

VITAMIN B12 DEFICIENCY- A RARE CASE OF MYELITIS

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ABSTRACT

Vitamin deficiencies are a common problem worldwide, and vitamin B12 deficiency has been identified as a health concern for nearly a century. People all over the world are frequently deficient in vitamin B12. A number of 30–50% of patients with cobalamin deficiency a kind of neurological involvement, for example neuropathy, myelopathy, dementia or optic neuropathy. We present the case of a 56 years old female patient, who suddenly developed gait difficulty due to motor deficit in the lower limbs. After hospitalization in the neurology ward and specialized investigations, thoracic myelitis and axonal sensorimotor polyneuropathy due to vitamin B12 deficiency are diagnosed. The patient underwent functional evaluation at Techirghiol Rehabilitation Sanatorium, where she received individualized treatment for her motor deficit. Despite longstanding recognition of vitamin B12 deficiency, accurately diagnosing the condition and determining the most appropriate management strategy remain challenging.

Keywords: vitamin B12 deficiency; myelitis; rehabilitation

Introduction

Non-traumatic myelopathies may arise from a variety of causes, like inflammatory, immune-mediated, metabolic, neoplastic, infections and vascular ones(1). Vitamin B12 deficiency is a rarer cause of myelitis described in the literature. We found in our search on PubMed, using the key-word combination “myelitis” AND “vitamin B12 deficiency”, without a time filter applied, only 3 matching articles reporting a total of 4 clinical cases (1–3).

Vitamin B12 (also named cobalamin) is a water-soluble vitamin primarily obtained from animal-derived foods, including meat,

particularly red meat, dairy products and eggs (Figure 1). Absorption occurs in the terminal ileum and depends on intrinsic factor, a glycoprotein secreted by gastric parietal cells. After absorption, it plays a role of a cofactor for enzymes involved in synthesis for deoxyribonucleic acid, fatty acids, and myelin (Figure 2) (4).

Vitamin deficiencies are a common problem throughout the world, and vitamin B12 deficiency has been identified as a health concern for nearly a century. People all over the world are frequently deficient in vitamin B12. With age, patients are more vulnerable to cobalamin deficiency (5). Vitamin B12 deficiency has 4 primary etiologies: autoimmune conditions, malabsorption, dietary

insufficiency and toxin exposure (4).

Studies in the literature demonstrated that vitamin B12 deficiency is a causative factor for a number of conditions, including neuropathy, optic neuropathy, myelopathy and dementia. The diagnosis is established by measuring the serum levels of vitamin B12, homocysteine and methylmalonic acid. Myelin and axonal degeneration in the white matter are the most common pathological findings in the spinal cord. The posterior columns of the cervical and thoracic regions are most frequently affected, and involvement of the anterior columns typically indicates advanced and severe disease (2). Psychiatric manifestations including mood disturbances, mental health disorders, and cognitive impairment are common seen in patients. Delayed recognition of these neuropsychological symptoms can lead to irreversible effects, with the timeframe for progression varying from several months to years (6).

A number of 30–50% of patients with cobalamin deficiency a kind of neurological involvement. 30–40% of patients with neurological or hematological symptoms may have normal vitamin B12 concentrations (6).

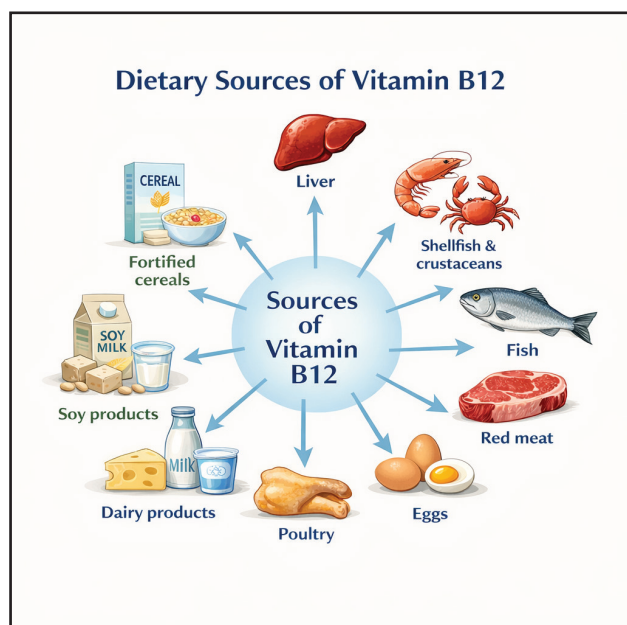


Figure 1: Sources of vitamin B12

Vitamin B12 is primarily obtained from animal-derived foods, including liver, fish, red meat, poultry, eggs, dairy products, and shellfish, as well as from plant-based products such as

cereals and soy products.

