Pseudo-Orthostatic Tremor as a Manifestation of Vitamin B12 Deficiency: A Case Report

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ABSTRACT

Vitamin B12 deficiency has long been known to present with various neurological manifestations, but only rarely presents as movement disorders, especially in adults. We present the case of a 30-year-old vegan male presenting with tremors on both legs when standing which was relieved by vitamin B12 supplementation. To the best of our knowledge, this is the first documented case of slow orthostatic tremor or pseudo-orthostatic tremor caused by vitamin B12 deficiency.

Key words Vitamin B12 Deficiency, Cobalamin, Vegan, Movement Disorders, Tremor, Orthostatic Tremor, Shaky-leg Syndrome, Electromyography

INTRODUCTION

Neurological damage due to vitamin B12 deficiency has been well documented, and the clinical manifestations are diverse, as our nervous system can be damaged from vitamin B12 deficiency at multiple locations. Among these manifestations, movement disorders resulting from cobalamin deficiency are relatively rare, especially in adults.[1]

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Tremors, myoclonus, cerebellar ataxia and extrapyramidal manifestations like dystonia and chorea are some that have been documented in adults.[1] This case report documents the case of vitamin B12 deficiency presenting as a slow orthostatic tremor (OT) or pseudo-orthostatic tremor.

Case Presentation

A 30-year-old male working as a bank manager in Jakarta presented with a tremor on both legs for a week prior to consultation. He reported dizziness, easy fatigability and occasional tingling sensation on both feet. He also felt unsteadiness when walking and required the support of a cane to walk. He has needed to take medical leave from work and requires someone to accompany him at all times because of the fear of falling.

There was no relevant past medical history and no family history of tremors or movement disorders. The patient does not smoke or drink alcoholic beverages and denies the use of illicit drugs. He has been a vegetarian for 5 years and started on a vegan diet 6 months ago.

On physical examination, a visible tremor in both thighs was found, emerging almost immediately upon standing. Leg strengths were 5/5 on manual muscle testing. There was no spasticity and deep tendon reflexes were normal. Cerebellar function tests were normal. Sensory tests for pain, temperature and touch were normal. The patient had a wide-based gait and often started to fall within several minutes of stance when he was without support. The tremor improved with use of support, and was relieved with sitting and lying down, but did not improve with walking.

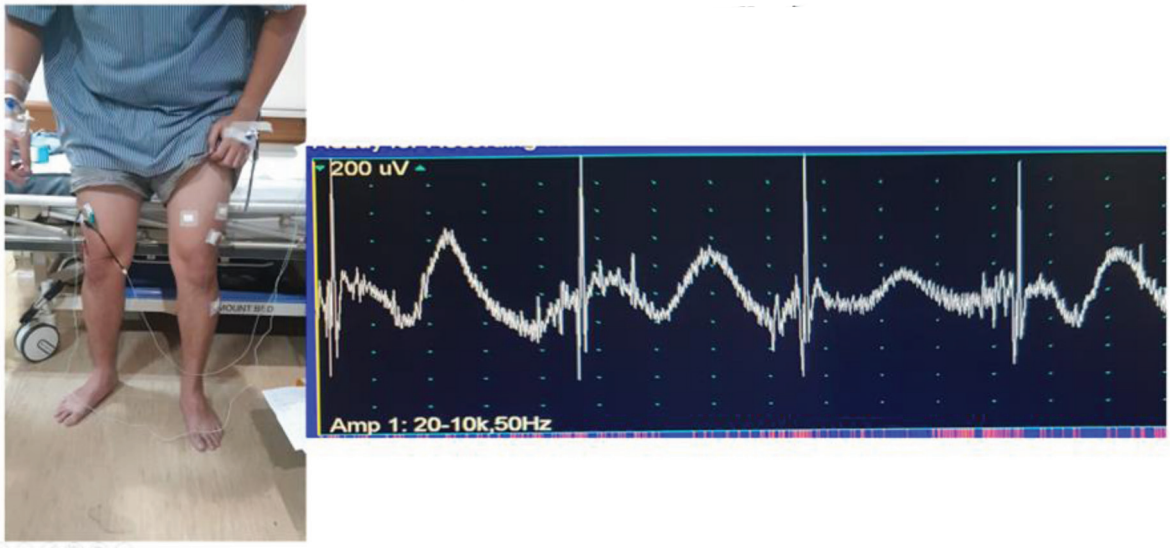

Figure 1 Surface Needle EMG of the left Quadriceps Femoris in the standing position showing compound muscle action potential of the Quadriceps muscle with high amplitude, high synchronized discharges of 5-6 Hz

