Psychiatric Case Series

Vol 5 | Issue 25 |2024

Dear Doctor,

Greetings from Micro Labs Limited.

We are happy to bring you the "PSYCHIATRY Case series", which contains the latest and most interesting cases in the field of Psychiatry.

This issue contains

1. **Case report on Delirious Mania**
2. **Case report on anorexia nervosa with focal cortical dysplasia**
3. **An uncommon case of spontaneous bladder rupture in a patient with catatonic schizophrenia**
4. **Case report on Psychosis in Hyperparathyroidism**

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

Looking forward to your valuable feedback

Best Regards,

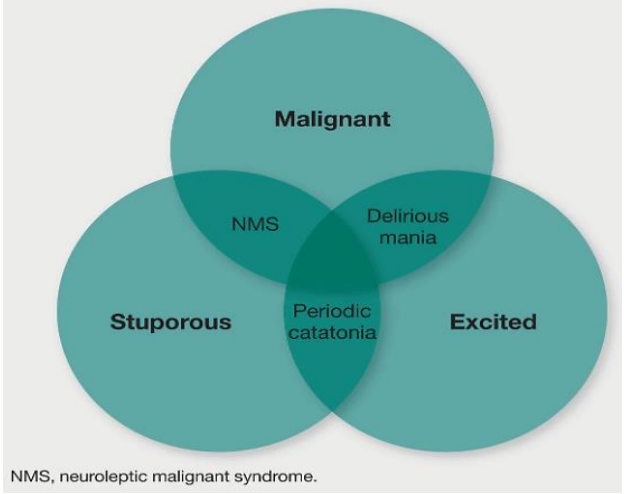
Medical Team,

1. Case report on Delirious Mania

Introduction:

Recognizing symptoms and medical history can help in diagnosis and treatment of Delirious mania (DM).

Delirious mania (DM) is a syndrome that causes sudden hyperactivity, psychosis, catatonia, and confusion. Patients with delirious mania experience quick onset of symptoms. Symptoms includes intense excitement, emotional lability, grandiose delusions, profound insomnia, pressured and rapid speech, auditory and visual hallucinations, hypersexuality and thought disorganization.[1] Delirious mania is not classified by the DSM-5 or ICD-10 systems. Formal diagnosis of DM is difficult since many people have previous medical or neurological conditions or use psychoactive medicines that can affect symptomology. However, based on clinical presentation and therapeutic efficacy, current cases can be diagnosed. The identification of clinical symptomology is crucial to treatment because there are currently no recognized therapeutic standards.[2] This case report describes a woman who presented with signs of manic episodes and delusions but had no past medical history of psychiatric illness or current use of psychoactive medicines.



Subtypes of catatonia

Case Presentation:

A 77-year-old female patient was spotted in an altered mental condition at a neighborhood petrol station and brought to the emergency room. The patient's blood pressure was as high as 227/92 mm Hg upon admission, indicating severe hypertension and dehydration. At the moment, the patient lives alone after retiring. She blames the government for her school taxes and claims that her financial circumstances are the cause of her depression. She was very tangential and quickly distracted, which made mental evaluation challenging. She couldn't recall why she had been brought in and exhibited confusion, disorientation, and

disorganization, including inappropriate loud laughter. She couldn't recall the time, her location, her last meal, or her residence. Despite being on multiple psychiatric drugs, she was unable to identify their dosages or prescriber. She eventually denied using any psychiatric drugs at all. During psychiatric consultation, she demonstrated impulsivity, paranoia, and fast mood and affect changes.

Throughout the conversation, she continued to laugh inappropriately. Her past psychiatric history included a four-month hospitalization for disturbed mental status. The patient was administered lithium and citalopram, but her blood work revealed a lithium level of 0, indicating non-adherence. She denied any previous psychiatric hospitalizations, hallucinations, or suicidal or homicidal thoughts. The patient has a history of bipolar disorder and cognitive deterioration.

