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 hyponatremia caused by psychogenic polydipsia and schizophrenia**
2. **Case report on managing major depressive disorder with shorter-duration hypomanic episodes**
3. **An uncommon case of antiphospholipid syndrome with Neuropsychiatric Characteristics**
4. **Case Report on Concussion-Related New-Onset Bipolar Disorder**

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 hyponatremia caused by psychogenic polydipsia and schizophrenia

Introduction:

Behavioural therapy, water restriction, medications, and salt replacement can all assist in managing psychogenic polydipsia.

Psychogenic polydipsia is a rare disorder characterized by excessive drinking of water. It can cause water intoxication, a potentially fatal situation. Furthermore, it is typically seen in patients with psychotic disorders, particularly schizophrenia, bipolar disorder, and depression with psychotic features. This serious condition occurs in 6% to 20% of psychiatric patients. [1] Psychogenic polydipsia can cause hyponatremia, resulting in neurological complications such seizures, cerebral edema, and even death. Clinicians face challenges in managing hyponatremia and psychogenic polydipsia due to limited therapeutic options available. Fluid restriction is the most effective treatment for this condition. However, it often fails due to compliance issues and excessive thirst. Behavioral therapy effectively treats psychogenic polydipsia and reduces water intake. So, this case report investigated a case of 45-year-old male with schizophrenia and psychogenic polydipsia, including potential management methods and challenges.

Psychogenic polydipsia

Case Presentation:

A 45-year-old single white Caucasian male who came in for psychiatric follow-up. He had Type 2 diabetes and hypothyroidism, which was managed with metformin and levothyroxine. His vital signs showed a height of 5′ 5″, weight of 189 lbs, blood pressure of 110/80 mm Hg, heart rate of 89 beats per minute, and BMI of 31.45. During his presentation, he stated smoking two packs of cigarettes and drinking three gallons of water daily. The patient had a psychiatric history since childhood, but was diagnosed with schizophrenia in his mid-20s. His schizophrenia symptoms included illusions about having 300 children, including two US presidents. He also discussed other business transactions in which he received a substantial sum of money. The patient had auditory hallucinations but tolerated them well. Clozapine was

administered to manage the patient's schizophrenia due to resistance to earlier antipsychotic medications. The patient was stable on the medication and had obtained complete blood tests to determine his absolute neutrophil count. The patient saw a case manager twice a week and participated in the assertive community treatment (ACT) program, receiving supportive assistance. During psychiatric follow-up for paranoid schizophrenia, the patient reported symptoms of dizziness, fatigue, and weakness. The patient had a history of compulsive water drinking, which had previously caused hyponatremic conditions. He had comprehensive metabolic panels (CMPs) to identify low serum sodium levels (Tables 1-2).

Investigation	Patient value	Normal range
Na	126 mmol/L	135-146 mmol/L
K	3.4 mmol/L	3.5-5.3 mmol/L
Cl	89 mmol/L	98-110 mmol/L
RBC	3.92 million/µL	4.2-5.8 million/µL
Hgb	12.5 g/dL	13.2-17.1 g/dL
Hct	36.60%	38.5-50%
MCV	93.4 fL	77-98 fL
Calculated osmolality	255 mos/kg	278-305 mos/kg

Table 1. May 2021 CMP results (Na: sodium; K: potassium; Cl: chloride; RBC: red blood cell; Hgb: hemoglobin; Hct: hematocrit; MCV: mean corpuscular volume; CMP: comprehensive metabolic panel)

The patient's primary care physician previously treated psychogenic polydipsia with salt tablets, which increased serum sodium levels and improved clinical presentation. The patient has been on and off salt medications for years due to low sodium levels. Once his sodium levels returned to normal, he continued to drink excessively, resulting in hyponatremia. The patient has previously tried water restriction and behavioral therapy without long-term success. The patient's mother expressed concern that his low sodium levels could occur again due to his symptoms. The patient did not see an issue with his water consumption and stated that he drinks till he was no longer thirsty. The patient was sent to his primary care physician for treatment of this problem, and he will receive a CMP at his next visit to assess serum sodium levels. If the patient is hyponatremic, he will be treated with salt tablets and water restriction to maintain

proper sodium levels. His symptoms will be followed in future appointments, and he will undergo more CMPs to monitor his sodium levels.

