Psychiatric Case Series

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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

Case report on Auditory Charles Bonnet syndrome and psychosis

1. **Case Report on the Psychiatric Presentation of a Severe Vitamin B12 Deficiency Related to Biermer's Disease**
2. **Case Report on Panic Disorder and Agoraphobia in a Patient with Post-Trauma Epilepsy**
3. **Case on acute mania associated with neuropsychiatric symptoms of arteriovenous malformation**
4. **Case report on spontaneous pneumomediastinum in a middle-aged female patient with schizophrenia**

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 the Psychiatric Presentation of a Severe Vitamin B12 Deficiency Related to Biermer's Disease

Introduction:

Vitamin B12 insufficiency can cause cognitive, mental, and neurological symptoms, with psychiatric symptoms being the most commonly diagnosed.

Biermer disease, commonly known as pernicious anemia, is a megaloblastic anemia caused by a lack of vitamin B12 (cobalamin), which is essential for blood cell formation, DNA synthesis, and brain functions.[1] Vitamin B12 insufficiency has been linked to a variety of neuropsychiatric symptoms, including cognitive deficits and severe psychiatric disorders (Table 1). Vitamin B12 deficiency can be caused by inadequate dietary intake, malabsorption disorders, or certain medicines. Vitamin B12 insufficiency can cause subtle neurological and mental symptoms that may resemble other medical conditions, making diagnosis difficult.[2] This was a case of patient who had neuropsychiatric symptoms and vitamin B12 encephalopathy that was caused by biermer's illness.

Neurological symptoms	Gastrointestinal symptoms	Psychological symptoms
• Memory loss; • Lack of concentration; • 'Brain fog'; • Pins and needles; • Reduced vibration sense; • Confusion; • Nominal aphasia; • Unexplained headaches; • Sleep difficulties; • Dizziness; • Balance problems; • 'Burning feet'; • Being unable to stand with eyes closed.	• Diarrhoea; • Indigestion.	• Irritability; • Impatience; • Mood swings; • Suicidal ideation; • Depression; • Anxiety; • Psychosis.

Table 1. Pernicious Anaemia: Symptoms

Case Presentation:

A 57-year-old female patient with type 2 diabetes, managed with insulin, was sent to clinic for a four-month-long developing behavioural disturbance. Symptoms include separation from family, social isolation, apathy, low mood, spasmodic crying and difficulty sleeping. The patient had gait problems, including restricted walking perimeter, tremors, and urine incontinence, as well as anorexia, asthenia, weight gain, and headaches without vomiting or fever. The patient denied any alcohol use or malnutrition. The patient's clinical examination revealed confusion in time and place, indifference, lack of facial emotions, slightly stained conjunctivae, and android obesity (BMI of 35). Eye closure worsened the ataxic gait. Romberg's sign was negative. The patient experienced spastic hypertonia on the left side, with

fast reflexes in all four limbs and bilateral Babinski sign. Muscle strength remained intact. Sensory functioning was normal, and no coordination issues were noted. Oculomotor examination revealed no ophthalmoplegia or nystagmus. Assessment of cognitive processes indicated attention and memory abnormalities, indicating anterograde amnesia. Brain MRI revealed symmetrical T2 and FLAIR hyperintensities in the periventricular and periaqueductal white matter, along with bilateral T1 hyperintensity in the striatum (Fig. 2). The EEG showed a widespread slowdown of background activity, without any paroxysmal abnormalities. Metabolic panel findings showed normal levels of thiamine, vitamins B6, and B9, but very low vitamin B12 levels (62.50 pg/ml) linked with macrocytic anemia. The metabolic and infectious workup showed normal values. Upper gastrointestinal endoscopy detected erosive pangastritis and positive anti-intrinsic factor and anti-parietal cell antibodies, indicating Biermer's illness. The patient was treated with a daily intramuscular injection of 5000µg of hydroxocobalamin for 10 days, followed by weekly doses for a month, and then monthly injections. After three months of vitamin B12 replacement therapy, the patient's mental symptoms improved and limb tremors resolved.

