National Institute for Mental Health and Sciences, Bangalore;

(ix) Indian Sign Language Research and Training Centre, New Delhi, Members, ex-officio;

(r) Members to be nominated by the Central Government,—

(i) five Members who are experts in the field of disability and rehabilitation;

(ii) ten Members, as far as practicable, being persons with disabilities, to represent non-Governmental Organisations concerned with disabilities or disabled persons organisations: Provided that out of the ten Members nominated, at least, five Members shall be women and atleast one person each shall be from the Scheduled Castes and the Scheduled Tribes;

(iii) upto three representatives of national level chambers of commerce and industry;

(s) Joint Secretary to the Government of India dealing with the subject of disability policy, Member-Secretary, ex-officio.

61. Terms and conditions of Service of members —

(1) Save as otherwise provided under this Act, a Member of the Central Advisory Board nominated under clause (r) of sub-section (2) of section 60 shall hold office for a term of three years from the date of his nomination:

Provided that such a Member shall, not with standing the expiration of his term, continue to hold office until his successor enters upon his office.

(2) The Central Government may, if it thinks fit, remove any Member nominated under clause (r) of sub-section (2) of section 60, before the expiry of his term of office after giving him a reasonable opportunity of showing cause against the same.

(3) A Member nominated under clause (r) of sub-section (2) of section 60 may at any time resign his office by writing under his hand addressed to the Central Government and the seat of the said Member shall thereupon becomes vacant.

(4) A casual vacancy in the Central Advisory Board shall be filled by a fresh nomination and the person nominated to fill the vacancy shall hold office only for the remainder of the term for which the Member in whose place he was so nominated.

(5) A Member nominated under sub-clause (i) or sub-clause (iii) of clause (r) of sub-section (2) of section 60 shall be eligible for renomination.

(6) The Members nominated under sub-clause (i) and sub-clause (ii) of clause (r) of sub-section (2) of section 60 shall receive such allowances as may be prescribed by the Central Government.

62. Disqualifications —

(1) No person shall be a Member of the Central Advisory Board, who —

(a) is, or at anytime has been, adjudged in solvent or has suspended payment of his debts or has compounded with his creditors, or

(b) is of unsound mind and stands so declared by a competent court, or

(c) is, or has been, convicted of an offence which, in the opinion of the Central Government, involves moral turpitude, or

(d) is, or at any time has been, convicted of an offence under this Act, or

(e) has so abused his position in the opinion of the Central Government as a Member so as to render his continuance in the office is prejudicial interests of the general public.

(2) No order of removal shall be made by the Central Government under this section unless the Member concerned has been given a reasonable opportunity of showing cause against the same.

(3) Notwithstanding anything contained in sub-section (1) or sub-section (5) of section 61, a

Member who has been removed under this section shall not be eligible for renomination as a Member.

63. Vacation of seats by Members —

If a Member of the Central Advisory Board becomes subject to any of the disqualifications specified in section 62, his seats hall become vacant.

64. Meetings of the Central Advisory Board on disability —

The Central Advisory Board shall meet at least once in every six months and shall observe such rules of procedure in regard to the transaction of business at its meetings as may be prescribed.

65. Functions of Central Advisory Board on disability —

(1) Subject to the provisions of this Act, the Central Advisory Board on disability shall be the national-level consultative and advisory body on disability matters, and shall facilitate the continuous evolution of a comprehensive policy for the empowerment of persons with disabilities and the full enjoyment of rights.

(2) In particular and without prejudice to the generality of the foregoing provisions, the Central Advisory Board on disability shall perform the following functions, namely:—

(a) advise the Central Government and the State Governments on policies, programmes, legislation and projects with respect to disability;

(b) develop a national policy to address issues concerning persons with disabilities;

(c) review and coordinate the activities of all Departments of the Government and other Government a land non-Governmental Organisations which are dealing with matters relating to persons with disabilities;

(d) takeup the cause of persons with disabilities with the concerned authorities and the international organisations with a view to provide for schemes and projects for the persons with disabilities in the national plans;

(e) recommend steps to ensure accessibility, reasonable accommodation, non-discrimination for persons with disabilities *vis-a-vis* information, services and the built environment and their participation in social life;

(f) monitor and evaluate the impact of laws, policies and programmes to achieve full participation of persons with disabilities; and

(g) such other functions as may be assigned from time to time by the Central Government.

66. State Advisory Board on disability —

(1) Every State Government shall, by notification, constitute a body to be known as the State Advisory Board on disability to exercise the powers conferred on, and to perform the function assigned to it, under this Act.

(2) The State Advisory Board shall consist of —

(a) the Minister in charge of the Department in the State Government dealing with disability matters, Chairperson, ex-officio;

(b) the Minister of State or the Deputy Minister incharge of the Department in the State Government dealing with disability matters, if any, Vice-Chairperson, ex-officio;

(c) secretaries to the State Government in charge of the Departments of Disability Affairs, School Education, Literacy and Higher Education, Women and Child Development, Finance, Personnel and Training, Health and Family Welfare, Rural Development, Panchayati Raj, Industrial Policy and Promotion, Labour and Employment, Urban Development, Housing and Urban Poverty Alleviation, Science and Technology, Information Technology, Public Enterprises, Youth Affairs and Sports, Road Transport and any other Department, which the State Government considers necessary, Members, ex-officio;

(d) three Members of the State Legislature of whom two shall be elected by the Legislative Assembly and one by the Legislative Council, if any, and where there is no Legislative Council, three Members shall be elected by the Legislative Assembly, Members, ex-officio;

(e) Members to be nominated by the State Government:—

(i) five Members who are experts in the field of disability and rehabilitation;

(ii) five Members to be nominated by the State Government by rotation to represent the districts in such manner as may be prescribed:
Provided that no nomination under this sub-clause shall be made excepton the recommendation of the district administration concerned;

(iii) ten persons as far as practicable, being persons with disabilities, to represent non-Governmental Organisations or associations which are concerned with disabilities:
Provided that out of the ten persons nominated under this clause, at least, five shall be women and atleast one person each shall be from the Scheduled Castes and the Scheduled Tribes;

(iv) not more than three representatives of the State Chamber of Commerce and Industry;

(f) officer not below the rank of Joint Secretary in the Department dealing with disability matters in the State Government, Member-Secretary, ex-officio.

67. Terms and conditions of service of Members —

(1) Save as other wise provided under this Act, a Member of the State Advisory Board nominated under clause (e) of sub-section (2) of section 66, shall hold office for a term of three years from the date of his nomination:

Provided that such a Member shall, not with standing the expiration of his term, continue to hold office until his successor enters upon his office.

(2) The State Government may, if it thinks fit, remove any Member nominated under clause (e) of sub-section (2) of section 66, before the expiry of his term of office after giving him a reasonable opportunity of showing cause against the same.

(3) A Member nominated under clause (e) of sub-section (2) of section 66 may at any time resign his office by writing under his hand addressed to the State Government and the seat of the said Member shall there upon become vacant.

(4) A casual vacancy in the State Advisory Board shall be filled by a fresh nomination and the person nominated to fill the vacancy shall hold office only for the remainder of the term for which the Member in whose place he was so nominated.

(5) A Member nominated under sub-clause (i) or sub-clause (iii) of clause (e) of sub-section (2) of section 66 shall be eligible for renomination.

(6) The Members nominated under sub-clause (i) and sub-clause (ii) of clause (e) of sub-section (2) of section 66 shall receive such allowances as may be prescribed by the State Government.

68. Disqualification —

(1) No person shall be a Member of the State Advisory Board, who —

(a) is, or at any time has been, adjudged in solvent or has suspended payment of his debts or has compounded with his creditors, or

(b) is of unsound mind and stands so declared by a competent court, or

(c) is, or has been, convicted of an offence which, in the opinion of the State Government, involves moral turpitude, or

(d) is, or at any time has been, convicted of an offence under this Act, or

(e) has so abused in the opinion of the State Government his position as a Member as to render his continuance in the State Advisory Board detrimental to the interests of the general public.

(2) No order of removal shall be made by the State Government under this section unless the Member concerned has been given areas on able opportunity of showing cause against the same.

(3) Not with standing anything contained in sub-section (1) or sub-section (5) of section 67, a Member who has been removed under this section shall not be eligible for renomination as a Member.

69. Vacation of seats —

If a Member of the State Advisory Board becomes subject to any of the disqualifications specified in section 68 his seat shall become vacant.

70. Meetings of State Advisory Board on disability — The State Advisory Board shall meet at leastonce in every six months and shall observe such rules or procedure in regard to the transaction of businessat its meetings as may be prescribed by the State Government.

71. Functions of State Advisory Board on disability —

(1) Subject to the provisions of this Act, the State Advisory Board shall be the State-level consultative and advisory body on disability matters, and shall facilitate the continuous evolution of a comprehensive policy for the empowerment of persons with disabilities and the full enjoyment of rights.