Investigation	Patient value	Normal range
Na	125 mmol/L	135-146 mmol/L
K	3.5 mmol/L	3.5-5.3 mmol/L
Cl	92 mmol/L	98-110 mmol/L
RBC	3.87 million/µL	4.2-5.8 million/µL
Hgb	12.3 g/dL	13.2-17.1 g/dL
Hct	36.00%	38.5-50%
MCV	93.0 fL	77-98 fL
Calculated osmolality	255 mos/kg	278-305 mos/kg

Table 2. CMP results in February 2022 (Na: sodium; K: potassium; Cl: chloride; RBC: red blood cell; Hgb: hemoglobin; Hct: hematocrit; MCV: mean corpuscular volume; CMP: comprehensive metabolic panel)

Discussion:

Psychogenic polydipsia is a potentially fatal medical condition that can cause seizures, cerebral edema, and rhabdomyolysis. There were several approaches to treat psychogenic polydipsia, but none were definitive. The patient's psychogenic polydipsia was treated with the use of salt tablets. Aside from pharmacological treatment, behavioral therapy can be utilized to treat psychogenic polydipsia.[2] In a previous case, Clozapine reduced obsessive drinking in a 68-year-old female with acute psychosis, improving her sodium levels from 113 to 143 mmol/L.[3]

Conclusion:

Psychogenic polydipsia is a life-threatening disorder that can progress to hyponatremia with serious consequences. Screening patients with psychiatric disorders and indications of hyponatremia is crucial for effective management. While behavioral therapy, water restriction, medicines, and salt replacement can help people with psychogenic polydipsia, more research, large-scale studies, and clinical trials are needed to develop a treatment guideline.

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2. Case report on managing major depressive disorder with shorter-duration hypomanic episodes

Introduction:

Major Depressive Disorder (MDD) is among the most debilitating conditions in the world, having a major negative influence on daily functioning, quality of life, employment status, and productivity at work. Researchers face challenges in investigating MDD due to its complicated nature and comorbidity with various chronic and acute illnesses, including physical and psychiatric disorders.[1] Subthreshold hypomania occurs in 30-55 percent of MDD patients. Scholars now consider bipolar disorder to be a continuous spectrum disorder, rather than a binary one. The DSM-5 includes more bipolar disorder categories beyond bipolar I and bipolar II. Despite improvements in clinical categorization, few guidelines support treating subthreshold bipolar disorder due to limited studies. As a result, psychiatrists face challenges in prescribing drugs to these patients.[2] This report described a case of 61-year-old female patient with a 7-year history of major depressive disorder with short-term hypomanic episodes.

For individuals with major depressive disorder who experience shorter-duration hypomanic episodes, mood stabilizers and atypical antipsychotics may be a better option than antidepressants.

Case Presentation:

A 61-year-old female was admitted to the emergency room for an overdose. Thirty years ago, the patient underwent a right ovariectomy for teratoma. She had hyperlipidemia for over 6 years and has been taking atorvastatin 20mg every night. She is also allergic to pollen, dust and mites. Following the death of a loved one 7 years ago, the patient had low mood, frequent crying, anhedonia, lack of energy and strength, back and neck discomfort, decreased appetite, feelings of life was meaningless, and denial of suicidal thoughts and behaviours. She was admitted in a psychiatric hospital, diagnosed with MDD, and prescribed duloxetine 120mg once daily and clonazepam 2mg before bedtime. Treatment successfully relieved her depression symptoms, including low mood and anhedonia. Despite daily medication following discharge from the hospital, duloxetine's effectiveness gradually decreased. Over the previous

7 years, the patient's mood has been variable, ranging from happiness to depression. She exhibited a range of emotions, including happiness and talkativeness, as well as irritability and a desire to consume alcohol. The husband recognized the patient's elevated state, which often lasted a few hours to a day. Over the previous 7 years, physicians prescribed her medications other than duloxetine. She couldn't recall the names of the antidepressants because they were ineffective and only taken for a short period of time. Five months after a COVID-19 infection, the patient's symptoms suddenly changed. Her relatives reported that she changed into a different person, becoming too talkative and lively despite only getting 4 hours of sleep per day. She was also confident but aggressive and vulnerable to losing her temper. She became involved in several hobbies, overspent on purchases, and struggled to stay at home. After 2 months, the patient stopped taking medicine as she believed she was cured and no longer needed it. Two months ago, she experienced depression, lost interest in life, felt weak, and had suicide thoughts. Symptoms did not improve after taking duloxetine 120mg once daily. Two days ago, took over 20 clonazepam tablets and attempted suicide. They were taken to the emergency room and then transferred for inpatient treatment. Upon admission, the patient's cranial MR revealed minor ischemia foci in the frontal parietal lobes bilaterally. The Hamilton Rating Scale for Depression (HAMD-17) (Figure 1) score was 22, while the Hamilton Anxiety Rating Scale (HAMA) score was 23. Additionally, the Pittsburgh Sleep Quality Index (PSQI) score was 10. The diagnosis has been changed to bipolar disorder with current major depressive episode and used a combination of lithium carbonate and quetiapine due to the patient's elevated risk of suicide and poor sleep quality. The doses of lithium carbonate were gradually increased to 0.3g three times per day, quetiapine to 0.3g once per night, and clonazepam to 2 mg before bedtime. After 9 days of hospitalization, the patient's mood gradually improved and re-examined for HAMD-