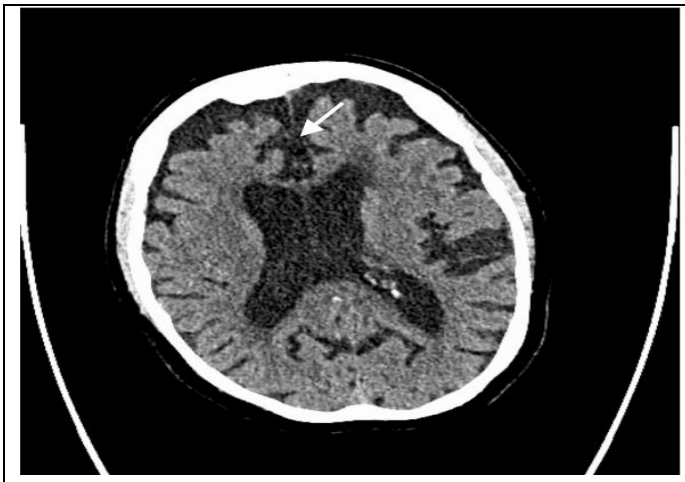


Figure 1: Head CT scan (The white arrow in the image indicates cortical atrophy).

The CT scan of her brain revealed no acute abnormalities. The patient's brain CT reveals cortical atrophy, in accordance with her age (Figure 1). The CT scan indicated persistent microvascular ischemic gliosis and an ancient lacunar infarction in the right basal ganglia. Her urinalysis revealed bacteria, WBCs, positive leukocyte esterase, and an unclear appearance. Due to a lack of clinical history, fosfomycin was administered as an empirical treatment for a potential UTI. Her electrocardiography (ECG) was consistent with her age and cardiovascular history (Figure 2). After the initial workup, the patient was started on nicardipine infusion therapy to reduce blood pressure. The patient's blood pressure has normalized. She

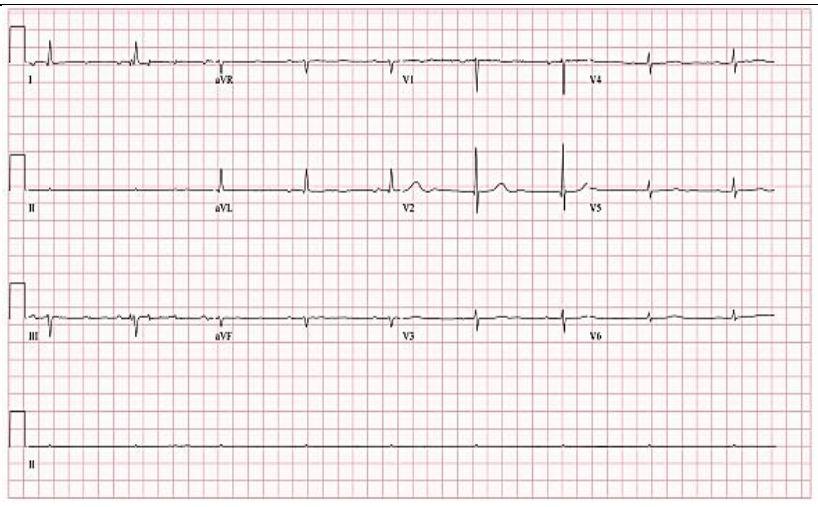


Figure 2: ECG at the time of admission revealing sinus bradycardia and non-specific T-wave abnormalities.

also started taking olanzapine medication. Medication improved her attention, mood swings, and confusion, but she continued to laugh inappropriately. Uncontrolled bipolar disorder and metabolic encephalopathy were taken into consideration. She was eventually discharged with a diagnosis of cognitive dysfunction. She was prescribed atorvastatin and lisinopril to treat her hypertension. She was restarted on citalopram and olanzapine to treat the bipolar disorder. After four days, the patient had been discharged.

