Vol 1|ISSUE 5|2022

Petril Times

NEWSLETTER

INFORMATION BULLETIN ISSUED AS A SERVICE TO THE MEDICAL PROFESSION

Volume 1 | Issue 5| 2022

This issue contains:

Should you require any article or, medical information, please feel free to contact us at medicalservices@microlabs.in

Looking forward to your valuable feedback.

Best Regards,

Medical Team, Micro Labs Limited.

Vol 1|ISSUE 5|2022
Petril Times
NEWSLETTER
INFORMATION BULLETIN ISSUED AS A SERVICE TO THE MEDICAL PROFESSION

GRAY MATTER REDUCTION IN BILATERAL INSULA MEDIATES THE ADVERSE PSYCHIATRIC EFECTS OF BODY MASS INDEX IN SCHIZOPHRENIA

Introduction:

Schizophrenia (SZ) is a severely debilitating mental disorder with a lifetime incidence of about 1% that imposes a massive impact on public health. Until recently, the pathophysiology and progression of SZ have remained unknown, and current evidence-based treatment methods are restricted. [1] Overweight/obesity is clinically prevalent in SZ, with about 40-60% of SZ patients being overweight or obese, a rate significantly higher than the general population (20-40%). [2] Overweight/obesity-related brain structural alterations have been identified in a variety of areas involved in emotion and memory (e.g., the amygdala, hippocampus), cognitive control (e.g., cingulate, prefrontal cortex), reward (e.g., the striatum, insula, orbitofrontal cortex), and energy homeostasis regulation (e.g., the hypothalamus). [3] Overlapping pathoetiologies include chronic low-grade infammation, increased oxidative stress, neurotransmitter imbalances, and hypothalamic-pituitary-adrenal axis disruptions, which may be linked to the prevalent neuroimaging abnormalities and high incidence of obesity in SZ patients. While the effects of overweight/obesity on brain structure are of major concern in SZ, few studies have used this approach to investigate whether overweight/obesity-related brain structural abnormalities directly overlap with brain areas linked with SZ. As a result, this study may fill a knowledge gap. Researchers expected that SZ and overweight/obesity would both be adversely associated with certain GMV abnormalities in homologous brain areas, and that the association between BMI and clinical symptoms would be mediated by GMV modifications.

Methods:

There were 290 participants in total, divided into four groups (n=69 SZ patients with OWB, SZ-OWB; n=74 SZ patients with normal weight, SZ-NW; n=54 healthy controls with OWB, HC-OWB; and n=53 HC with NW, HC-NW). A high-resolution T1-weighted sequence was used to scan all individuals. To analyse the GMV modifications, whole-brain voxel-based morphometry was used, and a 22 complete factorial analysis of variance was used to determine the main effects of diagnosis (SZ vs HC), BMI (NW vs OWB) variables, and their interactions. A post hoc analysis was also performed to compare the pairwise differences in GMV changes.

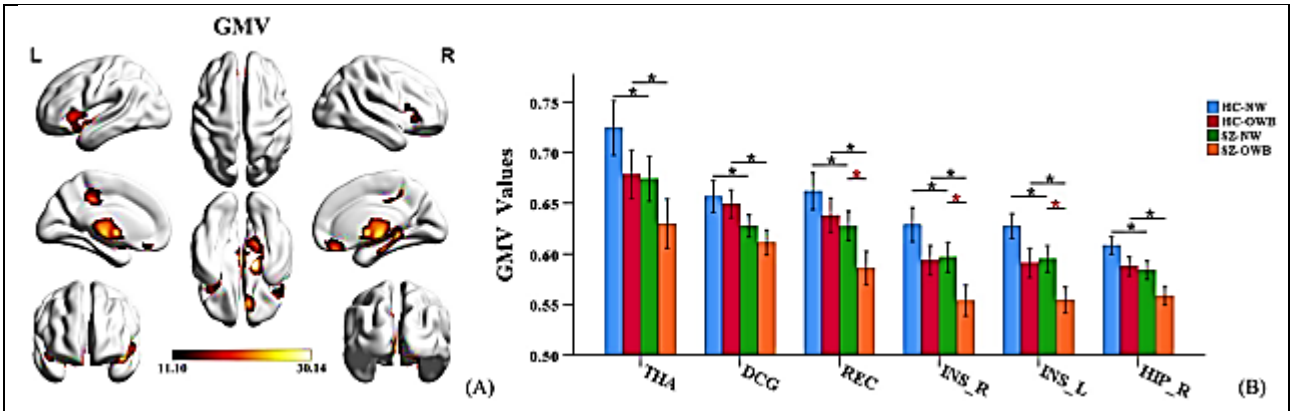
Results:

Vol 1|ISSUE 5|2022
Petril Times
NEWSLETTER
INFORMATION BULLETIN ISSUED AS A SERVICE TO THE MEDICAL PROFESSION

The primary effects of diagnosis were found in the right hippocampus, bilateral insula, rectus, median cingulate/paracingulate gyri, and thalamus (SZHC), whereas the primary effects of BMI were found in the right amygdala, left hippocampus, bilateral insula, left lingual gyrus, and right superior temporal gyrus (OWBNW). There were no significant diagnosis-by-BMI interaction effects in this investigation, although both SZ and OWB were shown to be additively linked with reduced GMV in bilateral insula. Furthermore, mediation analyses demonstrated that BMI has an indirect effect on unpleasant symptoms via GMV decrease in bilateral insula.

Discussion:

Lower GMV in the right hippocampus, bilateral insula, rectus, median cingulate/paracingulate gyri, and thalamus were associated with SZ, while lower GMV in the right amygdala, left hippocampus, bilateral insula, left lingual gyrus, and right superior temporal gyrus were associated with being overweight/obese regardless of SZ diagnosis. In this investigation, there were no significant diagnosis-by-BMI interaction effects, however both SZ and OWB were shown to be additively linked with reduced GMV in the contralateral insula. In SZ, mediation analysis demonstrated that BMI had an indirect effect on negative symptoms through GMV decrease in the contralateral insula.



Brain regions showing signifcant main efects of diagnosis (p<0.001, FWE corrected). A Results in the whole-brain VBM analysis: the red/orange indicates brain regions with lower GMV in SZ compared to HC group (SZ<HC), the color scales represent F-values. B A post hoc ROI analysis shows the pairwise comparisons in GMV, error bars indicate the standard error of the mean. *p<0.001, FWE corrected. Abbreviations: SZ=schizophrenia; HC=healthy controls; L=left; R=right; HIP=hippocampus; INS=insula; REC=gyrus rectus; DCG=median cingulate and paracingulate gyri; THA=thalamus

The study discovered substantial main effects of SZ diagnosis in the frontal, temporal, insular, and thalamus areas, which are thought to be the most replicated in SZ. The rectus and median cingulate/paracingulate gyri are components of the frontal brain; anatomical anomalies in these two areas are responsible for impulsive behaviour in SZ. [4] The thalamus is a significant information hub

involved in a variety of conscious integration processes such as cognition, emotion, and sensory information processing. This study also found that reduced GMV in the thalamus was linked to cognitive impairment (including visuospatial/constructional ability and attention) in SZ patients. [5]

Another obesity-affected area in this study was the left lingual gyrus, which is important for visual information processing. Based on this results, it is probable that GMV loss in the left lingual gyrus underpins the decreased brain activity in this area, resulting in a larger increase in food seeking in overweight/obese patients. The majority of the SZ-related and overweight/obesity-related brain regions identified in the current study were in limbic regions associated with reward information processing, namely, bilateral insula (sensory processing), amygdala-hippocampus (reward learning and memory), rectus and median cingulate/paracingulate gyri (reward value appraisal, executive control and decision-making).

Furthermore, the bilateral insula identified in SZ and OWB was linked to attention deficits in both disorders. Furthermore, mediation analysis demonstrated an indirect effect of BMI on unpleasant symptoms via GMV changes in the contralateral insula. These findings are very therapeutically significant, as both SZ and OWB are associated with poor cognitive performance, and comorbidity with overweight/obesity frequently contributes to poor mental outcomes in SZ patients.

Conclusion:

In conclusion, the current study adds to the evidence that both SZ and OWB have a negative impact on comparable brain areas related with reward processing, with the shared brain structural deficits in the bilateral insula perhaps serving as a significant site of intersection. This similarity in the morphometric deficit pattern might explain why people with SZ have a greater prevalence of overweight/obesity. Furthermore, this study offers early support for the involvement of the bilateral insula in mediating the indirect effect of BMI on negative symptoms, indicating that the bilateral insula plays an important role in the neuropathology of SZ.

