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

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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. Impact of bright-light therapy on depression and anxiety of a patient with Alzheimer's disease and sleep disorder

Introduction:

Bright-light treatment is an effective non-pharmacological treatment for improving sleep quality in elderly adults with Alzheimer's disease.

Alzheimer's disease is a progressive, irreversible neurological condition that affects cognition, function, and behavior. Alzheimer's disease advances from preclinical disease to mild cognitive and behavioral impairment, dementia and sleep disturbances.[1,2] Studies have shown a relationship between AD and sleep abnormalities, with sleep disturbances being a risk factor for the condition. Thus, increasing the quality of sleep may help individuals with AD. Several pharmacological and non-pharmacological treatments have been proposed for clinical management. Bright-light treatment (BLT) is a non-pharmacological approach that is commonly used with older persons who have difficulty sleeping. BLT is increasingly recommended as a first-line treatment for sleep disturbances in dementia patients, due to its safety and effectiveness.[2] This report describes a patient who got AD along with anxiety, depression, and sleep disturbances. BLT helped the patient's sleep quality. BLT is more effective than prescription drugs at enhancing sleep quality and reducing the amount of time spent sleeping each day and experiencing restlessness at night.

Case Presentation:

The present case involves a 68-year-old woman who has had AD for two years. She first showed signs of memory loss, which got worse over time and made her depressed and anxious. Clinical symptoms included significant sleep disturbances. Given the patient's age and current health, bright light therapy—a non-pharmacological treatment—was employed to enhance the quality of her sleep. Later, the patient's symptoms progressively deteriorated, and she began to lose her memory, become unable to go home after a walk, and need police help and family monitoring in her day-to-day activities. She regularly thought her family had stolen her belongings, which made her unhappy, agitated, and suspicious. She also constantly scolded her family members without cause. The patient denied having any AD in their family. The patient

was alert and cooperative at the first appointment, but she was unable to remember details of her previous meal or the events that had occurred, and she was unaware that she was in the hospital. She lacked self-awareness, had little volitional activity, and was disorientated in place and time as well as toward other people. Biochemistry, blood counts, and ultrasensitive C-reactive protein were all normal. There were no additional organic lesions or cerebral infarct foci on a head computed tomography scan, which clearly indicated cerebral atrophy. She had no history of hypertension, diabetes mellitus, or stroke, and no other physical conditions that could contribute to mental issues were identified. The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition criteria were used to make the diagnosis. Two research psychiatrists provided informed consent after diagnosing the patient with AD. The Mini-Mental State Examination (MMSE) scores of less than 17, 20, and 24 were used to assess the cognitive level of patients with primary school, junior high school, and illiteracy, respectively. The illness lasted more than three months. The cognitive level was raised by using memantine or donepezil. No prior history of severe mental disease exists for the patient. The patient's MMSE score was 17. The patient was hospitalized after receiving a diagnosis of AD with psychobehavioral symptoms based on these results. The phototherapy apparatus was mounted on a moveable cart and the light intensity could be adjusted from 0 to 20000 lux. Twice daily, from 8:30 am to 9:00 am and from 16.30 pm to 17:00 pm, the treatment was given for 30 minutes each time. For a 4-week course of treatment, the patient was positioned 0.5–1 m away from the light source, with a light intensity of 14,000 lux. The patient rested comfortably in a chair facing the light source. The nurse then puts on the light for the patient, secures the portable cart, and notifies the patient that therapy is ready to start. The patient was instructed to keep quiet and not to get up and move around during treatment. For intellectual stimulation, the patient received 7.5 mL of memantine oral solution, 3 mg of carboplatin capsules per day, and 5 mg of olanzapine tablets daily for antipsychotic therapy. She also had light treatment to help with her poor quality of sleep at night. After four weeks in the hospital, the family stated that the patient's mood had improved and that their paranoia had vanished, although the cognitive level remained stable (MMSE: 18). With a noticeable drop in daytime sleep, an increase in overnight sleep, and no nocturnal activity, the patient's sleep quality significantly improved.

