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Case Report

Vitamin B12 Deficiency Precipitating Mania, Exhaustion with Hypersomnolence – A Unique Perspective

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ABSTRACT

Vitamin B12 deficiency is implicated in various neurological, psychiatric, and deficit syndromes. Here, we present a unique case where a deficiency of vitamin B12 is the causative factor behind manic episodes with severe exhaustion and hypersomnolence.

Keywords: Vitamin B12, Hypersomnolence, Mania

INTRODUCTION

Vitamin B12, also known as cobalamin, is a water-soluble vitamin implicated in various neurological, psychiatric, and deficit syndromes. Normal levels of B12 range between 200 and 950 pg/ml.[1] Level below 200 pg/ml can precipitate mania, depression, dementia, chronic fatigue, etc., in the absence of clinical and investigative evidence of anemia.[2]

CASE REPORT

A 33/M with no known medical comorbidities or addictions, working as a bank manager, diagnosed with bipolar affective disorder 5 years ago with three manic and two depressive episodes presented with a 9-day history of persistent irritable mood, excessive non-productive, non-goal-directed activity, profound insomnia, and unprovoked violence to the extent that he broke the windshield of his brand new car and in the process sustained a hairline fracture to his right arm. Prior to the episode, the patient was on irregular treatment with Lamotrigine 100 mg and Olanzapine-fluoxetine 5/20 mg combination for 3 months. The patient was referred to the casualty of a general hospital in West Bengal, where he was injected with 1000 mg sodium valproate. Sedation was achieved, and the patient was admitted to the orthopedics ward for stabilization of the arm. Inpatient treatment was administered for 3 days, during which the patient remained lethargic with excessive perspiration, mild abdominal pain with constipation, and the sensation of bloating. His blood pressure was measured in the low 90/60 mmHg with a pulse rate of 126/min, low volume, and thready. Investigations like complete blood count (CBC), random blood sugar (RBS), liver function test (LFT), renal function test (RFT), serum potassium and sodium, thyroid assay (TFT), serum amylase, and lipase were all within normal limits. A baseline electrocardiography (ECG) and ultrasound abdomen were

also normal. Notwithstanding conservative management, he was prescribed clonazepam 1 mg. He was referred to a psychiatrist prior to discharge, who advised restarting his combination medication, but the patient was non-compliant. Post-discharge, he began sleeping for longer durations at night, and sleepiness progressively encroached during the daytime over a week. He was sleeping for 12–16 hours a day and, when awake, would be excessively lazy, so much so that he spilled hot tea over himself and was taken to a neurologist. At presentation, the patient was febrile (104°F) with no neck rigidity or headache. A complete neurological examination showed no focal neurological deficits. Magnetic resonance imaging (MRI) brain and ambulatory Electroencephalogram (EEG) were done with no significant findings. Cerobrospinal fluid (CSF) analysis showed clear fluid, nil RBCs and WBCs, proteins 20 mg/dl, and glucose 55 mg/dl. Paracetamol was administered intravenously, and the temperature subsided. Patient was sent home with multivitamins and pantoprazole 40 mg. He continued to sleep excessively and sustained a fall from bed with a soft tissue injury to the back. The patient was referred to a psychiatrist, who placed him on modafinil 50 mg bid with no improvement. On presentation, the patient was drowsy and febrile (99°F) and was constipated for 2 days. Pupils were bilaterally equal and reactive to light, and the patient followed commands with great difficulty. A vitamin B12 assay was ordered, which was 100 pg/ml. We prescribed a vitamin B12 injection of 750 mcg/ml intramuscularly thrice a day for 5 days, after which the patient was shifted to oral B12 supplementation of 500 mcg thrice a day for 5 days. Within 10 days, the patient's symptoms subsided, and sleep regularized. B12 levels increased to 250 pg/ml, and supplementation was reduced to twice daily dosing. Amisulpride 100 mg and oxcarbazepine 300 mg were started and continued for the next 2 weeks. Currently, the patient is symptom-free and well maintained on the same.

DISCUSSION

Psychotic patients with continuous mental and motor excitement are known to precipitate exhaustion, which, untreated, can lead to death. The exhaustion syndrome, between 2 days and 2 weeks, is also said to entail a rapid, thready pulse with a fall in blood pressure, acute loss of body weight, profuse clammy perspiration, and hyperpyrexia.[3] Exhaustion post-psychoses is most common in mania and catatonic schizophrenia. Etiological factors identified are predisposing parasympathetic nervous system ascendancy in the neural and vascular interrelations, accumulation of toxemic catabolites, malnourishment leading to deficiencies, dehydration, sodium loss, and lowered secretion of adrenal cortical hormones, with sustained small blood vessel dilatation and blood stasis.[3] Our patient presented similarly but had a unique presentation of hypersomnolence without the use of chemicals or physical restraints. The orthomolecular/megavitamin hypothesis posits various deficiencies in the etiology of affective states. Yet, this hypothesis undermines the importance of cobalamin deficiency in precipitating psychiatric illness.[4] One of the reasons for this oversight is the confounding normalcy appreciated in bone marrow and blood cell values of B12-deficient individuals with coexisting psychiatric morbidities.[2] The most common indications to test for vitamin B12 deficiency include anemia with macrocytosis with mean corpuscular volume (MCV) > 100, known gastrointestinal disorders associated with vitamin B12 deficiency, or a vegan diet.[5] Our patient presented with normal blood cell values, but the persistent complaint of gastrointestinal symptoms prompted the test for vitamin B12.

Several established reports are suggesting the co-occurrence of B12 deficiency and mania.[6–8] A postulated mechanism for the same is the glutamate-mediated excitotoxic effects on demyelinated neurons, especially in the dorsolateral pre frontal cortex (DLPFC).[9] There have also been reports eliciting the link between B12 and hypersomnolence.[10,11] The proposed mechanism for B12 action is the increase in sensitivity to the environmental conditions (light stimulation) that phase-advances the circadian rhythm and causes improved levels of consciousness.[11] Further, they highlight the poor response to tranquilizers, antidepressants, and anticonvulsants, as well as stimulants for hypersomnolence with behavioral disturbances.[11] A similar picture was observed in our patient as well.Mania is associated with a reduced need for sleep, and sleep deprivation is known to precipitate an episode of mania. Furthermore, prolonged sleep at initial hospitalization, post-an acute episode, is an early marker for mania resolution.[11] Adequate sedation is also the mainstay of treatment in exhaustion syndrome post psychoses.[3] While vitamin B12 deficiency is implicated in both conditions, in our patient, the natural onset of hypersomnolence seems to have had a protective effect. Further, the correction of B12 levels seems to have resolved not only the hypersomnolence but also affective psychoses.

In the present case, a probable explanation for our findings could be a chronic undetected B12 deficiency that not only predisposed the onset of the first episode of mania 5 years ago but also perpetuated the relapses along with inadequate compliance to treatment. The current episode of mania is proposed to have a bidirectional relationship with B12 deficiency, with increasing severity of symptoms worsening the deficiency as evidenced by the occurrence of concurrent gastrointestinal symptoms. Also, correcting the B12 levels alone has shown marked improvement in symptoms where all other treatment modalities failed, further strengthening the association.

CONCLUSION

While B12 deficiency has been linked with mania and hypersomnolence individually in different reports, this is the first case report, to our knowledge, that links all three. We suppose that the deficiency led to both mania as well as hypersomnolence, but coincidentally, the latter presentation worked as a protective mechanism for the former. Further studies are warranted to conclude whether this is a one-off phenomenon or just a scratch on the surface of something deeper.

Ethical approval

Institutional Review Board approval is not required.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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