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Psychiatric Presentation of a Severe Vitamin B12 Deficiency Associated with Biermer's Disease: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. Author EAY designed the study, and wrote the manuscript. Authors EAY, SS, MN and HM analyzed and interpreted the patient data regarding case presentation. Authors BY and BA supervised the study. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

This case report highlights a 57-year-old female patient presenting with psychiatric symptoms such as depressive mood, social withdrawal, and apathy followed by gait disturbances and tremor, which led to the diagnosis of severe Vitamin B12 deficiency-induced encephalopathy. Despite initially displaying only psychiatric symptoms, MRI revealed cerebral leukoencephalopathy. Metabolic and auto-immune evaluation confirmed profound Vitamin B12 deficiency secondary to Biermer's disease. Treatment with Vitamin B12 replacement therapy resulted in a favorable outcome. This

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case underscores the importance of considering Vitamin B12 deficiency in patients with neuropsychiatric symptoms, as timely diagnosis and treatment can prevent long-term complications.

Keywords: Vitamin B12 deficiency; cobalamin; psychiatric presentation; encephalopathy; biermer's disease.

1. INTRODUCTION

Vitamin B12 deficiency is associated with a wide range of neuropsychiatric manifestations, ranging from subtle cognitive impairments to severe psychiatric disorders [1,2]. Deficiency in vitamin B12 can result from various factors, including inadequate dietary intake, malabsorption syndromes, and certain medications [3]. The neurological and psychiatric manifestations of vitamin B12 deficiency can manifest insidiously and mimic other conditions, posing diagnostic challenges. We report the case of a patient presenting with neuropsychiatric manifestations revealing a vitamin B12 encephalopathy secondary to biermer's disease.

2. CASE PRESENTATION

A 57-year-old female patient, with a history of type 2 diabetes treated with insulin, was referred to our department due to a progressive behavioral disorder progressing over the past four months. characterized by withdrawal from family, social isolation, apathy, depressed mood with episodes of spasmodic crying, and difficulty falling asleep. Subsequently, the patient experienced gait disturbances, including reduced walking perimeter, axial and limb tremors, and urinary incontinence, alongside total anorexia, asthenia, significant weight gain, and headaches without vomiting or fever. The patient denied any alcohol consumption or malnutrition.

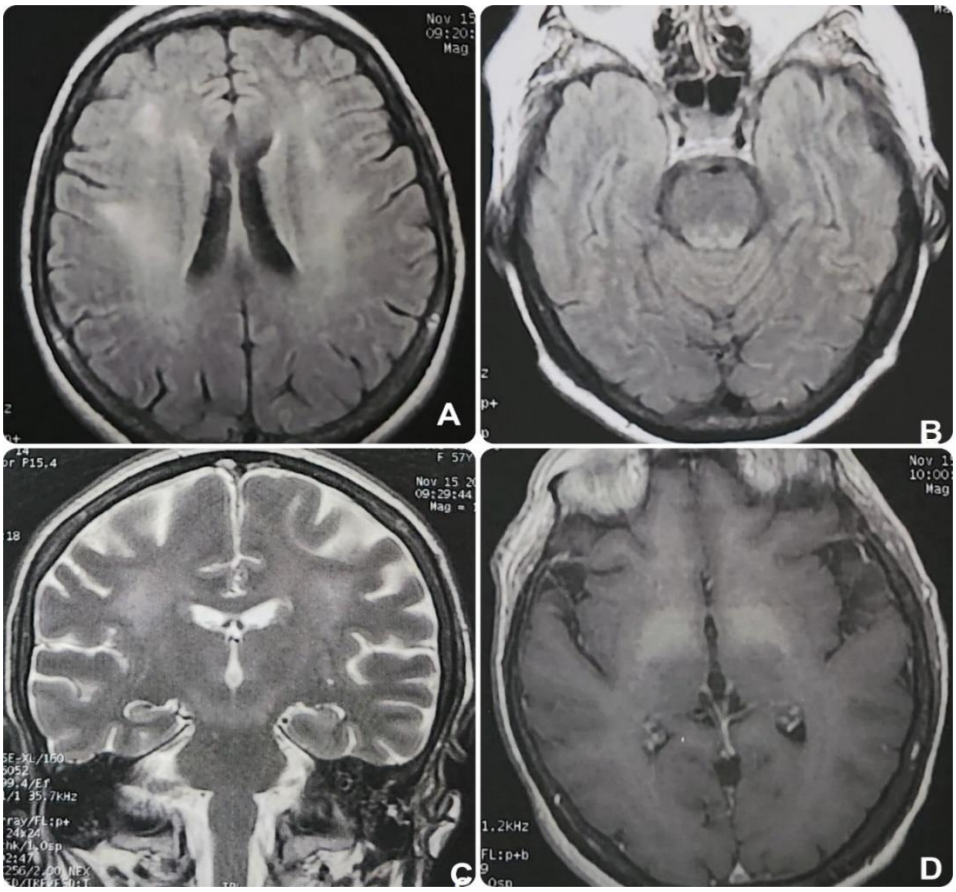

Fig. 1. Cerebral MRI in axial T2 FLAIR (A,B) and in coronal T2 (C) showing periventricular and periacqueductal white matter hyperintensities. Axial T1 sequence (D) showing bilateral pallidal hyperintensity