		Pre-treatment	1st Follow-up	2nd Follow-up
HAM-D Rating Scale Symptoms		Date	Date	Date
1	Depressed mood	0 1 2 3 4	0 1 2 3 4	0 1 2 3 4
2	Guilt feelings	0 1 2 3 4	0 1 2 3 4	0 1 2 3 4
3	Suicide	0 1 2 3 4	0 1 2 3 4	0 1 2 3 4
4	Insomnia - early	0 1 2	0 1 2	0 1 2
5	Insomnia - middle	0 1 2	0 1 2	0 1 2
6	Insomnia - late	0 1 2	0 1 2	0 1 2
7	Work and activities	0 1 2 3 4	0 1 2 3 4	0 1 2 3 4
8	Retardation - psychomotor	0 1 2 3 4	0 1 2 3 4	0 1 2 3 4
9	Agitation	0 1 2 3 4	0 1 2 3 4	0 1 2 3 4
10	Anxiety - psychological	0 1 2 3 4	0 1 2 3 4	0 1 2 3 4
11	Anxiety - somatic	0 1 2 3 4	0 1 2 3 4	0 1 2 3 4
12	Somatic symptoms GI	0 1 2	0 1 2	0 1 2
13	Somatic symptoms - General	0 1 2	0 1 2	0 1 2
14	Sexual dysfunction - menstrual disturbance	0 1 2	0 1 2	0 1 2
15	Hypochondrias	0 1 2 3 4	0 1 2 3 4	0 1 2 3 4
16	Weight loss by history	0 1 2	0 1 2	0 1 2
	- by scales	0 1 2	0 1 2	0 1 2
17	Insight	0 1 2	0 1 2	0 1 2

Fig 1. HAMD-17 Rating Scale Symptoms

17 (10 points) and HAMA (10 points) before discharge. After 6 months of follow-up, the patient demonstrated mental stability with HAMD-17 and HAMA scores of 9, 8 (2 months after discharge), 7, 6 (4 months after discharge), and 8, 6 (6 months after discharge).

Discussion:

The patient had been diagnosed with severe depressive illness for 7 years prior to COVID-19 infection, as their previous hypomanic episodes did not satisfy the diagnostic criteria for a hypomanic episode. As a result, the antidepressant that the doctor prescribed seemed to be the ineffective. Clinical phenomenology indicated that patients with major depressive episodes and short-duration hypomanic episodes form a complex phenotype, similar to unipolar depressive episodes and bipolar II disorder as defined by DSM-5.[2] A previous study found that MDD patients with subthreshold manic syndrome had clinical presentations more similar to bipolar II disorder than to MDD alone. Patients with only manic symptoms were intermediate between the two.[3]

Conclusion:

In patients with severe depressive disorder who experience shorter-duration hypomanic episodes, mood stabilizers and atypical antipsychotics may be a better option than antidepressants.

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3. An uncommon case of antiphospholipid syndrome with Neuropsychiatric Characteristics

Introduction:

Common neurological manifestations in Antiphospholipid syndrome (APS) include strokes and transient ischemic episodes.

Antiphospholipid syndrome (APS) is a thrombo-inflammatory condition caused by autoantibodies that target cell surface phospholipids and phospholipid binding proteins. Antiphospholipid syndrome affects 40 to 50 people per 100,000, with an annual incidence of 1 to 2 per 100,000.[1] The pathophysiology of APS involves the activation of platelets, endothelial cells, monocytes, and the complement system, all of which contribute to a prothrombotic state. Antiphospholipid antibodies can directly interact with neural tissue, disrupting normal function, but thrombotic events are the most common neurological manifestation of APS. APS has been linked to neuropsychiatric symptoms such as psychosis and hallucinations, however these are uncommon. Early detection of APS in patients with neuropsychiatric symptoms was critical for effective care and prevention of consequences.[2] This case described a 31-year-old female who needed medical attention at the emergency room due to psychosis and later suffered a thromboembolic stroke while hospitalized.