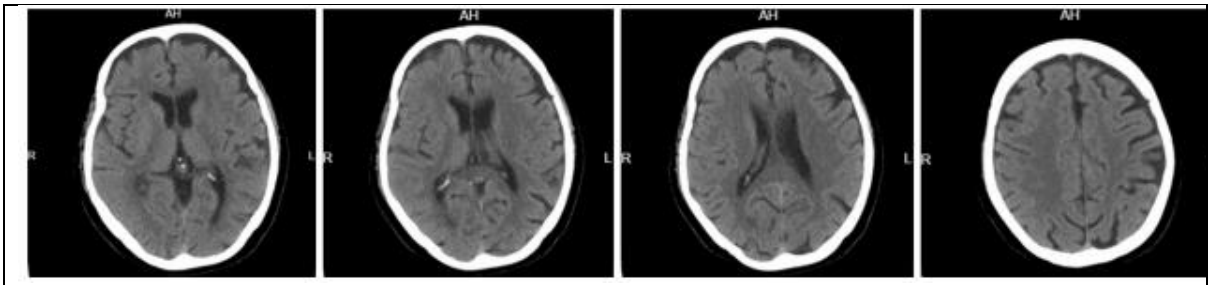

Figure 1: Head computed tomography. The cerebral hemispheres are symmetrical. The brain volume is diminished, and the sulci become deeper and wider. The ventricular system and sulcal fissure have enlarged. The midline structures are centered.

Discussion:

This is a case of severe Alzheimer's disease worsened by sleep difficulties, which was treated using BLT. Several drugs can be used to prolong and enhance sleep, but there is a chance that they can cause adverse reactions. BLT is an effective and safe non-pharmacological treatment that can improve the quality of sleep for older AD patients.[2] A previous study showed that BLT may help regulate the sleep-wake cycle in elderly persons with dementia.[3]

Conclusion:

According to this case study, BLT can be used as a safe and efficient non-pharmacological treatment for AD in older individuals and improve their quality of sleep.

References:

1. Porsteinsson AP et al. Diagnosis of early Alzheimer's disease: clinical practice in 2021. The journal of prevention of Alzheimer's disease. 2021 Mar;8:371-86.
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2. Case report on electroconvulsive therapy-induced manic episodes in bipolar disorder

Introduction:

Electroconvulsive therapy (ECT) is an effective treatment for bipolar disorder at all phases.

Bipolar disorder is a chronic and severe psychiatric disorder that significantly impacts patients' quality of life and has a high risk of recurrence. It is frequently diagnosed only after several years.[1] 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.[2] Treatment options include medications, lifestyle changes, supplementary psychotherapy, and electroconvulsive therapy (ECT). ECT is particularly useful for severely depressed or mixed states that are resistant to medication. ECT can cause manic or hypomanic episodes in patients with bipolar disorder, a condition known as ECT-induced mania. Despite its relevance, ECT-induced mania is poorly understood and rarely discussed in the literature, leaving doctors with minimal preventative and therapy guidelines.[1] This report analyzes a case of ECT-induced mania in a bipolar illness patient after discontinuing valproate and to identify risk variables and best methods for treatment.

Case Presentation:

A 56-year-old man is admitted to the psychiatric hospital's High Intensive Care (HIC) unit for the third time. He was admitted to this service twice before, both times under compulsory measures. After somatic exams ruled out organicity, the patient was diagnosed with late-onset bipolar disorder and a manic episode during his initial stay. He was discharged after taking lorazepam, olanzapine, and valproate for about a month and a half. A few days following the first admission, the patient developed a psychotic-depressive state that included fatalistic beliefs, self-injury, and a suicide attempt on the ward. Lorazepam and valproate dosages were increased (to 4 mg/day and 2000 mg/day, respectively), and sertraline treatment began at 50 mg. The patient improved and was discharged from the hospital after two months. At the third admission, the patient had hypomania symptoms such as sleeplessness, irresponsible behavior, and depression. He chose to get admitted voluntarily on this occasion. The olanzapine dosage is increased to 30 mg, and the valproate dosage is increased to 2300 mg (blood level control at 81 mg/l). The lorazepam dosage is increased to 4 mg per day, and sertraline is withdrawn. Despite pharmaceutical modifications, there is an increase in excitability and suspiciousness,