(2) In particular and without prejudice to the generality of the foregoing provisions, the State Advisory Board on disability shall perform the following functions, namely:—

(a) advise the State Government on policies, programmes, legislation and projects with respect to disability;

(b) develop a State policy to address issues concerning persons with disabilities;

(c) review and coordinate the activities of all Departments of the State Government and other Governmental and non-Governmental Organisations in the State which are dealing with matters relating to persons with disabilities;

(d) takeup the cause of persons with disabilities with the concerned authorities and the international organisations with a view to provide for schemes and projects for the persons with disabilities in the State plans;

(e) recommend steps to ensure accessibility, reasonable accommodation, non-discrimination for persons with disabilities, services and the built environment and their participation in social life on an equal basis with others;

(f) monitor and evaluate the impact of laws, policies and programmes designed to achieve full participation of persons with disabilities; and

(g) such other functions as may be assigned from time to time by the State Government.

72. District-level Committee on disability —

The State Government shall constitute District-level Committee on disability to perform such functions as may be prescribed by it.

73. Vacancies not to invalidate proceedings —

No act or proceeding of the Central Advisory Board on disability, a State Advisory Board on disability, or a District-level Committee on disability shall be called in question on the ground merely of the existence of any vacancy in or any defect in the constitution of such Board or Committee, as the case may be.

CHAPTER-XII
CHIEF COMMISSIONER AND STATE COMMISSIONER FOR PERSONS WITH DISABILITIES

74. Appointment of Chief Commissioner and Commissioners —

(1) The Central Government may, by notification, appoint a Chief Commissioner for Persons with Disabilities (hereinafter referred to as the" Chief Commissioner") for the purposes of this Act.

(2) The Central Government may, by notification appoint two Commissioners to assist the Chief Commissioner, of which one Commissioner shall be a persons with disability.

(3) A person shall not be qualified for appointment as the Chief Commissioner or Commissioner unless he has special knowledge or practical experience in respect of matters relating to rehabilitation.

(4) The salary and allowances payable to and other terms and conditions of service (including pension, gratuity and other retirement benefits) of the Chief Commissioner and Commissioners shall be such as may be prescribed by the Central Government.

(5) The Central Government shall determine the nature and categories of officers and other employees required to assist the Chief Commissioner in the discharge of his functions and provide the Chief Commissioner with such officers and other employees as it thinks fit.

(6) The officers and employees provided to the Chief Commissioner shall discharge their functions under the general super in tendence and control of the Chief Commissioner.

(7) The salaries and allowances and other conditions of service of officers and employees shall be such as may be prescribed by the Central Government.

(8) The Chief Commissioner shall be assisted by an advisory committee comprising of not more than eleven members drawn from the experts from different disabilities in such manner as may be prescribed by the Central Government.

75. Functions of Chief Commissioner —

(1) The Chief Commissioner shall —

(a) identify, *suomoto* or otherwise, the provisions of any law or policy, programme and procedures, which are in consistent with this Act and recommend necessary corrective steps;

(b) inquire, *suomoto* or otherwise, deprivation of rights of persons with disabilities and safe guards available to them in respect of matters for which the Central Government is the appropriate Government and takeup the matter with appropriate authorities for corrective action;

(c) review the safeguards provided by or under this Act or any other law for the time being inforce for the protection of rights of persons with disabilities and recommend measures for their effective implementation;

(d) review the factors that inhibit the enjoyment of rights of persons with disabilities and recommend appropriate remedial measures;

(e) study treaties and other international instruments on the rights of persons with disabilities and make recommendations for their effective implementation;

(f) undertake and promote research in the field of the rights of persons with disabilities;

(g) promote awareness of the rights of persons with disabilities and the safeguards available for their protection;

(h) monitor implementation of the provisions of this Act and schemes, programmes meant for persons with disabilities;

(i) monitor utilisation of funds disbursed by the Central Government for the benefit of persons with disabilities; and

(j) perform such other functions as the Central Government may assign.

(2) The Chief Commissioner shall consult the Commissioners on any matter while discharging its functions under this Act.

76. Action of appropriate authorities on recommendation of Chief Commissioner —

Whenever the Chief Commissioner makes a recommendation to an authority in pursuance of clause (b) of sub-section (1) of section 75, that authority shall take necessary action on it, and in form the Chief Commissioner of the action taken within three months from the date of receipt of the recommendation:

Provided that where an authority does not accept a recommendation, it shall convey reasons for non-acceptance to the Chief Commissioner within a period of three months, and shall also inform the aggrieved person.

77. Powers of Chief Commissioner —

(1) The Chief Commissioner shall, for the purpose of discharging his functions under this Act, have

the same powers of a civil court as are vested in a courtunder the Code of Civil Procedure, 1908 (5 of 1908) while trying a suit, in respect of the following matters, namely : —

(a) summoning and enforcing the attendance of witnesses;
(b) requiring the discovery and production of any documents;
(c) requisitioning any public recordor copy thereof from any court or office;
(d) receiving evidence on affidavits; and
(e) issuing commissions for the examination of witnesses or documents.

(2) Every proceeding before the Chief Commissioner shall be ajudicial proceeding within the meaning of sections 193 and 228 of the Indian Penal Code (45 of 1860) and the Chief Commissioner shall be deemed to be a civil court for the purposes of section 195 and Chapter XXVI of the Code of Criminal Procedure, 1973 (2 of 1974).

1. Ins. by Act 4 of 2018, s.3 and the second Schedule (w.e.f.5-1-2018).

78. Annual and special reports by Chief Commissioner —

(1) The Chief Commissioner shall submit an annual report to the Central Government and may at any time submit special reports on anymatter, which, in his opinion, is of such urgency or importance that it shall not be deferred till submission of the annual report.
(2) The Central Government shall cause the annual and the special reports of the Chief Commissioner to be laid before each House of Parliament, along with a memorandum of action taken or proposed to be taken on his recommendations and the reasons for non-acceptance the recommendations, if any.
(3) The annual and special reports shall be prepared in such form, manner and contain such details as may be prescribed by the Central Government.

79. Appointment of State Commissioner in States —

(1) The State Government may, by notification, appoint a State Commissioner for Persons with Disabilities (hereinafter referred to as the "State Commissioner") for the purposes of this Act.
(2) A person shall not be qualified for appointment as the State Commissioner unless he has special knowledge or practical experience in respect of matters relating to rehabilitation.
(3) The salary and allowances payable to and other terms and conditions of service (including pension, gratuity and other retirement benefits) of the State Commissioner shall be such as may be prescribed by the State Government.
(4) The State Government shall determine the nature and categories of officers and other employ eesrequired to assist the State Commissioner in the discharge of his functions and provide the State Commissioner with such officers and other employees as it thinks fit.
(5) The officers and employees provided to the State Commissioner shall discharge his functions under the general superintendence and control of the State Commissioner.
(6) The salaries and allowances and other conditions of service of officers and employees shall be such as may be prescribed by the State Government.
(7) The State Commissioner shall be assisted by an advisory committee comprising of not more than five members drawn from the experts in the disability sector in such manner as may be prescribed by the State Government.

80. Functions of State Commissioner —

The State Commissioner shall —

(a) identify, *suo motu* or otherwise, provision of any law or policy, programme and procedures, which are in consistent with this Act, and recommend necessary corrective steps;
(b) inquire, *suo motu* or otherwise deprivation of rights of persons with disabilities and safeguards available to them in respect of matters for which the State Government is the appropriate Government and take upthe matter with appropriate authorities for corrective action;
(c) review the safeguards provided by or under this Act or any other law for the time being inforce for the protection of rights of persons with disabilities and recommend measures for their effective implementation;
(d) review the factors that in hibit the enjoyment of rights of persons with disabilities and recommend appropriate remedial measures;
(e) under take and promote research in the field of the rights of persons with disabilities;
(f) promote awareness of the rights of persons with disabilities and the safeguards available for their protection;
(g) monitor implementation of the provisions of this Act and schemes, programmes meant for persons with disabilities;
(h) monitor utilisation of funds disbursed by the State Government for the benefits of persons with disabilities; and
(i) perform such other functions as the State Government may assign.

81. Action by appropriate authorities on recommendation of State Commissioner —

Whenever the State Commissioner makes a recommendation to an authority in pursuance of clause (b) of section 80, that authority shall take necessary action on it, and inform the State Commissioner of the action taken with in three months from the date of receipt of the recommendation:

Provided that where an authority does not accept a

recommendation, it shall convey reasons for non-acceptance to the State Commissioner for Persons with Disabilities within the period of three months, and shall also inform the aggrieved person.

82. Powers of State Commissioner —

(1) The State Commissioner shall, for the purpose of discharging their functions under this Act, have the same powers of a civil court as are vested in a court under the Code of Civil Procedure, 1908 (5 of 1908) while trying a suit, in respect of the following matters, namely: —
   (a) summoning and enforcing the attendance of witnesses;
   (b) requiring the discovery and production of any documents;
   (c) requisitioning any public record or copy there of from any court or office;
   (d) receiving evidence on affidavits; and
   (e) issuing commissions for the examination of witnesses or documents.

(2) Every proceeding before the State Commissioner shall be ajudicial proceeding within the meaning of sections 193 and 228 of the Indian Penal Code (45 of 1860) and the State Commissioners shall be deemed to be a civil court for the purposes of section 195 and Chapter XXVI of the Code of Criminal Procedure, 1973 (2 of 1974).

