INDIAN JOURNAL OF
CLINICAL PSYCHIATRY

MRI Publication Pvt. Ltd

Available online - www.ijocp.com

Crossref

Google Scholar

INTAS

Rexipra

Escitalopram 5/10/15/20 mg tabs

Provides more... with less !

Asprito

Aripiprazole 2/5/10/15/20/30 mg Melt-in-Mouth Tabs

Aspire for Uniqueness & Versatility

Qutan QUETIAPINE

25
50
100
200
300

Confidence of Lasting **Care**

DEPRAN 10

Escitalopram 10 mg + Clonazepam 0.5 mg Tabs.

No Substitute to Happiness

Olimelt

Olanzapine 2.5 / 5 / 7.5 / 10 / 15 / 20 mg Melt-in-Mouth Tabs

Melts into convenience and compliance

Zonalta

Endoxifen 8 mg Tablets

The World's First Direct→Protein Kinase C Inhibitor

Association of Clinical Psychiatry

www.ijocp.com

IJOCP

Indian Journal of
Clinical Psychiatry

An Official Journal of **Association of Clinical Psychiatry**
(Indian Psychiatric Society- Uttar Pradesh State Branch (IPS-UPSB))

About the Journal

Indian Journal of Clinical Psychiatry (IJOCP) is the official journal of the Indian Psychiatric Society, Uttar Pradesh Branch. It is published biannually. It is a comprehensive journal for mental health professionals, professionals of allied disciplines, and mental health policymakers. Emerging knowledge of the highest quality in the field of psychiatry, the latest technological enhancements, and developments of psychological interventions are shared in a timely and practical manner for clinical and professional use. The journal publishes peer-reviewed original research articles; review articles; commentaries on significant articles; case reports, perspectives, view-points (opinions), and letters to the editor. All papers are peer-reviewed before publication. The journal provides immediate free access to all the published articles. The journal does not charge the authors for submission, processing or publication of the articles.

About Society

Association of Clinical Psychiatry is the newly formed branch of the Indian Psychiatric Society. The resolution for formation of the IPS UP state branch was passed in the Annual General Body meeting of the IPS- Central Zone at the IPS-Central Zonal Conference held at Agra in 2018. Subsequent to this, the application for approval of formation of this new branch was sent to the Executive council of the Indian Psychiatric Society. IPS EC approved the formation of new branch at ANCIPS 2020 held at Kolkata.

Aim and Scope

The journal is aimed to-
- Promote research and publication in the field of psychiatry.
- Save the scientific update, knowledge among the researchers and mental health professionals free of cost.
- Promote research in mental health in Northern and other parts of India.

We encourage authors to publish their research related to Mental health and allied discipline. The journal encompasses all the subspecialties of Psychiatry (Child-adolescent Psychiatry, Social Psychiatry, Neuropsychiatry, Geriatric Psychiatry, Deaddiction Psychiatry, psychosexual Psychiatry, Perinatal Psychiatry, Preventive and Forensic Psychiatry).

The journal publishes Editorials, Original Articles, Case Studies, Reviews, Correspondence, and Book Reviews, etc. pertaining to Psychiatry with the special focus on Original and Review Articles on the following:

- Clinical Research.
- Fundamental research on various aspects of Psychiatry.
- Health promotion.
- Studies on Health Seeking Attitude and Medico-Social aspects including medical anthropology.
- Development of methodologies of Clinical Research Related to Psychiatry
- Art in Psychiatry
- Any other areas of related field etc.

Support Contact

Susheel Kumar
editor@mripub.com

Peer Review Process

The journal follows double-blind peer review policy.
- The IJOCP is very sensitive to plagiarism-related issues. The journal does not allow a similarity index of >10%. Any adoption of material (figure, diagnostic criteria, figures, etc.) from previous research work or publications needs to be produced after taking permission from publisher/authors of original publication and proper credits to the original research.
- The copyright of the published work lies with the author. However, when the author cites any published work with the journal it needs to be duly cited.
- As the journal is a non-profit journal and does not charge the author for submission, we do not allow the published material to be used for commercial purposes without permission.

Websites:
The electronic edition of the journal is available at **www.ijocp.com**
www.myresearchjournals.com
The manuscripts can be submitted at **www.ijocp.com**

Editorial Contact
Dr. Vipul Singh
Prof., Department of Psychiatry,
GMC, Kannauj, UP, India,
drvipulsingh@yahoo.co.in

Dr. Sujita Kumar Kar
Additional Professor, Department of Psychiatry, KGMU, Lucknow,
Uttar Pradesh, India
drsujita@gmail.com

Index

Guest Editorial

Research Articles

Review Articles

Case Study

Case Report

View Point

Moral Injury and mental health of youth: Reflections on Nepal's 'Gen Z Revolution'

Rashmi Shukla*, Ritwick Tripathi

Department of Psychiatry King George's Medical University, Lucknow 226003, Uttar Pradesh, India

Introduction

When thousands of young Nepalese filled Kathmandu's streets in September 2025, they were not only protesting a ban on social media, they were expressing the moral injury of a generation. Across South Asia, young people have become both witnesses to and victims of corruption and inequality. What unfolded in Nepal was not merely a political upheaval but a psychosocial event that revealed the deep interconnection between social injustice and mental health. Prior to the Nepal protests, digitally native, globally connected youth movements erupted in Sri Lanka (2022) and Bangladesh (2024), mobilizing against perceived corruption and nepotism.[1,2]

Thousands of youth flooded the streets of Kathmandu to oppose the ban on 26 social media sites. [1,2] However, the root cause of this protest was the widespread frustration about elite privilege and limited job opportunities for the youth. For example, approximately 20.8% youth unemployment existed in Nepal in 2024.[3] Moreover, even though the protests were largely decentralized and lacked visible leaders, most mainstream news accounts attributed the organization of the protests to online spaces. Nepal-based news organizations reported that the use of Discord (in addition to Viber/TikTok) was used for organizing the protests.[1,4] This reflected how digital spaces have become new sites of belonging and civic expression.

For clinicians, these events represented symbolized psychological distress as much as, if not more than, routine political contestation. For example, the themes expressed in the protests (i.e., helplessness, anger, frustrated aspirations) are similar to those that many of us have heard in our clinics. Soon after the protests, colleagues at Kanti Children's Hospital in Kathmandu reported an increase in acute stress, severe anxiety, and sleep disturbance in children and adolescents, many of whom lived away from where the protests took place. For example, a widely circulated report described an 8-year-old girl whose panic and screaming occurred when her parents closed a bathroom door because she feared that her home would be set on fire. [5] This representation is consistent with post-traumatic arousal. A study assessing symptoms of depression and anxiety in adults following the Gorkha earthquake in Nepal (2015) reported

that nearly 30% of adults experienced significant symptomatology in the months immediately following the earthquake.[6] Just as natural disasters leave invisible scars on collective memory, so too can political and social turbulence.

Current trauma treatment models fall short if they fail to account for the social conditions that contribute to the development of trauma. As such, treatments should extend beyond the delivery of trauma protocols. Mental health professionals must avoid pathologizing rational responses to unstable environments. The concept of moral injury, originally developed in the context of military psychiatry, offers a useful lens here.[7] Similar to post-traumatic stress disorder (PTSD), moral injury results from the experience of events that contradict an individual's core values (as enacted, unprevented, or witnessed); however, whereas PTSD focuses on fear, moral injury emphasizes shame, guilt, anger, and disloyalty/loss of trust.[7] When young people observe corruption, nepotism, and impunity closing off their futures, they experience a breach of moral expectation. They often experience institutional betrayal, which manifests as anomie, cynicism, and withdrawal from civic involvement, rather than flashbacks. Research to date indicates that chronic corruption is correlated with lower levels of well-being and internal struggle in civilian populations; these findings should be interpreted cautiously when applied to youth.[8]

This psychosocial shock also hits an under-resourced mental health infrastructure. At present, there is only one full-time Child & Adolescent Psychiatry OPD in Nepal (located at Kanti Children's Hospital since 2015).[9] The National Mental Health Survey (2020) reports high burdens of mental illness among both adults and adolescents in Nepal, including adolescent suicidal ideation and behavior; this demonstrates a substantial pre-existing treatment gap, which now intersects with a substantial trauma-driven demand.[10] South Asia as a region faces a similar scale challenge. India's national mental health and district mental health programmes (NMHP/DMHP) demonstrated that task-sharing and integration of mental health into primary care can extend reach. The Tele-MANAS initiative, a 24/7 two-tier helpline, is a potential way to increase access to mental health when a surge in demand occurs. By April 2025, Tele MANAS had established 53 service centers in 36 States/union territories and had already received over 2 million calls, with trained counselors available at Tier 1 and specialist escalation available at Tier 2.[11,12] Though imperfect, such scalable and technology-enabled approaches offer a glimpse of how systems can adapt when collective distress surges.

The psychological aftershocks of moral injury persist long after the protest period. Chronic despair and perceived corruption can limit identity formation, fostering confusion about roles, premature closure of options, or oppositional identities in young people.[13] Studies of youth living in areas of ongoing political conflict demonstrate that such youth exhibit enduring effects on trust and civic engagement, even after their acute symptoms resolve.[2,14] Furthermore, intergenerational studies have shown that the fears and survival strategies of a cohort of youth can be passed down to subsequent generations through attachment and family climate, thereby limiting the extent to which the next generation of youth will feel their own emotions or distrust others.[13,15]

When ambition meets violence or institutional apathy, many of the brightest leave. The selective emigration of educated youth, i.e., "brain drain," represents not only an economic loss but a form of avoidance. Each departure removes not just technical skill but a portion of a nation's mental capital, the capacity for civic hope, resilience, and moral repair. Nepal's Gen-Z uprising underscores that social and political crises are also public mental health crises. Mental health professionals must extend their gaze beyond the individual, recognizing how despair, disillusionment, and institutional betrayal shape emotional life. Mental-health responses that remain confined to clinical spaces will fail to meet the moral depth of this injury.

The lesson for South Asia is urgent and clear: to heal minds, we must also rebuild trust. Trauma care cannot be separated from the social conditions that generate trauma. Public mental health must therefore be both compassionate and civic, grounded in the recognition that every society's psychological well-being depends on the justice and integrity of its institutions. Nepal's Gen Z revolution will leave

lasting impacts on the mental health of its people. To treat our patients and communities effectively, we must recognize that a nation's psyche is inextricably linked to its social environment. The health of minds and the health of democracies are, in truth, one and the same.

References

1. Reuters. How 'Gen Z' protests over corruption and jobs ousted Nepal PM Oli [Internet]. 2025 Sep 9 [cited 2025 Nov 2] Available from: https://www.reuters.com/world/asia-pacific/how-gen-z-protests-over-corruption-jobs-ousted-nepal-pm-oli-2025-09-09/

2. Financial Times. The Gen Z revolution spreading in Asia [Internet]. 2025 Sep 14 [cited 2025 Nov 2] Available from: https://www.ft.com/content/31ac1faf-9348-4ee1-a129-4be6f9dd002d

3. World Bank via FRED. Youth Unemployment Rate for Nepal (SLUEM1524ZSNPL) [Internet]. 2025 [cited 2025 Nov 2] Available from: https://fred.stlouisfed.org/series/SLUEM1524ZSNPL

4. The Annapurna Express. The rise of digital natives: GenZ protest and Nepal's future [Internet]. 2025 Oct 7 [cited 2025 Nov 2] Available from: https://theannapurnaexpress.com/story/59097/

5. The Kathmandu Post. Children, teens show mental health problems after Gen Z protest [Internet]. 2025 Sep 18 [cited 2025 Nov 2] Available from: https://kathmandupost.com/health/2025/09/18/children-teens-show-mental-health-problems-after-gen-z-protest

6. Kane JC, Luitel NP, Jordans MJD, Kohrt BA, Weissbecker I, Tol WA. Mental health and psychosocial problems in the aftermath of the Nepal earthquakes: Findings from a representative cluster sample survey. Epidemiol Psychiatr Sci. 2017;26(6):1–10.

7. Litz BT, Stein N, Delaney E, Lebowitz L, Nash WP, Silva C, et al. Moral injury and moral repair in war veterans: A preliminary model and intervention strategy. Clin Psychol Rev. 2009;29(8):695–706.

8. Tay L, Herian MN, Diener E. Detrimental effects of corruption and subjective well-being: whether, how, and when. Soc Psychol Personal Sci. 2014;5(7):751–9.

9. IACAPAP. Child and Adolescent Mental Health in Nepal [Internet]. 2020–2025 update [cited 2025 Nov 2] Available from: https://iacapap.org/news/child-and-adolescent-mental-health-in-nepal.html

10. Nepal Health Research Council (NHRC). National Mental Health Survey, Nepal—2020: Report [Internet]. Kathmandu: NHRC; 2020 [cited 2025 Nov 2] Available from: https://nhrc.gov.np/wp-content/uploads/2022/10/National-Mental-Health-Survey-Report2020.pdf

11. Press Information Bureau, Government of India. Update on National Tele Mental Health Programme (NTMHP) [Internet]. 2025 Apr 4 [cited 2025 Nov 2]. Available from: https://pib.gov.in/PressReleasePage.aspx?PRID=2118790

12. Ministry of Health and Family Welfare, Government of India. National Mental Health Programme [Internet]. New Delhi: MoHFW; [cited 2025 Nov 2]. Available from: https://dghs.mohfw.gov.in/national-mental-health-programme.php

13. Erikson EH. Identity: Youth and crisis. New York: WW Norton; 1968.

14. Betancourt TS, Brennan RT, Rubin-Smith J, Fitzmaurice GM, Gilman SE. Longitudinal studies of mental health and psychosocial adjustment of children and youth in situations of political violence. Soc Sci Med. 2010;70(1):17–26.

15. Punamäki RL. Intergenerational transmission of trauma. In: Pat-Horenczyk R, Brom D, Vogel J, editors. Helping children cope with trauma: Individual, family, and community perspectives. New York: Routledge; 2013. p. 163–80.

Childhood Sexual Abuse among Adults with First-Episode Depression and Generalized Anxiety Disorder: Comparison with Healthy Controls

Shradha Khatri[1*], Sunil Goyal[2]

[1]Graded Specialist, MD, DNB (Psychiatry), AFMS, Pune, Maharashtra, India.
[2]Former Professor and Head, Dept of Psychiatry, Institute of Naval Medicine, Mumbai, India.

Abstract

Background: Childhood Sexual Abuse (CSA) has been identified as a serious public health concern and has been a global challenge. The severe, chronic and frequent the abuse, more likely the abused adults are to show symptoms of Psychiatric disorders.

Aim: To study the prevalence of CSA among adults presenting first time with a depressive episode or Generalized Anxiety Disorder.

Methods: 64 consecutive consenting adults among Armed Forces personnel and their families, diagnosed as first depressive episode or Generalized Anxiety Disorder as per International Classification of Diseases, 10th edition (ICD-10) checklist and a control group of gender and age-group matched adults were assessed for CSA using a 28-item retrospective, self-report Childhood Trauma Questionnaire (CTQ-28) at an Armed Forces hospital.

Results: Majority of cases reported no CSA, whereas majority of the control group reported moderate severity of CSA. Among the cases, statistically significant co-relation was neither observed between the diagnosis and severity of CSA nor the severity of illness with severity of CSA.

Conclusion: Absence of any signs of psychopathology could perhaps be due to psychological endurance, resilience and higher stress tolerance developed in the aftermath of early age traumatic incidents.

INTRODUCTION

The World Health Organization (WHO) defines Child Sexual Abuse (CSA) as a coercive act involving a child who is unable to comprehend or provide consent, often resulting in serious physical and psychological harm.[1] CSA is widely recognized as a major public health concern with significant developmental and mental health consequences.[2] It includes a broad spectrum of acts such as inappropriate touching, molestation, sodomy, exhibitionism, pornography, and cyber-sexual activities.[3] Such acts are regarded as offensive across all cultures. Prevalence rates vary between 8–31% among females and 3–17% among males, with some studies indicating higher rates among boys.[4] Childhood abuse has cumulative effects on the developing brain, mind, and

interpersonal relationships and is an important determinant of health outcomes.[5] Early traumatic experiences are known contributors to the etio-pathogenesis of depression, anxiety, and other stress-related disorders.[6] The severity of abuse has a direct correlation with the intensity of depressive and anxiety symptoms in later life.[7] Multiple meta-analyses have consistently demonstrated a strong association between CSA and a broad range of adult psychological disorders.[8]

Trauma has been conceptualized as a relational phenomenon that links an external event to its specific consequences for the inner psychic world.[9] Cooper described psychic trauma as an event that overwhelms an individual's capacity to maintain safety and psychological integration, resulting in enduring psychic change.[10] Chronic or repeated trauma often has compounded effects, leading to a higher lifetime prevalence of depression and anxiety disorders.[11]

In India, children below 18 years constitute nearly 37% of the total population, many of whom lack adequate nutrition, education, and access to healthcare.[12] A national survey reported that 53% of Indian children have experienced some form of abuse, including sexual abuse.[13] In primary care settings, 20–50% of adults report experiences of childhood physical or sexual abuse, and this prevalence rises to up to 70% among individuals with depression, post-traumatic stress disorder (PTSD), chronic pain, or substance use disorders.[14,15] Notable gender differences exist in both prevalence and severity of CSA experiences, and a temporal relationship has been observed between the number and severity of childhood abuse and the subsequent development of psychiatric disorders.[11,16]

Over the past two decades, extensive evidence has confirmed the relationship between childhood trauma and the onset, symptom severity, and clinical course of depressive and anxiety disorders.[17] Although definitions of abuse and age cut-offs differ across studies, the cumulative evidence fulfills multiple criteria for a causal association between CSA and later psychopathology.[18]

Despite high national rates of child abuse, Indian studies focusing on CSA among adults presenting with first-episode depressive disorder or generalized anxiety disorder (GAD) remain limited. The present study aims to estimate the prevalence and severity of CSA among such patients, compare these findings with age- and gender-matched healthy controls, and explore correlations between CSA severity, psychiatric diagnosis, and illness severity. Additionally, it seeks to identify socio-demographic and contextual factors such as education, income, family structure, and peer support that may confer resilience or vulnerability among individuals with CSA histories.

Materials and Methods

This was a comparative, cross-sectional observational study conducted in the psychiatry department of a tertiary care hospital in Mumbai. Data were collected between September 2019 and March 2021.

The case group comprised 64 consecutive adults who presented for the first time with a diagnosis of either a Depressive episode (ICD-10 F32) or GAD (ICD-10 F41.1). Diagnoses were established by a consultant psychiatrist using a clinical interview anchored to the ICD-10 symptom checklist. Individuals with past psychiatric diagnoses were excluded to focus on first-episode presentations and to reduce recall or reporting distortions associated with prior psychiatric care.

The control group consisted of 64 age- and gender-matched healthy adults drawn from the same principal population, comprising of the patient attendants and volunteers, who consented for the study. They were screened using the General Health Questionnaire-12 (GHQ-12) and were included only if there was no indication of current psychopathology.

Inclusion criteria for cases were: adults aged 18 years and above; first-episode depressive disorder or GAD; ability and willingness to provide informed consent. Exclusion criteria for both cases and controls included: history of psychiatric illness (for controls) or comorbid severe psychiatric conditions (psychotic disorders, bipolar disorder), significant cognitive impairment, chronic debilitating medical illness, or refusal to provide informed consent.

Measures

- A Socio-demographic and Clinical Proforma was used to capture age, gender, marital status, educational attainment, occupation, monthly income, family type (nuclear/joint), and rural/urban/semi-urban background.
- Beck Depression Inventory (BDI)[19] and Hamilton Anxiety Rating Scale (HAM-A) [20] were used to assess the severity of depressive and anxiety severity respectively.
- Childhood Trauma Questionnaire (CTQ-28),[21] a 28-item retrospective self-report instrument was used to assess multiple subtypes of childhood maltreatment, including sexual abuse, on a five-point Likert scale. Higher scores indicate greater severity of traumatic experiences.
- A study-specific Semi-Structured Interview on CSA was conducted to elicit age at first incident, number of episodes, nature of abuse, relationship to perpetrator, setting of the abuse, disclosure patterns, and responses received from confidants.
- No scale was used to measure the objective or the subjective parameters of resilience for cases or controls.

After obtaining written informed consent, participants underwent structured clinical assessment. Cases completed the BDI and HAM-A to quantify symptom severity. All participants completed the CTQ-28 and the semi-structured CSA interview. Information with clinical implications was communicated to the treating psychiatrist to ensure appropriate care. Index individual's distress related to recall of CSA was addressed in an empathetic and sensitive manner. The individual psychotherapy sessions were are tailor-made to address the distress. The study was approved by the Institutional Ethics Committee with reference number MECM/17 obtained on 29 August 2019. Data were entered and analysed using GNU PSPP (version 1.2.0).[22] A p-value of <0.05 was considered statistically significant.

Results

From September 2019 to August 2021, a total of 128 adults participated: 64 cases and 64 controls. The mean age of the overall sample was 29.5 years (SD =

3.48). Males constituted 64.1% of the sample, and the majority were aged 25–29 years (57.8%). Socio-demographic differences between cases and controls were notable as cases had a lower mean monthly income (₹29,680) and years of education (14.15 years) compared with controls (₹46,870, 15.78 years). Most cases were married (56.3%) and came from nuclear families (64.1%) and rural backgrounds (50%). Most controls were unmarried (67.2%), were living in occupational settings with colleagues (34.4%) and half originated from semi-urban backgrounds. These socio-demographic differences were statistically significant, highlighting potentially important contextual disparities between groups (Table 1).

Among the 64 cases, a majority (75.0%) reported no history of CSA. The remainder reported mild (12.5%), moderate (9.4%), or severe (3.1%) CSA. In stark contrast, among the 64 controls, only 14.1% denied CSA; 26.6% reported mild, 37.5% moderate, and 21.9% severe CSA. The resilience measures were hypothesized to be the reason for this paradoxical finding, and this between-group difference in CSA severity distribution was statistically significant (p < 0.001). Additional confounding details emerged from the semi-structured interviews revealed that among the 44 cases who reported any form of childhood trauma (not limited to sexual abuse), 25% reported the first incident occurred between the ages of 6 and 8 years. Of those, 15.6% identified sexual abuse as the specific nature of trauma. A large majority (77.3%) reported more than six incidents. Abuse most commonly took place at home (88.6%), followed by school and outdoor locations (4.54% each), and other/unknown areas (2.27%). Only four participants reported disclosing the abuse to a family member before age 8, most commonly to the mother. In each of these cases, no action was reportedly taken and the abuse continued. (Table 2) (Table 3) (Graph 1) However, the multivariate of these numerous factors was not carried out.

Within the case group, 35 (54.7%) had a depressive episode and 29 (45.3%) had GAD. When stratified by diagnosis, 48 presented with moderate and 16 with severe illness severity. The distribution of CSA severity across diagnoses did not differ significantly (p = 0.438). Similarly, no significant association was detected between illness severity (moderate vs.

Table 1: Descriptive data of cases and (age & gender matched) controls

Monthly Income (Rs)	Cases (n=64) n (%)	Controls (n=64) n (%)	Social support	Cases (n=64) n (%)	Controls (n=64) n (%)
Nil	8 (12.5)	00	Nuclear Family	41 (64.1)	20 (31.3)
<20,000	15 (23.4)	4 (6.3)	Joint Family	9 (14.1)	5 (7.8)
20,000-50,000	29 (45.3)	18 (28.1)	Living in Occupational Setting	4 (6.3)	22 (34.4)
>50,000	12 (18.8)	42 (65.6)	Chi-Square Test, P Value <0.001, Significant		
Mean	29.68	46.87	Place of Origin		
Chi-Square Test, P Value <0.001, Significant			Urban	11 (17.2)	10 (15.6)
Education			Rural	32 (50.0)	22 (34.4)
10th std	1 (1.6)	00	Semi-Urban	21 (32.8)	32 (50.0)
12th std	11 (17.2)	00	Chi-Square Test, P Value = 0.124, Not Significant		
12th std-Graduate	40 (62.5)	26 (40.6)	Marital Status		
Post-graduate	12 (18.8)	38 (59.4)	Single	26 (40.6)	43 (67.2)
Chi-Square Test, P Value <0.001, Significant			Married	36 (56.3)	21 (32.8)
Social support			Others	2 (3.1)	00
Living Alone	10 (15.6)	17 (26.6)	Chi-Square Test, P Value = 0.006, Significant		

Table 2: Diagnosis and self-report data of cases

Diagnosis	No.	Percent	Nature of abuse	No.	Percent
Depression	35	54.7	None	20	31.3
Generalized Anxiety Disorder	29	45.3	Sexual Abuse	10	15.6
Stressors			Other abuse/neglect	34	53.12
Acute	42	65.6	Self-reported no of incidents: (N=44)		
Long-Standing	22	34.4	1	3	6.8
Cases self-reporting abuse:			2-3	7	15.9
During data collection	44	68.7	>6	34	77.3
Cases self-reporting abuse during childhood: (N = 44)			Perpetrators (Multiple incidents)		
Reported to familymember	4	9.1	1st degree relative	2	4.5
Reported before age 8	4	100.0	2nd degree relative	2	4.5
Action taken	0	0.0	Parent	32	72.7
Abuse stopped later	0	0.00	Friend	7	15.9
1st Incident age (Inyears)			Sibling	10	22.7
Did not report	20	31.3	Place of incidents		
3-5 yrs.	5	7.8	Home	39	88.6
6-8 yrs.	16	25.0	School	2	4.54
9-12 yrs.	13	20.3	Outdoors	2	4.54
13-15 yrs.	10	15.6	Unknown	1	2.27
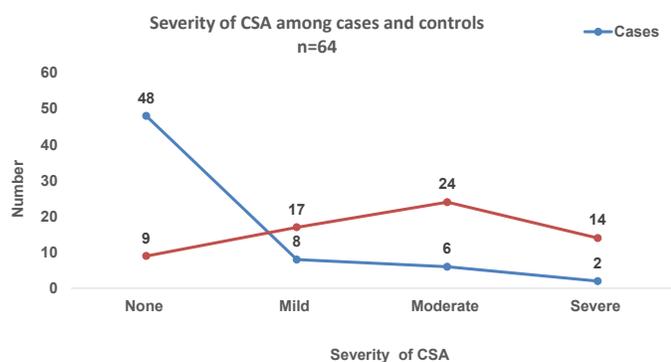
Graph 1: Severity of childhood sexual abuse between cases and controls

Table 3: Severity of childhood sexual abuse

Severity of Sexual Abuse	Group	
	Cases (n=64) n (%)	Controls (n=64) n (%)
None	48 (75.0)	9 (14.1)
Mild	8 (12.5)	17 (26.6)
Moderate	6 (9.4)	24 (37.5)
Severe	2 (3.1)	14 (21.9)

Chi-Square Test, P Value <0.001, Significant

severe) and CSA severity (p = 0.149). Thus, in this sample, neither diagnostic category nor symptom severity among cases correlated with the severity of CSA (Table 4).

Discussion

This study examined the prevalence and severity of childhood sexual abuse (CSA) among adults with first-episode depressive disorder or generalized anxiety disorder (GAD), compared with matched healthy controls. Contrary to existing literature linking CSA with psychopathology, the control group in our sample reported higher CSA prevalence and severity, despite absence of psychiatric morbidity. Within the clinical group, CSA severity did not significantly correlate with diagnosis or illness severity.

Several explanations may account for this paradoxical observation. First, resilience and adaptive coping may have mitigated adverse effects of CSA in controls. Higher education, income, and social support among controls possibly enhanced affect regulation and stress tolerance. Second, differential reporting bias could have influenced results, as individuals with psychopathology might underreport CSA due to shame, avoidance, or stigma, while psychologically stable individuals could disclose more freely. Third, diagnostic limitation to depression and GAD may have excluded other psychopathologies, such as subclinical PTSD or personality disorder traits, not captured by GHQ-12 screening. Finally, the cross-sectional design precludes temporal inference; some controls may develop psychopathology later, while some cases may have been influenced by non-CSA stressors, including genetic and environmental vulnerabilities.

Socio-demographic differences further contextualize findings. Cases were predominantly married, less educated, and from rural, nuclear families, whereas controls were unmarried, with higher income and education, and often lived with peers. Peer-based social networks might promote adaptive functioning and resilience, contrasting with literature suggesting marriage as protective against stress-related disorders.[23]

Table 4: Outcome variables: Severity of childhood sexual abuse and illness

Severity of Sexual Abuse	Diagnosis		Severity of Illness	
	Depression (n = 35) n (%)	Anxiety (n = 29) n (%)	Moderate (n=48) n (%)	Severe (n=16) n (%)
None	29 (82.9)	19 (65.5)	36 (75.0)	12 (75.0)
Mild	3 (8.6)	5 (17.2)	4 (8.3)	4 (25.0)
Moderate	2 (5.7)	4 (13.8)	6 (12.5)	00
Severe	1 (2.9)	1 (3.4)	2 (4.2)	00
Chi-Square Test	P Value = 0.438, Not Significant		P Value = 0.149, Not Significant	

The absence of a dose–response relationship between CSA severity and psychopathology within the case group may reflect limited statistical power, narrow trauma focus, and retrospective self-report limitations. Additional unmeasured variables—such as temperament, family psychiatric history, substance use, or ongoing stressors—could have moderated outcomes.

Previous studies affirm strong associations between childhood adversities and adult psychiatric disorders. Kessler et al[24] found robust correlations between childhood adversities and all DSM-IV disorder classes. Huh et al[25] reported significant links between sexual abuse and interpersonal distress among adults with depression and anxiety. Khan et al[26] identified sensitive developmental windows where abuse exposure maximally increased depression risk. Kessler and McLaughlin[27] estimated that nearly 30% of psychiatric disorders arise from early adversities. CSA has further been implicated in intimacy dysfunction, social maladjustment, and poor treatment outcomes.[3,28]

However, resilience studies indicate that trauma exposure does not universally result in psychopathology. Cicchetti et al[23] highlighted that maltreated individuals may develop adaptive coping, emotion regulation, and self-organization capacities under chronic stress. Rehan et al[29] similarly observed that most severely abused individuals (72%) did not exhibit clinical depression or anxiety, attributing this to resilience mechanisms.

Consistent with these frameworks, the present findings suggest that socio-demographic and contextual variables—particularly education, income, and peer support—significantly influence whether CSA leads to psychopathology or adaptive functioning. While CSA remains a potent risk factor for mental illness, resilience and social context appear equally critical in determining psychological outcomes.

This study included first-episode patients with closely matched controls and employed validated instruments to assess depression, anxiety, and childhood trauma. Recruiting controls from the same institutional population minimized sampling bias, and the focus on first-episode presentations reduced confounding from chronicity or treatment exposure.

However, certain limitations warrant consideration. The cross-sectional design precludes causal inference, and the modest, single-centre sample limits generalizability. Significant socio-demographic differences between cases and controls and unmeasured resilience-related variables (e.g., coping, social support, temperament) may have influenced findings. The reliance on retrospective self-report introduces potential recall and response bias, possibly leading to minimization or exaggeration of CSA experiences. Restricting inclusion to depressive and anxiety disorders may have excluded conditions where CSA exerts stronger effects, such as PTSD, dissociative, or personality disorders. Furthermore, variables such as personality organization, family psychiatric history, and ongoing stressors were not systematically assessed, constraining interpretation of resilience and vulnerability mechanisms.

Future research should adopt longitudinal, multi-centre designs with larger samples and multivariate modelling to examine developmental trajectories and delayed psychopathology. Expanding diagnostic coverage to include PTSD, complex PTSD, dissociative and substance use disorders, and incorporating biomarkers, neurocognitive profiling, and neuroimaging could enhance understanding of biological underpinnings. Assessing resilience, coping strategies, and social support through validated tools would further clarify protective factors mitigating psychopathology after CSA.

Conclusion

Despite its limitations, this study represents one of the first efforts to explore trauma-informed psychiatric assessment among adults with first-episode depression and GAD in an Indian tertiary care context. The findings underscore that a history of CSA does not invariably predict psychopathology and that socio-demographic and contextual resilience factors may buffer psychological outcomes. Routine trauma-informed screening should therefore be integrated into psychiatric and primary care settings, even among asymptomatic individuals reporting CSA. Clinicians should maintain a low threshold for CSA inquiry while recognizing that absence of overt symptoms does not equate to absence of distress.

Community and clinical interventions must empha-size education, peer support, and socio-economic stability as protective domains. Strengthening resilience-oriented public health strategies may hold promise in reducing psychiatric morbidity and enhancing adaptive recovery among CSA survivors.

Financial Support and Sponsorship

Nil.

Conflicts of Interest

There are no conflicts of interest.

References

1. Mathews B, Collin-Vézina D. Child sexual abuse: Toward a conceptual model and definition. Trauma Violence Abuse. 2019;20(2):131–48.

2. Shenk CE, Noll JG, Putnam FW, Trickett PK. A prospec-tive examination of the role of childhood sexual abuse and physiological asymmetry in the development of psychopathology. Child Abuse Negl. 2010;34(10):752–61. Available from: http://www.ncbi.nlm.nih.gov/pubmed/20850183

3. Rowan EL. Understanding child sexual abuse. Jackson (MS): University Press of Mississippi; 2006.

4. Barth J, Bermetz L, Heim E, Trelle S, Tonia T. The current prevalence of child sexual abuse worldwide: a sys-tematic review and meta-analysis. Int J Public Health. 2013;58(3):469–83.

5. Danese A, Pariante CM, Caspi A, Taylor A, Poulton R. Childhood maltreatment predicts adult inflamma-tion in a life-course study. Proc Natl Acad Sci U S A. 2007;104(4):1319–24. Available from: http://www.pnas.org/cgi/doi/10.1073/pnas.0610362104

6. Hill J. Developmental perspectives on adult depression. Psychoanal Psychother. 2009;23(3):200–12.

7. Leuzinger-Bohleber M. Working with severely trauma-tized, chronically depressed analysands. Int J Psycho-anal. 2015;96(3):611–36. Available from: https://www.tandfonline.com/doi/full/10.1111/1745-8315.12238

8. Chen LP, Murad MH, Paras ML, Colbenson KM, Sattler AL, Goranson EN, et al. Sexual abuse and lifetime diag-nosis of psychiatric disorders: systematic review and meta-analysis. Mayo Clin Proc. 2010;85(7):618–29.

9. Zepf S, Zepf FD. Trauma and traumatic neurosis: Freud's concepts revisited. Int J Psychoanal. 2008;89(2):331–53.

10. Cooper AM. Toward a limited definition of psychic trauma. In: The reconstruction of trauma: Its signifi-cance in clinical work. New York: Basic Books; 1986. p. 41–56.

11. Negele A, Kaufhold J, Kallenbach L, Leuzinger-Bohle-ber M. Childhood trauma and its relation to chronic depression in adulthood. Depress Res Treat. [Internet]. Available from: https://www.hindawi.com/journals/drt/

12. Baru R, Acharya A, Acharya S, Kumar AS, Nagaraj K. Inequities in access to health services in India: caste, class and region. Econ Polit Wkly. 2010;45(38):49–58.

13. Choudhary V, Satapathy S, Sagar R. Qualitative study on the impact of child sexual abuse: perspectives of children, caregivers, and professionals in Indian context. J Child Sex Abus. 2019;28(4):489–510.

14. Flaherty EG, Jones R, Sege R; Child Abuse Recognition Experience Study Research Group. Telling their stories: primary care practitioners' experience evaluating and reporting injuries caused by child abuse. Child Abuse Negl. 2004;28(9):939–45.

15. Blanchard EB, Keefer L, Lackner JM, Galovski TE, Krasner S, Sykes MA. The role of childhood abuse in Axis I and Axis II psychiatric disorders and medical disorders of unknown origin among irritable bowel syndrome patients. J Psychosom Res. 2004;56(4):431–6.

16. Teicher MH, Samson JA, Polcari A, McGreenery CE. Sticks, stones, and hurtful words: relative effects of various forms of childhood maltreatment. Am J Psychi-atry. 2006;163(6):993–1000.

17. Hovens JG, Giltay EJ, Wiersma JE, Spinhoven P, Penninx BW, Zitman FG. Impact of childhood life events and trauma on the course of depressive and anxiety disor-ders. Acta Psychiatr Scand. 2012;126(3):198–207.

18. Springer KW, Sheridan J, Kuo D, Carnes M. The long-term health outcomes of childhood abuse. J Gen Intern Med. 2003;18(10):864–70.

19. Beck AT. Depression: Causes and treatment. Philadel-phia: University of Pennsylvania Press; 1972.

20. Hamilton M. The assessment of anxiety states by rating. Br J Med Psychol. 1959;32:50–5.

21. Bernstein DP, Stein JA, Newcomb MD, Walker E, Pogge D, Ahluvalia T, et al. Development and validation of a brief screening version of the Childhood Trauma Ques-tionnaire. Child Abuse Negl. 2003;27(2):169–90.

22. GNU Project. GNU PSPP statistical analysis software, version 1.2.0. Free Software Foundation; 2019.

23. Cicchetti D, Rogosch FA. Adaptive coping under con-ditions of extreme stress: multilevel influences on the determinants of resilience in maltreated children. New Dir Child Adolesc Dev. 2009;(124):47–59.

24. Kessler RC, McLaughlin KA, Green JG, Gruber MJ, Sampson NA, Zaslavsky AM, et al. Childhood adversities and adult psychopathology in the WHO World Mental Health Surveys. Br J Psychiatry. 2010;197(5):378–85.

25. Huh HJ, Kim SY, Yu JJ, Chae JH. Childhood trauma and adult interpersonal relationship problems in patients with depression and anxiety disorders. Ann Gen Psy-chiatry. 2014;13(1):1–3.

26. Khan A, McCormack HC, Bolger EA, McGreenery CE,

Vitaliano G, Polcari A, et al. Childhood maltreatment, depression, and suicidal ideation: critical importance of parental and peer emotional abuse during developmentally sensitive periods in males and females. Front Psychiatry. 2015;6:42.

27. Green JG, McLaughlin KA, Berglund PA, Gruber MJ, Sampson NA, Zaslavsky AM, et al. Childhood adversities and adult psychiatric disorders in the National Comorbidity Survey Replication I. Arch Gen Psychiatry.

2010;67(2):113–23.

28. Blanco C, Rubio J, Wall M, Wang S, Jiu CJ, Kendler KS. Risk factors for anxiety disorders: common and specific effects in a national sample. Depress Anxiety. 2014;31(9):756–64.

29. Rehan W, Antfolk J, Johansson A, Jern P, Santtila P. Experiences of severe childhood maltreatment, depression, anxiety and alcohol abuse among adults in Finland. PLoS One. 2017;12(5):e0177252.

Exploring The Relationship of Symptom Dimensions with Insight and Functioning in Obsessive-Compulsive Disorder: A Cross-Sectional Study

Paramjeet Singh[1], Mahendra Kumar[2*], Niharika Bawankar[3], Saloni Gupta[4], Harshpreet Kour[5]

[1]Senior Professor, Department of Psychiatry, SMS Medical College, Jaipur 302004, India
[2]MD Psychiatry, Department of Psychiatry, SMS Medical College, Jaipur 302004, India
[3]Junior Resident, Department of Psychiatry, SMS Medical College, Jaipur 302004, India
[4]MD Psychiatry, Department of Psychiatry, SMS Medical College, Jaipur 302004, India
[5]Junior Resident, Department of Psychiatry, SMS Medical College, Jaipur 302004, India

Abstract

Background: In patients with obsessive-compulsive disorder (OCD), insight is a reflection of their understanding that their symptoms are irrational. Variations in insight have been associated with differences in symptom dimensions, severity, and overall prognosis. Moreover, specific psychopathological dimensions may differentially impact social and occupational functioning in OCD patients.

Aim: To explore the association of symptom dimensions with insight levels and to examine their impact on social and occupational functioning in patients with obsessive-compulsive disorder (OCD).

Methods: This cross-sectional study included 100 patients diagnosed with OCD according to DSM-5 criteria, aged 18–60 years, at a tertiary care center. Based on the Y-BOCS Item-11, patients were categorized as having good or poor insight. For assessing symptom dimensions, the Dimensional Yale-Brown Obsessive-Compulsive Scale (DY-BOCS) was used, while the Social and Occupational Functioning Assessment Scale (SOFAS) was utilized to evaluate functioning.

Results: Of the 100 patients, 78% had good insight, while 22% exhibited poor insight. Overall, no statistically significant difference was found in the presence or severity of symptom dimensions between the two groups. A significant negative correlation was observed between the sexual/religious symptom dimension and SOFAS scores (p<0.05), indicating that greater severity in this domain was linked to poorer social and occupational functioning.

Conclusions: Poor insight was associated with higher symptom severity, divorce/separation and longer duration of untreated illness. Insight levels did not significantly differ across symptom dimensions. However, sexual-religious obsessions were strongly associated with poorer functioning, underscoring the need for domain-specific interventions beyond insight-based management.

INTRODUCTION

The concept of insight in the context of OCD refers to the extent to which an individual recognizes that their obsessions and compulsions are unreasonable,

excessive, and not based in reality. DSM-5 (American Psychiatric Association, 2013) provides three specifiers for the level of insight: with good or fair insight, with poor insight, and with absent insight/ delusional beliefs.[1]

Psychopathology is defined as the study of mental illness, behaviours, and experiences that are indicative of mental illness or psychological impairment. It encompasses the signs and symptoms associated with mental disorders. A common tool for assessing OCD symptom dimensions is the Dimensional Yale-Brown Obsessive–Compulsive Scale (DY-BOCS), which identifies six dimensions: [1] symmetry, 'just right' feelings, counting, and arranging; [2] contamination and cleaning; [3] harm-related content, including aggression, injury, violence, and accidents; [4] moral, sexual, and religious content; [5] hoarding; and [6] miscellaneous, including somatic concerns and superstitions.[2]

According to studies, insight varies across different OCD symptom dimensions.[3-6] Certain dimensions, such as hoarding,[3-5,7,8] have been consistently associated with poor insight. Some data also suggest that the need for symmetry may be linked to poor insight.[5,9] However, there is conflicting evidence regarding the relationship between contamination/washing symptoms and insight, as these symptoms have been associated with both good[4] and poor insight.[5] Thus, the association between specific symptom dimensions and insight remains inconsistent, highlighting the need for further research in this area.

OCD is a disabling condition that adversely affects the quality of life of both patients and their families, and it significantly impairs social and occupational functioning.[10-12] Social functioning has been found to negatively correlate with sexual and religious symptom dimensions of OCD.[13] Additionally, another study reported specific associations between OCD symptom dimensions and various aspects of quality of life: symmetry/ordering symptoms were associated with poorer social quality of life; forbidden thought symptoms with diminished health-related quality of life; and contamination/ cleaning symptoms with reductions in leisure time, social interaction, and health-related quality of life. [14]Since insight is a modifiable and potentially treat-

able characteristic that may influence therapeutic outcomes in OCD, it is an important clinical variable that should be considered in the comprehensive evaluation and management of the disorder.[15] Although insight varies across symptom dimensions of OCD, the existing data are inconsistent, and there is a notable lack of research on this topic from India. Therefore, more research is needed to explore insight, psychopathology, and functioning in OCD patients. Hence, the current study was undertaken to investigate the relationship of symptom dimensions with insight and functioning in patients with OCD.

Materials And Methods

Ethical Considerations

Ethical approval for the study from the Institutional Ethical Committee (approval number: 187/MC/EC/2023) was obtained before the start of the study (on April 05, 2023). The study was conducted in accordance with the ethical principles outlined in the World Medical Association's Declaration of Helsinki for research involving human participants.

Patient Consent

Written informed consent was taken from each participant in the study. They were assured of complete confidentiality of their identity, information, and the option of withdrawing from the study at any point in time.

Study Design

This observational study, employing a descriptive cross-sectional design, was conducted at a tertiary care centre in northwestern India over a period of one year, from June 2023 to May 2024. Participants were recruited using a convenience sampling method from outpatient and inpatient departments of the hospital. They were diagnosed with OCD as per DSM-5 criteria and confirmed by 2 independent interviewers.

Inclusion and Exclusion Criteria: Adults aged between 18 and 60 years who met DSM-5 diagnostic criteria for obsessive-compulsive disorder and had a Yale-Brown Obsessive Compulsive Scale (Y-BOCS)

score of 8 or above were eligible for inclusion after obtaining written informed consent. Exclusion criteria comprised individuals with acute medical illnesses requiring immediate intervention, those with intellectual developmental disorder (IDD), comorbid psychiatric disorders (other than nicotine dependence), or refusal to consent.

Data collection tool

Patient Information Sheet and Informed Consent Form

Provided in both Hindi and English to ensure comprehension and voluntary participation.

Sociodemographic and Clinical Profile Proforma:

Developed for the study to record sociodemographic characteristics and clinical variables, including family history, age of onset, and illness duration.

Yale–Brown Obsessive Compulsive Scale (Y-BOCS II)

A validated instrument used to quantify the severity of obsessions and compulsions. The first 10 items contribute to a total score ranging from 0 to 40, with higher scores indicating greater symptom severity. Severity was classified as mild (8–15), moderate (16–23), severe (24–31), and extreme (32–40).[16]

Y-BOCS Item-11

This specific item is used to assess the extent of the individual's insight into their obsessive-compulsive symptoms. Insight is rated on a scale ranging from 0 to 4, where 0 indicates excellent insight, 1 denotes good insight, 2 reflects fair insight, 3 suggests poor insight, and 4 signifies a complete lack of insight.[17]

Dimensional Yale–Brown Obsessive–Compulsive Scale (DY-BOCS)

This clinician-administered (objective) tool was applied to identify and evaluate the severity of symptoms across various dimensions of OCD. The DY-BOCS organizes symptoms into six distinct domains: harm/aggression, sexual/religious obsessions, contamination/cleaning, symmetry/ordering/counting/arranging, hoarding/collecting, and a miscellaneous category. Each domain's clinical severity is assessed across three parameters—frequency, distress, and interference—each scored out of 5, allowing for a maximum score of 15 per dimension.[2]

Social and Occupational Functioning Assessment Scale (SOFAS)

This scale specifically focuses on the individual's performance in social and occupational settings. It is designed to assess functioning independently of the overall severity of psychological symptoms, ensuring that it reflects real-world functional ability rather than symptom burden. It is scored on a continuum from 0 to 100, with higher scores indicating better functioning.[18]

Statistical analysis

The data were first entered into Microsoft Excel and later transferred to IBM SPSS version 20.0.0 (IBM, New York, USA) for detailed statistical analysis. Descriptive statistics for quantitative variables are expressed as mean ± standard deviation (SD), while qualitative variables are summarized using frequency distributions. Pearson's correlation analysis was conducted to examine relationships between variables. A p-value of ≤ .05 was considered statistically significant for all analyses.

RESULTS

The study included 100 patients with obsessive–compulsive disorder. The mean age was 31.9 years, and most participants were between 18 and 44 years of age as shown in Table 1. Males constituted 57% of the sample, and 55% were married. Nearly two-fifths were graduates or had higher education, while 84% were non-earning individuals. A majority of participants resided in rural areas (61%), were Hindu by religion (90%), and lived in nuclear families (59%). A family history of OCD was present in 22% of cases. The mean age of onset was 24.6 years, and the mean illness duration was 7.3 years. Most patients had severe (41%) to extreme (32%) symptom severity according to the Y-BOCS score.

The comparison of sociodemographic factors between the two OCD groups with good and poor insight is shown in Table 2. There was no statistically significant difference between the two insight groups, except for marital status. A higher percentage of married individuals were in the good insight

Table 1: Categorization of the sample into two groups of OCD with good and poor insight

Number of patients (n=100)	*Group*	
n (%)	OCD with Good Insight (Y-BOCS Item-11 Score 0, 1 & 2)	OCD with Poor Insight (Y-BOCS Item-11 Score 3 & 4)
	(78%)	22 (22%)

Table 2: Comparison of sociodemographic variables between good and poor insight groups

Variables	*Subgroups*	*OCD with Good Insight n (%)*	*OCD with Poor Insight n (%)*	*$\chi 2$/Fisher's exact/ t-test*	*p-value*
Age (Years)	Mean (SD)	32.49 (9.58)	30.0 (9.28)	1.103	0.278
Gender	Male	44 (56.4%)	13 (59.1%)	0.050	0.823
	Female	34 (43.6%)	9 (40.9%)		
Marital status	Married	47 (60.3%)	8 (36.4%)	6.274	0.034*
	Unmarried	29 (37.2%)	11 (50%)		
	Divorced/Separated	2 (2.6%)	3 (13.6%)		
Education	Up to Primary	6 (7.7%)	2 (9.1%)	1.370	0.873
	Middle class	14 (17.9%)	5 (22.7%)		
	Secondary	13 (16.7%)	2 (9.1%)		
	Higher secondary	14 (17.9%)	5 (22.7%)		
	Graduate and above	31 (39.7%)	8 (36.4%)		
Employment status	Nonearning	62 (79.5%)	22 (100%)	3.816	0.256
	Unskilled/Semi-skilled	8 (10.3%)	0 (0.0%)		
	Skilled	4 (5.1%)	0 (0.0%)		
	Semi-professional/Professional	4 (5.1%)	0 (0.0%)		
Locality	Urban	34 (43.6%)	5 (22.7%)	3.139	0.088
	Rural	44 (56.4%)	17 (77.3%)		
Religion	Hindu	69 (88.5%)	21 (95.5%)	1.091	0.452
	Muslim	9 (11.5%)	1 (4.5%)		
Type of family	Nuclear	45 (57.7%)	14 (63.6%)	1.196	0.596
	Joint	14 (17.9%)	5 (22.7%)		
	Others	19 (24.4%)	3 (13.6%)		
Monthly family income (in Rs)	≤ 10000	20 (25.6%)	7 (31.8%)	1.107	0.785
	10001-20000	24 (30.8%)	6 (27.3%)		
	20001-30000	17 (21.8%)	6 (27.3%)		
	>30000	17 (21.8%)	3 (13.6%)		
Family history	Present	16 (20.5%)	6 (27.3%)	0.441	0.563
	Absent	62 (79.5%)	16 (72.7%)		

*Significant p-value (≤0.05)

group, while a higher percentage of unmarried and Divorced/Separated individuals were in the poor insight group compared to the good insight group.

Table 3 compares various clinical variables between the groups. Patients with poor insight had a significantly longer period of untreated illness and a shorter duration of treatment compared to those with good insight. Also, the poor insight group had a significantly higher total Y-BOCS score and compulsion subscale score.

The presence of symptom dimensions in OCD patients with good and poor insight is compared in Table 4. Overall, no statistically significant differences were observed across various symptom dimensions between the two groups as shown in Table 5.

Sexual/religious symptoms showed a statistically significant negative correlation with functioning, as shown in Table 6.

Discussion

The present study was conducted on 100 OCD patients to evaluate the relationship of symptom dimensions with insight and functioning. 78% of the study sample had good insight, while 22% had poor insight, which is consistent with earlier studies reporting that 9–45% of OCD patients have poor insight.[3-7,19-25]

Patients with poor insight also had longer untreated illness and shorter treatment duration, which highlights the impact of insight on treatment-seeking behavior and adherence. Poor insight could result in delay, including recognizing the need for help and therefore delay in the initiation of effective treatment. This is consistent with the finding in the previous study[22] which also reported that poor insight patients had a long span of untreated illness. Symptom severity was significantly higher in poor insight patients, as evidenced by higher Y-BOCS total scores and compulsion subscale scores, as patients with poor insight have reduced resistance to compulsions, which leads to increased symptom severity. These findings are in harmony with previous studies.[4,6,7,12]

In this study, no significant differences were observed in symptom dimensions between good and poor insight groups, suggesting that insight level does not meaningfully influence the type of OCD symptoms. Although contamination and cleaning symptoms were somewhat more frequent in the poor-insight group, and aggression, symmetry, and sexual/religious obsessions appeared more common in the good-insight group, none reached statistical significance. Cherian *et al.* (2012)[6] similarly reported higher rates of contamination fears and washing compulsions in poor-insight patients ($p < 0.001$) and more aggressive obsessions in those with good insight ($p < 0.001$). Jakubovski *et al.* (2011)[5], in a larger sample (n = 824), also found no significant correlation between insight and DY-BOCS dimension scores, except for a link between poor insight and hoarding. While several studies noted greater hoarding symptoms among poor-insight patients,[3,6,12,26] however, no participant in the present study exhibited hoarding features. Alonso *et al.* (2008)[4] observed more contamination and cleaning symptoms in good-insight patients, but no significant differences across other dimensions. Overall, the relationship between insight and OCD symptom dimensions still remains inconclusive.