as well as signs of depression and anxiety. A mixed episode is recognized, characterized by disorganized psychotic-depressive symptoms, guilt delusions, catatonia (scored 18 on the Bush-Francis Catatonia Rating Scale), and suicide ideation. After several weeks of unsuccessful pharmaceutical treatments, electroconvulsive therapy (ECT) was initiated. Etomidate and succinylcholine were used to deliver anesthetic during a bitemporal electrode implantation. The patient was treated with a 30% charge (151.2 mC) using the LOW 0.5 protocol on the Thymatron System IV. The first two sessions were ineffective in terms of both clinical efficacy and EEG results. The dosage was increased to 50%, lorazepam was reduced, and valproate was withdrawn. The anticipated result was obtained during the third session. The next day, the patient's mood improved significantly, but they also had manic symptoms such as megalomania, religious delusions, agitation, disinhibition, flight of ideas, and prolixity. The patient declined the fourth treatment of electroconvulsive therapy (ECT) scheduled for a few days later, claiming to have recovered. Therefore, considering lithium's antimanic and mood-stabilizing qualities, it was decided to stop the ECT and start treatment with a high dose lithium (3x400 mg/day). Additionally, clotiapine is given four times each day. Approximately one week later, the patient is willing to consider additional ECT treatment. Resuming treatment (bitemporal, 50%) results in a progressive reduction of manic symptoms. After eight acceptable sessions, the frequency of ECT is reduced from twice to once per week. After 13 sessions, the patient can move to an open treatment unit in a stable psychiatric condition. A few months later, he may be released from that unit as well. Lithium is used as a long-term medicine (400 mg, 1.5 times a day). Outpatient ECT treatment is tapered over six months. A complete patient timeline is presented in Figure 1.

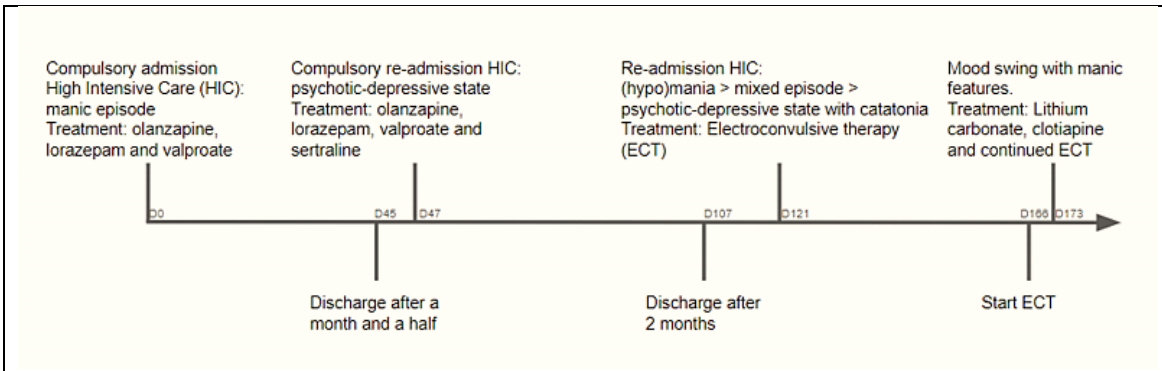

Figure 1: Timeline

Discussion:

This case study highlights the danger of manic transition after electroconvulsive therapy (ECT) for bipolar illness patients. In the present case, the patient's manic symptoms were stabilized by temporarily discontinuing ECT and adding lithium to the treatment. ECT was continued with lithium maintenance, resulting in increased mood stability.[1] A previous study showed that Lower lithium doses, shorter pulse width treatments, and right-unilateral electrode implantation reduce the risk of cognitive impairment and delirium in patients. The combination of ECT and lithium may reduce cognitive impairment and delirium in patients with bipolar illness compared to those with unipolar disease.[3] To avoid relapse, lithium should be taken after the affective episode has ended.[4]

