MICRO LABS SYNAPSE

Vol 4|Issue 19|2023

Interesting Cases PSYCHIATRY

Psychiatric Case Series

Vol 4 | Issue 19 |2023

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 Catastrophic Effects of Psychogenic Polydipsia**
2. **Case report on Marchiafava-Bignami disease with psychiatric signs**
3. **Abdominal Epilepsy in an adult male- A rare case report**
4. **Case report on Wernicke Encephalopathy in a Bipolar Disorder Patient**

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,

MICRO LABS SYNAPSE
Vol 4|Issue 19|2023
Interesting Cases PSYCHIATRY

Case Report on Catastrophic Effects of Psychogenic Polydipsia

Introduction:

Psychogenic polydipsia is a well-known condition marked by increased fluid intake and frequent urination. It is prevalent in 11-20% of schizophrenia spectrum disorder patients.[1] Polydipsia raises the probability of mortality in schizophrenia patients by 74%.[2] Depending on availability and consumption of water, a patient with both psychogenic polydipsia and nephrogenic DI may develop hypernatremia, hyponatremia, or swing rapidly between these two conditions.[1] This case report describes a 71-year-old man was brought to the emergency department (ED) with a history of mental illness that included anxiety and bipolar type schizoaffective disorder.

In order to properly manage psychogenic polydipsia and avoid its fatal consequences, a multimodal strategy that involves fluid restriction, medication modifications, and early clinical suspicion is required.

Psychogenic polydipsia

Case Presentation:

A 71-year-old male admitted in the emergency department (ED) of a community hospital with a psychiatric medical history that included schizoaffective disorder-bipolar type and anxiety. Before his admission, he was characterised as exhibiting strange and agitated behaviour and was not able to provide suitable answers to queries. He had no conscious and hematemesis on his chest. At first, the symptoms were suggestive of a stroke. Prophylaxis against stroke and suitable medical attention were given, including injection of 4 grams of magnesium sulphate in the ED. The results of the CT and MRI were both normal. The results of the electrocardiogram (EKG) showed a generic ST abnormalities and sinus rhythm with premature ventricular complexes (Figure 1). Seven hours later, a first-degree atrioventricular (AV) block and sinus rhythm were observed on an EKG (Figure 2). The medical team received collateral information regarding the patient's pharmaceutical regimen, which included lithium 300 mg three times daily for four years, lorazepam 1 mg overnight, and paliperidone palmitate 234 mg/1.5 mL syringe once monthly. Furthermore, the patient had a history of hyponatremia caused by excessive fluid intake, with a daily water consumption of 16-17 glasses. The primary diagnoses that were explored were hypovolemic hyponatremia and syndrome of inappropriate antidiuretic hormone secretion (SIADH), which was determined by the patient's first laboratory workup. Considering the patient's psychological background, psychogenic polydipsia was also seen as a likely diagnosis. Certain drugs have the potential to produce SIADH, which manifests as euvolemic hyponatremia, low urine osmolality, high serum osmolality, and poor urine output with concentrated urine. The patient satisfied all other requirements, including taking lithium, despite having a low urine osmolality.

MICRO LABS SYNAPSE
Vol 4|Issue 19|2023
Interesting cases PSYCHIATRY

A psychiatric diagnosis, an unusually high urine output, excessive fluid consumption, and diluted urine with low urine osmolality are all signs of psychogenic polydipsia. The onset of a tremor worsened the patient's hospital stay about eight hours after presentation, along with 4700 mL of urine output. Due to continuous emesis and obtundation, aspiration pneumonia was a concern, necessitating intubation for airway protection. An electroencephalogram (EEG) showed widespread background slowing, which might be caused by anomalies in the brain's metabolism, multifocal abnormalities, or toxicity.