Discussion:

Delirium is characterized by acute disorientation and altered consciousness, including easy distraction and concentration changes in the patient. This case study found that electroconvulsive therapy and high-dose benzodiazepines were the most effective therapies.[2] Although benzodiazepine medication may help manage bipolar disorder after discharge, it has been linked to exacerbating delirium in the elderly due to pharmacological and physiological effects.[3]

Conclusion:

DM is challenging to diagnose and treat due to a lack of clear treatment standards. Recognizing symptoms and medical history can be helpful, as seen in this patient with a history of bipolar disorder and symptoms of delirium and mania, such as distractibility, inappropriate laughter, and mood swings. The diagnosis of 'cognitive disease' reflects a range of issues affecting cognition, including metabolic encephalopathy from severe hypertension, untreated bipolar disorder, and diabetes symptoms. The treatment plan aimed to address these relevant variables.

References:

1. Reinfeld S, Yacoub A. Delirious mania: Presentation, pathogenesis, and management: Rapid recognition is critical to avoid initiating inappropriate treatment. Current Psychiatry. 2023 Dec 1;22(12):27-34
2. Espiridion ED, Deng A, Charron L. Delirious Mania in a 77-Year-Old Female. Cureus. 2024 Jul 29;16(7):e65619.
3. Ettcheto M, Olloquequi J, Sánchez-López E, et al. Benzodiazepines and Related Drugs as a Risk Factor in Alzheimer's Disease Dementia. Front Aging Neurosci. 2020 Jan 8;11:344.

2. Case report on anorexia nervosa with focal cortical dysplasia

Introduction:

There was a relationship between Anorexia nervosa (AN) and Focal cortical dysplasia (FCD), both have been linked to autism spectrum disorder.

Anorexia nervosa (AN) is a fatal disorder characterized by severe underweight and malnutrition. It can induce multiple organ failure and has a high mortality rate. AN is more prevalent in young females than in males.[1] Anorexia Nervosa is characterized by extremely low body weight, fear of weight gain, activities that prevent weight increase, and a lack of awareness of the condition's severity. AN can be classified into two subtypes: 1) restricting (characterized by restrictive eating, dieting, fasting, and excessive exercise) and 2) binge eating/purging. Mortality rates are high, and incomplete recovery puts people at risk of relapse. Clinical guidelines and evidence-based approaches prioritize therapeutic renourishment and weight restoration as primary treatment goals.[2] Focal cortical dysplasia (FCD) is characterized by cerebral cortex dysplasia and is a prevalent cause of epilepsy that is resistant to medication. However, there has never been a report of FCD related to AN.[1] This is the first incidence of AN in a 12-year-old male diagnosed with FCD-type 2 using head magnetic resonance imaging (MRI).

Case Presentation:

A 12-year-old male with no medical history was concerned about lower abdomen distention and reduced his food consumption 3 months before the appointment. He exercised repeatedly based on his dietary intake. Despite decreasing weight, he expressed dissatisfaction with his abdominal fat and a desire to reduce it. He was extremely lean at his first consultation, with a body mass index (BMI) of 11.5 kg/m^2. To enhance his weight, it was recommended to take enteral nutritional supplements while diagnosing underlying problems using MRI and abdominal ultrasound. However, he became disoriented and required emergency hospitalization 7 days following his first visit. He was 145 cm tall, weighed 20.9 kg (BMI: 9.9 kg/m^2), and had a Glasgow Coma Scale (GCS) of $E_3V_4M_6$ for impaired awareness when he was admitted. Hypercapnia, hypoglycemia, and increased levels of creatinine, blood urea nitrogen, and transaminases were found by blood gas analysis and blood testing. On the second day of admission, the patient had impaired consciousness, GCS of $E_1V_1M_1$, and low blood pressure, prompting transfer to the intensive care unit (ICU). Severe hypoglycemia and dehydration were recognized as the causes of the circulatory collapse and impaired awareness.