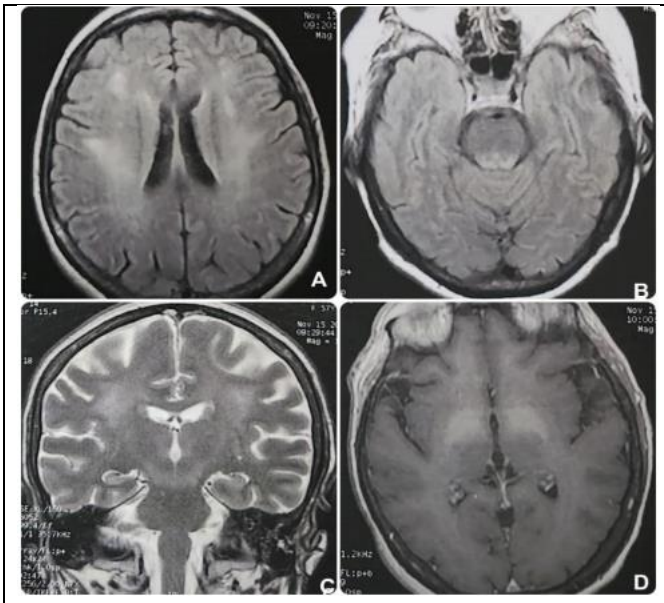

Figure 2. Periventricular and periacqueductal white matter hyperintensities noted in cerebral MRI in axial T2 FLAIR (A,B) and coronal T2 (C). Axial T1 sequence (D) demonstrates bilateral pallidal hyperintensity

Discussion:

Vitamin B12 is essential for the proper growth and function of the central nervous system, including myelination. Vitamin B12 insufficiency can cause neuropsychiatric symptoms across several cognitive and affective domains. Cognitive impairments often affect memory, attention, executive function, and processing speed.[2] Psychiatric symptoms can vary from mild mood changes like irritation and apathy to severe conditions like depression, psychosis, and dementia.[3] Neurological symptoms might include peripheral neuropathy, myelopathy, and optic neuropathy.[4]

Conclusion:

Vitamin B12 insufficiency can cause cognitive, psychiatric, and neurological symptoms. Psychiatric symptoms were frequently the most prominent in diagnosis. This case study highlights the need of early detection and treatment of vitamin B12 deficiency to prevent long-term consequences and improve outcomes for affected persons. A multidisciplinary approach is necessary for accurate diagnosis.

References:

1. Roger ND, Yannick FF, Wilson NN, Mathurin KP, Firmin AA, Pierre CS. Biermer Disease in an Unusual Neurological Presentation without Anemia: A Case Report. Open Journal of Gastroenterology. 2022 May 31;12(5):129-35.
2. Yassine, El Adraoui, et al. Psychiatric Presentation of a Severe Vitamin B12 Deficiency Associated with Biermer's Disease: A Case Report. International Journal of Medical and Pharmaceutical Case Reports.2024 17 (3):1-4.
3. Mota A, Lázaro M, Alves R, Moreira A, Nobre M. Neuropsychiatric manifestations of vitamin B12 and folate deficiencies: Data from an inpatient psychiatric department. European Psychiatry. 2016 Mar;33(S1):S371-.
4. Wolffenbuttel BHR, Wouters HJCM, Heiner-Fokkema MR, van der Klauw MM. The Many Faces of Cobalamin (Vitamin B_{12}) Deficiency. Mayo Clin Proc Innov Qual Outcomes. 2019 May 27;3(2):200-214.

2. Case Report on Panic Disorder and Agoraphobia in a Patient with Post-Trauma Epilepsy

Introduction:

Epilepsy is a chronic neurological illness characterized by a range of seizure patterns. Epilepsy patients have a greater risk of impairment, poor quality of life, stigma, and early death compared to the general population. Approximately 20-30% of people with epilepsy have psychiatric comorbidities. Psychotic illness was 6-12 times more prevalent in this demographic compared to the general population. Its complications may affect epilepsy care, leading to decreased health-seeking behavior, poor adherence to antiepileptic drugs, increased seizure frequency, and poor surgical outcomes.[1] Depression and anxiety are common psychiatric symptoms that go undiagnosed and untreated, negatively impacting quality of life. Also, Epilepsy was frequently accompanied by psychiatric symptoms including anxiety and mood disorders. Epilepsy stigma and negative perceptions can impair the patient's prognosis and make social reintegration harder. Therefore, increased awareness of psychiatric problems in people with epilepsy was essential.[2] This case described a post-traumatic epilepsy patient with panic disorder and agoraphobia.

