

Introduction: Arachnoid cysts are intra-arachnoid space-occupying brain lesions, typically of a benign, congenital nature. Such cysts are quite rare, accounting for only 1% of all lesions in the intracranial space. In most cases, they are diagnosed accidentally by neuroimaging. Treatment-resistant schizophrenia (TRS) has a high burden both for patients and healthcare services. There is a need to identify treatment resistance earlier in the course of the illness, in order that effective treatment can be offered promptly. Recently, the co-occurrence of arachnoid cysts and schizophrenia has captured the popular attention about possible relevancy.

Objectives: Through a case report and a review of the literature, we hypothesize that arachnoid cyst is the cause of resistance in a patient with treatment-resistant schizophrenia.

Methods: Starting from a case report, we conducted a literature review on “PubMed”, using key words “arachnoid cyst, arachnoid cyst and psychosis”, “arachnoid cyst and treatment-resistant schizophrenia”,

Results: We present a 47-year-old who is single and unemployed. His past psychiatric history revealed a diagnosis of schizophrenia, having been admitted several times in different inpatient psychiatric wards. In the psychiatric examination, the presence of auditory hallucinations, dissociated thinking, and predominantly negative symptoms was observed. His symptoms showed only minimal responsiveness. He was diagnosed with TRS owing to the inadequate response to two sequential antipsychotic trials (with adequate dose, duration, and adherence). Our evaluation of TRS began with a thorough review of the patient’s psychiatric and treatment history. All nonpsychiatric causes, including untreated medical problems, that may contribute to ongoing psychotic symptoms have been ruled out. Physical examination and blood tests were unrevealing. Electroencephalography showed no signs of seizure activity. Following the evaluation process, a head CT scan showed a left paramedian cystic lesion at the level of the pineal gland. A cerebral MRI was performed in order to get a more detailed image. It confirmed the nature of the lesion and revealed the existence of an arachnoid cyst about 2.5 cm × 3.5 cm × 2.0 cm in size, centered on the quadrigeminal cistern with triventricular dilatation. This neurological tumor didn’t require neurosurgery.

Conclusions: Our case emphasises the importance of considering an organic cause like any space-occupying lesion in the brain (an arachnoid cyst in our case) for the induction of psychopathological symptoms, even those of treatment-resistant schizophrenia, which represents a major clinical challenge. This also underlines the interest of neuroimaging in the initial workup and supports the hypothesis of psychosis as a global network.

Disclosure of Interest: None Declared

EPV0414

Obstructive hydrocephalus caused by colloid cyst and treatment –resistant schizophrenia

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Introduction: Treatment-resistant schizophrenia can be of primary or secondary etiology. Systematic and thorough differential diagnostics is essential to exclude organic causes for treatment-resistant

schizophrenia. Colloid cysts are congenital benign tumor accounting for 15-20 % of intraventricular mass but only about 1% of intracranial ones. They frequently cause psychiatric disturbances. The pathology behind these psychiatric symptoms remains unclear.

Objectives: Through a case report and a review of the literature, we hypothesize that a colloid cyst in the third brain ventricle is the cause of resistance in a patient with treatment-resistant schizophrenia.

Methods: Starting from a case report, we conducted a literature review on “PubMed”, using key words “colloid cyst and psychosis”, “colloid cyst and treatment-resistant schizophrenia”,

Results: We present a 48-year-old male who has a family history of malignant neoplasm. There was no history of physical illness. His past psychiatric history revealed a diagnosis of schizophrenia, having been admitted several times in different inpatient psychiatric wards. In the psychiatric examination, the presence of auditory hallucinations, dissociated thinking, and predominantly negative symptoms was observed. Recently, he has been diagnosed with treatment-resistant schizophrenia owing to the inadequate response to two sequential antipsychotic trials (with adequate dose, duration, and adherence). After a 2-month hospitalization, the severity of the psychotic symptoms had decreased but did not show remission. With no prodromes or triggering factors, our patient presented a drop attack without loss of consciousness and with instantaneous recovery to baseline status. He did not have any of the same experience previously. The physical and neurological examination did not reveal any positive findings. All biochemistry parameters were reported as normal range. Following the evaluation process, an urgent head CT scan showed a colloidal cyst at the anterior end of the third ventricle with dilatation of the lateral ventricles. A cerebral MRI was performed in order to get a more detailed image; it confirmed the diagnosis of a third ventricle colloid cyst immediately adjacent to the foramen of Monro with obstructive hydrocephalus. The patient was referred to the neurosurgical department for further evaluation. This neurological tumor didn’t require neurosurgery.

Conclusions: Our case implies the importance of neuroimaging in patients with treatment-resistant schizophrenia to rule out any underlying organic cause. It also emphasises the importance of considering an organic cause like any space occupying lesion in the brain (colloid cyst in the third brain ventricle in our case) for induction of psychopathological symptoms, even those of treatment-resistant schizophrenia.

Disclosure of Interest: None Declared

EPV0418

Post-Traumatic Diabetes: Focus on Psychiatric Trauma

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Introduction: Physical and/or psychological trauma may contribute to the onset of type 1 diabetes. Forensic medicine experts recognize post-traumatic diabetes in rare cases that meet specific criteria, including the severity of the trauma, its occurrence within a short timeframe before diabetes onset, and the absence of prior diabetes indicators.