83. Annual and special reports by State Commissioner

(1) The State Commissioner shall submit an annual report to the State Government and may at anytime submit special reports on any matter, which, in its opinion, is of such urgency or importance that it shall not be deferred till submission of the annual report.

(2) The State Government shall cause the annual and the special reports of the State Commissioner for persons with disabilities to be laid before each House of State Legislature where it consists of two Houses or where such Legislature consist of one House, before that House along with a memorandum of action taken or proposed to be taken on the recommendation of the State Commissioner and the reasons for non-acceptance there commendations, if any.

(3) The annual and special reports shall be prepared in such form, manner and contain such details as may be prescribed by the State Government.

CHAPTER-XIII
SPECIAL COURT

84. Special Court —

For the purpose of providing speedy trial, the State Government shall, with the concurrence of the Chief Justice of the High Court, by notification, specify for each district, a Court of Session to be a Special Court to try the offences under this Act.

85. Special Public Prosecutor —

(1) For every Special Court, the State Government may, by notification, specify a Public Prosecutor or appoint an advocate, who has been in practice as an advocate for not less than seven years, as a Special Public Prosecutor for the purpose of conducting cases in that Court.

(2) The Special Public Prosecutor appointed under sub-section (1) shall be entitled to receive such fees or remuneration as may be prescribed by the State Government.

CHAPTER-XIV
NATIONAL FUND FOR PERSONS WITH DISABILITIES

86. National Fund for persons with disabilities —

(1) There shall be constituted a Fund to be called the National Fund for persons with disabilities and there shall be credited there to —
   (a) all sums available under the Fund for people with disabilities, constituted vide notification No. S.O. 573 (E), dated the 11th August, 1983 and the Trust Fund for Empowerment of Persons with Disabilities, constituted vide notification No.30-03/2004-DDII, dated the 21st November, 2006, under the Charitable Endowment Act, 1890 (6 of 1890).
   (b) all sums payable by banks, corporations, financial institutions in pursuance of judgment dated the 16th April, 2004 of the Hon'ble Supreme Court in Civil Appeal Nos. 4655 and 5218 of 2000;
   (c) all sums received by way of grant, gifts, donations, benefactions, be quests or transfers;
   (d) all sums received from the Central Government including grants-in-aid;
   (e) all sums from such other sources as may be decided by the Central Government.

(2) The Fund for persons with disabilities shall be utilised and managed in such manner as may be prescribed.

87. Accounts and audit —

(1) The Central Government shall maintain proper accounts and other relevant records and prepare an annual statement of accounts of the Fund including the income and expenditure accounts in such form as may be prescribed in consultation with the Comptroller and Auditor-General of India.

(2) The accounts of the Fund shall be audited by the Comptroller and Auditor-General of India at such intervals as may be specified by him and any expenditure incurred by him in connection with such audit shall be payable from the Fund to the Comptroller and Auditor-General of India.

(3) The Comptroller and Auditor-General of India and any other person appointed by him in connection with the audit of the accounts of the Fund shall have the same rights, privileges and authority in connection with such audit as the Comptroller and Auditor-General of India generally has in connection with the audit of the Government accounts, and in particular, shall have the right to demand production of books of account, c onnected vouchers and other documents and papers and to inspect any of the offices of the Fund.

(4) The accounts of the Fund as certified by the Comptroller and Auditor-General of India or anyother person appointed by him in this behalf, together with the audit report thereon, shall be laid before each House of Parliament by the Central Government.

CHAPTER-XV
STATE FUND FOR PERSONS WITH DISABILITIES

88. State Fund for persons with disabilities —

(1) There shall be constituted a Fund to be called the State Fund for persons with disabilities by a State Government in such manner as may be prescribed by the State Government.

(2) The State Fund for persons with disabilities shall be utilised and managed in such manner as may be prescribed by the State Government.

(3) Every State Government shall maintain proper accounts and other relevant records of the State Fund for persons with disabilities including the income and expenditure accounts in such form as may be prescribed by the State Government in consultation with the Comptroller and Auditor-General of India.

(4) The accounts of the State Fund for persons with disabilities shall be audited by the Comptroller and Auditor-General of India at such intervals as may be specified by him and any expenditure incurred by him in connection with such audit shall be payable from the State Fund to the Comptroller and Auditor-General of India.

(5) The Comptroller and Auditor-General of India and any person appointed by him in connection with the audit of the accounts of the State Fund for persons with disabilities shall have the same rights, privileges and authority in connection with such audit as the Comptroller and Auditor-General of India generally has in connection with the audit of the Government accounts, and in particular, shall have right to demand production of books of accounts, connected vouchers and other documents and papers and to inspect any of the offices of the State Fund.

(6) The accounts of the State Fund for persons with disabilities as certified by the Comptroller and Auditor-General of India or any other person appointed by him in this be half to get her with the audit report thereon shall be laid before each House of the State Legislature where it consists of two Houses or where such Legislature consists of one House before that House.

CHAPTER-XVI
OFFENCES AND PENALTIES

89. Punishment for contravention of provisions of Act or rules or regulations made there under —

Any person who contravenes any of the provisions of this Act, or of any rule made there under shall for first contravention be punishable with fine which may extend to ten thousand rupees and for any subsequent contravention with fine which shall not be less than fifty thousand rupees but which may extend to five lakh rupees.

90. Offences by companies —

(1) Where an offence under this Act has been committed by a company, every person who at the time the offence was committed, was in charge of, and was responsible to, the company for the conduct of the business of the company, as well as the company, shall be deemed to be guilty of the offence and shall be liable to be proceeded against and punished accordingly:

Provided that nothing contained in this sub-section shall render any such personliable to any punishment provided in this Act, if he proves that the offence was committed without his knowledge orthat he had exercised all due diligence to prevent the commission of such offence.

(2) Notwithstanding anything contained in sub-section (1), where an offence under this Act has been committed by a company and it is proved that the offence has been committed with the consent or connivance of, or is attributable to any neglect on the part of any director, manager, secretary or other officer of the company, such director, manager, secretary or other officer shall also be deemed to be guilty of that offence and shall beliable to be proceeded against and punished accordingly.

Explanation. — For the purposes of this section, —

(a) "company" means any body corporate and includes a firm or other association of individuals; and

(b) "director", in relation to afirm, means a partner in the firm.

91. Punishment for fraudulently availing any benefit meant for persons with benchmark disabilities —

Whoever, fraudulently avails or attempts to avail any benefit meant for persons with benchmark

disabilities, shall be punishable with imprisonment for a term which may extend to two years or with fine which may extend to one lakh rupees or with both.

92. Punishment for offences of atrocities —

Whoever, —

(a) intentionally insults or intimidates with intent to humiliate a person with disability in any place within public view;

(b) assaults or uses force to any person with disability with intent to dishonour him or outrage the modesty of a woman with disability;

(c) having the actual charge or control over a person with disability voluntarily or knowingly denies food or fluids to him or her;

(d) being in a position to dominate the will of a child or woman with disability and uses that position to exploit her sexually;

(e) voluntarily injures, damages or interferes with the use of any limb or sense or any supporting device of a person with disability;

(f) performs, conducts or directs any medical procedure to be performed on a woman with disability which leads to or is likely to lead to termination of pregnancy without her express consent except in cases where medical procedure for termination of pregnancy is done in severe cases of disability and with the opinion of a registered medical practitioner and also with the consent of the guardian of the woman with disability, shall be punishable with imprisonment for a term which shall not be less than six months but which may extend to five years and with fine.

93. Punishment for failure to furnish information —

Whoever, fails to produce any book, account or other documents or to furnish any statement, information or particulars which, under this Act or any order, or direction made or given there under, is duty bound to produce or furnish or to answer any question put in pursuance of the provisions of this Act or of any order, or direction made or given there under, shall be punishable with fine which may extend to twenty-five thousand rupees in respect of each offence, and in case of continued failure or refusal, with further fine which may extend to one thousand rupees for each day, of continued failure or refusal after the date of original order imposing punishment of fine.

94. Previous sanction of appropriate Government —

No Court shall take cognizance of an offence alleged to have been committed by an employee of the appropriate Government under this Chapter, except with the previous sanction of the appropriate Government or a complaint is filed by an officer authorised by it in this behalf.

95. Alternative punishments —

Where an act or omission constitutes an offence punishable under this Act and also under any other Central or State Act, then, notwithstanding anything contained in any other law for the time being in force, the offender found guilty of such offence shall beliable to punishment only under such Act as provides for punishment which is greater indegree.

CHAPTER-XVII
MISCELLANEOUS

96. Application of other laws not barred —

The provisions of this Act shall be in addition to, and not in derogation of, the provisions of any other law for the time being inforce.

97. Protection of action taken in good faith —

No suit, prosecution or other legal proceeding shall lie against the appropriate Government or any officer of the appropriate Government or any officer or employee of the Chief Commissioner or the State Commissioner for anything which is in good faith done or intended to be done under this Actor the rules made thereunder.

