

Case Report

A Case of Macrocytic Anaemia with Paraplegia due to Tuberculosis

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ABSTRACT

A case of macrocytic anaemia with tuberculosis and paraplegia is very rare. This paper discusses the case of a patient who presented with fever and paraplegia, duly diagnosed as spinal tuberculosis, and showed considerable improvement with anti-tubercular regimen and treatment with vitamin B12.

KEYWORDS: Macrocytic anaemia, Tuberculosis, Paraplegia, Anti-tubercular regimen, Vitamin B12

INTRODUCTION

Vitamin B12 deficiency leads to delayed Deoxyribonucleic acid (DNA) synthesis in rapidly growing hematopoietic cells and can result in macrocytic anaemia. Neurologic abnormalities of vitamin B12 deficiency include paresthesia, sensory deficits, loss of deep tendon reflexes, movement disorders, developmental regression, dementia and neuropsychiatric changes[1]. Magnetic resonance imaging (MRI) has demonstrated brain atrophy and delayed myelination in these cases[2]. Possible causes of vitamin B12 deficiency in childhood include decreased intake, abnormal absorption and defects in vitamin B12 transport and metabolism.

CASE REPORT

A 20-year-old girl presented with complaints of fatigue, inability to walk, urinary incontinence, dysarthria and ataxia that had worsened in the last 3 weeks. She also had fever, weight loss and decreased appetite. In her past medical history, she had been treated for seizures with valproic acid (15 mg/kg/day) for 3 years. Her seizures were documented by Electroencephalogram (EEG) findings. On physical examination, her blood pressure was 110/60 mmHg, pulse rate was 88 beats/

min, respiratory rate was 14 breaths/min and oral temperature was 100.4°F. She reached menarche at the age of 13, followed by regular cycles until 2 months before, when she had irregular and scanty cycles. Her weight was 48 kg and her height was 154 cm. She was vaccinated in childhood. Her family had no significant medical history. She was conscious, had no neck stiffness but confused. The pupils were normal in size and equally reactive to light. The motor system examination revealed upper limb spasticity, muscle strength of 2–3/5 and normal upper limb reflexes and sensory examinations. However, the lower limbs were flaccid and wasted, muscle strength was 0/5, knee and ankle reflexes were absent and the plantar reflex equivocal. Lower limb sensory examination showed lost senses of light touch, pain, temperature, vibration and joint position. As she could not stand or walk, gait and cerebellar examinations were not carried out. Other systemic examinations did not reveal any significant findings. Her haemoglobin level was 8.3 g/dl, but all other blood tests were normal. Her cerebrospinal fluid (CSF) examination and chest X-ray were also normal. Vitamin B12 deficiency was documented as her serum vitamin B12 level was 84 pg/ml (normal 160–970 pg/ml) and peripheral blood smear showed hypersegmented neutrophils. The MRI demonstrated

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senile dilatation in the CSF space and sulci of both hemispheres, a finding compatible with mild atrophic changes.

DISCUSSION

Vitamin B12 deficiency causes neurologic abnormalities, such as paresthesia, movement disorders, developmental regression and neuropsychiatric changes^[1]. Our patient suffered from fatigue, inability to walk, urinary incontinence, dysarthria and ataxia. These manifestations could be attributed to vitamin B12 deficiency. In a study carried out on 50 patients with vitamin B12 deficiency and megaloblastic anaemia, the commonest finding was peripheral neuropathy, but subacute combined degeneration of the cord was uncommon. About a quarter of these patients had either cognitive impairment or an affective disorder, but a third had no detectable nervous system involvement^[3]. The mechanism of neurological effects in cobalamin deficiency is not fully elucidated. Impaired methionine synthesis may lead to depletion of S-adenosylmethionine, which is required for the synthesis of myelin phospholipids. The second hypothesis is that deficit of succinyl-CoA leads to the generation of odd-chained fatty acids, which may get incorporated into the myelin and cause the neurological syndrome of vitamin B12 deficiency^[4]. As in our patient, brain atrophy and delayed myelination can be observed on MRI^[2]. Vitamin B12 deficiency may also cause seizures. The exact mechanism of epileptogenesis in cobalamin deficiency is not clear. It is likely that cerebral neurons with destroyed myelin sheaths are more susceptible to the excitatory effects of glutamate^[5]. Serum vitamin B12 levels should be checked, especially in patients who present with other known neuropsychiatric features of vitamin B12 deficiency. She also suffered from fever, especially at night, weight loss and reduced appetite. Fever is an unusual finding in vitamin B12 deficiency, unless it is accompanied with another disease^[6]. Her fever workup showed acid-fast bacilli in her bone marrow aspiration smear, further confirmed by a positive culture after 48 days. After treatment with anti-tuberculosis drugs, her

fever was terminated, her haemoglobin levels elevated and her general condition improved. This response to therapy was a good indication, confirming the involvement of tuberculosis. In the histological workup for megaloblastic anaemia, macrocytosis, mainly by reducing serum folic acid levels^[7]. Possible causes of vitamin B12 deficiency in childhood include decreased intake, abnormal absorption and defects in vitamin B12 transport and metabolism^[8]. Vitamin B12 deficiency should be considered in patients with neurologic features such as paresthesia, sensory deficits, urinary incontinence, dysarthria and ataxia.

Treatment administered for the above-mentioned conditions resulted in the termination of fever on the second day, further elevation of the haemoglobin level and improvements in her general health. Her upper limb force became normal and she was able to eat by herself. Her lower limb force improved and the plantar reflex was now flexor. To find the cause of megaloblastic anaemia, we reassessed the case. She was not a vegetarian and she had no sign or symptoms of malabsorption or malnutrition. Her growth and development were normal. We concluded that her vitamin B12 deficiency may be of a gastrointestinal origin. As the Schilling test was not available at our centre, it could not be carried out. At present, our patient has been treated for vitamin B12 deficiency and for *Mycobacterium tuberculosis* infection. After being treated with anti-tuberculosis drugs, her fever terminated and her haemoglobin levels returned to normal. At present, 10 months after her admission to the hospital, she can eat, write and speak normally, as well as walk and ride a bicycle, but she still has some degree of foot drop. Her latest tests showed a haemoglobin level of 13.7 g/dl, Haematocrit value of 39.4 and her Mean Corpuscular Volume (MCV) was 89.3 fl.

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