In the current study, among the symptom dimensions, sexual and religious obsessions had the most substantial negative impact on functioning. A possible explanation for this could be that such obsessions- particularly those involving fears of blasphemy or inappropriate sexual thoughts- can cause intense distress. This may lead patients to avoid situations where they fear these thoughts might be triggered. Additionally, these obsessions are often accompanied by feelings of deep shame or guilt, making it difficult for individuals to seek help or engage in social interactions. Other symptoms, such as aggression-related obsessions, showed a moderate trend toward impairing functioning, although this was not statistically significant in the present study. This may be because aggressive thoughts often result in avoidant behaviors. In contrast, contamination, symmetry, and miscellaneous symptoms showed only minimal effects on functioning in this study. This suggests that individuals with these symptoms may still be able to maintain some level of social and occupational functioning, possibly because these symptom types may attract less societal stigma

Table 3: Comparison of clinical variables between groups

Clinical variable (in Years)	Group		Independent t test	
	OCD with Good Insight Mean (SD)	OCD with Poor Insight Mean (SD)	t	p-value
Age of onset	25.24 (8.619)	22.36 (7.480)	1.540	0.132
Duration of illness	7.15 (6.302)	7.64 (5.242)	-0.364	0.718
Duration of untreated illness	2.22 (1.877)	6.36 (4.124)	-6.782	0.000*
Duration of treatment	4.94 (5.560)	1.27 (1.804)	3.036	0.003*
Y-BOCS Total score (Range: 0-40)	26.72 (7.599)	30.09 (3.490)	-2.017	0.046*
Y-BOCS Obsession subscale score (Range: 0-20)	14.14 (3.702)	13.50 (2.198)	0.773	0.441
Y-BOCS Compulsion subscale score (Range: 0-20)	12.58 (4.232)	16.59 (1.764)	-4.331	0.000*

Table 4: Comparison of the presence of different symptom dimensions of DY-BOCS between groups

Symptom dimensions of DY-BOCS	Group		Chi square/Fisher's Exact test	
	OCD with Good Insight n (%)	OCD with Poor Insight n (%)	X2	p value
Contamination and cleaning	52 (66.7%)	19(86.4%)	3.233	0.109
Hoarding and collecting	0 (0%)	0(0%)	-	-
Symmetry and ordering	22 (28.2%)	3 (13.6%)	1.943	0.264
Aggression	25 (32.1%)	3 (13.6%)	2.886	0.111
Sexual and religious	19 (24.4%)	3 (13.6%)	1.248	0.388
Miscellaneous	6 (7.7%)	2 (9.1%)	0.044	1.000

Table 5: Correlation among the severity of symptom dimensions of DY-BOCS and Y-BOCS Item-11 (Insight) in patients of OCD

Symptom dimensions of DY-BOCS	Y-BOCS Item-11 (Insight)	
	Pearson Correlation Coefficient (r)	p-value
Contamination and cleaning	0.037	0.712
Hoarding and collecting	-	-
Symmetry and ordering	-0.143	0.157
Aggression	-0.078	0.440
Sexual and religious	-0.091	0.368
Miscellaneous	0.068	0.502

None: of the symptom dimensions demonstrated a statistically significant correlation with insight, as shown in Table 5.

or cause fewer direct disruptions in work-related activities. These findings are comparable to a study done by Rosa *et al.* (2012), who reported that sexual/religious and hoarding symptoms had the strongest negative effects on social functioning.[13]The study's cross-sectional design precludes causal inference between insight, symptom profile, and functioning. The modest sample size and absence of hoarding cases may limit generalizability. Self-reported avoidance behaviors and treatment adherence were not quantified, which might have clarified mediating pathways. Future longitudinal research with larger

Table 6: Correlation among symptom dimensions of DY-BOCS and functioning (SOFAS score) in patients with OCD

Symptom dimensions of DY-BOCS	SOFAS score	
	Pearson Correlation Coefficient (r)	p-value
Contamination and cleaning	-0.128	0.203
Hoarding and collecting	-	-
Symmetry and ordering	-0.060	0.552
Aggression	-0.183	0.068
Sexual and religious	-0.343	0.000*
Miscellaneous	-0.043	0.674

*Correlation is significant (at p-value ≤0.05)

and more diverse samples should examine neuro-cognitive and cultural moderators of insight and functioning in OCD.

Conclusion

In this study, poor insight was associated with higher symptom severity and compulsions, divorce/separation, longer duration of untreated illness and shorter duration of treatment. Level of insight is not significantly associated with either the presence or severity of specific symptom dimensions, reinforcing the inconclusive nature of the relationship between insight and OCD symptomatology. Notably, the study found that certain symptom dimensions, particularly sexual-religious obsessions, were significantly associated with poorer functioning. This underscores the functional burden imposed by specific types of obsessive-compulsive symptoms, independent of overall illness severity or level of insight. These findings highlight the importance of comprehensive symptom assessment in routine clinical practice to more effectively address functional outcomes in the management of OCD.

Ethical Considerations

Ethical approval for the study from the Institutional Ethical Committee (approval number: 187/MC/EC/2023) was obtained before the start of the study (on April 05, 2023). Participants were informed about the purpose of the study, and written informed consent was obtained from all participants prior to the study. The study was conducted in accordance with the ethical principles outlined in the World Medical Association's Declaration of Helsinki for research involving human participants.

Patient Consent

Written informed consent was taken from each participant in the study. They were assured of the complete confidentiality of their identity, information, and the option of withdrawing from the study at any point in time.

Acknowledgment

Author Approval Statement

All authors have read and approved the final manuscript. Each author meets the authorship criteria, and the manuscript represents honest and original work.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

Sources of Funding

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author's Contributions

Concept, design, literature search: Singh P, Kumar M; Data acquisition: Kumar M, Bawankar N, Kour H; Data analysis: Gupta S; Manuscript preparation: Kumar M, Bawankar N; Manuscript editing and manuscript review: Singh P; Guarantor: Kumar M

References

1. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders: DSM-5*. 5th ed. Arlington (VA): American Psychiatric Publishing; 2013, p.237.

2. Rosario-Campos MC, Miguel EC, Quatrano S, *et al*. The Dimensional Yale-Brown Obsessive-Compulsive Scale (DY-BOCS): an instrument for assessing obsessive-compulsive symptom dimensions. Mol Psychiatry 2006; 11: 495–504.

3. Kishore VR, Samar R, Reddy YCJ, *et al*. Clinical characteristics and treatment response in poor and good insight obsessive-compulsive disorder. Eur Psychiatry 2004; 19: 202–8.

4. Alonso P, Menchón JM, Segalàs C, *et al*. Clinical implications of insight assessment in obsessive-compulsive disorder. Compr Psychiatry 2008; 49: 305–12.

5. Jakubovski E, Pittenger C, Torres AR, *et al*. Dimensional correlates of poor insight in obsessive-compulsive disorder. Prog Neuropsychopharmacol Biol Psychiatry 2011; 35: 1677–81.

6. Cherian AV, Narayanaswamy JC, Srinivasaraju R, *et al*. Does insight have specific correlation with symptom dimensions in OCD? J Affect Disord. 2012; 138: 352–9.

7. Matsunaga H, Kiriike N, Matsui T, *et al*. Obsessive-compulsive disorder with poor insight. Compr Psychiatry 2002; 43: 150–7.

8. De Berardis D, Campanella D, Gambi F, *et al*. Insight and alexithymia in adult outpatients with obsessive-compulsive disorder. Eur Arch Psychiatry Clin Neurosci. 2005; 255(5): 350–8.

9. Elvish J, Simpson J and Ball LJ. Which clinical and demographic factors predict poor insight in individuals with obsessions and/or compulsions? J Anxiety Disord. 2010; 24(2): 231–7.

10. Stengler-Wenzke K, Kroll M, Matschinger H, *et al*. Subjective quality of life of patients with obsessive-compulsive disorder. Soc Psychiatry Psychiatr Epidemiol. 2006; 41: 662–8.

11. Srivastava S, Bhatia MS, Thawani R, *et al*. Quality of life in patients with obsessive-compulsive disorder: A longitudinal study from India. Asian J Psychiatr. 2011; 4: 178–82.

12. Fontenelle JM, Harrison BJ, Santana L, *et al*. Correlates of insight into different symptom dimensions in obsessive-compulsive disorder. Ann Clin Psychiatry 2013; 25: 11–6.

13. Rosa AC, Diniz JB, Fossaluza V, *et al*. Clinical correlates of social adjustment in patients with obsessive-compulsive disorder. J Psychiatr Res. 2012; 46: 1286–92.

14. Schwartzman CM, Boisseau CL, Sibrava NJ, *et al*. Symptom subtype and quality of life in obsessive-compulsive disorder. Psychiatry Res. 2017; 249: 307–10.

15. Guillen-Font MA, Cervera M, Puigoriol E, *et al*. Insight in obsessive-compulsive disorder: Relationship with socio-demographic and clinical characteristics. J Psychiatr Pract. 2021; 27(6): 427–38.

16. Goodman WK, Price LH, Rasmussen SA, Mazure C, Fleischmann RL, Hill CL, *et al*. The Yale–Brown Obsessive Compulsive Scale. II. Validity. Arch Gen Psychiatry. 1989;46(11):1012-6.

17. Storch EA, Rasmussen SA, Price LH, Larson MJ, Murphy TK, Goodman WK. Development and psychometric evaluation of the Yale-Brown Obsessive–Compulsive Scale – Second Edition. Psychol Assess. 2010;22(2):223-32.

18. Morosini PL, Magliano L, Brambilla L, Ugolini S, Pioli R. Development, reliability and acceptability of a new version of the DSM-IV Social and Occupational Functioning Assessment Scale (SOFAS) to assess routine social functioning. Acta Psychiatr Scand. 2000;101(4):323-9.

19. Marazziti D, Dell'Osso L, Di Nasso E, *et al*. Insight in obsessive-compulsive disorder: A study of an Italian sample. Eur Psychiatry 2002; 17: 407–10.

20. Eisen JL, Rasmussen SA, Phillips KA, *et al*. Insight and treatment outcome in obsessive-compulsive disorder. Compr Psychiatry 2001; 42: 494–7.

21. Catapano F, Perris F, Fabrazzo M, *et al*. Obsessive-compulsive disorder with poor insight: A three-year prospective study. Prog Neuropsychopharmacol Biol Psychiatry 2010; 34: 323–30.

22. Prasad C, Gupta B, Nischal A, *et al*. A comparative study of psychopathology and functioning in patients of obsessive–compulsive disorder with good and poor insight from a tertiary care center in North India. Indian J Health Sci Biomed Res. 2020.

23. Foa EB, Kozak MJ, Goodman WK, *et al*. DSM-IV field trial: Obsessive-compulsive disorder. Am J Psychiatry 1995; 152: 90–6.

24. Storch EA, Milsom VA, Merlo LJ, *et al*. Insight in pediatric obsessive-compulsive disorder: associations with clinical presentation. Psychiatry Res. 2008; 160: 212–20.

25. Jacob ML, Larson MJ and Storch EA. Insight in adults with obsessive-compulsive disorder. Compr Psychiatry 2014; 55(4): 896–903.

26. de Avila RCS, do Nascimento LG, Porto RLM, *et al*. Level of insight in patients with obsessive-compulsive disorder: An exploratory comparative study between patients with "good insight" and "poor insight". Front Psychiatry 2019; 10: 413.

A Qualitative Study of Gender Disparity of Substance Use in Children: Understanding Diverging Pathways and Influences

Anshu Sharma*, Satyananda Panda

Sikkim University, Gangtok, Sikkim, India.

Abstract

In India, substance abuse among children is a growing health concern with significant impact on the mental well-being, health, academic performance and social functioning. This study examines the gender disparity of substance abuse among Indian children, with the focus on the initiating factors, pattern of use, reason for continuation, and psychosocial outcomes between both boys and girls. The present study focuses on the qualitative approach and draws on semi-structured questionnaires. The sample consists of 20 participants (10 girls and 10 boys) receiving de-addiction rehabilitation treatment in SPYM organization, Delhi. Findings of the study reveal a notable gender- based disparity in both types of substance abuse and socio-environmental triggers that influence the consumption of drugs. The study reveals that while boys exhibit higher deviant behavior linked to substance use, both genders are equally vulnerable on an individual level. Environmental factors influence addiction risk in both genders, with peer pressure and family dynamics having a greater impact on boys. These findings highlight the need for gender-sensitive, context-specific prevention and intervention strategies.

INTRODUCTION

Worldwide, there has been a rapid increase in the consumption of drugs, especially among underage children. Substance abuse is defined as 'harmful and hazardous use of psychoactive substances', such as illicit drugs and alcohol.[1] A person consuming the substance is habituated and uses substances in a way that is extremely risky as well as damaging for the user and the people around them. As per the World Drug Report (2021), the year 2019 has observed a rapid rise of 22% of people who took substances was observed from the year 2010 and there has been an increase in drug trafficking and substance abuse worldwide.[2] India got caught in this destructive cycle of abuse, with the number increasing day by day. It is estimated that 1.58 crore children between the ages of 10 and 17 in India struggle with substance addiction, including cannabis.[2] In addition, according to a Ministry of Social Justice and Empowerment (2019) report, 2.8% of Indians between the ages of 10 and 75 (or 31 million people) already consume cannabis.[3] Substance abuse is one of the main reasons that

claim lives on the streets, especially among children. According to the Save the Children, India report,[4] substance abuse is reported as a major health problem in this segment of the population. These children indulged in substance use at any time in their lives, and it started at the minimum age of 5 years old.[5]

Substance use affects both men and women differently. There are various aspects in which gender disparity is observed, such as biological, psychological and social aspects. Although it is observed that substance-use disorder is reported higher among men as compared to women, women with substance-use disorder represent a more vulnerable profile, where they are less likely to undergo treatment.[6] Substance abuse among women was not recognised until recently, as most studies focused on the context of substance abuse among males.[7] Men and women also differ differently for their consumption of substances. Talking about the prevalence and pattern of substance abuse, men are observed to have a higher prevalence and abuse illicit drugs, whereas women are observed to rely on licit or prescriptive drugs. Considering the difference in terms of the psychological and emotional triggers, men usually consume drugs due to social pressure, peer pressure, or thrill seeking, but women consume substances as a self-medicated way to overcome stress, trauma, anxiety, or depression. For social and environmental reasons, men are accepted in society for substances such as alcohol or recreational drugs, hence allowing them to seek treatment, whereas substance abuse among women is considered taboo, which further restricts them from seeking treatment. Therefore, gender has implications for health issues across the lifespan of a person in terms of norms, roles, and relations. It also influences a person's willingness to take risks and health-seeking behavior, exposure to health risks, and vulnerability to disease.

Substance abuse among children is a global concern; context-specific patterns shaped by the socio-cultural and economic factors need to be taken into consideration. Although existing literature addresses the growing incidence of substance abuse, there is a scarcity of literature on the gender disparity of substance abuse, especially among chil-

dren. The impact of gender disparity on substance abuse is complex and depends on multiple factors. These areas are not explored much, especially in the Indian context, where substance abuse among girls is considered taboo and is often neglected, hence minimally researched. Gender plays a crucial role in shaping adolescent exposure to substances, their coping mechanism, peer influences, and prevention and rehabilitation services. Therefore, the aim of the study is to understand the gender disparity in the diverse pathways and influence of substance abuse. The first objective of the study is to focus on the different pathways that lead to gender differences in substance abuse and the second objective of the study is to understand the influence of substance abuse on both genders.

Methodology

Study Design

The participants were selected through purposive sampling according to the purpose of the study and to ensure representativeness in the study. The study uses a qualitative multiple case study where multiple participants (10 boys and 10 girls) were interviewed. Case study design helps in in-depth exploration of complex issues, multiple cases ensure the comparability of the data within and across data and it helps in gaining a holistic perspective. It also involves an investigation of an exclusive system, which is used to examine a situation inside a certain context and time frame.[8,9] Hence, it is a more suitable approach in this study. A retrospective approach was used as where the participants were required to recall their substance abuse experience, family situations and peer relations.

Participants

The participants selected were the children from the lower socioeconomic strata in Delhi, where families were involved in jobs like daily wage workers, labor, house helpers, etc. 20 children (10 girls and 10 boys) were selected. The children selected in the study have been cannabis consumers for more than 1 year. The participants selected in the study are the newly admitted children (to avoid the influence of

the treatment on the study) in the SPYM organisation, Delhi. There are two separate centres for the treatment of boys and girls with addiction. The mean age of the participants is 14.4 years for girls and 13 years for boys.

Ethical Approval

This study has received ethical approval from the Institutional Ethical Committee (IEC) of Sikkim University. Informed consent was obtained from participant parents and the organization prior to inclusion in the study.

Procedure

First, permission was sought from the centre's coordinator. Children who are newly admitted (1–2 days of admission) were identified and studied, to minimize the compounding bias dues to treatment. After that, each participant was interviewed separately in a separate room so they wouldn't feel uncomfortable disclosing their prior experiences. After being informed about the study, the participants were given the assurance that they could leave the study at any moment if they so desired. As a result, the interviewer read and clarified the consent letter to the parents and the organization's project manager in a preferred language (Hindi and English). Before they signed the consent form, they were asked if they had any questions.

Initially, questions concerning personal information and family history were given to the participants. The interviewer then moved on to discuss the participants' past substance use and the factors that led to drug misuse. A semi-structured questionnaire was developed focusing on the initiation of the substance, peer pressure, school environment, family issues or conflicts, behavior, personal issues, and others. The interviews were all conducted in the morning between 10 a.m. to 12 noon. The duration of each interview ranges from 30 to 40 minutes.

Data Collection and Analysis

Approval from the selected university's research committees to conduct the study was obtained. In-depth interviews were used in this study to gather data in order to look into people's opinions, experiences, and/or motivations about particular topics.[10]

A semi-structured schedule guided the interview process. The data were analysed using qualitative thematic analysis according to Braun and Clarke's (2006) six-step method.[11] The transcripts were read many times and the initial memo was taken. A data-driven approach was used to generate verbatim codes and identify themes.

A scheduled method was used to collect the interview responses and was analysed using Braun and Clarke's sex step method.[11] The transcripts were read many times and the initial memo was taken. A data-driven approach was used to generate verbatim codes and identify themes.

Results

Six main themes have emerged from the data and each theme has further sub-themes. These themes and sub-themes help in identifying the factors responsible for the gender disparity among children in substance use. The main themes that emerged were: Individual vulnerability, family as a factor into substance abuse, Deviant behavior, Environmental facilitation, Peer relation, and Positive insight into the future. A thematic network (Figure 1) of the themes and sub-themes is created to create an illustration for the factors associated with substance abuse.

Discussion

The aim of the study is to understand gender disparity and diverging pathways and influences that affect childhood substance use. The prevalence of the gender disparity in substance abuse is significantly higher in the Indian context; significantly, boys consume illicit drugs higher rate in comparison to girls.[12]

Theme 1: Individual vulnerability to substance abuse

The primary impact of the substance abuse is observed on the participants, both boys and girls, is that they themselves become vulnerable to the substance abuse.

Sub-theme 1.1. Early initiation

The present study shows that both girls and boys started consuming substances below the age of 12
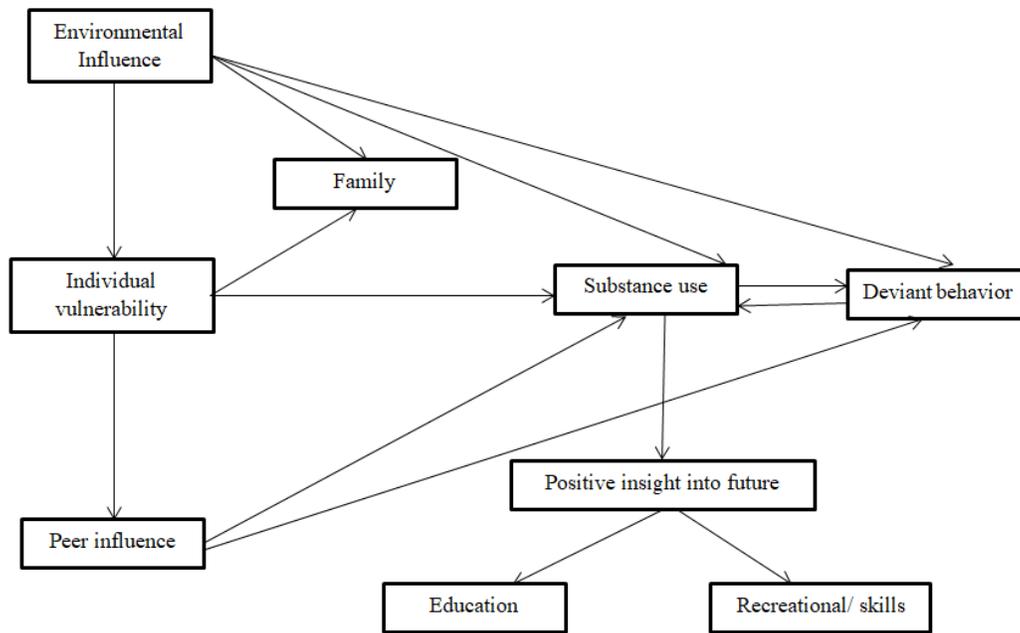
Figure 1: A thematic network of the factors associated with substance use

years. The average age of initiation in boys is 10.5 years, and in girls is 10.7 years. Early onset is related to early exposure to unhealthy and harmful habits. Children at this stage are not mentally developed to understand what they are doing and the consequences of their actions. Hence, this leads to unhealthy dependence on the drugs at an early stage.

Sub-theme 1.2. Withdrawn from education

The dropout rate is common among both genders. It is observed that education is the first thing that is most affected once the child is addicted. Schooling is not given much importance by the children after they go into addiction. The study shows that the majority of children have reached middle school and drop out after that. Children whose parents are into addiction, in such households too, education is not given much importance, as the dropout rate is observed to be higher in the families where education is not given much importance.[13]

Theme 2: Family as a Factor in Substance Abuse

Children observe behaviour from their surroundings. When they observe that a particular behaviour is normal in their surroundings, they themselves get involved in the behaviour without understanding the consequences of the behaviour.[14] The study reveals that family as a facilitator for addiction is observed in both genders.

Sub-theme 2.1. Familial facilitation

Family impacts both girls and boys equally, but through different approaches. Boys who develop attachment issues with family members are inclined toward substance abuse to fill the hole in their relationship.[15] Boys are more likely to observe a father who is into addiction, some with only daily tobacco consumption, some of them being occasional drinkers, or some who drinker regularly and become more fascinated with it than girls. One of the children's fathers died due to overconsumption of alcohol. Apart from the parents, the siblings of the children also engaged in the addiction. Some of them are consuming legal (alcohol) and illegal (smack, ganja, etc.) after reaching the legal age, whereas siblings who have not reached the legal age still consume drugs. Girls who experienced Adverse Childhood Experiences (ACEs) such as parental substance abuse and neglect are strongly linked to substance abuse that leads to developing low self-esteem, impulsivity, and attachment issues.[16]

Sub-theme 2.2. Familial relationship

The majority of children, especially boys, reported having a good and warm relationship with either parent, mostly with the mother, where the child reported that *"papa ke saath rishta accha nahi hai sirf mummy ke saath hai, papa nasha karke aate hai ladai hoti hai ghar me"* (I have a good relationship with my mother but not with my father, father is an alcoholic and every time he drinks we have a fight). Disrupted familial relationships are reported more by the girls as compared to the boys.[17] The majority of girls reported an association of shame and addiction. A girl reported that *"Parivar sharminda hai, aas paas ke logo ko bol diya hai mai rishtedaro ke ghar me hu"* (My family is ashamed of me, they have told the neighbours that I live separately from my relatives). Children mostly share a warm relationship with their siblings, but some children reported that consumption of the drugs has strained their relationship with their siblings, as one child reported that *"Bhai ke saath theek hai behen ke saath nahi, behen mana karti hai nasha karne se nahi manne se baat nahi karti hai"* (My relationship is good with my brother, not with my sister, my sister told me not to consume, but I didn't listen, so she doesn't talk to me now).

Sub-theme 2.3. Conflict between parents

Both the girls and boys reported that their parents shared abusive relationships, especially due to the father's alcoholic nature as One child reported that *"jab bhi papa nasha karke aate hai mummy ke saath ladai hoti hai"* (There is a fight between parents whenever father drinks). Apart from the addiction, financial strain is also the main important reason for the conflict, as a child reported that *"paise ko wajah se ladai hoti hai roz"* (Conflicts related to money occur).

Theme 3: Deviant Behaviour

Deviancy is observed more among boys as compared to girls. This is due to the fact that girls are under parental supervision, despite both young girls and boys spending equal amounts of time with their peers. Girls' deviant behavior decreases when they spend time with their parents, whereas spending time with parents does not have any impact on the boys' deviant behavior.[18]

Sub-theme 3.1. Running away tendency

Boys and girls have reported that at least once in their lifetime, they have run away from their home. Running away tendency is often a display of the child psychologically escaping from the environment that is toxic. The child tries to escape a difficult home environment and the negative consequences of the child's addiction that have been left on the family. Girls who ran away were more likely to have PTSD and have sex to survive, while boys were more likely to commit violent and drug-related crimes.[19] NCRB reported that children who run away from home are the ones who are often neglected and abused at their home or surrounding. One child reported *"haan, jab mai 6 saal ka tha tab ghar se bhaaga tha dosto ke chakkar me"* (Yes, when I was 6 years old, I ran away from my home due to my friends), another child reported that *"Haan baga tha 5-6 baar 2-2 mahine ke liye"* (Yes, I ran away from my home for 5-6 times that too for 2 months every time).

Sub-theme 3.2. Impulsivity

Children reported that they do not think before they act and take actions without much thought. Impulsivity increases the risk for drug abuse, and conversely, substance use produces acute and chronic changes in impulsivity.[20] Boys with histories of substance use show increased impulsivity and decreased inhibition, especially among users of cannabis or alcohol,[21] whereas girls with a history of substance abuse show increased levels of urgency-related impulsivity (i.e., acting on impulse during distress).[22] One of the participants reported that *"haan mann toh abhi bhi karta hai. Nahi toh apne aap ko nuksan pahuchane ka mann karta hai"* (Yes, I still feel like consuming it, otherwise I feel like hurting myself). Another child who stays with friends and consumes drugs reported that *"haan, chori se cheena hai bina kuch soche samjhe"* (Yes, I steal without thinking much).

Sub-theme 3.3. Guilty

Guilt plays a critical and gender differentiated role. Boys are more likely to experience behavioral guilt, such as about their actions and their consequences, like hurting someone, whereas girls are more likely to experience relational/ emotional guilt, like disap-

pointing others, breaking trust, or being a burden on loved ones.[23] In this study, a boy responded that *"log nashedi nashedi bolte hai isiliye wapas nahi jaane ka mann karta hai" (People labelled me as 'addict', that's why I don't feel like going back).* A girl reported that *"gharwale sharminda hai, logoko bola hai bahar rishtedaaro ke ghar pe hai" (Family is ashamed and they told everyone that I am at a relatives place).* This study also observes the behavioral guilt experienced by boys and relational guilt experienced by girls.

Sub-theme 3.4. Antisocial activities

Both boys and girls are likely to become involved in antisocial activities, as drugs push the children into criminal activities due to the need to support them financially. Boys exhibit a higher rate of overt aggression, such as violence, vandalism, and gang activities,[23] whereas girls show more covert or relational antisocial activity, such as lying, risky sexual behavior, or manipulating adults.[24] In the present study, the majority of the men were involved in antisocial activities such as snatching, robbery, gambling, etc., whereas a few girls were involved in gang activities and often involved in lying to their family or engaging in risky sexual behavior.

Theme 4: Environmental facilitation

Research suggests that environmental factors influence substance use behaviors. Differences in racial and socioeconomic status are examples of environmental factors that might contribute to substance abuse, including exposure and access to substances, neighbourhood disadvantage and disorder, and environmental barriers in treatment that contribute to the growth of substance abuse in the society.[25]

Sub-theme 4.1. Unsafe environment

The majority of the children are living in an environment where it is unsafe for their personal growth of the person. Children living in such conditions become numb to the outside crime and consider it a 'normal day-to-day activity'. Boys who grow up in high-risk environments are often involved in early substance use and externalising antisocial behavior.[23] One child reported, *"Area accha nahi hai, waha pe bohot saare log nasha karte hai" (Area is*

not good; there are a lot of people in my area who consume substances). However, the environment has an equivalent impact on the child's substance abuse despite gender. It is observed that girls report more higher risk in early substance initiation as compared to the boys.[26]

Sub-theme 4.2. Social taboo

Social taboo is associated with people becoming addicted. Once a person consumes drugs, they become an addict for their whole life. Even if they want to leave, the taboo is not letting them. These children are labelled as delinquent and troubled by society, which makes the child difficult to come out of the taboo. Social taboo is more relevant amongst girls' substance use despite being the ones who consume less than boys. One girl reported that *"parivaar wale sharminda hai, logo ko bol rakha hai padhne bahar bheja hai" (Family is ashamed they have told everyone that I have sent me to study outside).* This explains the cultural shame attached to girls that leads to facing harsher judgment and "moral panic".[27] A boy reported that *"haan, padosi pareshaan karte hai, nahi bhi nasha karne pe tokte hai" (Yes, neighbours tease a lot, even if I don't consume, they tease that I have consumed).* Social taboos among boys might make them receive more severe punishment from law and order. Social taboo often creates gender specific roles in society, hence creating gender differences.

Sub-theme 4.3. Weak law and order

Weak law and order often leads to a lack of supervision and enforcement of the law and provides liability to those who sell drugs to minors. This leads to the normalcy of substance abuse in society. According to the UNODC report (2024) countries with porous law enforcement, children as young as 10 years are exposed to the street- level substance sale and abuse.[28] This also leads to the peer-led exploration of harmful and illicit substances like cannabis or inhalants. Boys are more likely to engage in distribution, increasing the addiction risk, whereas girls, especially runaways, are pulled into drug-for-sex exchanges or exploitation. Hence, having a strong law and order is important to provide stability, safety and prosperity in the society.

Theme 5: Peer relations

Cultural norms highlight the discrepancy between genders as they dictate a double standard for the monitoring and punishment of deviance for girls and boys. Peer substance use is one of the strongest predictors of a child's substance initiation across different cultures.[29] This discrepancy is observed to play different roles between boys and girls. On one hand, boys have a greater opportunity to engage with classmates who encourage them into substance abuse, while female adolescents are often protected from engagement with their peers.[30]

Sub-theme 5.1. Peer influence on addiction

Children, especially boys who are into substance abuse, have shown that they have been negatively influenced by their peers to engage in the risk-taking behavior, with one of the children stating that *"nahi wo humhe galat raste pe le jaa rahe hai, galat kaam karwa rahe hai jaise chori nasha" (No, they are misguiding us, they make us steal and do drugs)*. Children's perception of the importance of peer approval and use is more often overestimated, which fuels the idea that "everyone is doing it". Girls often start consuming substances through their close relationships, such as boyfriends or female friends.[31] One girl reported that *"mere boyfriend ke saath kiya tha pehli baar" (Started consuming it with my boyfriend first)*.

Sub-theme 5.2. Lack of a good friend

Children whose friend circle involves both high levels of positive and negative friendships are more likely to become involved in substance abuse.[32] Other research also talks about that friendship intimacy has potential risk for adolescents regarding substance abuse, depending upon factors such as the characteristics of a friend.[33] Behavioral issues are more likely to lead to substance use when close friends are also users. In the absence of strong connections with peers who do not use substances, at-risk adolescents may gravitate towards deviant social groups. This study reveals that the majority of the participants, both girls and boys, have friends who are addicted, due to which they gravitate towards substance abuse themselves.

Theme 6: Positive insight into the Future

During addiction recovery, moving forward with a positive outlook or positive mindset plays a crucial role. It is grounded in purpose, hope and realistic goals. This psychological orientation leads to a reduction in the relapse rate and improves resilience and motivation. During the treatment, encouraging the patients to set small personal goals, engaging in skill-building, and discovering the purpose of life shifts the focus from the past trauma or failure towards a positive future.

Sub-theme 6.1. Education as a factor in leaving addiction

School-based prevention is found to be an effective strategy for the reduction of substance abuse among children.[34] Research shows that both boys and girls who have lower educational attainment are linked with more drug abuse, whereas higher educational attainment leads to a decreased likelihood of becoming involved in substance abuse.[35,36] Education helps in developing awareness, critical thinking, decision-making skills, and provides a structured, positive environment in the life of a child. Education can also be integrated into the rehabilitation program, which helps them to recover and reintegrate into society.

Sub-theme 6.2. Learning recreational activities to help them leave addiction and become financially independent

Recreational activities can contribute to their recovery and future stability by focusing on redirecting focus and energy, building self-esteem and confidence, social connection and social support, and skill development. By developing and enhancing their skills, they can turn those skills into professionalism and become financially independent. This study shows that where the majority of boys are involved in playing outdoor games, girls are involved in household work such as cooking. Research has shown that children participating in multiple leisure activities exhibit lower rates of substance abuse and better mental health outcomes as compared to those who are not involved in leisure activities.[37-38]

Limitation

Social desirability will be observed as higher on the sensitive topics like substance use and family behaviors. This might be experienced among the girls, who may experience greater stigma. Secondly, due to geographical limitations from lower socioeconomic strata in Delhi, it limits the generalizability of the results to other urban or rural settings in India or different socioeconomic contexts. Third, the small sample size in the study limits the generalizability of the findings among children from the community. Fourth, contextual variables such as the availability of substances, school environments, and community-level interventions were not studied comprehensively, which may have influenced the outcomes. Finally, the study does not consider environmental and familial factors that might influence mental health issues, trauma exposure, or developmental disorders, which can co-occur with substance use and vary by gender.

Conclusion

The findings show gender-specific differences in substance use among children from disadvantaged socioeconomic backgrounds in Delhi. The research shows that although both boys and girls are individually vulnerable to substance use, boys exhibit a notably higher prevalence of deviant behaviors associated with substance use. Factors in the environment- such as exposure, availability, and lenient social norms- significantly contributed to early initiation and continued use. Peer influence emerged as a more significant factor, reflecting children's stronger reliance on social and peer connections within their communities. Additionally, familial substance use indicates distinct gender pathways of risk within the family setting, influencing both girls and boys equally but differently. Other factors like early initiation, deviant behavior, or environmental facilitation show a considerable amount of impact on both boys and girls. These findings show the importance of developing intervention strategies that are sensitive to gender and specific to the context, taking into account the actual experiences of children living in marginalized urban areas. Programs aimed at prevention and treatment must consider the distinct

social and family structures that influence substance use behavior among boys and girls in these communities. By integrating gender-specific risk factors and local socio-environmental contexts, public health initiatives can be more effectively designed to reduce early substance use and its long-term effects on at-risk populations.

Availability of Data and Materials

Due to the potential for participant identification, the datasets developed and evaluated during the present study are not publically available; however, they can be obtained from the corresponding author upon reasonable request.

References

1. World Health Organization. Substance Abuse. 2025, July. Available from: http://www.who.int/topics/substance_abuse/en/.
2. Ahmad N, Bano R, Agarwal VK, Kalakoti P. Substance abuse in India. 2009.
3. Ambekar A, Agrawal A, Rao R, Mishra AK, Khandelwal SK, Chadda RK. National Survey on Extent and Pattern of Substance Use in India. Magnitude of Substance Use in India. New Delhi: Ministry of Social Justice and Empowerment, Government of India. 2019
4. Sharma K. Two lakh street children lured into substance abuse in Delhi. The Hindu. 2021, November. https://www.thehindu.com/news/cities/Delhi/Two-lakh-street-children-lured-into-substance-abuse-in-Delhi/article60343777.ece
5. Rao U. Making the global city: Urban citizenship at the margins of Delhi. Ethnos. 2010;75:402-424. Available from: https://doi.org/10.1080/00141844.2010.532227
6. Fonseca Casals F, Robles-Martínez M, Tirado Muñoz J, Alías i Ferri M, Mestre-Pintó JI, Coratu AM, Torrens M. A gender perspective of addictive disorders. Current Addiction Reports. 2021;8:89–99. Available from: https://doi.org/10.1007/s40429-021-00357-9
7. Agrawal A, Neale MC, Prescott CA, Kendler KS. Cannabis and other illicit drugs: co-morbid use and abuse/dependence in males and females. Behavior genetics. 2004;34:217-228. Available from: https://doi.org/10.1023/B:BEGE.0000017868.07829.45
8. Babbie E. The practice of social research (14th ed.). Cengage Learning. 2016
9. Denscombe M. The good research guide for small-scale social research projects (5th ed.). Open University Press. 2014
10. Silverman D. Doing qualitative research. Sage Publications. 2000
11. Braun V, Clarke V. Using thematic analysis in psychology.

Qualitative research in psychology. 2006;3:77-101. Available from: https://doi.org/10.1191/1478088706qp063oa

12. Jasani PK, Jadeja YM, Patel NM, Jadeja DY, Shrimali JB, Purani SK. Prevalence of substance abuse among adolescents of urban and rural community in Surendranagar district, Gujarat. International Journal Of Community Medicine And Public Health. 2019;6:1970–1974. Available from: https://doi.org/10.18203/2394-6040.ijcmph20191453

13. Casas-Gil MJ, Navarro-Guzman JI. School characteristics among children of alcoholic parents. Psychological Reports. 2002;90:341-348. Available from: doi:10.2466/pr0.2002.90.1.341.

14. Lander L, Howsare J, Byrne M. The impact of substance use disorders on families and children: from theory to practice. Social work in public health. 2013;28:194–205. Available from: https://doi.org/10.1080/19371918.2013.759005

15. Schindler A. Attachment and substance use disorders-theoretical models, empirical evidence, and implications for treatment. Frontiers in psychiatry. 2019;10: 727. Available from: https://doi.org/10.3389/fpsyt.2019.00727

16. Anda RF, Felitti VJ, Bremner JD. The enduring effects of abuse and related adverse experiences in childhood: A convergence of evidence from neurobiology and epidemiology. European archives of psychiatry and clinical neuroscience. 2006;256:174-186. Available from: https://doi.org/10.1007/s00406-005-0624-4

17. Skeer M R, McCormick MC, Normand SLT, Mimiaga MJ, Buka SL, Gilman SE. Gender differences in the association between family conflict and adolescent substance use disorders. Journal of adolescent health. 2011;49:187-192. Available from: doi: 10.1016/j.jadohealth.2010.12.003

18. Worthen MG. Gender differences in parent–child bonding: Implications for understanding the gender gap in delinquency. Journal of Crime and Justice. 2011;34:3-23. Available from: https://doi.org/10.1080/0735648X.2011.554744

19. Whitbeck LB, Hoyt DR, Johnson K, Chen X. Victimization and posttraumatic stress disorder among runaway and homeless adolescents. Violence and Victims. 2007;22:721- 734

20. Perry JL, Carroll ME. The role of impulsive behavior in drug abuse. Psychopharmacology. 2008;200:1-26. Available from: doi: 10.1007/s00213-008-1173-0.

21. Clark DB, Cornelius JR, Kirisci L, Tarter RE. Childhood risk categories for adolescent substance involvement: a general liability typology. Drug and alcohol dependence. 2005;77:13-21. Available from: https://doi.org/10.1016/j.drugalcdep.2004.06.008

22. Stoltenberg SF, Batien BD, Birgenheir DG. Does gender moderate associations among impulsivity and health-risk behaviors?. Addictive behaviors. 2008;33: 252-265. Available from: https://doi.org/10.1016/j.addbeh.2007.09.004

23. Zucker RA, Heitzeg MM, Nigg JT. Parsing the under control disinhibition pathway to substance use disorders: A multilevel developmental problem. Child development perspectives. 2011;5: 248-255. Available from: https://doi.org/10.1111/j.1750-8606.2011.00172.x

24. Schulz N, Murphy B, Verona E. Gender differences in psychopathy links to drug use. Law and human behavior. 2016;2:159–168. Available from: https://doi.org/10.1037/lhb0000165

25. Mennis J, Stahler GJ, Mason MJ. Risky Substance Use Environments and Addiction: A New Frontier for Environmental Justice Research. International journal of environmental research and public health. 2016;13:607. Available from: https://doi.org/10.3390/ijerph13060607

26. Anderberg M, Dahlberg M. Gender differences among adolescents with substance abuse problems at Maria clinics in Sweden. Nordic Studies on Alcohol and drugs. 2016; 35:24-38. Available from: https://doi.org/10.1177/1455072517751263

27. Miller-Day M. Talking to youth about drugs: What do late adolescents say about parental strategies?. Family Relations. 2008;57:1-12. Available from: https://doi.org/10.1111/j.1741-3729.2007.00478.x

28. United Nations Office on Drugs. World Drug Report 2024 (Set of 3 Booklets). Stylus Publishing, LLC. 2024.

29. Simons-Morton BG, Farhat T. Recent Findings on Peer Group Influences on Adolescent Smoking. J Primary Prevent. 2010;31:191–208. Available from: https://doi.org/10.1007/s10935-010-0220-x

30. Byrnes JP, Miller DC, Schafer WD. Gender differences in risk taking: A meta-analysis. Psychological Bulletin. 1999;125:367–383. Available from: https://doi.org/10.1037/0033-2909.125.3.367

31. United Nations Office on Drugs and Crime (UNODC). World drug report 2021 eISBN: 9789210058032). 2021. Available from: https://www.unodc.org/res/wdr2021/field/WDR21_Booklet_2.pdf (open in a new window)

32. Hussong AM. Perceived peer context and adolescent adjustment. Journal of Research on Adolescence. 2000;10:391-415. Available from: doi:10.1207/SRA1004_02.

33. Ennett ST, Bauman KE, Hussong A, Faris R, Foshee VA, Cai L, DuRant RH. The peer context of adolescent substance use: Findings from social network analysis. Journal of research on adolescence. 2006;16:159-186. Available from: doi:10.1111/j.1532-7795.2006.00127.x.

34. Alarcó-Rosales R, Sánchez-SanSegundo M, Ferrer-Cascales R, Albaladejo-Blazquez N, Lordan O, Zaragoza-Martí A. Effects of a School-Based Intervention for Preventing Substance Use among Adolescents at Risk of Academic Failure: A Pilot Study of the Reasoning and Rehabilitation V2 Program. Healthcare (Basel, Switzerland). 2021;9:1488. Available from: https://doi.org/10.3390/healthcare9111488

35. Carliner H, Sarvet AL, Gordon AR, Hasin DS. Gender discrimination, educational attainment, and illicit drug use among US women. Social psychiatry and psychiatric epidemiology. 2017;52:279-289. Available from: https://

doi.org/10.1007/s00127-016-1329-x

36. Srivastava S, Kumar P, Rashmi Paul R, Dhillon P. Does substance use by family members and community affect the substance use among adolescent boys? Evidence from UDAYA study, India. BMC Public Health. 2021;21:1- 10. Available from: https://doi.org/10.1186/s12889-021-11911-5

37. Santini ZI, Meilstrup C, Hinrichsen C, Nielsen L, Koyanagi A, Koushede V, Madsen KR. Associations between multiple leisure activities, mental health and substance use among adolescents in Denmark: A nationwide cross-sectional study. Frontiers in behavioral neuroscience. 2020;14:593340. Available from: https://doi.org/10.3389/fnbeh.2020.593340

38. More A, Jackson B, Dimmock JA, Thornton AL, Colthart A, Furzer BJ. Exercise in the treatment of youth substance use disorders: Review and recommendations. Frontiers in Psychology. 2017;8:1839.

Effect of Yoga Practices on Anxiety, Forms of Adjustment and Suicidal Ideation among Young Adults

Pramod Kumar[1], Tanya Sharma[1], Pankaj Kumar[2], Madhu[2], Ravi P. Pandey[3*], Naveen[2], Swati[5], Vivek Singh[1], Deepak Kumar[1] and Komal Bumra[1]

[1]Department of Psychology, Central University of Haryana, Mahendragarh, Haryana, India.
[2]Department of Yoga, Central University of Haryana, Mahendragarh, Haryana, India.
[3]Department of Psychology, Central University of Haryana, Mahendragarh, Haryana, India.
[4]Department of Psychology, University of Lucknow, Lucknow, Uttar Pradesh, India.

Abstract

Background: Nowadays, it is being observed that yoga practices work as a protective factor against psychological and physical concerns such as stress, depression, anxiety, suicidal ideation, drug addiction, osteoarthritis, multiple sclerosis, asthma, irritable bowel syndrome, lymphoma, hypertension, and mental health problems. The experimental design of this research is a result of the literature suggesting a causal relationship between the positive impact of yoga and psychological issues. The present study involves variables, viz., anxiety, forms of adjustment, suicide ideation, and yoga practices.

Aim: The aim of this study was to explore the effect of yogic practices on anxiety, forms of adjustment, and suicide ideation of young adults.

Method: A Total of 31 participants participated in this experiment. The age range of the participants was 20 to 25 years, with a mean age of 22.32 years and SD was of 1.37. Within-group experimental design was applied to conduct the experiment. Anxiety questionnaire, Bell's Adjustment Inventory, and Rudd's Suicidal Ideation Scale measures were used to collect data. Descriptive statistics and paired sample t-test were calculated for the analysis.

Results: Results disclosed that yogic practices helped participants to reduce their suicidal thoughts and improve home adjustment.

Discussion: The findings of this study showed that yogic practices might play a crucial role in preventing suicide ideation and enhancing the health adjustment of the participants. Limitations and implications of the study have been discussed in the conclusion.

INTRODUCTION

Every individual has difficulties. The key factor in evaluating an individual's efficacy is not how much difficulty or bad luck they experience, but rather how they handle or adapt to life's obstacles. "An index of integration between needs and satisfaction, remains related to achievement, social acceptance, age, sex, economic security, and moral standards" is the definition of adjustment, a crucial psychological attribute.[1] Adjustment means regulating, adapting, or

settling. The word 'adjustment' comes from the late Latin ad-juxtare, derived from juxta, near, but early confused with a purported derivation from Justus, right. The behavioural process of balancing competing wants, or needs, versus environmental barriers, is referred to as adjustment in psychology. Occasionally, a person cannot change in position to the point where it seems unlikely that his wants would ever be met.

Human behaviour is reflected in adjustment, which is the means by which individuals attempt to preserve social and personal balance. It is necessary to make adjustments in order to meet diverse needs and preserve social harmony. With their inherent qualities, humans always strive to live in harmony with the natural world. Happiness and harmony within the family can be preserved with the ability to adapt to both society and other family members.[1]

However, students these days like to play on their phones rather than outside. It is possible that they might prefer to engage in less demanding yoga techniques instead of physically demanding exercise. Yoga plays a significant part in systematising the many bodily systems, which improves the body's capacity for adjustment. Numerous studies in the yoga sector have shown that practicing yoga improves one's capacity for adaptability. Research indicates that researchers who studied a variety of populations, including college students, blind kids, and school students, discovered that yoga had a beneficial impact on adjustment.[2,3]

The term "complementary medicine" describes a group of procedures and therapies that have not been explored by contemporary medicine.[4] Yoga, which means "unity of mind and body" in Sanskrit, has been practiced for 5,000 years in Eastern societies and has drawn a lot of interest from Western nations lately.[5] Numerous scientific and medical studies on yoga conducted in the last several decades have shown how effective it can be in treating a number of illnesses.[6] Research has shown that yoga or yogic practices can help with a variety of medical and psychological illnesses, such as drug addiction, osteoarthritis, multiple sclerosis, asthma, irritable bowel syndrome, lymphoma, hypertension, mental health problems, suicidal thoughts, adjustment problems, stress, and anxiety.[7-14]

Yoga is a mental, spiritual, and physical practice. It involves the application of asana (physical postures), pranayama (breathing exercises), niyama (social ethics), yama (personal ethics), and meditation (the discipline of calming the mind).[15] Ernst, Pittler, Stevinson, & White (2001) have described yoga as a three-part practice that includes mild stretching, breath-control exercises, and meditation as a mind-body intervention.[16] The form of yoga that is most commonly practiced in the West is called hatha yoga, and it incorporates meditation, pranayama (a breathing exercise), and asana (postures).[17] Yoga is a secular practice, despite its roots in Indian culture and religion.[16]

Forms of Adjustment

The family and society play a prominent role in an individual's physical and mental well-being; hence, the following forms of adjustment can be used to encounter the problems of individuals. These are mentioned below.[18]

- Family adjustment.
- Emotional adjustment.
- Social adjustment.
- Health adjustment (Figure 1).

Family issues pertain to specific aspects such as the acceptance, overprotection, or negligence of an individual towards family members. People use family adjustment tools to handle family problems. People experience, such as anger, violence, melancholy, anxiety, and an inferiority complex, are referred to as emotional difficulties. To tackle these emotional difficulties, individuals can use emotional adjustment techniques (Figure 2).[18]

Yogic Practices and Adjustment

According to research, yoga has a positive effect on adjustment in a range of populations, including college students, blind children, and school students.[2,3], while Roy, Mukhopadhyay, and Ghosh (2022) reported that there was no significant impact of yoga on social, emotional, and educational adjustment of the participants.[3] Bhuyan and Vineeta (2022) discovered that yogic practices have a positive effect on the adjustment of secondary school students. Previous research findings have demonstrated a discrepancy.[19] Therefore, empirical data are required to
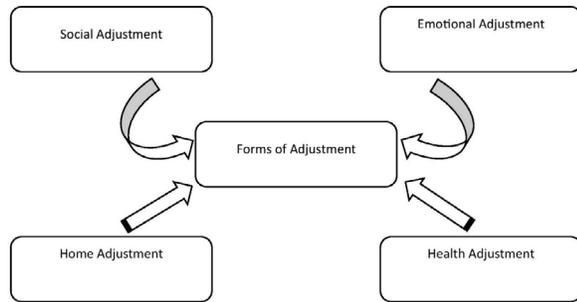
Figure 1: Represents forms of adjustment
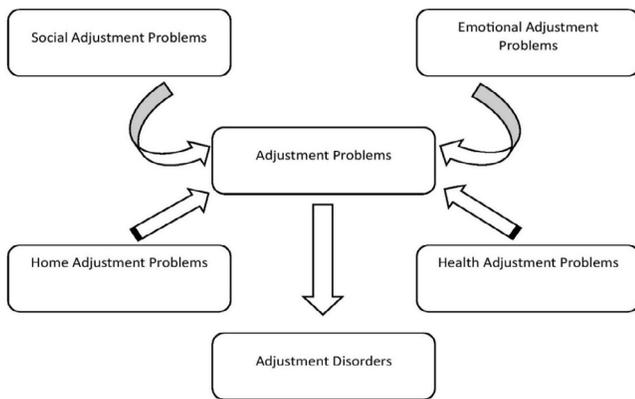
Figure 2: Represents the adjustment problems develop adjustment disorders

have a comprehensive understanding of the impact of yogic practices on adjustment.