This work adds to the evidence that a greater BMI is related with reduced GMV, which may raise the likelihood of SZ illness progression.

Reference:

1. Wu H, Dai G, Aizezi M, Tang J, Zou K, Wu Y, Wu X. Gray matter reduction in bilateral insula mediating adverse psychiatric effects of body mass index in schizophrenia. BMC psychiatry. 2022 Dec;22(1):1-2.

2. Tian Y, Wang D, Wei G, Wang J, Zhou H, Xu H, Dai Q, Xiu M, Chen D, Wang L, Zhang XY. Prevalence of obesity and clinical and metabolic correlates in first-episode schizophrenia relative to healthy controls. Psychopharmacology. 2021 Mar;238(3):745-53
3. García-García I, Michaud A, Dadar M, Zeighami Y, Neseliler S, Collins DL, Evans AC, Dagher A. Neuroanatomical differences in obesity: meta-analytic findings and their validation in an independent dataset. International Journal of Obesity. 2019 May;43(5):943-51.
4. Barry AB, Koeppel JA, Ho BC. Impulsive decision making, brain cortical thickness and familial schizophrenia risk. Schizophrenia Research. 2020 Jun 1;220:54-60.
5. Wolff M, Vann SD. The cognitive thalamus as a gateway to mental representations. Journal of Neuroscience. 2019 Jan 2;39(1):3-14.

IMPULSIVITY LEVELS IN INDIVIDUALS WITH CANNABIS USE DISORDER

Introduction:

Substance use disorder is defined by the existence of a collection of cognitive, behavioural, and physiological symptoms that suggest the individual's continued use despite substance-related issues. Continuous and long-term exposure to the substance increases dependence, accumulating psychological, neurobiological, and social components. [1] According to the United Nations Office on Drugs and Crime (UNODC), the usage of Cannabis sativa, popularly known as marijuana or cannabis, has grown in many regions of the world, making it the world's third most consumed narcotic after alcohol and cigarettes. Cannabis is also the most often used illegal substance in Brazil, with 3.2% of the Brazilian population, or 4.9 million individuals, having used psychoactive substances. [2] Despite their varied nature, they are recognised as quick activities, conducted without context evaluation, and associated with difficulty regulating the reaction. In turn, impulsiveness is linked to such actions, and impulsive people tend to act rashly and with little preparation. [3] The purpose of this study was to look at the existence and severity of impulsivity in people with cannabis use disorder to see how much impulsive conduct is linked to drug use. Furthermore, it sought to examine the relationship between sociodemographic and clinical factors (age, education, married status, cohabitation, and use of other psychoactive drugs) and impulsivity. This study should help to elucidate aspects linked with impulsivity in people with cannabis use disorder, giving data that may be used to create more successful intervention methods for this clinical condition.

Methods:

The study's goal was to look at the existence and severity of impulsivity in people with cannabis use disorder, as well as the relationships between sociodemographic and clinical variables and impulsivity. The Barratt Impulsiveness Scale and a sociodemographic data sheet were completed by

participants (BIS-11). A total of 122 patients with a cannabis use disorder diagnosis took part in the study, with a mean age of 34.46 years (standard deviation = 9.62).

Results:

The BIS-11 total score was substantially linked with cohabitation and alcohol consumption; the prevalence of high levels of impulsivity in the group varied from 30 to 33%. The BIS-11 scores for motor impulsivity and attentional impulsivity were also linked to alcohol use. There were no relationships observed between impulsivity and the variables age, education, cigarette usage, or cocaine/crack use.