Initial assessment typically includes a complete blood count with a peripheral smear, alongside measurement of serum B12 and folate concentrations. Characteristic laboratory findings of B12 deficiency include anemia with reduced levels of hemoglobin and hematocrit, an elevated mean corpuscular volume (over 100 fL), and hypersegmented neutrophils. When serum B12 levels fall within a borderline range (with a value of 200–300 pg/mL), evaluation of methylmalonic acid and homocysteine is recommended. Both are generally elevated in B12 deficiency, whereas folate deficiency tends to raise only homocysteine levels. Once the diagnosis is positive, determining the underlying cause is essential. Surgical history, gastrointestinal disorders causing malabsorption and nutritional deficit should be evaluated first. If those are excluded, autoimmune affections, like pernicious anemia, should be taken into consideration, by testing anti-intrinsic factor antibodies (4).

The treatment and prognosis depend on the stage of cobalamin deficiency and deterioration at the beginning of treatment (2). Vitamin B12 therapy can be administered in two different ways, orally or by injection. The treatment regimen is intramuscular vitamin B12 injections, with a dosage of 100–1000 mcg every day, for 5 days, followed by 100 mcg to 1000 mcg every week, 4 weeks in total, with a maintenance therapy of 100–1000 mcg every 1–3 months. Oral cobalamin is an alternative with similar results for its intramuscular administration, according to a number of studies. A daily dose containing 1000 mcg is efficient in maintaining a normal serum value. Residual neurological abnormalities may occur even though the majority of patients respond well to vitamin B12 treatment. Early stages deficiencies produce better outcomes because, if treatment is not received, the brain may undergo structural and irreversible changes (7).

Case report

We submit the case of a 56 year old active woman, living in the urban environment, who was admitted in the Techirghiol Rehabilitation Sanatorium to receive individualized treatment

for her motor deficit.

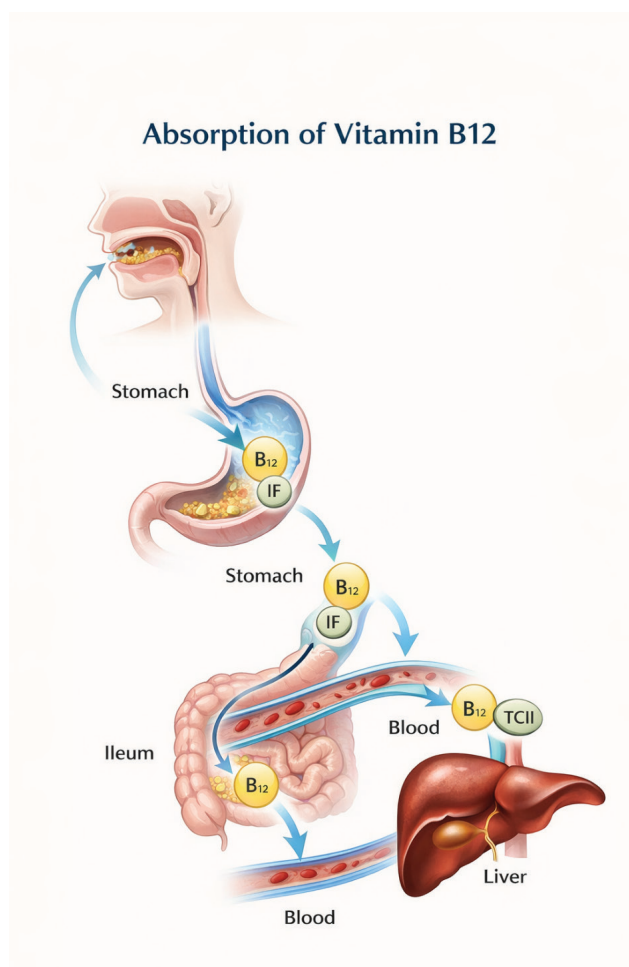


Figure 2: Absorption of vitamin B12. Following ingestion, vitamin B12 binds to intrinsic factor in the stomach, forming a complex that is absorbed in the terminal ileum. After absorption, vitamin B12 enters the bloodstream bound to transcobalamin II and is transported to the liver for storage and distribution to peripheral tissues.

Two months before presentation in our clinic, she suddenly developed gait difficulty and an altered cutaneous sensibility. A few days later she was admitted in an Emergency Care Unit, in the Neurology department for further evaluation.

The laboratory investigations showed elevated LDL cholesterol levels, the presence of leucocytes in urine and a significantly low level of B12 vitamin (60pg/mL with reference ranges between 197-771 pg/mL).

The cerebral and spinal cord Magnetic Resonance Imaging (MRI) showed signs of thoracic myelitis. The electromyography results indicated an axonal sensorimotor polyneuropathy caused by the low levels of B12 vitamin in the patient's body.

During her admission in the Neurology department she received 7 days of intramuscular 1000 mcg vitamin B12 injections and the recommendation to continue after discharge with 1 dose per week for the next four weeks, then 1 dose per month.