98. Power to remove difficulties —

(1) If any difficulty arises in giving effect to the provisions of this Act, the Central Government may, by order, published in the Official Gazette, make such provisions or give such directions, not inconsistent with the provisions of this Act, as may appear to it to be necessary or expedient for removing the difficulty:

Provided that no such order shall be made under this section after the expiry of the period of two years from the date of commencement of this Act.

(2) Every order made under this section shall be laid as soon as may be, after it is made, before each House of Parliament.

99. Power to amend Schedule —

(1) On the recommendations made by the appropriate Governmentor otherwise, if the Central Government is satisfied that it is necessary or expedient so to do, it may, by notification, amend the Schedule and any such notification being issued, the Schedule shall be deemed to have been amended accordingly.

(2) Every such notification shall, as soon as possible after it is issued, shall be laid before each House of Parliament.

100. Power of Central Government to make rules —

(1) The Central Government may, subject to the condition of previous publication, by notification, make rules for carrying out the provisions of this Act.

(2) In particular, and without prejudice to the generality of the foregoing power, such rules may provide for all or any of the following matters, namely: —

(a) the manner of constituting the Committee for Research on Disability under sub-section (*2*) of section 6;

(b) the manner of notifying the equal opportunity policy under sub-section (1) of section 21;

(c) the form and manner of maintaining records by every establishment under sub-section (1) of section 22;
(d) the manner of maintenance of register of complaints by grievance redressal officer under sub-section (3) of section 23;
(e) the manner of furnishing information and return by establishment to the Special Employment Exchange under section 36;
(f) the composition of the Assessment Board under sub-section (2) and manner of assessment to be made by the Assessment Board under sub-section (3) of section 38;
(g) rules for person with disabilities laying down the standards of accessibility under section 40;
(h) the manner of application for issuance of certificate of disability under sub-section (1) and form of certificate of disability under sub-section (2) of section 58;
(i) the allowances to be paid to nominated Members of the Central Advisory Board under sub-section (6) of section 61;
(j) the rules of procedure for transaction of business in the meetings of the Central Advisory Board under section 64;
(k) the salaries and allowances and other conditions of services of Chief Commissioner and Commissioners under sub-section (4) of section 74;
(l) the salaries and allowances and conditions of services of officers and staff of the Chief Commissioner under sub-section (7) of section 74;
(m) the composition and manner of appointment of experts in the advisory committee under sub-section (8) of section 74;
(n) the form, manner and content of annual report to be prepared and submitted by the Chief Commissioner under sub-section (3) of section 78;
(o) the procedure, manner of utilisation and management of the Fund under sub-section (2) of section 86; and
(p) the form for preparation of accounts of Fund under sub-section (1) of section 87.

(3) Every rule made under this Act shall be laid, as soon as may be after it is made, before each House of Parliament while it is in session, for a total period of thirty days which may be comprised in one session or in two or more successive sessions, and if, before the expiry of the session immediately following the session or the successive sessions aforesaid, both Houses agree in making any modification in the rule or both Houses agree that the rule should not be made, the rule shall thereafter have effect only in such modified form or be of no effect, as the case may be; so, however, that any such modification or an nulment shall be without prejudice to the validity of any thing previously done under that rule.

101. Power of State Government to make rules —

(1) The State Government may, subject to the condition of previous publication, by notification, make rules for carrying out the provisions of this Act, not later than six months from the date of commencement of this Act.

(2) In particular, and without prejudice to the generality of foregoing powers, such rules may provide for all or anyof the following matters, namely:—
(a) the manner of constituting the Committee for Research on Disability under sub-section (2) of section 5;
(b) the manner of providing support of a limited guardian under sub-section (1) of section 14;
(c) the form and manner of making an application for certificate of registration under sub-section (1) of section 51;
(d) the facilities to be provided and standards to be met by institutions for grant of certificate of registration under sub-section (3) of section 51;
(e) the validity of certificate of registration, the form of, and conditions attached to, certificate of registration under sub-section (4) of section 51;
(f) the period of disposal of application for certificate of registration under sub-section (7) of section 51;
(g) the period with in which an appeal to be made under sub-section (1) of section 53;
(h) the time and manner of appealing against the order of certifying authority under sub-section (1) and manner of disposal of such appeal under sub-section (2) of section 59;
(i) the allowances to be paid to nominated Members of the State Advisory Board under sub-section (6) of section 67;
(j) the rules of procedure for transaction of business in the me etings of the State Advisory Board under section 70;
(k) the composition and functions of District Level Committee under section 72;
(l) salaries, allowances and other conditions of services of the State Commissioner under sub-section (3) of section 79;
(m) the salaries, allowances and conditions of services of officers and staff of the State Commissioner under sub-section (3) of section 79;
(n) the composition and manner of appointment of experts in the advisory committee under sub-section (7) of section 79;

(o) the form, manner and content of annual and special reports to be prepared and submitted by the State Commissioner under sub-section (3) of section 83;
(p) the fee or remuneration to be paid to the Special Public Prosecutor under sub-section (2) of section 85;
(q) the manner of constitution of State Fund for persons with disabilities under sub-section (1), and the manner of utilisation and management of State Fund under sub-section (2) of section 88;
(r) the form for preparation of accounts of the State Fund for persons with disabilities under sub-section (3) of section 88.

(3) Every rule made by the State Government under this Act shall be laid, as soon as may be after it ismade, before each House of the State Legislature where it consists of two Houses, or where such State Legislature consists of one House, before that House.

102. Repeal and savings —

(1) The Persons with Disabilities (Equal Opportunity Protection of Rights and Full Participation) Act, 1995 (1 of 1996) is hereby repealed.

(2) Notwithstanding the repeal of the said Act, anything done or any action taken under the said Act, shall be deemed to have been done or taken under the corresponding provisions of this Act.

1. Physical disability —

THE SCHEDULE
[*See* clause (zc) of section 2]
SPECIFIED DISABILITY

A. Locomotor disability (a person's inability to execute distinctive activities associated with movement of self and objects resulting from affliction of musculoskeletal or nervous system or both), including —
(a) "leprosy cured person" means a person who has been cured of leprosy but is suffering from —
(i) loss of sensation in hands or feet as well as loss of sensation and paresis in the eye and eye-lid but with no manifest deformity;
(ii) manifest deformity and paresis but having sufficient mobility in their hands and feet to enable them to engage in normal economic activity;
(iii) extreme physical deformity as well as advanced age which prevents him/her from undertaking any gainful occupation, and the expression" leprosy cured" shall construed accordingly;
(b) "cerebral palsy" means a Group of non-progressive neurological condition affecting body movements and muscle coordination, caused by damage to one or more specific areas of the brain, usually occurring before, during or shortly after birth;
(c) "dwarfism" means a medical or genetic condition resulting in an adult height of 4 feet 10 inches (147 centimeters) or less;
(d) "muscular dystrophy" means a group of hereditary genetic muscle disease that weakens the muscles that move the human body and persons with multiple dystrophy have incorrect and missing information in their genes, which prevents them from making the proteins they need for healthy muscles. It is characterised by progressive skeletal muscle weakness, defects in muscle proteins, and the death of muscle cells and tissue;
(e) "acid attack victims" means a person disfigured due to violent assaults by throwing of acid or similar corrosive substance.

B. Visual impairment —
(a) "blindness" means a condition where a person has any of the following conditions, after best correction —
(i) total absence of sight; or
(ii) visual acuity less than 3/60 or less than10/ 200 (Snellen) in the better eye with best possible correction; or
(iii) limitation of the field of vision sub tending an angle of less than 10 degree.
(b) "low-vision" means acondition where a person has any of the following conditons, namely :—
(i) visual acuity not exceeding 6/18 or less than 20/60 upto 3/60 or upto 10/200 (Snellen) in the better eye with best possible corrections; or
(ii) limitation of the field of vision subtending an angle of less than 40 degree upto 10 degree.

C. Hearing impairment —
(a) "deaf"means persons having 70 DB hearingloss in speech frequencies in both ears;
(b) "hard of hearing" means person having 60 DB to 70 DB hearing loss in speech frequencies in both ears;

D. "speech and language disability" means a permanent disability arising out of conditions such as laryngectomy or aphasia affecting one or more components of speech and language due to organic or neurological causes.

2. Intellectual disability, a condition characterised by significant limitation both in intellectual functioning (reasoning, learning, problem solving) and in adaptive behaviour which covers arange of everyday, social and practical skills, including —
(a) "specific learning disabilities" means a heterogeneous group of conditions where in there is a deficit in processing language, spoken or written, that may manifest itself as a

difficulty to comprehend, speak, read, write, s pell, or to do mathematical calculations and includes such conditions as perceptual disabilities, dyslexia, dysgraphia, dyscalculia, dyspraxia and development alaphasia;

(b) "autism spectrum disorder" means a neurodevelopmental condition typically appearing in the first three years of life that significantly affects a person's ability to communicate, understand relationships and relate to others, and is frequently associated with unusal or stereotypical rituals or behaviours.

