Case Report

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Metabolic Encephalopathy From VitaminB12 Deficiency Case Report

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Author contribution: Juan Yang, Hao Huang ,Ya Chen ,Renfang Hao,Yutao Yang,Yaqin Zhang.Hao Huang:Conceptualization original. Ya Chen :conceptualization and investigation. , Renfang Hao, Yaqin Zhang:conceptualization. Yutao Yang: Writing – Original Draft Preparation. Juan Yang, Hao Huang ,Ya Chen ,Renfang Hao: supervision and conceptualization. Juan Yang,: Writing – Review & Editing.All authors contributed to the article and approved the submitted version.

Received Date: 22 Dec 2025
Accepted Date: 08 Jan 2026
Published Date: 16 Jan 2026

Citation:
Juan Yang, Metabolic Encephalopathy From VitaminB12 Deficiency Case Report.International Journal Clinical and Medical Case Reports, 2025; 7: 1-4

1. Abstract

Vitamin B12 deficiency represents a prevalent nutritional disorder, primarily arising from insufficient dietary intake, malabsorption, or impaired metabolic utilization. Populations at elevated risk comprise older adults, individuals adhering to vegetarian diets, patients with gastrointestinal disorders—such as atrophic gastritis and Crohn's disease—and those with a history of gastrointestinal surgery. Vitamin B12 is essential for DNA synthesis, erythropoiesis, and neurological integrity. Deficiency may result in megaloblastic anemia, metabolic disturbances, and impaired neurological function. Metabolic encephalopathy attributable to vitamin B12 deficiency is a neurometabolic disorder arising from inadequate dietary intake, malabsorption, or impaired metabolic conversion of the vitamin. Prompt administration of vitamin B12 can lead to substantial clinical improvement, while delayed intervention is associated with an increased risk of irreversible neurological sequelae

2. Keywords: Metabolic encephalopathy, Vitamin B12 deficiency, Anti-intrinsic factor antibodies, Megaloblastic anemia

3. Introduction

Metabolic encephalopathy represents a serious neurological manifestation frequently induced by systemic metabolic imbalances. Notably, cobalamin (vitamin B12)—an essential micronutrient implicated in numerous metabolic pathways—is synthesized exclusively by certain bacteria and archaea in the natural environment[1]. Metabolic encephalopathy secondary to its deficiency is clinically characterized by cognitive impairment, depression, confusion, and progressive dementia[2][3]. Furthermore, vitamin B12 deficiency constitutes a leading cause of pernicious anemia, often characterized by highly specific elevations in anti-intrinsic factor antibody (IFA) titers. Although awareness of vitamin B12 deficiency-associated metabolic encephalopathy is increasing, it remains frequently underrecognized in clinical settings. Through a illustrative case report and supporting literature review, this article aims to improve recognition of this condition and facilitate earlier, more precise diagnostic and therapeutic interventions.

4. Case Presentation

On January 13, 2025, a 48-year-old female was admitted to the Affiliated Hospital of Zunyi Medical University presenting with abnormal psychiatric behavior of over one month's duration, which had recently worsened accompanied by limb weakness for more than two weeks. Approximately one month before admission, the patient began experiencing intermittent psychiatric symptoms in the absence of identifiable precipitating factors. These episodes were characterized primarily by visual hallucinations and persecutory delusions, with notable exacerbation during nighttime. Following a two-week course of treatment with olanzapine and sodium valproate (dose and frequency unspecified) at a local hospital, the patient's family reported further deterioration of her psychiatric condition.

The patient exhibited persistent neuropsychiatric symptoms with progressive frequency, characterized by impaired facial recognition, episodes of incoherent speech, and choreiform movements of the upper limbs. Concurrently, she developed bilateral lower limb weakness that resulted in loss of ambulation. For further diagnostic evaluation and management, the patient was evaluated in the neurology outpatient clinic of our institution and subsequently hospitalized in the neurology department with a provisional diagnosis of "mental disorder, etiology undetermined". The patient had a history of hyperthyroidism diagnosed over two decades ago and anemia identified five years ago, both of which were managed irregularly with unclear therapeutic outcomes. Temperature 36.5°C, heart rate 77 beats/min, respiratory rate 20 breaths/min, blood pressure 99/65 mmHg. The patient exhibited a cachectic appearance and clinical signs consistent with anemia. Physical examination of the cardiac, pulmonary, and abdominal

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systems revealed no abnormalities.

