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Vitamin B 12 Deficiency Precipitating Mania, Exhaustion with Hypersomnolence - A Unique Perspective

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Abstract: ***Vitamin B12, or cobalamin, plays a crucial role in neurological and psychiatric health. This case report presents a 33 - year - old male with Bipolar Affective Disorder and severe mania, which included irritable mood, insomnia, and violent behavior. Despite initial treatments, he developed hypersomnolence and lethargy. Comprehensive testing revealed a significant vitamin B12 deficiency 100 picogramsmL. After intensive vitamin B12 therapy, the patients symptoms improved markedly. This report underscores the importance of recognizing and treating vitamin B12 deficiency in psychiatric conditions, highlighting a unique case where B12 deficiency was linked to both mania and hypersomnolence.***

Keywords: Vitamin B12 deficiency, Bipolar Affective Disorder, mania, hypersomnolence, psychiatric symptoms

1. 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 - 950 picograms/mL[1.] . Levels below 200pg/mL can precipitate mania, depression, dementia, chronic fatigue etc. in the absence of clinical and investigative evidence of anaemia [2].

2. Case Report

A 33/M with no known medical comorbidities or addictions, working as a bank manager, diagnosed with Bipolar Affective Disorder, five years ago, with three manic and two depressive episodes presented with a nine 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 patient was on irregular treatment with lamotrigine 100mg and olanzapine fluoxetine 5/20 mg combination for three months. Patient was referred to the casualty of a general hospital in West Bengal, where he was injected with 1000mg sodium valproate. Sedation was achieved and patient was admitted to the orthopaedics ward for stabilisation of the arm. Inpatient treatment was administered for three days during which patient remained lethargic with excessive perspiration, mild abdominal pain with constipation and sensation of bloating. His blood pressure measured in the low 90/60 mmHg with 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 ECG and ultrasound abdomen done were also normal. Notwithstanding conservative management, he was prescribed clonazepam 1mg. He was referred to a psychiatrist prior to discharge who advised restarting his combination medication but patient was non - compliant.

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

3. Discussion

Psychotic patients in continuous mental and motor excitement are known to precipitate exhaustion, which untreated, can lead to death. The exhaustion syndrome, between two days to two weeks, is said to also entail rapid thready pulse with 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 anaemia with macrocytosis with 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.

There are several established reports suggesting the co - occurrence of B12 deficiency and mania [6, 7, 8]. A postulated mechanism for the same is the glutamate mediated excitotoxic effects on demyelinated neurons especially in the 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, anticonvulsants as well as stimulants for hypersomnolence with behavioural 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 hospitalisation, post an acute episode, is an early marker for mania resolution [12]. 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, 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 first episode of mania five years ago, but also perpetuated the relapses along with the 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 occurrence of concurrent gastrointestinal symptoms. Also, correcting the B12 levels alone has shown marked improvement in symptoms were all other treatment modalities failed, further strengthening the association.

4. 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.

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