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Vitamin B12 deficiency mimicking AIDP: A case report

Fatima Rawas*; Saad Abdul Halim; Raja Sawaya

Department of Neurology, American University of Beirut Medical Center, Beirut, Lebanon.

***Corresponding Author: Fatima Rawas**

Department of Neurology, American University of Beirut Medical Center, Beirut, Lebanon.

Tel: 0096170270194;

Email: rawas1995@hotmail.com

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Abstract

Acute inflammatory demyelinating polyneuropathy typically presents with progressive weakness and numbness in the lower extremities, along with gait abnormalities. However, other conditions can mimic these symptoms, potentially delaying diagnosis and appropriate treatment. We present the case of a 67-year-old man, who did not smoke, drink alcohol, or have diabetes or hypertension, who developed acute progressive weakness and numbness in his lower extremities over five days, along with oscillopsia and unsteady gait. Examination revealed bilateral gaze-evoked horizontal nystagmus, areflexic weakness in both the proximal and distal lower extremities, and a sensory level at T10. MRI scans of the brain and spine were normal, as was a PET scan. EMG (Electromyography) and NCS (Nerve Conduction Studies) conducted upon admission and repeated on the fourth and sixth days showed normal latencies and conduction velocities, with no signs of demyelination or conduction blocks. However, posterior tibial Somatosensory Evoked Potential (SSEP) responses indicated severe delays in cortical responses bilaterally. The Cerebrospinal Fluid (CSF) was acellular with elevated protein levels. Blood tests revealed a significant Vitamin B12 deficiency and elevated homocysteine levels. The patient was diagnosed with Vitamin B12 deficiency and received only parenteral Vitamin B12 replacement during his stay, leading to significant improvement. This case highlights that atypical acute presentations of Vitamin B12 deficiency can resemble demyelinating inflammatory polyneuropathy, underscoring the importance of incorporating Vitamin B12 testing to enable early detection and treatment.

Keywords: AIDP; Vitamin B12; Deficiency; Demyelination; Nerve conduction; Weakness; Numbness.

Abbreviations: AIDP: Acute Inflammatory Demyelinating Polyneuropathy; MRI: Magnetic Resonance Imaging; PET: Positron Emission Tomography; EMG: Electromyography; NCS: Nerve Conduction Studies; CSF: Cerebrospinal Fluid; SCD: Subacute Combined Degeneration; PPIs: Proton Pump Inhibitors; HBA1C: Hemoglobin A1C; Anti-MOG: Anti-Myelin Oligodendrocyte Glycoprotein; CT: Computed Tomography; IV: Intravenous; GIT: Gastrointestinal Tract; SSEP: Somatosensory Evoked Potential; MFS: Miller Fisher syndrome; GBS: Guillain-Barre syndrome.

Introduction

Patients with Acute Inflammatory Demyelinating Polyneuropathy (AIDP) typically present with ascending numbness in the distal extremities and predominantly proximal weakness in the lower extremities. In contrast, patients with vitamin B12 deficiency present with loss of dorsal column sensory modalities and motor weakness due to corticospinal tract involvement. Myeloneuropathy and Subacute Combined Degeneration (SCD) are well-known complications of Vitamin B12 deficiency. While polyneuropathy related to Vitamin B12 deficiency can occur independently of myelopathy, it typically presents with gradual progression, making it relatively easy to differentiate from AIDP both clinically and electrophysiologic ally. However, in cases where Vitamin B12 deficiency presents acutely, distinguishing it from AIDP becomes more difficult [1]. Vitamin B12 deficiency typically leads to neurological symptoms that develop sub acutely, with the median time before diagnosis and treatment being around 4 months. However, there are documented cases of acute Vitamin B12 deficiency, where five patients with SCD presented with symptoms lasting 6 to 15 days and improved after parenteral Vitamin B12 treatment [2].

This case describes a patient with acute, progressive, predominantly proximal weakness of the lower extremities and ascending numbness in the lower extremities. NCS did not confirm AIDP. However, the patient's weakness progressed resulting in quadriparesis. Vitamin B12 supplements led to a dramatic improvement in both motor weakness and sensory deficits.