She was admitted to our rehabilitation centre due to continued difficulties in walking. We learned that she had a right knee arthroscopic meniscectomy 5 months ago and surgical decompression of carpal tunnel syndrome on both hands 8 years ago.

Physical examination showed us a normoponderal, conscious, cooperative patient with a normal mental status, temporal and spatial oriented, without neck pain or stiffness, with normal oculomotricity, without double vision, facial asymmetry or difficulty of speech; the upper extremities showed no motor deficits or coordination disorders; both lower extremities showed a motor deficit of 4/5 on the MRC (Medical Research Council) scale; tactile, algic

and thermal hypoesthesia below T6 dermatome, vibratory hyperesthesia on both lower limbs and no peroneal nerve palsy; proprioception was absent on both legs; hyperreactive osteodendinous reflexes on both lower limbs; normal reaction to cutaneous-plantar reflex; gait difficulty with bilateral external support.

She was hospitalized for 11 days, while she had an individualized kinethotherapy program, to help her improve walking, limb coordination, postural equilibrium and muscle strength training. She also benefited of massage therapy and magnetotherapy, with an improved functional ability and independence in daily activities, with slightly favorable clinical evolution.

3 weeks after discharge, the patient presented to another Neurology Unit for clinical and biologic re-evaluation.

The objective clinical examination: good general condition, conscious, cooperative, temporal, spatial and al- lopsychic oriented, no meningeal syndrome, no oculomotor disorder, no cranial nerve deficit, tetramelic acropares- thesias lower limbs> upper limbs, paraparesis, sensory disorder with T6-T8 level, muscle atrophy of the lower limbs, gait is possible with unilateral support.

The patient was re-evaluated endoscopically

with gastric mucosal biopsy, which showed: normal esophagus with normal mucosa, normal anterior cardia; stomach with normal distension on insufflation, with vertical portion with visible submucosal vessels, suggestive of corpus atrophy; normal gastric and antral angle, D1 and 2 normal.

Laboratory tests show correction of vitamin B12 deficiency (>2000 pg/mL). Anti gastric parietal cell antibodies and gastric biopsy-currently in work-up.

During hospitalization the evolution was favorable, with improvement of general condition and symptoms.

Discussion

At the onset of gait difficulty caused by reduced muscle strength and altered cutaneous sensation, vitamin B12 deficiency is not typically considered the primary diagnostic option. The symptoms appeared in the context of otherwise apparent good health and had a relatively abrupt onset, prompting the patient to seek emergency care for clinical and functional evaluation and diagnosis. Following a comprehensive rehabilitation program over a 3-week period, the patient showed functional improvement but still required continued rehabilitation interventions to recover muscle strength, sensory function, and the ability to walk without difficulty.

It is widely acknowledged that cobalamin deficiency represents a significant public health concern, especially in developing countries. Cobalamin plays an indispensable role in DNA (deoxyribonucleic acid) synthesis, which is essential for maintaining a normal neurological function. Vitamin B12 forms and maintains a normal function for both central and peripheral nervous system (7).

Although vitamin B12 deficiency was first identified more than a century ago, there are challenges even in the present day to discover the right diagnosis and recommend the best course of action. Vitamin B12 deficiency may be easily overlooked if total serum B12 is the only marker assessed. Moreover, the natural history of B12 deficiency remains incompletely understood.

The process of diagnosis and treatment does not present an important challenge in

typical cases where patients are presenting signs and symptoms which indicate to polyneuropathy and/or megaloblastic anemia, accompanied by low serum B12 levels (8).

As vitamin B12 deficiency can cause neurological symptoms without anemia, so it's critical to diagnose and treat it early to avoid irreversible damage. It is postulated in the literature that treatment should be initiated in patients with serum cobalamin levels below 300 pg/mL. Doctors should be on the lookout for neurological signs or symptoms specific for cobalamin deficiency, within high-risk demographics, even when hematologic abnormalities are absent. Management is particularly critical during pregnancy and lactation due to its role in fetal and infant brain development (7).

Conclusion

Vitamin B12 deficiency has traditionally been associated with hematologic abnormalities. However, its neurological effects are gaining more and more attention, as left untreated can lead to progressive and potentially irreversible neurologic damage. Neurological consequences usually develop subtly, delaying both diagnosis and intervention.

Factors like symptom variability, a lack of knowledge about the condition, failure to detect the variable symptoms in patients and contemporary laboratory serum markers, which can be difficult to access, could all be contributing factors in diagnosis delay of vitamin B12 deficiency. It is generally accepted that educating patients and physicians can help to remove some of these barriers. Symptoms affecting the central and peripheral nervous system are the most challenging to link to this affection, emphasizing the need for more specific awareness in the medical field and an enhancement in the range of examining options available to patients.

Conflict of interest statement: The authors declare no conflict of interest.

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