Yogic Practices, Anxiety and Stress

Consistent yogic practice is the best strategy for developing a steady sympathovagal balance. Yoga poses, meditation, and pranayama all help the body relax and balance the sympathetic nervous system. Thus, achieving sympathovagal equilibrium is the main physiological goal of yoga.[20] According to studies by West (1979), Dighore and Gadkari (2013), meditation is a yogic technique that can lower anxiety and arousal.[21,22] Yoga was found to lessen anxiety in students, but only in male students, according to US Ray, Purkayastha, Asnani, Tomer, Prashad, Thakur, & Selvamurthy (2001).[23] Yoga is beneficial for reducing stress, anxiety, and depression, as per the study by Shohani *et al.* (2018).[24] Yoga has been shown to reduce stress by promoting relaxation, emotional regulation, and resilience. According to a study done by Castellote-Caballero *et al.* (2024), a 12-week yoga intervention can

significantly reduce perceived stress and anxiety and improve emotional well-being in university students.[15] Chauhan *et al.* (2025) concluded that by including yoga and meditation practice in the daily lives of medical students, educational institutes can provide a more supportive environment that focuses on students' overall health.[17] Yoga, defined by the practice of postures, generally leads to some reductions in anxiety and depression in youth regardless of health status and intervention characteristics, according to a systematic review by James-Palmer *et al.* (2020).[34] From the perspective of the Cognitive Appraisal Model (Lazarus & Folkman, 1984), yoga helps individuals interpret stressors with less threat perception.[39]

Yogic Practices and Suicidal Ideation

Suicide is an actual, common, and worldwide social incident. It is the act of choosing to end one's own life. It is produced in a state of extreme psychological stress when a person is unable to look at their issues clearly or come up with a different plan of action. It is morally neutral, neither right nor evil, nor does it represent a flaw in one's character. It's just an imbalance between coping mechanisms and suffering.

Thoughts to take one's own life are considered suicidal ideation, also known as suicidal thought. These might be just as detailed as a well-thought-out plan, only without the suicide part. While most individuals experiencing suicidal thoughts do not really attempt suicide, others do. Suicidal ideation can take many forms, from brainstorming to detailed planning, role-playing, and futile efforts that are either fully intended to succeed but are let down by reality or they can be purposefully intended to fail. Appropriately enough, suicidal ideation refers to wishing to terminate one's life or considering suicide but not really having plans to do so.[25]

Suicidal thoughts can arise when a child's anxiety over getting better grades leads to failure or when they don't meet the expectations of their parents and teachers. Recently published research showed that suicidal ideation or thoughts, tendency, and attempts were high among students.[26-31]

Elevated levels of adjustment problems, stress, depression, anxiety, and suicidal ideation characterise modern living.[26,32,14] In order to avoid these

issues, effort in this specific area is required. Yogic practices might be helpful to prevent these kinds of psychological concerns.

Gahlawat's (2017) research on students suggests that yoga exercises have a beneficial impact on students' prevalence of suicidal thoughts.[33] Ashish and Sharma (2023) concluded in their review article that regular practice of Hatha Yoga reduced suicidal thoughts of the participants.[34]

A person's cognitive, behavioural, autonomic, and somatic functioning can all be significantly altered by yogic practice, which combines 'surya namaskar, standing and sitting/meditation asanas, pranayama, mudra, and bandha'. This reduces anxiety, suicidal thoughts, and enhances sensory-motor coordination and adjustment. The aim of the current study is to determine how 2-week yoga and yogic practice sessions affect the mental health and well-being of 31 male and female volunteers, aged between 20 to 25 years, studying in the central university of Haryana.

Method

Priori Analysis

A priori power analysis for a one-sample t-test was conducted using G*Power 3.1.9.4 to determine the required sample size. The analysis was based on a medium effect size (d = .5), an alpha level of 0.05, and a desired power of 0.80. The results indicated that a total sample size of 27 participants was required to detect a significant difference from a constant with a power of 0.811. The critical t-value for this analysis was 1.70 with 26 degrees of freedom.

Participants and Research Design

The convenient sampling method was used to collect data. A total of 40 (20 Male and 20 Female) participants were selected from various departments of the Central University of Haryana, Mahendergarh, who consented to participate in the study. Some participants dropped out of the study due to personal reasons and some participants dropped out of the study due to time conflicts. A total of 9 participants dropped out. The remaining 31 participants (17 Male and 14 Female) filled pre- and post-test

questionnaires. The age range of the participants was 20 to 25 years, with a mean age of 22.32 and SD 1.37 years. Within-group research design was applied for this study.

Measures

Multiaxial Anxiety Questionnaire

The questionnaire developed by Reynolds (1999) is a self-report measure of anxiety designed for individuals aged 18 to 89 years. It comprises 40 items rated on a four-point Likert-type scale and assesses anxiety across four subscales: Negative Affectivity, Social Phobia, Physiologic-Panic, and Worry-Fears. Six items (2, 15, 23, 31, 36, and 40) are reverse-scored. The Negative Affectivity subscale includes 9 items (2, 15, 21, 23, 24, 31, 35, 36, 38), Social Phobia 9 items (1, 3, 4, 5, 8, 22, 25, 29, 40), Physiologic-Panic 12 items (9, 11, 12, 13, 14, 16, 17, 19, 20, 28, 29, 33), and Worry-Fears 10 items (6, 7, 10, 18, 26, 27, 30, 32, 34, 37). Sample items include statements such as "I worried that people thought I was bad," "I felt good," and "I felt nervous when I was with people." The scale has high internal consistency, with a Cronbach's alpha of .96 for the full scale and subscale alphas ranging from .88 to .91. Test-retest reliability is also strong, with coefficients of .95 for the total scale and .90 to .93 for the subscales.[35]

Suicidal Ideation Scale

SIS was developed by Rud (1989). It contains a total 10 items (For example, I have been thinking of ways to kill myself; I have told someone I want to kill myself; etc.). It is a five-point Likert-type scale. The total score of the scale ranges from 10 to 50. 15 and above scores of the participants indicate higher suicidal ideation. The computed Cronbach's Alpha (.86) of this scale indicates high internal consistency. It contains good inter-item correlation ranging from .45 to .74.[36] The author of the Suicide Ideation Scale had not suggested any referral protocols for the participants who had high suicidal ideation risk because this scale is mostly used for screening purposes in clinical settings. It helps clinical psychologists/psychotherapists to identify the suicidal risk in the population.

Table 1: Demographic details of the participants

Gender	Male	17	N = 31
	Female	14	
Education	Under Graduate	18	N = 31
	Post Graduate	13	
Stream	Arts	11	N = 31
	Science	5	
	Commerce	6	
	Engineering	9	
Types of Family	Joint Family	15	N = 31
	Nuclear Family	16	
Age	20 years	2	N = 31
	21 years	9	
	22 years	5	
	23 years	9	
	24 years	4	
	25 years	2	

Note. N = Total Number of Participants

Bell's Adjustment Inventory (Student Form)

The scale was adapted for the Indian population by Ojha (2006). There are 140 items (for example, Do you ever feel that your parents are not satisfied with you; Does your mother have supremacy in your family; etc.), in all, covering the four categories of adjustment: emotional, social, health, and home. Every domain has the same number of items—35—in total. A Yes/No scale with a score of two was used to assess the responses. Every yes response of the participant was scored as one, while every no response was scored as zero. Low scores on the inventory imply better adjustment overall and in all forms of adjustment, while high scores on the inventory indicate poor adjustment. The test was found to be quite reliable based on the computation of reliability using the split-half and test-retest techniques. The results showed that the reliability of the test was 0.91, 0.90, 0.89, and 0.92 by the test-retest method, and 0.84, 0.81, 0.87, and 0.89 by the split-half method. The home, health, social, and emotional domains of the questionnaire had validity coefficients of 0.72, 0.79, 0.82, and 0.81, in that order.[37]

Procedure

Before starting the study, the researchers had taken verbal feedback about past/current physical and mental illness, consumption of any medications or yoga practices from the respondents. The researchers followed all the required ethical guidelines. The chance of a confounding variable effect was controlled by the researchers in the initial stage of the study by obtaining the details on prior psychiatric or physical illnesses, psychosocial stressors and previous exposure to yoga.

Firstly, a good rapport was established with all participants, followed by obtaining informed consent from each individual. The researcher then provided clear and systematic instructions for the study, after which the pre-test was administered. The completed responses were collected from the participants. In the evening at 04:00 PM, all participants come to the peaceful grounds of the university for the yoga practices. The researchers practiced planned yoga exercises with the participants for approximately 35 to 40 minutes every day for two weeks. Consequently, the intervention involving yogic practices was introduced. After two weeks of practicing yoga, the post-test was administered, and the data were collected once again. After collecting data, those participants who were at risk of suicidal ideation the clinical management strategies and suicidal ideation risk mitigation plans were told by the researchers. The details of the planned yogic practices and interventions are presented in Table 1. The present study was conducted in 2024 in the month of May to September. In this study, researchers have not used any invasive techniques, and all aspects of the research were completed without harming to participants. Additionally, all ethical guidelines issued by the American Psychological Association (APA) have been followed diligently throughout the entire process, from data collection to writing the manuscript.

Statistical Analysis

The paired t-test was conducted to analyse the data, and the detailed findings of the study are comprehensively presented in Table 2.

Result

A paired sample *t*-test was conducted to determine whether yogic practices led to improvements or declines in adjustment, anxiety, and suicidal ideation among participants (*N = 31*) from pre-test to post-test. The results revealed that yogic practices significantly improved the *health adjustment* of participants from pre-test (*M = 10.19, SD = 4.76*) to post-test (*M = 8.84, SD = 5.11*), *t(30) = 2.46, p < .001.* Additionally, yogic practices significantly reduced *suicidal ideation* from pre-test (*M = 14.23, SD = 5.89*) to post-test (*M = 11.39, SD = 3.25*), *t(30) = 3.68, p < .001.* However, the effect of yogic practices was not found to be significant for anxiety and its dimensions (psychological-panic, social phobia, worry, fear, and negative affectivity), nor for total adjustment and its dimensions (home, social, and emotional adjustment), except for health adjustment.

Discussion

The aim of the study was to examine the impact of yogic practices on anxiety, adjustment, and suicidal ideation among participants. The findings revealed that yogic practices significantly reduced suicidal ideation and improved the health adjustment of participants. These results are supported by previous research.[38,3] Prior research has also highlighted a decrease in suicidality among patients with Major Depressive Disorder after the intervention of Iyengar yoga, which aligns with the current study's findings.[39] Studies suggest a link between suicidal ideation and factors such as academic stress and depression[40], as well as the role of life stressors in the suicidal ideation of adolescents.[41-44]

Consistent with past research by Ashish and Sharma (2023), the findings of this study indicate that yogic practices have a negative impact on suicidal thoughts.[34] A significant improvement in health adjustment following yoga interventions was also observed, which is consistent with findings from Kumar (2016), and Ghosh, Roy, and Malay (2021).[2,3] This may be due to the positive effects of yoga in reducing stress, inducing relaxation, and promoting positive attitudes toward stress[20,45,46] enhancing self-awareness[47], improving coping mechanisms[48-50], fostering self-compassion[51] increasing positive affect[52], inducing calmness[53], and promoting mindfulness.[54,55] Many researchers suggest that mindfulness is a crucial link between yoga practice and stress reduction.[56,57]

However, the present study also indicates no significant impact of yogic practices on home, social, and emotional adjustment, which is consistent with a prior study by Roy, Mukhopadhyay, and Ghosh (2020), suggesting no impact of yogic practices on the adjustment of college students.[14] Additionally, the study found no significant effect of yogic intervention on anxiety, which contrasts with existing literature that reports positive outcomes of yogic interventions on anxiety and stress levels among students (Figure 3). Possible explanations for these discrepancies could include the initial sample size, which was reduced to 31 due to dropouts, potentially being insufficient to detect significant effects given the diverse backgrounds of the students, including variations in disciplines and family backgrounds. The dropout rate (9 out of 40) might have impacted the study's power and introduced bias if the participants who dropped out differed systematically from

Table 2: Details of Yogic practices, time durations provided to participants

Sr. No.	Name of the Yogic Practices or Interventions	Time Duration
1	Suryanamaskara	15 minutes every day for two weeks
2	Standing Asanas	one different asana per day in two weeks, about two minutes with a complementary pose.
3	Sitting or Meditative Asanas	Four to five minutes for two weeks
4	Pranayama	one pranayama daily for five to seven minutes per day for weeks
5	Mudra	Four to five minutes per day for two weeks
6	Bandha	Four to five minutes per day for two weeks

Table 3: Mean, SD, and t-score of the study variables

Variables	Mean	Sd	t scores
Adjustment			
Home Adjustment Pre	13.29	5.68	1.74
Home Adjustment Post	12.13	5.61	
Health Adjustment Pre	10.19	4.76	2.46*
Health Adjustment Post	8.84	5.11	
Social Adjustment Pre	17.19	3.77	-.48
Social Adjustment Post	17.65	4.69	
Emotional Adjustment Pre	14.45	8.32	1.16
Emotional Adjustment Post	13.19	9.61	
Total Adjustment Pre	54.81	16.89	1.02
Total Adjustment Post	51.84	20.50	
Anxiety			
Psychological-Panic Pre	20.48	5.20	-.79
Psychological-Panic Post	21.26	5.72	
Social Phobia Pre	17.74	5.26	-.15
Social Phobia Post	17.84	5.31	
Worry, Fear Pre	19.00	5.66	.52
Worry, Fear Post	18.55	5.10	
Negative Affectivity Pre	19.87	5.04	-1.18
Negative Affectivity Post	20.90	4.43	
Total Anxiety Pre	76.61	18.32	-.58
Total Anxiety Post	78.23	18.89	
Suicidal Ideation			
Suicidal Ideation Pre	14.23	5.89	3.68***
Suicidal Ideation Post	11.39	3.25	

Note. N=31, *p<.05, ***p<.01.

those who completed the study. Furthermore, the duration of the yoga intervention may have been too short to produce measurable changes. Additionally, uncontrolled external factors, such as academic pressures, family issues, or the social environment, might have influenced participants' anxiety and adjustment levels, overshadowing the effects of the yoga intervention.

The planned yoga practices do not have a significant impact on other forms of adjustment, i.e., home, social, and emotional, except for health. However, the planned yoga practices have reduced the overall maladjustment of the participants. The planned yoga practices had also not a significant impact on the various dimensions of anxiety, i.e., psychological-panic, social-phobia, worry-fear, and negative affectivity. However, the planned yoga practices had reduced the anxiety of the respondents. This study was based on a short-term yoga practice, which is why no significant results were found over the forms of adjustment and dimensions of anxiety. If in the future, long-term intervention may give a significant outcome over the remaining non-significant study variables' forms and dimensions.

Implications and Limitations

Based on the findings, it can be concluded that yogic practices may contribute to enhancing health adjustment and reducing suicidal ideation among participants. The results suggested that yogic practices hold potential as a supportive intervention in mental health care, particularly in addressing issues related to maladjustment and suicidality. Mental health practitioners may consider integrating yogic practices into therapeutic programs to complement existing treatment strategies.

However, the study is not without limitations. First, the intervention period was relatively short, only two weeks, which may have been insufficient to observe substantial changes in anxiety and adjustment levels. Longer and more sustained yogic interventions might yield more conclusive outcomes. Second, the study faced challenges related to sample size. Although it began with 40 participants, a dropout rate of approximately 22.5% reduced the final sample to 31, which could have compromised the statistical power and generalizability of the results. Additionally, the sample was homogenous, consisting exclusively of students from a single central university in Haryana. This limited geographical and demographic scope restricts the applicability of the findings to broader populations. The absence of a control group further weakens the ability to establish causal relationships, as improvements observed may not be solely attributable to the yogic intervention. Finally, the study relied solely on a quantitative research design, which may have overlooked nuanced psychological or

experiential aspects that could be captured through qualitative methods. Future research should aim for a more diverse and larger sample, include a control or comparison group, extend the duration of intervention, and consider employing a mixed-method approach to obtain a more comprehensive understanding of the effects of yogic practices on mental health outcomes.

References

1. Agarwal A, Kaul N, Gandhi N. A comparative study of adjustment between boys and girls at undergraduate level. International Journal of Indian Psychology. 2017;4(4):31-40. Available from: doi.org/10.25215/0404.124

2. Ang RP, Huan VS. Relationship between academic stress and suicidal ideation: Testing for depression as a mediator using multiple regression. Child Psychiatry and Human Development. 2006; 37:133-143. Available from: doi.org/10.1007/s10578-006-0023-8

3. Arora S, Bhattacharjee J. Modulation of immune responses in stress by yoga. International Journal of Yoga. 2008;1(2):45-55. Available from: doi.org/10.4103/0973-6131.43541

4. Arria AM, O'Grady KE, Caldeira KM, Vincent KB, Wilcox HC, Wish ED. Suicide ideation among college students: A multivariate analysis. Archives of Suicide Research. 2009;13(3):230-246. Available from: doi.org/10.1080/13811110903044351

5. Arun P, Chavan BS. Stress and suicidal ideas in adolescent students in Chandigarh. Indian Journal of Medical Sciences. 2009;63(7):281-287.

6. Ashish, Sharma NK. Impact of Hatha Yoga on Suicidal Ideation among Transgender People. International Journal of Yoga and Allied Sciences. 2023;12(1):82-87.

7. Baig SA. Exploring Indian Knowledge System: Veda and Life Science. SGSH Publications. 2024.

8. Barnes PM, Powell-Griner E, McFann K, Nahin RL. Complementary and alternative medicine use among adults: United States, 2002. Advance Data. 2004;1-19.

9. Bell HM. Bell Adjustment Inventory: Manual. Consulting Psychologists Press. 1963.

10. Borji M, Otaghi M, Salimi E, Sanei P. Investigating the effect of performing the quiet time protocol on the sleep quality of cardiac patients. Biomedical Research. 2017;28(16):7076-7080.

11. Brown KW, Ryan RM. The benefits of being present: mindfulness and its role in psychological well-being. Journal of Personality and Social Psychology. 2003;84(4):822-848. Available from: doi.org/10.1037/0022-3514.84.4.822

12. Brown RP, Gerbarg PL. Sudarshan Kriya Yogic breathing in the treatment of stress, anxiety, and depression. Part II—clinical applications and guidelines. Journal of Alternative & Complementary Medicine. 2005;11(4):711-717. Available from: doi.org/10.1089/acm.2005.11.711

13. Bruton A. Breathing exercises for adults with asthma. Drug and Therapeutics Bulletin. 2015;53(11):126-129. Available from: doi.org/10.1136/dtb.2015.11.0361

14. Buitron V, Hill RM, Pettit JW, Green KL, Hatkevich C, Sharp C. Interpersonal stress and suicidal ideation in adolescence: An indirect association through perceived burdensomeness toward others. Journal of Affective Disorders. 2016;190:143-149. Available from: doi.org/10.1016/j. jad.2015.09.077

15. Castellote-Caballero Y, Carcelén-Fraile MC, Aibar-Almazán A, Rivas-Campo Y, González-Martín AM. Yoga as a therapeutic approach to mental health in university students: a randomized controlled trial. Frontiers in Public Health 2024;12:1406937. Available from: doi.org/10.3389/fpubh.2024.1406937

16. Chakradhari KR, Verma S. The effect of Yogic Practices on Adjustment level of visually Challenged School going student. (Unpublished Manuscript)

17. Chauhan S, Najaf SS, Lukács G, Király AK, Karsai I, Prémusz V. Impact of 10 weeks of yoga intervention on mental health and overall well-being among medical students: GSY study. Sports. 2025;13(4):114. Available from: doi.org/10.3390/sports13040114

18. Chiesa A, Serretti A. Mindfulness-based stress reduction for stress management in healthy people: a review and meta-analysis. The Journal of Alternative and Complementary Medicine. 2009;15(5):593-600. Available from: doi.org/10.1089/acm.2008.0495

19. Chu P, Gotink RA, Yeh GY, Goldie SJ, Hunink MM. The effectiveness of yoga in modifying risk factors for cardiovascular disease and metabolic syndrome: A systematic review and meta-analysis of randomized controlled trials. European Journal of Preventive Cardiology. 2016;23(3):291-307. Available from: https://www.ncbi.nlm.nih.gov/books/NBK292110/

20. Cohen L, Warneke C, Fouladi RT, Rodriguez MA, Chaoul-Reich A. Psychological adjustment and sleep quality in a randomized trial of the effects of a Tibetan yoga intervention in patients with lymphoma. Cancer. 2004;100(10):2253-2260. Available from: doi.org/10.1002/cncr.20236

21. CU Denver News. The Effects of Yoga on Student Mental Health. 2020. Retrieved November 24, 2023, From https://news.ucdenver.edu/the-effects-of-yogaon-student-mental-health/

22. Dighore M, Gadkari JV. Reducing Anxiety: Effect of Yogic Exercises and Meditation in the Improvement of Anxiety Score, Visual Reaction Time and Finger Dexterity Score. International Journal of Recent Trends in Science and Technology. 2013;7(1):52-55.

23. Duan-Porter W, Coeytaux RR, McDuffie JR, Goode AP, Sharma P, Mennella H, Nagi A, Williams JW. Evidence map of yoga for depression, anxiety, and posttraumatic stress disorder. Journal of Physical Activity and Health. 2016;13(3):281-288. Available from: doi.org/10.1123/

jpah.2015-0027

24. Dunn KD. A review of the literature examining the physiological processes underlying the therapeutic benefits of Hatha yoga. Advances in Mind-Body Medicine. 2008;23(3):10-18.

25. Ernst E, Pittler MH, Stevinson C, White A. The desktop guide to complementary and alternative medicine: an evidence-based approach. 2001.

26. Gahlawat S. Effect of yogic practices on prevalence of suicidal ideation among depressive students. International Journal of Applied Research. 2017;3(6):1160-1162.

27. Gard T, Brach N, Hölzel BK, Noggle JJ, Conboy LA, Lazar SW. Effects of a yoga-based intervention for young adults on quality of life and perceived stress: the potential mediating roles of mindfulness and self-compassion. The Journal of Positive Psychology. 2012;7(3):165-175. Available from: doi.org/10.1080/17439760.2012.667144

28. Garfinkel MS, Schumacher Jr HR, Husain AB, Levy M, Reshetar RA. Evaluation of a yoga based regimen for treatment of osteoarthritis of the hands. The Journal of Rheumatology. 1994;21(12):2341-2343.

29. Gilbert D, Waltz J. Mindfulness and health behaviors. Mindfulness. 2010;1(4):227-234. Available from: doi.org/10.1007/s12671-010-0032-3

30. Gliatto MF, Rai AK. Evaluation and treatment of patients with suicidal ideation. American Family Physician. 1999;59(6):1500-1506.

31. Grover KE, Green KL, Pettit JW, Monteith LL, Garza MJ, Venta A. Problem solving moderates the effects of life event stress and chronic stress on suicidal behaviors in adolescence. Journal of Clinical Psychology. 2009;65(12):1281-1290. Available from: doi.org/10.1002/jclp.20632

32. Heilbronn FS. The use of hatha yoga as a strategy for coping with stress in management development. Management Education and Development. 1992;23(2):131-139. Available from: doi.org/10.1177/135050769202300210

33. Hidaka BH. Depression as a disease of modernity: explanations for increasing prevalence. Journal of Affective Disorders. 2012;140(3):205-14. Available from: doi.org/10.1016/j.jad.2011.12.036

34. James-Palmer A, Anderson EZ, Zucker L, Kofman Y, Daneault, J-F. Yoga as an intervention for the reduction of symptoms of anxiety and depression in children and adolescents: A systematic review. Frontiers in Pediatrics. 2020;8:78. Available from: doi.org/10.3389/fped.2020.00078

35. Jayabalan R. A Study on Suicidal Tendency among PG Students in DRBCCC Hindu College, Pattabiram, Chennai-72. REDEFINING PROFESSIONAL SOCIAL WORK EDUCATION AND PRACTICE IN THE PRESENT SCENARIO: A Multi-disciplinary Perspective. 2019;9(2).

36. Kiecolt-Glaser JK, Christian L, Preston H, Houts CR, Malarkey WB, Emery CF, Glaser R. Stress, inflammation, and yoga practice. Biopsychosocial Science and Medicine. 2010;72(2):113-121. Available from: doi.org/10.1097/PSY.0b013e3181cb9377

37. Kinser PA, Bourguignon C, Whaley D, Hauenstein E, Taylor AG. Feasibility, acceptability, and effects of gentle hatha yoga for women with major depression: findings from a randomized controlled mixed-methods study. Archives of Psychiatric Nursing. 2013;27(3):137-147. Available from: doi.org/10.1016/j.apnu.2013.01.003

38. Kumar K, Tyagi AK. Approach of Yoga based lifestyle towards Social adjustment among Students. International Journal of Yoga and Allied Sciences. 2016;5(1):18-23.

39. Lazarus RS, Folkman S. Stress, appraisal and coping. Springer. 1984.

40. Makwana MD, Kaji SM. Adjustment of secondary school students in relation to their gender. The International Journal of Indian Psychology. 2014;2(1):5-12. Available from: doi.org/10.25215/0201.041

41. Malathi A, Damodaran A. Stress due to exams in medical students-role of yoga. Indian Journal of Physiology and Pharmacology. 1999;43(2):218-224.

42. Nagendra K, Sanjay D, Gouli C, Kalappanavar NK, Kumar CV. Prevalence and association of depression and suicidal tendency among adolescent students. 2012.

43. Nyer M, Gerbarg PL, Silveri MM, Johnston J, Scott TM, Nauphal M, Owen L, Nielsen GH, Mischoulon D, Brown RP, Fava M. A randomized controlled dosing study of Iyengar yoga and coherent breathing for the treatment of major depressive disorder: Impact on suicidal ideation and safety findings. Complementary Therapies in Medicine. 2018;37:136-142. Avaialble from: doi.org/10.1016/j.ctim.2018.02.006.

44. Oken BS, Kishiyama S, Zajdel D, Bourdette D, Carlsen J, Haas M, Hugos C, Kraemer DF, Lawrence J, Mass M. Randomized controlled trial of yoga and exercise in multiple sclerosis. Neurology. 2004;62(11):2058-2064. Available from: doi.org/10.1212/01.wnl.0000129534.88602.5c

45. Overholser J. Predisposing factors in suicide attempts: life stressors. In Evaluating and treating adolescent suicide attempters. Academic Press. 2003;41-52.

46. Pettit JW, Green KL, Grover KE, Schatte DJ, Morgan ST. Domains of chronic stress and suicidal behaviors among inpatient adolescents. Journal of Clinical Child & Adolescent Psychology: the official journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53. 2011;40(3):494-9. Available from: doi.org/10.1080/15374416.2011.563466

47. Reynolds WM. Multidimensional anxiety questionnaire. Odessa, FL: Psychological Assessment Resources. 1999.

48. Riley D. Hatha yoga and the treatment of illness. Alternative Therapies in Health and Medicine. 2004;10(2):20-21.

49. Rizzolo D, Zipp GP, Stiskal D, Simpkins S. Stress management strategies for students: The immediate effects of yoga, humor, and reading on stress. Journal of College Teaching & Learning. 2009;6(8):79-88. Available from: doi.org/10.19030/tlc.v6i8.1117

50. Roy R, Mukhopadhyay MK, Ghosh P. A comparative study of effect of yoga practices on adjustment of res-

idential college students. 2021.

51. Rudd MD. The prevalence of suicidal ideation among college students. Suicide and Life-Threatening Behavior. 1989;19(2):173-183. Available from: doi.org/10.1111/j.1943-278x.1989.tb01031.x

52. Shaffer HJ, LaSalvia TA, Stein JP. Comparing Hatha yoga with dynamic group psychotherapy for enhancing methadone maintenance treatment: a randomized clinical trial. Alternative Therapies in Health and Medicine. 1997;3(4):57-66.

53. Shohani M, Badfar G, Nasirkandy MP, Kaikhavani S, Rahmati S, Modmeli Y, Soleymani A, Azami M. The effect of yoga on stress, anxiety, and depression in women. International journal of preventive medicine. 2018;9:21. Available from: doi.org/10.4103/ijpvm.IJPVM_242_16

54. Singh S, Malhotra V, Singh KP, Sharma SB, Madhu SV, Tandon OP. A preliminary report on the role of yoga asanas on oxidative stress in non-insulin dependent diabetes mellitus. Indian Journal of Clinical Biochemistry. 2001; 16(2):216-220. Available from: doi.org/10.1007/BF02864866

55. Taneja I, Deepak KK, Poojary G, Acharya IN, Pandey RM, Sharma MP. Yogic versus conventional treatment in diarrhea-predominant irritable bowel syndrome: a randomized control study. Applied Psychophysiology and Biofeedback. 2004;29(1):19-33. Available from: doi.org/10.1023/b:apbi.0000017861.60439.95

56. Taylor MJ. Yoga therapeutics: an ancient, dynamic systems theory. Techniques in Orthopaedics. 2003;18(1):115-125. Available from: doi.org/10.1097/00013611-200303000-00017

57. Ray US,Mukhopadhyaya S, Purkayastha SS, Asnani V, Tomer OS, Prashad R, Thakur L, Selvamurthy W. Effect of yogic exercises on physical and mental health of young fellowship course trainees. Indian Journal of Physiology and Pharmacology. 2001;45(1):37-53.

Behavior and Academic Performance in Adolescents with ADHD in the Home Environment

Metali Bhatti[1], Priti Arun[2], Shivangi Mehta[3*], Smriti Mahajan[4]

Department of Psychiatry, Government Medical College and Hospital, Chandigarh, India.

How to Cite:
Bhatti M, Arun P, Mehta S, Mahajan S. Behavior and Academic Performance in Adolescents with ADHD in the Home Environment. Indian Journal of Clinical Psychiatry.2025;5(2):40-49.
doi: 10.54169/ijocp.v5i02.06

Abstract

Background: During the COVID pandemic, children and adolescents with ADHD had to struggle with a lack of structure and routine, resulting in an increase in irritability, oppositionality and challenging behaviours.

Aim: To assess the behaviour, academic performance, and problematic internet use in adolescents with ADHD in the home environment and stress coping strategies in their parents.

Methodology: A total of 96 adolescents with ADHD (11–17 years) were recruited after exclusion and inclusion criteria and were assessed either physically in the OPD or telephonically along with their parents for the purpose of assessing behaviour and academic performance, problematic internet use in the adolescents with ADHD in the home environment during Covid-19 pandemic lockdown and difficulties faced by their parents and stress coping strategies used by them using various assessment scales.
Results: Twenty-three (24.0%) were diagnosed with Hyperactive/impulsive symptoms, 18 (18.8%) with inattentive symptoms and 55 (57.3%) with combined type ADHD. The SAICA showed that the problems were more prevalent in males under the 14-17 age group. The problematic internet use and excessive internet use in the home during lockdown were found in two-thirds of the adolescents with ADHD (67.7%). The maximum number of parents had active coping, which depicts their adaptive behaviour (22.14 ± 3.19), compared to the parents who had maladaptive behaviour (12.24 ± 1.65).
Conclusion: From the current study, it was concluded that during the pandemic, the adolescents with ADHD faced many behavioural issues and problems in academic performance, which differed with socio-demographic data.

Introduction

Attention-deficit hyperactivity disorder (ADHD) is a common childhood neurodevelopmental disorder with a prevalence of 5% according to the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5).[1] Clinically diagnosed childhood ADHD persists into adolescence in 50-80 % of cases and into adulthood in 30 to 50% of cases.[2] About 3 to 7% of the childhood population is affected by ADHD, with boys being overrepresented, on average, approximately in the proportion of 3:1.[3]

According to the DSM-5, ADHD is characterised by a persistent pattern of inattention and/or hyperactivity/impulsivity that interferes with functioning or

development. The symptoms should have persisted for at least 6 months to a degree that is inconsistent with the developmental level and negatively impacts social/academic/occupational activities. Three types of presentations of ADHD are combined, predominantly inattentive and predominantly hyperactive/impulsive.[1] ADHD is associated with substantially lower school performance, independent of socioeconomic background factors. According to the research, children and adolescents with ADHD generally have poor academic performance. There is an increased risk of lower scores on reading and arithmetic achievement tests, lower grade point average (GPA), grade repetition and placement in special education classes as compared to the controls. As compared to the test controls, the children and adolescents with ADHD fail more grades and their performance is worse on standardised academic achievement tests. The teacher assessment differs by ADHD status as evidenced by the lower test scores and the grade points in ADHD as compared to non-ADHD.[4] During the pandemic, all schools were closed and classes were suspended; however, online classes resumed in schools gradually. Hence, it became extremely difficult for the ADHD adolescents to spend time at home. Making these children sit beyond a certain timeframe can cause them significant distress.[5] As schools and the day care centres were closed, access to resource material, peer group interactions and chances of learning and developing important social and behavioural skills in due time were lacking, which could lead to relapse of their symptoms. Although before the pandemic, these adolescents did face difficulties while attending school, but in due course, they had learnt to develop a schedule to adhere to for most of the time of the day.[4] Due to the lack of professional expertise during the pandemic, it became difficult for parents to handle the children and adolescents on their own, as they were previously mostly relying on school and therapists to help them out. Sometimes it became difficult for the caregivers to engage these adolescents in meaningful activities, as due to being confined in one place, the chances of their hyperactivity increased along with the heightened impulses. These adolescents can become a constant source of disturbance to their family members, and it can become difficult for the families to manage them, adding significant distress to the parents. Usually, parents do not have time to supervise the ongoing online classes and to carry out behavioural intervention due to their work pressure and busy schedules.[5]

Substantial distress is reported by the parents of these children and adolescents with ADHD, reasons for the distress being difficulty of living with family and the societal judgements related to their child's diagnosis, behaviours and the use of medications. Hence, they feel "isolated" and „ostracised" as the society does not understand ADHD, its consequences and the value of the medication. Therefore, the parents attribute their high levels of stress to their children's behaviour and social difficulties, their unmet needs for support, and their experiences of social stigma. Adolescents with ADHD are more prone to becoming dependent on the internet, and in-home confinement, it is more likely that they tend to overuse it due to the lack of outdoor activities. Internet overuse leads to additional behavioural problems and becomes a matter of concern and adding to the stress of their parents.

The coping strategies used by the parents of the adolescents with ADHD showed that they used more avoidant-focused coping strategies, contrary to the parents of the typical children. The parents of the ADHD adolescents sought significantly more support and used indirect means than the mothers of typically developing children.[5]

There is a need to assess the behavioural problems that arise in adolescents with ADHD due to disturbance in the structured routine at home, which was earlier maintained in the school environment, changes in the academic performance due to the change in the study pattern (with the introduction of the online classes) and how parents coped with stress while managing their online academics and behaviour.

This study was planned with the objectives of assessing behaviour and academic performance, and problematic internet use in adolescents with ADHD in the home environment and assessing coping strategies in parents of these adolescents with ADHD.

Methods

Study Design

This was an observational, cross-sectional study. Institutional ethics committee clearance was obtained before initiating recruitment. Participants were assessed either in person or by telephone/via video conference with due consent and with prior appointment.

Participants

Adolescents enrolled in the child guidance clinic run by the Psychiatry department of a tertiary-care teaching hospital in North India were assessed for eligibility. Those with a DSM-5 diagnosis of ADHD who were enrolled in school, aged between 11 and 17 years, with either parent giving written informed consent, were included. Verbal (till 12 years) or written assent (12–17 years) was also taken for the study. Those with comorbid autism spectrum disorder, specific learning disability, intellectual disability or other psychiatric disorders, and those with medical illnesses preventing the participant from pursuing education for a period of at least 1 month out of the previous 6 months were excluded from the study. The parents who refused to consent were excluded along with their adolescent.

Assessment Parameters

A basic socio-demographic proforma and a clinical profile proforma were filled out for all participants. The tools used were the Vanderbilt ADHD diagnostic parent rating scale (VADPRS),[6] Strengths and Difficulties Questionnaire (SDQ)[7]- parent and adolescent versions, Social Adjustment Inventory for Children and Adolescents[8] (SAICA), Problematic Internet Use Scale in Adolescents[9] (PIUS), and Stress Coping Behaviour Scale, Indian adaptation by Janghel and Srivastava(2017) (SCBS).[10]

Sample Size

The sample size was calculated using the prevalence of behavioural difficulties in adolescents, which is

Table 1: Relationship of the type of ADHD with socio-demographic variables

		VADPRS				Chi- Square	p-value
		Hyperactive/ Impulsive n (%)	Inattentive n (%)	Combined n (%)	Total n (%)		
Age (Years)	11-13	16 (25.4%)	11 (17.5%)	36 (57.1%)	63 (65.6%)	0.322	0.851
	14-17	7 (21.2%)	7 (21.2%)	19 (57.6%)	33 (34.4%)		
Gender	Male	22 (26.5%)	16 (19.3%)	45 (54.2%)	83 (86.5%)	2.763	0.251
	Female	1 (7.7%)	2 (15.4%)	10(76.9%)	13 (13.5%)		
Education	Primary School	7 (21.2%)	5 (15.2%)	21(63.6%)	33 (34.4%)	1.308	0.860
	Middle School	7 (21.9%)	7 (21.9%)	18(56.3%)	32 (33.3%)		
	High School	9 (29.0%)	6 (19.4%)	16(51.6%)	31 (32.3%)		
Type of family	Nuclear	12 (19.0%)	16 (25.4%)	35(55.6%)	63 (65.6%)	6.345	0.175
	Joint	10 (33.3%)	2 (6.7%)	18(60.0%)	30 (31.3%)		
	Extended	1 (33.3%)	0 (0.0%)	2 (66.7%)	3 (3.1%)		
Family income	0-3500	0 (0.0%)	1 (20.0%)	4 (80.0%)	5 (5.2%)	1.770	0.778
	3501-7000	3 (27.3%)	2 (18.2%)	6 (54.5%)	11 (11.5%)		
	7001 & above	20 (25.0%)	15 (18.8%)	45(56.3%)	80 (80.3%)		
Locality	Rural	6 (15.4%)	10 (25.6%)	23(59.0%)	39 (40.6%)	3.711	0.156
	Urban	17 (29.8%)	8 (14.0%)	32(56.1%)	57 (59.4%)		
	Total	23 (24.0%)	18 (18.8%)	55(57.3%)	96 (100.0%)		

10.1%.[11] The sample size was calculated as 96 subjects, with 5% confidence level and a margin of error of 1-unit standard deviation (SD).

Statistical Methods

Data was coded and analysed using SPSS version 20.0. Mean and SD for ordinal variables and frequency tabulation for categorical variables were carried out. An unpaired t-test was applied for ordinal variables, and a chi-square test was applied for categorical variables. In case of skewed data, non-parametric tests like the Kruskal-Wallis test were applied. Repeated measures ANOVA was applied to analyse repeated intra-group measurements. Baseline comparison of quantitative parameters was done using the Mann-Whitney U test.

Results

A total of 250 case files belonging to patients registered with the clinic since 2015 were screened, out of which 180 fulfilled the inclusion and exclusion criteria. 150 of these 180 could be contacted telephonically and were ready to participate. Out of the 150 contacted, 25 were completely assessed in OPD, 71 were assessed telephonically, and 54 could not complete the study and were excluded. Eventually, 96 patients were included in the completed study.

Sample Characteristics

A total of 96 participants completed the study, out of which 83 (86.5%) were males and 13 (13.5%) were females. 63 (65.6%) participants were aged between

Table 2: Relationship between the Domains of SAICA and age group of study sample

	Age (in years)				t-value	p-value
	11-13		14-17			
	n	Mean ± SD	n	Mean ± SD		
Academic performance	63	2.11 ± 0.41	33	2.36 ± 0.55	2.557	.012*
Attitude towards schoolwork	63	2.57 ± 0.53	33	2.70 ± 0.73	.966	.337
Attitude towards teachers	63	2.14 ± 0.47	33	2.39 ± 0.66	2.157	.034*
Teachers attitude towards child	63	2.32 ± 0.59	33	2.21 ± 0.60	.825	.411
Relationships with classmates	63	2.30 ± 0.59	33	2.39 ± 0.66	.703	.484
Problems at school	63	2.16 ± 0.45	33	2.18 ± 0.216	.216	.830
Spare time activities	63	22.33 ± 0.54	33	2.33 ± 0.65	0.000	1.000
Spare time spent mostly with others	63	2.35 ± 0.51	33	2.21 ± 0.48	1.267	.208
Spare time problems	63	2.24 ± 0.50	33	2.39 ± 0.61	1.346	.182
Peer relationships	63	2.25 ± 0.54	33	2.21 ± 0.60	.348	.729
Peer problems	63	2.17 ± 0.52	33	2.09 ± 0.68	.670	.505
Boy girl relationships	63	.92 ± 1.34	33	2.00 ± 1.12	3.968	.0001*
Boy girl problems	63	.83 ± 0.19	33	2.06 ± 0.93	5.198	.0001*
Sibling relationships	63	2.21 ± 0.63	33	2.21 ± 0.60	.044	.965
Siblings' problems	63	2.13 ± 0.71	33	2.18 ± 0.64	.373	.710
Relationship with mother	63	2.22 ± 0.68	33	2.21 ± 0.70	.068	.946
Relationship with father	63	2.14 ± 0.56	33	2.15 ± 0.89	.069	.945
Problems with parent	63	2.13 ± 33	33	2.09 ± 0.90	.289	.773

*- significant

Table 3: Relationship between gender and the subdomains of SAICA

	Gender				t-value	p-value
	Male		Female			
	n	Mean ± SD	n	Mean ± SD		
Academic performance	83	2.19 ± 0.48	13	2.23 ± 0.44	.268	.789
Attitude towards schoolwork	83	2.65 ± 0.57	13	2.38 ± 0.77	1.484	.141
Attitude towards teachers	83	2.24 ± 0.55	13	2.15 ± 0.55	.527	.599
Teachers attitude towards child	83	2.29 ± 0.60	13	2.23 ± 0.60	.329	.743
Relationships with classmates	83	2.33 ± 0.59	13	2.38 ± 0.77	.325	.746
Problems at school	83	2.19 ± 0.48	13	2.00 ± 0.58	1.309	.194
Spare time activities	83	2.40 ± 0.56	13	1.92 ± 0.49	2.874	.005*
Spare time spent mostly with others	83	2.35 ± 0.50	13	2.00 ± 0.41	2.375	.020*
Spare time problems	83	2.30 ± 0.53	13	2.23 ± 0.60	.434	.665
Peer relationships	83	2.27 ± 0.54	13	2.08 ± 0.64	1.134	.260
Peer problems	83	2.14 ± 0.57	13	2.15 ± 0.69	.053	.958
Boy girl relationships	83	1.22 ± 1.37	13	1.77 ± 1.24	1.367	.175
Boy girl problems	83	1.17 ± 1.25	13	1.77 ± 1.17	1.627	.107
Sibling relationships	83	2.18 ± 0.63	13	2.38 ± 0.51	1.114	.268
Siblings problems	83	2.19 ± 0.67	13	1.85 ± 0.69	1.726	.088
Relationship with mother	83	2.28 ± 0.69	13	1.85 ± 0.55	2.153	.034*
Relationship with father	83	2.17 ± 0.60	13	2.00 ± 0.41	.975	.332
Problems with parent	83	2.13 ± 0.60	13	2.00 ± 0.41	.767	.445

*- significant

11 to 13 years and 33 (34.4%) were between 14 to 17 years. 57 (59.4%) belonged to urban areas and 39 (40.6%) were from rural areas. 68 (70.8%) participants were fluent in Hindi, followed by Punjabi (n = 25, 26%) and English (n = 3, 3.1%). Most of the participants lived in nuclear families (n = 63, 65.6%), followed by joint families (n = 30, 31.3%) and extended families (n = 3, 3.1%).

On VADPRS, 23 (24%) participants were found to have a hyperactive/impulsive type, 18 (18.8%) had an inattentive type, and 55 (57.3%) had a combined type of ADHD. Hyperactive/impulsive symptoms were more prevalent in the age group 11 to 13 years (n=16, 25.4%), inattentive symptoms were more prevalent in the age group 14 to 17 years (n=7, 21.2%) and combined type of ADHD had similar prevalence in both the age groups i.e 57.1% in 11 to 13 years and 57.6% in 14 to 17 years. Further correlating with gender, the hyperactive/impulsive and inattentive types were mostly seen in males (n=22, 26.5% and n=16, 19.3% respectively), and combined symptoms were more prevalent in females (n=10, 76.9%) (as depicted in Table 1).

On assessing the behavior and academic performance using SAICA scale and comparing with socio-demographic data; it was found that older age group, i.e., 14 to 17 years had more academic problems (2.36 ± 0.55), a negative attitude towards teachers (2.39 ± 0.66), boy girl relationship (2.00 ± 1.12) and boy girl problems (2.06 ± 0.93) than the younger age group i.e 11-13 years. (*p <0.05) (As depicted in Table 2).

On correlating the SAICA score with gender, it was seen that males tend to participate more in

Table 4: Strength and Difficulty Questionnaires (Adolescent version)

		Frequency (n)	Percent (%)
SDQA (total)	Clinically sig. problems in this area are unlikely	4	4.2%
	Score is slightly raised, which may reflect clinically sig. problems	7	7.3%
	This score is high – there is a substantial risk of clinically. Significant problem in this area.	85	88.5%
Emotional symptoms	Clinically sig. problems in this area are unlikely	62	64.6%
	Score is slightly raised, which may reflect clinically sig. problems	22	22.9%
	This score is high – there is a substantial risk of a clinically significant problem in this area.	12	12.5%
Conduct problem	Clinically sig. problems in this area are unlikely	5	5.2%
	Score is slightly raised, which may reflect clinically sig. problems	11	11.5%
	This score is high – there is a substantial risk of a clinically significant problem in this area.	80	83.3%
Hyperactivity	Clinically sig. problems in this area are unlikely	62	64.6%
	Score is slightly raised, which may reflect clinically sig. problems	21	21.9%
	This score is high – there is a substantial risk of a clinically significant problem in this area.	13	13.5%
Peer problem	Clinically sig. problems in this area are unlikely	11	11.5%
	Score is slightly raised, which may reflect clinically sig. problems	15	15.6%
	This score is slightly raised, which may reflect clinically	70	72.9%
Pro Social Behaviour	Clinically sig. problems in this area are unlikely	35	36.5%
	The score is slightly raised, which may reflect clinically significant problems	28	29.2%
	This score is high – there is a substantial risk of clinically. significant problem in this area.	33	34.4%
	Total	96	100.0%

spare-time activities, and spare time was mostly spent with others. Also, the relationship with the mother was more disturbed in the case of males as compared to females (*p = 0.05). (As depicted in Table 3).

When the total SDQ- adolescent version was compared with socio-demographic variables, it was found that substantial risk of clinically significant problems was significantly associated with gender, education and locality and was found higher in the males (n=75, 90.4%), those studying in the primary school (n=33, 100%) and belonging to urban areas (n=52, 91.2%). (Table 4)

The total SDQ (parent version) depicts that the maximum number of parents of participants facing difficulties fell under the category of substantial risk of clinically significant problems (n=94, 97.7%), with significant problems in domains of emotions (68.8%), conduct problems (90.6%) and peer problems (85.4%). (as depicted in Table 5).

Problematic internet use is more prevalent in the older age group (75.8%), participants going to high school (n=22, 71.0%), belonging to extended family (n=3, 100.0%) with family income of 3501-7000 (n=10, 90.9%) belonging to the urban area (n=57, 100.0%) than in the corresponding groups, and comparable in females and males (69.2 and 67.5%, respectively) using problematic internet use scale (As depicted in Table 6).

The stress coping behaviour scale comprises adaptive and maladaptive behaviour domains. Under the adaptive behaviour domain, the participants with adaptive behaviour were more (22.34 ± 3.16) than the participants with maladaptive behaviour (12.07 ± 1.67). (As depicted in Table 7)

Table 5: Relationship between the total SDQ score of the adolescents and the socio-demographic variables.

		SDQA				Chi-Square	p-value
		Clinically sig. problems in this area are unlikely n (%)	Score is slightly raised which may reflect clinically sig. problems n (%)	This score is high – there is a substantial risk of clinically significant problem in this area n (%)	Total		
Age	11-13	1 (1.6%)	6 (9.5%)	56 (88.9%)	63 (65.6%)	4.181	.124
	14-17	3 (9.1%)	1 (3.0%)	29 (87.9%)	33 (34.4%)		
Gender	Male	1 (1.2%)	7 (8.4%)	75 (90.4%)	83 (86.5%)	14.230	.001*
	Female	3 (23.1%)	0 (0.0%)	10 (76.9%)	13 (13.5%)		
Education	Primary School	0 (0.0%)	0 (0.0%)	33 (100.0%)	33 (34.4%)	9.959	.041*
	Middle School	1 (3.1%)	5 (15.6%)	26 (81.3%)	32 (33.3%)		
	High School	3 (9.7%)	2 (6.5%)	26 (83.9%)	31 (32.3%)		
Type of family	Nuclear	3 (4.8%)	4 (6.3%)	56 (88.9%)	63 (65.6%)	0.883	0.927
	Joint	1 (3.3%)	3 (10.0%)	26 (86.7%)	30 (31.3%)		
	Extended	0 (0.0%)	0 (0.0%)	3 (100.0%)	3 (3.1%)		
Family income	0-3500	0 (0.0%)	0 (0.0%)	5 (100.0%)	5 (5.2%)	1.309	0.86
	3501-7000	0 (0.0%)	1 (9.1%)	10 (90.9%)	11 (11.5%)		
	7001 & above	4 (5.0%)	6 (7.5%)	70 (87.5%)	80 (80.3%)		
Locality	Rural	4 (10.3%)	2 (5.1%)	33 (84.6%)	39 (40.6%)	6.382	.041*
	Urban	0 (0.0%)	5 (8.8%)	52 (91.2%)	57 (59.4%)		
	Total	4 (4.2%)	7 (7.3%)	85 (88.5%)	96 (100.0%)		

Table 6: Relationship between internet use and the socio-demographic variables

		PIUS			Chi-Square	p-value
		No problematic internet n(%)	*Problematic internet n(%)*	*Total*		
Age	11-13	23 (36.5%)	40 (63.5%)	63 (65.6%)	1.490	.222
	14-17	8 (24.2%)	25 (75.8%)	33 (34.4%)		
Gender	Male	27 (32.5%)	56 (67.5%)	83 (86.5%)	.016	0.9
	Female	4 (30.8%)	9 (69.2%)	13 (13.5%)		
Education	Primary School	12 (36.4%)	21 (63.6%)	33 (34.4%)	.417	.812
	Middle School	10 (31.3%)	22 (68.8%)	32 (33.3%)		
	High School	9 (29.0%)	22 (71.0%)	31 (32.3%)		
Type of family	Nuclear	22 (34.9%)	41 (65.1%)	63 (65.6%)	1.702	0.427
	Joint	9 (30.0%)	21 (70.0%)	30 (31.3%)		
	Extended	0 (0.0%)	3 (100.0%)	3 (3.1%)		
Family income	0-3500	2 (40.0%)	3 (60.0%)	5 (5.2%)	3.112	0.211
	3501-7000	1 (9.1%)	10 (90.9%)	11 (11.5%)		
	7001 & above	28 (35.0%)	52 (65.0%)	80 (80.3%)		
Locality	Rural	13 (33.3%)	26 (66.7%)	39 (40.6%)	.033	.857
	Urban	18 (31.6%)	39 (68.4%)	57 (59.4%)		
	Total	31 (32.3%)	65 (67.7%)	96 (100.0%)		

Table 7: Stress coping behaviour scale

	N	Mean ± SD	Minimum	Maximum
Active coping	96	3.27 ± 0.62	2.00	4.00
Use of emotional support	96	2.81 ± .73	2.00	4.00
Use of instrumental support	96	1.51 ± .50	1.00	2.00
Positive refraining	96	3.07 ± .80	2.00	4.00
Planning	96	2.96 ± 0.71	2.00	4.00
Humour	96	2.81 ± 0.71	2.00	4.00
Acceptance	96	2.95 ± 0.70	2.00	4.00
Religion	96	2.96 ± 0.89	2.00	4.00
Adaptive Behaviour	96	22.34 ± 3.16	16.00	29.00
	N	*Mean ± SD*	*Minimum*	*Maximum*
Self-distraction	96	3.46 ± 0.58	2.00	4.00
Denial	96	1.63 ± 0.59	1.00	2.00
Substance use	96	1.15 ± 0.35	1.00	2.00
Behavioural disengagement	96	1.44 ± 0.50	1.00	2.00
Venting	96	2.93 ± 0.76	2.00	4.00
Self-blame	96	1.48 ± 0.50	1.00	2.00
Maladaptive behaviour	96	12.07 ± 0.50	9.00	15.00

Discussion

The VADPRS scale was used to assess the type of ADHD (Hyperactive/Impulsive, Inattentive or Combined). In this study, 57% of participants had a combined type of ADHD, 24% had predominantly hyperactive/impulsive symptoms and 19% predominantly inattentive type of ADHD. Earlier research describes that the inattentive type of ADHD is present in 20 to 30% of cases, the hyperactive-impulsive type has a prevalence of less than 15% of cases, and the most common type is the combined type, present in 50 to 75% of cases.[12] Hyperactive symptoms were more prevalent than inattentive symptoms, with a slight difference in the percentage between the two (5%). The reason that the current study showed a high prevalence in the hyperactivity/impulsive subtype of ADHD could be due to disturbance in the structured routine attributed to the home environment and online classes. There is no significant difference in the prevalence of ADHD type when compared with studies carried out in the past.[13] Some studies show that, on average, inattention declines, while others show stable or rising inattention.[13,14] Comparison of subdomains of Social adjustment inventory with socio-demographic parameters showed that academic performance, attitude towards teachers, boy-girl relationship and boy-girl problems subdomains were significantly more prevalent in the 14 to 17 years age group. The subdomains of spare time activities and spare time spent with others showed higher scores in males than in females. In past studies, the t-tests of difference in mean SAICA subscale and overall scores of children ages 6 through 11, and those 12 through 18 years, by mothers' and children's reports had significant differences ($p < 0.05$) on more subscales and overall scores.[15] When compared with socio-demographic variables, it was found that the total score on SDQ- SDQ-adolescent showed a significant association with gender, education and locality. Clinically significant problems were more likely to be found in males studying in primary school and belonging to an urban area. When comparing the subdomains with socio-demographic variables, no significant association was found between the subdomains and the socio-demographic variables, except that the prosocial behavior problems were found to have a significant association with the locality. SDQ-parent version showed that emotional symptoms were more prevalent in the age group 14 to 17 years. There were no significant differences between the community sample and clinical sample with respect to gender differences, missing information about the gender, mean age of the boys and girls, and missing information about age.[16] Though there were no significant differences between the ages, it showed that a higher number of participants with problematic internet use fall under the category of the older age group, i.e, 14 to 17 years (75.8%), than the younger age group, i.e., 11 to 13 years (65.5%). This finding is consistent with previous research, which showed that no significant differences were found by sex, but there was significantly higher problematic internet use in the older age group.[17] Our results showed that a greater number of parents had active coping, which depicts their adaptive behaviour (22.14 ± 3.19), than the parents who had maladaptive behaviour (12.24 ± 1.65). In previous studies, some parents of adolescents with ADHD reported worsening of general well-being in their children, and this manifested as oppositional/defiant attitudes and emotional outbursts. Parents also cited sleep problems and anxiety in this context.[18,19]

The objectives of this study were to assess the behavioural and academic performance in adolescents with ADHD in the home environment; to assess the problematic internet use in these adolescents, parental stress associated with it and the coping strategies used by them. The various strengths of this study include both online and physical data collection, which minimised dropout due to fear of getting infected; it included adolescents along with their parents to provide a multidimensional view of the problems faced by the adolescents through the comparison of the self-reported symptoms and those reported by the parents. The parents were also assessed for adaptive or maladaptive coping skills in response to the problems encountered during lockdown. Standardised scales were used for assessment. However, this study is not without its limitations. The study design was cross-sectional; hence, a temporal link between the exposure and the outcome cannot be determined. Recall bias could be there as some assessment scales required reporting of behaviour or academic performance of the adolescent during the previous 6 months.