The patient was diagnosed with post-traumatic epilepsy, panic disorder, agoraphobia, and a moderate depressive episode with somatic symptoms and treated with antiseizure medication, sleeping pills, antidepressants, cognitive behavioral therapy, psychotherapy, relaxation therapy, and family psychoeducation.

Case Presentation:

A 31-year-old adult male patient complained of anxiety and chest palpitations. He is a neuropsychiatric patient with post-traumatic epilepsy. The patient is currently experiencing regular symptoms of anxiety and a pounding chest. When nervous, he gets symptoms such as shortness of breath, sweating, palpitation, and dizziness, which can lead to stiffness and seizures. The patient stated that he began to have similar symptoms in 2014, when his father died. According to the patient, only his father understands his condition. After the father died, the patient married. Although the patient feels calmer when his wife is there, he still experiences anxiety occasionally. The patient feels depressed and anxious as a result of his lack of improvement. The patient's seizures have decreased significantly to 3-4 times per month. The patient has anxiety on a daily basis and was hesitant to engage in activities without his wife's presence. The patient was treated collaboratively by neurology and psychiatry colleagues

due to behavioural changes (anxiety) following the death of his father. The patient had experienced seizures following an injury in 2008. Since 2014, the patient has been receiving treatment from a psychiatrist. He has post-traumatic epilepsy and diagnosed with panic disorder, agoraphobia, and mild depression with somatic symptoms. The following stages of management have been completed: 1) Following a psychiatric history, a manual psychometric examination of mood and anxiety was conducted using the Hamilton Anxiety Rating Scale (35 for severe anxiety) and the Hamilton Depression Rating Scale (21 for moderate depression, HDRS score 18-24). The patient was given 1x50 mg Sertraline in the morning and 2x10 mg Clobazam orally in the evening, 2) A neurology colleague examined the patient who had been complaining about seizures for six years. Seizures continue to occur, although with a unique pattern. The pattern has shifted since 2014, with regular emotions of anxiety. In 2013, electroencephalogram (EEG) findings indicated functional abnormalities in the left temporal area, potentially leading to epilepsy. The CT-Scan revealed gliosis in the right frontotemporal cortex, a right temporal bone defect, and no evidence of intracerebral bleeding. The patient has been diagnosed with post-traumatic epilepsy. The patient received 500mg-500mg-1000mg divalproex sodium treatment, 3) A vital sign assessment revealed a body weight of 92 kg, blood pressure of 140/92 mm Hg, pulse rate of 100/minute, RR of 22/minute, and no signs of fever, anemia, jaundice, cyanosis, or dyspnea. There was some ptosis on the right eyelid. The abdomen was flexible, with normal bowel sounds and peristaltic movements. 4) Despite receiving Cognitive Behavioral treatment, relaxation treatment, and ventilation/catharsis, the patient failed to express his emotions on several occasions. 5) Family members, including uncles, receive psychoeducation to monitor treatment progress and side effects, prevent self-harm, report suicidal thoughts, improve communication, monitor seizure intensity, keep seizure records, and identify potential triggers. The outcomes noticed were from physical, psychological, social, and interpersonal dimensions. The patient reported fewer problems after being given sedatives by hospital staff. Adding analgesics and multivitamins from neurology, as well as antidepressants from psychiatry, which have been linked to tension-type headaches (TTH), was expected to reduce symptoms greatly. After less than a week of therapy, the patient reported decreased headaches and improved sleep quality. The healthcare team provides family psychoeducation to encourage patients to continue their therapy successfully. Communicate with neurology colleagues that the patient's panic disorder, agoraphobia, and depression may overlap with epilepsy, necessitating a comprehensive assessment and treatment plan. Communicate psychiatric medication to prevent overlap with other therapies.

