Vitamin B12 deficiency presenting with unsteady gait: a case report and review of literature

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ABSTRACT

Background: Vitamin b12 deficiency is associated with wide spectrum of neurological manifestations

Case Presentation: We are reporting a case of 63 years old male presented with history of unsteady gait and was found to have severely low level of vitamin B12, Total resolution of the symptoms occurred following parenteral vitamin B12 replacement therapy.

Conclusion: This case report highlights one of the neurological presentations of vitamin B12 deficiency in a previously healthy individual.

Keywords: Vitamin B12 deficiency, unsteady gait.

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Background

Vitamin B12 deficiency can present with several hematological, gastrointestinal, and neuropsychiatric manifestations. Regarding neurological manifestations, peripheral nerves, spinal cord, optic nerves, and brain are affected by vitamin B12 deficiency but spinal cord is affected early and often exclusively [1].

The major neurological symptoms are typically insidious in onset and can progress in a subacute or chronic manner. Vitamin B12 deficiency presents with gradual onset of paresthesia in hands and feet then patients start to develop weakness and unsteadiness of gait [2].

The recent review by World Health Organization illustrated that most of data on the prevalence of folate and vitamin B12 deficiencies are derived from relatively small and local studies, however, these national survey data from a few countries suggest that deficiencies of both of these vitamins may be a public health problem that could affect many millions of people throughout the world [3].

The risk factor to develop vitamin B12 deficiency can be divided into several categories including decreased ileal absorption, decreased intrinsic factors, genetic causes, prolonged medication use, and inadequate intake.

Case Presentation

A 63 years old Chadian man presented to our primary care clinic complaining of 2 weeks duration of unsteady gait this was preceded with generalized weakness and fatigability associated with numbness sensations of both upper and lower limbs, the numbness in the upper limbs is aggravated by neck flexion, he also reported symptoms of anosmia that started years before this presentation

He does not have any chronic illnesses, no history suggestive of autoimmune disease and no previous gastric or ileal surgeries, His diet was well balanced with no specific restrictions

On physical examination the patient was fully alert, awake and oriented with pallor more pronounced at the palpebral conjunctiva, central nervous system examination revealed positive Romberg test with diminished reflexes, sensation, tone, power and cranial nerve examinations were normal except for loss of smell on olfactory never examination

Hematological tests included a complete blood count (CBC) which showed moderate anemia of 8.9 g/dl (normal: 12–16) with a raised mean cell volume of 115 fl (normal: 80–100) normal white blood cell count.

Serum vitamin B12 levels were very low at 100 pg/ml (normal: above than 300 pg/ml), folate level 33 nmol/l (normal: above than 9.1 nmol/l) liver and renal function tests were all normal.

After the first visit we started the patient on parenteral cyanocobalamin 1,000 mcq deep intramuscular injection every other day for 2 weeks, follow up showed improvement regarding symptoms of unsteady gait and paresthesia, then we continued with preantral cyanocobalamin once weekly for another 2 weeks, then we repeated the

CBC which showed the following improvements hemoglobin 13 g/dl, mean cell volume 90 fl, repeated vitamin B12 is 1,500 pg/ml and intrinsic factor antibodies was negative.

After 6 weeks of the original presentation the patient was seen in the clinic and he reported total resolution of his symptoms of unsteady gait, paresthesia, and anosmia.

Discussion

This case report illustrates the presenting symptoms of combined subacute degeneration of the spinal cord because of vitamin B12 deficiency.

Subacute combined degeneration of spinal cord is due to preferential degeneration of white matter tracts of posterior and lateral column of lower cervical and upper thoracic cord segments. Histologically the first event seen in posterolateral column is myelin edema followed by collection of small foci of tissue destruction into larger ones, giving vacuolated appearance of the tissue. Similar changes are seen in AIDS myelopathy and rarely in systemic lupus erythematosus. Early on there is little gliosis of tissue but later in the chronic state, gliosis is prominent [4].

The importance of this case report is that subacute combined degeneration of the spinal cord is a rare neurological manifestation in Vitamin B12 deficiency, and in our patient the presentation was classical and early in the course of disease and if left untreated the Clinical manifestations may progress to impair position sense and vibration, gait ataxia and up to spastic paraparesis or tetraparesis.

The recommended laboratory evaluation for patients with suspected vitamin B12 deficiency includes a complete blood count and serum vitamin B12 level. A level of less than 150 pg per ml (111 pmol per l) is diagnostic for deficiency [5,6].

There are multiple differential diagnosis that were considered after the initial evaluation of the patient including spinal cord disorders, neuropathies but after reviewing the CBC picture which was requested immediately and it showed a picture of macrocytic anemia raising the suspicion of Vitamin B12 deficiency or folate deficiency which was confirmed later by low levels of vitamin B12 and normal level of folate.

The presumed cause of vitamin B12 deficiency in this patient was inadequate dietary intake of Vitamin B12 rich foods. He also did not have any history of chronic Proton pump inhibitor (PPI) or metformin use or any clinical evidence suggestive of auto immune conditions and malabsorption syndrome.

Both parenteral and oral vitamin B12 replacement therapies have been demonstrated to have equivalent effectiveness in achieving the desirable hematological and neurological remissions regardless of the etiology of vitamin B12 deficiency [7,8].

Approximately 10% of the standard injectable dose of 1 mg is absorbed, which allows for rapid replacement in patients with severe deficiency or severe neurologic symptoms [6].

Intramuscular vitamin B12 treatment was preferred in this patient due to severe deficiency and to improve compliance.

Conclusion

Symptoms of Subacute combined degeneration of the spinal cord due to vitamin B12 deficiency are usually reversible if diagnosed and treated early. However, diagnostic delay or discontinuation of therapy may result in permanent irreversible injury to the spinal cord with disabling neurological impairments, hence, all primary care providers should be familiar with its presentation.

What is new?

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List of Abbreviations

AIDS	Acquired immunodeficiency syndrome
CBC	Complete blood count

Consent for publication

Written informed consent was obtained from the patient for his anonymized information to be published in this article.

Ethics approval

Ethical approval to report this case was obtained from the Primary Health Care Corporation PHCC Research Subcommittee and the Department of Clinical Research in Doha, Qatar.

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Summary of the case

1	**Patient (gender, age)**	Male, 63
2	**Final diagnosis**	Vitamin B12 deficiency
3	**Symptoms**	Unsteady gait, numbness and anosmia
4	**Medications**	Cyanocobalamin 1,000 mcq IM injections
5	**Clinical procedure**	None
6	**Specialty**	Primary care, neurology