Conclusion

The current study found that adolescents with ADHD faced many behavioural issues and problems in academic performance during the pandemic. These issues differed with socio-demographic parameters. ADHD was more prevalent in males of younger age belonging to lower socioeconomic status, and significant problems were found in these groups. Assessment of strengths and difficulties of adolescents and parents found that adolescents reported fewer problems faced by themselves than what their parents reported of them. Problematic internet use was also highly prevalent during lockdown and was more common in adolescent males. The parents of these adolescents faced significant stress due to disturbance in structured routine, and most parents showed adaptive coping behaviour in dealing with the stress associated with this.

Financial Support and Sponsorship

References

1. American Psychiatric Association. The diagnostic and statistical manual of mental disorders. 5 edition. Arlington, VA: American Psychiatric Association 2013;59-65.
2. Barkley RA. Behavioral inhibition, sustained attention and executive functions: constructing a unifying theory of ADHD. Psychol Bull 1997;12:65-94.
3. Wolraich ML, Hagan JF Jr, Allan C, Chan E, Davison D, Earls M, et al. Clinical Practice Guideline for the Diagnosis, Evaluation, and Treatment of Attention-Deficit/Hyperactivity Disorder in Children and Adolescents. Pediatrics. 2019 Oct;144(4):e20192528. doi: 10.1542/peds.2019-2528. Erratum in: Pediatrics. 2020 Mar;145(3):e20193997. doi: 10.1542/peds.2019-3997. PMID: 31570648; PMCID: PMC7067282
4. Jangmo A, Stålhandske A, Chang Z, Chen Q, Almqvist C, Feldman I et al. Attention-deficit/hyperactivity disorder, school performance, and effect of medication. J Am Acad Child Adolesc Psychiatry 2019;58:423-432.
5. Lai WW, Mahony OM, Mulligan A. The home measure of the environment is associated with the symptoms of ADHD and Oppositionality in a CAMHS Sample. Clin Child Psychol Psychiatry 2017;23:503-513.
6. Wolraich ML, Lambert W, Doffing MA, Bickman L, Simmons T, Worley K et al. Psychometric properties of the Vanderbilt ADHD diagnostic parent rating scale in a referred population. J Pediatr Psychol 2003;28:559-567.
7. Stone LL, Janssens JM, Otten R. The strength and difficulties Questionare: psychometric properties of the parent and teacher version in children. BMC Psychol 2015;3:1-10.
8. John K, Gammon GD, Prusoff BA, Warner V. The social adjustment inventory for children and adolescent (SAICA) : testing of a new semi structured interview. J Am Acad Adolesc Psychiatry 1987:26;898-911.
9. Boubeta RA, Salgado GP, Folgar MI, Mallou JV. PIUS-a: A problematic internet use scale in adolescents. Development and psychometric Validation. Adicciones 2015;27:47-63.
10. Janghel G, Srivastava P. Coping behaviour assessment Scale (Indian adaptation) : establishing psychometric properties. The International Journal of Indian Psychology 2017;4:151-156.
11. Aboujaoude E. Problematic Internet use: an overview. World Psychiatry 2010;9:85-90.
12. Jensen PS. Clinical considerations for the diagnosis and treatment of ADHD in the managed care setting. Am J Manag Care 2009;15:129-140.
13. O'Neill S, Rajendran K, Mahbubani SM, Halperin JM. Preschool predictors of ADHD symptoms and impairment during childhood and adolescence. Cur Psychiatry Rep 2017 Oct 30;19(12):95. doi: 10.1007/s11920-017-0853-z. PMID: 29082443; PMCID: PMC6349372.
14. Sasser TR, Beekman CR, Bierman KL. Preschool executive functions, single-parent status, and school quality predict diverging trajectories of classroom inattention in elementary school. Dev Psychopathol. 2015;27:681–963.
15. Cerrillo-Urbina AJ, García-Hermoso A, Martínez-Vizcaíno V, Pardo-Guijarro MJ, Ruiz-Hermosa A, Sánchez-López M et al. Prevalence of probable attention-deficit/hyperactivity disorder symptoms: result from a spanish sample of children. BMC Pediatr 2018;18:111-119.
16. Reilly C, Atkinson P, Memon A, Jones C, Dabydeen L, Helen Cross J et al. Autism, ADHD and parent-reported behavioural difficulties in young children with epilepsy. Seizure 2019 ;71:233-239.
17. Ioannidis K, Treder MS, Chamberlain SR, Kiraly F, Redden SA, Stein DJ, et al. Problematic internet use as an age-related multifaceted problem: Evidence from a two-site survey. Addict Behav. 2018 Jun;81:157-66. doi: 10.1016/j.addbeh.2018.02.017. Epub 2018 Feb 12. PMID: 29459201; PMCID: PMC5849299.
18. Bobo E, Lin L, Acquaviva E, Caci H, Franc N, Gamon L, et al. How do children and adolescents with attention deficit hyperactivity disorder (ADHD) experience lockdown during the COVID-19 outbreak? Encephale. 2020:46:85-92.
19. Giannakopoulos G. Adolescents with ADHD in the school environment: A comprehensive review of academic, social, and emotional challenges and interventions. J Clin Images Med Case Rep. 2025; 6(3): 3528

Cognitive Dysfunctions and Neurological Soft Signs in Drug-Naïve Schizophrenia: A Cross-Sectional Study

Shruti Singh[1*], Om Prakash Raichandani[2], Neha Ramsinghani[3]

[1]*Netaji Shubhash Chandra Bose Medical College, Jabalpur, Madhya Pradesh, India.*

[2]*Professor and Head of Department, Department of Psychiatry, NSCB Medical College and Hospital, Jabalpur, Madhya Pradesh, India.*

[3]*Assisstant Professor, Department of Psychiatry, NSCB Medical College and Hospital, Jabalpur, Madhya Pradesh, India.*

Abstract

Background: Cognitive impairments and neurological soft signs (NSS) are common in schizophrenia, often evident prior to treatment. This study investigates the relationship between cognitive deficits and NSS in drug-naïve schizophrenia patients.

Methods: A cross-sectional study was conducted with 60 drug-naïve schizophrenia patients. Cognitive performance was evaluated using the Montreal Cognitive Assessment (MoCA), with scores ≤26 indicating cognitive impairment. NSS were assessed via the Neurological Evaluation Scale (NES). Pearson Chi-square tests analysed associations between MoCA scores and specific NSS, with significance set at $p < 0.05$.

Results: Of the 60 patients, 39 (65%) showed cognitive impairment (MoCA ≤26). Significant correlations were found between cognitive deficits and several NSS, including Audio-Visual Integration ($p = 0.001$), graphesthesia ($p = 0.027$), fist-ring test ($p = 0.001$), and fist-edge-palm test ($p = 0.001$). Additional associations were noted with memory ($p = 0.001$), rhythm tapping test part A ($p = 0.001$), and rapid alternating movements ($p = 0.001$). No significant correlations were observed for cerebral dominance ($p = 0.435$), stereognosis ($p = 0.459$), or extinction ($p = 0.459$). Patients exhibited an average of 4.6 NSS, with 86.7% showing at least one NSS.

Conclusion: Cognitive impairments in drug-naïve schizophrenia patients are significantly associated with specific NSS, suggesting shared neurobiological underpinnings. These findings emphasize the need for early cognitive and neurological evaluations to guide treatment. Longitudinal studies are required to examine the progression of these deficits.

INTRODUCTION

Schizophrenia is a profound mental disorder marked by symptoms such as hallucinations, delusions, negative symptoms, and cognitive impairments. Cognitive deficits, a central characteristic of schizophrenia, often emerge at the illness's onset and persist despite treatment [1]. These impairments disrupt attention, memory, executive functioning, and processing speed, profoundly affecting patients' daily functioning and quality of life [2]. Alongside cognitive challenges, schizophrenia is linked to neurological soft signs (NSS), subtle neurological irregularities that indicate dysfunction in sensory integra-

tion, motor coordination, and task sequencing [3]. NSS are commonly observed in schizophrenia and are viewed as potential endophenotypes, signalling underlying neurodevelopmental disruptions.[4] These signs include challenges in motor coordination (e.g., Fist-Edge-Palm Test), sensory processing (e.g., Audio-Visual Integration), and graphesthesia. Notably, NSS often appears in drug-naïve patients, suggesting they are inherent to the disorder rather than a consequence of antipsychotic treatment [5]. The presence of NSS in first-episode, untreated patients supports the neurodevelopmental hypothesis, which attributes schizophrenia to early brain development anomalies [6].

Globally, schizophrenia affects approximately 24 million people, or 0.32% of the population (1 in 300), with a slightly higher prevalence among adults at 0.45% (1 in 222) [7]. The Global Burden of Disease (GBD) Study 2019 reports that from 1990 to 2019, the raw prevalence of schizophrenia increased by over 65%, from 14.2 million to 23.6 million, though age-standardized prevalence rates remained stable [8]. Schizophrenia ranks among the top 15 leading causes of disability worldwide, contributing significantly to disability-adjusted life years (DALYs) due to its chronic nature and associated impairments [8]. In India, the National Mental Health Survey (NMHS) conducted between 2015 and 2016 across 12 states reported a lifetime prevalence of schizophrenia spectrum disorders at 1.41% and a current prevalence of 0.42%, with a substantial treatment gap of 72% [9]. Another study analyzing data from 2008 to 2017 indicated a 19% increase in schizophrenia cases in India, from 2.91 million to 3.46 million, with higher prevalence among males and individuals aged 25–49 [10]. These figures highlight the significant burden of schizophrenia in India, where genetic, environmental, and cultural factors may influence its clinical presentation and outcomes [9]. The connection between cognitive impairments and NSS in schizophrenia has attracted increasing research interest. Studies suggest NSS may indicate cognitive deficits, likely due to shared neurobiological disruptions in frontocerebellar and subcortical pathways [11]. However, this relationship remains underexplored in drug-naïve patients, particularly in India, where genetic and cultural factors may shape clinical manifestations [12]. This study examines the association between cognitive impairments, assessed by the Montreal Cognitive Assessment (MoCA; score ≤26 indicating dysfunction) [13], and NSS, evaluated using the Neurological Evaluation Scale (NES) [14], in drug-naïve schizophrenia patients. By focusing on untreated individuals, the study aims to clarify the inherent link between cognitive and neurological deficits.

Methodology

This cross-sectional study involved 60 drug-naïve patients diagnosed with schizophrenia as per the International Classification of Diseases (ICD-10) criteria. The sample size was calculated as follows.

Sample Size

The sample size was estimated using the formula

$$n = \frac{Z^2 S^2}{d^2}$$

where
n is denoted for the required minimum sample size
z = 1.96 at 95% confidence interval
S = Standard deviation of GAF total score 4.18
d = 3% Relative error of mean (mean of GAF Total score=36.54)
d = 1.09

$$n = \frac{(1.96)^2 * 4.18 * 4.18}{(1.09)^2} = 59 \text{ minimum sample}$$

Participants were enrolled from the outpatient department of a tertiary care center in India from July 2023 to March 2025. Eligibility criteria included ages 18–45 years, no prior antipsychotic treatment, and no history of neurological conditions, substance dependence, or significant head trauma. The institutional ethics committee approved the study, and all participants provided informed consent. Cognitive performance was evaluated using the montreal cognitive assessment (MoCA), a validated instrument for identifying mild cognitive impairment as shown in Table 1. The MoCA assesses domains such as attention, memory, language, visuospatial abilities, and executive function, with scores ranging from 0 to 30. A score of ≤26 indicated cognitive impairment, as recommended for psychiatric populations [13]. The MoCA was con-

Table 1: Distribution of cognitive function based on MoCA scores

MoCA score	Frequency	Percent
< 26	39	65.0
> or = 26	21	35.0
Total	60	100.0

Table 2: Distribution of patients according to number of soft signs present

Number of Soft signs Present	Frequency	Percent
0	8	13.3
1	10	16.7
2	6	10.0
3	1	1.7
4	0	0
5	7	11.7
6	8	13.3
7	5	8.3
8	8	13.3
9	4	6.7
10	2	3.3
11	1	1.7
Total	60	100.0

ducted in a controlled, distraction-free setting. Neurological soft signs (NSS) were assessed using the neurological evaluation scale (NES), a validated tool for detecting subtle neurological irregularities in psychiatric patients in India [14-16]. The NES evaluates sensory integration (e.g., Audio-Visual Integration, Graphesthesia), motor coordination (e.g., Fist-Ring Test, Fist-Edge-Palm Test), and domains like memory and rhythm tapping. Tests were scored based on abnormality presence and severity, with higher scores indicating greater impairment. Neurologists, unaware of MoCA scores, performed NES assessments to ensure objectivity. Data analysis was conducted using SPSS version 25.0. Descriptive statistics summarized cognitive impairment and NSS prevalence as shown in Table 2. Pearson Chi-square tests explored associations between MoCA scores (≤26 vs. >26) and specific NSS, with significance set at p < 0.05. The average NSS per patient was calculated by dividing the total NSS by the sample size.

Note

The Study has been reviewed and approved by the Institutional Ethics Committee. (No.IEC/2023/7335-70)

RESULTS

Relevant Sociodemographic Details

The study comprised 60 participants with a mean age of approximately 29.5 years (range 18-45). The largest age group was 26 to 35 years (45.0%), followed by 18 to 25 (43.3%). Most were male (58.3%) and single (53.3%), with Hinduism as the predominant religion (80.0%). Educationally, half had middle or high school qualifications (50.0%). Unemployment was the most common occupation (33.3%), and the highest income bracket was ₹4110 to 8219 (33.3%). Nuclear families prevailed (45.0%), with urban residence slightly more common (51.7%). Illness duration was mostly 7 to 12 months (53.3%), family psychiatric history was absent in most cases (81.7%), and tobacco was the leading substance abuse (46.7%). The majority of participants (65%) demonstrated cognitive impairment on the Montreal Cognitive Assessment (MoCA), with scores below 26, while 35% exhibited normal cognitive function (scores ≥26).

Number of soft signs present in the patients as determined by testing on according to NES scale.

Correlations between cognitive dysfunction (MoCA ≤26) and specific NSS were analyzed using Pearson Chi-square tests, as shown in Table 3. Correlation between cognitive dysfunctions and soft signs present (Based on MoCA rating interpretation and NES)

Pearson Chi-square analyses revealed significant associations (*p* <0.05) between lower MoCA scores (<26, indicating cognitive impairment) and poorer performance across multiple neurological domains in a sample of 60 participants. Specifically, those with impaired MoCA scores showed higher rates of errors or disruptions in:

Sensory Integration and Perception

Audio-visual integration (*p* = 0.001), graphesthesia (*p*=0.027), right/left confusion (*p* = 0.001), and gaze persistence (*p*=0.01).

Table 3a: Cognitive dysfunctions and difficulties with sensory integration

Neurological evaluation scale tests		MoCA score			Pearson Chi-Square Tests
Test	Rating	<26	> OR = 26	Total	p-value
Cerebral dominance	Right	28 (62.2%)	17 (37.8%)	45 (100.0%)	0.435
	Left	11 (73.3%)	4 (26.7%)	100.0%)	
Audio-visual integration	0 = no error	3 (12.5%)	21 (87.5%)	24 (100.0%)	0.001
	1 = one error	14 (100.0%)	0 (0.0%)	14 (100.0%)	
	2 = two or more errors	22 (100.0%)	0 (0.0%)	100.0%)	
Stereognosis	0 = no errors	38 (64.4%)	21 (35.6%)	59 (100.0%)	0.459
	1 = one error	1 (100.0%)	0 (0.0%)	(100.0%)	
Graphesthesia	0 = no errors	28 (57.1%)	21 (42.9%)	49 (100.0%)	0.027
	1 = one error	5 (100.0%)	0 (0.0%)	5 (100.0%)	
	2 = more than one error	6 (100.0%)	0 (0.0%)	6 (100.0%)	
Extinction (Face-Hand Test)	0 = no errors	38 (64.4%)	21 (35.6%)	59 (100.0%)	0.459
	1 = one error	1 (100.0%)	0 (0.0%)	1 (100.0%)	
Right/left confusion	0 = no errors	13 (38.2%)	21 (61.8%)	34 (100.0%)	0.001
	1 = one error	14 (100.0%)	0 (0.0%)	14 (100.0%)	
	2 = two or more errors	12 (100.0%)	0 (0.0%)	12 (100.0%)	
Gaze impersistence	0 = no deviation from fixation	26 (55.3%)	21 (44.7%)	47 (100.0%)	0.01
	1 = deviation from fixation after 20 secs	6 (100.0%)	0 (0.0%)	6 (100.0%)	
	2 = deviation from fixation before 20 secs	7 (100.0%)	0 (0.0%)	7 (100.0%)	

Motor Coordination and Sequencing

Fist-ring test (*p*=0.001), fist-edge-palm test (*p*=0.001), Ozeretski test (*p*=0.001), rapid alternating movements (*p* = 0.001), and rhythm tapping (Parts A and B, both *p*=0.001).

Memory

Immediate word recall (*p*=0.001).

No significant differences were observed in cerebral dominance (*p*=0.435), stereognosis (*p*=0.459), or extinction/face-hand test (*p*=0.459). Overall, these findings suggest that cognitive impairment correlates strongly with deficits in sensory-motor integration, praxis, and memory functions.

DISCUSSION

This study reveals a strong association between cognitive impairments and neurological soft signs (NSS) in drug-naïve schizophrenia patients. The 65% prevalence of cognitive dysfunction (MoCA ≤26) in drug-naïve schizophrenia patients is consistent with recent research highlighting cognitive deficits as a core feature of the disorder, often present at illness onset. For instance, Ugwuonye *et al*.[17] reported an 82.9% prevalence of neurocognitive deficits in first-episode schizophrenia patients using the SCIP tool. Similarly, Goonathilake *et al*. [18] found an 89.3% prevalence of cognitive impairment in schizophrenia patients. Tang *et al*. [19] demonstrated significant cognitive deficits in first-episode drug-naïve schizophrenia patients, with improvements post-treatment. Deng *et al*.[20] revealed significant cognitive impairments in first-episode drug-naïve male schizophrenia patients associated with BMI. Additionally, Khedr *et al*. [21] showed lower MoCA scores in schizophrenia patients compared to controls, underscoring the early onset of these deficits. The average of 4.6 NSS per patient and the 86.7% prevalence of at least one NSS reinforce that

Table 3B: Cognitive dysfunctions and difficulties with sequencing of complex motor tasks

Fist-Ring Test	*0 = no major disruption of motion after first repetition*	*1 (11.1%)*	*8 (88.9%)*	*9 (100.0%)*	*0.001*
	1 = no major disruption of motion after first repetition or complete breakdown of motion	9 (42.9%)	12 (57.1%)	21 (100.0%)	
	2 = major disruption of movement or complete breakdown of motion, or more than four fist ring hesitations or confusions.	29 (96.7%)	1 (3.3%)	30 (100.0%)	
Fist-Edge-Palm Test	0 = no major disruption of motion after first repetition	1 (25.0%)	3 (75.0%)	4 (100.0%)	0.001
	1 = no major disruption of motion after first repetition or complete breakdown of motion	1 (7.1%)	13 (92.9%)	14 (100.0%)	
	2 = major disruption of movement or complete breakdown of motion, or more than four hesitations or position confusions.	37 (88.1%)	5 (11.9%)	42 (100.0%)	
Ozeretski Test	0 = no major disruption of motion after first repetition	0 (0.0%)	2 (100.0%)	2 (100.0%)	0.001
	1 = no major disruption of motion after first repetition or complete breakdown of motion	1 (11.1%)	8 (88.9%)	9 (100.0%)	
	2 = major disruption of movement or complete breakdown of motion, or more than four hesitations or position confusions.	38 (77.6%)	11 (22.4%)	49 (100.0%)	

NSS are inherent to schizophrenia, independent of antipsychotic treatment effects [5].

The study highlights a strong link between cognitive impairments and neurological soft signs (NSS) in drug-naïve schizophrenia patients, suggesting shared neurobiological underpinnings rooted in disrupted brain connectivity and neurodevelopmental abnormalities, including dysfunction in frontocerebellar, frontoparietal, and corticostriatal circuits implicated in sensory integration, motor coordination, and executive function. Recent research by Petrescu et al. [22] supports these findings, reporting higher NSS prevalence in drug-naïve patients with deficits in motor coordination and sensory integration tied to prefrontal and cerebellar changes, aligning with observed correlations in tasks like the Fist-Ring and Fist-Edge-Palm Tests. Similarly, Zhang et al. [23] identified pronounced cognitive deficits in first-episode patients, particularly in processing speed and attention, linked to negative symptoms and reduced cortical thickness in frontal and parietal regions, consistent with graphesthesia impairments. Tsapakis et al. [24] further reinforce this, noting NSS as trait markers reflecting cerebellar and basal ganglia atrophy, underscoring schizophrenia's neurodevelopmental basis with NSS and cognitive impairments as endophenotypes. Dong et al. [25] confirm severe deficits in working memory and verbal learning in first-episode cases, while Petrescu et al. [26] highlight associations with negative symptoms independent of antipsychotics.

Significant differences in Rhythm Tapping and Rapid Alternating Movements (p = 0.001) underscore schizophrenia-specific motor timing deficits, likely tied to cerebellar dysfunction.[27] The absence of correlation with Extinction (p = 0.459) may indicate its lesser relevance to cognitive deficits, possibly due to distinct neural pathways like the temporoparietal junction.[28] These findings suggest that cognitive dysfunction and NSS are integral to schizophrenia, detectable before treatment. Early detection could inform tailored interventions, such as cognitive remediation or motor-focused therapies [29]. NSS may also serve as endophenotypes, aiding in risk identification and genetic research [4]. The drug-naïve focus minimizes medication-related confounds, but the cross-sectional design limits insights into impairment progression. Longitudinal studies and larger, diverse samples are needed to explore these dynamics. Cul-

Table 3C: Cognitive dysfunctions and difficulty in motor coordination

(a) Rhythm Tapping Test Part A	*0 = no errors*	*2 (10.5%)*	*17 (89.5%)*	*19 (100.0%)*	*0.001*
	I = one error of either non-discrimination between soft and hard sounds, rhythm, or error in number of taps	3 (50.0%)	3 (50.0%)	6 (100.0%)	
	2 = more than one error	34 (97.1%)	1 (2.9%)	(100.0%)	
1.(b) Rhythm Tapping Test Part B	0 = no errors	3 (14.3%)	18 (85.7%)	21 (100.0%)	0.001
	I = one error	4 (57.1%)	3 (42.9%)	7 (100.0%)	
	2 = more than one error	32 (100.0%)	0 (0.0%)	32 (100.0%)	
Rapid Alternating Movements	0 = no major disruption of motion, hesitation, or mistake in hand placement	2 (14.3%)	12 (85.7%)	14 (100.0%)	0.001
	1= no major disruption of motion or one to two hesitations or mistakes in hand placement	17 (65.4%)	9 (34.6%)	26 (100.0%)	
	2 = major disruption of motion or three or more hesitations or mistakes in hand placement.	20 (100.0%)	0 (0.0%)	20 (100.0%)	

Table 3D: Cognitive dysfunctions and difficulties with memory

Memory	*0 = Subject remembers all words*	*7 (25.9%)*	*20 (74.1%)*	*27 (100.0%)*	*0.001*
	1 = Subject remembers three words	13 (92.9%)	1 (7.1%)	14 (100.0%)	
	2 = Subject remembers fewer than three words	19 (100.0%)	0 (0.0%)	19 (100.0%)	

tural and genetic factors, such as dopamine pathway variations in Indian populations, may influence these impairments, warranting further investigation [30].

Conclusion

In conclusion, this study underscores the intrinsic connection between cognitive impairments and neurological soft signs in drug-naïve schizophrenia patients, reinforcing the neurodevelopmental origins of the disorder through shared disruptions in fronto-cerebellar and subcortical pathways. Clinically, these insights advocate for early, integrated assessments to inform personalized interventions, potentially improving patient outcomes, while highlighting the potential of neurological soft signs as biomarkers for risk stratification and monitoring. However, the study's limitations include its cross-sectional design, which precludes causal inferences; a relatively small sample size, limiting generalizability; reliance on a single cognitive screening tool, potentially overlooking domain-specific deficits; and recruitment from a single center in India, which may not capture broader sociocultural or genetic variations. Future research should prioritize longitudinal designs with larger, multicultural cohorts to track impairment trajectories and treatment impacts. There are no conflicts of interest.

References

1. Green MF. Cognitive impairment and functional outcome in schizophrenia and bipolar disorder. J Clin Psychiatry. 2006;67(Suppl 9):3-8. https://pubmed.ncbi.nlm.nih.gov/16965182/

2. Keefe RS, Fenton WS. How does cognitive impairment in schizophrenia impact real-world functioning? CNS Spectr. 2007;12(10):741-745.

3. Bombin I, Arango C, Buchanan RW. Significance and meaning of neurological signs in schizophrenia: a review. Schizophr Bull. 2005;31(4):962-977. https://doi.org/10.1093/schbul/sbi028

4. Chan RC, Gottesman II. Neurological soft signs as candidate endophenotypes for schizophrenia: a shooting gallery of new ideas. Neurosci Biobehav Rev. 2008;32(3):558-569. https://doi.org/10.1016/j.neubiorev.2007.12.001

5. Dazzan P, Murray RM. Neurological soft signs in first-episode psychosis: a systematic review. Br J Psychiatry. 2002;181(Suppl 43):s50-s57. https://doi.org/10.1192/bjp.181.43.s50

6. Weinberger DR. Implications of normal brain development for the pathogenesis of schizophrenia. Arch Gen Psychiatry. 1987;44(7):660-669. https://doi.org/10.1001/archpsyc.1987.01800190066013

7. World Health Organization. Schizophrenia. WHO fact sheet. January 10, 2022. https://www.who.int/newsroom/fact-sheets/detail/schizophrenia

8. GBD 2019 Mental Disorders Collaborators. Global, regional, and national burden of 12 mental disorders in 204 countries and territories, 1990–2019: A systematic analysis for the Global Burden of Disease Study 2019. The Lancet Psychiatry. 2022;9(2):137–150. https://doi.org/10.1016/S2215-0366(21)00395-3

9. Gururaj G, Varghese M, Benegal V, Rao GN, Pathak K, Singh LK, … & NMHS Collaborators Group. National Mental Health Survey of India, 2015–16: Prevalence, patterns and outcomes. Bengaluru: National Institute of Mental Health and Neuro Sciences, NIMHANS Publication No. 129; 2016. https://indianmhs.nimhans.ac.in/phase1/Docs/Report2.pdf

10. Goyal S, Goyal V. Increasing prevalence of patients with Schizophrenia in India. Journal of Hospital Pharmacy. 2021;16(1):1-5. https://www.researchgate.net/publication/348884326_Increasing_prevalence_of_patients_with_Schizophrenia_in_India

11. Andreasen NC, Paradiso S, O'Leary DS. "Cognitive dysmetria" as an integrative theory of schizophrenia: a dysfunction in cortical-subcortical-cerebellar circuitry? Schizophr Bull. 1998;24(2):203-218. https://doi.org/10.1093/oxfordjournals.schbul.a033321

12. Thara R, Eaton WW. Outcome of schizophrenia: the Madras longitudinal study. Aust N Z J Psychiatry. 1996;30(4):516-522. https://doi.org/10.3109/00048679609065026

13. Nasreddine ZS, Phillips NA, Bédirian V, et al. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. J Am Geriatr Soc. 2005;53(4):695-699. https://doi.org/10.1111/j.1532-5415.2005.53221.x

14. Buchanan RW, Heinrichs DW. The Neurological Evaluation Scale (NES): a structured instrument for the assessment of neurological signs in schizophrenia. Psychiatry Res. 1989;27(3):335-350. https://doi.org/10.1016/0165-1781(89)90148-0

15. Varambally S, Venkatasubramanian G, Sharma S, Gangadhar BN. Neurological soft signs in schizophrenia: A cross-sectional study from India. Indian Journal of Psychiatry. 2006;48(3):161–165. https://doi.org/10.4103/0019-5545.31579

16. Rao NP, Venkatasubramanian G, Arasappa R, Gangadhar BN. Relationship between neurological soft signs and neurocognitive deficits in schizophrenia: An Indian study. Asian Journal of Psychiatry. 2010;3(4):218–221. https://doi.org/10.1016/j.ajp.2010.09.002

17. Ugwuonye OK, et al. Prevalence of neurocognitive deficits in patients with first-episode schizophrenia in an African sample and its relationship with dimensions of psychopathology and psychosocial outcome. BMC Psychiatry. 2024;24:659. https://doi.org/10.1186/s12888-024-06315-9

18. Goonathilake P, et al. Prevalence and correlates of cognitive impairment in schizophrenia: a cross-sectional study from a teaching hospital southern Sri Lanka. BMC Psychiatry. 2022;22:670. https://doi.org/10.1186/s12888-022-04368-2

19. Tang C, et al. Comparison of cognitive performance in first-episode drug-naïve schizophrenia, bipolar II disorder, and major depressive disorder patients after treatment. BMC Psychiatry. 2024;24:468. https://doi.org/10.1186/s12888-024-05897-8

20. Deng X, et al. Association between increased BMI and cognitive function in first-episode drug-naïve male schizophrenia. Frontiers in Psychiatry. 2024;15:1362674. https://doi.org/10.3389/fpsyt.2024.1362674

21. Khedr EM, et al. Cognitive impairment among an Egyptian sample of patients with schizophrenia and bipolar disorders: a comparative study. Middle East Current Psychiatry. 2023;30:78. https://doi.org/10.1186/s43045-023-00344-y

22. Petrescu C, et al. Neurological Soft Signs in Schizophrenia, a Picture of the Knowledge in the Last Decade: A Scoping Review. Healthcare. 2023;11(10):1471. https://doi.org/10.3390/healthcare11101471

23. Zhang T, et al. Cognitive Impairment in First-Episode Psychosis With Predominant Negative Symptoms. JAMA Network Open. 2024;7(6):e2415110. https://doi.org/10.1001/jamanetworkopen.2024.15110

24. Tsapakis EM, et al. Neurological soft signs and schizophrenia. CNS Spectrums. 2023;28(5):539-550. https://doi.org/10.1017/S109285292300002X

25. Dong F, et al. Cognitive deficits profiles in the first-episode of schizophrenia, clinical high risk of psychosis, and genetically high-risk of psychosis. Front. Psychiatry. 2023;14:1292141. https://doi.org/10.3389/fpsyt.2023.1292141

26. Petrescu C, et al. Clinical and Sociodemographic Correlations with Neurological Soft Signs in Hospitalized Patients with Schizophrenia: A Preliminary Longitudinal Study. Biomedicines. 2024;12(4):787. https://doi.org/10.3390/biomedicines12040787

27. Torrey EF. Schizophrenia and the cerebellum: a new hypothesis. Schizophr Res. 2007;96(1-3):1-3.

28. Karnath HO, Fruhmann Berger M, Küker W, et al. The anatomy of spatial neglect based on voxelwise statistical analysis: a study of 140 patients. Cereb Cortex. 2004;14(10):1164-1172. https://doi.org/10.1093/cercor/bhh076

29. Wykes T, Huddy V, Cellard C, et al. A meta-analysis of cognitive remediation for schizophrenia: methodology and effect sizes. Am J Psychiatry. 2011;168(5):472-485. https://doi.org/10.1176/appi.ajp.2010.10060855

30. Howes OD, Kapur S. The dopamine hypothesis of schizophrenia: version III—the final common pathway. Schizophr Bull. 2009;35(3):549-562. https://doi.org/10.1093/schbul/sbp018

Body Image and "Sexy" in India

Rehan Haider[1*], Zameer Ahmed[2], Geetha Kumari Das[3]

[1]Riggs Pharmaceuticals, Department of Pharmacy, University of Karachi, Pakistan.
[2]University of Health Sciences, Karachi, Pakistan.
[3]GD Pharmaceutical Inc. OPJS University, Rajasthan, India.

Abstract

In India, women's perceptions of body image and sexual attractiveness are shaped by the interplay of traditional cultural norms, modern media influences, and shifting societal values. Attributes such as breast size, body shape, and nipple appearance have become central to how female sexuality is interpreted. Historically, beauty ideals emphasized modesty, humility, and grace, aligning with family-centered and conservative values. With globalization and increased media exposure, however, these ideals are evolving, and notions of what is considered "sexy" are changing.

In contemporary society, body ideals often highlight larger breasts and slim, hourglass figures as markers of femininity and sexual appeal, particularly in urban contexts. Nevertheless, these standards are not universally accepted, as many women continue to value traditional and natural representations of beauty. Nipple size, though rarely discussed, also contributes to body image concerns, adding further pressure on women to conform to narrowly defined ideals.

The tension between modern expressions of sexuality and cultural traditions creates conflicting expectations for Indian women. While media and cosmetic surgery promote new standards, social and familial norms continue to emphasize modesty. This paper examines how cultural values, media portrayals, and physiological features influence Indian women's perceptions of their bodies, sexuality, and evolving standards of attractiveness.

INTRODUCTION

In India, women's perceptions of body image and attractiveness are shaped by a combination of biological, cultural, and social influences. Traditionally, Indian society emphasized modesty, humility, and family-centered values as markers of feminine identity, with limited focus on external appearance.[1] Historically, female bodies were often assessed through their roles in fertility, nurturing, and strength rather than physical beauty or sexual appeal. In recent decades, however, globalization and modern media have contributed to a shift toward ideals that prioritize external features such as body shape, size, and sexual attractiveness.[2]

Among the most debated aspects of body image in contemporary India is breast size, which increasingly influences perceptions of femininity and sexual-

ity. Whereas smaller breasts were once considered modest and ideal within traditional frameworks, larger breasts are now more commonly associated with desirability, especially in urban areas. This change reflects the rising visibility of Western beauty standards and the availability of cosmetic enhancements, such as breast augmentation.[3] This trend diverges significantly from earlier Indian beauty norms, which valued natural and unaltered body types.[4]

Body shape also plays a crucial role in beauty standards. Media portrayals often highlight hourglass figures and fuller breasts as the ideal, reinforcing social pressures on women to conform to these expectations.[2] Yet, these ideals frequently clash with conservative views that emphasize modesty and natural physique.[5] Bollywood has further amplified the sexualization of women's bodies, making cosmetic surgery, particularly breast enhancement, increasingly attractive to urban women.[6] Nonetheless, this pursuit of media-driven ideals is not universally accepted, particularly in rural or conservative communities where modesty and family obligations continue to dominate cultural expectations.[7]

This tension between traditional and modern ideals creates a layered understanding of body image in India. Women in urban contexts are more likely to internalize Westernized notions of attractiveness, while women in rural regions tend to adhere to traditional ideals.[9] The growing demand for body modifications, such as cosmetic surgery, suggests a shift toward globalized beauty ideals.[10] However, cultural, religious, and familial influences continue to mediate how women define beauty and acceptability.[11]

The discourse on sexuality among Indian women remains complex. Although there is increasing openness toward sexual self-expression, social norms and conservative family expectations still play a significant role in shaping attitudes toward female sexuality ^1. This creates a dual reality where women navigate both external pressures to conform to evolving beauty standards and internalized values rooted in tradition.[12]

The objective of this study is to examine the shifting perspectives on body image and sexuality among Indian women, with particular attention to breast size, body shape, and sexual appeal. By analyzing the role of media, cultural traditions, and social expectations, this research aims to highlight the complexity of body representation in contemporary India.[13] Importantly, the balance between preserving traditional values and embracing liberated expressions of sexuality continues to influence how Indian women view their bodies and femininity.[14]

This paper explores these evolving concepts of body image and sexuality, with a focus on breast size, body shape, and cultural expectations, within the intersecting contexts of media, social pressure, and tradition.[15-17]

Literature Review

The idea of body image has existed widely intentionally across cultures, revealing how societal beliefs influence women's self-perceptions. In the Indian framework, historically established beliefs placed better emphasis on women's ancestral and social duties, alternatively, their physical image.[1] With the proliferation and expansion of radio, nevertheless, the importance of beauty has aged significantly, particularly in urban environments. The influence of Western moral beliefs has risked a pivotal function in changing local guidelines, leading many women to attempt body types that stress a physical restriction, larger breasts, and different body shapes.[3]

Breast Size and Body Shape

Breast augmentation has arisen as one of the most effective features in forming ideas of femininity and sexual appeal across many cultures, including India. In conservative domains, tinier breasts were, as a rule, visualized as appropriate and ideal, yet growing exposure to global principles has fluctuated preferences toward the best breast sizes. Bollywood and different media outlets commonly represent women with more adequate breasts as symbols of beauty, reinforcing social pressures to reach these ideals.[13]

Similarly, the ideal body shape has progressed over time, accompanying the watch figure gaining extensive acknowledgment. The desire to match these standards has contributed to the rising demand for beautifying processes, including breast enlargement, specifically among urban women.[6]

Cultural and Social Influences

Cultural and societal determinants play an essential in forming body countenance. In rural communities, established ethics still emphasize modesty, normal physique, and less devoted effort to something sexualized beauty, whereas urban women are increasingly affected by globalized averages that prioritize unconcealed girlishness and sexuality ^8. This urban–rural divide underlines the complicatedness of the body concept in India, emphasizing how educational principles mediate the agreement of worldwide beauty principles.[9]

Research Methodology

This study uses an assorted-methods design to inspect progressing perceptions of physique countenance and sexuality between Indian women. A determinable component was used to capture large-scale styles, while subjective interviews provided deeper insight into enlightening beliefs and private happenings.

Quantitative Approach

Survey data were collected from 500 women, evenly divided between urban and rural domains, and aged 18–40 years. The survey included legalized tools such as the Body Shape Questionnaire (BSQ) and the Sociocultural Attitudes Towards Appearance Questionnaire (SATAQ) to determine satisfaction accompanying body countenance, breast size, and crowd shape.

Qualitative Approach

Semi-organized interviews were conducted with a subsample of partners. Open-ended questions surveyed women's ideas of beauty, sexuality, and friendly beliefs, along with their views on beautifying processes. This approach admits to a richer circumstantial dossier on how enlightening principles and media uncovering communicate accompanying individual self-image.

Ethical Considerations

All members were cognizant of the purpose of the study and provided informed consent. Confidentiality was upheld throughout the dossier group and reasoning processes. Ethical approval was obtained from the Institutional Review Board.

Statistical Analysis

Quantitative data were resolved utilizing SPSS version 26.0. Descriptive enumerations (repetitions, percentages, and methods) summarized participants' answers. Chi-square tests were applied to equate dissimilarities in perceptions between urban and rural groups, while independent t-tests evaluated differences in body concept delight across age types.

For the qualitative dossier, having a theme analysis was working. Interview transcripts were systematized, and recurrent ideas were recognized to capture enlightening influences, media impact, and ideas of material ideals. The unification of the two datasets allowed triangulation, heartening the dependability of the judgments.

Results

The analysis provided both quantitative and qualitative insights into women's perceptions of body image in India. Survey findings demonstrated that breast size, nipple diameter, and body shape varied across age groups, with a clear trend of increasing breast size and nipple diameter as women aged as shown in Table 1. For instance, women aged 18–24 most frequently reported small to medium breast sizes, nipple diameters of 1–2 cm, and hourglass or pear-shaped bodies, with 75% satisfaction. In contrast, women aged 45 and above commonly reported larger breasts (3–4 cm nipple diameter) and pear or apple body shapes, though only half reported being satisfied. This indicates a decline in body satisfaction with age despite natural physiological changes.

Urban women expressed greater dissatisfaction, with 70% desiring larger breasts and slimmer waistlines, often referencing Bollywood and Western media as influencing factors. Meanwhile, rural participants emphasized modesty and cultural ideals, with 55% satisfied with their natural body type. Interview responses highlighted generational differences, as younger women were more likely to

Table 1: Breast sizes, nipple sizes, and body shape according to age groups

Age Group	Average Breast Size	Average Nipple Size (Diameter)	Common Body Shape	Percent of Women Reporting Satisfaction with Size
18–24	Small to Medium	1–2 cm	Hourglass, Pear	75%
25–34	Medium to Large	2–3 cm	Hourglass, Apple	68%
35–44	Medium to Large	2–3 cm	Apple, Pear	60%
45–54	Large	3–4 cm	Pear, Apple	50%
55+	Medium to Large	2–3 cm	Pear, Inverted Triangle	40%

Source: Data collected from a survey of 500 women, with an even split between urban and rural populations. A survey was conducted in 2023 across various regions of India.
Source: Data from the Indian Body Image Survey 2023, conducted in collaboration with Dr Rehan Haider Riggs Pharmaceuticals, a study examining women's body image perceptions and physical attributes across different age groups in India.
Nipple size distribution varied with age groups, ranging from 1–2 cm in women aged 18–24 years to 3–4 cm in women aged 45 years and older. These findings suggest that nipple size increases gradually with age.
Source: Data from Indian Body Image Survey 2023, part of an ongoing longitudinal study on body image and cosmetic surgery trends in urban and rural India
Source: Data sourced from interviews with 500 women (rural and urban), part of the broader study on body image and self-perception trends in India over the last two decades (2003-2023).

idealize the hourglass figure, while older women accepted natural variations such as pear or apple shapes. Plastic surgery and cosmetic interventions were reported or considered by 35% of urban women, while rural women largely rejected such practices.

The analysis showed meaningful contrasts between urban and rural participants. Among urban women, 70% expressed dissatisfaction with the size of their breasts, accompanied most of whom favor larger breasts. Approximately 65% stated that the impression was pressured to persuade Western ideals, specifically those represented in Bollywood.

In contrast, rural women were less affected by these globalized factors. Over half (55%) expressed delight accompanying their natural physique shape, and 80% stressed the importance of modesty and kin-concentrated values over outside presentation.

Thematic analysis of interviews further supports these verdicts. Urban women frequently noted plastic surgery—particularly breast augmentation—as an alternative, with 35% newsgathering they had either sustained or considered it. Rural women, still, frequently resisted the plan of surgical augmentation, aligning with established values of normal beauty and simplicity.

Discussion

These results reveal how body image ideals are strongly mediated by age, geography, and cultural orientation. The quantitative trends highlight that body satisfaction declines as women age, corresponding to physiological changes in breast size and shape. This aligns with prior research that links body image concerns with aging and media exposure. Urban women's greater interest in cosmetic surgery underscores the influence of globalization and Bollywood portrayals of femininity. By contrast, rural women continue to adhere to traditional frameworks that value modesty, natural beauty, and family honor.

Qualitative interview data reinforced this divergence. Younger participants emphasized Westernized ideals such as the hourglass figure and associated breast size with attractiveness. Older and rural women, however, placed greater emphasis on inner values, traditional norms, and resilience. These contrasting views illustrate the cultural negotiation that Indian women undergo in reconciling modern and traditional expectations of beauty. The dual pressures contribute to psychological challenges, including body dissatisfaction and anxiety over appearance. Thus, while globalized ideals are becoming more prominent, cultural and familial norms remain powerful mediators of women's body image in India.

The judgments climax a tension between worldwide beauty ideals and established Indian principles. Urban women are more affected by Westernized standards, which stress the best breasts and waist

figures. This joins with prior studies connecting media exposure to raised physique dissatisfaction [3]. By contrast, rural women continue to support modesty and natural habit as principles, copying research on cultural causes of body image [5].

The growing interest in plastic surgery among urban participants suggests that globalization and Bollywood's portrayals of girlishness are changing women's self-image. However, this shift is not everywhere recognized; traditional anticipations about family honor and modesty remain central in many countries' societies. These two-fold pressures contribute to mental challenges as women navigate contradictory goals of beauty and sensuality.

Conclusion

This study shows that party image ideas among Indian women are enduring, meaningful transformations. Urban women are increasingly adopting globalized beauty standards affected by television, while rural women embrace established principles of modesty and natural beauty. The climbing demand for cosmetic surgery highlights the desire to follow the agreement or instructions of new ideals, though these practices are disputed in conservative settings.

Future research endeavors to investigate the psychological results of these changeable standards, particularly their effect insane health, pride, and public relationships. Understanding these movements is critical for advancing healthier stances toward body image and for cultivating with regard for the welfare of mankind, sensitive invasions that balance established principles with existing pressures.

Acknowledgment

Authors Contribution

Conflict of Interest

Funding and Financial Support

References

1. Schilder P. The Image and Appearance of the Human Body. London: Kegan Paul, Trench, Trubner; 1935.
2. Tiggemann M, Lynch JE. Body image across the life span in adult women: The role of self-objectification. Dev Psychol. 2001;37(2):243–53.
3. Grabe S, Ward LM, Hyde JS. The role of the media in body image concerns among women: A meta-analysis of experimental and correlational studies. Psychol Bull. 2008;134(3):460–76.
4. Dittmar H, Howard S. Thin-ideal media images and women's body dissatisfaction: Association, moderation, and mediation in the UK and the US. J Soc Clin Psychol. 2004;23(1):1–24.
5. Swami V, Tovée MJ. Male and female physical attractiveness in Britain and Malaysia: A cross-cultural study. Body Image. 2005;2(2):115–28.
6. Bessenoff GR. Can the media affect us? Social comparison, self-discrepancy, and the thin-ideal. Psychol Women Q. 2006;30(3):239–51.
7. Cash TF. Body image: Past, present, and future. In: Cash TF, Pruzinsky T, editors. Body Image: A Handbook of Theory, Research, and Clinical Practice. New York: Guilford Press; 2002. p. 3–10.
8. Stewart TM. The relationship between body image and cultural values: Understanding the connection. J Cross Cult Psychol. 2008;39(3):123–37.
9. Goffman E. The Presentation of Self in Everyday Life. Garden City, NY: Doubleday Anchor; 1959.
10. Maloney MJ, McGuire JB, Daniels AE. Eating disorders and body image in women: The impact of societal standards. Int J Eat Disord. 1999;26(1):79–88.

11. Swami V. Body appreciation, media influence, and weight discrepancy in female adolescents. Body Image. 2009;6(3):199–203.

12. Striegel-Moore RH, Smolak L. Understanding Eating Disorders: Integrating Etiology, Prevention, and Treatment. Mahwah, NJ: Lawrence Erlbaum Associates; 2001.

13. Tiggemann, M. Considerations of body image and eating disorders in the media. Sex Roles. 2013;69(11–12):613–21.

14. Kumanyika SK, Charleston JB. Media influences on body image and health behavior. Am J Health Behav. 2001;25(5):498–508.

15. Striegel-Moore RH, Smolak L, editors. Eating Disorders: Innovative Directions in Research and Practice. Washington, DC: American Psychological Association; 2001.

16. Tiggemann M, Lynch JE. Self-objectification in women: Causes, consequences, and prevention. Dev Psychol. 2001;37(2):243–53.

17. Cash TF, Pruzinsky T, editors. Body Image: A Handbook of Theory, Research, and Clinical Practice. 2nd ed. New York: Guilford Press; 2002.

A Cross-sectional, Multicenter, Observational Study to Assess the Safety and Usage Pattern of Antidepressants in the Management of Indian Patients with Major Depressive Disorder

Dharmesh Choksi[1], Vishal Chhabra[2], K S Pavitra[3], Ramani P N[4], Sharmistha Chakroborty[5], Yakshdeep Dave[6], Zahraan Qureshi[6], Girish Kulkarni[6*]

[1]Mamta Hospital & Research Center, Medicare centre, Near M J Library, Ellisbridge, Ahmedabad, Gujarat, 380006, India
[2]Chhabra Psychiatry Centre, 107-108, 1st Floor, RG-1 Complex, Prashant Vihar Sector 14, New Delhi, India
[3]Sridhar nursing home complex, Rajendranagar, Shimoga, Karnataka – 577204, India
[4]Sita Laxmi Hospital, 21 Koweli Brown Road, R.S.Puram East, Coimbatore, Tamil Nadu 641002, India
[5]Samikshani 37 Soythend Park, Bhakuria, Dover Lane, Kolkata, West Bengal, India
[6]Medical Affairs, Torrent Pharmaceuticals Ltd, Ahmedabad, India

How to Cite:

Choksi D, Chhabra V, Pavitra KS, Ramani PN, Chakroborty S, Dave Y, Qureshi Z, Kulkarni G. A Cross-sectional, Multicenter, Observational Study to Assess the Safety and Usage Pattern of Antidepressants in the Management of Indian Patients with Major Depressive Disorder. Indian Journal of Clinical Psychiatry. 2025;5(2): 63-71.

doi: 10.54169/ijocp.v5i02.09

Abstract

Background: Major depressive disorder (MDD) is a leading cause of disability worldwide, with rising prevalence. Antidepressants (ADs) remain the cornerstone of treatment for depression and anxiety. However, data on AD prescribing patterns in India are limited, which is essential for optimizing treatment strategies.