Discussion:

Means, standard deviations and Barratt Impulsiveness Scale (BIS-11) score classifications

	BIS-11			
	Total	Motor impulsivity	Attentional impulsivity	Impulsivity due to lack of planning
Mean	70.12	23.53	18.66	27.93
Standard deviation	12.90	5.47	4.74	5.49
Minimum	32	13	8	11
Maximum	101	39	30	41
Percentile (%)				
< 25	12 (10)	12 (10)	19 (16)	11 (10)
25	19 (16)	13 (11)	13 (11)	24 (19)
50	25 (20)	23 (19)	29 (24)	36 (30)
75	26 (21)	21 (17)	28 (23)	18 (15)
90	30 (25)	43 (35)	31 (25)	24 (19)
99	10 (8)	10 (8)	2 (1)	9 (7)

Chi-square (X^2) tests for associations between sociodemographic variables and presence of impulsivity deficits according to the Barratt Impulsiveness Scale (BIS-11)

	Total		Motor		Attentional		Lack of planning	
	X^2	p	X^2	p	X^2	p	X^2	p
Age	0.403	0.525	0.024	0.877	1.072	0.300	0.933	0.334
Education	1.513	0.469	1.377	0.502	3.418	0.181	2.008	0.366
Marital status	4.146	0.246	2.619	0.454	0.727	0.867	2.604	0.457
Cohabitation	12.068	0.017*	4.441	0.350	6.924	0.140	4.384	0.357
Alcohol use	5.004	0.025*	4.872	0.027*	4.163	0.041*	2.688	0.101
Tobacco use	0.297	0.586	1. 262	0.261	0.296	0.587	0.311	0.577
Cocaine/crack use	0.007	0.935	1.168	0.280	1.149	0.284	0.008	0.927

* $p < 0.05$.

The primary goal of this study was to look at the presence and severity of impulsivity in people with drug use disorders, especially cannabis users. As a result, participant performance on the BIS-11 scale was verified, and normative data for the Brazilian context were categorised using z scores and percentile rankings. The proportion of high impulsivity scores in this study's sample was between 30 and 33%, depending on the categorization used (z score or percentile). The motor impulsivity component had the largest proportion of impairment among individuals (37% by z score; 43% by percentile) of the BIS-11 variables. Another goal of this study was to look for links between sociodemographic and clinical variables (such as age, education, marital status, cohabitation, and use of other psychoactive drugs) and impulsivity. Participants who live alone and take alcohol in addition to cannabis seem to be more impulsive. Furthermore, alcohol use was linked to increased levels of motor and attentional impulsivity.

The incidence of impulsive difficulties in the research sample above the levels predicted for the general population (about 17%), while no specific values were reported for the clinical group with substance use disorder, mostly linked to cannabis use. College students who use cannabis, on the other hand, have significant levels of impulsivity. This conclusion complements the findings in the

literature, which show that impulsive behaviours are quite frequent in a variety of diseases, including drug use disorder. [4] A similar age profile for adults consuming cannabis was also documented in a research. However, there was no connection between greater levels of impulsivity and age in this investigation, despite the fact that deficits in executive components, including impulsivity, caused by cannabis use were more common among adolescents than adults. [5] According to the findings of this study, individuals who live alone and consume alcohol alongside cannabis had greater rates of impulsivity. According to the literature, the use of numerous psychoactive drugs is connected with changes in impulsivity and might be linked to family problems. As a result, social isolation and loneliness may be risk factors for the development of drug use disorders.

Conclusion:

This study adds to the understanding of drug dependence, particularly cannabis dependence. The majority of the cannabis use disorder group showed high levels of impulsivity, which is supported by the literature. This emphasises the significance of conducting research to undertake comparative evaluations of impulsivity in people with cannabis use disorder and the general population, in order to add to existing therapies and build relevant and effective interventions. Professionals who specialise in drug abuse treatment would be more positioned to support these persons in their process of behavioural change, with the goal of reducing and eventually discontinuing substance use, expanding competency in interpersonal interactions, and improving quality of life.

This study adds to the understanding of drug dependence, particularly cannabis dependence. It discovered impulsive conduct in people with cannabis use disorder, which is consistent with previous research.