His consciousness improved when they were corrected, and on the third day of his hospital stay, he was moved from the intensive care unit to the general ward. His hepatic transaminases peaked on the fourth day of his hospital stay. Additionally, coagulopathy was noted (prothrombin time (%): 28%). A condition like acute liver failure was taken into consideration. He received enteral nutrition and glucose infusion as part of liver support therapy, and his AST and ALT values steadily improved. When his general condition stabilized six weeks after hospitalization, a head MRI scan was conducted. T2-weighted and fluid-attenuated inversion recovery images revealed a concentrated high-signal region close to the left hippocampus, extending from the cortex to the white matter of the fusiform gyrus to the parahippocampal gyrus (Figure 1). The imaging results were typical of FCD type II, and there was no contrast enhancement. There were no notable findings from cerebral blood flow scintigraphy or electroencephalography. His full-scale intelligence quotient (IQ), as determined by the Wechsler Intelligence Scale for Children, Fourth Edition, was 119, which was higher than usual for his age group. According to the synthetic House-Tree-Person test, he lacked positive thinking and personality richness and had low internal energy. The Rorschach test revealed that he exhibited excessive vigilance and anxiety. This suggests a risk of impaired psychological performance in complicated or ambiguous situations. His mother also observed that he was very precise, following the school schedule even on days when classes weren't in session, which may be indicative of ASD tendencies. His communication with others was distinctive, but not to the point that it interfered with his day-to-day activities or satisfied the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria A of ASD. Due to the patient's poor general health upon admission, his caloric intake was gradually raised by intravenous nutrition before beginning enteral nutrition. Titrating the intake would not result in electrolyte problems, which are known to happen as refeeding syndrome. Enteral feeding was started and progressively increased following the resolution of intestinal edema, which was confirmed by sonography. Despite not vomiting, he had a slow oral intake and

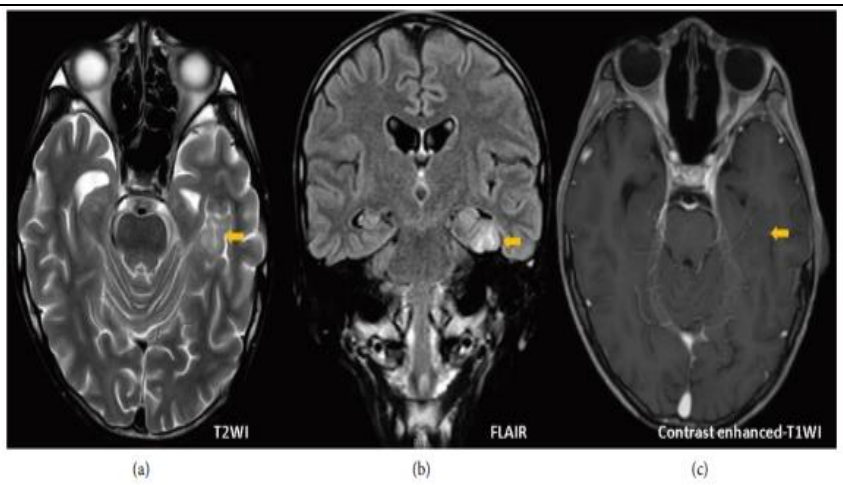


Figure 1: Six weeks following admission, a head MRI showing (a) T2-weighted, (b) FLAIR, and (c) contrast enhanced-T1 weighted (right).
MRI, magnetic resonance imaging; FLAIR, fluid attenuated inversion recovery.

needed to be fed through a nasogastric tube to meet his desired intake. After being advised that his life was at risk due to acute emaciation, the patient steadily increased his oral intake, and the nasogastric tube was removed by week 9. After hospitalization, the BMI dropped to 8.9 kg/m^2. By week 12 of his hospital stay, his BMI had returned to 13 kg/m^2. By the 20^{th} week, the patient had reached 15 and was discharged. He maintained a steady BMI of about 16 kg/m^2. Psychiatric counseling and disease education were started once the patient's physical condition had stabilized. After two years of follow-up, the patient has not experienced any repeat eating disorders or weight loss. Neither the start of epilepsy nor morphological abnormalities on imaging were observed at follow-up.