Objective/Aim: To evaluate the usage and safety patterns of commonly prescribed ADs (escitalopram, sertraline, and vortioxetine) in Indian patients with MDD.

Methods: This retrospective, observational study analyzed data of 3,321 Indian patients having MDD. Data were collected by 306 psychiatrists and clinicians using structured DCFs, and analyses were performed using SPSS (v29.0.1.0) and Microsoft Excel 2019. Ethics approval was obtained before study initiation.

Results: The mean (SD) age of the study population was 44.0 (12.9) years, with males comprising 64.8% of the cohort. Most patients (70.6%) reported no family history of MDD. Anxiety was the most common psychiatric comorbidity (59.0%), while diabetes (22.9%) was the most frequent non-psychiatric comorbidity. Among antidepressants, escitalopram was the most prescribed agent (50.6%), followed by vortioxetine (28.1%) and sertraline (21.0%). Clonazepam was the most frequently co-prescribed benzodiazepine (36.0%). Escitalopram was commonly initiated at 10 mg once daily (80.0%), predominantly as monotherapy (55.2%), with treatment duration of 2–6 months (39.7%). Nausea (24.5%) and dizziness (13.9%) were the most reported adverse events.

Conclusion: Escitalopram emerged as the preferred AD, with frequent clonazepam co-prescription for comorbid anxiety. These patterns reflect common clinical practice and highlight the need for individualized treatment strategies in MDD management.

INTRODUCTION

Major Depressive Disorder (MDD), as defined by DSM-5 and ICD-10/11, is characterized by a prolonged episode of depressed mood or loss of interest, accompanied by symptoms such as changes in appetite or weight, sleep distur-

bances, fatigue,[1,2] feelings of worthlessness, impaired concentration, and recurrent thoughts of death or suicide. Globally, depression affects more than 300 million people and is recognized as a leading contributor to disability, with the World Health Organization ranking it the third highest cause of global disease burden in 2018 and projecting it to become the leading cause by 2030. Vulnerable populations such as women, elderly, and adolescents show a particularly high risk, influenced by genetic, psychological, and socio-environmental factors.[3] In India, the National Mental Health Survey (NMHS) 2015–2016, covering over 34,000 adults across 12 states, reported a lifetime prevalence of depressive disorders of 5.25% and a current prevalence of 2.68%. Contributing factors include urbanization, lifestyle changes, and rising comorbid non-communicable diseases. The disability burden is considerable, with most affected individuals experiencing impairment in work, family, or social functioning, while nearly 80% remain untreated, reflecting significant gaps between prevalence and effective clinical management.[4] Comorbid conditions are common in MDD, with frequent co-occurrence of anxiety disorders, cardiovascular disease, diabetes, and other chronic illnesses.[5] Such multimorbidity often necessitates polypharmacy, which increases the risk of drug–drug interactions, adverse events, and reduced adherence due to complex treatment regimens and side effects.[6,7] A wide range of antidepressant classes are available, including tricyclic antidepressants (TCAs), monoamine oxidase inhibitors (MAOIs), selective serotonin reuptake inhibitors (SSRIs), serotonin–noradrenaline reuptake inhibitors (SNRIs), and other newer agents [8,9] Globally, the growth in antidepressant prescriptions has been driven largely by the increasing use of SSRIs and newer drugs. However, challenges persist, including variability in prescribing practices, issues of underuse or overuse, poor adherence, and safety concerns.[10,11] Despite the clinical importance of antidepressant therapy, real-world evidence on prescribing patterns and safety in India remains limited, underscoring the need for systematic evaluation in local practice settings. The aim of this study was to evaluate the safety and utility patterns of the antidepressants (vortioxetine, escitalopram, and sertraline) in patients having MDD.

Material And Methods

Study Design

This was a non-interventional, retrospective, observational study conducted across 306 outpatient sites in India between January 2023 and January 2024. Male and female patients with a confirmed diagnosis of major depressive disorder (MDD) who had received treatment with at least one of the antidepressants (vortioxetine, escitalopram, or sertraline) were included in the study.

The study protocol was reviewed and approved by the Sangini Hospital Ethics Committee, Ahmedabad, Gujarat, India (Registration no: ECR/147/Inst/GJ/2013/RR-19 and IORG0007258). The study was conducted in accordance with the principles outlined in the Declaration of Helsinki (2013 revision), the Indian Council of Medical Research (ICMR) National Ethical Guidelines for Biomedical and Health Research Involving Human Participants (2017), and all applicable regulatory requirements.

Study Objective

The primary objective of the study was to evaluate the safety and usage pattern of commonly used antidepressants with a focus on vortioxetine, escitalopram, and sertraline in the management of MDD in Indian patients.

The secondary objective of the study was to understand and document the prevalence and medications used in other co-morbid conditions along with depression in Indian patients.

Study Procedures

Data were first extracted from patient medical records and transcribed into standardized data collection forms (DCFs) by the clinicians and psychiatrists. The completed DCFs were cross verified against the original medical records to ensure accuracy. Following this, the verified data were entered into the excel. A second round of quality checks was performed by comparing the entered data with the respected DCFs to confirm completeness and consistency before statistical analysis. Incomplete data or partially filled DCFs were excluded in the analysis.

Data Collection

Patients' demographic data including their age, gender, height, body weight, marital status, smoking, and alcohol history were captured. Additionally, family history along with the duration of depression and the risk factors were noted with the psychiatric and non-psychiatric comorbidities, if any.

Antidepressant drug details were captured which included the antidepressant name, dose, and its frequency with duration and type of therapy and co-prescribed medicines, if any. Lastly, the safety profile was documented based on past medical records.

Sample Size Calculation

No formal sample size calculation was performed, being a real-world, retrospective observational study, sample size was carried out pragmatically,[12] based on the number of eligible patient medical records available during the study period (January 2023–January 2024), a total of 3,500 patient records were analysed across 306 outpatient sites in India.

Statistical Analysis

Statistical analyses were performed using SPSS software (version 29.0.1.0; IBM, USA) and Microsoft Excel 2019. Data from subjects who met eligibility criteria and had complete records were included in the analysis. Descriptive statistics were used to summarize the data. Categorical variables were presented as frequencies and percentages, while continuous variables were summarized using mean and standard deviation (SD).

RESULTS

A total of 3,500 patient records were screened for eligibility. Of these, 179 records were excluded due to incomplete data. Consequently, complete case analysis was performed, and 3,321 patient records were included in the final analysis set. The study process flow is depicted in Figure 1.

Demographic Distribution

The demographic distribution indicated that the male-to-female ratio was nearly 2:1 in the study.
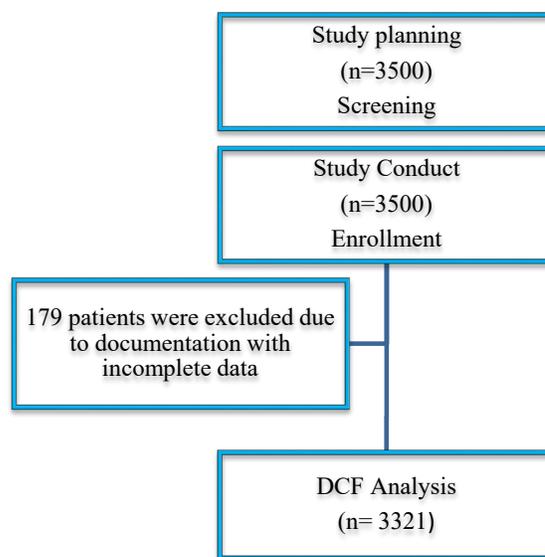
Figure 1: Study Process Flow (CONSORT diagram)

The mean age of the study population was 44 (12.9) years. Almost 74% of the patients belonged to the age group 30-60 years. A summary of demographic distribution is shown in Table 1.

Antidepressant Usage Pattern

Of the total study population, 1682 (50.6%) patients were prescribed escitalopram, while 932 (28.1%), and 697 (21.0%) patients were prescribed vortioxetine and sertraline, respectively. Most common starting and maintenance dose for escitalopram was 10mg, for vortioxetine 5 and 10mg, and for sertraline 50 and 25mg, respectively. For vortioxetine and sertraline, patients were down titrated to a lower dose when it came to maintenance.

Most of the subjects (2658 (80%)) were managed with a once-daily dose. Of the total study population, 1320 (39.7%) received antidepressant therapy for the duration of 2-6 months, and 886 (26.7%) received it for 6-12 months.

The trend for monotherapy was slightly higher, at 55%, compared to the combination therapy. The antidepressant usage pattern is depicted in Figure 2.

Drugs Co-prescribed with Antidepressants

Clonazepam was the most commonly co-prescribed drug. It was present in 1195 (36%) of prescriptions. A

Table 1: Demographic distribution

Characteristic		N
Total Study Population		3321 (100%)
Gender	Female	1168 (35.2%)
	Male	2153 (64.8%)
Age Group (years)	<30	446 (13.4%)
	30-44	1392 (41.9%)
	45-60	1075 (32.4%)
	>60	408 (12.3%)
Age (years)	Mean (SD)	44.0(12.9)
Weight (kg)	Mean (SD)	67.0(11.3)
Height (cm)	Mean (SD)	163.4(8.9)

summary of the distribution of co-prescribed drugs is mentioned in Table 2.

Patient Characteristics

A family history of depression was seen in 977 (29.4%) patients. Trauma or negative life events (1755 (52.8%)) emerged as a common risk factor. The duration of depression in more than 85% of patients was less than 3 years. Rural and urban settings of patients showed a similar distribution of prevalence. Most patients were married (2528 (76.2%)). A summary of the patient's characteristics is mentioned in Table 3.

Prevalence of Co-morbid Conditions

Anxiety (59%) was noted as the most common psychiatric comorbidity amongst the study population followed by schizophrenia (11.1%), and substance abuse (10.1%). Lifestyle diseases such as diabetes (22.9%) and hypertension (19.7%) were the commonest non-psychiatric comorbidities. A summary of the distribution of co-morbid conditions is presented in Table 4.

Safety Profile

Nausea (814 (24.5%)) was the most commonly reported adverse reaction followed by dizziness (462 (13.9%), and vomiting (422 (12.7%)).

DISCUSSION

This large-scale, retrospective pharmacoepidemiologic study assessed the safety and usage patterns
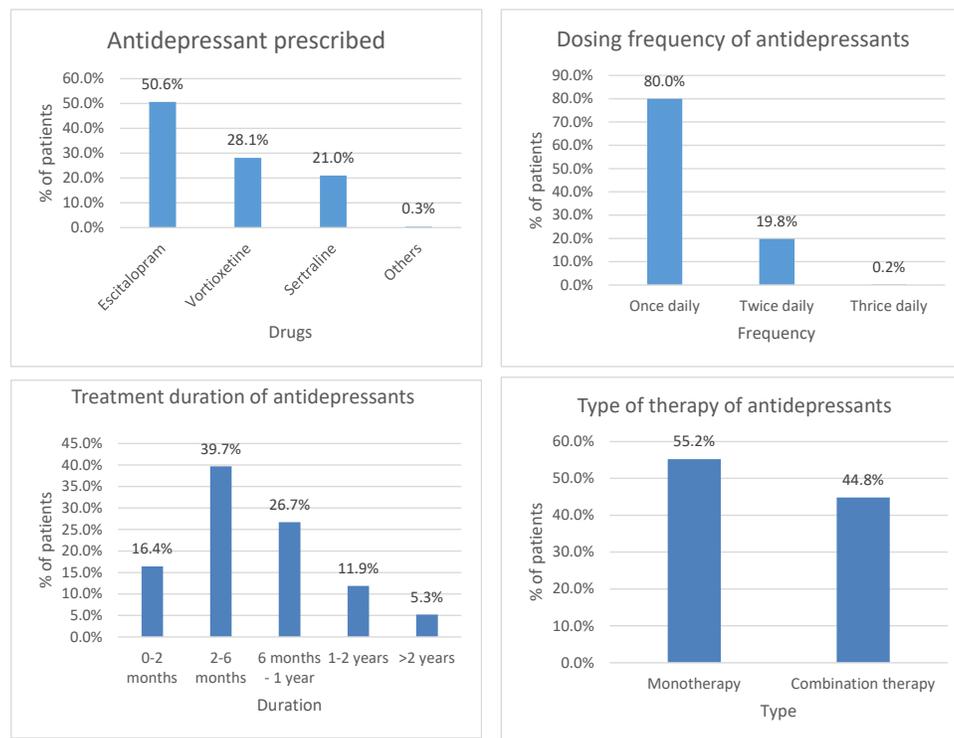
Figure 2: Usage pattern of Antidepressants

Table 2: Summary of co-prescribed drugs

Co-prescribed Drugs		N	%
If combination therapy, commonly combined with	Clonazepam	1195	36.0%
	Alprazolam	142	4.3%
	Etizolam	106	3.2%
	Diazepam	96	2.9%
	Tofisopam	84	2.5%
	Others	57	1.7%

Table 3: Patient characteristics

Characteristic		N	%
Family History of Depression	Yes	977	29.4%
	No	2344	70.6%
Risk Factors	Trauma/Negative life event	1755	52.8%
	Genetics	690	20.8%
	Health Condition	652	19.6%
	Age	494	14.9%
	Gender	442	13.3%
	Others	126	3.8%
Duration of Depression	0-1 years	1368	41.2%
	1-3 years	1498	45.1%
	3-5 years	363	10.9%
	>5 years	92	2.8%
Residence	Rural	1541	46.4%
	Urban	1780	53.6%
Marital Status	Unmarried	529	15.9%
	Married	2528	76.2%
	Widowed	157	4.7%
	Divorced/Separated	107	3.2%
Smoking	Yes	1356	40.8%
	No	1965	59.2%
Alcoholic	Yes	1280	38.5%
	No	2041	61.5%

of antidepressants, with a focus on escitalopram, vortioxetine, and sertraline, for treating MDD within the Indian population.

In our study, the prevalence rate of MDD was higher in males (64.8%) than in female (35.2%). Similar results were found in study done by Vishal et al, Mohamand et al and Ghosh et al, with the prevalence of depression in males being 55.63%, 51.8% and 54.67% and in females being 44.36%, 48.2%, and 45.33% respectively.[13-15]

In the present study, MDD prevalence was highest among individuals aged 30–60 years, followed by those <30 years, and lowest in those >60 years. A population-based Indian study by Arvind et al. similarly reported the highest prevalence in the 40–59-year age group, followed by those >65 years, and the lowest in individuals <30 years.[4] In contrast, global data generally indicate the peak burden in younger adults (<30 years), with prevalence declining with age. This variation may reflect sociocultural factors, delayed diagnosis in younger individuals, and greater utilization of healthcare services among midlife adults, along with increased psychosocial stressors and comorbidities during midlife.[16-17]

In our study, family history of depression was reported in 29.4% of patients. This aligns with recent genetic studies showing MDD heritability of about 30–59%, underscoring the genetic contribution and the relevance of family history in clinical populations.[18] Another recent meta-analysis of genetic polymorphism studies estimated heritability at about 37% (95% CI 31-42%) based on twin and family data.[19] Among risk factors, trauma or negative life events were most frequent (52.8%), consistent with Wang's review highlighting stressful life events such as abuse, bereavement, or divorce as significant triggers for depressive episodes.[20] Urban residency was slightly more common (53.6%), aligning with meta-analyses showing higher depression prevalence in urban areas due to psychosocial stressors.[21] Additionally, smoking (40.8%) and alcohol use (38.5%) were notable, which agrees with systematic reviews reporting strong associations between substance use and depression.[22] These findings underscore the multifactorial nature of MDD, influenced by genetic, environmental, and lifestyle factors, and are broadly consistent with patterns observed in Indian and global studies. Treatment duration in our study showed that 39.7% of patients were on antidepressants for 2–6 months, while 26.7% continued for 6–12

Table 4: A summary of the distribution of co-morbid conditions

Co-morbid conditions		N	%
Psychiatric Comorbidity	Anxiety	1960	59.0%
	Schizophrenia	369	11.1%
	Substance Abuse	336	10.1%
	Others	35	1.1%
	None	882	26.6%
Non-Psychiatric Comorbidity	Diabetes	760	22.9%
	Hypertension	655	19.7%
	Thyroid Disorder	344	10.4%
	Dyslipidemia	318	9.6%
	Asthma	239	7.2%
	Others	59	1.8%

months. This is consistent with Indian Council of Medical Research (ICMR) guidelines recommending continuation of therapy for 6–9 months after achieving remission to prevent relapse [23]

Among the prescriptions analysed, escitalopram (50.6%) emerged as the most prescribed drug for major depressive disorder (MDD), followed by vortioxetine (28.1%) and sertraline (21%). This finding aligns with previous Indian studies highlighting the dominance of selective serotonin reuptake inhibitors (SSRIs) in clinical practice. Kulkarni et al. reported that SSRIs constituted 32.18% of prescriptions, making them the most commonly prescribed class, followed by atypical antidepressants (0.64%). Similarly, a study from North India observed that SSRIs were the preferred class (61.26%), with escitalopram (58.4%) being the most frequently prescribed agent, followed by sertraline (7%).[13,24] The Indian Psychiatric Society (IPS) multicentric study further corroborates these findings, reporting that escitalopram accounted for 40% of antidepressant prescriptions, followed by sertraline (17.6%) and fluoxetine (16.3%). Overall, SSRIs represented 79.2% of all prescriptions, underscoring their role as first-line therapy in India.[25] Recent expert opinion surveys also reinforce the preference for escitalopram, with 87% of psychiatrists favouring it over other SSRIs due to its efficacy and tolerability profile. Approximately 64% of respondents reported significant improvement in

50–75% of patients treated with escitalopram, and nearly half indicated that remission typically occurs within 8 weeks.[26] Emerging evidence on vortioxetine suggests its growing role in Indian practice. A randomized comparative study demonstrated that vortioxetine is as effective as escitalopram in reducing depressive symptoms, with a potentially faster onset of action and a lower incidence of sexual dysfunction and weight gain, which may influence future prescribing trends.[27]

In our study, escitalopram was most often prescribed at 10 mg for both initiation and maintenance, consistent with guideline-recommended dosing. Vortioxetine was commonly started at 5 mg and maintained at 10 mg, while sertraline was initiated at 50 mg and maintained at 25 mg, possibly reflecting individualized titration for tolerability.[28-29] Similar dosing patterns have been reported in Indian and global literature, where SSRIs and vortioxetine are typically administered once daily due to favorable pharmacokinetics.

Regarding dosing frequency, 80% of patients in our cohort received once-daily administration, which is in line with the pharmacokinetic profiles of SSRIs and vortioxetine that allow for single daily dosing. Comparable findings have been reported in Indian studies, where once-daily dosing predominates due to improved adherence and convenience.[10,30]

In terms of therapy type, 55.2% of patients received monotherapy, while 44.8% were on combination therapy. Although monotherapy remains the first-line approach, combination therapy is increasingly used in clinical practice for partial responders or severe cases. Evidence from a meta-analysis and clinical study indicates that combination therapy can provide superior efficacy compared to monotherapy without significantly increasing dropout rates, particularly when combining agents with complementary mechanisms.[31,32]

In our study, the most common adverse effects were nausea (24.5%) and dizziness (13.9%). These findings are consistent with published evidence indicating that gastrointestinal symptoms, particularly nausea, are among the most frequently reported side effects of SSRIs and vortioxetine. Uher et al. (2009) reported that nausea is the most common early-onset adverse effect of antidepressants, especially SSRIs, often occurring within the

first weeks of treatment and typically resolving over time.[33] a cross-sectional study on SSRIs (sertraline, escitalopram, fluoxetine) found dizziness and light-headedness among the common adverse effects, particularly with escitalopram.[34] For vortioxetine, clinical trial data confirm that nausea is the most frequent adverse event, followed by dizziness and headache, with most events occurring within the first month of therapy.[35,36]

In our study, anxiety (59.0%) emerged as the most frequent psychiatric comorbidity in MDD, which is consistent with recent evidence showing that 50–75% of patients with MDD meet DSM-5 criteria for anxious depression, and this comorbidity is associated with poorer outcomes and delayed remission.[37] Among non-psychiatric comorbidities, diabetes (22.9%) was most common, aligning with recent meta-analyses confirming a bidirectional association between depression and type 2 diabetes.[38] Hypertension (19.7%) and dyslipidemia (9.6%) were also notable, reflecting the metabolic burden frequently observed in MDD and supported by recent studies linking depression with metabolic syndrome and cardiovascular risk factors.[39] Thyroid disorders (10.4%) in our cohort are comparable to recent findings showing a significant association between depression and thyroid dysfunction, particularly hypothyroidism and subclinical hypothyroidism.[40]

Conclusion

This study highlights current antidepressant prescribing trends in India, with escitalopram as the preferred agent and frequent co-prescription of clonazepam for managing comorbid anxiety. These findings reflect a common clinical practice of short-term benzodiazepine use alongside antidepressants for symptomatic relief, while underscoring the importance of individualized treatment strategies in MDD management.

Acknowledgment

Conflict of Interest

References

1. Cerbo AD. Letter to the Editor: CONVERGENCES AND DIVERGENCES IN THE ICD-11 VS. DSM-5 CLASSIFICATION OF MOOD DISORDERS. Turk Psikiyatri Derg. 2021;32(4):293–5.

2. Health (UK) NCC for M. The classification of depression and depression rating scales/questionnaires. In: Depression in Adults with a Chronic Physical Health Problem: Treatment and Management. British Psychological Society (UK); 2010.

3. Cui L, Li S, Wang S, Wu X, Liu Y, Yu W, et al. Major depressive disorder: hypothesis, mechanism, prevention and treatment. Sig Transduct Target Ther. 2024 Feb 9;9(1):30.

4. Arvind BA, Gururaj G, Loganathan S, Amudhan S, Varghese M, Benegal V, et al. Prevalence and socioeconomic impact of depressive disorders in India: multisite population-based cross-sectional study. BMJ Open. 2019 Jun 27;9(6): e027250.

5. Berk M, Köhler-Forsberg O, Turner M, Penninx BWJH, Wrobel A, Firth J, et al. Comorbidity between major depressive disorder and physical diseases: a comprehensive review of epidemiology, mechanisms and management. World Psychiatry. 2023 Oct;22(3):366–87.

6. Wolff J, Reißner P, Hefner G, Normann C, Kaier K, Binder H, et al. Pharmacotherapy, drug-drug interactions and potentially inappropriate medication in depressive disorders. PLoS One. 2021;16(7):e0255192.

7. Dołoto A, Bąk E, Batóg G, Piątkowska-Chmiel I, Herbet M. Interactions of antidepressants with concomitant medications-safety of complex therapies in multimorbidities. Pharmacol Rep. 2024 Aug;76(4):714–39.

8. Park LT, Zarate CA. Depression in the Primary Care Setting. N Engl J Med. 2019 Feb 7;380(6):559–68.

9. Sheffler ZM, Patel P, Abdijadid S. Antidepressants. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025. Available from: http://www.ncbi.nlm.nih.gov/books/NBK538182/

10. Pillai A, Keyes KM, Susser E. Antidepressant prescriptions and adherence in primary care in India: Insights from a cluster randomized control trial. PLOS ONE. 2021 Mar 19;16(3):e0248641.

11. Cebron Lipovec N, Anderlic A, Locatelli I. General antidepressants prescribing trends 2009-2018 in Slovenia: a cross-sectional retrospective database study. Int J Psychiatry Clin Pract. 2022 Nov;26(4):401–5.

12. Capili B, Anastasi JK. Pragmatic Clinical Trials and Real-World Evidence: An Introduction. Am J Nurs. 2025 Feb 1;125(2):56–8.

13. admin. PATTERN OF ANTIDEPRESSANT PRESCRIPTION IN PATIENTS SUFFERING FROM DEPRESSIVE DISORDER IN A TERTIARY CARE HOSPITAL | INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES AND RESEARCH. 2022 Feb 1;13(2):942-947.

14. Kulkarni VA, Baig MS, Deshpande P. Study of the prescription pattern of antidepressants in a tertiary care hospital: a prospective observational study. International Journal of Research in Medical Sciences. 2023 Mar 29;11(4):1251–6.

15. Dar MM, Tarfarosh SFA, Kullah SM, Mushtaq R, Manzoor M, Maqbool S. Socio-Demographic and Clinical Profile of Patients Suffering from Severe Depressive Disorders in Kashmir Valley. 2016;3(11).

16. Major Depression - National Institute of Mental Health (NIMH) [Internet]. [cited 2025 Sep 17]. Available from: https://www.nimh.nih.gov/health/statistics/major-depression

17. Brody DJ. Depression Prevalence in Adolescents and Adults: United States, August 2021–August 2023. 2025;(527).

18. Nguyen TD, Kowalec K, Pasman J, Larsson H, Lichtenstein P, Dalman C, et al. Genetic Contribution to the Heterogeneity of Major Depressive Disorder: Evidence From a Sibling-Based Design Using Swedish National Registers. AJP. 2023 Oct;180(10):714–22.

19. Suktas A, Ekalaksananan T, Aromseree S, Bumrungthai S, Songserm N, Pientong C. Genetic polymorphism involved in major depressive disorder: a systemic review and meta-analysis. BMC Psychiatry. 2024 Oct 22;24(1):716.

20. Wang SK, Feng M, Fang Y, Lv L, Sun GL, Yang SL, et al. psychological trauma, posttraumatic stress disorder and trauma-related depression: A mini-review. World J Psychiatry. 2023 Jun 19;13(6):331–9.

21. Peen J, Schoevers RA, Beekman AT, Dekker J. The current status of urban-rural differences in psychiatric disorders. Acta Psychiatr Scand. 2010 Feb;121(2):84–93.

22. Hu Z, Cui E, Chen B, Zhang M. Association between cigarette smoking and the risk of major psychiatric disorders: a systematic review and meta-analysis in depression, schizophrenia, and bipolar disorder. Front Med. 2025 Feb 13;12.

23. Depression [Internet]. Available from: https://www.icmr.gov.in/icmrobject/uploads/STWs/1725952332_psychiatry_depression.pdf

24. Sirajo I, Manaal A, Khan R, Aliyu A, Sada M, Ajmal M, et al. Drug utilization pattern of antidepressant used in depression disorder in psychiatric department in Northern India: a prospective observational study. International Journal of Basic & Clinical Pharmacology 2024 Aug 28;13(5):691–5.

25. Grover S, Avasth A, Kalita K, Dalal PK, Rao GP, Chadda RK, et al. IPS multicentric study: Antidepressant prescription patterns. Indian J Psychiatry. 2013;55(1):41–5.

26. Manjula S, Krishna Kumar M. Expert Opinion on the Prescription Practice of Selective Serotonin Reuptake Inhibitors for Major Depressive Disorder in Indian Settings. SAR J Psychiatry Neurosci, 5(1), 13-18

27. Aziz S, Reyazuddin M, Shaan F. Comparative efficacy and safety of vortioxetine versus escitalopram in major depressive disorder: A randomized, open-label, rater-blinded study. International Journal of Psychiatry Research 2025; 7(2): 15-18

28. Gautam S, Jain A, Gautam M, Vahia VN, Grover S. Clinical Practice Guidelines for the management of Depression. Indian J Psychiatry. 2017 Jan; 59(Suppl 1): S34–S50

29. Pharmacology antidepressants - UpToDate [Internet]. Available from: https://www.uptodate.com/contents/image?imageKey=PC/53818

30. Chen G, Højer AM, Areberg J, Nomikos G. Vortioxetine: Clinical Pharmacokinetics and Drug Interactions. Clin Pharmacokinet. 2018 Jun;57(6):673–86.

31. Trivedi JK, Dhyani M, Sareen H, Yadav VS, Rai SB. Antidepressant drug prescription pattern for depression at a tertiary health care center of Northern India. lobal Journal of Medicine and Medical Sciences.2019 April 6.

32. Henssler J, Alexander D, Schwarzer G, Bschor T, Baethge C. Combining Antidepressants vs Antidepressant Monotherapy for Treatment of Patients With Acute Depression: A Systematic Review and Meta-analysis. JAMA Psychiatry. 2022 Apr 1;79(4):300–12.

33. Uher R, Farmer A, Henigsberg N, Rietschel M, Mors O, Maier W, et al. Adverse reactions to antidepressants. Br J Psychiatry. 2009; 195, 202-210.

34. Anagha K, Shihabudheen P, Uvais NA. Side Effect Profiles of Selective Serotonin Reuptake Inhibitors: A Cross-Sectional Study in a Naturalistic Setting. Prim Care Companion CNS Disord. 2021 Jul 29;23(4) 20m02747.

35. Findling RL, DelBello MP, Zuddas A, Emslie GJ, Ettrup A, Petersen ML, et al. Vortioxetine for Major Depressive Disorder in Adolescents: 12-Week Randomized, Placebo-Controlled, Fluoxetine-Referenced, Fixed-Dose Study. J Am Acad Child Adolesc Psychiatry. 2022 Sep;61(9):1106-1118.e2.

36. Li L, Xu Q, Pang L, Liu Y, Lu Y. Comprehensive analysis of adverse events associated with vortioxetine using the FDA adverse event reporting system. Front Pharmacol. 2025 May 2 ;16.

37. Hopwood M. Anxiety Symptoms in Patients with Major Depressive Disorder: Commentary on Prevalence and

Clinical Implications. Neurol Ther. 2023 Apr 28;12(Suppl 1):5–12.

38. Fang T, Zhang Q, Wang Z, Liu JP. Bidirectional association between depression and diabetic nephropathy by meta-analysis. PLoS One. 2022 Dec 20;17(12):e0278489.

39. Tang Z, Zhen Y, Zhang L, Liu X, Ma J. Prevalence and factors associated with metabolic syndrome in first hospitalization for major depression disorder patients. Sci Rep. 2023 Sep 19;13(1):15496.

40. Ma Y, Wang M, Zhang Z. The association between depression and thyroid function. Front Endocrinol (Lausanne). 2024;15:1454744.

Machine Learning Applications in Schizophrenia: A Comprehensive Review

Jamuna Das*, **Jitendriya Biswal, Asish Asutosh Choudhury**

Department of Psychiatry, Siksha' O' Anusandhan, IMS and SUM Hospital, Bhubaneshwar, India.

Abstract

Schizophrenia is a severe and heterogeneous neuropsychiatric disorder characterised by complex symptoms, uncertain aetiology, and variable treatment outcomes. Traditional diagnostic methods relying on clinical observation and self-report often fail to capture the underlying biological diversity of the illness. Recent advances in machine learning (ML) have introduced powerful tools for analysing multimodal data and improving diagnostic precision, risk prediction, and treatment outcomes in schizophrenia. This comprehensive review summarises studies published between 2010 and 2025 that applied ML methods to schizophrenia across diverse data modalities, including neuroimaging, genomics, electronic health records, cognitive assessments, and digital phenotyping. Evidence shows that ML models, particularly deep learning and multimodal fusion techniques, can effectively distinguish schizophrenia from other psychiatric conditions, identify individuals at ultra-high risk for psychosis, predict treatment response, and uncover biologically meaningful subtypes. Despite these advances, major challenges remain, including small and imbalanced datasets, limited generalisability, model opacity, and ethical concerns related to privacy and bias. Addressing these limitations through large-scale, diverse datasets, explainable AI, and ethical frameworks will be essential for clinical translation. Integrating ML into psychiatric decision-support systems may enable earlier diagnosis, personalised treatment, and better long-term outcomes. With continued development and responsible implementation, ML holds the potential to transform schizophrenia care and advance the realisation of precision psychiatry.

INTRODUCTION

Schizophrenia is a chronic, severe, and heterogeneous mental disorder that profoundly affects thought, perception, emotion, and behaviour.[1] It remains one of the most disabling psychiatric conditions worldwide, with significant clinical, social, and economic implications. The disorder's complex aetiology, encompassing genetic, neurodevelopmental, neurobiological, and environmental factors, creates challenges in achieving early and accurate diagnosis, predicting disease trajectory, and optimising treatment [2]. Traditional psychiatric assessments, which rely on clinical interviews and observable symptoms, often lack the precision needed to detect subtle biological markers or forecast individual responses to treatment. [3]

Current psychiatric assessments rely heavily on clinical interviews and behavioural observations, which are often useful, and clinical judgement, but typically lack the precision required to capture subtle biological signatures or prognostic information for an individual. These complex and multifaceted diagnostic problems have prompted researchers and clinicians to seek to employ computational methods and machine learning (ML) methods and analytic systems in psychiatric research, diagnosis, and treatment. Machine learning, which is a branch of artificial intelligence, refers to algorithms that can find hidden structures within high-dimensional data and use them to make predictions or classifications without programming the algorithm explicitly.[4] In psychiatry, machine learning has been increasingly utilised on multimodal data sources such as neuroimaging (structural and functional MRI, EEG), genomics, transcriptomics, electronic health records, and digital phenotyping data obtained from smartphones and wearables. Support vector machines (SVMs), random forests, logistic regression, ensemble learning, and deep learning architectures (e.g., convolutional neural networks and recurrent neural networks) have all demonstrated an ability to discriminate patients with schizophrenia from healthy controls, identify individuals at ultra-high risk for developing a psychotic disorder, and forecast their trajectory of treatment response or relapse.[5-9]

In addition to classification, unsupervised methods such as clustering and dimensionality reduction (including principal component analysis, t-SNE, and autoencoders) have been used to identify subtypes within schizophrenia and schizophrenia-related syndromes, adding evidence to suggest that psychiatric disorders occur on a continuous distribution as opposed to discrete categories[10,11]. Finally, supervised methods have also used natural language processing (NLP) to analyse (early) clinical notes and transcripts of speech and interactions on social media data to identify linguistically based early indications of psychosis[12-14]. Despite the advances in ML, there are important limitations to utilisation within psychiatry; small sample sizes or imbalanced datasets, high risk of overfitting, difficulty with complex and opaque inferences from the

model, and limited generalizability are all important considerations with ML approaches in psychiatry. Furthermore, ethical considerations, including data privacy, algorithm-proof bias, and clinical account-ability (i.e., clinical decisions based on ML), are points of consideration prior to implementation of ML into clinical practice[15-18].

ML refers to an assortment of algorithms that can identify underlying structures within high-dimensional data and make predictions or classify data without specific programming. In the field of psychiatry, ML has been employed increasingly with multimodal data sources, supporting analysis of complex data that may come from neuroimaging, genomics, clinical records, or digital phenotyping. [5,19-21] In general, ML approaches can be classified into three groups: supervised learning methods (e.g., support vector machines (SVMs), logistic regression, and random forests), unsupervised methods (e.g., clustering, principal component analysis (PCA), and autoencoders), and deep learning techniques (e.g., convolutional neural networks (CNNs), recurrent neural networks (RNNs), and transformers). Ensemble learning options (i.e., boosting and bagging) can help with prediction and improve performance by using multiple models.[22-32] A common workflow for all methods follows a similar pattern of data gathering, pre-processing, feature extraction, training and fine-tuning the model, and validating and evaluating the model. This framework is represented in most applications currently being conducted for psychiatric prediction.[15,33]

In recent years, advances in computational psychiatry and artificial intelligence (AI) have introduced new opportunities for understanding and managing schizophrenia. Among these, machine learning (ML)—a subset of AI that enables algorithms to identify patterns and make predictions from high-dimensional data without explicit programming—has shown substantial promise.[2,34,35] ML techniques have been applied across a range of data modalities, including neuroimaging, genomics, transcriptomics, electronic health records, cognitive assessments, and digital phenotyping from smartphones and wearable devices. These approaches have demonstrated the potential to differentiate patients with schizophrenia from healthy controls,

identify individuals at high risk for psychosis, predict treatment response, and uncover biologically distinct subtypes.[2]

Despite encouraging results, several methodological and ethical challenges limit the clinical translation of ML in schizophrenia. These include small and imbalanced datasets, variability in data acquisition, limited model interpretability, and concerns regarding privacy, bias, and accountability. Addressing these challenges is critical for the responsible implementation of ML-driven tools in psychiatry. This review provides a comprehensive synthesis of current ML applications specifically in schizophrenia, summarising methodological advances, key findings, and ongoing challenges. It also highlights future directions aimed at developing interpretable, equitable, and clinically useful models that could transform schizophrenia care through precision psychiatry.

Methods

This review was conducted to summarise and critically evaluate current applications of ML in the prediction, diagnosis, and treatment of schizophrenia. A comprehensive literature search was performed in PubMed, Scopus, and Google Scholar for studies published between January 2010 and September 2025. The search terms included combinations of *"machine learning," "artificial intelligence," "deep learning," "neuroimaging," "genomics," "electronic health records," "digital phenotyping,"* and *"schizophrenia."*

Original research articles, systematic reviews, and meta-analyses focusing specifically on ML applications in schizophrenia were included. Studies exclusively addressing other psychiatric disorders were excluded unless they provided comparative insights relevant to schizophrenia. References of key publications were manually screened to identify additional eligible studies.

Extracted information included the ML technique used (e.g., support vector machines, random forests, convolutional neural networks), data modality (e.g., MRI, EEG, genetic, clinical, or behavioural data), study objectives, sample size, and key outcomes. Findings were organised thematically into major application areas—early risk identification, diagnostic classifi-

cation, treatment response prediction, and clinical decision-support integration.

The review was structured following a narrative synthesis approach, highlighting methodological trends, limitations, ethical considerations, and emerging directions toward precision psychiatry in schizophrenia.

RESULT

Machine Learning Applications in Schizophrenia

ML has been increasingly applied in schizophrenia research to improve prediction, diagnosis, and treatment personalisation. Across multiple data modalities, neuroimaging, genomics, clinical records, and digital phenotyping, ML methods have demonstrated promising performance in identifying biomarkers and forecasting clinical outcomes.

Machine learning has been increasingly applied across different stages of psychiatric care, from early risk identification to treatment optimisation. These applications highlight the potential of ML to bridge the gap between research and clinical practice, enabling more precise and personalised care. Below are the key domains where ML has shown substantial promise.[36,37] One of the most critical applications of ML in psychiatry is the early identification of individuals at risk of developing schizophrenia or other psychotic disorders.[37,38]

Several studies have used ML algorithms such as support vector machines (SVMs), random forests, and deep learning networks to identify individuals at ultra-high risk (UHR) for psychosis. Models trained on structural and functional MRI data have predicted transition to schizophrenia with greater accuracy than traditional clinical assessments.[39]

Natural language processing (NLP) applied to speech and text data has detected early signs of thought disorder years before onset. Similarly, smartphone-based monitoring and digital phenotyping have revealed behavioural and social activity patterns indicative of early psychosis risk. Digital phenotyping: Smartphone-based monitoring of activity, mobility, and communication patterns has been used to identify behavioural shifts signalling

early psychiatric deterioration.[40] By identifying at-risk individuals earlier, ML can support preventive interventions and reduce the duration of untreated psychosis, which is strongly linked to better long-term outcomes.

ML methods have also been applied to improve the accuracy of psychiatric diagnosis, which traditionally relies on subjective clinical evaluation. Subtyping within schizophrenia: Clustering and dimensionality reduction approaches have uncovered biologically distinct subgroups, such as patients with predominant cognitive impairment versus those with affective symptoms. Multimodal classification: Combining imaging, genomic, and clinical data has yielded robust classifiers capable of outperforming single-modality approaches.[41,42]

Improved classification supports the movement toward data-driven diagnostic frameworks and may inform future revisions of psychiatric nosology beyond the DSM/ICD categories.[43]

Predicting treatment outcomes remains one of the most challenging areas in psychiatry, where trial-and-error prescribing is common. ML has been applied to: Medication response: Models trained on clinical and genomic data have predicted which patients are likely to respond to specific antipsychotic medications (e.g., risperidone, clozapine), paving the way for personalised pharmacotherapy. [44–46]

Algorithms using genetic and metabolic profiles have estimated the risk of adverse effects, such as weight gain or extrapyramidal symptoms.[47]

Prognosis and relapse prediction: Longitudinal ML models leveraging EHRs, wearable sensor data, and social behaviour features have forecasted relapse events, hospital readmissions, and long-term functional outcomes.[48] These predictive tools can help clinicians personalise treatment plans, reduce unnecessary medication trials, and anticipate relapses before they occur. Beyond prediction, ML has contributed to understanding the biological mechanisms underlying schizophrenia and related disorders.[49]

Graph-theory-based ML approaches have revealed disrupted brain networks and altered connectivity hubs in schizophrenia, pointing toward dysconnectivity as a central feature of the disorder.

[50,51] ML models applied to polygenic risk scores and gene expression data have identified pathways involved in synaptic signalling, immune function, and neurodevelopment as key contributors. Endophenotype discovery by clustering patients on the basis of cognitive, imaging, and genetic data, ML has uncovered intermediate phenotypes that may better explain heterogeneity in clinical presentation. [52,53]

These insights contribute to the shift from a purely symptom-based understanding of schizophrenia to a mechanistic, biology-informed perspective, which is essential for developing targeted therapies.[54]

Increasingly, ML applications are being integrated into comprehensive clinical decision-support systems (CDSS). These systems combine risk prediction, diagnostic classification, treatment response forecasting, and relapse monitoring into unified platforms. While still in early stages, pilot studies suggest that such systems may significantly aid psychiatrists in tailoring interventions, improving efficiency, and enhancing patient engagement.[55]

The effectiveness of machine learning to foresee and to classify schizophrenia and other mental disorders, to a great extent, relies upon the nature and the quality of the input data. Every modality provides one-of-a-kind clues, and nowadays a combination of modalities is being used not only for greater accuracy but also for wider understanding. [56-58]

Neuroimaging is still considered to be the most preferred data source in Machine Learning (ML) applications in psychiatry.[17,19] Some of the techniques used for this purpose are structural magnetic resonance imaging (sMRI), functional MRI (fMRI), diffusion tensor imaging (DTI), and electroencephalography (EEG), which provide biomarkers of brain structure, connectivity, and activity.[59-65] Through sMRI, several changes have been reported, such as cortical thinning, reduction of gray matter, and volumetric abnormalities associated with schizophrenia. [66,67] ML models like support vector machines (SVMs) and random forests have been utilised to perform the classification of patients vs. healthy controls by using these structural features as a basis. fMRI measures resting-state and task-related functional connectivity patterns. Therefore, models

such as convolutional neural networks (CNNs) and graph-based learning methods can support the identification of disrupted brain networks that are potentially linked to psychosis. DTI has been utilised to examine white matter integrity, while ML algorithms have pointed out the microstructural changes in tracts like the corpus callosum. EEG/MEG offers high temporal resolution, and the use of recurrent neural networks (RNNs) and temporal pattern recognition techniques has made it possible to identify abnormal oscillatory activity that predicts the occurrence of schizophrenia.[66-70]

Genomic data provide another powerful perspective, shedding light on the heritability and biological roots of psychiatric disorders. Genome-wide association studies (GWAS)[71] have identified numerous single-nucleotide polymorphisms (SNPs) associated with schizophrenia risk. ML approaches such as regularised regression models (LASSO, elastic net) and ensemble methods have been used to construct polygenic risk scores that improve risk prediction. Transcriptomic and epigenetic data have also been analysed with deep learning models like autoencoders, uncovering genetic signatures tied to psychiatric outcomes. Integrating genomic data with imaging and clinical features is an emerging and promising direction.[72–76]

Electronic health records (EHRs) offer rich, longitudinal clinical information, including diagnoses, treatments, and hospital admissions. Natural language processing has been applied to unstructured notes, allowing the early identification of psychosis risk factors. ML models trained on EHR data have also shown promise in predicting hospital readmissions, treatment adherence, and relapse risk. [74,77–79]

Behavioural and cognitive assessments, such as psychometric tests and structured interviews, further support classification tasks. ML models built on these inputs have successfully distinguished schizophrenia disorders.[80]

Digital phenotyping, enabled by smartphones and wearable devices, has emerged as a novel and rapidly growing data source. Passive measures like GPS tracking, call/text activity, and screen time can reveal patterns of social withdrawal, cognitive impairment, or circadian rhythm disruptions, which

are common in schizophrenia.[81] Additionally, speech and language analysis through NLP and acoustic methods has proven effective in detecting early markers of thought disorder and predicting psychosis onset. Social media data, including posting behaviour and language use, have also been studied, though concerns about privacy remain significant.[82]

Because schizophrenia is highly heterogeneous, relying on a single data source often proves insufficient.[81,83] Recent research highlights the benefits of multimodal approaches, which combine neuroimaging, genomic, clinical, and digital data. Methods such as multimodal deep learning, feature fusion, and ensemble modelling consistently outperform single-modality approaches. For example, combining fMRI connectivity data with genomic risk scores has improved classification accuracy, while integrating EHR and digital phenotyping has enhanced relapse prediction.[81–86]

Discussion

While machine learning has shown significant promise in predicting schizophrenia, several methodological and practical challenges limit its clinical translation. These challenges span issues related to data, model development, interpretability, reproducibility, and real-world deployment.[87]

Many datasets have skewed class distributions (e.g., far fewer individuals who transition from high-risk to schizophrenia compared to those who do not). Imbalanced data can bias models toward the majority class, leading to poor sensitivity for clinically critical outcomes like relapse or psychosis onset.[88]

Schizophrenia is inherently heterogeneous, and data collection protocols vary widely across studies, scanners, and clinical settings. Lack of standardisation complicates model training and reduces generalizability across populations. Missing data, noise in electronic health records, motion artefacts in neuroimaging, and incomplete genomic datasets are common. ML models trained on noisy data risk producing unreliable outputs.[88,89]

Overly complex models (e.g., deep neural networks with many parameters) may fit the idiosyncrasies of a training dataset rather than true underlying

patterns. Without rigorous external validation, such models often fail when applied to new populations. [90]

Unlike in computer vision or natural language processing, psychiatry lacks large benchmark datasets and standardised evaluation frameworks. This makes it difficult to compare models across studies and slows scientific progress. Many studies report high accuracy on internal datasets but fail to replicate results in independent cohorts. Variability in pre-processing pipelines, feature extraction methods, and performance metrics contributes to this problem. Deep learning models often achieve high accuracy but provide little insight into how predictions are made. In psychiatry, where clinical decisions can have profound consequences, the lack of transparency is a major barrier to adoption.[91,92]

Many ML studies in psychiatry are based on data from Western, educated, industrialised, rich, and democratic (WEIRD) populations. Models trained in such contexts may not generalise to diverse cultural or socioeconomic settings, raising concerns about bias and inequity. Variability in scanner hardware, imaging protocols, and clinical diagnostic practices introduces systematic differences that can degrade model performance when applied across sites. [91,93,94]

Psychiatric data is highly sensitive. ML applications relying on digital phenotyping, EHRs, or social media raise concerns about privacy breaches and informed consent. Biased training data can lead to unfair predictions, for instance, overdiagnosing or underdiagnosing certain demographic groups. This exacerbates existing disparities in mental health care. Determining responsibility for decisions made with ML support remains unclear, whether the clinician, institution, or algorithm developer should be accountable for adverse outcomes. Few ML-based psychiatric tools have passed through rigorous regulatory approval processes (e.g., FDA, EMA), limiting their availability in clinical practice.[49,88,90–92,95]

Ethical, Legal, and Social Considerations

The integration of machine learning into psychiatry introduces not only technical and methodological challenges but also a range of ethical, legal, and

social issues. Because psychiatric data is deeply personal and stigmatised, these challenges must be carefully addressed to ensure that the benefits of ML are realised without compromising patient rights, equity, or trust in mental health systems.[49,93,95]

Unlike many other medical domains, psychiatric data often contains subjective accounts of thoughts, emotions, and behaviours, which are particularly vulnerable to misuse. For example, digital phenotyping through smartphone monitoring captures intimate behavioural patterns such as sleep cycles, mobility, and social interactions.[52,93]

Storing and processing psychiatric data, especially when linked across modalities (EHRs, neuroimaging, genomics, and digital behaviour), raises the risk of breaches that could expose sensitive information. Strong encryption, federated learning (where data never leaves the local institution), and anonymisation are necessary to ensure secure handling of psychiatric data.[92,93]

Many ML applications rely on continuous data collection from smartphones or wearables. Patients may not fully understand the scope of data being collected or how it will be used. Continuous digital tracking raises questions about whether patients truly have the option to opt out without feeling coerced, particularly in clinical or institutional settings. Emerging frameworks propose allowing patients to update or withdraw consent in real time, thereby preserving autonomy and trust.[55,88,95]

Model Interpretability and Clinical Trust

Deep learning models, though powerful, often function as "black boxes," offering little explanation for their predictions. Clinicians require interpretable models that highlight key features—such as specific brain regions or genetic markers—to build confidence in their use. Techniques like SHAP values, attention mechanisms, and explainable AI are being explored but remain underused in psychiatric research.[96]

When ML models are used to inform diagnosis or treatment, questions arise about who is accountable for errors: the clinician, the healthcare institution, or the algorithm developers. Most argue that ML should be a decision-support tool rather than an

autonomous decision-maker in psychiatry. However, even in supportive roles, reliance on ML outputs could shift responsibility in ambiguous ways.[96,97]

Regulatory agencies such as the U.S. Food and Drug Administration (FDA) and European Medicines Agency (EMA) have begun developing frameworks for AI/ML in healthcare. However, few psychiatric applications have yet undergone formal regulatory approval.[98]

Predictive tools that label individuals as "high risk" for schizophrenia could unintentionally increase stigma or discrimination in employment, insurance, and education. Access to ML-driven tools (e.g., mobile health apps, wearable sensors) may be limited in low-resource settings, potentially widening existing inequities in mental health care.[97]

Transparency, interpretability, and clear communication of model limitations are essential to build trust among patients, clinicians, and the public. Without trust, adoption of ML in psychiatry will remain limited.[98]

Future Directions and Opportunities

Progress in large-scale, multi-site collaborations such as ENIGMA and PsychENCODE will support the development of more representative datasets. Integrating multimodal data—neuroimaging, genetics, EHR, and digital behaviour—using advanced fusion models may enhance accuracy and reveal new schizophrenia subtypes. The next generation of ML research must prioritise fairness-aware algorithms, explainability, and transparency to facilitate regulatory approval and clinician adoption. Embedding ML tools within clinical decision-support systems could help realise the promise of precision psychiatry, offering earlier diagnosis, individualised treatment, and improved patient outcomes.

Conclusion

Schizophrenia remains one of the most complex and disabling psychiatric disorders, with challenges in early detection, diagnosis, and treatment prediction. Machine learning (ML) has emerged as a transformative approach capable of analysing high-dimensional, multimodal data to reveal hidden biological and behavioural patterns underlying the illness. Evidence from neuroimaging, genomic, and digital phenotyping studies demonstrates that ML can enhance diagnostic precision, predict treatment outcomes, and support the development of personalised interventions. However, the field still faces critical barriers, including small and heterogeneous datasets, limited model interpretability, and ethical concerns related to privacy and bias. Future research must prioritise large, diverse, and longitudinal datasets alongside explainable and ethically responsible AI frameworks. With continued innovation and clinical integration, ML holds significant promise for advancing precision psychiatry and improving long-term outcomes for individuals with schizophrenia.

