Case report

Vitamin B12 Deficiency with Sub Acute Combined Degeneration of Cord: A Case Report

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Abstract

Vitamin b12 deficiency is a common nutritional deficiency. It is generally seen in vegetarians. It may have varied manifestations from asymptomatic to neurological involvement. Vitamin b12 deficiency may occur due to multiple causes varying from nutritional to autoimmune. Vitamin B12 deficiency may present as myelopathy and peripheral neuropathy. Vitamin b12 deficiency leading to sub acute combined degeneration is not common. Sub acute combined degeneration rarely presents with loss of consciousness. We describe such an uncommon case.

Key Words: Vitamin b12; deficiency; SACD; parietal cell

Introduction

Vitamin B12 deficiency is a common ailment in India. Vitamin B12 is an ingredient of mainly animal and dairy products. So, its deficiency is commonly seen among vegetarians. Various manifestations of its deficiency include megaloblastic anaemia, peripheral neuropathy, and sub acute combined degeneration of spinal cord (SACD), gastroenterological and neuropsychiatric symptoms. SACD is a known complication of vitamin B12 deficiency. The other common neurological manifestations of vitamin B12 deficiency include polyneuropathy, but rarely dementia and optic neuropathy [1]. SACD characteristically involves demyelisation of the posterior column and the lateral tracts of the cervical and thoracic spine. The brainstem involvement in SACD occurs rarely. Loss of consciousness is rarely encountered in SACD. We describe such an unusual presentation of vitamin B12 deficiencies with SACD.

Case

A 45 year lady presented to us with complaints of fever for 2 months, numbness in distal part of bilateral lower limb associated with dull back ache for one and a half month, difficulty in walking for 20 days and altered sensorium for 1 day. No history of cough, headache, vomiting, seizures or weakness of any part of body. No history suggestive of any cranial nerve or bladder/bowel involvement. No significant past, personal or family history. Patient took treatment from a private practitioner where she was given injectables for dimorphic anemia.

On examination patient was E3V2M5 maintaining all her vitals? Her body mass index was 22.7 kg/m. [2] General physical examination was unremarkable. CNS examination revealed increased tone with power of 2/5 at all the joints in the bilateral lower limb. Deep tendon reflexes in bilateral knee were exaggerated along with clonus. However, ankle joint reflexes were absent. Babinski sign was positive. Sensory and cerebella functions could not be assessed at presentation as the patient was not fully conscious. Cardiovascular, respiratory and abdominal examination was unremarkable.

Investigation showed anemia with haemoglobin of 8.9 gm%, peripheral smear showing predominantly macrocytic cells and MCV of 114 fl. Rest of routine investigations were normal. The thyroid function tests were normal. Anti nuclear antibody was negative. Serum vitamin B12 levels were 20 ng/ml and folic acid were > 2 ng/ml. Nerve conduction study revealed axonal involvement of bilateral lower limb (motor>sensory) and left median nerve (motor>sensory) in the upper limb.

MRI of cervical spine showed non enhancing T2 hyper intensities in the cord with posterior predilection consistent with sub acute combined degeneration of cord. MRI brain with contrast was a normal study. CSF analysis was non contributory. Patient was managed conservatively along with inject able vitamin b12 and folic acid supplements. Patient showed clinical improvement and became conscious and oriented to time, place and person. After 6 months of follow up, patient became completely normal and regained full power. Her NCV and MRI also showed improvement.

Discussion

Nutritional deficiencies are very common problem in country like India. Vitamin B12 deficiency is one of them. Vitamin B12 deficiency may result from insufficient ingestion, impaired intestinal absorption of vitamin B12 or use of certain drugs. Insufficient ingestion may occur due to insufficient uptake - namely, chronic conditions resulting from alcoholism, anorexia nervosa, or

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an imbalanced diet. Impaired absorption is caused by one of the following: [1] intrinsic factor deficiency due to parietal cell atrophy, gastric carcinoma, or partial or total gastrectomy; [2] antibody production against vitamin B12, intrinsic factor, or parietal cells; [3] malabsorption due to ileitis terminals, colitis ulcerosa, intestinal tuberculosis, resection of the ileum, pancreas insufficiency etc. Drugs such as colchicines, anticonvulsants, metformin, cytotoxic drugs etc can cause vitamin B12 deficiency. The peak incidence of vitamin B12 deficiency occurs in the sixth and seventh decades [2,3,4]. As anemia is a common early symptom leading to the diagnosis of vitamin B12 deficiency, neurological symptoms have often been considered to be late manifestations and typically occur after the development of anemia [5]. Neuropathological studies show the main lesions in SACD occur in the posterior and lateral columns, predominantly in the upper thoracic and midthoracic regions [6]. Brain lesions of vitamin B12 deficiency involving the medulla oblongata, Pons, me encephalon and crus cerebella have also been reported [7].

The diagnosis of B12 deficiency is made by a haematological changes, low serum B12 level, or if the B12 level is borderline, elevated levels of the metabolites homocysteine and methylmalonic acid. Although, more than one quarter of patients with neurologic syndromes will have a normal complete blood count [8].

The myelopathy of vitamin B12 deficiency (or SACD) is characterized neuropathologically by degeneration of myelin and axonal loss [9]. Lesions in SACD are due to overproduction of myelinolytic tumour necrosis factor a (TNF-a) and to the reduced synthesis of the two neurotrophic agents epidermal growth factor (EGF) and interleukin-6. This imbalance between TNF-a and EGF synthesis occurs due to cobalamin deficiency [10]. The changes manifest as high-signal lesions on T2-weighted MRI scans due to increased water content secondary to edema [11,12]. They present as demyelisation, Waller an degeneration, and glossies [12]

The diagnosis of sub acute combined degeneration is based on clinical diagnosis which includes sub acute onset of spastic par paresis and impaired sensation, in addition to history of pernicious anemia or any antecedent cause of vitamin B12 deficiency [13].

If SACD is suspected, early treatment must be initiated with inject able cobalamine to avoid irreversible damage as the condition is reversible depending on the extent of neurological damage of spinal cord [14]. The absence of sensory deficits and Romberg and Babinski signs is associated with a higher complete resolution rate [15].

Our patient treated with injects able cobalamine recovered completely.

References