Discussion:

AN is a potentially fatal illness that damages systemic organs because the patient is severely underweight and malnourished as a result of inadequate nutrition. Female patients are more likely to be impacted, with male patients showing only one-tenth of the frequency reported in female patients. Although there is currently no evidence of FCD and AN co-occurring, the shared factor of ASD might offer a consistent possible explanation. Complications from AN and ASD occur frequently.[1] A previous research indicates that ASD complicates 18% of AN case.[3]

Conclusion:

This was the first case report that suggests a connection between FCD and AN. To determine whether FCD and eating problems are closely related, more cases are required. Additionally, it might contribute to a better understanding of the pathophysiology of eating disorders in children who have tendencies toward ASD.

References:

1. Nemoto H et al. A Case of Anorexia Nervosa with Focal Cortical Dysplasia. Case Rep Psychiatry. 2024;2024(1):7478666.
2. Bulik CM, Carroll IM, Mehler P. Reframing anorexia nervosa as a metabo-psychiatric disorder. Trends Endocrinol Metab. 2021 Oct;32(10):752-761.
3. Watson H. J., Yilmaz Z., Thornton L. M., et al. Genome-wide association study identifies eight risk loci and implications Metabo-psychiatric origins for anorexia nervosa. Nature Genetics . 2019;51(8):1207–1214.

3. An uncommon case of spontaneous bladder rupture in a patient with catatonic schizophrenia

Introduction:

When patients with catatonia experience abdominal pain, psychiatrists should include bladder rupture in the differential diagnosis rather than ignoring urinary retention.

Catatonia is a syndrome associated with mental illnesses and other medical issues. It is described as a collection of symptoms that include a lack of both speech and movement. It can cause agitation, disorientation, and restlessness. Although catatonia is known to be linked to schizophrenia and other mental illnesses like mania and depression, there have been several case reports of medical conditions like liver transplantation, hyponatremia, and cerebral venous sinus thrombosis that can lead to catatonia.[1] In schizophrenia, catatonia is a psychiatric emergency that frequently results in an excessive level of sympathetic nervous system activity. Urinary retention in catatonia is frequently underestimated but can have serious effects. The percentage of bladder ruptures that occur spontaneously is just 3.4%. There is a very small chance of spontaneous bladder rupture—roughly 1 in 50,000–126,000.[2] In this case, a patient with catatonia experienced bladder rupture due to urinary retention, which was successfully treated by an early transport to an emergency hospital.

Case Presentation:

A 40-year-old female patient was diagnosed with schizophrenia. She had a cesarean section but no other noteworthy medical history. She was not a regular smoker or drinker. There was no family history of psychiatric disorders. The patient started suffering auditory-verbal hallucinations and delusions in her 20s and was treated with antipsychotics for several years. However, discontinuing the drugs resulted in a relapse and catatonia. Upon admission, the patient displayed a catatonic stupor. An IM injection of 10 mg of diazepam relieved the patient's stupor, followed by a 3-day continuous intravenous infusion of 5 mg haloperidol for acute psychosis, which improved her symptoms. She improved her communication skills and gained the ability to consume food. She was prescribed aripiprazole at a daily dose of 12 mg. On the eighth day of hospitalization, the patient's symptoms worsened, including auditory hallucinations, delusions, and unusual behaviors such as walking with closed eyelids, staring, tweeting, and fluttering hands. The patient's symptoms improved after taking haloperidol (5