3. Mental behaviour, —
"mental illness" means a substantial disorder of thinking, mood, perception, orientation or memory that grossly impairs judgment, behaviour, capacity to recognise reality or ability to meet the ordinary demands of life, but does not include retardation which is a conditon of arrested or incomplete development of mind of a person, specially characterised by subnormality of intelligence.
4. Disability caused due to —
   (a) chronic neurological conditions, such as —
      (i) "multiple sclerosis" means an inflammatory, nervous system disease in which the myelin sheaths around the axons of nerve cells of the brain and spinal cord are damaged, leading to demyelination and affecting the ability of nerve cells in the brain and spinal cord to communicate with each other;
      (ii) "parkinson's disease" means a progressive disease of the nervous system marked by tremor, muscular rigidity, and slow, imprecise movement, chiefly affecting middle-aged and elderly people associated with degeneration of the basal ganglia of the brain and a deficiency of the neurotransmitter dopamine.
   (b) Blood disorder —
      (i) "haemophilia" means an inheritable disease, usually affecting only male but transmitted by women to their male children, characterised by lossor impairment of the normal clotting ability of blood so that a minor would may result in fatal bleeding;
      (ii) "thalassemia" means a group of inherited disorders characterised by reduced or absent amounts of haemoglobin.
      (iii) "sickle cell disease" means a hemolytic disorder characterised by chronic anemia, painful events, and various complications due to associated tissue and organ damage; "hemolytic" refers to the destruction of the cell membrane of red blood cells resulting in the release of hemoglobin.
5. Multiple Disabilities (more than one of the above specified disabilities) including deaf blindness which means a condition in which a person may have combination of hearing and visual impairments causing severe communication, developmental, and educational problems.
6. Any other category as may be notified by the Central Government.

Case Report

Woes of Domestic Violence: An Intervention Based Case Report on Complex Post Traumatic Stress Disorder

Akanksha Arora,[1] Prerna Sharma,[2] Varun Kalakoti,[3] Anuj Mittal[4]
[1,2]Amity Institute of Behavioural (Health) and Allied Sciences, Amity University Uttar Pradesh, Noida
[3,4]Deen Dayal Upadhyay Hospital, New Delhi
Contact: Anuj Mittal, E-mail: drmittalanuj@gmail.com

Introduction

According to ICD-11 complex post-traumatic stress disorder (CPTSD) is a new disorder that describes the more complex reactions that are typical of individuals exposed to chronic trauma.[1] Involving long histories of clinical observation of individuals who experience chronic, repeated and prolonged traumas, such as childhood sexual abuse or domestic violence, they tend to experience more complex reactions extending beyond those typically observed in PTSD and include disruptions in emotion regulation, self-identity and relational capacities.[2]

Domestic violence is a pattern of abusive behaviour in any relationship that is used by one partner to gain or maintain power and control over another intimate partner.[3] Domestic violence can be physical, sexual, emotional, economic, psychological, or technological actions or threats of actions or other patterns of coercive behaviour that influence another person within an intimate partner relationship.[4,5]

This research article highlights a case report of a women suffering from domestic violence and thereby developing C-PTSD (Complex Post-Traumatic Stress Disorder).

ICD-11[1] states that complex PTSD develops after exposure to a traumatic event or a sequence of severely frightening experiences that are typically lengthy or recurring and from which escape is typically difficult or impossible. CTPD is diagnosed when the patient experiences all the core symptoms of PTSD. In addition, Complex PTSD is characterized by having severe and pervasive problems in affect regulation, persistent beliefs about oneself as diminished, defeated or worthless, accompanied by deep and pervasive feelings of shame, guilt or failure related to the traumatic event and persistent difficulties in sustaining relationships and in feeling close to others. The disturbance causes significant impairment in personal, family, social, educational, occupational or other important areas of functioning. Management of CPTSD involves a combination of pharmacotherapy and psychotherapy for efficient results in the patient's overall functioning.[6]

Case History

Ms. G, 26-year-old, divorced female, a homemaker, hailing from a Hindu middle socioeconomic strata, living in a nuclear family, presented with complaints tracing back 4.5 years ago to a traumatic experience with her marriage involving domestic violence. Early in the marriage, Ms. G experienced unwelcoming attitude from the husband and in-laws with frequent arguments leading to physical and verbal abuse. She reported being increasingly anxious, experiencing increased heart rate and palpitations; feeling of disgust, negative ideations about self, easily frightened, feeling low on mood and energy, feelings of helplessness. She experienced a difficult time during and post her pregnancy physically and mentally; nausea, palpitations, breathlessness, bp fluctuations, crying spells, negative ideations, death wishes for the baby, increased discomfort and family's unsupportive attitudes. Information from the patient and family revealed a collaborative decision for filing a divorce case due to the patient feeling vulnerable having increasing fear of death, fear of harm or injury to her and the baby, helplessness, hopelessness and frequent nightmares.

Taking note of the birth history indicated no significant complications reported during the pregnancy, delivery, or the post-natal period. No significant delay in developmental milestones was confirmed. The patient reported herself to be an anxious person as a child, often finding it difficult to open up and socialize. She liked to stay indoors. She liked going to school, reading and meeting her few close friends. Tying the knot in an arranged setup at the age of 21 years, the patient reported intimacy issues and diminished emotional connect to the husband, in line with the experience of domestic violence.

The premorbid personality of Ms. G indicated anxious traits, with a tendency to overthink and analysing everything.

Clinical Features

Ms. G, at the time of referral, presented with complaints of experiencing nightmares, scary flashbacks with vivid movie like scenes, intruding thoughts, constant anxiety while mobilizing. She reported feeling breathless, low on mood and self-confidence, anhedonia, having trust issues, developing avoidance. She mentioned memory difficulties, deteriorating self-care and weight gain. The complaints were subsequently in line with patient's life after divorce. The onset of her symptoms was insidious, with a continuous course and deteriorating progress.

Mental Status Examination

Ms. G was well-kempt and tidy; she was slouching while sitting and sobbing. Eye contact was established a little late but was later maintained. Rapport was established a little late. Psychomotor activity was within normal range and she was cooperative. Speech was coherent, relevant and goal directed with adequate reaction time.

Mood was depressed and affect was congruent to mood. No perceptual disturbances were elicited. Thought patterns revealed feelings of helplessness, guilt and low self-esteem.

Ms. G was oriented to time, place and person. Attention and concentration aroused and sustained. Her memory was intact with her having adequate fund of knowledge. She maintained conceptual level of abstraction, impaired judgment and an insight level of 4.

Treatment History

There was no past psychiatric or medical history reported. The patient at the time of referral was prescribed Escitalopram (10 mg) plus clonazepam (0.5 mg), Fluoxetine 20 mg, Propranolol 20 mg, Amitriptyline 25 mg and Clonazepam 0.5 mg to alleviate symptoms of anxiety and depression. Ms. G was also recommended to psychotherapy along with drug therapy.

Psychological Assessment

PTSD Symptom Scale–Interview (PSS-I) to assess the presence and severity of PTSD symptoms,[7] Hamilton Depression Rating Scale (HAM D) to assess the severity of depressive symptoms[8] and Hamilton Anxiety Rating Scale (HAM A) to assess the severity of anxiety symptoms.[9]

On PSS-I, Ms. G received a score of 46, indicative of high severity of Ptsd symptoms, with presence of high amount of distress, interfering with everyday life. On HAM-D, she received a score of 30, which is indicative of moderate Severity. On HAM-A, she received a score of 25, which is indicative of severe anxiety.

Intervention

The goals of the intervention focused on providing the patient with capabilities to work on coping strategies and address the underlying trauma that lead to CPTSD and help the patient to recover in overall daily life functioning. An integrative approach was applied for the index patient involving psycho-education, supportive psychotherapy, relaxation, behavioural activation, EMDR.

Psychoeducation is a systematic, structured and didactic transfer of knowledge about an illness and its treatment, integrating emotional and motivational aspects to enable patients to cope with the illness and to improve its treatment adherence and efficacy.[10] The patient and the family members were psycho-educated about the disorder, its symptoms, treatment, how to deal with this disorder, and how these processes will progress to the family as well.

The family was also psycho-educated about the need of a positive and stimulating family environment. Techniques were practiced with them to reinforce positive changes, managing medication compliance and adherence, importance of timely

consultations and providing complete up to date information. No suicide contract was signed and the resources were duly informed and kept in loop.

Relaxation techniques involving deep breathing, 4-7-8 breathing were practiced with the patient to deal with anxiety, restlessness and overthinking. the patient was instructed to practice relaxation all through the tenure of therapy as apart of her daily life activity schedule.

Supportive psychotherapy, that uses direct measures to ameliorate symptoms and to maintain, restore, or improve self-esteem, ego functions, and adaptive skills,[11] was provided to the patient. A strong therapeutic alliance was at the focus, along with encouragement to vent emotions and techniques of positive enforcement.

Behavioural activation and goal setting was achieved through activity scheduling,[12] helping Ms. G gain mastery and pleasure in activities of daily life. Starting with very small goal of maintaining daily self-care routine, we progressed towards adding her participation in home chores and family interactions. Once maintained, little additions to her leisure activities were included in everyday routine to maintain pleasure.