Reference:

1. Wagner MF, Oliveira CR, Paloski LH. Levels of impulsivity in individuals with cannabis use disorder. Trends in psychiatry and psychotherapy. 2022 Jul 8;44.
2. Pereira JR, Veloso C, Shigaki HB, Lara JE. Cannabis Sativa: Aspectos relacionados ao consumo de maconha no contexto brasileiro. RAHIS-Revista de Administração Hospitalar e Inovação em Saúde. 2018 Jun 11;15(1).
3. Kozak K, Lucatch AM, Lowe DJ, Balodis IM, MacKillop J, George TP. The neurobiology of impulsivity and substance use disorders: implications for treatment. Annals of the New York Academy of Sciences. 2019 Sep;1451(1):71-91.
4. Pilatti A, Prince MA, Bravo AJ, Pearson MR, Mezquita L, Pautassi RM, Cross-Cultural Addictions Study Team. Cannabis-Related Perceptions as Mediators of the Association Between Trait Impulsivity and Cannabis Outcomes. Journal of Studies on Alcohol and Drugs. 2021 Jul;82(4):522-35.
5. Goodman S, Fischer B, Hammond D. Lower-risk cannabis use guidelines: adherence in Canada and the US. American journal of preventive medicine. 2020 Dec 1;59(6):e211-20.

Vol 1|ISSUE 5|2022
Petril Times
NEWSLETTER
INFORMATION BULLETIN ISSUED AS A SERVICE TO THE MEDICAL PROFESSION

EFFECTS OF EPILEPSY INSOMNIA AND LEVELS OF DEPRESSION AND ANXIETY SYMPTOMS ON QUALITY OF LIFE

Introduction:

Epilepsy is a dangerous chronic neurological illness that affects more than 70 million people globally. It is distinguished by recurring and unexpected seizures. PWE frequently describe psychological symptoms as well as sleep issues including insomnia. Insomnia is the most prevalent sleep-related complaint among epilepsy patients, with 25% to 54% reporting insomnia symptoms. Insomnia has been linked to lower QOL in PWE, and PWE with insomnia had considerable QOL impairment. However, there is little research on how insomnia affects QOL. Furthermore, the QOL of PWE patients is highly related to their knowledge, attitude, and perceived stigma. [1] Previous research has found that PWE with insomnia had higher depressive symptoms than those without insomnia. Insomnia is associated with depressive symptoms. Furthermore, depression has been identified as the most important indicator of low QOL in people with Parkinson's disease. Anxiety and depression are quite common in PWE. Anxiety symptoms are also a powerful predictor of low quality of life. As a result, we expect that anxiety and depression symptom levels would interact to impact the association between sleeplessness and QOL in patients. [2] The purpose of this study was to look at the effects of sleeplessness, anxiety levels, and depressive symptoms on QOL in PWE. Two hypotheses (H) are proposed in the study: H1: depression symptom levels influence the indirect relationship between insomnia and QOL; and H2: anxiety symptom levels moderate the indirect relationship between insomnia and QOL via depression symptom levels.

Methods:

A total of 179 PWE were enrolled in a single cohort. Data on sleeplessness, degrees of sadness and anxiety symptoms, and QOL were obtained. The following questionnaires were used: The Insomnia Severity Index (ISI), the Depression Inventory for Epilepsy (NDDI-E), the Generalized Anxiety Disorder-7 (GAD-7), and the QOL in Epilepsy Inventory (QOLIE-31). A moderated mediation model was used to evaluate the direct, indirect, and total impacts of insomnia on QOL.

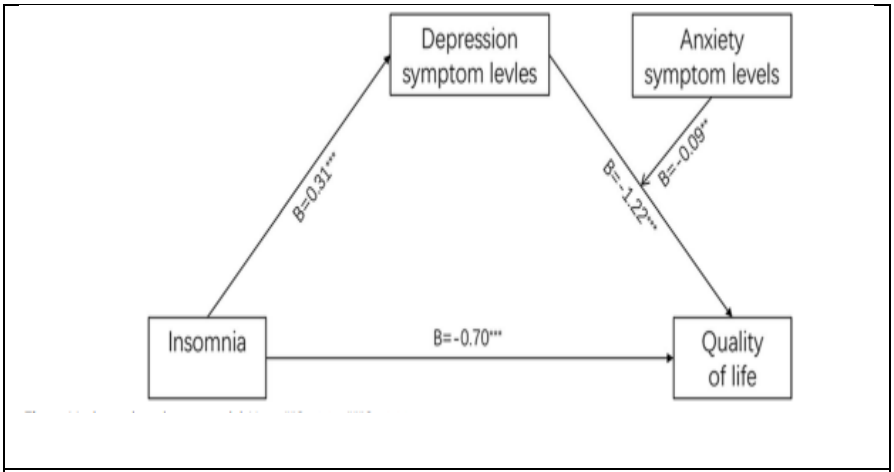
Results:

The relationship between sleeplessness and QOL was mediated by depression symptom levels (B=0.09 SE=0.03, p=0.01). Depression symptom levels were responsible for 34.7% of the overall

effect of sleeplessness on QOL. Anxiety symptom levels positively moderated the mediating effect of depression symptom levels (B=0.09, SE=0.03, p=0.01).