Case Presentation:

A 31-year-old Caucasian female presented to the hospital with erratic conduct in a public environment. At admission, she was angry, agitated, and physically aggressive toward nearby objects. The patient was referred to a comprehensive psychiatric emergency treatment. During the interview, she claimed to have used K2 and an unknown chemical. The patient's medical history includes borderline personality disorder, post-traumatic stress disorder, and severe depressive disorder. She was a daily tobacco cigarette smoker and used marijuana, cocaine, and K2. The patient had an allergy to aripiprazole, trimethoprim-sulfamethoxazole, haloperidol, and penicillin. The patient was given risperidone 1 mg twice daily to treat psychosis. On the second day of admission, the patient exhibited restless movements and tossing in bed, leading to a fall. A thorough neurological evaluation indicated right-sided facial drooping and paralysis. Her stroke score was 5 on the National Institutes of Health (NIH) scale. A CT scan of the head and neck without contrast revealed no evidence of acute transcortical

infarction, cerebral bleeding, or mass effect. A contrast CT angiography of the head and neck revealed no stenosis or dissection in the neck or brain, but a stenosis in the left middle cerebral artery (MCA) with a sub-occlusive thrombus. In order to manage her condition, the patient was started on a treatment plan that included three mainstay doses: 325 mg of aspirin once, 81 mg twice a day through a nasogastric (NG) tube, which was supplemented with 300 mg of clopidogrel once, and 75 mg four times a day using the same NG tube. An EKG showed normal sinus rhythm with a QTc of 366 m/s. A non-contrast MRI of the head revealed limited diffusion in the left MCA distribution. A Holter monitor examination excluded atrial fibrillation or flutter as contributing factors. After a brief stay in the psychiatric unit, the patient was transferred to the medical intensive care unit (MICU) to address her condition. During the first day in the MICU, the patient had dilated reactive pupils and aphasia. The NIH stroke scale increased to 15, suggesting worsening neurological condition. A CT scan of the head without contrast revealed acute infarctions in the left frontal, parietal, caudate nucleus, and basal ganglia. CT angiography showed lower contrast opacification in the distal right M2 segments of the right MCA compared to the left. Patchy subcortical white matter hypodensities were observed in the bilateral and parasagittal frontal lobes. The patient was referred to the interventional neurology unit for mechanical thrombectomy. Injection of the left common carotid artery revealed a thrombus near the origin of the ACA and MCA. The MCA was entirely occluded, whereas the ACA was only slightly occluded, allowing for visibility of its branches and left leptomeningeal flow into MCA regions. While the left MCA thrombus was successfully removed, attempting to extract the left ACA thrombus was unsuccessful. A post-procedure CT scan of the head without contrast revealed cytotoxic edema in the left frontal lobe, basal ganglia, and posterior frontoparietal lobe, suggesting acute ischemia infarctions (Figure 1). The transthoracic echocardiography revealed a left ventricular ejection fraction above 70% and a patent foramen ovale. A panel of coagulation investigations was initiated when a suspicion of hypercoagulable disease was raised. Both beta-2 glycoprotein I antibody IgG and

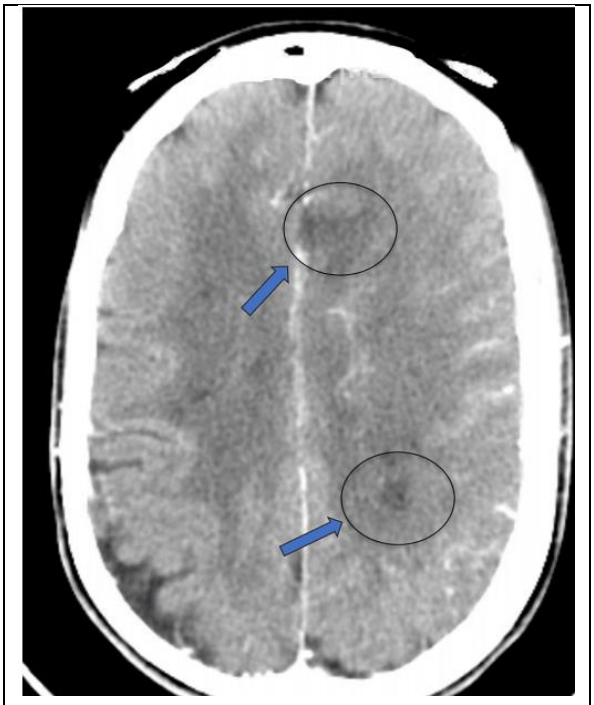

Figure 1. A CT scan taken 48 hours after a mechanical thrombectomy revealed the existence of further infarctions (blue arrows).