Upon clinical examination, the patient presented disorientation in time and space, apathy, and absence of facial expressions, with slightly discolored conjunctivae and android obesity resulting in a BMI of 35. Gait was ataxic, exacerbated by eye closure. Romberg's sign was negative. Muscle strength remained intact with spastic hypertonia on the left side, accompanied by brisk reflexes in all four limbs and bilateral Babinski sign. Sensory functions were intact, and no coordination disorders were observed. Oculomotor examination showed no ophthalmoplegia or nystagmus. Assessment of higher cognitive functions revealed attention and memory deficits consistent with anterograde amnesia.

Brain MRI displayed bilateral and roughly symmetrical T2 and fluid attenuated inversion recovery (FLAIR) hyperintensities in the periventricular and periaqueductal white matter, associated with bilateral T1 hyperintensity of the striatum (Fig. 1). EEG indicated a global slowing of background activity without paroxysmal anomalies. Lumbar puncture findings were unremarkable. Metabolic panel results demonstrated normal levels of thiamine, vitamins B6, and B9, but severely decreased vitamin B12 levels at 62.50 pg/ml, associated with macrocytic anemia. The remainder of the metabolic and infectious workup returned normal results. Upper gastrointestinal endoscopy revealed erosive pan-gastritis alongside positive anti-intrinsic factor and anti-parietal cell antibodies, suggestive of biermer’s disease.

The patient was treated with a regimen of daily intramuscular injections of 5000ug of hydroxocobalamin for 10 days, followed by weekly injections for a month, and then monthly injections. The patient positively responded to the vitamin B12 replacement therapy, experiencing improvement in psychiatric symptoms and resolution of limb tremors within a three-month period.

3. DISCUSSION

Vitamin B12, also known as hydroxocobalamin, serves as a pivotal cofactor for the enzymes methionine synthase and L-methylmalonyl–coenzyme A mutase. This vitamin is crucial for the development and initial myelination of the central nervous system, as well as for maintaining its normal function [3]. Deficiency in Vitamin B12 represents a significant global health issue, with prevalence rates varying across different populations and regions [3,4]. Biermer’s disease, also called pernicious anemia, an autoimmune disorder characterized by the presence of anti-gastric parietal cell and anti-intrinsic factor antibodies, is the predominant cause of Vitamin B12 malabsorption [5].

Vitamin B12 deficiency can often be overlooked in patients if only total serum vitamin B12 is used as a status marker. Consequently, measurement of additional functional biomarkers is recommended, such as methylmalonic acid (MMA) and homocysteine, to accurately diagnose vitamin B12 deficiency. This is particularly pertinent for individuals with borderline vitamin B12 levels, defined as rates between 140 and 300 pmol/L, where elevated levels of MMA and/or homocysteine may provide crucial diagnostic evidence of a deficiency [6,7].

The neuropsychiatric manifestations of vitamin B12 deficiency are diverse and can affect multiple cognitive and affective domains. Cognitive impairments commonly include deficits in memory, attention, executive function, and processing speed. Psychiatric symptoms may range from subtle mood disturbances such as irritability and apathy to more severe presentations resembling depression, psychosis, or even dementia [8]. Neurological manifestations may include peripheral neuropathy, myelopathy, and optic neuropathy [9]. The heterogeneity of clinical manifestations emphasizes the necessity of conducting a comprehensive evaluation, including meticulous history-taking, thorough physical examination, and relevant laboratory testing.

Cerebral MRI findings are often nonspecific and should be interpreted in conjunction with the clinical context. It may reveal leukoencephalopathy with diffuse hyperintensities on T2 and FLAIR sequences in the periventricular white matter, occasionally accompanied by involvement of the basal ganglia [10,11].

In our patient, psychiatric and behavioral manifestations preceded neurological symptoms. Metabolic and autoimmune investigations, including digestive explorations, led to the diagnosis of encephalopathy due to profound Vitamin B12 deficiency secondary to biermer’s disease. Additionally, cerebral imaging in our case revealed lesions in the periaqueductal gray matter, suggesting the possibility of an association with Wernicke's encephalopathy in the setting of normal thiamine levels [12].

4. CONCLUSION

Vitamin B12 deficiency can present with a spectrum of cognitive, psychiatric, and neurological symptoms, with psychiatric manifestations often playing a prominent role in diagnosis. Our case report underscores the necessity of a thorough and multidisciplinary diagnostic approach to vitamin B12 deficiency, emphasizing the importance of early recognition and treatment to prevent long-term complications and improve outcomes for affected individuals.

CONSENT

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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