Discussion:

Epilepsy is a disorder that commonly affects children. Factors contributing to this condition include head trauma, brain tumors, brain inflammation, a history of pregnancy issues, and febrile seizures. Social phobia can be related to epilepsy. Another kind of phobia that was rarely mentioned in relation to epilepsy was seizure phobia. The ILAE recognizes seizure phobia, social phobia, and agoraphobia as unique anxiety disorders associated with epilepsy.[2] A previous study on 69 epilepsy patients found a 27.5% incidence of seizure phobia. Female gender, a history of severe depressive episodes, and post-traumatic stress disorder all have significant associations. This finding confirmed the significance of psychiatric intervention alongside neurology epilepsy care to improving patients' quality of life and facilitating reintegration into society.[3]

Conclusion:

The patient was diagnosed with post-traumatic epilepsy, panic disorder with agoraphobia, and moderate depression with somatic symptoms. The patient has had CBT, supportive psychotherapy, family psychoeducation, psychopharmaceuticals, and communication with neurological colleagues on their psychological status and treatment plan. Psychiatric problems are comorbidities that frequently occur in epilepsy patients. To prevent or minimize the occurrence of severe psychiatric disorders and recurrent illness, interventions and collaboration with neurology colleagues, as well as effective communication with patients and their families, are crucial.

References:

1. Lu E, Pyatka N, Burant CJ, Sajatovic M. Systematic Literature Review of Psychiatric Comorbidities in Adults with Epilepsy. J Clin Neurol. 2021 Apr;17(2):176-186.
2. Fitriani F, Putra GF, Syulthoni ZB. Panic Disorder with Agoraphobia in Post-Trauma Epilepsy Patient: a Case Report. Journal of Medicine and Health Technology. 2024 May 30;1(1):9-15.
3. Weiss A, Canetti L, David SB, Reuveni I, Ekstein D. Seizure phobia: A distinct psychiatric disorder among people with epilepsy. Seizure. 2022 Feb;95:26-32.

3. Case on acute mania associated with neuropsychiatric symptoms of arteriovenous malformation

Introduction:

Differentiating between primary psychiatric conditions and secondary medical causes in acute mania patients might greatly affect their management and prognosis.

Mania, an acute psychiatric condition, has been reported from long ago. This condition has a lifetime incidence rate of 0.8% to 1.6%, resulting in considerable morbidity and mortality.[1] Organic causes of acute mania should be addressed, especially in cases with unusual presentation, such as a lack of prior psychiatric history or age beyond 40. Studies indicate that mania was caused by brain lesions that impact sleep, hunger, energy, and libido regulation. The limbic system, thalamus, hypothalamus, frontal lobe, temporal lobe, and basal ganglia all interact in intricate ways. Toxic insults can disturb the balance of neurotransmitters and chemicals necessary for sustaining neurological and psychological equilibrium, in addition to structural lesions affecting these regions. There were limited evidence that mania can be caused by vascular abnormalities, particularly arteriovenous malformations (AVM). [2] This case report described a 46-year-old male patient with a history of left AVM who had severe mania and behavioral abnormalities after radiation therapy.

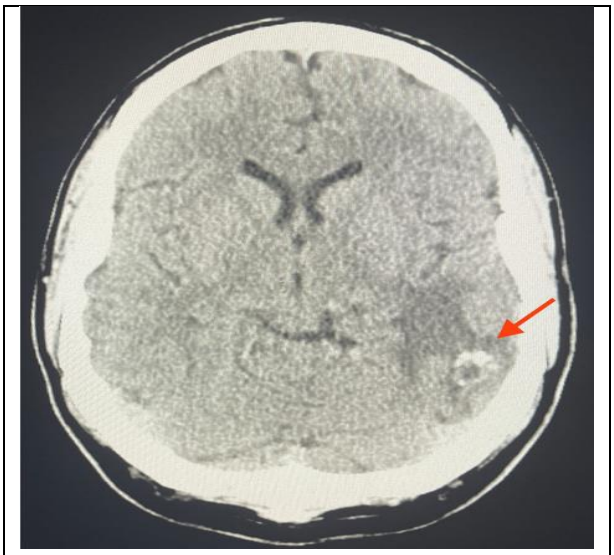

Figure 1. Non-contrast CT conducted during admission revealing a left temporal lobe AVM (red arrow), with no evidence of acute infarctions, intracranial bleeding, or tumor.