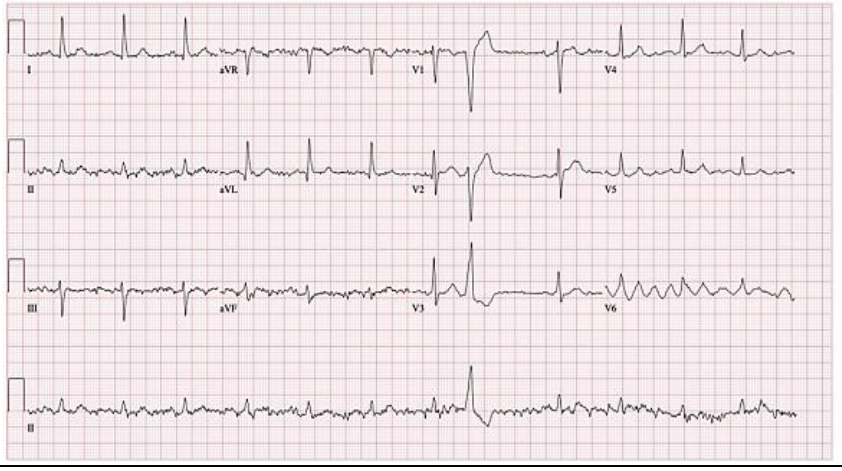

Figure 1. Initial EKG shows normal sinus rhythm with premature ventricular complexes

This is symptomatic of extensive cerebral dysfunction. The major issue was found to be encephalopathy. After receiving 100 mL/h of 0.9% normal saline, the patient's sodium level increased to 125 mEq/L in 12 hours. The correction rate was slowed by the administration of desmopressin acetate (DDAVP). After correction had been achieved, D5W was added and isotonic intravenous fluid (IVF) hydration was stopped to prevent overcorrection. When allowing for self-correction, fluid restriction was introduced. After two days, post-obstructive diuresis was suspected due to urine retention, which resulted in the implantation of a Foley catheter that produced an output of 6000 mL in five hours. Limiting fluid intake to 2 L and avoiding any IV fluids were part of the therapy. Ultimately, polyuria was shown to be the cause of hyponatremia, leading to the diagnosis of psychogenic polydipsia in the patient. The patient's history of mental illnesses and the noted episodes of delirium supported the psychological cause of polyuria, which was highlighted by a good reaction to fluid restriction. These clinical indicators confirmed the

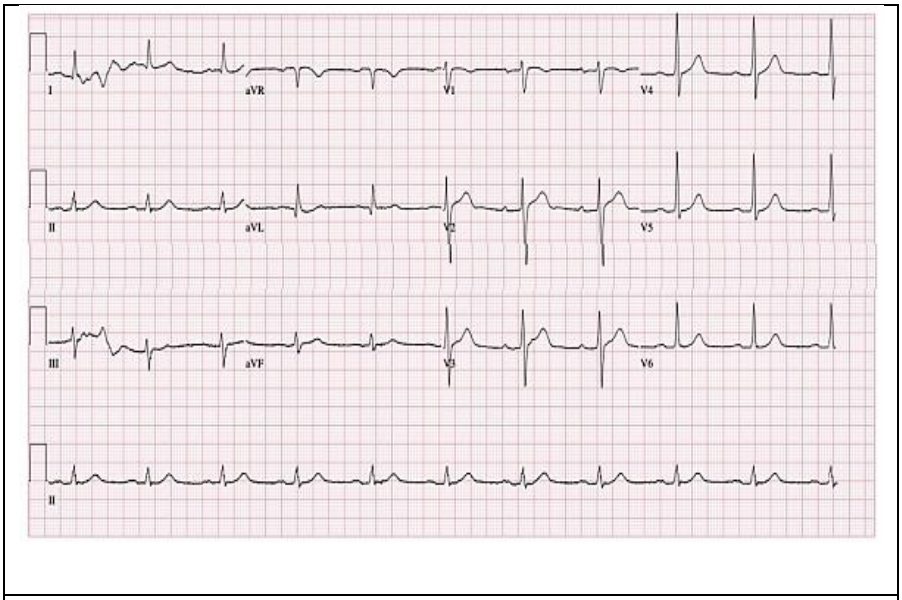

Figure 2. Repeat EKG shows sinus rhythm with first-degree atrioventricular (AV) block

diagnosis of psychogenic polydipsia as the most likely cause of the patient's hyponatremia, together with the laboratory findings and symptomology.