mg IM) and levomepromazine (5 mg IM), increasing the dose of aripiprazole to 24 mg/day, and administering flunitrazepam (1 mg/day). On the tenth day of hospitalization, the patient reported significant abdominal pain. The patient presented with mild rhabdomyolysis at admission. On the third day of hospitalization, CT scans of the chest and abdomen revealed no abnormalities, except for a significant amount of urine accumulated in the bladder. The patient experienced significant abdominal pain and had a conscious level of 15 on the Glasgow coma scale. The physical examination indicated abdominal distension, negative guarding, and positive rebound tenderness. A CT scan performed at the referring institution revealed a bladder rupture (Figure 1). The radiological report showed a thick bladder wall, no expansion, a discontinuity in the posterior wall, and significant ascites. Additionally, abdominal paracentesis confirmed that the contents of her ascites matched the results of her urine test. The patient was diagnosed with spontaneous bladder rupture, as there was no history of trauma. In the urology department, emergency repair surgery was carried out. A median lower abdominal incision revealed a 5 cm-long rupture in the bladder's posterior wall. After cutting the damaged tissue, the bladder wall was sutured with two layers of 3-0 monofilament absorbable thread and intermittent sutures. For conventional infection management during the preoperative phase, 3 g/day of ampicillin/sulbactam was given for 3 days. During postoperative care, the patient received an indwelling urinary catheter to manage voiding. After surgery, the patient was readmitted to the hospital and was discharged one month later after gaining stability on aripiprazole at up to 30 mg/day. The patient's Bush-Francis Catatonia Rating Scale score improved from 29 at admission and 26 after symptoms worsened to 0. After symptom alleviation, the patient's vital values stabilized at 112/86 mmHg, 80 beats/min, and 36.0°C, respectively, compared to the acute psychiatric phase. Catatonia may have caused hyperactivation of the sympathetic nervous system during the acute phase of psychosis. After discharge, she needed to follow up with the psychiatry and urology departments at a nearby hospital. Given her bladder health, the possibility of exaggerated symptoms, and her

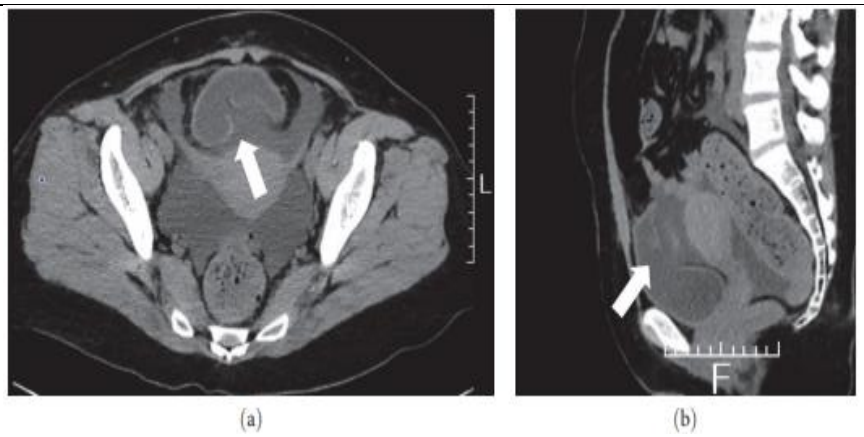


Figure 1: Computed tomography images of the bladder indicate a rupture from the posterior wall: (a) axial and (b) sagittal views. Arrows point to the ruptured wall.

preferences, decided to keep the catheter in place for ongoing use with planned monthly replacements.

Discussion:

Bladder rupture can be fatal if not diagnosed and treated promptly. Early transfer from psychiatric health facility to the emergency room, contributed to a successful outcome. In the present case, patient's bladder rupture may have been caused by a certain illness that is characterized as catatonia.[2] Numerous reasons for spontaneous bladder rupture have been noted, including alcohol usage, surgeries, and tumors; some of them include cases of depression and schizophrenia.[3,4] Bladder rupture in individuals with psychiatric disorders is typically caused by urine retention from psychotropic medicines.[4]

Conclusion:

This case report highlights the importance of considering bladder rupture in patients with catatonia who have abdominal pain, rather than ignoring urinary retention.