anticardiolipin IgG were positive. The antibody findings led to a diagnosis of antiphospholipid antibody syndrome. Four days after the procedure, a follow-up CT scan of the head without contrast revealed the predicted progression of the left MCA territory infarction, including increased hypodensity in the left frontal, frontoparietal, basal ganglia, and posterior parietal regions. The upper left Doppler ultrasound revealed clots in the left basilic vein's proximal, mid, and distal forearm regions, as well as one in the cephalic vein's elbow region. The patient underwent multiple X-rays during the hospital stay, but the results were not notable. After a 15-day hospitalization, the patient was discharged with a prescription for apixaban 5 mg tablets, to be taken orally every 12 hours for 30 days. In addition, a single aspirin 81 mg tablet was advised to be taken once a day, along with chlorpromazine 25 mg tablets taken orally three times a day, and quetiapine 25 mg tablets twice a day.

Discussion:

APS is a multifactorial autoimmune disease that causes arterial and venous thrombosis and is associated with the presence of at least one autoimmune antibody. In the present case, APS is managed with apixaban 5 mg tablets, a single dose of 81 mg aspirin tablet along with chlorpromazine 25 mg tablets taken orally three times a day, and quetiapine 25 mg tablets twice a day.[2] Previous study suggested that APS can be treated with DOACs, vitamin K antagonists, heparin, or antiplatelet medications.[3]

Conclusion:

Early detection of APS in individuals with neuropsychiatric symptoms is crucial for effective treatments and improved outcomes. More study is needed to identify the pathophysiological mechanisms that link APS to neuropsychiatric symptoms, leading to better understanding and management of this complex disorder.

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4. Case Report on Concussion-Related New-Onset Bipolar Disorder

Introduction:

Psychiatric evaluation and screening for risk factors of psychiatric problems after traumatic brain injury is crucial for early diagnosis and management.

Bipolar disorder is characterized by significant mood swings, ranging from mania or hypomania to depression. Bipolar disorder is a life-threatening condition, with 15-20% committing suicide and one-third to half attempting it.[1] Emotional dysregulation after a concussion is extensively reported. The development of serious psychiatric disorders like bipolar disorder (BPD) after a concussion has not been studied. BPD often begins with a depressed episode and progresses to mania, with concurrent depressive and manic states. There are other theories describing the origin of BPD, such as the diathesis-stress model (DiSM), however, no single acceptable theory has been developed.[2] In this case report, a genetically vulnerable individual developed new-onset bipolar disorder with mixed presentation after a traumatic brain injury, leading to serious medical consequences.

Case Presentation:

A 50-year-old male ambidextrous patient arrived at an outpatient neurorehabilitation clinic with a history of imbalances, dizziness, and behavioral, and emotional problems after an vehicle accident (20 months before the assessment). Previous medical conditions included obsessive-compulsive disorder, ADHD, and hypertension. The patient's family history includes a sister and brother with bipolar disorder, as well as a mother who had a stroke. The patient sustained a grade II concussion after striking his forehead on the driving, causing hematoma, dizziness, and tinnitus but no loss of consciousness. Two months after MVC, brain MRI revealed negative susceptibility weighted imaging (SWI), mildly increased fluid-attenuated inversion recovery (FLAIR) signal in frontal and parietal periventricular regions, and (not shown) abnormal diffusion tensor imaging (DTI) with a C-FAST score of 4, indicating grade II concussion with diffuse axonal injury (Fig 1). Vestibular treatment helped alleviate post-concussion symptoms such as dizziness and balance disruption. The patient's caregiver reported worsening mood disturbances and personality changes over the next year. One year after the MVA, the patient developed irritability, anxiety, paranoia, rapid speech, hypersexuality, and violence, leading to involuntary admission to a psychiatric center and