Discussion:

Due to the significant overlap with other illnesses that have similar clinical symptoms, diagnosing psychogenic polydipsia can be difficult. On the other hand, early detection is essential to prevent possibly fatal consequences. The patient in present case was taking lithium, a drug that has a variety of impacts on electrolyte balance and fluid balance. [2] Similarly, Gitlin M et al. demonstrated that lithium has been associated with side effects such as polyuria and polydipsia in up to 70% of individuals.[3] Over time, there has been a noticeable variation in the method to treat psychogenic polydipsia, utilizing various forms of intervention.[2] A study by So Kirino et al. found that the effectiveness of medication-based therapies has been inconsistent, however, new research has indicated that drugs such as clozapine and vasopressin type 2 antagonists may be useful.[4]

Conclusion:

There may be potentially fatal outcomes from the intricate interactions that exist between pharmaceutical regimens, fluid-electrolyte imbalances, and mental diseases. For an accurate diagnosis and suitable therapies, a thorough history-taking, careful clinical investigations, and collateral information gathering are essential. This case study highlights the need for a multimodal strategy that includes fluid restriction, medication modification, and early clinical suspicion in order to successfully manage psychogenic polydipsia and avoid its fatal consequences.

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MICRO LABS SYNAPSE
Vol 4|Issue 19|2023
Interesting Cases PSYCHIATRY

Case report on Marchiafava-Bignami disease with psychiatric signs

Psychiatrists, neurologists, internists, and dietitians should be part of a multidisciplinary team that manages MBD therapy.

Introduction:

Marchiafava-Bignami disease (MBD) is a rare neurological condition characterized by corpus callosum necrosis and demyelination.[1] Necrosis of the corpus callosum was first identified in a post-mortem examination of an Italian alcoholic patient in 1903.[2] MBD is accompanied with persistent drinking, however there have been reports of non-alcoholic aetiologies such as diabetes, malnutrition, and paraneoplastic disease. The usual age of onset of MBD is in the mid to late 40s, and it primarily affects men.[1] Despite certain problems in diagnosis and differential diagnosis, early detection and suitable treatment techniques can prevent lifelong impairment and coma development. High dosages of thiamine, vitamin B complexes, and steroids are thought to be useful.[2] This study aimed to contribute to the present literature by reporting a case that was brought to the psychiatry outpatient clinic with unusual mental symptoms and was later diagnosed with MBD.

Case Presentation:

A 56-year-old married man who had completed primary school and was retired was brought to the outpatient psychiatric ward by his daughter. Based on information provided by the patient's daughter, who had complaints of forgetfulness and loss of appetite, it was found that the patient had not behaved normally in recent months, that his increased libido, and he had taken out a bank loan and overspent on needless shopping and having been speaking a lot in the past few months. He was responding to the questions asked in a thoughtful manner. He spoke very loud and speaking at a rate of around 300 words per minute. The affect was dysphoric and the mood was somewhat raised. There were no psychotic findings in the perception test. The patient was noted to move quickly across the space and to have trouble holding one posture for an extended period of time. Until the last two months, the patient had been consuming 200 cc of beer every day for thirty years. Furthermore, it was found that the patient occasionally drank 80–100 cc of 70% ethanol and wine three–four days a week. With a pre-diagnosis of bipolar I disorder's manic phase based on the DSM-5, the patient began therapy with olanzapine 5 mg/day. After a month, the patient's mood symptoms showed a clinical regression; nonetheless, he remained bedridden, experienced loss of bladder control, lost his appetite, fell, struggled to talk, and had trouble walking. A popular test for assessing cognitive abilities, "Mini-Mental State Examination" (MMSE) or "Standardised Mini-Mental State Examination" (SMMSE) was used to evaluate cognitive functioning and identify challenges with language, orientation, memory, attention, and visuospatial skills. A score of 16 was evaluated on the MMSE. There was a decline in orientation, memory, language, and executive function scores on the MMSE. A score of 4 received on the Young Mania Rating Scale (YMRS) and 42 points on the Brief Psychiatric Rating Scale (BPRS). There was low vitamin B12 levels. Upon examination of blood tests for viral meningitis, no agent was discovered. Both the serum acetylcholine receptor antibody and the Brucella tests found negative. The amount of vitamin B12 was low. Widespread signal increases in T2-FLAIR series have been reported in the perisupraventricular white matter as a