Neurological Examination: The patient is conscious and partially responsive to questions. Cooperation could not be achieved for higher cognitive function assessment. Both pupils are round and equal in size, approximately 3.0 mm in diameter, with prompt direct and indirect light reflexes. The frontal wrinkles and nasolabial grooves are symmetrical bilaterally. Involuntary movements are observed in all four limbs. Muscle strength testing in the extremities could not be performed due to lack of cooperation. Muscle tone is increased in both upper limbs. Tendon reflexes are present and symmetrical in all four limbs. Bilateral Babinski signs are equivocal. Further examination could not be completed due to lack of cooperation.

4.1. Laboratory Findings

Complete blood count revealed leukopenia (white blood cell count: 2.51×10^9/L), anemia (red blood cell count: 2.56×10^{12}/L; hemoglobin: 73.0 g/L), and thrombocytopenia (platelet count: 74×10^9/L; plateletcrit: 0.07%). Serum biochemical analysis demonstrated hypocalcemia (calcium: 2.17 mmol/L), decreased free T3 (2.3 pmol/L), and elevated homocysteine (66.6 μmol/L). Potassium, sodium, and chloride levels were within normal limits. Non-contrast Cranial MRI: Neuroimaging revealed symmetrical areas of high signal intensity in the bilateral cerebellum on diffusion-weighted imaging (DWI). Additional lesions were observed in the bilateral frontal and parietal lobes, with subtle signal alterations also noted in the left insular region. The overall imaging findings are consistent with:1.Metabolic or toxic encephalopathy, although encephalitis remains a diagnostic consideration;2.Bilateral cerebral white matter hyperintensities (Fazekas grade 1) and/or chronic cerebral infarcts;3.Mild cerebral atrophy.Contrast-Enhanced Cranial MRI:

Magnetic resonance imaging (MRI) demonstrated linear and nodular areas of contrast enhancement within the right cerebellar dentate nucleus. Additionally, generalized cerebral atrophy was observed.January 16, 2025: Lumbar puncture was performed, revealing an intracranial pressure of 150 mmH_2O. Cerebrospinal fluid appeared clear, with lactate dehydrogenase level of 55 U/L and CSF protein quantification of 506 mg/L. No other significant abnormalities were detected.January 17, 2025: Vitamin B_{12} level was 28 pg/mL. Two atrophic gastritis antibodies: IFA 1618.28 U/mL. D-dimer 0.82 μg/mL, absolute reticulocyte count 12.6×10^9/L. Bone marrow aspiration demonstrated morphological features consistent with megaloblastic anemia, with toxic changes in granulocytes. No significant abnormalities were found in liver/kidney function, viral serology (four items), rheumatoid disease-related tests, female tumor-associated antigens, genetic metabolic disease amino acid and acylcarnitine profile analysis, or comprehensive urine organic acid analysis. Blood ammonia and autoimmune encephalitis panel showed no notable abnormalities. Etiological and Topographic Diagnosis Based on History and Auxiliary.Magnetic resonance imaging (MRI) demonstrated linear and nodular areas of contrast enhancement within the right cerebellar dentate nucleus. Additionally, generalized cerebral atrophy was observed.

4.2. Treatment Course

Following admission, comprehensive diagnostic workup was completed. Based on physical examination, laboratory/imaging findings, and prior medical history, the patient was ultimately diagnosed with metabolic encephalopathy secondary to vitamin B12 deficiency. Treatment regimen included:1.Vitamin B1 injection (1 mg) + Vitamin B12 injection (1 mg) intramuscularly once daily, 2.Oral methylcobalamin tablets (0.5 mg three times daily) to address B12 deficiency, 3.Shengxuebao mixture (15 mL three times daily), 4.Leucogen tablets (20 mg three times daily), 5.Diyu Sheng Bai tablets (0.2 g three times daily) to ameliorate trimethylcytopenia. After 12 days of structured treatment, the patient demonstrated gradual improvement with intermittent speech recovery. Follow-up contrast-enhanced cranial MRI on February 4, 2025, showed notable reduction in lesion size and number compared to the January 15, 2025 MRI study.