Conclusion:

Electroconvulsive therapy (ECT) is a safe and effective treatment for bipolar disorder at all phases. In people with a history of bipolar disorder, a more decisive approach would be preferable. Options include starting pharmacological treatment (ideally with lithium), continuing ECT, or combining both. Patients with a history of ECT-induced mania or bipolar disorder should start taking lithium before undergoing ECT to avoid hypomania.

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1. Van Eenoo S, Bollen O, Janssens S. Electroconvulsive Therapy-Induced Mania in Bipolar Disorder: A Case Report. Cureus. 2024 Dec 7;16(12):e75255.
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3. Youssef NA, Madangarli N, Bachu A, Patel RS. Electroconvulsive therapy plus lithium is associated with less cognitive impairment and drug-induced delirium in bipolar depression compared to unipolar depression. Ann Clin Psychiatry. 2023 May;35(2):103-108.
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3. Case report on Pineal cyst in bipolar patient with normolithiaemia and positive fibromyalgic tender points

Introduction:

Chronic inflammation especially in systemic rheumatoid arthritis diseases like fibromyalgia may lead to cyst growth.

Pineal cysts (PCs) are non-cancerous cysts that originate from the pineal gland. These are relatively common, appearing in 0.5-5% of brain MRI scans and 20% of autopsies.[1] Although inflammation and post-traumatic mechanical stress have been linked to the development of acquired pineal cysts, the natural history and pathophysiology of these lesions remain unclear. Chronic lithium accumulation may lead to cyst formation in many organs, such as kidneys, uterus, and arachnoid. Given the incomplete blood-brain barrier of the pineal gland, it is hypothesized that lithium deposition may promote pineal cyst formation in patients on chronic lithium therapy. Further study was necessitated to understand the role of lithium in pineal cystogenesis.[2] This case report describes a lithium-treated patient with bipolar disorder who developed a pineal cyst with symptoms like fibromyalgia.

Case Presentation:

A 49-year-old female with a history of bipolar disorder and long-term lithium medication presented to the emergency room with severe bilateral headaches particularly affecting the occipital-cervical and parietal regions. The headache intensity was assessed as 7/10 on the Visual Analogue Scale (VAS). In addition to the headache, the patient complained of difficulties sleeping and muscle tightness in both the scapular and pelvic girdles. Upon arrival, her Glasgow Coma Scale (GCS) score was 15/15, indicating complete consciousness. Despite having no prior diagnosis of fibromyalgia, the patient had considerable pain while percussing the trapezius and latissimus dorsi muscles, indicating tender spots associated with the condition. Applying pressure to these areas caused significant pain. The laboratory results were within normal ranges, with lithium levels at 0.71 mEq/L (reference range: 0.5-1.2 mEq/L). Serum β-HCG and α-fetoprotein levels were within normal limits at 2.10 mIU/mL and 0.97 ng/mL, respectively. The neurological evaluation was generally normal. The patient exhibited no motor impairments, undamaged cranial nerves, and no evidence of myelopathy. A brain MRI with gadolinium contrast showed a cystic lesion in the pineal region. There was no sign of third-ventricle obstruction or compression of surrounding tissues (Fig. 1). The patient was advised to go for clinical and neuroradiological follow-up if there were no compression signals over the parenchymal ventricular structures or a midline shift. The patient was given a scaled dose of corticosteroid medicine (Dexamethasone), which provided moderate relief from her symptoms.