References:

1. Edinoff AN et al. Catatonia: Clinical Overview of the Diagnosis, Treatment, and Clinical Challenges. Neurol Int. 2021 Nov 8;13(4):570-586.
2. Miyakoshi M et al. Spontaneous Bladder Rupture in a Catatonic Schizophrenia Patient. Case Rep Psychiatry. 2023 Nov 21;2023:4277372.
3. Reddy D, Laher AE, Lawrentschuk N, Adam A. Spontaneous (idiopathic) rupture of the urinary bladder: a systematic review of case series and reports. BJU international. 2023 Jun;131(6):660-74.
4. Zhang Y et al. Spontaneous rupture of urinary bladder: two case reports and review of literature. Frontiers in Surgery. 2021 Nov 2;8:721705.

4. Case report on Psychosis in Hyperparathyroidism

Introduction:

Recognizing the association between primary hyperparathyroidism and psychiatric symptoms has crucial implications for diagnosis and

Psychosis is a complicated psychiatric illness with multiple causes, including primary psychiatric disorders, substance abuse, environmental variables, and organic reasons. Hyperparathyroidism is defined by high parathyroid hormone production and subsequent hypercalcemia, is an uncommon but significant biological cause of psychosis. Symptoms vary depending on the level of hypercalcemia.[1] In recent years, the incidence of psychosis among adolescents has increased.

According to the World Health Organization (WHO) estimates, around one in every five children and adolescents globally suffers from a mental condition, with half exhibiting symptoms before the age of 14.[2] Primary hyperparathyroidism (PHPT) and the resultant hypercalcemia have been linked to psychosis.[1] This case report features a 28-year-old cannabis user who required psychiatric care due to emotional instability, unusual conduct, and difficulty doing daily tasks.

Case Presentation:

A 28-year-old man with no past psychiatric treatment was admitted to the hospital by his wife after experiencing mood instability, psychosis, unusual behavior, and restricted everyday activities for three weeks. The patient previously experienced a similar episode in college, which resolved after a period of sleep and did not require psychiatric treatment. Psychosocial stresses, such as his mother's recent death, his wife's pregnancy, relocation, and lack of social support, were identified. The mother had a history of alcoholism and bipolar disorder. The patient expressed no suicidal or homicidal thoughts, or auditory or visual hallucinations, and reported feeling safe in the hospital. The patient exhibited intermittent and agitated conduct, including yelling and cursing at his family, which was unusual for him. The patient's previous experience had a significant influence on his emotional well-being, causing him to appear upset and socially withdrawn during a holiday gathering with family, which was rare. During his voluntary hospitalization, the patient demonstrated disorganized thinking and conduct, including heightened distrust and paranoia toward personnel and peers on the unit. He frequently complained that the hospital was attempting to poison him. He paid special attention to other patients and nurses throughout the day. Initially, the patient avoided group therapy due to social anxiety. He reported intense thoughts about "an important symbol," as well as a lack of emotional responses. Despite his frequent use of mathematical symbols since admission, he struggled to express his current emotions. The patient occasionally exhibited rapid blinking during talks with staff. The patient was diagnosed with Bipolar 1 illness, experiencing manic episodes and psychotic conduct. The goal was to identify any underlying medical conditions that could be causing these symptoms. In the inpatient psychiatric facility, the patient was