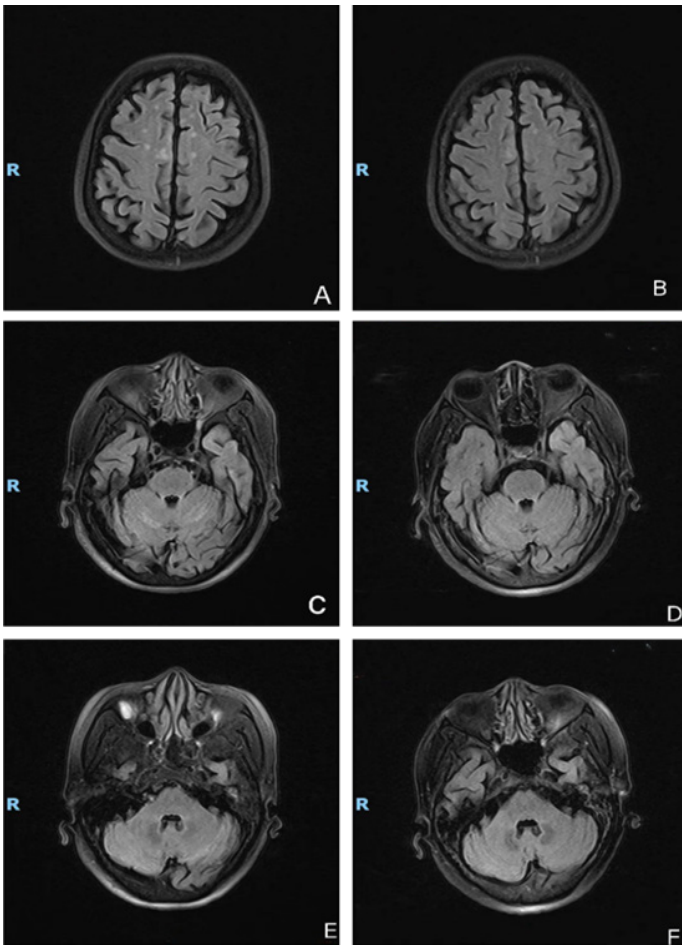

Figure 1Serial cranial magnetic resonance imaging (MRI) of a patient with vitamin B12 deficiency-induced metabolic encephalopathy, before and after treatment.

A, C, E: MRI scans obtained at admission prior to treatment.

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Axial T2-weighted image demonstrates symmetrical hyperintense signals (white arrows) within the bilateral cerebellar dentate nuclei. Axial Fluid-Attenuated Inversion Recovery (FLAIR) image shows patchy and punctate hyperintense signals (arrowheads) scattered in the cerebral white matter.Axial Diffusion-Weighted Imaging (DWI) reveals prominent hyperintensity (restricted diffusion) in the bilateral cerebellar dentate nuclei (arrows).

B, D, F: Follow-up MRI scans after treatment with parenteral vitamin B12 supplementation.

Corresponding axial T2-weighted, FLAIR, and DWI sequences demonstrate significant reduction in the size and extent of the hyperintense lesions in both the cerebellum and cerebral white matter, indicating marked radiological improvement.

5. Discussion

The patient presented with subacute onset of neuropsychiatric symptoms and limb weakness, along with detected vitamin B12 deficiency. Since vitamin B12 deficiency can lead to central nervous system abnormalities, and based on findings from MRI and other examinations suggesting metabolic encephalopathy, the patient was ultimately diagnosed with metabolic encephalopathy caused by vitamin B12 deficiency.

Vitamin B_{12} is an essential water-soluble vitamin for humans, playing an indispensable role in the development of the central nervous system, initial myelination, and the maintenance of normal neurological function[4]. Vitamin B_{12} directly scavenges free radicals, inhibits oxidative damage, supports glutathione activity, and reduces endoplasmic reticulum stress, thereby protecting against oxidative injury and inflammatory stress[1]. It also prevents neuronal apoptosis in various neurological disorders. Consequently, vitamin B_{12} deficiency can lead to neuropsychiatric symptoms[6].