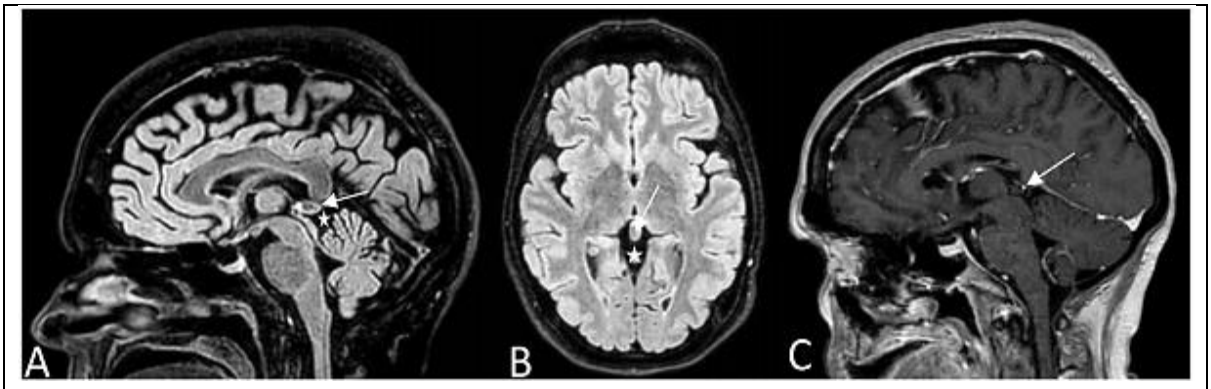

Figure 1: MRI study of the pineal cyst. (A, B) A hyperintense lesion in FLAIR sequences. (C) T1 contrast-enhanced sagittal image of the lesion. (A) The white arrow points to the cyst wall, which seems slightly hyperintense. (B-C) A white arrow shows a cystic lesion in the pineal area. The white star indicates the quadrigeminal cistern (Bichat's canal) that surrounds the pineal gland, the splenium of the corpus callosum, the quadrigeminal tubercles, and the superior surface of the cerebellar vermis (A-B).

Discussion:

Acquired pineal cysts are regulated by multiple variables, including inflammatory and post-traumatic mechanical stimulation. In the present case, the patient's lithium levels were within normal ranges, which lessens the possibility that lithium has a significant anti-inflammatory effect on the widespread inflammation observed in fibromyalgia.[2] A previous study also showed the ability of lithium, which has anti-inflammatory qualities to change the collagen architectures of the pachymeninges and leptomeninges. [3]

Conclusion:

Lithium is known to cause hyperplastic effects on glandular epithelia and organs. While there is no direct evidence linking lithium usage to the production of pineal cysts in people, chronic inflammation, especially in systemic rheumatoid arthritis diseases like fibromyalgia, may lead to cyst growth. Pro-inflammatory stimuli in these disorders may promote epithelial proliferation, leading to late-onset pineal cystogenesis.

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4. A rare case of thiamine-deficient neuropathy in a patient suffering from malnourishment due to melancholic depression

Introduction:

It is important to detect and treat thiamine deficiency in individuals with severe depression, particularly in those who are experiencing melancholy symptoms and have lost a large amount of weight.

Although certain symptoms are used to diagnose the condition, depression is a complicated disorder with varying biological backgrounds depending on the stage of the disease. Depression is now recognized as a systemic disorder caused by several biological mechanisms, such as inflammation, hypothalamic-pituitary-adrenal (HPA) axis dysfunction, and imbalances in neurotransmitters and neurotrophic systems.[1] In 2015, the World Health Organization (WHO) reported that 4.4% of the world's population suffers from depression. According to the DSM-5 classification, "melancholic features" indicate a severe depressive episode. The main clinical symptoms of melancholic depression are psychomotor retardation and lack of emotional responsiveness. Other symptoms may include psychomotor agitation, early awakening, and loss of appetite/weight. Depressive episodes can cause considerable weight loss or undernutrition.[2] This case described an unexpected neurological complication caused by a melancholy condition and dietary insufficiency.

