Case report

Psychiatric presentation of a severe vitamin B12 deficiency associated with biermer's disease:a case report

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 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 service 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.

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 (Figure 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 positively responded to vitamin B12 replacement therapy, experiencing improvement in psychiatric symptoms and resolution of limb tremors within a three-month period.

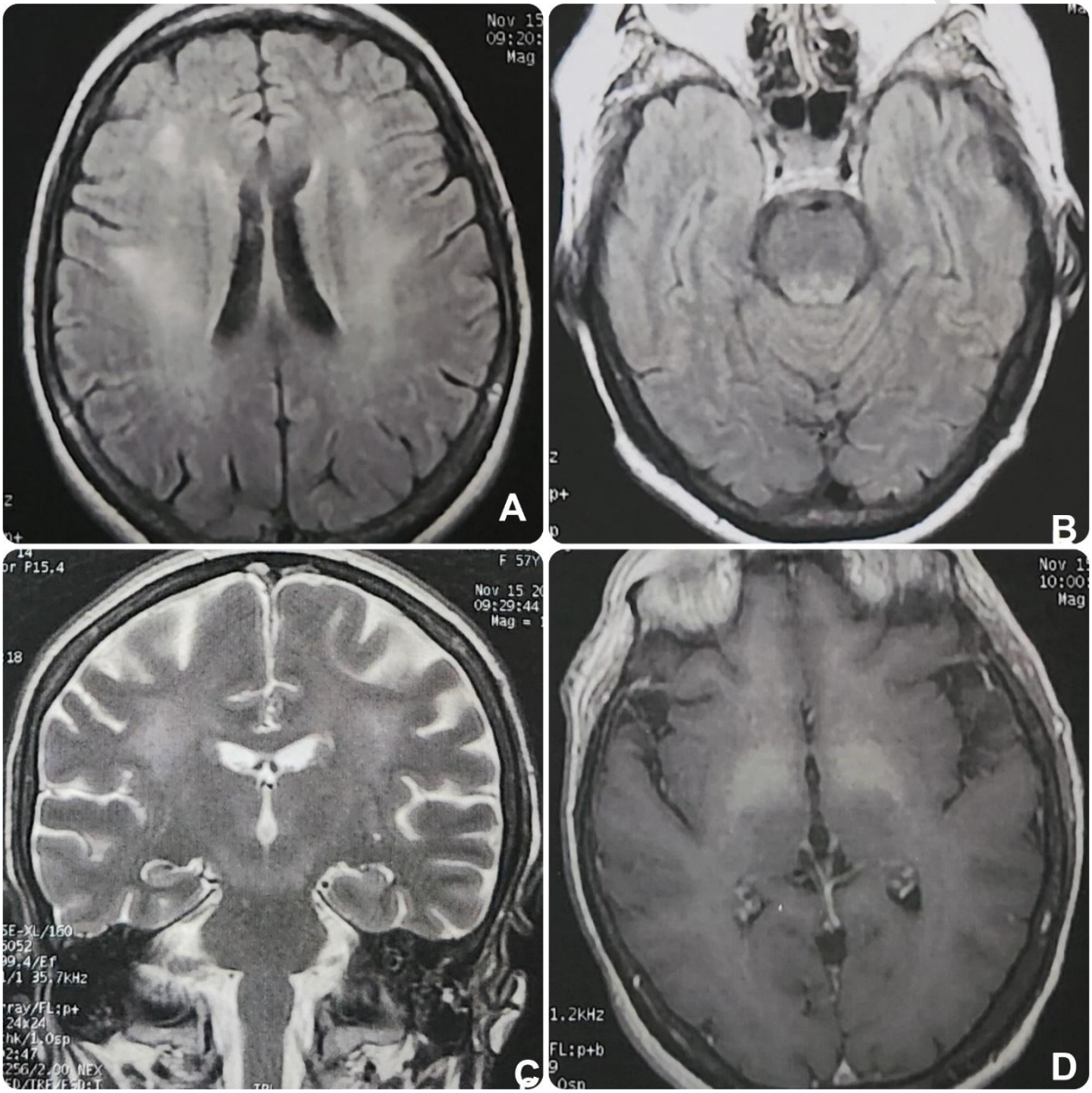

Figure 1: Cerebral MRI in axial T2 FLAIR (A,B) and in coronal T2 (C) showing periventricular and periacqueductal white matter hyperintensities. Axial T1 sequence (D) showing bilateral pallidal hyperintensity.

3) Discussion :

Vitamin B12 deficiency is a global health concern, with varying prevalence rates across different populations and regions [3]. 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 [4]. Neurological manifestations may include peripheral neuropathy, myelopathy, and optic neuropathy [5]. The heterogeneity of clinical manifestations emphasizes the necessity of conducting a comprehensiveevaluation, including meticulous history-taking, thorough physical examination, and relevant laboratory testing.

Although serum levels of vitamin B12 are frequently measured, they may not consistently reflect tissue reserves or functional insufficiency. Supplementary examinations such as methylmalonic acid (MMA) and homocysteine levels may provide supplementary insights, particularly in instances of borderline vitamin B12 levels. 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 [6, 7].

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 [8].

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. Early recognition and treatment are essential to prevent long-term complications and improve outcomes for affected individuals.

Consent:

As per international standards, the patient's written consent has been collected and preserved by the authors.

Ethical approval:

Not applicable.

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