Discussion:

The researchers used a moderated mediation model to determine if depression symptom levels mediate the relationship between insomnia and QOL and whether anxiety symptom levels exacerbate the indirect effect of insomnia on QOL via depression symptom levels. This is the first exploratory study to use a moderated mediation model to evaluate the causal relationship between insomnia and QOL. We discovered that the influence of sleeplessness on QOL may be explained by the mediation of depression symptom levels, and anxiety symptom levels can exacerbate the indirect effect of insomnia on QOL via depression symptom levels. These findings add to our understanding of the relationship between insomnia and QOL.



Moderated mediation model.

According to the accepted moderated mediation model, sleeplessness may enhance depressive symptom levels, resulting in a lower QOL. As a result, insomnia had an indirect effect on low QOL, and depression symptom levels modulate insomnia's effects on QOL in PWE. This significant discovery has clinical implications. Managing depression symptoms may be equally as crucial as addressing sleeplessness symptoms, according to patients. Interventions targeting this mediator (depression symptom levels), which is related with QOL, might enhance QOL in individuals with insomnia. The finding that there is an indirect link between sleeplessness and QOL via depression symptom levels highlights the limits of traditional regression analysis in determining the true interplay among risk variables for QOL in PWE. [3]

In the literature, there is a complicated link between depression and anxiety. Anhedonia may be a major element in the development of these two mental diseases, and anxiety may deteriorate into depression as a result of anhedonia. The effect of anxiety symptom levels on the relationship between sleeplessness and QOL via depression symptom levels was also investigated. For the first time,

research findings revealed that the favourable relationship between depression and QOL gets greater in individuals with increasing degrees of anxiety symptoms. The findings indicate that reducing anxiety symptoms may help lessen the influence of depressive symptoms on QOL in PWE.

Finally, the effects of depression and anxiety symptoms on quality of life may be influenced by other epilepsy-related characteristics, such as the number of ASMs and seizure frequency, which were not investigated in this study. Aside from depression and anxiety symptoms, several other factors might contribute to the incidence of insomnia, including age and economic and marital factors, among others, which may influence the established link.

Conclusion:

In the literature, there is a complicated link between depression and anxiety. Anhedonia may be a major element in the development of these two mental diseases, and anxiety may deteriorate into depression as a result of anhedonia. The effect of anxiety symptom levels on the relationship between sleeplessness and QOL via depression symptom levels was also investigated. For the first time, research findings revealed that the favourable relationship between depression and QOL gets greater

Insomnia's effect on QOL can be explained in part by the mediation of depressive symptom levels. Furthermore, reducing anxiety symptoms may mitigate the indirect effect of sleeplessness on QOL via depression symptom levels.

Reference:

1. Zhong R, Li Z, Chen Q, Zhang H, Zhang X, Lin W. Effects of insomnia and levels of depression and anxiety symptoms on quality of life in people with epilepsy. BMC psychiatry. 2022 Dec;22(1):1-6.
2. Zhong R, Lu Y, Chen Q, Li M, Zhao Q, Zhang X, Lin W. Sex differences in factors associated with quality of life in patients with epilepsy in Northeast China. Epilepsy & Behavior. 2021 Aug 1;121:108076.
3. Siarava E, Hyphantis T, Katsanos AH, Pelidou SH, Kyritsis AP, Markoula S. Depression and quality of life in patients with epilepsy in Northwest Greece. Seizure. 2019 Mar 1;66:93-8.
4. Kawata Y, Maeda M, Sato T, Maruyama K, Wada H, Ikeda A, Tanigawa T. Association between marital status and insomnia-related symptoms: findings from a population-based survey in Japan. European Journal of Public Health. 2020 Feb 1;30(1):144-9.