In this case, the patient exhibited neuropsychiatric symptoms such as disrupted sleep-wake cycles and hallucinations (including visual and auditory hallucinations) over one month prior to admission. However, due to limited medical resources, the underlying cause was not promptly identified, and the patient's condition progressively worsened. Upon admission, comprehensive laboratory tests revealed severely low vitamin B12 levels (28 pg/mL). Additionally, vitamin B_{12} deficiency-induced pernicious anemia is often associated with significantly elevated anti-intrinsic factor antibody (IFA) levels (nearly 100% specificity). Intrinsic factor, a carrier protein for vitamin B_{12} in the intestines, is essential for active intestinal absorption via binding to the cubilin-amnion receptor[5]. IFA is a well-established serological biomarker for the diagnosis of autoimmune gastritis[6]. In this patient, IFA levels were markedly elevated at 1618.28 U/mL. Elevated IFA reduces the availability of intrinsic factor necessary for vitamin B_{12} absorption, leading to deficiency[7]and subsequent metabolic encephalopathy.

Pre-treatment cranial MRI revealed symmetrical lesions in the bilateral cerebellum, showing high signal intensity on FLAIR and DWI sequences, along with scattered patchy and punctate prolonged T2 signals in the cerebral white matter with high FLAIR signal intensity. Based on the patient's history, clinical signs, laboratory results, and imaging findings, the diagnosis of vitamin B_{12} deficiency-induced metabolic encephalopathy was established. These changes may be reversible following vitamin B_{12} supplementation therapy. Ultimately, after structured vitamin B_{12} treatment, the patient's clinical symptoms significantly improved.

The patient's clinical presentation and cranial MRI features show considerable similarity to those of methylmalonic acidemia and Wernicke encephalopathy. In methylmalonic acidemia, cranial CT and MRI often reveal bilateral symmetrical basal ganglia lesions and white matter abnormalities[8][9][10], whereas brain MRI in Wernicke encephalopathy typically demonstrates high signal intensity in the dorsomedial thalamic nuclei, periaqueductal gray matter, and the regions around the third or fourth ventricles[11].

In this patient, the anti-intrinsic factor antibody (IFA) level was significantly elevated at 1618.28 U/mL, and vitamin B_{12} was markedly reduced to 28 pg/mL. In contrast, genetic metabolic disease amino acid/acylcarnitine profiling and comprehensive urine organic acid analysis yielded normal results. Based on these parameters, combined with imaging and other laboratory findings, comprehensive analysis of all data is crucial for differentiating other diseases that resemble vitamin B12 deficiency-related metabolic encephalopathy, serving as the cornerstone for definitive diagnosis.

For patients presenting with neuropsychiatric abnormalities and pyramidal tract signs, multiple etiologies must be considered, necessitating thorough examination and precise diagnosis. The diagnosis of vitamin B12 deficiency-induced metabolic encephalopathy is established by integrating a detailed clinical history with pertinent laboratory and neuroimaging findings. Given the nature of this disorder, early diagnosis and prompt intervention are paramount, as they can effectively halt disease progression, improve functional outcomes, and prevent irreversible neurological damage. Consequently, accurate etiological identification, rigorous differential diagnosis, and proactive management form the cornerstone of effective clinical care for this condition.Therefore, this case report aims to share diagnostic and therapeutic experiences, improve clinicians' awareness of this disease, and ultimately guide clinical practice more effectively.

We believe this case provides valuable clinical insights; however, its academic impact could be enhanced in two key areas to better align with the standards of international journals. First, the Discussion section could be strengthened by more directly contextualizing the novel aspects of this case—such as the striking radiological resolution and the markedly elevated anti-intrinsic factor antibody titers—within the existing literature. This

would help clarify the case's unique contributions and reinforce the diagnostic rationale. Second, a more systematic delineation of the diagnostic pathway—from initial suspicion based on neuropsychiatric symptoms to the confirmatory roles of serological and imaging biomarkers—would offer readers a clearer, more transferable clinical framework. Addressing these aspects would significantly elevate the manuscript's scholarly value and practical applicability.

Funding: the Natural Science Foundation of China (32160190); the Natural Science Foundation of China (32500866); the Science and Technology Project of Guizhou Health Commission (gzwkj2021-020)

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