Case presentation

A 67-year-old male nondiabetic, non-hypertensive, non-smoker nonalcoholic, presented to the hospital with acute progressive proximal lower extremity weakness of five days duration, accompanied by numbness in the lower extremities. He had hip flexion rated 2/5, knee flexion and extension, and ankle dorsiflexion and plantarflexion rated 3/5. He was areflexic and had no Babinski signs. The neurological exam was normal in the upper extremities and the anal tone. The patient had no sensation in all modalities in the lower extremities, and eventually had a sensory level at T10. Additionally, he had bilateral gaze-evoked horizontal beating nystagmus with oscillopsia. The progressive predominant proximal weakness, unsteady gait, areflexia and gaze-evoked nystagmus suggested Miller Fisher Syndrome (MFS). Lower extremity weakness and sensory level suggested a myelopathy despite a normal anal tone. Additionally, a paraneoplastic process was considered because of the combined involvement of the central and peripheral nervous systems. Hematology and chemistry were normal including normal Hemoglobin 14.8 g/dl (13.0-18.0 g/dL) and normal mean corpuscular volume 85 fl (80.0-94.0 fl). Copper, Zinc, Vitamin E and HBA1C serum levels were normal. Serum ganglioside panel, Anti-MOG and Anti-Aquaporin 4 antibodies were all negative. The serum B12 level was low at 125 pg/ml (243-894 pg/mL) with elevated homocysteine level 16.6 µmol/L (5.0-15.0 µmol/L). CSF was acellular with elevated protein 0.69 g/L (0.10-0.50 g/L). 3T MR imaging of the brain and entire spine with gadolinium contrast was normal. Whole body CT and PET scans were normal. EMG and NCS studies conducted on day 1, and repeated on days 4 and 6 of admission, revealed normal latencies and conduction velocities of all sensory and motor fibers in the upper and lower extremities, normal and symmetrical posterior tibial F-responses in all studies, with no evidence of demyelination or conduction blocks. Posterior tibial somatosensory evoked responses showed severe delay in the cortical responses bilaterally. The patient continued to deteriorate in his motor and sensory functions every day. He became bedridden and his sensory level reached T7-8. He developed constipation and was maintained on daily laxatives. Despite the progressive worsening in his presentation, NCS was against an acute or subacute demyelinating peripheral neuropathy, ruling out GBS (Guillain-Barre syndrome) and Miller-Fisher syndrome. The abnormal tibial SSEP, sensory level and the presence of nystagmus rather than ophthalmoplegia with low serum vitamin B12 level suggested subacute combined degeneration with spinal, cerebellar and brain stem involvement. We initiated treatment with IV cyanocobalamin 1000 mcg daily for 7 days, the nystagmus resolved by day 1 and motor power improved (hip flexion increased to 4/5) by day 7. He was discharged walking with a walker on oral cobalamin 1000 mcg daily and after one month he began ambulating with a cane and showed improvement in bilateral lower extremity sensation. To complete the workup, the patient's vitamin B12 deficiency was investigated, revealing normal results from an endoscopic gastro-duodenoscopy with biopsy and a negative malabsorption workup, including endomysial and transglutaminase antibody serology. Upon review, it was found that our patient had been on Proton Pump Inhibitors (PPIs) for the past five years to manage his hiatal hernia, which is likely the primary factor contributing to his deficiency. It has been reported that using PPIs for more than six months can cause vitamin B12 deficiency [3].

Discussion

Our case presented with acute proximal lower extremity weakness, areflexia, and sensory level suggesting initially AIDP. The presence of eye movement disorder suggested Miller-Fisher syndrome, but ophthalmoplegia in MFS is not in the form of horizontal nystagmus. All investigations were normal including repeated NCS even 1 week into the illness. This goes against a peripheral nervous system disorder. His negative brain MRI rules out a central etiology.

The only abnormality noted was his low serum vitamin B12 level. The only treatment our patient received was IV vitamin B12 which directly led to arrest of deterioration and clinical improvement. Vitamin B12 deficiency leads to decreased activity of the cobalamin-dependent methylcobalamin esterase enzyme, resulting in elevated levels of methylmalonic acid, which is toxic to myelin. The neurological manifestations of vitamin B12 deficiency are varied and can occur even without hematologic abnormalities. These manifestations include myelopathy, known as subacute combined degeneration, neuropathy, neuropsychiatric abnormalities, and, less commonly, optic nerve atrophy [4]. Jain et al. noted that advanced MR imaging techniques, such as diffusion tensor imaging, can detect microstructural changes in tissues that appear normal on conventional MRI [5]. Eye movement disorders are infrequently associated with vitamin B12 deficiency. Akdal et al. (2007) described two cases featuring bilateral internuclear ophthalmoplegia and downbeat nystagmus linked to vitamin B12 deficiency [6]. Katsaros et al. (1998) reported a case of subacute combined degeneration in a patient with bilateral horizontal gaze-evoked nystagmus, which was accompanied by abnormal signals in the medulla oblon-

gata, pons, and mesencephalon on T2-weighted MRI images [7]. Cao et al. found that a shorter clinical course of Subacute Combined Degeneration (SCD) is associated with a higher rate of complete symptom resolution [8]. Puntambekar et al. reported on a 43-year-old patient with vitamin B12 deficiency due to pernicious anemia, who presented with a sharp sensory level at T11, bowel and bladder dysfunction, and unusual autonomic symptoms [9].

Conclusion

When a patient presents with acute worsening of areflexic proximal lower extremity weakness, a sensory level deficit, bilateral nystagmus, and normal NCS alongside a normal brain MRI, vitamin B12 deficiency should be strongly considered as a potential diagnosis. Despite the rarity of eye movement disorders and sensory levels in vitamin B12 deficiency, these symptoms can still occur and should prompt early testing of vitamin B12 serum levels. Conventional MRI often has low sensitivity (less than 50%) for detecting the microstructural damage associated with this deficiency, making it an unreliable tool for early diagnosis. Given the potential for severe and irreversible neurological damage, if left untreated, early diagnosis and prompt supplementation of vitamin B12 are crucial. The atypical symptoms, such as bilateral nystagmus and sensory level, should not rule out B12 deficiency, as the consequences of delayed treatment can be devastating. Therefore, clinicians should maintain a high index of suspicion and prioritize testing for vitamin B12 levels in similar clinical scenarios to prevent long-term complications.

Declarations

Disclosure: Written informed consent was obtained from the individual for the publication of data included in this article. Nothing to disclose.

Conflict of interest: The authors declare that they have no conflict of interest.

Contributions: Dr Fatima Rawas: Writing- review & editing, Writing-Original draft Dr Saad Abdul Halim: Writing -original draft Dr Raja Sawaya: Writing-review & editing, Supervision.

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