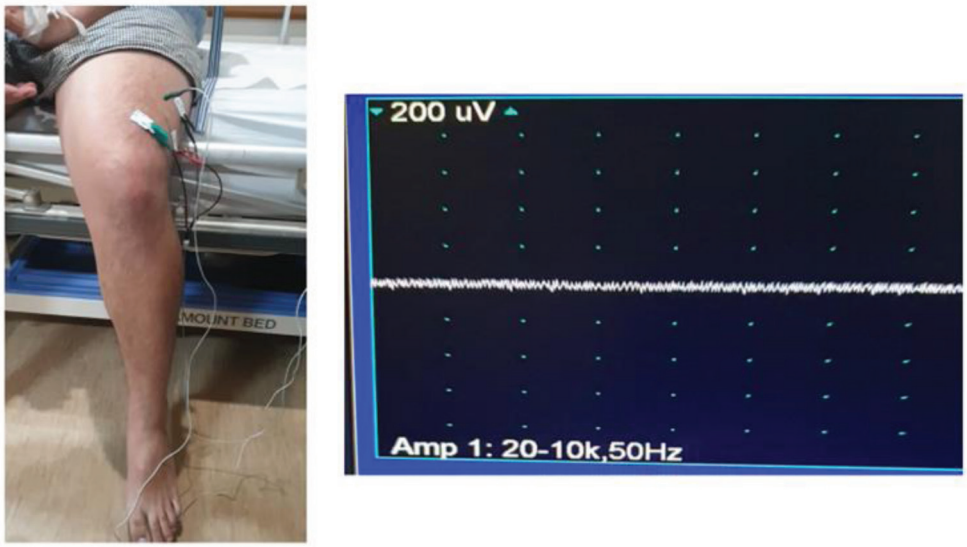

Figure 2 Surface Needle EMG of the left Quadriceps Femoris in the sitting position showing no sign of tremor or muscle activity

On ancillary investigations, laboratory findings revealed megaloblastic anemia with hemoglobin level of 11.2 g/dL and mean corpuscular volume (MCV) of 101 fL/RBC with vitamin B12 level <150 pg/L. Non-contrast cranial and whole spine MRI were unremarkable.

On surface electromyography, we found high amplitude synchronous slow tremor of 5-6 Hz on both quadriceps femoris muscles when the patient was standing (Figure 1), which was absent during sitting or lying down (Figure 2). Nerve conduction studies of the peroneal and tibial nerves showed normal compound muscle action potentials (CMAP) and f-waves. Sensory nerve action potentials were normal for both sural nerves. H-reflex were also normal for both tibialis nerves.

Methylcobalamin 1000 µg intramuscular injections were given every other day with gradual relief of symptoms. After 3 weeks of treatment, the patient was then switched to oral supplementation of methylcobalamin 500 mcg daily. He was also sent to a nutritionist to consult about his dietary needs.

DISCUSSION

To the best of our knowledge, this is the first documented case of slow orthostatic tremor or pseudo-orthostatic tremor caused by vitamin B12 deficiency.

Orthostatic tremor (OT), also referred to as shaky leg syndrome, is a rare condition characterized by high frequency (13-18 Hz) tremor of the legs during stance, with a subjective feeling of unsteadiness. In 2018, The International Parkinson and Movement Disorder Society proposed the term "pseudo-orthostatic tremor" to describe orthostatic tremors with a frequency of <13 Hz, suggesting it to be a separate entity from classic orthostatic tremor.[2] Despite that, there is still disagreement on the proper nomenclature and classification of lower frequency tremors of <13 Hz, alluding to its obscure etiology and pathophysiology.[3]

Pseudo-OT or slow OT, unlike the classic orthostatic tremor, is more often associated with unsteadiness during walking, falls and other neurological signs[3], as found in our patient. Additionally, pseudo-OT has also been shown to have neurophysiologic features distinguishable from classic orthostatic tremors, with longer duration discharges and lower inter-muscular coherence.[4]

Pseudo-OT may be idiopathic or associated with other neurological conditions, but is commonly reported as a symptom of another condition. Among them are cerebellar ataxia, dystonia, myelopathy, Parkinson's disease, parkinsonism, multiple sclerosis, Graves' disease and familial essential tremor.[4,5] In our case, we determined that the pseudo-orthostatic tremor was related to vitamin B12 deficiency.

Numerous factors may lead to vitamin B12 deficiency, including atrophic gastritis and lack of intrinsic factor. In vegetarians or vegans, however, inadequate dietary vitamin B12 intake is usually the main cause. Vitamin B12 is mainly found in animal-based products including meat, milk and eggs, as it is produced in the large intestine of animals. Plants are typically not a source of this vitamin. Consequently, high rates of vitamin B12 deficiency have been seen among vegetarian and vegan populations.[6,7]

The exact mechanism by which vitamin B12 deficiency causes neurological problems, particularly movement disorders, is still unknown. The current hypothesis suggests that it may be caused by combined accumulation of homocysteine, methyltetrahydrofolate, and methylmalonic acid, resulting in basal ganglia dysfunction.[1]

Vitamin B12 supplementation is the mainstay of treatment in movement disorders caused by vitamin B12 deficiency, and when initiated early enough, may reverse neurologic dysfunction.[1] Our patient's symptoms were resolved by administering vitamin B12 supplementation.

Nutritional deficiencies, including B12 deficiency, should not be overlooked as a differential for patients presenting with movement disorder, especially since they respond well to supplementation.[8] When diagnosis is unclear, a careful history, including a thorough review of the patient's diet may provide the answer.

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