MICRO LABS SYNAPSE
Vol 4|Issue 19|2023
Interesting cases PSYCHIATRY

result of cranial magnetic resonance imaging (MRI). These signals were more common in the frontoparietal area, bilateral capsules and interna, externa, thalamic nucleus and mesencephalon, periaqueductal area, mammillary body, and pons level. AdditionallyThe mammillary body, corpus callosum genus, optic chiasm, and splenium isthmus level all showed an increase in diffuse signal. Furthermore, patchy enhancement was noted in the lesions at the level of the corpus callosum splenium, the interna posterior crus on the right, and the capsule following the intravenous contrast agent. Results from a cranial MRI were in line with MBD. The patient's neurological examination revealed that they are cooperative, somewhat oriented in space, conscious, and responded to instructions. Dysarthria is noticeable and He speaks in echo.

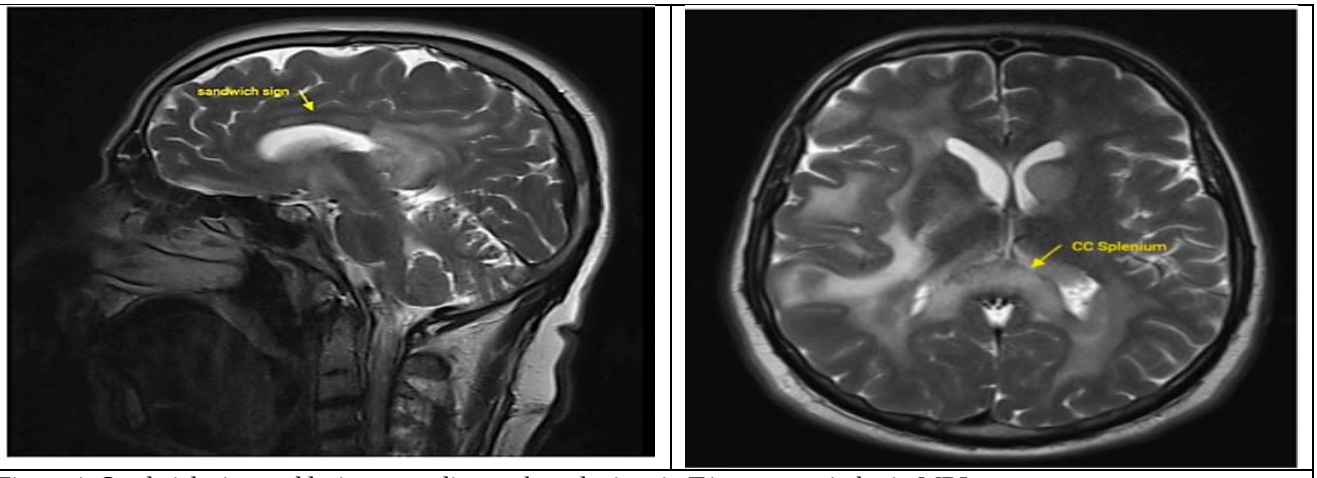

Figure 1. Sandwich sign and lesion extending to the splenium in T1 sequence in brain MRI