EMDR (eye movement desensitization and reprocessing) can help you process upsetting memories, thoughts, and feelings related to the trauma. By processing these experiences, you can get relief from CPTSD symptoms.[2] The first few sessions of EMDR therapy involved grounding techniques and coming up with a safe space in the client's mind to face up the trauma she experienced. A timeline was worked through for key traumatic events that needed to be worked upon. The client was advised to keep contact with some supportive alliance which in the client's case was her friend. With subsequent sessions, target memories and thoughts were worked upon.

Progress and Outcome

Ms. G was not complaint to medications which proved to be a major hinderance in the maintenance of normal functioning at home and in society. Psychotherapy was effective once the patient was compliant. She reported being able to effectively deal with issues and not feeling stuck up. She was able to consider having perspective on things and understand her feelings and accept them better. She was able to process the traumatic events with better regulation of thoughts, emotions and behaviour.

Discussion

C-PTSD is a mental health condition when someone has experienced a traumatic event. C-PTSD shares many symptoms in common with PTSD, including re-experiencing, avoidance, and hyper-arousal, as described above. However, C-PTSD also includes problems in emotion regulation, self-image and interpersonal problems.[14]

As in this case, Ms. G was diagnosed with CPTSD following the experiences of traumatic marriage and divorce and the life after. She suffers from experiencing nightmares, scary flashbacks with vivid movie like scenes, intruding thoughts, constant anxiety while mobilizing, breathlessness, poor self-image, anhedonia, trust issues, developing avoidance. Her symptomatology being more chronic than ptsd puts PTSD as a consideration for differential diagnosis.

For the management of symptoms, pharmacotherapy and psychotherapy were utilized. psychoeducation was provided to the patient and family members at the start of therapy and during sessions as and when necessary. Psychotherapy included supportive therapy, relaxation and behavioural activation and EMDR.[14]

Ms. G, in the initial stages was not compliant to the medications and therapy which was a major hinderance in the treatment process. Later, when compliance was achieved, drug therapy and psychotherapy were proved effective.

CPTSD can be effectively treated with pharmacotherapy and psychotherapy provided a good doctor-patient relationship is established and adverse medication effects are effectively dealt with.[15] Utilizing a biopsychosocial approach is essential so as to integrate all aspects of the patients' history in a meaningful way in order to provide adequate and timely help.

Informed Consent

Ethical consideration were considered crucial i.e. informed consent and confidentiality. Consent of the participant was taken for the publication of this case report.

Data which can reveal the identity of the client has not been provided here.

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Case Report

Non-communicating Hydrocephalus in Kartagener Syndrome – Case Report

M.S. Bhatia, Bushra Zahoor, Dimple Gupta, Sandeep Sekhon, Nimmi A. Jose
Department of Psychiatry, HIMSR & HAHC Hospital, Guru Ravidas Marg, New Delhi-110062

Kartagener syndrome (KS), also known as primary ciliary dyskinesia, refers to the triad of Situs inversus, sinusitis and bronchiectasis, infertility in males (due to immotile sperms) and females can also be seen. The Incidence ranges from 1 in 20,000 to 1 in 60,000.[1] Only half the patients present with all the symptoms. viz., situs inversus, infertility, sinusitis, and bronchiectasis, a condition designated as complete KS, compared with incomplete KS, typically defined as cases in which situs inverus does not occur.[2] Associations with Polysplenia, asplenia, and congenital heart diseases have been described. It is autosomal recessive. The severity of symptoms can vary for each person, but they usually start at birth.[3]

A Kartagener syndrome diagnosis starts with a physical exam and history of typical symptoms. If one suspects diagnosis of KS, one can go for various tests. These include: Electron microscopy where they take a tissue sample or scrape your nose and look at how the cilia move; X-rays to look at your lungs and internal organs; Computed tomography (CT) scan; Nitric oxide test which measures how much nitric oxide one breathes out, typically lower in people with ciliary problems and hearing tests.[3-5]

Common differential diagnosis include: Cystic fibrosis; Immunodeficiency, such as immunoglobulin G (IgG) subclass deficiency; Allergic rhinitis; Gastroesophageal reflux disease; Wegener's granulomatosis (upper- and lower-airway disease).

Specific Kartagener syndrome treatment includes: Regular sinus washes; Regular ear canal washes; Steroids; Mucus thinners, called mucolytics; Antibiotics; Bronchodilators to relax lung muscles for easier breathing; Ear tubes; Speech therapy; Hearing aids; Heart surgery for defects; Lung transplant in severe cases.[3-5]

Hydrocephalus may occur on rare occasion in individuals with primary ciliary dyskinesia and may reflect dysfunctional ependymal cilia.[7-9] We present a case presenting with persistent headache who on detailed history and investigations found to be suffering from Kartagener syndrome and hydrocephalus.

Case Report

A 39 year old male presented with persistent frontal headache and dizziness. Headache was mild to moderate and was throbbing at times. Initially, it was relieved on taking paracetamol but later on becomes unresponsive. There was no nausea or visual disturbances. On detailed history there were recurrent episodes of coryza, nasal congestion, facial pain and primary infertility and anxiety symptoms for the last 4 years.

History of the Present Illness

There were complaints of recurrent episodes of nasal congestion, facial pain and frontal headache. He got married but inability to yield a child that led to recurrent episodes of palpitations, choking sensation, and a feeling of intense fear; each attack lasting 3 to 4 minutes. He also sought a surgeon's opinion regarding mild chordee he had developed, which however did not interfere with his ability to perform sexual intercourse. Semen analysis revealed oligospermia and immotile sperm ranging from 80% to 95%.

The patient was referred to the medicine department for some somatic complaints wherein a detailed

work up of the patient was sought including an ultrasonogram of whole abdomen and ECG.

Negative History

No history of having burning micturition was present. Pedigree analysis did not reveal any positive case. Thus the patient came to us with numerous reports showing oligospermia and immotile sperms. ENT diagnosis was of recurrent facial and maxillary sinusitis, X ray chest PA view with the radiologist's opinion showed wrong placement of the markers, and there were features of generalized anxiety disorder.

Examination

Psychiatric evaluation and Medical examination showed sinus tenderness and normal size of testis. The secondary sexual characters were well developed.

Investigations

What was initially passed off as 'wrong placement of the marker' on the chest X ray was confirmed on EKG findings to be 'dextrocardia' EKG showed right axis deviation, no progressive increase in R wave height across the chest leads, and inverted P wave in lead I. Chest roentgenogram then made sense with the Aortic knuckle and stomach bubble on the right side; and the right hemidiaphragm lower than the left.Lung fields and costodiaphragmatic and the costophrenic angles were clear. No changes like increased bronchial markings or any change consistent with the destruction of the lung parenchyma were noted.

An X ray for paranasal sinuses revealed bilateral frontal and maxillary sinusitis.

Whole abdomen ultrasonogram confirmed 'Situs inversus', with the liver and the gallbladder visualized in the left hypochondrium, and the spleen and the pancreas in the right. The Aorta and the Inferior vena cava were also transposed. No evidence of Polysplenia was seen.

Semen analysis revealed a count of 5 million/ cumm, with a remarkable 82% immotile sperms, and 13% and 5%, sluggish and active sperms, respectively. In addition, there were 50 to 60 pus cells per cumm. However, repeated AFB stain for mycobacterium tuberculosis and cultures yielded no positive results. VDRL for Syphilis and HIV ELISA were also negative. Bilateral testicular aspirate revealed normal histology and spermatogenesis.

Transrectal ultrasonogram revealed grade I prostate hyperplasia Hormone assay for FSH yielded a higher value of 14.5 mIU/ml (normal value: 1.5 to 12.4 mIU/ml), raised LH (13.41 U/L; normal: 1.7 to 8.6U/L). Tesosterone and prolactin levels were with in the normal range. Echocardiogram was normal.

He was initially diagnosed as having Psychogenic headache but on the suggestion of a neurosurgeon, a MRI brain was done which revealed marked dilatation of lateral ventricles, foramina of Monroe and third ventricle with subtle thinning of cerebral parenchyma, suggestive of non-communicating hydrocephalus (Figure).

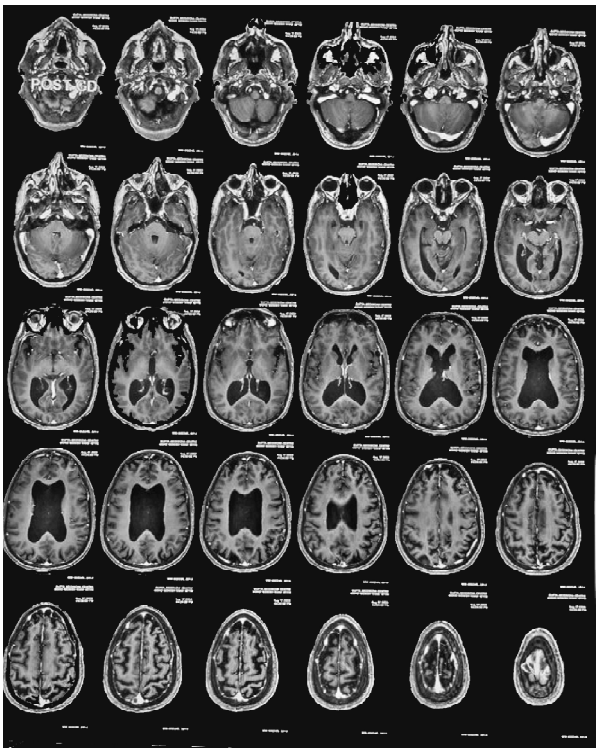

Fig. MRI Scan (Brain) showing hydrocephalus

Discussion

Atypical nature of the case is highlighted by the following points.