Case Presentation:

The patient, a 46-year-old male with a medical history of hypertension, gastroesophageal reflux disease, hyperlipidemia, and spinal fractures from a car accident, was presented to the emergency department (ED) of a local community hospital by police after being restrained outside his wife and children's home. His wife said that he had been beating on the door of the house, wanted to see his children, after she had left for safety reasons a few days previously. The patient's wife reported a diagnosis of an AVM in the left temporal lobe a year before admission. He had radiation therapy the same year with no issues. Following

therapy, he had a range of symptoms that were unexpected given his baseline. Symptoms included irritation, mood fluctuations, frequent anger toward children, and suicidal ideation. The wife reported that he was obsessed about her adultery and physically hostile to her. He frequently spent days without sleeping. This conduct persisted, prompting the wife to take the children to a relative's place. Prior to being diagnosed with AVM, the patient experienced seizures and began taking levetiracetam. He had no history of psychiatric disorder, but acquired depression due to his AVM and began taking escitalopram. After a few months, his neurologists switched him from levetiracetam to brivaracetam due to concerns about potential mood changes. The patient's escitalopram dose was raised from 10 mg to 20 mg nine days before presenting to the ED. He had a history of taking beer four to five times per week and vaping cannabis twice per week. The neurological examination and initial laboratory values were within the normal range. Laboratory testing were conducted to rule out frequent causes of subsequent mania and psychosis, including drug use and metabolic abnormalities. Serum and urine toxicology screenings detected marijuana but not alcohol, salicylates, acetaminophen, or other recreational drugs. The patient was transferred from the ED to psychiatric emergency services (PES) for further examination and imaging. During the admission, brain imaging was repeated and discussed between attendings at both institutions. The results of the non-contrast CT scan and contrast MRI were consistent with previous studies of an AVM in remission after radiation treatment. The non-contrast CT revealed the left temporal lobe AVM, with no evidence of acute infarctions, cerebral bleeding, or mass (Figure 1). The contrast-enhanced MRI revealed that the treated AVM in the left temporal lobe was less visible than in prior imaging at the other institution. Additionally, the left temporal perilesional edema and mass effect persisted but diminished (Figure 2). During the PES, he continued to display acute emotional instability, paranoia, and episodes of aggression. He cried frequently over his situation and had a negative attitude on life, including verbal violence towards his wife. The patient stayed in PES for three days and gradually improved. After being transported to the medical unit, he was monitored for stability and treatment plans. After consultation with neurology, neurosurgery, and

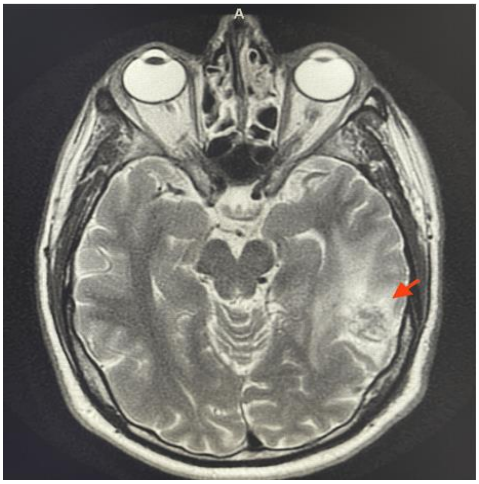

Figure 2. MRI with contrast conducted during hospitalization revealing post-radiation AVM status in the left temporal lobe, including peri-lesional edema and mass effect (red arrow).

medicine, the patient was discharged after a few days. His attitude had normalized, and he and his wife felt secure returning home. The patient's escitalopram dosage was lowered to 10 mg, and valproate was introduced for anti-epileptic and mood stabilizing effects. He was scheduled for an outpatient follow-up with his neurologist at another facility.