After receiving advice from the neurology department, the patient was admitted to the neurology unit. Intravenous vitamin B1, B6, B12 complex, and 4000 mg of dexpanthenol replacement treatment were started for the patient. The patient received steroid therapy and daily dose of olanzapine at 5 mg was continued. The patient had some improvement in their neurological and psychiatric symptoms after receiving therapy for 14 days. During the control assessment, it was noted that the patient had some clinical progress in improving his speech difficulties and was able to walk with assistance. Prior to discharge, it was assessed for 22 points on the MMSE, 5 points on the YMRS, and 12 points on the BPRS. With olanzapine 5 mg/day, the patient's therapy and follow-up continued on an outpatient basis. In the current case the patient was treated with intravenous thiamine, folate, vitamin B complexes, high-dose corticosteroids, and olanzapine 5 mg/day for mood and psychotic symptoms. Although there was a partial improvement in the symptoms, there was no regression in the corpus callosum lesions (Figure 1).

Discussion:

MICRO LABS SYNAPSE
Vol 4|Issue 19|2023
Interesting cases PSYCHIATRY

MBD is an uncommon demyelinating condition that arises in persons who have consumed alcohol for an extended period of time. While it can occur in non-drinking individuals as well, it is more common in those who have a history of long-term alcohol consumption.[1] Similarly, in the current case, a male patient of age 56 who had drunk 100 cc of alcohol every day for thirty years diagnosed with MBD.[2] Al-Witri et al. demonstrated that degenerations in the corpus and centre of the carpus callosum, as well as a profusion of macrophages and fat deposits in the lesion sites, are visible in postmortem histological investigations.[3] Among the diagnostic procedures carried out on this case, the cerebral MRI revealed a low vitamin B12 level and typical degenerative lesions in the corpus callosum. In this case diagnosis was aided by the patient's history of prolonged alcohol use, clinical symptoms, inadequate vitamin B12, and—most importantly—typical lesions on cerebral MRI.[2]

Conclusion:

MBD is an uncommon dilemma of long-term alcohol intake. Early detection and intervention are crucial in the management of this fatal condition. Nevertheless, in the clinical scenario, improvement is possible with prompt diagnosis and care. Patients with abnormal mental symptoms, behavioural abnormalities, neurologic symptoms, and a history of continuous alcohol consumption, like patient of this case, should always been taken into consideration for MBD when making a differential diagnosis. MBD, an uncommon consequence of prolonged alcohol consumption, is as difficult to cure as it is to diagnose. A multidisciplinary team that monitors MBD therapy should include psychiatrists, neurologists, internists, and nutritionists.

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Abdominal Epilepsy in an adult male- A rare case report

MICRO LABS SYNAPSE
Vol 4|Issue 19|2023
Interesting cases PSYCHIATRY

Introduction:

Abdominal epilepsy (AE) is an uncommon seizure disorder characterized by episodic gastrointestinal symptoms and abnormal electroencephalogram (EEG). The majority of the cases that are currently accessible in the literature describe onset during infancy. The diagnosis of AE is frequently delayed because of unclear and intermittent symptoms. A strong index of suspicion and an EEG recorded during an abrupt abdominal seizure are crucial for the diagnosis.[1] AE can manifest with unclear, severe, and recurring gastrointestinal symptoms such as paroxysmal severe pain, nausea, vomiting, bloating, and diarrhoea that resolve with antiepileptics. It is frequently associated with alterations in the temporal lobes of the electroencephalogram (EEG) and symptoms such as altered awareness, disorientation, and fatigue that indicate the involvement of the central nervous system (CNS).[2] This case study describes a forty-year-old man who had a misdiagnosis of psychogenic stomach discomfort following many tests involving various medical specialties in multiple hospital departments.

When patients appear with episodic, recurring, and paroxysmal GI problems and symptoms suggestive of CNS disturbance that do not improve when conventional treatment guidelines are followed, it is advisable to evaluate Abdominal epilepsy (AE), since it is frequently ignored or misinterpreted.