diagnosis of BPD with mixed presentation. He was treated and discharged with oxcarbazepine, risperidone, and trazodone. After discharge, the patient lost his 36-year-old business, became homeless, and stopped taking prescriptions. Two months after discharge, the patient was taken to the hospital for a syncopal episode and hypertensive crisis, resulting in aortic dissection and stroke. Embolic stroke caused by aortic dissection resulted in enhanced FLAIR signal in the right frontal, parietal, and occipital lobes, accompanied by frontal encephalomalacia (Fig 2). SWI blooming and clustering can be observed in locations with higher FLAIR signal. Following the stroke, the patient developed dysarthria and visual disruption, which exacerbated their vestibular and mental problems. A year later, the patient was taken to a psychiatric center for a second time due to a suicide attempt and significant depressed symptoms, indicating acute bipolar depression. The patient was stabilized and discharged after receiving additional drugs, including sertraline, trazodone, and lorazepam. The patient continued to have BPD symptoms till he was lost to follow-up.

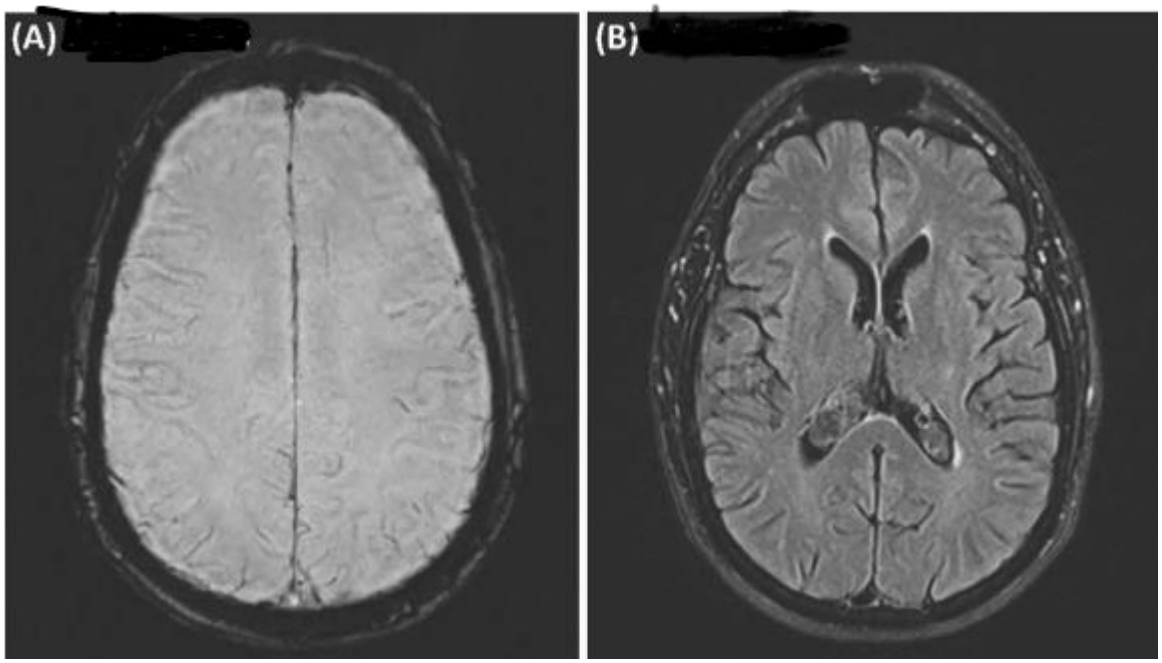

FIGURE 1: Brain MRI post-concussion: (A) The susceptibility-weighted imaging (SWI) sequence shows no hemosiderin deposition. (B) FLAIR sequence imaging reveals minor hyperintensities in the frontal and parietal periventricular white matter.

Discussion:

The DSM-5 defines BPD as a mood disorder that encompasses type I, type II, and cyclothymia, distinguished by the lack or presence of manic or major depressive episodes. There is an

increased risk of a history of TBI in patients admitted for BPD.[2] Patients with a history of neuropsychiatric disorder are five times more likely to develop post-concussion syndrome than the general population.[3] So, a bidirectional association is likely to occur, establishing a positive feedback loop that raises the frequency of TBI and BPD.

Conclusion:

This case demonstrated how the diathesis-stress model (DiSM) and traumatic brain damage might lead to a new diagnosis of bipolar disorder (BPD). Undiagnosed or delayed diagnosis of new-onset psychiatric illnesses can pose a substantial health risk to patients. Psychiatric evaluation and screening for risk factors of psychiatric problems after traumatic brain injury is crucial for early diagnosis and management.

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