Discussion:

Few cases have been described of acute mania in individuals with an AVM and no prior psychiatric history. This patient's mania may be due to the temporal-parietal cortex becoming accustomed to the vascular malformation. After treatment, increased blood flow to the brain may have altered activity levels in surrounding regions, leading to the presenting symptoms.[2] Radiation treatment to reduce the AVM may have changed blood flow patterns to the brain, leading to hypoactivity and hyperactivity in areas linked with manic symptoms.[3] A previous study compared bipolar disorder patients' regional cerebral blood flow (rCBF) to that of healthy individuals as a negative control and patients with major depressive disorder as a positive control. The study demonstrated a substantial increase in rCBF to the left temporal lobe and reduction in rCBF to the right hippocampus. The regional cerebral blood flow velocity (rCBFV) fluctuated correspondingly.[4]

Conclusion:

The present case emphasized the necessity of examining medical causes of psychiatric symptoms, especially in emergency situations. Identifying the primary psychiatric disorders with secondary medical reasons for acute mania patients might greatly affect their therapy and prognosis. Further study was needed to better understand long-term results and patient prognoses for arteriovenous malformations, given their complicated biology and the impact of therapies such as radiation. To diagnose and manage acute mania, doctors should examine organic causes, especially in individuals with unusual presentations.

References:

1. Elias A, Thomas N, Sackeim HA. Electroconvulsive therapy in mania: a review of 80 years of clinical experience. American Journal of Psychiatry. 2021 Mar 1;178(3):229-39.
2. Villa NAE, Espiridion ED. Neuropsychiatric Manifestations of Arteriovenous Malformation: A Case of Acute Mania. Cureus. 2024 Apr 15;16(4):e58297.
3. Lee I, Nielsen K, Nawaz U, Hall MH, Öngür D, Keshavan M, Brady R Jr. Diverse pathophysiological processes converge on network disruption in mania. J Affect Disord. 2019 Feb 1;244:115-123.
4. Wang Y, Liu X, Li P, Zhou H, Yang L, Zheng L, Xie P, Li L, Liao DJ, Liu Q, Fang D. Regional Cerebral Blood Flow in Mania: Assessment Using 320-Slice Computed Tomography. Front Psychiatry. 2018 Jul 4;9:296.

4. Case report on spontaneous pneumomediastinum in a middle-aged female patient with schizophrenia

Introduction:

Spontaneous pneumomediastinum (SPM) should be considered as a differential diagnosis in schizophrenia patients who have unexplained chest discomfort, neck pain, or dysphagia. A chest CT scan may help in diagnosis.

Spontaneous pneumomediastinum (SPM) is an uncommon condition characterized by the presence of free air in the mediastinum without any obvious cause, such as chest trauma. It has been suggested that the Macklin effect—a sudden rise in alveolar pressure—was responsible for the pathophysiology of SPM by rupturing the alveoli.[1] Symptoms including chest discomfort, neck pain, and difficulties swallowing can be confusing and difficult to diagnose. SPM was linked to psychiatric diseases, including anorexia nervosa was reported previously. This was the first known case of SPM in schizophrenia.[2] This case report involved a middle-aged female patient with schizophrenia who had neck discomfort and dysphagia due to SPM.

Case Presentation:

A 60-year-old female patient with schizophrenia and osteoporosis, on olanzapine 20 mg/day and eldecalcitol 0.75 µg/day, attended a psychiatrist five days earlier to report neck and chest pain, trouble swallowing, and food poisoning. The patient was suspected of having a relapse of delusions, but due to her inability to eat for several days, she was admitted to the emergency department. She had a BMI of 17.1 kg/m^2, height of 153 cm and weight of 40 kg. She did not smoke and had no history of drug usage. She did not have a history of asthma, COPD, or other lung illnesses that might cause secondary pneumomediastinum. When she arrived, her consciousness was clear and her vital signs were normal. Although her chest discomfort disappeared, she still experienced a painful throat and difficulty swallowing. The electrocardiogram (ECG) was normal, and the chest X-ray revealed no evident abnormalities. An upper gastrointestinal endoscopy revealed no evident abnormalities. Blood testing indicated a normal white blood cell count and negative C-reactive protein levels. A chest CT scan revealed open air in the normal-sized mediastinum and subcutaneous emphysema in the soft tissue of the dorsal upper chest, indicating SPM (Figure 1). The patient was admitted to the hospital and treated with intravenous fluids. On day 3 of hospitalization, the patient's dysphagia and neck discomfort reduced and she was able to eat. As she swallowed the meal, she lost the belief that it was poisonous. On day 5 of hospitalization, the patient was discharged after a

chest x-ray revealed no abnormalities or recurrence of SPM. At one month post-discharge, she reported no illusions and was eating well.

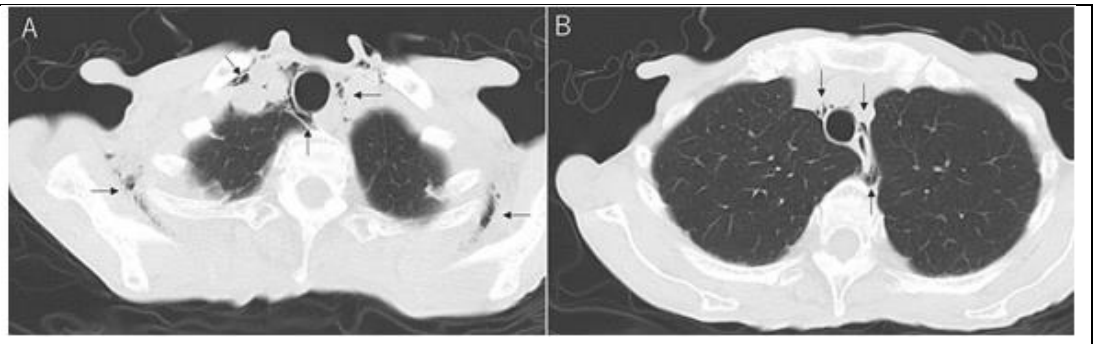

Figure 1: Chest computed tomography (A) The picture around the lung apex shows open air in the mediastinum and subcutaneous emphysema in the back's soft tissues. (B) Free air is visible from the spine to the mediastinum that surrounds the bronchi.

Discussion:

This was the first case of SPM in a schizophrenic patient presenting with neck discomfort, chest pain, and dysphagia. In the present case, a chest CT scan revealed open air in the normal-sized mediastinum and subcutaneous emphysema in the dorsal upper chest, resulting in the diagnosis of SPM.[2] Amari K et al. showed that chest CT scans can identify underlying causes of dysphagia and chest symptoms, such as a tracheal foreign body in a patient with schizophrenia.[3]

Conclusion:

SPM is an uncommon condition that causes free air in the mediastinum without any known cause. This case of SPM in schizophrenia is unique in two ways: (1) it occurred at a higher age than normal, and (2) the onset was caused by a shift in mediastinum pressure and gas inflow. SPM is primarily associated with male adolescents, but thin middle-aged females were also at risk due to lower mediastinal pressure. For schizophrenia patients with unexplained chest discomfort, neck pain, and dysphagia, SPM should be evaluated as a differential condition. A chest CT scan may assist in diagnosis.

References:

1. Kumeda H, Saito G, Eguchi T, Hara D, Shimizu K. Clinical features of recurrent spontaneous pneumomediastinum. J Thorac Dis. 2023 Feb 28;15(2):462-471.
2. agamine T. Spontaneous Pneumomediastinum in a Middle-Aged Female Patient With Schizophrenia: A Case Report. Cureus. 2024 Jan 8;16(1):e51887.
3. Amari K, Hirata R, Tago M. Recurrent Transient Hypoxemia in a Schizophrenic Patient Caused by Upper Airway Foreign Body Obstruction by Watermelon. Cureus. 2023 Nov 14;15(11):e48809.