Case Presentation:

A 40-year-old right-handed male patient, who arrived to the gastrointestinal clinic after experiencing significant acute epigastric abdominal discomfort on a frequent basis for two months. These episodes were accompanied with nausea and occasionally vomiting, which was followed by a few minutes of lethargy or confusion. There were five to eight episodes of abdominal pain every day, each lasting 15 to 20 minutes. It occurred unexpectedly and resolved spontaneously. There were no remarkable results from the general and local examination of the abdomen and neurological system. Proton pump inhibitors were administered to him, and a week later, he was reassessed. There has been a notable reduction in his symptoms. Barium swallow tests and upper GIT endoscopy were performed and were found normal (Figure 1). After that, he was referred to the clinical immunology section, where he was conducted with several laboratory investigations ((ESR – CRP – Anti_ DNA – Anti_CCP IgG – RNP_ab). All the results were normal. Then, due of the psychogenic stomach discomfort, he referred to a psychiatrist. After using antipsychotics and antidepressants for a while without observing any change, he was referred

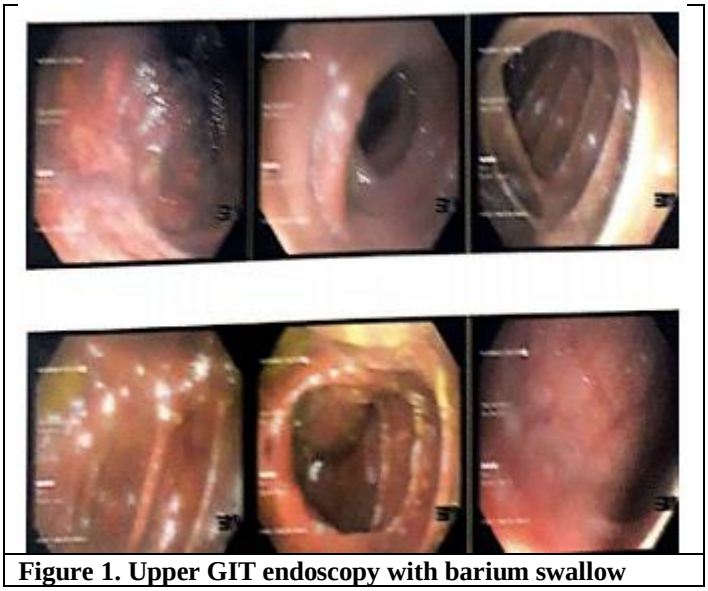

Figure 1. Upper GIT endoscopy with barium swallow

to the neurology department for a second opinion. The brain MRI revealed no abnormalities. An epileptic aetiology was suspected at this point. An abnormality in the form of recurring sharp waves in the right temporal leads was found during a 30-minute awake EEG. These waves were strongly indicative of an epileptogenic focus originating from the right temporal lobe. Following the diagnosis of AE, he was started on 450 mg of oxcarbazepine twice a day. He was taking his medicine as prescribed and had routine follow-up appointments every two weeks. There have been no symptoms and a gradual decrease in the frequency of seizures after six-month follow-up.

Discussion:

Due to its uncommon nature and ambiguous symptoms, abdominal epilepsy (AE) is frequently misdiagnosed or overlooked in medical evaluations. Although it is most frequently seen in children, it has also been seen in adults. In the current study following the diagnosis of AE, patient was prescribed 450 mg BID of oxcarbazepine.[2] In Giuliano Lo Bianco et al., patient was prescribed with pregabalin 75 mg BID, fluoxetine 20 mg BID, carbamazepine 200 mg at the morning and 400 mg at night, and codeine/paracetamol 30 mg/500 mg up to eight tablets once daily if needed. The patient stated that she considered the inclusion of pregabalin to be the most significant component in her recovery.[3]

Conclusion:

This case study demonstrated how difficult and time-consuming it may be to get a definitive diagnosis of AE, particularly in adults, due to the lack of suspicion and low occurrence of the condition's symptoms. Since AE is frequently overlooked or misdiagnosed, it should be considered when patients present with episodic, recurrent, and paroxysmal GI complaints and symptoms suggestive of CNS disturbance that do not improve after following standard treatment guidelines. In order to prevent such patients' symptoms from being mistakenly classified as psychogenic, EEG—ideally a video-EEG—should be considered. Antiepileptic medications are prescribed along with routine follow-up if the electroencephalogram results show abnormalities. In the present case, 450 mg of oxcarbazepine BID was useful in managing AE.

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Case report on Wernicke Encephalopathy in a Bipolar Disorder Patient

MICRO LABS SYNAPSE
Vol 4|Issue 19|2023
Interesting cases PSYCHIATRY

Introduction:

It takes a high degree of clinical suspicion to differentiate Wernicke encephalopathy from psychotic features in psychiatric patients who have altered mental state. Additionally, early thiamine supplementation can reduce morbidities.

Wernicke's encephalopathy (WE) is a neuropsychiatric disorder brought on by thiamine, a vitamin B1 deficiency. Thiamine is an essential coenzyme in several biochemical processes. Low thiamine levels can damage thiamine-dependent enzymes such as transketolase and pyruvate dehydrogenase, which frequently results in lesions in the mesencephalon, thalami, and mammillary bodies. These selective lesions can cause a triad of WE symptoms, including nystagmus/ophthalmoplegia, gait ataxia, and disorientation.[1] Patients who present with increased alcohol intake and impaired mental status are more likely to be clinically suspected since it is most typically linked to alcohol consumption. Since this has the potential to cause many nonalcoholic individuals to go undiagnosed with WE, which can then proceed to the irreversible and highly fatal Korsakoff syndrome. The frequency of WE in non-alcoholics ranged from 0.8% to 2.8% in a research based an autopsy specimens.[2] This case study describes a 59-year-old female patient with decompensated bipolar disease who presented with several syncopal episodes and disturbed mental state.

Case Presentation:

A 59-year-old female patient with a history of asthma, hyperlipidemia, and bipolar disorder was admitted to the hospital after experiencing syncope at home and experiencing mental alterations such as disorientation, confusion, and malnutrition. According to information gathered from the patient's family members, the patient was living alone and working. She had, however, behaved confused and different from her usual behaviour over the last few months. She didn't pick up the phone and seemed confused while speaking. They also observed a substantial weight reduction since their last visit, and the patient complained of not sleeping for several days in a row. The patient's physical examination revealed that she was 60 kg in weight, BMI of 23, temperature of 36.7°C, 127/73 mmHg in blood pressure, and pulse rate 88 beats per minute.

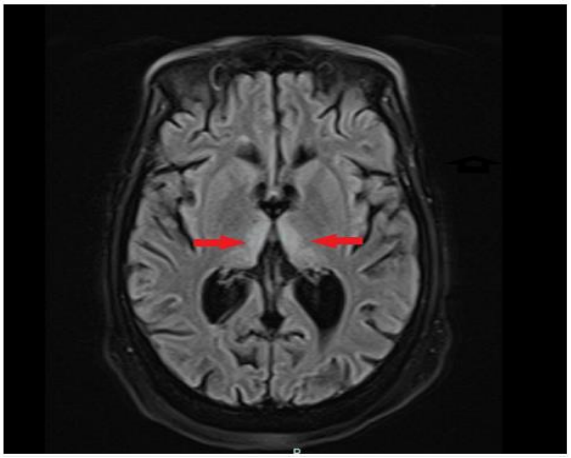

Figure 1. Magnetic resonance imaging (MRI) scan of the brain; red arrows show a symmetric hyperintense signal within the bilateral medial thalami region.