1. Hydrocephalus has been reported as an uncommon presentation. If patient presents with persistent headache or dizziness, MRI/ CT Scan is warranted.
2. Although the presence of immotile sperms

is seen in many cases of Primary Ciliary Dyskinesia, Oligospermia has not been reported with it.

3. This is a variant from what most incomplete KS cases are; as already incomplete KS cases are classically defined as those which do not have situs inversus. So neither does this patient classify as a complete KS case as he did not have the classic triad of KS (he had no evidence of recurrent pneumonias or bronchiectasis), nor is he a typical incomplete KS (as the presence of situs inversus defies the definition of incomplete KS).
4. Deranged Hormone profile; cause for oligospermia could not be discerned.
5. Patient had symptoms of Generalized Anxiety Disorder with Primary Infertility as the underlying stressor, as incriminated by the patient and as was also evident from the complete Psychiatric work up of the patient.

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Case Report

ARFID: A Case Report

Sana Usmani, Deepak Gupta, Nebanita Sengupta
Pediatric Psychiatry Services, Institute of Child Health, Sir Ganga Ram Hospital, New Delhi
Contact: Sana Usmani, Email: sana.usmani.psy@gmail.com

Introduction

Avoidant/Restrictive Food Intake Disorder (ARFID), is a complex and heterogeneous disorder, which can present with symptoms including lack of interest in eating sensory sensitivity and fear of aversive consequences.[1,2]

Individual case reports/series have shown promising approaches for older children, adolescents, and adults with ARFID, using as a base either family-based treatment (FBT),[3-7] cognitive behavioral therapy (CBT);[8,9] or other novel approaches.[10] Despite varied approaches being tried and studied, no published, randomized controlled trials have evaluated their efficacy in the treatment of ARFID.[2] There is a deficiency in addressing the concurrently existing high rates of comorbid mood and anxiety disorders in patients with ARFID in the existing treatment models.[11,12]

There are lacunae in diagnosis of ARFID in children below 5 year of age, in this case report we present the case of a 4 year old male presenting with food avoidance, highlighting on the diagnostic challenges along with presenting novel treatment approach which keeps the emotional and psychological needs of the child along with the symptoms of ARFID.

Case Report

Patient Y is a 4-year-old male; he was born at 37th week by C-section. He has no history neurodevelopmental disorder. All motor mile stones were achieved on time. At the age of 1 year 5 months, Y had complains of vomiting after feeding for which the parents took him to a doctor on examination he was found to have a tongue tie for which he was operated, this helped in his speech clarity but did not solve the presenting complains. The complaints persisted, parents would try to feed him but the child would refuse feed and would regurgitate what was fed to him. He would be extremely irritable and sad during feeding. It would take hours to feed him one meal which he, he would cry scream and refuse food at all times in any form. Parents would have to force feed him mainly liquid diet. At the age of 2 years an endoscopy was done but no abnormality or pathology was detected. In the dietary history it was seen that the child was on cow milk for the first six months which led to constipation and bloating. From 10 months of age formula feed and buffalo milk was started which the child vomited after feed. Bloating with constipation was a constant issue. The parents went to various doctors who advised them to start normal food so that constipation is resolved which the parents tried to start but the child would refuse to eat. The child was still unwilling to eat anything, parents would have to force feed him liquid diet. When solid food was given the child did not chew and would take it out of his mouth. Even after liquid diet child would end up vomiting majority of it. For 3 years parents went to multiple doctors across cities but neither a diagnosis was made nor was the issue resolved. In November 2023 child was given tablet Risperidone 0.5 mg half tablet by a doctor to help with the irritability. Parents gave the same for 2 months but noticed no change. Following which tablet Aripiprazole 1.25mg was started which was given only for 2 days as the child had fainting episodes and even refused to drink water so the medication was stopped. Parents even tried therapy 15 sessions of occupational and sensory therapy but

it did not help either. His recent investigations revealed decreased Vitamin D and Ferritin levels.

According to the parents child was willing to go to school and performed well there. He was made the class monitor which reflects that the child had no problems in social or educational fields. Parents did complain of weak immunity that could be a result of insufficient diet.

Discussion

The introduction of the ARFID diagnosis in the DSM-5 is a pivotal advancement in achieving diagnostic criteria and improving the recognition of patients who had previously been marginalized in clinical settings. ARFID is a challenging disorder with various presentations and aspects, making its diagnosis and treatment a multidisciplinary issue. In our case report the patient and the family member had visited multiple doctors and underwent varied procedures and yet faced disappointment, thus showing the rarity of diagnosis of ARFID in a 4 year old. The treatment modalities for ARFID include pediatric behavioral therapy, dietary plans, along with family and group therapy. In few cases psychopharmacology has been. CBT-based approaches[13-18] have also shown decrease in symptom severity, intensity of food phobia and family accommodation.[14,15] Since the treat modalities are varied and not specified, for our case keeping in mind the severity of symptoms and parents anguish, we started the patient on psychopharmacology, play therapy and dietary therapy. The modality chosen for the child among the array of options was based on studies showing that treatments have been effective in increasing food variety[13] and achieving a healthy body weight and nutritional intake.[15] Moreover other studies confirm these results,[19-21] and have shown improvement even in psychosocial functioning, yet seeing their effect on a 4 year child would be noteworthy. While interceptive exposure could decrease ARFID symptoms and increase general self-regulation skills,[17] extinction-based treatment and exposure to disgust treatment should still be further investigated and none of them could be used in our case as the age of child was less and such measures have not been tried often.[22,23]

Studies that have investigated the effects of family-based treatment have found that it is applicable to various clinical presentations of ARFID with a positive effect on symptoms and weight gain.[24-27] FBT is also highly acceptable for families and better than usual care.[28] FBT has been identified as a promising area to investigate further in the ARFID context.[29] Le Grange et al.[28] in their work made a comparison between CBT-E and FBT and found that FBT improved weight gain better than CBT-E, but not at follow-up. However, Rienecke et al.[30] state that different ARFID subtypes benefit more from different strategies. It seems that patients with limited intake benefit most from FBT, while aversive patients benefit most from CBT for anxiety. Finally, patients with a limited variety of foods benefit more from multidisciplinary behavioral interventions. After all the reviews and evidence based examples FBT would be one of our chosen modality after the child's eating habits are improved and is easier to handle by the parents.

There are currently no specific pharmacological treatments approved for ED. The only exception is fluoxetine for bulimia nervosa. ARFID patients are treated for the concomitant disease that exist with ARFID instead of the disorder itself or are treated with drugs which have been shown to have positive effects on ED.[31] Dolman et al.[32] for example, in their work, treated a young patient with olanzapine and sertraline in order to manage anxiety, nausea, and to stimulate appetite. The authors have combined this pharmacological treatment with CBT and FBT with good results.[32] With these reviews in mind and considering the age of the patient we started him on Olanzapine 1.25 mg half tablet twice day. We also added an appetite stimulant along with a probiotic in 100 ml water in the evening to help with constipation and also help improve sleep. Lansoprazole was given 15 mg tablet once a day empty stomach this was added keeping in mind the frequent reflux episode the child had after feeding. For therapy we started with play therapy since the child is merely 4 years of age any other therapy would be difficult to conduct. Hence play therapy was chosen so that it would help relax the child and it would also help in him verbalize his emotions. For dietary advice parents were attached to a dietician. This was important as child with ARFID can suffer from deficiencies which would in turn affect the growth and overall development of the child.

Future Directions

Due to its multifaceted nature, ARFID is a complex disorder that requires polytherapeutic treatment involving a multidisciplinary team working and collaborating for the well-being of the patient. ARFID's various presentations and comorbidity may require psychotherapy, a dietetic approach, and FBT. In some cases, psychopharmacology can be very useful but should be carefully considered. A multi-dimensional approach proves to be the best choice, as it allows for due consideration to be given to many aspects of the disease that affects not only the patient, but also the family.

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Case Report

ADHD and Conversion Disorder

Sana Usmani, Deepak Gupta, Nebanita Sengupta
Pediatric Psychiatry Services, Institute of Child Health, Sir Ganga Ram Hospital, New Delhi.
Contact: Sana Usmani, Email: sana.usmani.psy@gmail.com

Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is often comorbid with other disruptive behavior disorders, mood disorders, stress/anxiety, and addictive disorder. Presentations are often complicated by environmental issues including parental discord, family situation, parenting and exposure to trauma.[1]

A large proportion of children and adolescents with ADHD have at least one comorbid psychiatric disorder.[2] It has also noted that children and adolescents with ADHD generally have varied medical disorders and physical symptoms.[3]

Here we are presenting a case of conversion disorder with comorbid ADHD in a 12-year-old male highlighting the diagnostic and therapeutic challenges in these comorbidities due to overlapping clinical presentations of these conditions.