References

1. Patel KR, Cherian J, Gohil K, Atkinson D. Schizophrenia: Overview and Treatment Options. Pharmacy and Therapeutics 2014;39:638.
2. Luvsannyam E, Jain MS, Pormento MKL, Siddiqui H, Balagtas ARA, Emuze BO, et al. Neurobiology of Schizophrenia: A Comprehensive Review. Cureus 2022;14:e23959. https://doi.org/10.7759/CUREUS.23959.
3. Insel TR, Cuthbert BN. Brain disorders? Precisely: Precision medicine comes to psychiatry. Science (1979) 2015;348:499–500. https://doi.org/10.1126/SCIENCE.AAB2358.
4. Chen ZS, Kulkarni P (Param), Galatzer-Levy IR, Bigio B, Nasca C, Zhang Y. Modern views of machine learning for precision psychiatry. Patterns 2022;3:100602. https://doi.org/10.1016/J.PATTER.2022.100602.
5. Reynolds T, Johnson EC, Huggett SB, Bubier JA, Palmer RHC, Agrawal A, et al. Interpretation of psychiatric genome-wide association studies with multispecies heterogeneous functional genomic data integration. Neuropsychopharmacology 2021;46:86–97. https://doi.org/10.1038/S41386-020-00795-5.
6. Lydon-Staley DM, Cornblath EJ, Blevins AS, Bassett DS. Modeling brain, symptom, and behavior in the winds of change. Neuropsychopharmacology 2021;46:20–32. https://doi.org/10.1038/S41386-020-00805-6.
7. von Ziegler L, Sturman O, Bohacek J. Big behavior: challenges and opportunities in a new era of deep behavior profiling. Neuropsychopharmacology 2021;46:33–44. https://doi.org/10.1038/S41386-020-0751-7.
8. Onnela JP. Opportunities and challenges in the collection and analysis of digital phenotyping data. Neuropsychopharmacology 2021;46:45–54. https://doi.org/10.1038/S41386-020-0771-3.

9. Ressler KJ, Williams LM. Big data in psychiatry: multiomics, neuroimaging, computational modeling, and digital phenotyping. Neuropsychopharmacology 2020;46:1. https://doi.org/10.1038/S41386-020-00862-X.

10. Feczko E, Miranda-Dominguez O, Marr M, Graham AM, Nigg JT, Fair DA. The Heterogeneity problem: Approaches to identify psychiatric subtypes. Trends Cogn Sci 2019;23:584. https://doi.org/10.1016/J.TICS.2019.03.009.

11. Gao CX, Dwyer D, Zhu Y, Smith CL, Du L, Filia KM, et al. An overview of clustering methods with guidelines for application in mental health research. Psychiatry Res 2023;327:115265. https://doi.org/10.1016/J.PSYCHRES.2023.115265.

12. Narendra Kumar Rao B, Hemanth Raga Sai P, Naseeba B, Venkata Phani Karthik B, Madhavi G. Multilingual Text Identification Using NLP and Machine Learning 2024:29–36. https://doi.org/10.1007/978-981-99-2832-3_5.

13. Mangla P, Singh G, Pathak N, Chawla S. Language Identification Using Multinomial Naive Bayes Technique. Lecture Notes in Networks and Systems 2024;786:307–16. https://doi.org/10.1007/978-981-99-6547-2_24.

14. Thu YK, Aung T, Supnithi T. Neural Sequence Labeling Based Sentence Segmentation for Myanmar Language. Lecture Notes in Networks and Systems 2023;734 LNNS:285–96. https://doi.org/10.1007/978-3-031-36886-8_24.

15. Jordan MI, Mitchell TM. Machine learning: Trends, perspectives, and prospects. Science (1979) 2015;349:255–60. https://doi.org/10.1126/SCIENCE.AAA8415.

16. Walter M, Alizadeh S, Jamalabadi H, Lueken U, Dannlowski U, Walter H, et al. Translational machine learning for psychiatric neuroimaging. Prog Neuropsychopharmacol Biol Psychiatry 2019;91:113–21. https://doi.org/10.1016/J.PNPBP.2018.09.014.

17. Sui J, Jiang R, Bustillo J, Calhoun V. Neuroimaging-based Individualized Prediction of Cognition and Behavior for Mental Disorders and Health: Methods and Promises. Biol Psychiatry 2020;88:818–28. https://doi.org/10.1016/J.BIOPSYCH.2020.02.016.

18. Eyre HA, Singh AB, Reynolds C. Tech giants enter mental health. World Psychiatry 2016;15:21–2. https://doi.org/10.1002/WPS.20297.

19. Rutherford S. The Promise of Machine Learning for Psychiatry. Biol Psychiatry 2020;88:e53–5. https://doi.org/10.1016/J.BIOPSYCH.2020.08.024.

20. Chen ZS, Kulkarni P (Param), Galatzer-Levy IR, Bigio B, Nasca C, Zhang Y. Modern views of machine learning for precision psychiatry. Patterns 2022;3:100602. https://doi.org/10.1016/J.PATTER.2022.100602.

21. Li G, Han D, Wang C, Hu W, Calhoun VD, Wang YP. Application of deep canonically correlated sparse autoencoder for the classification of schizophrenia. Comput Methods Programs Biomed 2020;183. https://doi.org/10.1016/J.CMPB.2019.105073.

22. Aslan MF, Unlersen MF, Sabanci K, Durdu A. CNN-based transfer learning–BiLSTM network: A novel approach for COVID-19 infection detection. Appl Soft Comput 2021;98. https://doi.org/10.1016/J.ASOC.2020.106912.

23. Zhang X, Yao L, Wang X, Monaghan J, Mcalpine D, Zhang Y. A survey on deep learning-based non-invasive brain signals: recent advances and new frontiers. J Neural Eng 2021;18. https://doi.org/10.1088/1741-2552/abc902.

24. Al-Qatf M, Lasheng Y, Al-Habib M, Al-Sabahi K. Deep Learning Approach Combining Sparse Autoencoder with SVM for Network Intrusion Detection. IEEE Access 2018;6:52843–56. https://doi.org/10.1109/ACCESS.2018.2869577.

25. Zhang X, Yao L, Huang C, Wang S, Tan M, Long G, et al. Multi-modality sensor data classification with selective attention. IJCAI International Joint Conference on Artificial Intelligence 2018;2018-July:3111–7. https://doi.org/10.24963/ijcai.2018/432.

26. Cho K, Van Merriënboer B, Gulcehre C, Bahdanau D, Bougares F, Schwenk H, et al. Learning phrase representations using RNN encoder-decoder for statistical machine translation. EMNLP 2014 - 2014 Conference on Empirical Methods in Natural Language Processing, Proceedings of the Conference 2014:1724–34. https://doi.org/10.3115/v1/d14-1179.

27. Minaee S, Azimi E, Abdolrashidi A. Deep-Sentiment: Sentiment Analysis Using Ensemble of CNN and Bi-LSTM Models 2019.

28. Shankar D, Narumanchi S, Ananya HA, Kompalli P, Chaudhury K. Deep Learning based Large Scale Visual Recommendation and Search for E-Commerce 2017.

29. Kingma DP, Welling M. Auto-encoding variational bayes. 2nd International Conference on Learning Representations, ICLR 2014 - Conference Track Proceedings 2014. https://doi.org/10.61603/ceas.v2i1.33.

30. Kingma DP, Welling M. An introduction to variational autoencoders. Foundations and Trends in Machine Learning 2019;12:307–92. https://doi.org/10.1561/2200000056.

31. Sarker IH. Deep Learning: A Comprehensive Overview on Techniques, Taxonomy, Applications and Research Directions. SN Comput Sci 2021;2:420. https://doi.org/10.1007/S42979-021-00815-1.

32. Pugliese R, Regondi S, Marini R. Machine learning-based approach: global trends, research directions, and regulatory standpoints. Data Science and Management 2021;4:19–29. https://doi.org/10.1016/J.DSM.2021.12.002.

33. Sarker IH. Machine Learning: Algorithms, Real-World Applications and Research Directions. SN Comput Sci 2021;2:160. https://doi.org/10.1007/S42979-021-00592-X.

34. Gashkarimov VR, Sultanova RI, Efremov IS, Asadullin A. Machine learning techniques in diagnostics and prediction of the clinical features of schizophrenia: a narrative review. Consortium Psychiatricum 2023;4:43. https://doi.org/10.17816/CP11030.

35. Chen Z, Hu B, Liu X, Becker B, Eickhoff SB, Miao K, et al. Sampling inequalities affect generalization of neu-

roimaging-based diagnostic classifiers in psychiatry. BMC Med 2023;21:241. https://doi.org/10.1186/S12916-023-02941-4.

36. Chen ZS, Kulkarni P (Param), Galatzer-Levy IR, Bigio B, Nasca C, Zhang Y. Modern views of machine learning for precision psychiatry. Patterns 2022;3:100602. https://doi.org/10.1016/J.PATTER.2022.100602.

37. Ebrahimzadeh E, Saharkhiz S, Rajabion L, Oskouei HB, Seraji M, Fayaz F, et al. Simultaneous electroencephalography-functional magnetic resonance imaging for assessment of human brain function. Front Syst Neurosci 2022;16. https://doi.org/10.3389/FNSYS.2022.934266.

38. Bringas-Vega ML, Michel CM, Saxena S, White T, Valdes-Sosa PA. Neuroimaging and global health. Neuroimage 2022;260. https://doi.org/10.1016/J.NEUROIMAGE.2022.119458.

39. Morita T, Asada M, Naito E. Contribution of neuroimaging studies to understanding development of human cognitive brain functions. Front Hum Neurosci 2016;10. https://doi.org/10.3389/FNHUM.2016.00464.

40. Yen C, Lin CL, Chiang MC. Exploring the Frontiers of Neuroimaging: A Review of Recent Advances in Understanding Brain Functioning and Disorders. Life 2023;13:1472. https://doi.org/10.3390/LIFE13071472.

41. Sepede G, De Berardis D, Campanella D, Perrucci MG, Ferretti A, Salerno RM, et al. Neural correlates of negative emotion processing in bipolar disorder. Prog Neuropsychopharmacol Biol Psychiatry 2015;60:1–10. https://doi.org/10.1016/J.PNPBP.2015.01.016.

42. Yen C, Lin CL, Chiang MC. Exploring the Frontiers of Neuroimaging: A Review of Recent Advances in Understanding Brain Functioning and Disorders. Life 2023;13:1472. https://doi.org/10.3390/LIFE13071472.

43. Oh SL, Vicnesh J, Ciaccio EJ, Yuvaraj R, Acharya UR. Deep convolutional neural network model for automated diagnosis of schizophrenia using EEG signals. Applied Sciences (Switzerland) 2019;9. https://doi.org/10.3390/APP9142870.

44. Vita A, De Peri L, Deste G, Sacchetti E. Progressive loss of cortical gray matter in schizophrenia: a meta-analysis and meta-regression of longitudinal MRI studies. Transl Psychiatry 2012;2:e190. https://doi.org/10.1038/TP.2012.116.

45. Zhang Y, Catts VS, Sheedy D, McCrossin T, Kril JJ, Shannon Weickert C. Cortical grey matter volume reduction in people with schizophrenia is associated with neuro-inflammation. Transl Psychiatry 2016;6:e982. https://doi.org/10.1038/TP.2016.238.

46. Aksoy G, Cattan G, Chakraborty S, Karabatak M. Quantum Machine-Based Decision Support System for the Detection of Schizophrenia from EEG Records. J Med Syst 2024;48. https://doi.org/10.1007/s10916-024-02048-0.

47. Zhao Z, Deng Y, Zhang Y, Zhang Y, Zhang X, Shao L. DeepFHR: Intelligent prediction of fetal Acidemia using fetal heart rate signals based on convolutional neural network. BMC Med Inform Decis Mak 2019;19. https://doi.org/10.1186/s12911-019-1007-5.

48. Mortier S, Turkeš R, De Winne J, Van Ransbeeck W, Botteldooren D, Devos P, et al. Classification of Targets and Distractors in an Audiovisual Attention Task Based on Electroencephalography. Sensors 2023;23. https://doi.org/10.3390/s23239588.

49. Bergen SE, Petryshen TL. Genome-wide association studies (GWAS) of schizophrenia: does bigger lead to better results? Curr Opin Psychiatry 2012;25:76. https://doi.org/10.1097/YCO.0B013E32835035DD.

50. Bracher-Smith M, Rees E, Menzies G, Walters JTR, O'Donovan MC, Owen MJ, et al. Machine learning for prediction of schizophrenia using genetic and demographic factors in the UK biobank. Schizophr Res 2022;246:156. https://doi.org/10.1016/J.SCHRES.2022.06.006.

51. Coors A, Imtiaz MA, Boenniger MM, Aziz NA, Breteler MMB, Ettinger U. Polygenic risk scores for schizophrenia are associated with oculomotor endophenotypes. Psychol Med 2021;53:1611. https://doi.org/10.1017/S0033291721003251.

52. Yang J, Benyamin B, McEvoy BP, Gordon S, Henders AK, Nyholt DR, et al. Common SNPs explain a large proportion of the heritability for human height. Nat Genet 2010;42:565–9. https://doi.org/10.1038/NG.608.

53. Zhao Z, Dorn S, Wu Y, Yang X, Jin J, Lu Q. One score to rule them all: regularized ensemble polygenic risk prediction with GWAS summary statistics. BioRxiv 2024:2024.11.27.625748. https://doi.org/10.1101/2024.11.27.625748.

54. Fong WJ, Tan HM, Garg R, Teh AL, Pan H, Gupta V, et al. Comparing feature selection and machine learning approaches for predicting CYP2D6 methylation from genetic variation. Front Neuroinform 2023;17:1244336. https://doi.org/10.3389/FNINF.2023.1244336/FULL.

55. Swinckels L, Bennis FC, Ziesemer KA, Scheerman JFM, Bijwaard H, de Keijzer A, et al. The Use of Deep Learning and Machine Learning on Longitudinal Electronic Health Records for the Early Detection and Prevention of Diseases: Scoping Review. J Med Internet Res 2024;26. https://doi.org/10.2196/48320.

56. hua yining, Blackley S, Shinn A, Skinner J, Moran L, Zhou L. Identifying Psychosis Episodes in Psychiatric Admission Notes via Rule-based Methods, Machine Learning, and Pre-Trained Language Models. Res Sq 2024:rs.3.rs-4126574. https://doi.org/10.21203/RS.3.RS-4126574/V1.

57. Garriga R, Buda TS, Guerreiro J, Omaña Iglesias J, Estella Aguerri I, Matić A. Combining clinical notes with structured electronic health records enhances the prediction of mental health crises. Cell Rep Med 2023;4:101260. https://doi.org/10.1016/J.XCRM.2023.101260.

58. Gashkarimov VR, Sultanova RI, Efremov IS, Asadullin A. Machine learning techniques in diagnostics and prediction of the clinical features of schizophrenia: a narrative review. Consortium Psychiatricum 2023;4:43. https://doi.org/10.17816/CP11030.

59. Chivilgina O, Elger BS, Jotterand F. Digital Technologies for Schizophrenia Management: A Descriptive Review. Sci Eng Ethics 2021;27. https://doi.org/10.1007/S11948-021-00302-Z.

60. Taipale H, Schneider-Thoma J, Pinzón-Espinosa J, Radua J, Efthimiou O, Vinkers CH, et al. Representation and Outcomes of Individuals with Schizophrenia Seen in Everyday Practice Who Are Ineligible for Randomized Clinical Trials. JAMA Psychiatry 2022;79:210–8. https://doi.org/10.1001/JAMAPSYCHIATRY.2021.3990.

61. Birk RH, Samuel G. Digital Phenotyping for Mental Health: Reviewing the Challenges of Using Data to Monitor and Predict Mental Health Problems. Curr Psychiatry Rep 2022;24:523–8. https://doi.org/10.1007/S11920-022-01358-9.

62. Chukka A, Choudhary S, Dutt S, Bondre A, Reddy P, Tugnawat D, et al. Digital Interventions for Relapse Prevention, Illness Self-Management, and Health Promotion In Schizophrenia: Recent Advances, Continued Challenges, and Future Opportunities. Curr Treat Options Psychiatry 2023;10:346–71. https://doi.org/10.1007/S40501-023-00309-2.

63. Lane E, D'Arcey J, Kidd S, Onyeaka H, Alon N, Joshi D, et al. Digital Phenotyping in Adults with Schizophrenia: A Narrative Review. Curr Psychiatry Rep 2023;25:699–706. https://doi.org/10.1007/S11920-023-01467-Z/FIGURES/1.

64. Yan W, Zhao M, Fu Z, Pearlson GD, Sui J, Calhoun VD. Mapping relationships among schizophrenia, bipolar and schizoaffective disorders: A deep classification and clustering framework using fMRI time series. Schizophr Res 2021;245:141. https://doi.org/10.1016/J.SCHRES.2021.02.007.

65. Ferrara M, Franchini G, Funaro M, Cutroni M, Valier B, Toffanin T, et al. Machine Learning and Non-Affective Psychosis: Identification, Differential Diagnosis, and Treatment. Curr Psychiatry Rep 2022;24:925. https://doi.org/10.1007/S11920-022-01399-0.

66. Ostojic D, Lalousis PA, Donohoe G, Morris DW. The challenges of using machine learning models in psychiatric research and clinical practice. European Neuropsychopharmacology 2024;88:53–65. https://doi.org/10.1016/J.EURONEURO.2024.08.005.

67. Sun J, Lu T, Shao X, Han Y, Xia Y, Zheng Y, et al. Practical AI application in psychiatry: historical review and future directions. Mol Psychiatry 2025;30:4399. https://doi.org/10.1038/S41380-025-03072-3.

68. Yassin W, Nakatani H, Zhu Y, Kojima M, Owada K, Kuwabara H, et al. Machine-learning classification using neuroimaging data in schizophrenia, autism, ultra-high risk and first-episode psychosis. Transl Psychiatry 2020;10:1–11. https://doi.org/10.1038/S41398-020-00965-5;TECHMETA.

69. Deneault A, Dumais A, Désilets M, Hudon A. Natural Language Processing and Schizophrenia: A Scoping Review of Uses and Challenges. J Pers Med 2024;14:744. https://doi.org/10.3390/JPM14070744/S1.

70. De Filippis R, Carbone EA, Gaetano R, Bruni A, Pugliese V, Segura-Garcia C, et al. Machine learning techniques in a structural and functional MRI diagnostic approach in schizophrenia: a systematic review. Neuropsychiatr Dis Treat 2019;15:1605. https://doi.org/10.2147/NDT.S202418.

71. Cao P, Li R, Li Y, Dong Y, Tang Y, Xu G, et al. Machine learning based differential diagnosis of schizophrenia, major depression disorder and bipolar disorder using structural magnetic resonance imaging. J Affect Disord 2025;383:20–31. https://doi.org/10.1016/J.JAD.2025.04.135.

72. Stein DJ, Lund C, Nesse RM. Classification Systems in Psychiatry: Diagnosis and Global Mental Health in the Era of DSM-5 and ICD-11. Curr Opin Psychiatry 2013;26:493. https://doi.org/10.1097/YCO.0B013E3283642DFD.

73. Ciapparelli A, Dell'Osso L, Di Poggio AB, Carmassi C, Cecconi D, Fenzi M, et al. Clozapine in treatment-resistant patients with schizophrenia, schizoaffective disorder, or psychotic bipolar disorder: A naturalistic 48-month follow-up study. Journal of Clinical Psychiatry 2003;64:451–8. https://doi.org/10.4088/JCP.v64n0416.

74. Fonseca de Freitas D, Kadra-Scalzo G, Agbedjro D, Francis E, Ridler I, Pritchard M, et al. Using a statistical learning approach to identify sociodemographic and clinical predictors of response to clozapine. J Psychopharmacol 2022;36:498–506. https://doi.org/10.1177/02698811221078746.

75. Del Fabro L, Bondi E, Serio F, Maggioni E, D'Agostino A, Brambilla P. Machine learning methods to predict outcomes of pharmacological treatment in psychosis. Transl Psychiatry 2023;13:75. https://doi.org/10.1038/S41398-023-02371-Z.

76. De Hert M, Schreurs V, Vancampfort D, Van Winkel R. Metabolic syndrome in people with schizophrenia: a review. World Psychiatry 2009;8:15. https://doi.org/10.1002/J.2051-5545.2009.TB00199.X.

77. Habehh H, Gohel S. Machine Learning in Healthcare. Curr Genomics 2021;22:291–300. https://doi.org/10.2174/1389202922666210705124359.

78. Del Fabro L, Bondi E, Serio F, Maggioni E, D'Agostino A, Brambilla P. Machine learning methods to predict outcomes of pharmacological treatment in psychosis. Transl Psychiatry 2023;13:75. https://doi.org/10.1038/S41398-023-02371-Z.

79. Wang X, Zhang Y, Wang PJ, Yan Q, Wang XX, Wu HS, et al. Altered Brain Network Dynamics in Schizophrenia Patients With Predominant Negative Symptoms: A Resting-State fMRI Study Using Co-Activation Pattern Analysis. Hum Brain Mapp 2025;46:e70369. https://doi.org/10.1002/HBM.70369.

80. Ottet MC, Schaer M, Debbané M, Cammoun L, Thiran JP, Eliez S. Graph theory reveals dysconnected hubs in 22q11DS and altered nodal efficiency in patients with hallucinations. Front Hum Neurosci 2013;7:402. https://doi.org/10.3389/FNHUM.2013.00402.

81. Mena N, Ab M. Machine Learning techniques and

Polygenic Risk Score application to prediction genetic diseases. ADCAIJ: Advances in Distributed Computing and Artificial Intelligence Journal 2020;9:5–14. https://doi.org/10.14201/ADCAIJ202091514.

82. Guerrin CGJ, Doorduin J, Sommer IE, de Vries EFJ. The dual hit hypothesis of schizophrenia: Evidence from animal models. Neurosci Biobehav Rev 2021;131:1150–68. https://doi.org/10.1016/J.NEUBIOREV.2021.10.025.

83. Lysaker PH, Pattison ML, Leonhardt BL, Phelps S, Vohs JL. Insight in schizophrenia spectrum disorders: relationship with behavior, mood and perceived quality of life, underlying causes and emerging treatments. World Psychiatry 2018;17:12. https://doi.org/10.1002/WPS.20508.

84. Zonayed M, Tasnim R, Jhara SS, Mimona MA, Hussein MR, Mobarak MH, et al. Machine learning and IoT in healthcare: Recent advancements, challenges & future direction. Adv Biomark Sci Technol 2025;7:335–64. https://doi.org/10.1016/J.ABST.2025.08.006.

85. Chen ZS, Kulkarni P (Param), Galatzer-Levy IR, Bigio B, Nasca C, Zhang Y. Modern views of machine learning for precision psychiatry. Patterns 2022;3:100602. https://doi.org/10.1016/J.PATTER.2022.100602.

86. Koppe G, Meyer-Lindenberg A, Durstewitz D. Deep learning for small and big data in psychiatry. Neuropsychopharmacology 2020;46:176. https://doi.org/10.1038/S41386-020-0767-Z.

87. Yasin S, Adeel M, Draz U, Ali T, Hijji M, Ayaz M, et al. A CNN-Transformer Fusion Model for Proactive Detection of Schizophrenia Relapse from EEG Signals. Bioengineering 2025;12:641. https://doi.org/10.3390/BIOENGINEERING12060641.

88. Li C, Chen J, Dong M, Yan H, Chen F, Mao N, et al. Classification of schizophrenia spectrum disorder using machine learning and functional connectivity: reconsidering the clinical application. BMC Psychiatry 2025;25:372. https://doi.org/10.1186/S12888-025-06817-0.

89. Cortes-Briones JA, Tapia-Rivas NI, D'Souza DC, Estevez PA. Going deep into schizophrenia with artificial intelligence. Schizophr Res 2022;245:122–40. https://doi.org/10.1016/J.SCHRES.2021.05.018.

90. Khan W, Daud A, Khan K, Muhammad S, Haq R. Exploring the frontiers of deep learning and natural language processing: A comprehensive overview of key challenges and emerging trends. Natural Language Processing Journal 2023;4:100026. https://doi.org/10.1016/J.NLP.2023.100026.

91. Abbas Q, Jeong W, Lee SW. Explainable AI in Clinical Decision Support Systems: A Meta-Analysis of Methods, Applications, and Usability Challenges. Healthcare 2025;13:2154. https://doi.org/10.3390/HEALTHCARE13172154.

92. Muthukrishna M, Bell A V., Henrich J, Curtin CM, Gedranovich A, McInerney J, et al. Beyond Western, Educated, Industrial, Rich, and Democratic (WEIRD) Psychology: Measuring and Mapping Scales of Cultural and Psychological Distance. Psychol Sci 2020;31:678–701. https://doi.org/10.1177/0956797620916782.

93. Susanto AP, Lyell D, Widyantoro B, Berkovsky S, Magrabi F. Effects of machine learning-based clinical decision support systems on decision-making, care delivery, and patient outcomes: a scoping review. J Am Med Inform Assoc 2023;30:2050. https://doi.org/10.1093/JAMIA/OCAD180.

94. Timmons AC, Duong JB, Simo Fiallo N, Lee T, Vo HPQ, Ahle MW, et al. A Call to Action on Assessing and Mitigating Bias in Artificial Intelligence Applications for Mental Health. Perspect Psychol Sci 2022;18:1062. https://doi.org/10.1177/17456916221134490.

95. Nouis SCE, Uren V, Jariwala S. Evaluating accountability, transparency, and bias in AI-assisted healthcare decision- making: a qualitative study of healthcare professionals' perspectives in the UK. BMC Med Ethics 2025;26:89. https://doi.org/10.1186/S12910-025-01243-Z.

96. Benjamens S, Dhunnoo P, Meskó B. The state of artificial intelligence-based FDA-approved medical devices and algorithms: an online database. NPJ Digit Med 2020;3:118. https://doi.org/10.1038/S41746-020-00324-0.

97. Torous J, Linardon J, Goldberg SB, Sun S, Bell I, Nicholas J, et al. The evolving field of digital mental health: current evidence and implementation issues for smartphone apps, generative artificial intelligence, and virtual reality. World Psychiatry 2025;24:156. https://doi.org/10.1002/WPS.21299.

98. Nouis SCE, Uren V, Jariwala S. Evaluating accountability, transparency, and bias in AI-assisted healthcare decision- making: a qualitative study of healthcare professionals' perspectives in the UK. BMC Med Ethics 2025;26:89. https://doi.org/10.1186/S12910-025-01243-Z.

Innovations in Brain Function: How Modern Science Is Redefining Human Thinking: Bridging Neuroscience and Psychology to Transform Human Cognition

Rehan Haider[1*], Zameer Ahmed[2]

[1]Riggs Pharmaceuticals, Department of Pharmacy, University of Karachi, Pakistan
[2]Assistant Professor, Department of Pathology, Dow University of Health Sciences, Karachi, Pakistan

Abstract

Recent advances in neuroscience and psychology have transformed our understanding of how the brain shapes thought, emotion, and behavior. Innovations such as neurostimulation, cognitive-behavioral interventions, neuropharmacology, and artificial intelligence (AI) have opened new frontiers in modifying brain function and redefining human thinking. This paper explores how these scientific and psychological approaches interact to enhance cognitive flexibility, emotional regulation, and problem-solving. By integrating neurobiological research with psychological frameworks, a more holistic understanding of human thinking emerges. The results demonstrate that interdisciplinary innovations are reshaping not only the science of cognition but also the philosophy of human thought itself.

INTRODUCTION

Understanding how modern science is transforming human thinking begins with appreciating the brain's remarkable adaptability, or neuroplasticity. This paper explores how contemporary neuroscience and psychology work together to enhance cognition, emotional regulation, and behavior. Advancements such as neurostimulation, neurofeedback, and artificial intelligence (AI) are reshaping how we understand thought and consciousness. The rationale for this study lies in the growing need to bridge biological and psychological insights to create a more integrated model of cognition. Therefore, the objective of this research is to investigate how interdisciplinary scientific innovations are redefining the human thought process.

The human brain's capacity for change, known as neuroplasticity, underlies modern efforts to improve cognitive and emotional performance. Scientific and technological innovations have provided new pathways to modify brain func-

tion, with implications for mental health, learning, and creativity [1]. Neurostimulation, neurofeedback, and AI-driven brain-computer interfaces (BCIs) now bridge the gap between biology and psychology, redefining what it means to think [2]. This research paper investigates how modern science reconfigures human thinking by combining neurobiological insights with psychological strategies.

Literature Review

Neurostimulation techniques such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) have been shown to enhance memory, learning, and mood regulation [3]. Meditation and mindfulness practices, rooted in ancient psychology, now have measurable neurobiological effects on cortical thickness and emotional stability [4]. Cognitive-behavioral therapy (CBT) modifies dysfunctional neural patterns through structured thought reappraisal [5]. Meanwhile, neuropharmacological interventions optimize neurotransmitter activity for improved cognition [6]. AI technologies analyze neural data to predict behavior and design personalized treatments [7]. Collectively, these studies illustrate that integrating neuroscience with psychology yields measurable changes in brain function and thinking

Research Methodology

A systematic review and analytical comparison were conducted using research published between 2015 and 2025. Databases such as PubMed, ScienceDirect, and PsycINFO were searched using key terms including 'neurostimulation', 'cognitive therapy', 'neuropharmacology', and 'AI in neuroscience'. Studies were included if they discussed measurable cognitive or neural outcomes related to these innovations. A total of 95 publications were initially screened; of these, 42 met the inclusion criteria and were analyzed in detail. The findings were categorized based on the type of intervention—neurostimulation, meditation and mindfulness, cognitive therapy, neuropharmacology, and artificial intelligence models—allowing for a structured comparison of their effects on human cognition.

This study utilizes a comparative analytical framework combining findings from neuroscience and

psychology literature published between 2015 and 2025. Peer-reviewed journals, clinical studies, and meta-analyses were reviewed to evaluate innovations affecting brain function. Data were categorized based on intervention type—neurostimulation, meditation, cognitive therapy, neuropharmacology, and AI-driven models—to assess their distinct and overlapping impacts on thinking and cognition.

RESULTS

The review revealed that all five categories of innovation have distinct yet complementary effects on cognitive processes. Neurostimulation techniques such as TMS and tDCS showed consistent improvements in working memory, focus, and emotional regulation. Meditation and mindfulness were linked to enhanced cortical thickness and reduced stress. Cognitive therapy, especially CBT, demonstrated success in reshaping maladaptive thought patterns, while neuropharmacology optimized neurotransmitter balance for better mental performance. Finally, artificial intelligence contributed to predictive modeling that helped identify individualized cognitive enhancement strategies. Table 1 and Figure 1 summarize the synthesized outcomes from the analyzed literature.

Figure 1 illustrates the conceptual model demonstrating the interaction between scientific innovation and psychological adaptation in redefining human thinking.
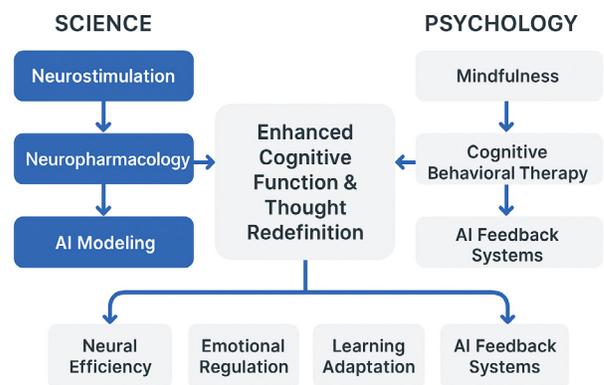

Figure 1: Conceptual model showing how science and psychology interact to redefine human thinking. Source: Author's illustration.
Source: Author's conceptual illustration based on Kolb & Gibb, 2019 [1]; Thibault et al., 2018 [2]; Friston, 2010 [9].

Table 1: Comparative summary of key innovations in brain function and their impact on cognitive processes

Innovation Type	Mechanism of Action	Impact on Thinking Patterns
Neurostimulation (TMS/tDCS)	Electrical or magnetic modulation of neural circuits	Enhances memory, focus, and mood regulation
Meditation and Mindfulness	Promotes neural plasticity and stress reduction	Improves emotional balance and cognitive clarity
Cognitive Therapy (CBT)	Reframes maladaptive thought patterns	Develops rational, goal-oriented thinking
Neuropharmacology	Regulates neurotransmitter activity	Optimizes mental performance and motivation
Artificial Intelligence (AI) Source: Author's conceptual illustration basedonKolb&Gibb,2019[1];Thibaultetal., 2018 [2]; Friston, 2010 [9].	Analyzes brain activity and predicts behavior	Enables adaptive learning and thought modeling

Discussion

This study highlights how neuroscience and psychology are converging to redefine the concept of thinking. Neurostimulation and neuropharmacological methods act directly on the neural circuits, while psychological techniques like CBT and mindfulness engage the brain's self-regulating capacity. AI-based models integrate these approaches by predicting and optimizing cognitive outcomes. The major finding is that cognitive enhancement is most effective when biological and psychological interventions are combined. The implications of this synthesis are significant for clinical practice, education, and artificial intelligence development. However, the study also acknowledges limitations—most notably, the reliance on secondary data and variability in study methodologies. Future research should focus on longitudinal and experimental designs to validate these integrative effects.

The convergence of neuroscience and psychology underscores that thinking is not merely a mental event but a neurobiological process shaped by external interventions. Neurostimulation and pharmacological methods provide direct modulation of neural pathways, while psychological approaches such as CBT and meditation engage the brain's intrinsic capacity for change [8]. AI models now predict how these interventions interact, offering precise cognitive enhancement tools [9]. Such innovations raise ethical and philosophical questions about human autonomy and the nature of thought itself. Nevertheless, these advancements pave the way toward a future where mental optimization becomes an integrated science of mind and brain.

Conclusion

Modern science is revolutionizing our understanding of how the brain shapes thought. The fusion of neuroscience, psychology, and artificial intelligence offers unprecedented insight into human cognition. By merging biological and psychological frameworks, researchers can redefine thinking as a dynamic, adaptable process. These innovations hold promise for mental health, education, and human development, heralding a new era in cognitive science.

Acknowledgment

References

1. Kolb B, Gibb R. Brain plasticity and behavior. Annu Rev Psychol. 2019;70:141–164.

2. Thibault RT, Lifshitz M, Raz A. The self-regulating brain and neurofeedback: experimental science and clinical promise. Cortex. 2018;109:93–118.

3. Lefaucheur JP, Aleman A, Baeken C, et al. Evidence-based guidelines on the therapeutic use of repetitive transcranial magnetic stimulation. Clin Neurophysiol. 2020;131(2):474–528.

4. Tang YY, Hölzel BK, Posner MI. The neuroscience of mindfulness meditation. Nat Rev Neurosci. 2015;16(4):213–225.

5. Beck JS. Cognitive Behavior Therapy: Basics and Beyond. 3rd ed. New York: Guilford Press; 2020.

6. Follow ND, Morales M. The brain on drugs: from reward to addiction. Cell. 2015;162(4):712–725.

7. Hassabis D, Kumaran D, Summerfield C, Botvinick M. Neuroscience-inspired artificial intelligence. Neuron. 2017;95(2):245–258.

8. Pascual-Leone A, Freitas C, Oberman L, et al. Characterizing neuroplasticity in humans: from brain stimulation to cognition. Brain Topogr. 2019;32(6):1011–1024.

9. Friston KJ. The free-energy principle: a unified brain theory? Nat Rev Neurosci. 2010;11(2):127–138.

Anxiety in Cancer Survivors

Simhachalam Gurugubelli[1], Prasad Rao[2]*, Nitesh Mutyala, Arvind Kunadi[3], Uma Devi Avasarala[4], Shivangini Singh[4]

[1]Clinical Assistant, Professor of Medicine, Indiana University School of Medicine
[2]Director of Schizophrenia Clinic and Psychopharmacology, Dean of Academic Affairs, Asha Hospital
[3]Assistant Program Director, McLaren Health Care
[4]Junior Research Officer, Asha Hospital

Abstract

People suffering from cancer often suffer from anxiety, which is fuelled by worries about the future, side effects from therapy, and fear of recurrence. Through molecular mechanisms linked to stress, these psychological difficulties may affect cancer outcomes and lower quality of life. Although overlapping physical symptoms might make diagnosing anxiety difficult, validated instruments like the HADS and GAD-7 help in early detection. Younger age, unemployment, comorbidities, adverse effects from therapy, a lack of social support, financial distress and type of cancer are important risk factors. Effective strategies for reducing anxiety and enhancing emotional health include mindfulness-based exercises, cognitive behavioural therapy (CBT), acceptance and commitment therapy (ACT), and alternative therapies like yoga and tai chi. In severe situations, pharmacotherapy may be necessary. It's critical to address obstacles like stigma, a lack of knowledge about mental health, and restricted access to care. Cancer survivors' psychological resilience and general health outcomes can be improved by incorporating mental health management into survivorship care.

INTRODUCTION

Psychological stress is a common occurrence in patients diagnosed with cancer.[1] Cancer diagnosis is often accompanied by a whirlwind of emotions—shock, disbelief, fear, and uncertainty about the future. These feelings can overwhelm patients and persist even after the initial stages of diagnosis and treatment. Anxiety and depression, in particular, are frequently reported in cancer survivors, often stemming from concerns about their future health, treatment side effects, and changes in their physical and emotional well-being.[2]

The emotional toll of cancer can be far-reaching, as survivors must often cope with the fear of cancer recurrence (FCR), a prevalent and significant contributor to psychological stress. This fear, which is deeply rooted in the unpredictability of cancer, can persist long after treatment ends. Patients may feel constant anxiety about every ache or pain, worrying that their cancer might have returned.[3] Such stress can be overwhelming, leading to chronic anxiety

and depression, both of which can negatively affect a survivor's quality of life.

In addition to its impact on emotional health, these psychological comorbidities—depression and anxiety—have been implicated in increasing the risk of cancer recurrence.[4] It has been suggested that stress-induced alterations in the immune system and inflammation may create a biological environment conducive to cancer growth or recurrence. Therefore, early detection and prompt treatment of mental health disorders in cancer survivors are crucial, as this can improve not only mental health outcomes but also long-term cancer outcomes, potentially reducing the risk of recurrence and enhancing overall survival rates.

Diagnosis of Anxiety

Identifying anxiety in cancer survivors can be particularly challenging due to the overlap between symptoms of anxiety and the physical effects of cancer and its treatment. For instance, fatigue, insomnia, and concentration difficulties can be symptoms of both anxiety and cancer treatment side effects. Thus, distinguishing between the two is essential but can be difficult for both patients and healthcare providers.

The Generalized Anxiety Disorder 7 (GAD-7) is a widely recognized and reliable tool for diagnosing anxiety.[5] This seven-item scale has been validated in various clinical settings and is effective in both primary care and specialized oncology practices. It is quick and easy to administer, making it an ideal choice for busy clinics. While the GAD-7 can help identify anxiety, it's important to recognize that not every cancer survivor will benefit from this screening tool. Knowing when to administer this tool requires careful observation and understanding of the patient's emotional state.

Self-reports are valuable tools for assessing anxiety, particularly in adult cancer survivors. These tools empower patients to articulate their emotional struggles, which might otherwise go unnoticed by healthcare providers. However, in pediatric cancer patients, self-reports may not be as reliable.[5] Children often lack the vocabulary to describe their feelings of anxiety, and their expressions of distress may manifest in behavioral changes rather than verbal

complaints. This is where observational assessments by parents and healthcare providers become crucial.

Due to the high prevalence of anxiety in cancer survivors, screening for anxiety using self-report questionnaires, such as the Hospital Anxiety and Depression Scale (HADS), is essential.[6] The HADS is a widely used screening tool in oncology settings and focuses on both anxiety and depression, offering a comprehensive view of a patient's emotional health. Its use can help identify patients who may require further psychological support and interventions.

Risk Factors

Several identifiable risk factors increase the likelihood of anxiety in cancer survivors. One study conducted on 1,323 patients with breast, lung, or gastrointestinal cancer found that younger age and unemployment were associated with greater levels of anxiety.[7] Younger survivors often face distinct challenges, such as concerns about future fertility, career prospects, and long-term health, which may not be as prevalent in older populations. Additionally, younger patients may feel a greater sense of isolation, as they are less likely to know peers who have experienced similar health challenges.

Unemployment exacerbates the financial and social stresses that can accompany cancer treatment, contributing to anxiety. Cancer survivors may struggle to return to work due to lingering physical side effects, or they may face workplace discrimination due to their medical history. The financial strain of unemployment, coupled with the high costs of cancer treatment, can lead to significant emotional distress, further increasing the risk of anxiety.

Another study conducted on 133 cancer survivors indicated that patients experiencing more side effects from treatment had a greater likelihood of developing an anxiety disorder.[3] Cancer treatments, such as chemotherapy, radiation therapy, and surgery, can cause significant physical side effects, including fatigue, pain, and cognitive changes (often referred to as "chemo brain"). These side effects can lead to a sense of helplessness and uncertainty about the future, which in turn contributes to heightened anxiety levels.

A cross-sectional study performed on colorectal cancer survivors suggested that survivors with

multiple comorbid diseases, shorter time since diagnosis, and female survivors might be at risk for higher levels of anxiety and/or depressive symptom severity.[8] Female survivors, in particular, may face unique challenges, such as changes in body image, reproductive health concerns, and caregiving responsibilities, all of which can contribute to emotional distress. Furthermore, those who have other chronic health conditions may feel overwhelmed by managing multiple illnesses at once.

Another cross-sectional study identified financial problems, global quality of life, and cognitive function as factors with the strongest association with depression and anxiety in cancer survivors[9]. Financial difficulties often arise from the high cost of cancer treatments, loss of income during treatment, and ongoing medical expenses related to follow-up care. Cognitive function, often impacted by cancer treatments, can also contribute to anxiety, as survivors may struggle to regain their pre-cancer cognitive abilities, which can affect their work and daily life.

Other risk factors include low emotional support, isolation, and low social status[10]. Social inequalities, such as low income or lack of access to healthcare, were also associated with poorer quality of life in cancer survivors[11]. Survivors with limited social support systems, such as those living alone or without family, may feel isolated and overwhelmed by their cancer journey. In contrast, those with strong support networks may have better emotional resilience and lower levels of anxiety.

The type of cancer is a significant predictor of anxiety and depression symptoms. These variations may be brought on by varying prognoses, pain thresholds, the extent of body image disturbance, and neuropsychiatric (side) effects associated with tumors or treatments.[12] In a cross-sectional study covering more than 13 different types of cancers, with over 7500 participants found lung and brain cancer patients to be particularly burdened, probably due to higher mortality rates. Patients with breast cancer, having better prognosis and social support, showed lower rates of anxiety[13]. However, a recent meta-analysis revealed that ovarian cancer had the highest prevalence of anxiety symptoms/disorders among cancers (43%), followed by breast cancer (27%), and lung cancer (26%).[14]

Treatment Approaches

The treatment of anxiety in cancer survivors is not markedly different from the treatment of anxiety arising from other causes. However, treating anxiety in this specific population is crucial, as it can lead to significant improvements in their quality of life. Additionally, some evidence suggests that managing anxiety may reduce the risk of cancer recurrence and all-cause mortality[4]. By alleviating psychological stress, patients may experience fewer negative health outcomes and be more resilient in the face of potential cancer-related challenges.

Cognitive behavioral therapy (CBT) is widely considered the first-line treatment for anxiety disorders, and it has been shown to improve quality of life in cancer survivors[15]. CBT helps patients challenge their negative thought patterns and develop healthier coping mechanisms for dealing with anxiety. In the context of cancer survivors, CBT may focus on helping patients manage their fears of recurrence, cope with physical side effects, and rebuild their sense of identity and purpose post-treatment.

Acceptance and commitment therapy (ACT) is particularly effective for cancer survivors[16]. Unlike CBT, which focuses on changing thought patterns, ACT encourages patients to accept their emotions and commit to actions that align with their values, even in the face of anxiety. This therapy can be especially beneficial for cancer survivors, who may need to come to terms with uncertainty and the possibility of recurrence. By focusing on values-driven action, survivors can build a meaningful life despite their fears.

Mindfulness has gained significant popularity as an effective adjunct therapy for managing anxiety and other behavioral symptoms related to cancer.[17] Mindfulness-based practices, such as meditation and deep-breathing exercises, help individuals stay grounded in the present moment, reducing the tendency to ruminate on past trauma or future uncertainties. Studies have shown that mindfulness-based interventions can reduce stress, improve emotional regulation, and enhance overall well-being in cancer survivors.

Some cancer survivors may require pharmacotherapy to manage their anxiety. These medica-

Table 1: Role of medications in managing anxiety in cancer survivors

Medication Class	Description	Study/Source
Benzodiazepines	Commonly used for short-term relief of anxiety. Typically limited to acute situations due to the risk of dependence.	Study [18]
Selective Serotonin Reuptake Inhibitors (SSRIs)	Often the most effective medications for treating anxiety with comorbid depression. Examples include sertraline and escitalopram. These medications help balance neurotransmitter levels.	Study [19]
Atypical Antipsychotics	Used in more severe cases, especially when anxiety is accompanied by psychotic symptoms. Examples include olanzapine and quetiapine.	Study [20]

tions can help balance neurotransmitter levels in the brain, leading to improvements in both mood and anxiety symptoms. The role of different classes of drugs in the management of anxiety in cancer patients has been discussed in Table 1.

Yoga and other relaxation techniques can also be helpful in reducing anxiety in cancer survivors. A randomized controlled study on cancer patients found that yoga therapy significantly reduced anxiety symptoms and fatigue compared to a control group that did not receive yoga therapy[21]. The gentle physical movement, combined with mindfulness and breath awareness, can help survivors reconnect with their bodies and reduce tension. Yoga and mindfulness have also been proven beneficial in reducing symptom load in cancer patients[22]. Tai Chi, especially Yang-style Tai Chi, has also been shown in studies to alleviate anxiety symptoms in cancer survivors.[23]

Barriers to Treatment

There are several barriers to the effective treatment of anxiety in cancer survivors. Mental health literacy—the ability to recognize mental health problems and seek appropriate treatment—is one of the most prominent barriers.[24] Many cancer survivors may not fully understand the symptoms of anxiety or may attribute them to physical side effects of treatment, leading to delayed diagnosis and treatment.

Experienced stigma and self-stigma are also major barriers to seeking mental health care[25]. Survivors may feel embarrassed or ashamed of their emotional struggles, believing that they should focus solely on their physical recovery. This stigma may be compounded by cultural beliefs or family pressures that discourage seeking mental health support. In some cases, survivors may view seeking mental health care as a sign of weakness or failure to "beat" cancer.

Negative attitudes toward professional help, high waiting times, and a shortage of mental health professionals also prevent many survivors from accessing the care they need.[26] The mental health system may be overwhelmed, leaving survivors waiting for weeks or months before receiving therapy. Additionally, the cost of mental health care can be prohibitive for many survivors, especially when combined with the already significant financial burden of cancer treatment.

CONCLUSION

Anxiety is highly prevalent in cancer survivors and has the potential to significantly impact their quality of life. The emotional toll of surviving cancer, compounded by fears of recurrence, physical side effects from treatment, and the social and financial challenges that survivors face, can lead to persistent anxiety that requires ongoing management. Prompt diagnosis and treatment of anxiety in cancer survivors are crucial, as they can lead to better emotional well-being, improved physical health outcomes, and potentially lower rates of cancer recurrence.

CBT, mindfulness-based approaches, and ACT have all been shown to be effective in treating anxiety in cancer survivors. Pharmacotherapy, including benzodiazepines, SSRIs, and atypical antipsychotics, may also be necessary for some patients, particularly those with severe anxiety or co-occurring mental health disorders. Complementary therapies, such as yoga and Tai Chi, can further enhance treatment by providing survivors with additional tools to manage stress and anxiety.

However, barriers to treatment, including mental health stigma, lack of awareness, and financial challenges, must be addressed to ensure that all cancer survivors have access to the care they need. By prioritizing mental health as a key component of survivorship care and addressing these barriers, healthcare providers can help cancer survivors lead healthier, more fulfilling lives, free from the burden of untreated anxiety.

References

1. Vargas-Román K, Díaz-Rodríguez CL, Cañadas-De la Fuente GA, Gómez-Urquiza JL, Ariza T, De la Fuente-Solana EI. Anxiety prevalence in lymphoma: A systematic review and meta-analysis. Health Psychol Off J Div Health Psychol Am Psychol Assoc 2020;39(7):580–8.
2. Suzuki M, Deno M, Myers M, Asakage T, Takahashi K, Saito K, et al. Anxiety and depression in patients after surgery for head and neck cancer in Japan. Palliat Support Care 2016;14(3):269–77.
3. Arch JJ, Genung SR, Ferris MC, Kirk A, Slivjak ET, Fishbein JN, et al. Presence and predictors of anxiety disorder onset following cancer diagnosis among anxious cancer survivors. Support Care Cancer 2020;28(9):4425–33.
4. Wang X, Wang N, Zhong L, Wang S, Zheng Y, Yang B, et al. Prognostic value of depression and anxiety on breast cancer recurrence and mortality: a systematic review and meta-analysis of 282,203 patients. Mol Psychiatry 2020;25(12):3186–97.
5. Spitzer RL, Kroenke K, Williams JBW, Löwe B. A brief measure for assessing generalized anxiety disorder: the GAD-7. Arch Intern Med 2006;166(10):1092–7.
6. Annunziata MA, Muzzatti B, Bidoli E, Flaiban C, Bomben F, Piccinin M, et al. Hospital Anxiety and Depression Scale (HADS) accuracy in cancer patients. Support Care Cancer Off J Multinatl Assoc Support Care Cancer 2020;28(8):3921–6.
7. Suskin JA, Paul S, Stuckey AR, Conley YP, Miaskowski C, Dunn LB. Risk factors for worse anxiety trajectories among patients undergoing cancer chemotherapy. Support Care Cancer Off J Multinatl Assoc Support Care Cancer 2022;31(1):32.
8. Braamse AMJ, van Turenhout ST, Terhaar Sive Droste JS, de Groot GH, van der Hulst RWM, Klemt-Kropp M, et al. Factors associated with anxiety and depressive symptoms in colorectal cancer survivors. Eur J Gastroenterol Hepatol 2016;28(7):831–5.
9. Götze H, Friedrich M, Taubenheim S, Dietz A, Lordick F, Mehnert A. Depression and anxiety in long-term survivors 5 and 10 years after cancer diagnosis. Support Care Cancer Off J Multinatl Assoc Support Care Cancer 2020;28(1):211–20.
10. Puigpinós-Riera R, Graells-Sans A, Serral G, Continente X, Bargalló X, Domènech M, et al. Anxiety and depression in women with breast cancer: Social and clinical determinants and influence of the social network and social support (DAMA cohort). Cancer Epidemiol 2018;55:123–9.
11. Graells-Sans A, Serral G, Puigpinós-Riera R, Grupo Cohort DAMA. Social inequalities in quality of life in a cohort of women diagnosed with breast cancer in Barcelona (DAMA Cohort). Cancer Epidemiol 2018;54:38–47.
12. Pitman A, Suleman S, Hyde N, Hodgkiss A. Depression and anxiety in patients with cancer. BMJ 2018;361:k1415.
13. Zeilinger EL, Oppenauer C, Knefel M, Kantor V, Schneckenreiter C, Lubowitzki S, et al. Prevalence of anxiety and depression in people with different types of cancer or haematologic malignancies: a cross-sectional study. Epidemiol Psychiatr Sci 2022;31:e74.
14. Amiri S. The prevalence of anxiety symptoms/disorders in cancer patients: a meta-analysis. Front Psychiatry 2024;15:1422540.
15. Ye M, Du K, Zhou J, Zhou Q, Shou M, Hu B, et al. A meta-analysis of the efficacy of cognitive behavior therapy on quality of life and psychological health of breast cancer survivors and patients. Psychooncology 2018;27(7):1695–703.
16. Mathew A, Doorenbos AZ, Jang MK, Hershberger PE. Acceptance and commitment therapy in adult cancer survivors: a systematic review and conceptual model. J Cancer Surviv Res Pract 2021;15(3):427–51.
17. Xunlin NG, Lau Y, Klainin-Yobas P. The effectiveness of mindfulness-based interventions among cancer patients and survivors: a systematic review and meta-analysis. Support Care Cancer Off J Multinatl Assoc Support Care Cancer 2020;28(4):1563–78.
18. Traeger L, Greer JA, Fernandez-Robles C, Temel JS, Pirl WF. Evidence-based treatment of anxiety in patients with cancer. J Clin Oncol Off J Am Soc Clin Oncol 2012;30(11):1197–205.
19. Popkin MK, Callies AL, Mackenzie TB. The outcome of antidepressant use in the medically ill. Arch Gen Psychiatry 1985;42(12):1160–3.
20. Sakamoto R, Koyama A. Effective Therapy Against Severe Anxiety Caused by Cancer: A Case Report and Review of the Literature. Cureus 12(6):e8414.
21. Hardoerfer K, Jentschke E. Effect of Yoga Therapy on Symptoms of Anxiety in Cancer Patients. Oncol Res Treat 2018;41(9):526–32.
22. Boppana SH, Kutikuppala LVS, Koirala SB, Konduru S, Thota V. Role of Mindfulness and Yoga in Cancer Management [Internet]. In: Anand A, editor. Neuroscience of Yoga: Theory and Practice: Part II. Singapore: Springer Nature; 2024 [cited 2025 Apr 14]. page 167–80.Available from: https://doi.org/10.1007/978-981-97-2855-8_7
23. Kuang X, Dong Y, Song L, Dong L, Chao G, Zhang X, et al. The effects of different types of Tai Chi exercise on anxiety and depression in older adults: a systematic review and network meta-analysis. Front Public Health 2023;11:1295342.