MICRO LABS SYNAPSE
Vol 4|Issue 19|2023
Interesting cases PSYCHIATRY

She struggled to answer questions and gave repeated response to various inquiries. The patient showed poor visual tracking and an inability to obey straightforward instructions. Her right pupil responded to light, however her left pupil was fixed and showed no light reaction. Her extremities showed signs of hyperreflexia and increased muscle tone. Rest of the neurologic examination was either incomplete or unremarkable. The patient seemed to be staring at the wall. Her initial test results for blood coagulation markers, glucose, amylase, lipase, sodium, creatinine, and other parameters were all within normal ranges, but she had electrolyte abnormalities, including low protein, low albumin, moderate hypokalemia, and hypomagnesemia. The electroencephalogram (EEG) showed no seizures or epileptiform discharges, and the departments of neurology and psychiatry were consulted for possible diagnoses. Microvascular ischemia and progressive volume loss were seen on the head's CT scan. The brain was then imaged using diffusion-weighted MRI (DWI) and fluid-attenuated inversion recovery (FLAIR), both of which showed symmetric hyperintense signals in the bilateral medial thalami and along the periaqueductal grey matter (Figure 1 and 2). These results indicated the possibility of Wernicke's encephalopathy sequelae. The patient was started on 500 mg of intravenous thiamine three times a day for three days. After that, they were given 250 mg of intravenous thiamine for three more days. The patient's confusion reduced after just one day of therapy, and she also become more conscious of her surroundings and her own identity. She recognised her family members and asked appropriate inquiries. Following the completion of intravenous thiamine therapy, the patient was switched to oral medicine and then discharged from the hospital on the same day.

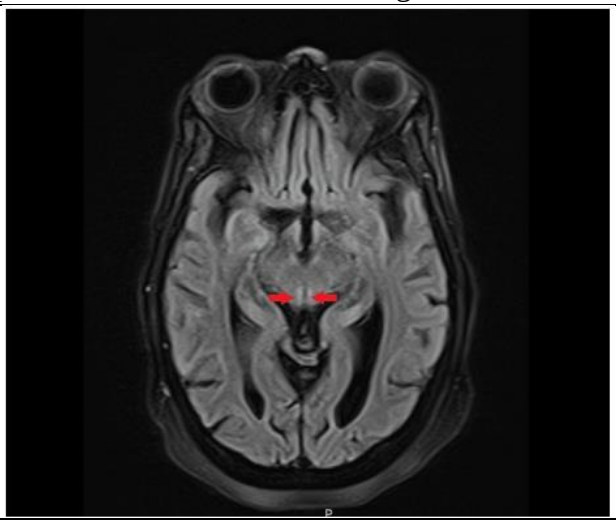

Figure 2. On magnetic resonance imaging (MRI) of the brain, the red arrows show a symmetric hypertense signal within the periaqueductal gray matter.

Discussion:

In this case, the patient had many syncopal episodes, severe psychosis, and ophthalmoplegia. Upon physical examination, it was found that the patient was very malnourished and dehydrated. The patient in this case given 500 mg of intravenous thiamine three times a day for three days. After that, they were given 250 mg of intravenous thiamine for three more days. The patient's confusion reduced after just one day of therapy, and She also became more conscious of her surroundings and her own individuality.[1] Erik Oudman et al concluded that prophylactic thiamine tests and therapy are important in individuals with schizophrenia, and if WE is suspected, sufficient parenteral thiamine supplementation is required.[3]

Conclusion:

MICRO LABS SYNAPSE
Vol 4|Issue 19|2023
Interesting cases PSYCHIATRY

Wernicke encephalopathy is a neurological disorder that can be irreversible and has a significant death rate if not treated early in the disease's progression. Many cases of Wernicke's encephalopathy remain undiagnosed because of the intrinsic association between the illness and alcoholism. When psychiatric patients exhibit altered mental status, a high level of clinical suspicion is required to distinguish Wernicke encephalopathy from psychotic characteristics. Early thiamine administration can also help minimise the morbidities.

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