Vol 1|ISSUE 5|2022
Petril Times
NEWSLETTER
INFORMATION BULLETIN ISSUED AS A SERVICE TO THE MEDICAL PROFESSION

A CASE REPORT ON THE 3 YEAR NEURORADIOLOGICAL SCREENING OF A PATIENT WITH FIRST-EPISODE PSYCHOSIS

Introduction:

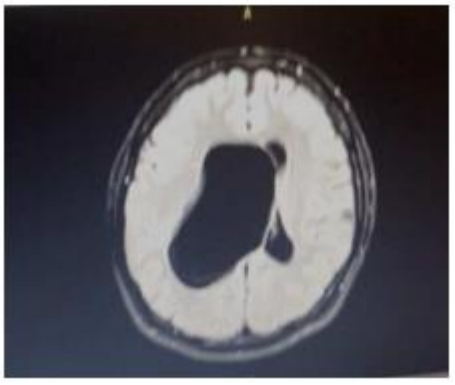
The majority of instances of first episode psychosis are caused by early onset schizophrenia. Early-onset schizophrenia is most common between the ages of 15 and 25. Very early onset schizophrenia is defined as occurring before the age of 13, and it is extremely rare. According to some writers, mania with psychotic signs comes within the category of first episode psychosis. Almost all practise recommendations urge neuroradiological screening to rule out or rule in reversible causes of first episode psychosis, particularly if they are operable. There are certain hurdles to carrying out the neuroradiological screening section of the practise recommendations for managing first episode psychosis. These barriers include a lack of needed facilities, restricted government financing, and insurance reimbursement restrictions for the radiological treatment. [1] Ultrasonography was a suggested procedure for the foetal brain throughout our patient's pregnancy in several circumstances, including this case report. The neurodevelopmental hypothesis of schizophrenia proposes that pathological neurodevelopmental processes that begin as early as the first and second trimesters result in neuronal circuits primed to generate psychotic symptoms during adolescence or early adulthood, often in the context of heightened biological and psychological stress. The neurodegenerative theory, on the other hand, is supported by several research' findings, since individuals with schizophrenia have accelerated age-related brain tissue loss following symptom onset when compared to healthy controls.

Case Report:

A 20-year-old female patient of Middle Eastern descent presented to the outpatient clinic with her family after an eight-month period of mental deterioration in which she developed paranoid delusions of persecution accusing her family of spying on her, poisoning her food and drink, and having perceptual abnormalities in the form of auditory hallucinations giving running commentary about her. The study initially assessed her for first episode psychosis using structural neuro-radiological scans and related blood tests. Simultaneously, a modest dosage of risperidone was started and subsequently increased with a limited initial response to oral risperidone doses up to 4 mg. ECG was used to monitor dose escalation, and her QTc was about 408 msec.

A clinical examination was performed, as well as an evaluation for probable adverse effects. We ensured that both spontaneously reported adverse effects by the patient and side effects witnessed by

her family and documented during our evaluation were discovered and addressed. The structural MRI indicated a massive chronic cyst in the right lateral ventricle, measuring 8.4 cm anteroposterior x 5.7 cm side to side x 4.6 cm craniocaudally, with cyst volume matching the CSF signal intensity. A dilated right temporal horn and right occipital horn were also seen, indicating outflow blockage owing to a blocked foramen of Monro.



Chronic thin walled intraventricular cyst crossing the midline and occupying the lateral ventricle.

The cyst was extending the right lateral ventricle and crossing the midline, compressing and narrowing the left lateral ventricle and third ventricle, with radiological markers of the cyst's chronicity, such as the lack of surrounding edoema and the existence of a thin capsule. This patient was and continues to be neurologically free in clinical neurological examination and has no neurological complaints at the time of writing this report. This patient was observed for three years and reacted effectively to the second generation antipsychotics Risperdione orally and Paliperdione monthly injection. She became pregnant and gave birth to a healthy baby who reached the developmental milestone of a 24-month-old toddler. She was followed for 36 months. A few weeks after her symptomatic recovery, the patient planned a traditional marriage, and after counselling the patient and her family, medications were gradually discontinued so that before her wedding day, she was completely free of Risperidone to avoid possible teratogenic effects despite the Risperidone's safety profile. She gave birth to a healthy girl who was bottle fed and reached the standard developmental milestone of 24 months. As her insight improved during the procedure, the patient was eager to stay on the same antipsychotic prescription. During her follow-up, there was no indication of risk presented to her infant as a result of neglect or disturbed conduct, and she provided reasonable care for her kid.