prescribed low dose olanzapine, which was gradually increased to 20 mg nightly to manage his mood and psychotic symptoms. Lorazepam was prescribed at 0.5 mg twice daily to reduce anxiety. The patient's condition improved, and he reported feeling "pretty good." His sleep and eating returned to normal, and he denied any depression, anxiety, or psychotic symptoms. He communicated regularly with his wife and father but struggled with self-evaluation and a lack of satisfaction. Despite the difficulty, he followed his medication schedule and was willing to change as needed by the treatment team. He was discharged from the inpatient facility. The patient's substance usage history was reviewed, including a positive urine drug screen for cannabis. The patient had been smoking cannabis for over a decade. During treatment, it was noted that continued cannabis usage may worsen psychiatric symptoms. He was encouraged to stop using drugs and to continue following up with an outpatient substance use disorder treatment provider. The patient's medical workup revealed persistent hypercalcemia, with plasma calcium levels at 12.5 mmol/L (reference range, 8.5-10.5 mg/dL). The patient had a history of kidney stones, which could be attributed to underlying endocrine abnormalities. Laboratory results showed increased levels of parathyroid hormone (PTH) at 112.7 (reference range, 10-65) pg/mL and a positive toxicological screen for cannabis. After reviewing the findings, an endocrinologist recommended a parathyroid scan to confirm the suspected PHPT diagnosis. After consulting with the endocrinology team, cinacalcet was prescribed at 30 mg each evening with dinner. A SPECT scan of his parathyroid revealed activity at the left thoracic inlet, confirming PHPT (Figure 1). A CT scan of the patient's head and an MRI of his brain were used for diagnosis and therapy planning. Within the fourth ventricle, there were two tiny regions of calcification measuring around 7 mm. After his inpatient psychiatric stay, he was encouraged to seek outpatient surgery to address hypercalcemia. After consulting with an endocrinologist, he underwent a parathyroidectomy. After surgery, the serum calcium levels returned to normal. Furthermore, the patient's mental state improved significantly. The patient did not get outpatient psychiatric care and had no new medication refills for almost 6 months following

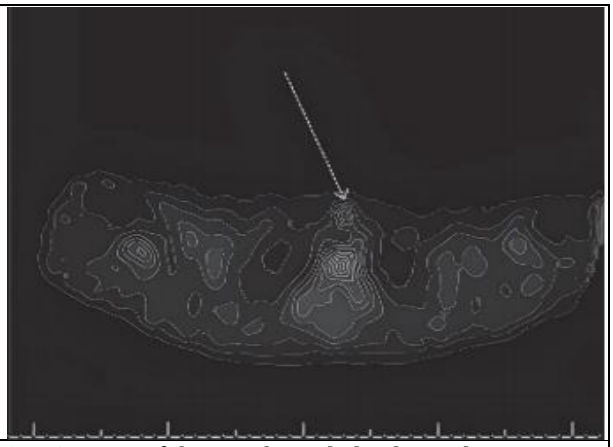


Figure 1: A SPECT scan of the parathyroid gland reveals activity at the left thoracic inlet.

his inpatient hospitalization. There was no information on the patient's post-surgery use of cannabinoids, making it unclear if this changed.

Discussion:

This case report highlights the significance of recognizing hyperparathyroidism as an intrinsic etiology of psychosis in young patients, even with substance use disorders. Although olanzapine helped treat the patient's symptoms, research suggests that surgical treatment of PHPT can significantly reduce or eliminate symptoms.[1] A previous study showed that to treat paranoid psychosis and delusions caused by hypercalcemia, blood calcium levels should be normalized, or a parathyroid adenoma should be removed. Patients with significant psychiatric symptoms, including psychosis, should undergo parathyroidectomy surgery.[3]

Conclusion:

This case highlights the complex association between hyperparathyroidism, hypercalcemia, and neuropsychiatric symptoms, including psychosis. Recognizing the link between PHPT and psychiatric symptoms has important implications for diagnosis and therapy, even though the exact pathophysiological pathways are yet unknown. Early detection of hyperparathyroidism and its therapy through endocrinology are crucial for better patient outcomes.

References:

1. Murphy RJ, Paul S, Primelo R. Parathyroid Paranoia: Unveiling Psychosis in Hyperparathyroidism. Case Rep Psychiatry. 2024 Jun 24;2024:8126125.
2. Cao XJ, Liu XQ. Artificial intelligence-assisted psychosis risk screening in adolescents: Practices and challenges. World J Psychiatry. 2022 Oct 19;12(10):1287-1297.
3. Otsuki K, Izuhara M, Miura S et al. Psychosis in a primary hyperparathyroidism patient with mild hypercalcemia: A case report. Medicine. 2021 Mar 26;100(12):e25248.