Objectives: This study aims to describe the clinical and biological characteristics of post-traumatic diabetes, with particular emphasis on the psychiatric aspects.

Methods: This retrospective, cross-sectional study analyzed cases of patients hospitalized for acute diabetes following physical or psychological trauma. The diagnosis of post-traumatic diabetes was based on the criteria of French forensic experts: acute autoimmune diabetes, trauma occurring within six months before diagnosis, and no signs suggesting pre-existing diabetes. Autoimmune involvement was confirmed by the presence of anti-pancreatic antibodies, specifically anti-glutamic acid decarboxylase (GAD) and/or anti-tyrosine phosphatase IA2.

Results: The study included 10 patients (8 men and 2 women) aged between 17 and 47 years (mean age 29.5 ± 11 years). Family history of autoimmune disease was present in 40% of cases, and type 2 diabetes in 60%. The average body mass index was 24 ± 6 kg/m², with obesity in 30% of cases. The mean blood glucose at admission was 14.54 ± 3.48 mmol/L, and the average HbA1C was $6.51 \pm 0.56\%$. Anti-GAD antibodies were present in all patients, while anti-IA2 antibodies were detected in 20%. Clinically, 60% of patients presented with ketosis, and 40% with ketoacidosis. Psychiatric trauma, including grief in 3 patients and divorce in 2 patients, was the triggering factor in 50% of cases. All patients required insulin therapy upon admission, with obese patients receiving additional metformin.

Conclusions: This study supports the hypothesis of post-traumatic diabetes, particularly in cases of severe psychiatric trauma. Although scientific literature remains inconclusive, the role of stress in the onset of diabetes warrants further investigation due to the heterogeneity of findings regarding stress episodes preceding diabetes development.

Disclosure of Interest: None Declared

EPV0419

Stunned Into Silence: Classical Catatonia secondary to Viral Myocarditis, a Case Report

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Introduction: Catatonia due to myocarditis is rare and poses a significant diagnostic challenge. We present a case of classical catatonia secondary to viral myocarditis.

Objectives: We want to highlight the clinical issues in diagnosing catatonia due to specific medical conditions, especially in a resource-limited setting like Pakistan.

Methods: The case is discussed, followed by suggestions and a literature review.

Results: A 22-year-old male with no known medical and psychiatric comorbidities presented to the emergency department in April 2023 with avolition, alogia and fever for 2 weeks. Patient was admitted to Neurology for workup; CSF analysis, CSF autoimmune screen, CSF culture, CSF HSV PCR, blood culture, urine culture, serum ceruloplasmin, MRI Brain were all unremarkable. He was treated with broad-spectrum antibiotics, following which his fever remitted.

On initial psychiatric exam, patient scored 13 on Bush Francis Catatonia Rating Scale for mutism, staring, posturing/catalepsy, stupor, grimacing, mitgehen, gegenhalten and rigidity. Initial work up included normal CBC, CMP, TSH, HIV/Syphilis, Urine toxicology, ESR and a CRP of 43 mg/L. He underwent echocardiography which revealed a severely reduced Ejection Fraction of 30% and dilated cardiomyopathy, consistent with viral myocarditis; which was considered in remission and thus managed conservatively by Cardiology. The patient then received IV infusion of Diazepam 10mg BID which adequately treated his catatonia over 3 days and he was discharged with instructions for close follow-up. On followup visits, the patient continued to display remission of catatonic symptoms, but exhibited new symptoms of acute psychosis including 2nd person auditory hallucinations and delusions of reference and persecution. These were treated with Olanzapine 5mg PO resulting in complete remission over 4 weeks. The patient continued to have residual symptoms of social withdrawal, decreased motivation and poor concentration on his most recent presentation. Patient was also noted to have an exaggerated startle reflex which was not associated with any other neurologically relevant symptoms. It prompted a workup to rule out seizure disorder, the results for which are pending at time of this submission.

Conclusions: This case underscores the clinical complexity and diagnostic challenge of catatonia linked to medical conditions. It also depicts the evolving presentation over time. It is important to note that the patient did not receive IV Lorazepam due to its unavailability in Pakistan. Electroconvulsive therapy should have been considered for his catatonic symptoms but was delayed due to his pending diagnosis. Limited findings from literature review related to catatonia associated with viral myocarditis demonstrate the need for further research to bridge the gaps in evaluation and management.

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EPV0423

Psychiatrists' practices on monitoring cardio-metabolic adverse effects: a narrative review

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Introduction: Several medications in use in the psychiatry field, particularly in severe mental illness (SMI), are associated with weight gain and other cardio-metabolic side effects and are usually prescribed long-term. Extensive research has been conducted on the contribution of these effects to poorer physical health and a 10- to 20-year mortality gap in patients with SMI. Many factors have been reported, such as patients' lifestyle behaviours, lower search for medical care, delayed diagnosis and treatment, diagnostic overshadowing by mental illness, and stigma towards this population. In this context, psychiatrists may have a role in monitoring medication's side effects their role is still to be defined.

Objectives: To access usual practices and barriers on cardio-metabolic monitoring of patients by psychiatrists.

Methods: We conducted a narrative review on general psychiatrists' practices of cardio-metabolic monitoring. A search was conducted in Pubmed and PsycNet using the keywords "practice",