Case Presentation:

A 36-year-old woman was apathetic and refused to eat. According to the patient's family, she was very sensitive and emotional. The situation started two months ago and quickly escalated into feelings of loneliness, agitation, and religious concerns. Negativity, self-deprecation, and an unwillingness to eat were the next signs to appear. The family had noticed a significant weight loss of 15 kg per month (15% of baseline weight) and vomiting episodes that did not respond to symptomatic therapy. The initial clinical assessment demonstrated extracellular dehydration, a BMI of 18 kg/m^2, and functional impotence. The psychiatric assessment indicated a patient who was attentive but apathetic, remained in bed, struggled with social contact, and had emotional numbness, anger, and anxiety. She had delusions of hopelessness, punishment, and suicide ideation. The patient was hospitalized in a psychiatric department and experienced gastrointestinal discomfort, diarrhea, tachypnea, and low oxygen levels after resuming oral nutrition. Biochemical tests revealed high blood sugar, low potassium, phosphate, and magnesium levels. The patient was diagnosed with inappropriate refeeding syndrome (IRS) and was sent to an intensive care unit for 4 days. She was given parenteral

nutrition and vitamin supplements (Vit B1, Vit B9, Vit D, and Vit E). The patient was subsequently readmitted to the psychiatric facility. A diagnosis of severe depression with psychotic and melancholy symptoms was made. The patient started taking Venlafaxine at 150 mg/day in combination with Olanzapine. After 3 weeks of therapy, the patient showed improved responsiveness and reduced irritability. Despite the disappearance of delusions, functional impotence remained. The neurological exam was hard as the patient was bedridden and had flaccid tetraparesis in the lower limbs. Vitamin insufficiency was suspected to cause metabolic neuropathy. During the patient's stay in the critical care unit, their thiamine levels dropped to 36 nmol/L (normal range: 70-180 nmol/L). The patient received intravenous Vit B1 300 mg three times each day for 7 days. She was subsequently given 100 mg of vitamin B1 orally daily. Electromyography (EMG) of all four limbs revealed significant axonal polyneuropathy, mostly affecting nerves in the lower limbs (Table 1). The brain and spinal cord MRI revealed no abnormalities. The patient had acute peripheral neuropathy, most likely caused by malnutrition and melancholy depression. The patient received antidepressant medication, oral polyvitamin supplementation, and multidisciplinary follow-up (physical medicine, neurology, psychiatry).

Nerve	Latency (ms)	Amplitude (μv)	Speed (m/s)	Surface (μV × ms)	Duration (ms)	Distance (mm)
Left median sensory						
Digit 2-wrist	3.94	9.7	35.5	15.2	3.9	140
Right superficial fibular sensory						
Leg–ankle	—	—	—	—	—	100
Left radial sensory						
Forearm-D1	3.46	3.5	28.9	1.39	2.1	100
Left sural sensory						
Leg–ankle	—	—	—	—	—	140
Right ulnar sensory						
D5-wrist	3.09	1.79	38.8	0.55	1.91	120

Table 1: Electromyography results demonstrating a considerable drop in the amplitudes of the upper limbs and an absence of the lower limbs' sensory nerve amplitude response

Discussion:

The symptoms of depression and thiamine deficiency can coexist. Thiamine deficiency first manifests as nonspecific symptoms that often appear gradually. These include anorexia, fatigue, irritability, nausea, weakness, apathy, lack of appetite, and disturbed sleep. Food intake and appetite might change because of depression. [2] A previous study showed that depressed cancer patients with borderline thiamine concentrations (BTC) can quickly develop thiamine deficiency (TD). Healthcare providers should always be aware of the possibility of TD and the necessity of routine thiamine-level testing or preventative replacement medication.[3]

Conclusion:

It was important to identify and treat thiamine deficiency in individuals with severe depression, particularly those who experienced melancholy symptoms and significant weight loss. Recent studies suggested that thiamine affects the development of mood disorders. This indicated the importance of thiamine in preventing complications and addressing the underlying causes of mood disorders. Early identification and management can prevent long-term impairment and improve the quality of life for those with severe depression and hypovitaminosis B1.

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