Case Report

Master V a 12-year-old male was born at term following an uneventful pregnancy. He has one elder sister who has nohistory of any neurodevelopmental disorder. All domains of development were appropriate for his and also had good academic achievement. V presented with complains of persistent unresponsive episodes he was apparently doing well till March 2023, when he started having unresponsive episodes (Frequency: 6-9 per day; Type: No stiffening of limbs, jerky movements, up rolling of eyes, incontinence, tongue bite, frothing; Duration: several seconds to 20 minutes) there was closing of eyes with hyperventilation followed by V coming back to consciousness and behaving normally, there was also increased irritability along with school absence. V was prescribed Antidepressants, Benzodiazepines and advised Individual Therapy sessions. Stressors were tried to be explored, but V would not sit for therapy, showed restlessness and would run out of the sessions.

The episodes still continued, fluctuating only in frequency (from 7 times in a day to >20 times on some days). V started complaining new symptoms which were polymorphic in nature, there were complains of frequent hand washing, checking of locks and fear that someone will harm him. They appeared in a few weeks difference to one another. But none of the complaints met the criteria of OCD or prodromal psychosis. These complaints lasted for several days to few weeks only to disappear on their own. V had stopped going to school in this interim.

V was admitted in the month of Jan 2024 and the past history was explored. It was then found that as a preschooler V was noted to be stubborn, showed temper tantrums, demanding, talkative and hyperactive. Complains from school were of V not sitting in his place and moving around the class, not paying attention and easy distractibility during classes. Even in social settings, V would show increased activity compared to the children around, he would be impatient about waiting at places and would interrupt conversations for his own needs, because of these reasons parents would have to constantly supervise his whereabouts, but these behavior patterns was never looked into and there was no formal diagnosis made. There were also parenting difficulties noted in the history, V and his parents had a communication gap, their expression of care and attention were not the same. There was also a temporal association of V facing problems in school with his classmates and

the onset of the unresponsive episodes. However the academic remained reasonable and teachers were happy with performance. On examination he was extremely restless with frequent leg shaking he had several unresponsive episodes during the interview and would complain of headache when he did not want to answer to the questions, would frequently shift place on the bed, had shifting gaze, decreased concentration in the interview and was suggestible to distractions in room. His blood investigations and Contrast Enhanced Magnetic Resonance Imaging of Brain were normal.

During hospitalization, he was prescribed Methylphenidate 18 mg once a day, and it was seen that there was a decrease in the frequency of his episodes also better interaction was noted with the parents and doctors. There was decrease in unresponsive episodes drastically. His psychomotor activity touched baseline and he would sit comfortably through individual counseling therapies, concentration had improved and his understanding of his immediate environment along with interaction with parents became more effective. V was discharged on the same medications as he showed improvement and is currently maintaining well.

Discussion

The reported prevalence and incidence of attention-deficit hyperactivity disorder (ADHD) has increased in recent years despite differences in rates between countries.[4] This case in particular is a mix of family and individual challenges which made the diagnosis unclear initially. Due to absence of a reliable biological marker for ADHD, the diagnosis completely relies on thorough neurodevelopmental history, longitudinal history of the patient's functioning seen over a course of time in varied situations, information from reliable sources, teacher- and parent-reported behaviour rating scale scores (based on ICD-10 and DSM-5 criteria), along with clinical skills.[5,6] A bio-psychosocial approach to understand the problems would be helpful in not only making appropriate diagnosis but also the planning appropriate management.[7,8] In DSM-5 the presence of inattentive and hyperactive-impulsive symptoms have to be present before the age of 12 years for a diagnosis of ADHD, there has also been mention of adolescent or adult-onset ADHD.[5,9]

The existing association between of ADHD and epilepsy is well documented.[10] However, the association of ADHD and pseudo-seizure appears to be less explored. In our patient, who had certain psychosocial challenges like school performance, difficulties in communication with parents along with poor parenting, conversion episodes appeared to be maladaptive coping mechanism. Factors like reasonable academic achievements and less knowledge about ADHD among teachers and family members were the barriers from referring him earlier. Conversion disorder are known to be higher in patients with family conflict, in families with poor communication or when patients feel that family members are less interested in their activities and values,[11] which in our case were the etiological factors. It is interesting to note that 60% of patients with conversion episodes learning problems as well,[11] this factor needed to be explored more to see if our patient was facing difficulties in academics cause of learning difficulties. Also there is a dilemma of using stimulants in patient's presenting with ADHD and seizures.[12]

In our patient the use of methylphenidate played a significant role. It helped in combating the symptoms by addressing the underlying ADHD. It also reconfirmed the diagnosis of ADHD presenting as conversion disorder due to inability to keep up with increasing psychosocial and educational demands. Our patient showed good improvement and is maintaining well on them. He has been attending, eye movement desensitization and reprocessing (EMDR) sessions whereas his parents are on family counselling and parent training. With this multimodal management we have not only relieved his symptoms but also addressed the bio-psychosocial stressors. All of this together led to a positive outcome.

Future Directions

Although it has been observed that conversion disorder can be comorbid with ADHD, further epidemiologic studies are needed to help in early diagnosis. There is need of specific diagnostic criteria particularly designed for this group of children to guide the clinicians to delineate these two overlapping conditions. Further studies need to be done on different medical modalities for treatment of conversion disorder in ADHD.

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Book Review

Short Textbook of Psychiatry

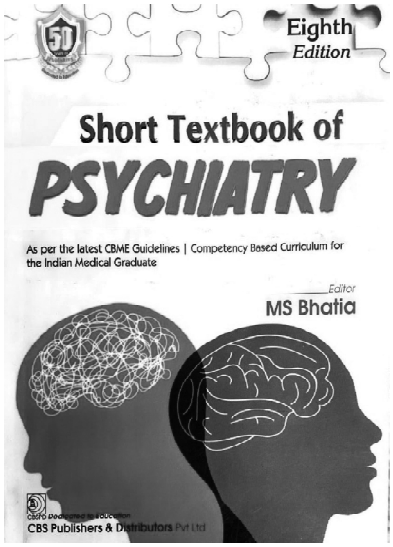
8th edition by CBS Publishers and Distributors Private Limited
Edited by Dr M.S. Bhatia

Dr M.S. Bhatia is currently Professor and Head Department of Psychiatry, Hamdard Institute of Medical Sciences and Research, New Delhi, Ex Professor University College of Medical Sciences and associated GTB Hospital, Delhi. He is an experienced senior teacher and examiner, associated with writing and editing of various articles, many popular books at undergraduate and postgraduate level for the students preparing for the competitive and university exams.

This book is a eighth edition as per latest CBME guidelines and Competency based Curriculum. It encompasses 32 chapters including Recent advances. Every chapter covers important aspects and has key points that are indicated in boxes which are important for viva-voce as well as MCQs. Every chapter is followed by review questions and professional exam questions of different Indian Universities.

This book is entirely focused on giving knowledge about different aspects and generating new skills. It is specifically prepared for easy comprehension of psychiatry in plain and clear language. Detailed chapters on historical aspects, symptomatology and examination have been included and there is an updated section on psychiatric treatments.Recent advances in diagnosis and treatment and new classifications (DSM 5 and ICD-11) have been elaborated. There is a list of psychotropic drugs in alphabetical order according to the trade name are included. Epidemiology of various psychiatric disorders, National Mental Health Programme, latest Acts on forensic psychiatry and cultural bound syndromes in India are added. Psychiatric glossary and psychological tests have been described in detail. Important appendices including management of overdose and toxicity of various psychotropic drugs and psychiatric quiz (based on difficult MCQ) have been added.

This book is also an attempt to fill some of the gaps in the field of psychiatric writing. A sincere



effort has been made to make this book simple, easy, comprehensive and practically oriented. Many available textbooks do not contain the cornerstone of Psychiatry and are devoted to the psychiatric practice in Western countries. This book lays emphasis on important topics related to Indian psychiatry topics such as epidemiology, forensic psychiatry, mental health program of India, contribution of Indian psychiatrists and also the important subspecialities of psychiatry such as Child Psychiatry, Psychiatry related to women, Community psychiatry. So this book is a sincere effort on psychiatric practice as applicable to Indian setting.It is a popular book for paper setting in undergraduate and postgraduate examinations.

Reviewers
Dimple Gupta
Sandeep Sekhon
Bushra Zahoor
Department of Psychiatry
HIMSR & HAHC Hospital
Guru Ravi Das Marg,
New Delhi-110062

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  2. Bremner JD, Shearer KD, McCaffery PJ. Retinoic acid and affective disorders: The evidence for an association. J Clin Psychiatry 2012; 73 : 37–50.
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  1. Stahl SM. The Prescriber's Guide (Stahl's Essential Psychopharmacology, 4th ed. Cambridge, U.K.: Cambridge University Press, 2011.
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