She remained mostly aware of the nature of her condition and participated when she experienced breakthrough symptoms by sharing her worries. She steadily gained weight, which might be attributed to her antipsychotic medication as well as a mix of other social variables such as a lack of exercise and having servants handle her domestic tasks for her. Fortunately, she had gastric sleeve surgery and dropped eighteen kg in the two months preceding her most recent check-up. Her most recent follow-up visit revealed that she was taking Palioeridone 150 mg and Risperidone 2 mg orally. She was in sustained full remission with no sign of relapse symptoms. Her performance at home was considered

as good. Her father claimed that a few days after the injection, her eyeballs rolled up in a perceptible fashion, which is not painful but lasts for many days. This was something new and had never been reported before. The goal was to restart the anticholinergic twice daily for better outcomes, reduce Risperidone by 1 mg/week over two weeks until she was solely on the long acting injectable, and then reassess her need for the anticholinergic before discontinuing it. The onset of the oculogyric crisis corresponded with the patient's weight decrease after bariatric surgery.

Discussion:

Several publications have identified brain abnormalities in adult schizophrenia patients, including medial temporal lobe reduction, lateral and third ventricle enlargement, reduced brain volume, basal ganglia enlargement, and thalamic abnormalities. The most commonly reproduced observation is lateral ventricle enlargement. The majority of first-episode psychosis patients will not have recognisable abnormalities with etiological relevance on neuroimaging, and only 3% of first-episode psychosis is organic in origin. [2] Brain results at the time of symptom start corroborate the neurodevelopmental theory, which is connected to a range of genetic and prenatal risk factors that may impair neurodevelopmental processes.

Following the emergence of symptoms, neurodevelopmental disorder may progress further. The neurodegenerative notion is backed by faster tissue loss in schizophrenia patients compared to controls. In schizophrenia, the word neuroprogression refers to neurodegeneration, apoptosis, and decreased neurogenesis/neuroprotection. There is an apparent overlap with the infection theory aetiology in this case report, since this is relevant to likely aetiology for stenosis of the foramen of Monro and viral infectious hypothesis of schizophrenia. Both have a neurodevelopmental origin, however it is unclear if this overlap is relevant. It is also important to note that other colleagues encountered a case with schizophrenia-like symptoms that was the only manifestation of an arachnoid cyst in the temporal fossa with full recovery and no relapse following neuro-surgical intervention resulting in brain decompression with full remission of psychosis without the use of antipsychotic medications. The same authors also reported seeing two individuals with arachnoid cyst pathology in the middle cranial fossa that did not require surgical intervention, and both cases continued to have psychotic symptoms while being on antipsychotic medicines. [2]

Conclusion:

Neuro-radiological screening is an important inquiry that must be performed in order to screen for first episode psychosis. The identification of neuro-radiological finding/s will aid in patient care and necessitates further investigation. The causal association between the neuro-radiological result and a first episode of psychosis is currently being studied. Discovering the possible relationship(s) between a neuro-radiological pathology or brain changes and the development of first episode psychosis will not only shed light on the various possible etiologies of first episode psychosis, but will also shed light on possible ways to manage first episode psychosis, predict its development earlier, and intervene earlier based on newer knowledge about its development. It is always crucial to remember that connection does not always imply causation.

This is a case report of a 21-year-old female patient with first-episode psychosis who met the DSM 5 criteria for paranoid schizophrenia and who had an unexpected neuro-radiological finding discovered during regular neuro-radiological screening for first-episode psychosis. A massive right lateral intraventricular thin walled cyst with symptoms of blockage of the foramen of Monro was discovered.

Reference:

1. Mohammed Allam, Khaled Hamolila, Yahya Tikriti. Case Report: Neuro Radiological Screening of First Episode Psychosis Patient Followed up for 3 Years. American Journal of Psychiatry and Neuroscience. Vol. 10, No. 2, 2022, pp. 77-81.
2. Baquero GA, Molero P, Pla J, Ortuño F. A schizophrenia-like psychotic disorder secondary to an arachnoid cyst remitted with neurosurgical treatment of the cyst. The open neuroimaging journal. 2014;8:1.