24. Kangas M, Heissel A. Mental health literacy, treatment preferences and the lived experience of mental health problems in an Australian cancer sample. Psychooncology 2020;29(11):1883–94.

25. Dubreucq J, Plasse J, Franck N. Self-stigma in Serious Mental Illness: A Systematic Review of Frequency, Correlates, and Consequences. Schizophr Bull 2021;47(5):1261–87.

26. Waumans RC, Muntingh ADT, Draisma S, Huijbregts KM, van Balkom AJLM, Batelaan NM. Barriers and facilitators for treatment-seeking in adults with a depressive or anxiety disorder in a Western-European health care setting: a qualitative study. BMC Psychiatry 2022;22(1):165.

Anhedonia: A Comprehensive Narrative Review

Dishita Jallan[1], Sara Wadhwa[2], Veenu Wadhwa[3], Reema Lamba[4*]

[1]*Bachelor's Student in Psychology (BS) and Human Health (BA), Emory College of Arts and Sciences, Emory University, Georgia, United States.*

[2]*Doctoral Student in Clinical Psychology (PhD), Palo Alto University, California, United States.*

[3]*Professor, Department of Human Development and Childhood Studies, Institute of Home Economics, University of Delhi, New Delhi, India.*

[4]*Professor, Department of Human Development and Family Empowerment, Aditi Mahavidyalaya, University of Delhi, New Delhi, India.*

Abstract

Anhedonia is a transdiagnostic symptom defined as the lack of pleasure due to disruptions in reward processing pathways, specifically in the prefrontal cortex and striatum. This paper aims to investigate the complex nature of anhedonia as observed in many psychiatric disorders, as well as analyze its neurobiological mechanisms, computational models, and treatment options. To do this, we completed a thorough review of recent literature, including developments in the field. The literature shows that current interventions aimed at treating anhedonia are also often inadequate, which leads to a greater need for novel treatments like neurostimulation and personalized therapeutic models. Many studies suggest that using genetic and behavioral data could also improve treatment efficacy. Further research is necessary to develop individualized models and interventions for anhedonia. Future studies in the field should be aimed at exploring the social context of anhedonia and integrating computational models with neurobiological components to improve treatment options.

ARTICLE INFO

INTRODUCTION

Théodule Ribot first defined anhedonia in 1897 as the inability to enjoy usually pleasurable activities or experiences.[1] More recently, the DSM-5-TR defined anhedonia as a loss of pleasure or interest in response to stimuli previously perceived as rewarding during a pre-morbid state.[2] The clinical manifestations of anhedonia include difficulty feeling pleasure, engaging in goal-directed activities, and adapting to changing circumstances. According to the DSM-5-TR, anhedonia, along with depressed mood, is one of the core symptoms for the diagnosis of a Major Depressive Episode and the "with melancholic features" specifier for Major Depressive Disorder (MDD).[2] It is also reported in other disorders such as schizophrenia, anxiety disorders, and post-traumatic stress disorder (PTSD). According to Snaith[3] and Treadway and Zald,[4] anhedonia has long been neglected in psychopathology, despite its pervasive impact on mood, motivation, and social and cognitive performance. Its significance has grown considerably in recent years as more researchers have been interested in investigating its transdiagnostic relevance in psychiatry.

The multidimensional nature of anhedonia, which includes a decrease in pleasure from physical or sensory experiences as well as a decreased desire

to engage in pleasurable activities, has important implications for understanding its role in psychiatric disorders. According to Pelizza and Ferrari,[5] anhedonia could be a state marker that fluctuates with mood and symptom severity, or a trait marker that reveals underlying vulnerabilities in persons predisposed to depression and schizophrenia. A state marker is a measure of a patient's current symptoms or condition and a trait marker is a biological factor that shows their natural risk for developing major mental disorders and plays a role in causing these conditions.[6] Fortunati *et al.*[7] highlight the subjective nature of anhedonia, where the individuals' inability to experience pleasure notably impacts their quality of life, leading to social withdrawal and diminished functioning.

Anhedonia is becoming an increasingly significant focus for study and clinical intervention because of its widespread influence across multiple life domains. Understanding the underlying causes of anhedonia may lead to more effective treatments, particularly as current research has started investigating its neurological basis and function in reward circuits.[8] As such, this paper aims to examine the underpinnings of anhedonia, its relationship with stress, how it manifests in depression and other related disorders, and current therapeutic techniques through an in-depth literature review of current research and developments in the field.

Methods

We performed a narrative review of the literature to consolidate evidence regarding the manifestation of anhedonia in various psychiatric disorders, including depression, schizophrenia, anxiety disorders, and PTSD, as well as the neurobiological underpinnings of anhedonia and its treatments. We performed systematic keyword searches across three primary databases: PubMed, PsycINFO, and Google Scholar. The databases were selected due to their comprehensive coverage of psychological, biomedical, and interdisciplinary research.

We searched for different combinations of the word "anhedonia" with related disorders and ideas to demonstrate that the review is transdiagnostic. The search terms encompassed examples such as: anhedonia, anhedonia AND depression, anhedonia AND schizophrenia, anhedonia AND anxiety, anhedonia AND PTSD, anhedonia AND treatments. These were chosen according to the topics discussed in the review and exemplify methodologies employed in prior literature searches involving anhedonia. Moreover, secondary searches integrated "anhedonia" with reward processing, neurobiology, or computational models to locate mechanistic studies.

We primarily searched for papers from 2000 to 2023 (the most recent studies at the time of writing), but we also included some important historical works (like Ribot's original definition and Snaith's early conceptual paper) to provide context. The search was not strictly limited by date, but we focused on research from the last 15 to 20 years to obtain the most up-to-date findings and developments.

The criteria for inclusion were as follows: Peer-reviewed journal articles written in English (including empirical studies, meta-analyses, systematic reviews, and conceptual/theoretical papers) that specifically focused on anhedonia or its underlying mechanisms. Empirical studies encompassed randomized controlled trials, observational and correlational studies, neuroimaging or neurophysiological investigations, and computational or modeling papers that measured or studied anhedonia. Review articles and book chapters that included evidence related to anhedonia were also incorporated. Studies needed to focus on human subjects (clinical or healthy populations) where anhedonia was evaluated or examined. Animal studies and other preclinical research were predominantly omitted unless they offered significant mechanistic insights. We did not include non-peer-reviewed works (like commentaries, editorials, dissertations, meeting abstracts) or works that were not written in English. We also excluded articles that did not focus on anhedonia, like general depression or anxiety papers that didn't explicitly measure or talk about pleasure or motivation deficits. These criteria (English language, human focus, peer-reviewed sources, and direct relevance to anhedonia) conform to established practices in narrative review methodology.

After our initial searches of the databases, we looked at the reference lists of all the relevant arti-

cles to find any other studies ("snowballing"). This iterative method helped us find important papers and new work that we might not have encountered with keyword searches alone. The final compilation of sources included in the corpus shows this wide, transdiagnostic search strategy, which is in line with best practices for narrative literature reviews.

Literature Review

Anhedonia in Different Psychiatric Disorders

Anhedonia is a phenomenon that manifests across various psychiatric disorders, each with a distinct symptomatology. This section analyzes the presentation of anhedonia in depression, schizophrenia, anxiety, and PTSD.

Anhedonia and Depression

Anhedonia, a core symptom of MDD, impairs the ability to feel pleasure and the motivation to participate in such activities. People affected by depression exhibit diminished activity in reward-processing brain regions, which leads to reduced interest in activities pleasurable previously.[4,8] Rizvi et al.[9] classified anhedonia in depression as either consummatory anhedonia, the inability to enjoy rewards in the moment, or anticipatory anhedonia, the inability to look forward to or expect pleasure from future events. Depressive anhedonia is also closely associated with stress-related mechanisms. A study shows that chronic stress and its neuroinflammatory responses can worsen anhedonia by further impairing dopamine transmission in the mesolimbic pathway.[10] The combined effects of dopamine dysregulation and stress-induced neuroinflammation highlight the complex neurobiological pathways that cause anhedonia in depression.

Anhedonia and Schizophrenia

Anhedonia is a key negative symptom of schizophrenia that can significantly affect life quality and social functioning. Individuals affected by schizophrenia often report difficulty deriving pleasure from social interactions and other previously enjoyable activities. A study suggests that unlike in depression, patients with schizophrenia may have the ability to feel pleasure during enjoyable experiences but struggle with the motivation to seek out these experiences.[11] This distinction is important for understanding the presentation of anhedonia in schizophrenia, which may be more closely linked to deficits in motivation rather than the feeling of pleasure itself.

According to Fortunati et al.,[7] subjective experiences in schizophrenia differ greatly, with some patients reporting extreme emotional blunting and others reporting fleeting moments of pleasure that are hard to sustain. Fortunati et al.[7] also classified social anhedonia, or the inability to feel pleasure in social settings, as a distinct type of anhedonia in schizophrenia. Additionally, another study found that anhedonia in schizophrenia is further worsened by impairments in social cognition and functioning because individuals with this disorder may have difficulty recognizing or interpreting rewarding social cues, leading to diminished interaction with other people.[12] Thus, understanding social anhedonia is important to develop interventions addressing both neurobiological and social deficits.

Anhedonia and Anxiety

While anhedonia is most commonly observed in depression and schizophrenia, it also manifests in anxiety disorders and PTSD. Taylor et al.[13] found that anhedonia often overlaps with feelings of worry and hypervigilance in anxiety disorders, reducing the ability to experience positive emotions. This can be further worsened by the state of persistent anxiety, leading to diminished pleasure in everyday activities and social withdrawal. Winer et al.[14] posited that the reduced capacity for pleasure is detrimental because it reduces the ability to engage in positive, mood-enhancing experiences that can buffer against anxiety symptoms, thereby potentially contributing to the worsening of symptoms. Additionally, Taylor et al.[13] found that anhedonia could also play a role in anxiety onset and maintenance, perpetuating avoidance behaviors, increasing threat reactivity, reducing tolerance for distress, and impairing learning. This suggests that anhedonia not only worsens anxiety but also limits opportunities for recovery and resilience.

Anhedonia may worsen anxiety in depression by disrupting sensory processing and environmental

perception.[15] Grillo[15] posits that intense pleasures can help restore these experiences, reducing both anxiety and depression temporarily. This suggests that targeting sensory and experiential deficits could be a key therapeutic strategy for alleviating co-occurring anxiety and depression. Thus, better understanding and targeting anhedonia in anxiety disorders could help alleviate persistent symptoms and improve overall treatment outcomes.

Anhedonia and PTSD

Anhedonia is a common symptom in PTSD because trauma-exposed individuals experience heightened sensitivity to stress. One study found that the neural circuit involved in reward processing is often disrupted in PTSD, with decreased activity in the brain's reward centers contributing to emotional numbing and avoidance-like symptoms.[16] Additionally, Kirshenbaum *et al.*[17] found that neural sensitivity to stress can predict the development of anhedonia in trauma survivors, with long-term disruptions in reward processing persisting for years after the traumatic event. This suggests that understanding and addressing reward processing disruptions in PTSD could be crucial for preventing and treating anhedonia in trauma survivors.

Anhedonia in PTSD is believed to result from inhibited emotional expression, heightened sensitivity to negative stimuli, and disruptions in the brain's reward system, with some suggesting it could also be a pre-existing risk factor due to early-life adversity.[18] Frewen *et al.*[19] explored anhedonia in women with PTSD, distinguishing between hedonic deficits (reduced positive affect) and negative affective interference (negative responses to positive stimuli). The study linked anhedonia to PTSD severity, depressive symptoms, emotional responses- particularly in social contexts- and observed differing neural responses between social and non-social stimuli, with trauma exposure and early-life abuse identified as key factors.[19]

Neurobiological and Computational Models

Neurobiological Models

The neurobiological mechanisms of anhedonia have been extensively studied, with most research focusing on the reward circuitry of the brain. The main component of this system is the mesolimbic dopamine pathway since it is important for reward and pleasure processing.[8] Research has found dopamine dysregulation in this circuit to be involved in the development of anhedonia, especially in cases of MDD.[8] A study indicated that a decrease in dopamine transmission in certain brain regions, the ventral tegmental area (VTA) and nucleus accumbens, can lead to reduced motivation and pleasure-seeking behavior, which are distinctive features of anhedonia.[20]

In their 2018 study, Cooper *et al.*[20] also reported that reward processing deficits are the primary biological mechanisms of anhedonia in depression, mainly because of their association with dopamine transmission and reinforcement learning. They hypothesized that anhedonia is linked to reduced dopamine transmission, which affects the experience of pleasure and the effectiveness of reinforcement learning. Typically, anhedonic symptoms in depression are often thought to be due to failures in reinforcement systems. However, this study's findings suggest that while depressed individuals may have a diminished ability to learn by utilizing rewards, the primary deficit may lie in reward sensitivity rather than learning, meaning that they may be less motivated by or responsive to rewards.[20]

Other than dopamine, neurotransmitters such as serotonin and glutamate are also involved in the manifestation of anhedonia. Research has linked serotonin to mood regulation and emotional processing, showing that disruptions in serotonergic signaling may lead to anhedonia in depression.[21] Another study suggests that glutamate dysregulation may alter synaptic plasticity and increase anhedonic symptoms, especially in brain regions related to reward processing and emotion.[22]

Stress-related mechanisms also play an important role in the onset and continued experience of anhedonia. A study showed that chronic stress impairs neuroplasticity and diminishes the ability of the brain to adjust to positive stimuli, contributing to the persistence of anhedonia.[23] Recent research also shows that stress-induced inflammation further disrupts the brain's reward

circuitry, especially in individuals with high neural sensitivity to stress.[10] This neuroinflammatory response worsens anhedonia by increasing dopamine dysregulation and impeding the operation of the reward system.

Neuroimaging studies have also provided insight into the structural and functional abnormalities in the brains of those with anhedonia. A study by Treadway and Zald[4] consistently observed reduced activity in the prefrontal cortex and ventral striatum, suggesting that anhedonia may arise from impaired top-down regulation of reward processing. These irregularities in brain function may indicate broader changes in neural circuitry, leading to a decreased ability to feel pleasure and motivation to partake in rewarding activities.

Understanding the neurobiological basis of anhedonia provides various opportunities for developing targeted interventions. In one study, pharmacological treatments targeting the mesolimbic pathway to enhance dopamine function showed potential in treating anhedonic symptoms in depression and schizophrenia.[22] Another study suggested anti-inflammatory treatments, a novel approach, to treat the stress-related components of anhedonia by reducing neuroinflammation and restoring reward processing.[16] As research in this area progresses, a better understanding of the neurobiology of anhedonia may lead to more promising interventions for individuals with various psychiatric disorders suffering from this symptom.

Computational Models

Computational models are becoming increasingly important in understanding the complex causes of anhedonia, providing insights that go beyond the traditional approaches. These models simulate brain processes involved in reward processing, decision-making, and motivation to help identify key disruptions linked with anhedonia. They offer a framework to link observable symptoms of anhedonia with underlying neural mechanisms by incorporating findings from neuroimaging studies and behavioral data.[20]

The reinforcement learning (RL) framework is the most commonly used computational model for studying anhedonia. It examines how individuals learn to associate actions with rewards and alter behavior based on feedback. In one study, Treadway and Zald[4] found that this process is disrupted in anhedonia, which results in decreased reward responsiveness and diminished motivation to seek pleasurable experiences. According to another study using the RL model, individuals with anhedonia have deficits in both reward sensitivity and the ability to learn from positive reinforcement, suggesting that the brain's reward prediction error signals, which measure the difference between expected and actual rewards, may be impaired.[20] This means that individuals with anhedonia fail to assess rewards and thus may not make optimal choices. These deficits can be simulated within RL models, allowing researchers to test different hypotheses about the underlying causes of anhedonia. Through this, future research can address the motivational deficits associated with anhedonia and lead to the development of targeted interventions that enhance reward sensitivity and improve the ability to learn from positive reinforcement.

Value-based decision-making models have also been used to study how anhedonia affects decision-making for delayed and immediate rewards. Pizzagalli[21] and Olson *et al.*[24] reported that individuals with anhedonia tend to favor smaller immediate incentives over larger delayed rewards, displaying a disruption in the brain's ability to anticipate future pleasure. Computational models can quantify these changes in the judgment of reward value and identify the neural circuits involved in the decision-making impairments seen in anhedonia by simulating decision-making processes. Even though computational models have significantly advanced our knowledge of anhedonia, there is a need for more individualized models, which serves as a big challenge. Current models often fail to record the heterogeneity of anhedonia across psychiatric disorders. To offer a more comprehensive view of the manifestation of anhedonia, future models should integrate personalized data, such as genetic, neuroimaging, and behavioral data.[17] Further, incorporating longitudinal data may improve the prediction of treatment outcomes and disease progression.[16] Moreover, computational models should study the role of inflammation and immune dysregulation in anhedonia. A recent study suggests that inflamma-

tion disrupts reward-related circuits, and incorporating inflammatory biomarkers into models could lead to new treatment avenues.[10]

A promising approach by Klein *et al.*[22] combined computational models with neurostimulation techniques. These models could lead to the development of targeted interventions, like deep brain stimulation (DBS) or transcranial magnetic stimulation (TMS), and optimize treatment parameters by identifying disrupted neural circuits. Additionally, Rizvi *et al.*[9] reported that machine learning also shows the potential to analyze large datasets to identify patterns and improve diagnostic accuracy and treatment plans. As computational models evolve, they can provide a better understanding of anhedonia, paving the way for individualized treatments and improved outcomes.

Treatment Approaches for Anhedonia

Given the complex neurobiological and psychological causes behind anhedonia, effective treatment approaches often require interventions that target both biological and cognitive factors. Traditional approaches to treating anhedonia focus on pharmacological interventions, while recent research explores psychotherapy and lifestyle-based interventions to treat this symptom.

Pharmacological interventions for anhedonia primarily focus on modulating the brain's dopamine and serotonin systems, which are critical for reward processing and motivation. Patients with anxiety and depression are frequently prescribed serotonin-norepinephrine reuptake inhibitors (SNRIs) and selective serotonin reuptake inhibitors (SSRIs).[22] However, Pizzagalli[21] found that while SSRIs and SNRIs are effective in alleviating general depressive symptoms, their efficacy in specifically treating anhedonia has been limited. This may be due to their indirect effect on the dopamine system, as they primarily target serotonin and norepinephrine pathways rather than directly enhancing dopamine transmission.

New pharmacological treatments target the dopamine system more directly. Treadway and Zald[4] reported that drugs such as Bupropion, which increases dopamine activity, have shown promise in treating anhedonic symptoms by improving motivation and reward sensitivity. Novel treatments such as ketamine, an NMDA receptor antagonist, have also shown immediate and lasting effects on anhedonia, potentially due to its effect on glutamatergic signaling and neural plasticity.[22] However, further study is required to determine the long-term safety and effectiveness of these treatments for anhedonia.

Psychological treatments for anhedonia aim to improve emotional and cognitive regulation while re-engaging patients with pleasurable activities. One such approach is Positive Affect Treatment (PAT), which encourages positive emotional experiences and focuses on reforming cognitive patterns that reduce motivation to directly boost positive affect and reduce anhedonic symptoms.[25] PAT combines behavioral activation with cognitive-behavioral therapy (CBT) techniques to encourage patients who are feeling unmotivated to resume enjoyable activities or indifferent initially.[9]

Behavioral activation alone has also been proven effective in treating anhedonia, especially when combined with mindfulness and relaxation techniques. These techniques help patients identify and pursue activities that match their previous interests, reinforcing reward-seeking behavior.[26] Structured psychotherapy, such as CBT and dialectical behavior therapy (DBT), have also been used to treat anhedonia by helping patients recognize harmful thought patterns and substituting them with more adaptive cognitive strategies.[27]

Additionally, mindfulness-based therapies have become increasingly popular as adjunctive treatments for anhedonia. Examples of these are mindfulness-based cognitive therapy (MBCT) and mindfulness-based stress reduction (MBSR). These therapies encourage present-moment awareness and acceptance, helping individuals disengage from ruminative thinking and re-engage with pleasurable experiences.[25]

Neurostimulation methods such as deep brain stimulation (DBS) and transcranial magnetic stimulation (TMS) have become increasingly popular in recent years as prospective treatments for anhedonia. TMS increases neural activity and enhances mood regulation by non-invasively stimulating particular brain areas, such as the dorsolateral prefrontal

cortex. In a 2022 study, Klein *et al.*[22] reported that TMS can improve reward responsiveness and reduce anhedonia in treatment-resistant depression. On the other hand, DBS is more invasive and targets deeper brain structures involved in reward processing, such as the nucleus accumbens. Preliminary studies show that DBS may treat anhedonia in individuals with severe treatment-resistant psychiatric disorders, but more research is needed to study its long-term efficacy and safety.[22]

Lifestyle interventions like exercise and diet modifications have also shown potential in reducing anhedonic symptoms. Regular physical activity has been shown to improve mood and increase dopamine signaling in the brain's reward pathways, to decrease anhedonia.[9] Dietary interventions targeting inflammation and neuroplasticity, like increasing omega-3 fatty acid intake, have also been suggested as complementary treatments for anhedonia, but more clinical trials are needed to confirm their efficacy.[10]

Discussion

Our findings emphasize the complex nature and role of anhedonia across various psychiatric disorders. In addition to being a hallmark of depression, anhedonia is also among the symptoms of schizophrenia, anxiety disorders, and PTSD. The presentation of anhedonia in these disorders exhibits its transdiagnostic nature, implying that shared underlying neurobiological mechanisms could be targeted for treatment. The findings from a multitude of studies have shed light onto the brain regions and circuits, such as the prefrontal cortex, striatum, and reward-related circuits, involved in anhedonia.[4,20,23] These neurological disruptions appear consistent across disorders, while the specific manifestations of anhedonia may differ depending on the diagnosis.

Anhedonia is still difficult to treat, especially because it is resistant to conventional antidepressants. Current pharmaceutical and psychotherapy treatments frequently fail to address the motivational and reward-related deficits associated with anhedonia.[27] Novel therapies, such as positive affect treatment, which focuses on improving positive emotions and increasing participation in rewarding activities, show promise but require more research.[25]

Neurostimulation approaches, including TMS and DBS, have emerged as possible treatments because they directly target the brain circuits involved in reward processing.[22] These techniques may provide hope for patients suffering from treatment-resistant forms of anhedonia, but further research is needed to improve their efficacy and assess long-term effects.

The use of computational models has provided a more structured framework for understanding the changes in reward processing caused by anhedonia. These models have helped us understand how anhedonia shows itself in various psychiatric populations by mimicking the impacts of decreased reward sensitivity, motivation, and decision-making.[20] Computational models also enable the discovery of possible biomarkers and neurological targets for treatment, facilitating the development of tailored therapies. However, current models are restricted in their ability to represent the diversity of anhedonia, particularly among mental illnesses. To gain a better understanding of anhedonia, future research should focus on constructing individualized computer models that include genetic, neuroimaging, and behavioral data.[17]

Conclusion

While considerable progress has been made in understanding the neurobiological and psychological mechanisms of anhedonia, much is left to be discovered, particularly in terms of incorporating individualized treatments into clinical practice. Future research should focus on bridging the gap between neurobiological insights and clinical applications, eventually leading to the development of more effective interventions for those suffering from this debilitating symptom.

References

1. APA Dictionary of Psychology [Internet]. dictionary.apa.org. Available from: dictionary.apa.org/anhedonia
2. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th-TR. American Psychiatric Association; 2022. Available from: doi.org/10.1176/appi.books.9780890425787
3. Snaith P. Anhedonia: a neglected symptom of psychopathology. Psychological Medicine. 1993;23(4):957–66.

Available from: doi.org/10.1017/s0033291700026428

4. Treadway MT, Zald DH. Reconsidering anhedonia in depression: lessons from translational neuroscience. Neuroscience and biobehavioral reviews. 2011;35(3):537–55. Available from: doi.org/10.1016/j.neubiorev.2010.06.006

5. Pelizza L, Ferrari A. Anhedonia in schizophrenia and major depression: state or trait? Annals of General Psychiatry. 2009;8(1). Available from: doi.org/10.1186/1744-859X-8-22

6. Lema YY, Gamo NJ, Yang K, Ishizuka K. Trait and state biomarkers for psychiatric disorders: Importance of infrastructure to bridge the gap between basic and clinical research and industry. Psychiatry and Clinical Neurosciences. 2018;72(7):482–9. Available from: doi.org/10.1111/pcn.12669

7. Fortunati R, Ossola P, Camerlengo A, Bettini E, De Panfilis C, Tonna M, et al. Anhedonia in schizophrenia: The role of subjective experiences. Comprehensive Psychiatry. 2015;62:152–60. Available from: doi.org/10.1016/j.comppsych.2015.07.011

8. Gorwood P. Neurobiological mechanisms of anhedonia. Dialogues in Clinical Neuroscience. 2008;10(3):291–9. Available from: doi.org/10.31887/DCNS.2008.10.3/pgorwood

9. Rizvi SJ, Pizzagalli DA, Sproule BA, Kennedy SH. Assessing anhedonia in depression: Potentials and pitfalls. Neuroscience & Biobehavioral Reviews. 2016;65:21–35. Available from: doi.org/10.1016/j.neubiorev.2016.03.004

10. Boyle CC, Bower JE, Eisenberger NI, Irwin MR. Stress to inflammation and anhedonia: Mechanistic insights from preclinical and clinical models. Neuroscience & Biobehavioral Reviews. 2023;152:105307. Available from: doi.org/10.1016/j.neubiorev.2023.105307

11. Strauss GP, Gold JM. A New Perspective on Anhedonia in Schizophrenia. American Journal of Psychiatry. 2012;169(4):364–73. Available from: doi.org/10.1176/appi.ajp.2011.11030447

12. Lambert C, Da Silva S, Ceniti AK, Rizvi SJ, Foussias G, Kennedy SH. Anhedonia in depression and schizophrenia: A transdiagnostic challenge. CNS Neuroscience & Therapeutics. 2018;24(7):615–23. Available from: doi.org/10.1111/cns.12854

13. Taylor CT, Hoffman SN, Khan AJ. Anhedonia in Anxiety Disorders. Current Topics in Behavioral Neurosciences. 2022;58:201–18. Available from: doi.org/10.1007/7854_2022_319

14. Winer ES, Bryant J, Bartoszek G, Rojas E, Nadorff MR, Kilgore J. Mapping the relationship between anxiety, anhedonia, and depression. Journal of Affective Disorders. 2017;221:289–96. Available from: doi.org/10.1016/j.jad.2017.06.006

15. Grillo L. A Possible Role of Anhedonia as Common Substrate for Depression and Anxiety. Depression Research and Treatment. 2016;2016:1–8. Available from: doi.org/10.1155/2016/1598130

16. Mehta ND, Stevens JS, Li Z, Gillespie CF, Fani N, Michopoulos V, et al. Inflammation, reward circuitry and symptoms of anhedonia and PTSD in trauma-exposed women. Social Cognitive and Affective Neuroscience. 2020;15(10):1046–55. Available from: doi.org/10.1093/scan/nsz100

17. Kirshenbaum JS, Pagliaccio D, Pizzagalli DA, Auerbach RP. Neural sensitivity following stress predicts anhedonia symptoms: a 2-year multi-wave, longitudinal study. Translational Psychiatry. 2024;14(1). Available from: doi.org/10.1038/s41398-024-02818-x

18. Vinograd M, Stout DM, Risbrough VB. Anhedonia in Posttraumatic Stress Disorder: Prevalence, Phenotypes, and Neural Circuitry. Current Topics in Behavioral Neurosciences. 2021; Available from: doi.org/10.1007/7854_2021_292

19. Frewen PA, Dozois DJA, Lanius RA. Assessment of anhedonia in psychological trauma: psychometric and neuroimaging perspectives. European Journal of Psychotraumatology. 2012;3(1):8587. Available from: doi.org/10.3402/ejpt.v3i0.8587

20. Cooper JA, Arulpragasam AR, Treadway MT. Anhedonia in depression: biological mechanisms and computational models. Current opinion in behavioral sciences. 2018;22(1):128–35. Available from: doi.org/10.1016/j.cobeha.2018.01.024

21. Pizzagalli DA. Depression, Stress, and Anhedonia: Toward a Synthesis and Integrated Model. Annual Review of Clinical Psychology. 2014;10(1):393–423. Available from: doi.org/10.1146/annurev-clinpsy-050212-185606

22. Klein ME, Grice AB, Sheth S, Go M, Murrough JW. Pharmacological Treatments for Anhedonia. Anhedonia: Preclinical, Translational, and Clinical Integration. 2022;467–89. Available from: doi.org/10.1007/7854_2022_357

23. Anisman H, Matheson K. Stress, depression, and anhedonia: Caveats concerning animal models. Neuroscience & Biobehavioral Reviews. 2005;29(4-5):525–46. Available from: doi.org/10.1016/j.neubiorev.2005.03.007

24. Olson EA, Kaiser RH, Pizzagalli DA, Rauch SL, Rosso IM. Anhedonia in Trauma-Exposed Individuals: Functional Connectivity and Decision-Making Correlates. Biological Psychiatry Cognitive Neuroscience and Neuroimaging. 2018;3(11):959–67. Available from: doi.org/10.1016/j.bpsc.2017.10.008

25. Craske MG, Meuret AE, Ritz T, Treanor M, Dour H, Rosenfield D. Positive affect treatment for depression and anxiety: A randomized clinical trial for a core feature of anhedonia. Journal of Consulting and Clinical Psychology. 2019;87(5):457–71. Available from: doi.org/10.1037/ccp0000396

26. Sandman CF, Craske MG. Psychological Treatments for Anhedonia. Current Topics in Behavioral Neurosciences. 2021;58. Available from: doi.org/10.1007/7854_2021_291

27. Craske MG, Meuret AE, Ritz T, Treanor M, Dour HJ. Treatment for Anhedonia: A Neuroscience Driven Approach. Depression and Anxiety. 2016;33(10):927–38.

Reimagining Recovery: Integrating Evidence-Based Rehabilitation, Technology, and Indigenous Wisdom in Severe Mental Illness

Venu Gopal Jhanwar, Padmanabh J*

Deva Institute of Healthcare & Research Pvt. Ltd., Varanasi, India

Abstract

Recovery in severe mental illnesses (SMI) such as schizophrenia and bipolar disorder has evolved from a focus on symptom remission to functional and personal recovery. Contemporary psychiatry demands integration of biological and psychosocial domains, using evidence-based rehabilitation strategies alongside technology and culturally contextualized practices. This paper critically examines modern rehabilitation models emphasizing cognitive remediation, neuromodulation, and measurement-based care. It further explores how integrating indigenous wisdom and digital tools within an Indian context can strengthen recovery-oriented psychiatry. The proposed framework, derived from clinical practice at Anvita Rehabilitation & Deaddiction, underscores a triadic approach combining evidence-based interventions, neurotechnology, and holistic wellness for sustainable functional outcomes.

INTRODUCTION

Recovery in severe mental illness (SMI) has transitioned from a narrow biomedical paradigm to a multidimensional model encompassing biological, psychological, and social restoration (Anthony, 1993). Despite advances in psychopharmacology, nearly 30-40% of patients with schizophrenia and bipolar disorder experience persistent cognitive and functional deficits (Green et al., 2019). These impairments underline the necessity of structured rehabilitation interventions beyond pharmacotherapy. The World Health Organization (WHO, 2022) and World Psychiatric Association (WPA, 2021) emphasize recovery-oriented, community-integrated care that focuses on empowerment, functionality, and autonomy. In India, where treatment gaps remain above 70% for major mental illnesses (Gururaj et al., 2016), integrating evidence-based rehabilitation within resource-constrained environments requires innovation and contextual adaptation. This paper explores how integrating evidence-based rehabilitation, neurotechnology, and indigenous wisdom can redefine recovery trajectories for persons with SMI.

Evidence-Based Rehabilitation: From Symptom Control to Function Restoration

Rehabilitation in psychiatry seeks to restore not only symptom stability but also autonomy, productivity, and social connectedness. Early rehabilitation models, such as the Boston University Approach (Anthony et al., 2002), proposed recovery as a personal process distinct from clinical remission. Recent meta-analyses support psychosocial interventions—including cognitive remediation, social skills training, supported employment, and family psychoeducation—as robust predictors of improved functioning (Wykes et al., 2011; Bond & Drake, 2014). Cognitive remediation therapy (CRT) and its structured variants, such as NEAR (Neuropsychological Educational Approach to Remediation) and CIRCuiTS (Computerized Interactive Remediation of Cognition in Schizophrenia), demonstrate medium-to-large effect sizes in improving executive function and vocational readiness (Medalia & Choi, 2009; Wykes & Reeder, 2018). Objective outcome tools such as the Social and Occupational Functioning Scale (SOFS), the Recovery Assessment Scale (RAS), and the WHOQOL-BREF facilitate quantifiable measurement of progress. These instruments enable clinicians to transition from subjective recovery narratives to data-driven rehabilitation tracking.

Technology as a Catalyst for Recovery

Technological innovation has transformed rehabilitation science. Neuromodulation techniques such as repetitive transcranial magnetic stimulation (rTMS), deep transcranial magnetic stimulation (Deep TMS), transcranial direct current stimulation (tDCS), and neurofeedback have shown potential in augmenting cognitive and affective recovery in SMI (George & Post, 2011; Lefaucheur et al., 2020). Deep TMS, in particular, influences distributed cortical networks, improving negative symptoms and cognitive processing speed in schizophrenia (Rapinesi et al., 2020). Similarly, tDCS has been demonstrated to enhance working memory and executive control (Kuo & Nitsche, 2012). Digital psychiatry complements biological rehabilitation through tele-rehabilitation platforms, cognitive training applications, and wearable monitoring systems. Virtual reality (VR) environments for social cognition training have been proven to enhance empathy and social integration (Rus-Calafell et al., 2018). Artificial intelligence (AI) is increasingly applied in early relapse prediction and personalized therapy adjustments, promoting measurement-based care (Insel, 2023). The convergence of neuroplasticity-based interventions and real-time digital monitoring constitutes the foundation of precision rehabilitation psychiatry.

Integrating Indigenous Wisdom: A Complementary Approach

India's indigenous traditions offer time-tested frameworks of holistic well-being that align with recovery-oriented psychiatry. Concepts from Ayurveda and Yoga emphasize the equilibrium between body, mind, and consciousness—paralleling biopsychosocial models. Empirical research supports yoga-based interventions in improving mood, cognition, and neuroendocrine balance among patients with schizophrenia and depression (Varambally et al., 2012; Thirthalli et al., 2013). Meditation and pranayama practices have demonstrated measurable effects on neuroplasticity and stress biomarkers (Goyal et al., 2014). Integrating these interventions within structured rehabilitation programs can address gaps in emotion regulation and self-efficacy. Importantly, cultural congruence enhances adherence and engagement, critical for long-term functional outcomes in low- and middle-income countries (LMICs). However, it is crucial to maintain evidence-based fidelity—combining indigenous modalities with validated therapeutic frameworks rather than replacing biomedical care.

An Integrated Recovery Framework for India

A comprehensive rehabilitation framework for SMI must integrate three synergistic pillars: clinical, functional, and spiritual recovery. At the Deva-Anvita model, recovery is conceptualized through measurable outcomes spanning symptom remission (PANSS, HAM-D), functional reintegration (SOFS, WHOQOL), and subjective empowerment (RAS). This triadic approach reflects the convergence of

evidence-based interventions, technological augmentation, and indigenous wisdom. Neuromodulation and pharmacotherapy form the biological base; cognitive remediation, occupational therapy, and Recovery College models provide the functional scaffold, while yoga, mindfulness, and meaning-making offer existential integration. Outcome monitoring through KPI dashboards enables continuous quality improvement and accountability in care delivery. The model aligns with the WHO's Comprehensive Mental Health Action Plan 2022-2030, emphasizing recovery-oriented, community-integrated, and technology-supported systems.

Implementation in the Indian Context

India's psychiatric rehabilitation landscape faces systemic challenges: an inadequate trained workforce, low public investment, and stigma-related disengagement. Nevertheless, models such as the Schizophrenia Research Foundation (SCARF, Chennai), NIMHANS, and private centers like Anvita exemplify scalable rehabilitation practices. Public-private partnerships (PPP) and CSR-funded recovery colleges could address accessibility gaps by offering structured psychoeducation, cognitive training, and peer-led learning environments. The integration of recovery metrics—SOFS, RAS, and WHOQOL-BREF—into electronic medical record systems can institutionalize measurement-based psychiatry. Emerging policy frameworks under India's Mental Healthcare Act (2017) provide a legislative basis for rights-based recovery services. Training psychiatry residents and psychologists in cognitive remediation, neuromodulation, and digital therapy should be prioritized for nationwide capacity-building.

Conclusion

Recovery in SMI demands a paradigm shift from episodic symptom control to continuous neuropsychological rehabilitation. Integrating evidence-based interventions such as cognitive remediation and neuromodulation with digital tools and culturally sensitive practices creates a multidimensional scaffold for sustained recovery. The

Indian mental health ecosystem, with its pluralistic traditions and emerging technological capacity, is uniquely positioned to pioneer an indigenous yet globally relevant model of recovery-oriented psychiatry. Future research must focus on longitudinal outcome data, cost-effectiveness analyses, and culturally nuanced rehabilitation metrics.

References

1. Anthony, W. A. (1993). Recovery from mental illness: The guiding vision of the mental health service system in the 1990s. Psychosocial Rehabilitation Journal, 16(4), 11-23.
2. Anthony, W. A., Cohen, M. R., & Farkas, M. (2002). Psychiatric Rehabilitation. Boston University Center for Psychiatric Rehabilitation.
3. Bond, G. R., & Drake, R. E. (2014). Making the case for IPS supported employment. Administration and Policy in Mental Health and Mental Health Services Research, 41(1), 69-73.
4. George, M. S., & Post, R. M. (2011). Daily left prefrontal repetitive transcranial magnetic stimulation for acute treatment of medication-resistant depression. American Journal of Psychiatry, 168(4), 356-364.
5. Green, M. F., Horan, W. P., & Lee, J. (2019). Social cognition in schizophrenia. Nature Reviews Neuroscience, 16(10), 620-631.
6. Goyal, M., Singh, S., Sibinga, E. M., Gould, N. F., Rowland-Seymour, A., Sharma, R., ... & Haythornthwaite, J. A. (2014). Meditation programs for psychological stress and well-being: A systematic review and meta-analysis. JAMA Internal Medicine, 174(3), 357-368.
7. Gururaj, G., Varghese, M., Benegal, V., Rao, G. N., Pathak, K., Singh, L. K., ... & NMHS Collaborators Group. (2016). National Mental Health Survey of India, 2015-16: Summary. NIMHANS Publication No. 128.
8. Insel, T. (2023). Healing: Our Path from Mental Illness to Mental Health. Penguin Press.
9. Kuo, M. F., & Nitsche, M. A. (2012). Effects of transcranial electrical stimulation on cognition. Clinical EEG and Neuroscience, 43(3), 192-199.
10. Lefaucheur, J. P., Aleman, A., Baeken, C., Benninger, D. H., Brunelin, J., Di Lazzaro, V., ... & Ziemann, U. (2020). Evidence-based guidelines on the therapeutic use of repetitive transcranial magnetic stimulation (rTMS). Clinical Neurophysiology, 131(2), 474-528.
11. Medalia, A., & Choi, J. (2009). Cognitive remediation in schizophrenia. Neuropsychology Review, 19(3), 353-364.
12. Rapinesi, C., Kotzalidis, G. D., Ferracuti, S., Sani, G., & Girardi, P. (2020). Efficacy of deep transcranial magnetic stimulation in the treatment of schizophrenia.

World Journal of Biological Psychiatry, 21(3), 222-232.

13. Rus-Calafell, M., Garety, P., Sason, E., Craig, T. K., & Valmaggia, L. R. (2018). Virtual reality in the assessment and treatment of psychosis: A systematic review. Psychological Medicine, 48(3), 362-391.

14. Thirthalli, J., Naveen, G. H., Rao, M. G., Varambally, S., Christopher, R., & Gangadhar, B. N. (2013). Cortisol and antidepressant effects of yoga. Indian Journal of Psychiatry, 55(S3), S405-S410.

15. Varambally, S., Gangadhar, B. N., Thirthalli, J., Jagannathan, A., & Keshavan, M. S. (2012). Therapeutic efficacy of add-on yoga in psychosis: Randomized controlled trial. Acta Psychiatrica Scandinavica, 126(5), 377-384.

16. WHO. (2022). World Mental Health Report: Transforming mental health for all. World Health Organization.

17. WPA. (2021). WPA Position Paper on Recovery-Oriented Practice. World Psychiatric Association.

18. Wykes, T., Huddy, V., Cellard, C., McGurk, S. R., & Czobor, P. (2011). A meta-analysis of cognitive remediation for schizophrenia: Methodology and effect sizes. American Journal of Psychiatry, 168(5), 472-485.

19. Wykes, T., & Reeder, C. (2018). Cognitive Remediation Therapy for Schizophrenia: Theory and Practice. Routledge.

Phineas of Punjab: A Young Contused Brain

Shradha Khatri*

Graded Specialist, MD, DNB (Psychiatry), AFMS, Pune, Maharashtra, India.

Abstract

Traumatic brain injury (TBI) can be defined as the disruption in brain function, or other evidence of brain pathology, caused by an external physical force. Its neuropsychiatric manifestations include a myriad of cognitive deficits, affective changes, apathy, psychosis and behavioral dyscontrol disorders. The case study aimed to discuss an interesting case of a young male had a deviation from his normalcy following head injury during an road traffic accident, under influence of alcohol. He was referred by his treating surgeon for evaluation of alcohol use, however, he had subtle behavioural changes in form of impulsivity, anger outbursts, perseverance and crying spells, a stark deviation from his pre-morbid self. Evaluation and identification of neuro-psychiatric consequences of traumatic brain injury requires a high suspicion, close monitoring, longitudinal follow ups along with pharmacotherapy, socio-occupational supportive measures and an empathetic-holistic approach.

INTRODUCTION

Phineas P. Gage (1823–1860) was an American railroad construction foreman who survived an accident where an iron rod was driven completely through his head, destroying his left frontal lobe, which eventually had profound effects on his personality and behaviour over the remaining twelve years of his life, making him "no longer Gage". Famously known as the "American Crowbar Case," it is one of the first documented cases suggesting the role of the brain in determining personality, and that damage to specific parts of the brain induces specific behavioural changes. [1]

A traumatic brain injury (TBI) [2] refers to a brain injury caused by an external force such as a forceful bump, blow, or jolt to the head or body, or from an object penetrating the brain. In some cases, the presenting problems are temporary or short-lasting with brain dysfunction, including problems with a person's cognition, emotions, behaviours and motor functions. Severe TBI often leads to profound and permanent disability, sometimes even death. [2]

Some injuries are considered primary, where the damage is immediate due to contact or inertial forces. (Acceleration, deceleration, rotation). In secondary TBI, damage evolves gradually over the course of hours, days, or weeks after injury. These secondary brain injuries are the result of reactive inflammatory

processes that occur after the initial head trauma. They can also be classified as penetrating (open) and non-penetrating (closed) TBI. [2] TBI often presents with a pleomorphic clinical presentation which includes physical symptoms (headache, convulsions, blurred vision, nausea/vomiting), neurological issues, etc), cognitive/behavioural symptoms (loss/change in consciousness, altered orientation, confusion, memory impairments, affective symptoms, deranged bio-drives, etc) and other systemic and sensory symptoms such as light-headedness, dizziness, vertigo, hearing problems, unexplained bad taste in the mouth, photophobia, fatigue or drowsiness, etc.

Organic Personality Disorder is a condition characterized by a significant change in personality and behaviours, due to an underlying TBI or another pathophysiological medical condition affecting the brain.[3]

Case Report

A 26-year-old Hindu male, the youngest of three siblings, hailing from a nuclear family of rural Punjab, educated till 12th standard, married for six years, had one three-year-old son and premorbid anxious and avoidant personality traits, presented to the emergency department of a tertiary care hospital following a road traffic accident with head trauma, under the influence of alcohol. During hospitalization in the Surgery ward, he was irritable and intermittently uncooperative. On day two of hospitalization, he was referred for Psychiatric evaluation on a consultation-liaison (CL) basis for alcohol use. Interviews with him and his wife did not reveal any history suggestive of a dependant pattern of alcohol use or withdrawal discomfort. Both refused consent for admission to the Psychiatry ward, so he was planned to be managed on a CL basis in the Surgical ward. Wife further described him to be a cheerful person, who had an active & outgoing social profile and managed stress in an adaptive and mature manner. In the Surgical ward, he continued to be irritable, pacing around, struggling with self-care, refusing meals, having disturbed sleep, with occasional crying spells. After one week of hospitalization, during visiting hours, he indulged in an inappropri-

ate, profane behaviour with his wife, in front of other patients and their families and an anger outburst towards his playful son, which was a gross deviation from his usual self. Thereafter, he was found to be overtalkative, repetitive and fidgety. Following the incident, the *now scared* wife re-visited the Psychiatrist and requested his management, stating that he is "*not my husband anymore",* precipitating his transfer to the Psychiatry ward as admission with high support needs as per section 89 of the Mental Health Care Act (MHCA), 2017.[4]

Mental status examination revealed an ill-kempt, uncooperative, fidgety, occasionally tearful, aggressive person with low tone, repetitive, but high rate and volume of speech. His affect fluctuated from being blunted to irritable to perplexed. He had an increased stream of thoughts, in the absence of delusions or perceptual abnormality. He had impaired personal and social judgement with intact memory, cognition and orientation.

Lab Investigations revealed normal Liver function tests (LFT), gamma-glutamyl transferase (GGT), mean corpuscular volume (MCV) and Ultrasonography of the abdomen. The Alcohol Use Disorders Identification Test (AUDIT) score was 7/40. [5] (rules out problematic alcohol use disorder) Montreal Cognitive Assessment (MOCA) revealed a score of 27/30, objectively suggestive of intact cognition. [6] On the Brief Psychiatric Rating Scale (BPRS) [7], he scored 46/126, suggestive of mild deviation from normalcy.

Serial non-contrast computed tomography (NCCT) was done. On the day of admission, it revealed bilateral frontal and left temporal haemorrhagic contusions. (Figure 1) On day two, following a head injury, NCCT revealed increased peri-lesional oedema. (Figure 2) After one month of trauma, NCCT showed prominent encephalomalacia with gliosis and a prominent temporal horn of the right lateral ventricle. (Figure 3)

A diagnosis of Organic Personality Disorder (ICD10 F07.0), now subsumed under the broader category of Personality change due to another medical condition (6E68) in ICD-11, [3,9] was made and Tablet Divalproate Sodium 750 mg in divided doses was started. He was also given the benefits of eclectic psychotherapy with components of supportive, behavioural and family psychotherapy along with
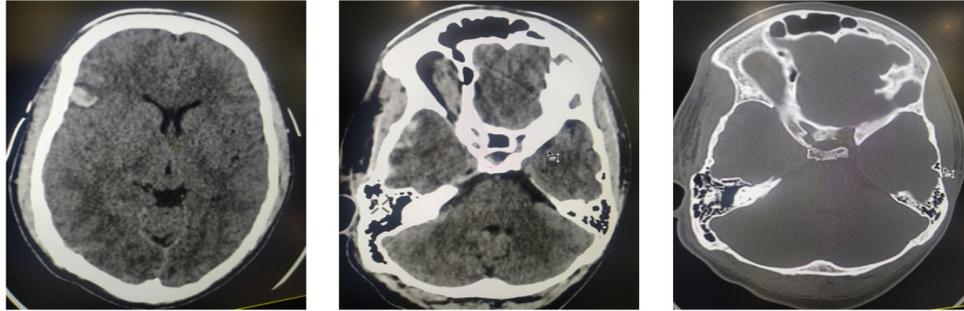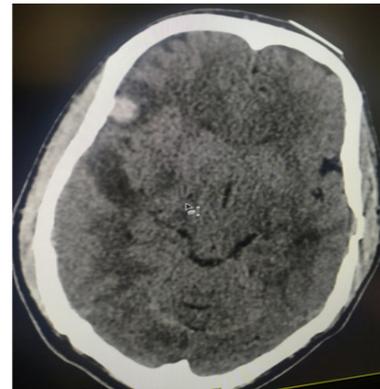
Figure 1: CT scan (Head) at Day 0: Right fronto-temporal contusion

psychoeducation. After nine weeks of intensive inpatient management, he was discharged with a follow-up plan of monthly review at the outpatient department, with a goal of treatment rationalization and vocational rehabilitation.
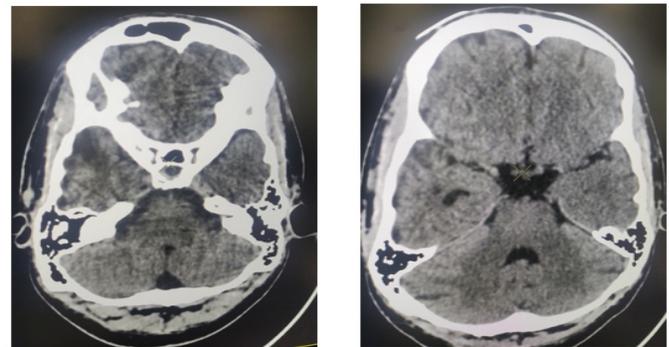
With regular follow-ups, treatment compliance and adequate social support, he continued to have clinical as well as socio-occupational improvement. During the last follow-up, he reported occasional dull aching headache after a perceived stressful and hectic day at his job, which would subside with Tab Paracetamol (500 mg) and rest. He is planned for a regular follow-up with maintenance medications, followed by gradual tapering and a period of drug-free observation.

Figure 2: CT scan (Head) at Day 5: Increased peri-lesional oedema

Discussion

Diagnostic and Statistical Manual of Mental Disorders (DSM) 5 defines TBI as brain trauma along with at least one of the features of loss of consciousness, post-traumatic amnesia, disorientation/confusion or neurological signs such as a new onset of seizure/worsening of pre-existing seizure disorder, visual field defects, anosmia, hemiparesis, or positive neuro-imaging.[8] In ICD-11, Personality change due to another medical condition is characterized by a persistent disturbance in personality that represents a marked deviation from the individual's premorbid functioning. This disturbance arises as a direct consequence of a medical condition affecting the brain, such as traumatic brain injury, epilepsy, cerebrovascular disease, or neurodegenerative disorders. The presentation includes reduced capacity for persistence in goal-directed activity and difficulty in sustaining efforts that require delayed gratification, emotional dysregulation, with lability,

Figure 3: CT scan (Head) at Day 60: Encephalomalacia with gliosis, Prominent temporal horn of right lateral ven-

irritability, or inappropriate affect, impulsivity and disinhibition, cognitive changes, and alterations in communication or behaviour, including changes in speech production or socially inappropriate sexual behaviours. These features represent a consistent and enduring change from the person's premorbid personality pattern and cannot be better explained by another mental, behavioural, or neurodevelopmental disorder. [9]

Neuropsychiatric syndromes are common after TBI, often underdiagnosed and have pleomorphic

presentations, causing a significant increase in distress, disability, and health care utilization. Most common psychiatric syndromes associated with TBI include Cognitive impairment, dementia, personality changes, affective disorders, psychosis, anxiety disorders, substance use disorder and post-concussion syndrome.[10]

A holistic and empathetic approach with high suspicion, close monitoring, longitudinal follow-up plans, rationalized pharmacotherapy, along with adequate socio-occupational supportive measures with focus on the biological, psychological and social profile of the individual with Psychiatric disorders is a must. Such cases often require a multi-disciplinary approach and long-term systematic monitoring.

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

Financial Support and Sponsorship

Nil.

Conflicts of Interest

There are no conflicts of interest.

References

1. Ratiu P, Talos IF, Haker S, Lieberman D, Everett P. The tale of Phineas Gage, digitally remastered. J Neurotrauma. 2004;21(5):637-43.
2. National Institute of Neurological Disorders and Stroke. Traumatic brain injury (TBI). Available from: https://www.ninds.nih.gov/health-information/disorders/traumatic-brain-injury-tbi [Accessed 2024 Oct 2].
3. Lang FU, Dudeck M, Becker T, Jaeger M. Organic personality disorder: conceptual principles, clinical symptoms and treatment. Nervenarzt. 2015;86:332-9.
4. Wikipedia contributors. Mental Healthcare Act, 2017 [Internet]. Wikipedia, The Free Encyclopedia; 2024. Available from: https://en.wikipedia.org/w/index.php?title=Mental_Healthcare_Act,_2017&oldid=1227139096
5. Babor TF, Higgins-Biddle JC, Saunders JB, Monteiro MG, World Health Organization. AUDIT: The Alcohol Use Disorders Identification Test: Guidelines for use in primary health care. Geneva: WHO; 2001.
6. Nasreddine ZS, Phillips NA, Bédirian V, Charbonneau S, Whitehead V, Collin I, et al. The Montreal Cognitive Assessment (MoCA): A brief screening tool for mild cognitive impairment. J Am Geriatr Soc. 2005;53(4):695-9.
7. Overall JE, Gorham DR. The Brief Psychiatric Rating Scale. Psychol Rep. 1962;10(3):799-812.
8. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders: DSM-5. 5th ed. Arlington (VA): APA Publishing; 2013.
9. World Health Organization. ICD-11: International classification of diseases for mortality and morbidity statistics. 11th revision. Geneva: WHO; 2019.
10. Fleminger S. Head injury. In: David AS, editor. Lishman's Organic Psychiatry. 4th ed. Wiley-Blackwell; 2009. p. 167-255.

Deep Cerebral Venous Sinus Thrombosis in an Alcohol Dependent Male: An Important Differential for Wernicke's Encephalopathy

Richa Tripathi[1*], Lalchhandama Hauhnar[1], Ashutosh Tiwary[2], Ayushi Taru[1]

[1]Department of Psychiatry, All India Institute of Medical Sciences, Gorakhpur, Uttar Pradesh, India.
[2]Department of Neurology, All India Institute of Medical Sciences, Gorakhpur, Uttar Pradesh, India.

Abstract

Wernicke's encephalopathy (WE) is suspected in patients with chronic alcoholism with poor nutrition presenting with walking difficulty, ocular problems and fluctuating orientation. However, one must quickly consider other possibilities if typical radiological features are absent or unexplained symptoms are present. A man in his mid-40s with an alcohol intake of ten years presented with walking difficulty, nystagmus, bilateral lateral rectus palsy and fluctuating orientation for ten days, along with deficits in recent memory. He had a history of poor nutrition for three months, diarrhoea for one month, and severe diffuse headache and slurred speech for ten days. Plain MRI-Brain revealed an acute non-haemorrhagic left thalamic infarct and chronic infarct/early gliosis in the left cerebellar hemisphere and left temporal lobe. Contrast-enhanced MR-Venography revealed cerebral venous sinus thrombosis. The patient showed rapid improvement with high-dose parenteral thiamine and low molecular weight heparin and is currently maintaining well on 3 milligrams of warfarin.

INTRODUCTION

Cerebral venous sinus thrombosis is a rare condition that includes thrombosis in cerebral veins or the dural sinuses. It has a highly variable presentation, including headaches, seizures and altered mental states. Any pro-thrombotic state, such as pregnancy, puerperium, oral contraceptive intake, or hormone replacement therapy, may precipitate the condition. Because of these gender-specific risk factors, the prevalence is three times higher among females compared to males.[1] Multiple studies have reported that alcoholism leads to dehydration and blood hyperviscosity, thereby becoming a unique risk factor for the condition among males with chronic alcoholism.[2-5] In any case of chronic alcoholism, Wernicke's encephalopathy (WE) is a severe but easily treatable metabolic disorder that must be looked for, since up to 80% of cases go undiagnosed and may lead to irreversible memory impairment if untreated, and the classic triad of symptoms appears in only 16–33% of cases.[6,7]

This case report focuses on the importance of considering these two entities and differentiating between them while evaluating patients presenting with chronic alcohol consumption.

Case Presentation

A man in his mid-40s presented to the psychiatry OPD along with his wife with complaints of difficulty walking, blurred vision and fluctuating orientation for 10 days associated with on-and-off severe diffuse headache. He also had a history of poor oral intake and frequent diarrhoea for one month. Ten days prior, he had two episodes of blood-tinged vomiting and agitation. The patient had a history of intake of alcohol intake for 10 years, with average consumption being 90 mL of Indian-made foreign liquor 2 to 3 times per week. The drinking became regular 5 years back, consuming 180 ml of whiskey every evening with friends due to an irresistible desire, especially after work. For the last two years, he also reported withdrawal symptoms. For the past three months, he had been trying to quit alcohol. At the time of presentation, he had been off alcohol for at least 10 days. The patient's past medical or psychiatric history was insignificant.

On admission, the patient's vital signs were within normal limits, and blood oxygen saturation was well above 97% in room air. His BMI was 24.6 kg/m^2. He was oriented to time, place and person but could not recollect events of the recent past. On mental status examination, the patient was apathetic, had slurred speech, mildly irritable affect, no thought/perceptual abnormalities, good insight into his illness and had a contemplation stage of motivation for alcohol use.

He was responding normally to verbal commands. He had horizontal nystagmus in both eyes, along with bilateral lateral rectus palsy. There was a mild blurring of disc margins on fundus examination. Power examination revealed Medical Research Council (MRC) grade 5/5 in all four limbs. All deep tendon reflexes were brisk. Bilateral plantar was equivocal. He was unable to walk on his own on the day of admission, but was able to do so with support.

Assessment for alcohol dependence and withdrawal: At the time of admission, he scored 8 (Anxiety-3; Headache-5) on the CIWA-Ar (Clinical Institute Withdrawal Assessment-Alcohol Revised) scale. The patient also scored 15 (mild dependence) on the Severity of Alcohol Dependence Questionnaire (SAD-Q) applied on the 4th day post-admission.

Investigations

Routine investigations showed raised MCV (107.5fL), raised TSH (5.8 mIU/L) and raised FBS (149.7 mg/dl), PPBS (314.5 mg/dl) and HbA1C (7.70%), and low vitamin D3 (8.88 ng/mL). Liver function tests were grossly within normal limits. Serum magnesium was normal (1.95 mg/dL). Serum homocysteine level was raised (28.30 µmol/L). MRI brain (plain) was done, which revealed an acute non-haemorrhagic infarct in the left thalamus (Figure 1C & 1D), chronic infarct/early gliosis with hemosiderin staining in the left cerebellar hemisphere and the left temporal lobe (Figure 1A), and loss of flow void in bilateral sigmoid sinuses and left transverse sinus. Due to the presence of a few red flags such history of headache and vomiting suggesting features of raised intracranial pressure, and the presence of apathetic attitude, slurred speech and mildly irritable affect which could not be explained by Wernicke's Encephalopathy alone, MR-venography with contrast was done which revealed attenuation of flow-related enhancement in the left transverse sinus, sigmoid sinus and internal jugular vein and a patchy filling defect in the anterior aspect of the superior sagittal sinus suggestive of venous thrombosis (Figure 1B).

Neuropsychological Assessment

A neuropsychological assessment of the patient was done using the NIMHANS (National Institute of Mental Health and Neurosciences) Neuropsychological battery on day four of admission. The patient scored low on verbal fluency (6.33 on the Controlled Oral Word Association Test) and categorical fluency (08 words on the animal naming test). The patient's scores on other domains of the test were within normal limits.

Diagnosis

Initially, the presence of a classic triad of gait disturbance, eye signs and fluctuating orientation against the backdrop of chronic alcohol intake and
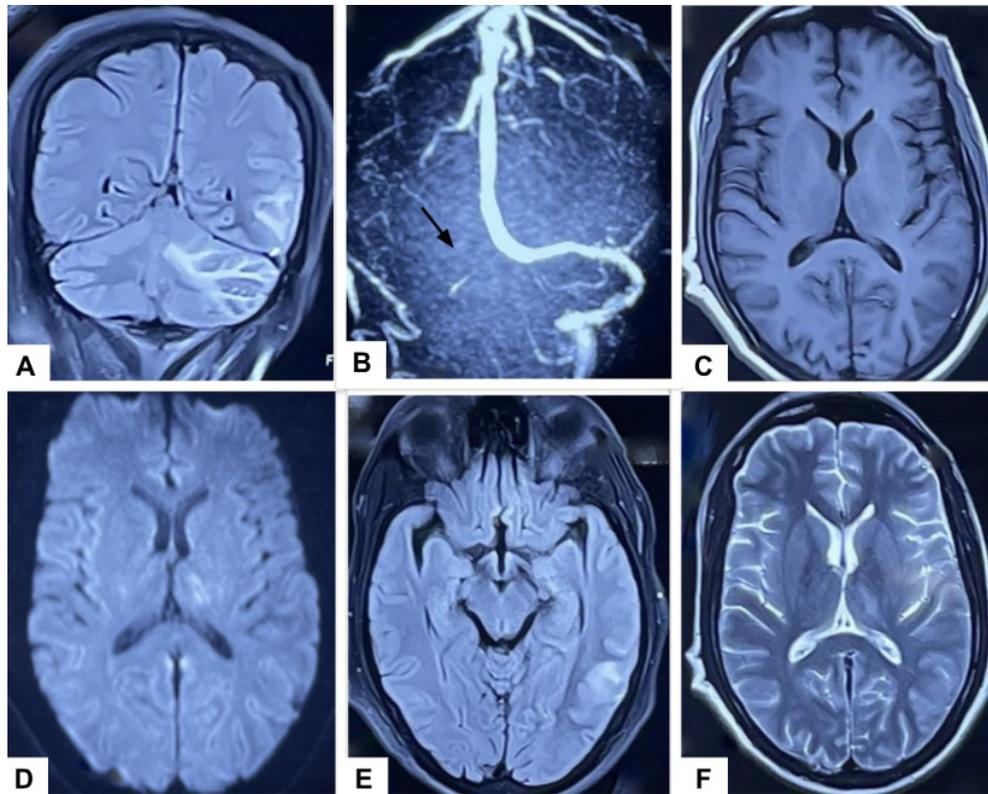
Figure 1: MRI Brain showing an acute non-haemorrhagic infarct in the left thalamus (C, D), chronic infarct/early gliosis with hemosiderin staining in the left cerebellar hemisphere and the left temporal lobe (A), and loss of flow void in bilateral sigmoid sinuses and left transverse sinus. Contrast-enhanced MR-Venography showing attenuation of flow-related enhancement in the left transverse sinus, sigmoid sinus and internal jugular vein and a patchy filling defect in the anterior aspect of the superior sagittal sinus, suggestive of venous thrombosis (B).

poor nutrition was highly suggestive of Wernicke's encephalopathy as per Caine's criteria. So, a provisional diagnosis of alcohol dependence syndrome and Wernicke's encephalopathy was made. However, after radiological evidence failed to show Wernicke's encephalopathy lesions, but instead showed venous sinus thrombosis and thalamic infarct, the diagnosis was appropriately revised to alcohol dependence syndrome and Cerebral venous sinus thrombosis (CVST) and Thalamic Infarct.

Treatment

The patient was started on high-dose parenteral thiamine 1500 mg/day, along with an injection of pantoprazole. Benzodiazepine was added on prn basis for mild anxiety and sleep. Following neurology referral and diagnosis of cerebral venous thrombosis, he was started on injection low molecular weight heparin (LMWH) 0.6 mL twice daily along with tablet warfarin 2 mg in the evening. The dose of tablet warfarin was optimised until the target INR was achieved, and LMWH was stopped after five days. The patient's INR was maintained on 3 milligrams of tablet warfarin. After the initial 5 days, the dose of parenteral thiamine was decreased to 750 mg/day for another five days, after which it was switched to oral formulation, 300 mg/day. Tab levothyroxine 25 mcg and tab metformin 500 + Glimepiride 1mg were started to manage his hyperglycemia and hypothyroidism. Vitamin D3 supplementation was also given. The headache subsided from the second day of admission, while his ocular signs almost completely resolved by the 5th day. From the 3rd day, he could walk on his own but had a prominent, wide-based ataxic gait with a tendency to sway on the left side, which also gradually resolved completely in 2 weeks. He was discharged after 2 weeks with residual symptoms of complaints of intermittent headache on the right temporal area lasting for 1 to 2 hours per day, mild in intensity.

Outcome and Follow-up

The patient was discharged on day 14 of admission. His only lingering symptom was a mild intermittent headache. The patient's warfarin was maintained at 3 mg with an INR of 2.9. Thiamine was continued, and so were the medications for diabetes and hypothyroidism. The patient has been regular with his follow-up for the last two months and has been abstinent from alcohol use.

Discussion

At face value, the patient had all the risk factors for developing Wernicke's Encephalopathy: chronic alcohol use, poor nutrition, and recurrent diarrhoea. Despite lacking typical radiological features, the history and symptoms still warranted high-dose thiamine (1500 mg/day for five days), which was duly given to the patient with no delays, as an untreated deficiency could lead to severe cellular damage resulting in permanent Korsakoff's syndrome or even death.[8] This is because thiamine is an essential cofactor in the TCA cycle and pentose-phosphate pathway, and its deficiency impairs ATP, DNA/RNA, and NADPH synthesis, leading to toxic metabolite accumulation.[9]

Our patient had cerebral venous sinus thrombosis and a left thalamic infarct. Nystagmus and gait disturbance could have resulted from the cerebellar involvement. The decreased verbal and categorical fluency test scores, the apathetic attitude and recent memory impairment were typical of left-sided thalamic infarct.[10] The involvement of multiple brain regions could explain all the symptoms in the patient, but the important question remains: what could have been primarily responsible for the cerebrovascular event? Especially given the fact that the patient had no history of any significant medical condition.

Elevated homocysteine level in the patient points to a pro-thrombotic state.[11] From as early as 1983, alcohol intake has been shown to increase blood viscosity.[2] A more recent study was conducted by Saribal D. 2019 among 20 males with chronic alcohol consumption and 20 age- and sex-matched controls.[4] He found that the mean blood viscosity of the study group was significantly higher than the mean viscosity of the control group. Alcohol causes blood hyperviscosity by increasing serum osmolality, which results in the shrinkage of red blood cells. Kumari and colleagues (2005) suggested that decreased blood fibrinogen causes blood hyperviscosity, while Mukamal and colleagues. 2001 claimed that alcohol can decrease blood fibrinogen levels.[12,13] Other mechanisms, such as decreased synthesis of thromboxane A2 and increased lipids & cholesterol by alcohol, also play a role.[14,15]

In 2014, a case of Wernicke's Encephalopathy mimicking stroke was reported by Bhan and colleagues. Our case seems to be the exact reverse of the above case report: a patient with chronic alcoholism presenting with a clinical history pointing strongly towards Wernicke's encephalopathy but no radiological evidence to support it, and instead showed evidence of stroke. Nonetheless, the case offers a unique learning point: how alcohol uniquely predisposes a patient with chronic alcoholism to develop cerebral venous sinus thrombosis by way of dehydration and increased blood viscosity, and as such, the possibility of comorbid cerebral venous thrombosis must always be entertained in chronic alcoholic patients.

Patient's Perspective

I was having a headache when I was brought to the hospital, and did not feel like talking with anyone. But the headache went away after a couple of days. I used to drink alcohol every day for the last 5 years or so. I was not drinking too much, only about 180 ml of whiskey in the evening. But I was largely unable to control myself from drinking, as I used to drink in the company of colleagues at the workplace. I was working as a supervisor for cooks at a canteen of an engineering college in Gorakhpur, while my family was back home in Deoria. I had not been eating well for the last 3 months, and also had frequent loose stools for 1 month. But I did not think it was anything serious, and as my family was not with me, I did not care to visit the hospital sooner. Then I think it was about 2 weeks back, one night I had a terrible headache and vomited as well. And the vomit even contained some blood on it. Thereafter, what happened after that is hazy in my memory. But I remember being brought here, the hospital.

I could not walk on my own, and my vision was all blurred. But with treatment, I gradually recovered. First thing, my headache subsided after a couple of days, and after a few more days, my vision improved. And towards the time of discharge, I was able to walk steadily on my own. I feel like I have been brought back from the depths of despair. I agree that alcohol played a major role in this illness, and from now I give my word that I will never touch alcohol again. I will also change my profession. I will stay at home with my family in Deoria and look for another job. I can open up a roadside food truck.

References

1. Ranjan R, Ken-Dror G, Sharma P. Pathophysiology, diagnosis and management of cerebral venous thrombosis: A comprehensive review. Medicine (Baltimore). 2023 Dec 1;102(48):e36366. Available from: doi. 10.1097/MD.0000000000036366.

2. Hamazaki T, Shishido H. Increase in blood viscosity due to alcohol drinking. Thromb Res. 1983 Jun 15;30(6):587-94. Available from: doi. 10.1016/0049-3848(83)90267-0.

3. Reinhart WH, Piety NZ, Goede JS, Shevkoplyas SS. Effect of osmolality on erythrocyte rheology and perfusion of an artificial microvascular network. Microvasc Res. 2015 Mar;98:102-7. Available from: doi. 10.1016/j.mvr.2015.01.010.

4. Saribal D. Blood viscosity of individuals with alcohol abuse. Ann Med Res. 2019;26(4):695-7.

5. Mathew T, Sarma GR, Kamath V, Roy AK. Subdural hematoma, subarachnoid haemorrhage and intracerebral parenchymal haemorrhage secondary to cerebral sinovenous thrombosis: a rare combination. Neurol India. 2007 Oct-Dec;55(4):438-9. Available from: doi. 10.4103/0028-3886.37110.

6. Day E, Bentham P, Callaghan R, Kuruvilla T, George S. Thiamine for Wernicke-Korsakoff Syndrome in people at risk from alcohol abuse. Cochrane Database Syst Rev. 2004;(1): CD004033. Available from: doi. 10.1002/14651858.CD004033.

7. Chandrakumar A, Bhardwaj A, 't Jong GW. Review of thiamine deficiency disorders: Wernicke encephalopathy and Korsakoff psychosis. J Basic Clin Physiol Pharmacol. 2018 Oct 2;30(2):153-162. Available from: doi. 10.1515/jbcpp-2018-0075.

8. Hazell AS, Todd KG, Butterworth RF. Mechanisms of neuronal cell death in Wernicke's encephalopathy. Metab Brain Dis. 1998 Jun;13(2):97-122. Available from: doi. 10.1023/a:1020657129593. PMID: 9699919.

9. Hazell AS, Butterworth RF. Update of cell damage mechanisms in thiamine deficiency: focus on oxidative stress, excitotoxicity and inflammation. Alcohol Alcohol. 2009 Mar-Apr;44(2):141-7. Available from: doi. 10.1093/alcalc/agn120. Epub 2009 Jan 16.

10. Chen XY, Wang Q, Wang X, Wong KS. Clinical Features of Thalamic Stroke. Curr Treat Options Neurol. 2017 Feb;19(2):5. Available from: doi. 10.1007/s11940-017-0441-x.

11. Kamat PK, Mallonee CJ, George AK, Tyagi SC, Tyagi N. Homocysteine, Alcoholism, and Its Potential Epigenetic Mechanism. Alcohol Clin Exp Res. 2016 Dec;40(12):2474-2481. Available from: doi. 10.1111/acer.13234.

12. Kumari M, Marmot M, Rumley A, Lowe G. Social, behavioural, and metabolic determinants of plasma viscosity in the Whitehall II Study. Ann Epidemiol. 2005 May;15(5):398-404. Available from: doi. 10.1016/j.annepidem.2005.01.004.

13. 13. Mukamal KJ, Jadhav PP, D'Agostino RB, Massaro JM, Mittleman MA, Lipinska I, Sutherland PA, Matheney T, Levy D, Wilson PW, Ellison RC, Silbershatz H, Muller JE, Tofler GH. Alcohol consumption and hemostatic factors: analysis of the Framingham Offspring cohort. Circulation. 2001 Sep 18;104(12):1367-73. Available from: doi. 10.1161/hc3701.096067.

14. 14. Mikhailidis DP, Barradas MA, Jeremy JY. The effect of ethanol on platelet function and vascular prostanoids. Alcohol. 1990 Mar-Apr;7(2):171-80. Available from: doi. 10.1016/0741-8329(90)90080-v.

15. 15. Wakabayashi I. Alcohol intake and triglycerides/high-density lipoprotein cholesterol ratio in men with hypertension. Am J Hypertens. 2013 Jul;26(7):888-95. Available from: doi.10.1093/ajh/hpt033.

Beyond Uniform Narratives: Rethinking the Mental Health Impact of War through Diversity, Gaps, and Future Directions

Raghavendra Kukkehalli[1], Chaithra KC[2]

[1]*PhD scholar, Department of Psychosocial Support in Disaster Management, National Institute of Mental Health and Neuro Sciences, Bangalore, India.*
[2]*Tele MANAS Counsellor, Department of Psychiatry, National Institute of Mental Health and Neuro Sciences (NIMHANS), Bengaluru, India.*

Dear Editors,

The article titled Beyond the battlefield: understanding the mental health impact of war, is very appropriate in the current context.[1] This article highlighted very important issues that the impact of war not only affects the physical health and economics of the country but also causes long-term damage to mental health. Typically, the experience of war is assessed based on the sacrifices of soldiers, battles fought along national borders, and political victories and defeats. But this article goes beyond that limited perspective. The article strongly emphasizes how the war impacts the mental health and well-being of people in society, including cohesion, child development, education, the health system, employment opportunities, and the long-term stability of the community. From this perspective, the article possesses its own uniqueness, and I sincerely appreciate the authors' contribution.

One of the article's greatest strengths is its refusal to confine mental health to a narrow clinical framework. Instead, it boldly examines the broader social determinants that shape mental well-being in conflict zones. The article convincingly demonstrates that the consequences of war go far beyond PTSD, depression, or anxiety; they can devastate healthcare systems, shut down schools, destroy jobs, drive poverty and displacement, and perpetuate intergenerational deprivation. This holistic perspective aligns perfectly with global conversations on social determinants of health and delivers an urgent message: war is a fundamental driver of mental health crises. Furthermore, the authors' focus on vulnerable populations, children, women, the elderly, people with disabilities, minorities, and communities most affected by conflict rightly centres the discussion on equity and justice.

The article also stressed how news in the media about war influences people to have serious mental health issues. The news about the war can lead to some kind of fear, anxiety, and psychological instability among the common people,

which can reduce the helping nature among the people.

The author also highlighted that the accessibility of the mental health service during war is very difficult. The author mentioned that the significant challenges of accessing healthcare services during the war, such as the demolition of healthcare infrastructure, distrust in medical institutions, the lack of qualified professionals, and financial constraints, are substantial barriers. That is how the authors emphasize to the readers the necessity of addressing these issues.

The article also focused on digitalized interventions for war survivors. The article also highlighted the importance of unique digitalized interventions for the survivors of wars. The article provided the example of chatbots in Ukraine, which offered mental health services. Additionally, this article mentioned the WHO's 'Doing What Matters in Times of Stress' (DWM). Digital guides empower individuals psychologically, even in times of crisis. These initiatives enhance a person's capacities through practices of mindfulness, learning, engagement in activities, and the restoration of social connection. In reality, in refugee camps or regions affected by terrorism where expert doctors, counsellors, and psychiatrists are not available, such digital solutions can at least provide a minimum level of psychosocial support to people. Recent studies also indicate that more than 80% of users have found chatbots to be useful, providing reassurance to readers. The main strength of the article focuses on the consequences of the wars, and it's not limited to clinical perspectives, but also encompasses social, economic, and educational aspects. Analysing through environmental factors is particularly distinctive. The authors have clearly explained the diverse impacts of war on women, minority communities, youth, and children. This raises awareness among readers and encourages community support.

The article tends to generalize the war experience. Not every war produces the same effects. For example, the border conflict in Ukraine, the civil war in Sudan, the prolonged conflict in Gaza, and the internal political unrest in Kashmir all have distinct psychological consequences.[2] The distress experienced by urban residents exposed to bomb blasts cannot be equated with the suffering of rural refugees. The article does not sufficiently reflect this diversity. As a result, readers may come to perceive the term 'war' as representing a uniform experience.[3] There is a lack of in-depth discussion on the experiences of minorities, women, children, and persons with disabilities.[4] In reality, women are more likely to experience sexual violence, forced displacement, heavy family responsibilities, and inadequate access to health care. Children lose access to education, their social networks are disrupted, and their long-term development is hindered. Rather than being addressed only in brief paragraphs, these issues required deeper exploration. The article does not adequately discuss the limitations of digital initiatives. In war-affected regions, the lack of electricity, limited internet connectivity, language barriers, and low digital literacy levels pose significant challenges. In addition, data privacy concerns, information security, misuse of apps, and lack of cultural adaptability are pressing issues. By overlooking these, the article appears overly optimistic. The recommendations remain very general. Suggestions such as 'rebuilding hospitals' or 'restoring social bonds' are valuable, but the article does not clarify how these can be practically implemented. For instance, training nurses and doctors in primary health centres to provide psychosocial support, setting up counselling rooms in schools, developing digital applications in local languages, and involving refugee communities in decision-making processes would constitute more concrete and actionable recommendations[5] with a specific focus on healthcare workers (HCWs.

Despite these gaps, the article has initiated an important discussion. Therefore, some points may be suggested for future research and writing. First, longitudinal studies are necessary, as the effects of war may not appear immediately but can persist for years. Second, implementation science should be employed to examine which interventions succeed in particular contexts and how they can be adapted to local cultures.[6] Third, involving refugee communities, local women's associations, and youth groups at the planning stage is essential. This would make interventions more credible and sustainable.

One of the recent analysis of the anti-quota movement in Bangladesh in 2024, support the

argument presented in beyond the battlefield. Even though the incident did not happen in a conventional war zone, civilians experienced equivalent psychological consequences, including anxiety, fear, depression and post-traumatic stress. The curfew, violence and communication blackouts disrupted safety, social connectedness and access to important mental health services. The findings highlight that the mental health toll of collective violence outspreads beyond armed conflict, disturbing populations exposed to political unrest and human rights violations. Integrating such civilian perspectives into discussions on war related mental health can strengthen trauma informed and inclusive psychosocial interventions.

In summation, *Beyond The Battlefield: Understanding The Mental Health Impact of War* has several strengths: a comprehensive perspective, recognition of digital interventions, and acowledgement of the long-term struggles of civilians. Yet, its weaknesses are also evident in insufficient depth of evidence, neglect of the diversity of wars, inadequate discussion of minority hardships, omission of the limitations of digital initiatives, and vague recommendations. If future works address these shortcomings, they can better position mental health as central to post-war reconstruction. Mental health should be understood as a right as fundamental as physical health, food, and shelter. Policymakers, researchers, and prac-titioners must therefore develop integrated action plans. War is not only fought across borders; it is also a prolonged battle within the human mind. Ensuring that we do not lose this battle is a responsibility shared by us all.

References

1. Osokina O, Silwal S, Bohdanova T, Hodes M, Sourander A, Skokauskas N. Impact of the Russian Invasion on Mental Health of Adolescents in Ukraine. J Am Acad Child Adolesc Psychiatry. 2023 Mar 1;62(3):335–43.
2. Kurapov A, Kalaitzaki A, Keller V, Danyliuk I, Kowatsch T. The mental health impact of the ongoing Russian-Ukrainian war 6 months after the Russian invasion of Ukraine. Front Psychiatry. 2023 July 27;14:1134780.
3. Xu W, Pavlova I, Chen X, Petrytsa P, Graf-Vlachy L, Zhang SX. Mental health symptoms and coping strategies among Ukrainians during the Russia-Ukraine war in March 2022. Int J Soc Psychiatry. 2023 June;69(4):957–66.
4. Hussein MF, Saleeb M, Tolba B, Mohamed YY, Gebreal A, Mohamed AOA, et al. Assessment of post-traumatic stress disorder and well-being among Sudanese during the ongoing war: a cross-sectional study. BMC Psychol. 2025 Mar 2;13(1):181.
5. Polyvianaia M, Yachnik Y, Fegert JM, Sitarski E, Stepanova N, Pinchuk I. Mental health of university students twenty months after the beginning of the full-scale Russian-Ukrainian war. BMC Psychiatry. 2025 Mar 12;25(1):236.
6. Arafat SMY, Kar SK, Kabir R, Hossain MS. Mental health challenges during mass trauma: Insights from the July mass uprising (2024) in Bangladesh. Geopsychiatry. 2025 Dec 1;2:100025.

Cannabidiol (CBD) in Psychiatry: Therapeutic Potential and Challenges in the Indian Context

Shobit Garg[1*], E. Mohandas[2]

[1]Department of Psychiatry, Shri Guru Ram Rai Institute of Medical and Health Sciences, Shri Guru Ram Rai University, Dehradun, Uttarakhand – 248001 India.
[2]Sun Medical and Research Centre, Thrissur, Kerala, India.

Abstract

Cannabidiol (CBD), a non-psychoactive component of the *Cannabis sativa* plant, is attracting increased attention for its potential therapeutic applications across various medical fields, including psychiatry. This viewpoint explores the current evidence regarding CBD's effectiveness and safety in treating psychiatric disorders, while also addressing the regulatory challenges and the need for further research, specifically within the Indian context. The discussion emphasizes the unique barriers faced by Indian researchers due to historical and legal factors, including colonial-era prohibition and modern regulatory ambiguity. The article discusses the pharmacological profile of CBD, its potential mechanisms of action, and the available clinical evidence for its use in conditions such as anxiety disorders, schizophrenia, and other neuropsychiatric conditions. It also highlights the complexities surrounding the legal status of cannabis in India and the implications for CBD research and clinical practice.

Introduction

The *Cannabis sativa* plant has a long and complex history, with applications ranging from ancient traditional medicine to modern-day therapeutic uses. While delta-9-tetrahydrocannabinol (THC), the primary psychoactive component of cannabis, has been extensively studied, cannabidiol (CBD), a non-psychoactive cannabinoid, is now at the forefront of research, demonstrating promise in various medical fields, including psychiatry.[1] This viewpoint aims to provide an overview of the current evidence on CBD's therapeutic potential in psychiatry, with a specific focus on the Indian context, where the legal and regulatory landscape surrounding cannabis remains intricate. In India, where cannabis has deep roots in Ayurvedic traditions but faces modern prohibition under the NDPS Act, CBD offers a promising yet under-explored avenue for mental health interventions.[2,3]

Pharmacology and Mechanisms of Action

CBD is a complex molecule with a unique pharmacological profile. Unlike THC, CBD exhibits low affinity for the canonical cannabinoid receptors CB1 and CB2. Instead, it interacts with a range of other receptors and ion channels, includ-

ing the transient receptor potential vanilloid type 1 (TRPV1) receptor, the 5-HT1A serotonin receptor, and G-protein coupled receptor 55 (GPR55).[4,5] These interactions are believed to underlie CBD's diverse effects, including its anxiolytic, anti-inflammatory, and anti-seizure properties.[5]

The precise mechanisms by which CBD exerts its therapeutic effects in psychiatric disorders are not fully elucidated, but several hypotheses have been proposed. CBD may modulate the endocannabinoid system (ECS), a complex network of endogenous cannabinoids, receptors, and enzymes that plays a crucial role in regulating various physiological and psychological processes. CBD may enhance the levels of endocannabinoids, such as anandamide, by inhibiting their degradation.[1,4] Additionally, CBD's interaction with the 5-HT1A receptor may contribute to its anxiolytic and antidepressant effects. Emerging evidence suggests that these mechanisms could be particularly relevant in diverse populations, including those in India with varying genetic polymorphisms affecting endocannabinoid metabolism.[6]

Evidence in Psychiatric Disorders

The evidence for CBD's effectiveness in treating psychiatric disorders is still evolving, with much of the research being preliminary. However, several studies have shown promising results in specific areas:

Anxiety Disorders

CBD has been most extensively studied for its potential in treating anxiety disorders. Multiple preclinical studies and some clinical trials suggest that CBD may reduce anxiety symptoms in conditions such as generalized anxiety disorder (GAD), social anxiety disorder (SAD), and panic disorder. A systematic review of preclinical and clinical studies concluded that CBD may have potential as a treatment for anxiety disorders.[7] Recent meta-analyses reinforce this, showing moderate effect sizes in real-world settings, though limited by variability in dosing and administration routes.[8]

Insomnia Disorder

Randomized controlled trials (RCTs) have shown that CBD alone or in a CBD:THC combination (1:1) may be beneficial in alleviating the symptoms of insomnia disorder.[9] However, long-term studies are lacking to assess dependency risks, particularly given the potential for tolerance with prolonged cannabinoid use.[10]

Schizophrenia

Preliminary studies suggest that CBD may have antipsychotic properties. Some clinical trials have investigated CBD as an adjunct to antipsychotic medication in patients with schizophrenia, with mixed results. While some studies have shown a reduction in psychotic symptoms, others have not found significant benefits.[11] More research is needed to determine the role of CBD in schizophrenia treatment.

Other Neuropsychiatric Conditions

CBD is also being investigated for its potential in other neuropsychiatric conditions, including:

Substance use disorders (SUD)

Some preclinical and clinical studies suggest that CBD (not alone but in combination with THC) may reduce craving and withdrawal symptoms in substance use disorders, including cannabis, opioid, and tobacco use disorders.[12] In the Indian context, where opioid misuse is prevalent, this could have significant public health implications, especially given the rising rates of substance dependence.[13]

Attention deficit hyperactivity disorder (ADHD)

There is limited evidence to support the use of CBD in ADHD, and more research is needed in this area.[14]

Autism spectrum disorder (ASD)

Some studies have explored the use of CBD in children with ASD, with some showing improvements in behavioral symptoms such as irritability and anxiety.[14]

Post-traumatic stress disorder (PTSD)

Preliminary research suggests that CBD may help to reduce symptoms of PTSD, such as nightmares and anxiety, though more robust clinical trials are needed.[15]

Bipolar disorder and OCD

The evidence for CBD use in bipolar disorder and OCD is limited. Additional trials focusing on these disorders are urgently required to fill evidence gaps, as current studies lack sufficient statistical power.[16]

It is important to note that much of the evidence for CBD's effectiveness in psychiatric disorders is based on small sample sizes and preliminary studies. Larger, well-designed clinical trials are needed to confirm these findings and establish the optimal dosage, route of administration, and long-term safety of CBD treatment. Limitations such as publication bias and heterogeneous study designs further complicate interpretations, necessitating rigorous methodological improvements.[17]

Safety and Tolerability

CBD is generally considered to be safe and well-tolerated, with relatively few side effects reported in clinical trials. The most commonly reported side effects include drowsiness, diarrhea, changes in appetite, and fatigue. However, it is important to note that the long-term safety of CBD use is still unknown, and more research is needed to assess its potential for drug interactions and other adverse effects.[18] In particular, interactions with commonly used psychiatric medications in India, such as benzodiazepines, warrant careful investigation due to potential cytochrome P450 enzyme inhibition.[19]

Regulatory and Legal Challenges in India

In India, the legal status of cannabis is governed by the Narcotic Drugs and Psychotropic Substances (NDPS) Act of 1985.[20] The NDPS Act defines "cannabis" in a specific way, which has significant implications for the legality of cannabis and its derivatives, including CBD.

While the NDPS Act does not explicitly prohibit the use of CBD derived from the seeds and leaves of the cannabis plant, the regulatory landscape remains complex and somewhat ambiguous. There is a lack of clear guidelines regarding the production, distribution, and sale of CBD products in India. This ambiguity creates challenges for both researchers and clinicians who are interested in exploring the therapeutic potential of CBD.[20] Recent advocacy efforts by medical bodies in India, such as the Indian Psychiatric Society, call for amendments to facilitate research while maintaining stringent oversight.[21]

Recently, the Food Safety and Standards Authority of India (FSSAI) has taken steps to regulate the use of hemp seed and hemp seed products, allowing their use as food as long as they contain less than 0.3% THC. However, the regulations surrounding CBD derived from other parts of the cannabis plant remain unclear.[22]

The Need for Further Research

There is a significant need for further research on CBD, particularly within the Indian context. This research should focus on several key areas:

Well-designed clinical trials

Large-scale, randomized, controlled trials are needed to evaluate the effectiveness and safety of CBD in treating various psychiatric disorders in the Indian population.

Pharmacokinetic and pharmacodynamic studies

Studies are needed to determine the optimal dosage, route of administration, and metabolism of CBD in Indian patients.

Exploration of indigenous knowledge

India has a long history of traditional use of cannabis in Ayurvedic medicine. Research should explore this indigenous knowledge to identify potential therapeutic applications of CBD.

Regulatory clarity

The Indian government needs to provide clear and comprehensive regulations regarding the production, distribution, and sale of CBD products to facilitate research and clinical use. Collaborations between academia, industry, and policymakers are essential to advance this agenda, leveraging public-private partnerships to streamline regulatory approvals.[23]

Conclusion

CBD has potential for treating psychiatric disorders like anxiety, insomnia, and SUDs, while noting that

more research is needed to confirm its effectiveness and safety. In India, the complex cannabis regulations pose challenges for CBD research and clinical use. Further investigation and clearer regulations are necessary to fully explore CBD's therapeutic benefits within the Indian context. A balanced, evidence-based approach is crucial, considering India's specific regulatory environment, as understanding of CBD advances. Ultimately, integrating CBD into psychiatric practice in India could revolutionize mental health care, provided that cultural, legal, and scientific hurdles are addressed proactively through collaborative and culturally sensitive research efforts.[24]

References

1. Mechoulam R, Parker LA, Abrams RM. Cannabidiol: an overview of some pharmacological aspects. Journal of Clinical Pharmacology. 2002;42(S1):11S-19S.
2. Chandra S, Radwan MM, Majumdar CG, Church JC, Freeman TP, ElSohly MA. New trends in cannabis potency in USA and Europe during the last decade (2008–2017). European Archives of Psychiatry and Clinical Neuroscience. 2019;269(1):5–15.
3. Sharma P, Murthy P. Cannabis use in India: Historical perspectives and contemporary challenges. Indian Journal of Psychiatry. 2020;62(5):483–490.
4. Zuardi AW. Cannabidiol: from an inactive cannabinoid to a drug with wide pharmacological potential. Brazilian Journal of Medical and Biological Research. 2008;41(8):664-670.
5. Watkins AR. Cannabinoid interactions with ion channels and receptors. Channels (Austin, Tex.). 2019;13(1):162–167.
6. Gaston TE, Szaflarski JP. Cannabis for the treatment of epilepsy: An update. Current Neurology and Neuroscience Reports. 2018;18(11):73.
7. Han K, Wang JY, Wang PY, Peng YC. Therapeutic potential of cannabidiol (CBD) in anxiety disorders: A systematic review and meta-analysis. Psychiatry Research. 2024;339:116049.
8. Skelley JW, Deas CM, Curren Z, Ennis J. Use of cannabidiol in anxiety and anxiety-related disorders. Journal of the American Pharmacists Association. 2020;60(1):253–261.
9. Ranum RM, Whipple MO, Croghan I, Bauer B, Toussaint LL, Vincent A. Use of cannabidiol in the management of insomnia: A systematic review. Cannabis and Cannabinoid Research. 2023;8(2):213–229.
10. Freeman TP, Lorenzetti V. A standard THC unit for the regulation and monitoring of cannabis products. Addiction. 2020;115(2):199–201.
11. Kopelli E, Samara M, Siargkas A, Goulas A, Papazisis G, Chourdakis M. The role of cannabidiol oil in schizophrenia treatment: A systematic review and meta-analysis. Psychiatry Research. 2020;291:113246.
12. Redonnet B, Eren F, Avenin G, Melchior M, Mary-Krause M. Efficacy of cannabidiol alone or in combination with Δ-9-tetrahydrocannabinol for the management of substance use disorders: An umbrella review of the evidence. Addiction (Abingdon, England). 2025;120(5):813–834.
13. Ambekar A, Agrawal A, Rao R, Mishra AK, Khandelwal SK, Chadda RK. Magnitude of substance use in India 2019. Ministry of Social Justice and Empowerment, Government of India; 2019.
14. Parrella NF, Hill AT, Enticott PG, Barhoun P, Bower IS, Ford TC. A systematic review of cannabidiol trials in neurodevelopmental disorders. Pharmacology, Biochemistry, and Behavior. 2023;230:173607.
15. Rehman Y, Saini A, Huang S, Sood E, Gill R, Yanikomeroglu S. Cannabis in the management of PTSD: A systematic review. AIMS Neuroscience. 2021;8(3):414–434.
16. Black N, Stockings E, Campbell G, Tran LT, Zagic D, Hall WD, et al. Cannabinoids for the treatment of mental disorders and symptoms of mental disorders: A systematic review and meta-analysis. The Lancet Psychiatry. 2019;6(12):995–1010.
17. Whiting PF, Wolff RF, Deshpande S, Di Nisio M, Duffy S, Hernandez AV, et al. Cannabinoids for medical use: A systematic review and meta-analysis. JAMA. 2015;313(24):2456–2473.
18. Iffland K, Grotenhermen F. An update on safety and side effects of cannabidiol: A review of clinical data and relevant animal studies. Cannabis and Cannabinoid Research. 2017;2(1):139–154.
19. Zendulka O, Dovrtělová G, Nosková K, Turjap M, Šulcová A, Hanuš L, et al. Cannabinoids and cytochrome P450 interactions. Current Drug Metabolism. 2016;17(3):206–220.
20. Parliament of India. The Narcotic Drugs and Psychotropic Substances Act, 1985 (Act No. 61 of 1985). 1985.
21. Indian Psychiatric Society. Position statement on cannabis and mental health. Indian Journal of Psychiatry. 2023;65(3):345–347.
22. Food Safety and Standards Authority of India. Regulation on Hemp Seed and Products. 2021.
23. Rathi A, Rathi S. Public-private partnerships in Indian healthcare: Addressing the cannabis research gap. Journal of Health Management. 2021;23(4):567–574.
24. Singh OP. Cannabis and mental health: A call for culturally sensitive research in India. Indian Journal of Psychological Medicine. 2022;44(1):1–3.

Author Guidelines

Guidelines for Authors

We encourage researchers, academicians, clinicians and students to contribute scientific articles for the Indian Journal of Clinical Psychiatry (IJOCP).
- Scope of the journal: We publish articles related to psychiatry and allied disciplines.
- Types of articles:

Type of article	Abstract	Word limit for text	References
1. Editorial	Not required	2000	15
2. Review article	Required (Unstructured)	6000	No limit
3. Original research article	Required (Structured)	4000	40
4. Case report/Series	Required (Unstructured)	1500	15
5. Letter to the Editor	Not required	1000	10
6. View point/ Opinion/ Perspectives	Not required	2000	20
7. Debate	Required (Unstructured)	4000	40
8. Book review	Not required	1500	10
9. Others (Art & mental health, UG corner, Announcements)	Not required	1500	15

The **"Article Text"** document should not have any identification data such as Author details, affiliations, sequence, and ack--nowledgment.

Author details must be shared in a separate file

Specific Instructions for Individual Categories of Articles

EDITORIAL

Editorial is to be written by the editor or the editorial team. However, the journal may invite guest editorials from prominent researchers in the field of psychiatry.

REVIEW ARTICLE

Review articles are usually unsolicited. It can be a narrative, scoping and systematic review related to any contemporary issue of psychiatry and allied sciences.

ORIGINAL ARTICLE

Original article should have a structured format that should include- Introduction, Methodology, Results, and Discussion. Details of study design, sampling techniques, ethical approval status, study analysis, major strengths and limitations and future implications should be covered. Maximum of 6 tables and figures are allowed. The tables should be numbered with legends and should be appended at the end of the manuscript after the references in a serial order. The figures should be in jpeg format and are to be produced separately along with manuscript draft.

CASE REPORT/SERIES

This article should have the following structure- Introduction, Case presentation, Discussion and Conclusion. Authors may include 1 figure/image.

PERSPECTIVE/VIEWPOINT/ OPINION

It should be on a current relevant topic. It can be structured (with headings) or unstructured (without headings). Upto 2 tables or figures are allowed.

References

Journal Articles

Authors separated by commas – Family name and initials. Title of article. Abbreviated journal title. Publication year, month, day (month & day only if available);volume(issue):full page range.

Example

McInnes D, Bollen J. Learning on the job: metaphors of choreography and the practice of sex in sex-on-premises venues. Venereology. 2000;13(1):27-36

For more authors; Note: list all 6 authors or alternatively list the first 3 and add "et al"

Electronic journal article with DOI number

Author(s) - Family name and initials. Title of article. Abbreviated journal title [Internet]. Publication year, month, day (month and day only if available) [cited date - year, month, day];volume number(issue number):page numbers. Available from: URL. doi: Note: - Months are abbreviated to their first 3 letters. - if no URL - just enter the DOI number

Example

Puri S, O'Brian MR. The hmuQ and hmuD genes from Bradyrhizobium japonicum encode heme-degrading enzymes. J Bacteriol [Internet]. 2006 Sep [cited 2012 Aug 2];188(18):6476-82. Available from: http://jb.asm.org/cgi/content/full/188/18/6476?view=lo ng&pmid=16952937. doi:10.1016/j.psychsport.2009.03.009

Book

Author(s) – Family name and initials (no more than 2 initials with no spaces between initials). Title of book. Edition of book if later than 1st ed. Place of publication: Publisher name; Year of publication.

Example

- Butler SW. Secrets from the black bag. London (GB): The Royal College of General Practitioners; 2005.
- Cheers B, Darracott R, Lonne B. Social care practice in rural communities. Sydney (AU): The Federation Press; 2007.
- Hofmeyr GJ, Neilson JP, Alfirevic Z, Crowther CA, Gulmezoglu AM, Hodnett ED et al. A Cochrane pocketbook: Pregnancy and childbirth. Chichester (England): John Wiley & Sons; 2008.

Web Pages

Author/organization's name. Title of the page [Internet]. Place of publication: Publisher's name; Publication date or year [updated date - year month day; cited date - year month day]. Available from: URL

If the place, publisher or date is unknown use [place unknown], [publisher unknown].

If neither the date of publication nor a date of copyright can be found - use the date of update/revision or date cited.

Example

Diabetes Australia. Gestational diabetes [Internet]. Canberra (AU): Diabetes Australia; 2015 [updated 2015; cited 2017 Nov 23]. Available from: https://www.diabetesaustralia.com.au/gestational-diabetes

National Center for Infectious Diseases [Internet]. Atlanta (GA): Centers for Disease Control and Prevention (US); [reviewed 2007 Feb 15; cited 2007 Feb 20]. Available from: http://www.cdc.gov/ncidod/index.htm

Webpages with no author:

Title [Internet]. Place of publication: Publisher's name (unless unknown); Publication date or year [updated date (if available); cited date]. Available from: URL

Example

The family impact of Attention Deficit Hyperactivity Disorder (ADHD) [Internet]. [place unknown: publisher unknown]; 2009 Nov 1 [updated 2010 Jan 1; cited 2010 Apr 8]. Available from:http://www.virtualmedicalcentre.com.au/healthandlifest yle.asp?sid=192&title=The-Family-Impact-of-Attention- Deficit-Hyperactivity-Disorder-%28ADH-D%29page=2

Or

Australian Medical Association [Internet]. Barton (AU): AMA; c2016. Junior doctors and medical students call for urgent solution to medical training crisis; 2012 Oct 22 [cited 2012 Nov 5]; Available from: https://ama.com.au/media/junior-doctors-and-medical-students-call-urgent-solution-medical-training-crisis

With best compliments from **India's** **1**st **CNS Company**

torrent
PHARMA

Serta®
Sertraline   25/50/100mg
No. 1 Prescribed
Sertraline in Indian patients
The Smile of India

CLONOTRIL
Clonazepam 0.25 / 0.5 / 1 / 2 mg orally disintegrating tablets
Your **Seizure Free** Companion

Feliz S
Escitalopram 5 / 10 / 20 mg tablets
Means Happiness…

Arip MT
Aripiprazole 5/10/15/20/30 mg
Restores Life back to normal

Amazeo OD
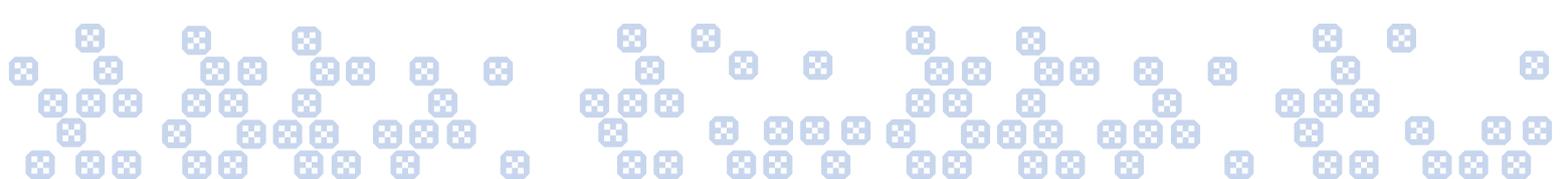Amisulpride Extended release 100 / 200 / 400 mg tablets
The amazing atypical

Tolaz LA
210mg/vial
300mg/vial
405mg/vial
Olanzapine Pamoate for extended release injectable suspension
Better adherence, Better outcome

SUN PHARMA

The favorable 1st line antipsychotic

OLEANZ

(Olanzapine 2.5 mg / 5 mg / 7.5 mg / 10 mg tablets)

Makes life easier for all schizophrenics

OLEANZ Forever

For synergistic additive benefits in co-morbid depression and anxiety

PANAZEP **LS**

Paroxetine Extended Release 12.5 mg & Clonazepam 0.25 mg tab

Delivers faster & sustained benefits

A NOVEL TRIMODAL psychotropic
D2 modulator + 5HT2A antagonist + Glu modulator

Nexluma™

Lumateperone 42 mg capsules

Helps them put light back into their lives

New

A Novel multimodal antidepressant that is DIFferent

REBORN
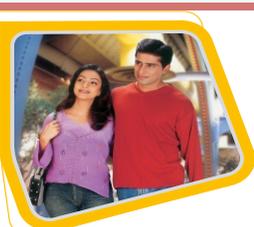
Vortidif

vortioxetine
5 / 10 / 20 m g Tablets

Makes a big DIFference in their life

Stabilize the lives of bipolar disorder patients

The once daily mood stabilizer

1 x 24

Dicorate ER

Divalproex sodium extended release 125/250/500/750mg/1g tablets

...the newer way the world prescribes valproate

Unique Dopamine System Stabilizer

Arpizol

Aripiprazole
/5/10/15/20/30 mg Tablets

Partial agonism... Total reintegration