Spinal MR imaging in Vitamin B12 deficiency: Case series; differential diagnosis of symmetrical posterior spinal cord lesions

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Abstract

We report three cases of Vitamin B12 deficiency with symmetrical posterior spinal cord lesions and discuss the differential diagnosis, some of which are not well known. Because the degree of resolution of the clinical symptoms in subacute combined degeneration depends on early detection, MRI findings should not be missed.

Key Words

Spinal MRI, symmetrical posterior spinal cord lesions, vitamin B12 deficiency

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Introduction

Vitamin B12 deficiency may present with dorsal spinal column involvement, clinically and on magnetic resonance imaging (MRI). MRI finding of symmetrical posterior spinal cord lesions have many differential diagnoses, some of which are not well known. Because the degree of resolution of the clinical symptoms in B12 deficiency depends on early detection, MR findings should not be missed.

Case Report

We report three cases of Vitamin B12 deficiency:

Patient 1, a non-vegetarian male, presented with symptoms of dorsal cord involvement and had a definite sensory level, below which sensations were reduced. MRI showed T2 hyperintensities involving the lateral [Figure 1a and b] and posterior [Figure 1b] columns of the spinal cord. The serum B12 level (68 pg/ml) was found to be low.

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Patient 2, a strict vegetarian male, presented with paresthesias involving the lower limbs more than the upper limbs and positive Romberg's sign. MRI showed posterior column [Figure 2a and b] involvement. B12 level (226 pg/mL) tested at referral centre was normal, possibly due to stat B12 injection administered at the peripheral centre after presumptive diagnosis.

Patient 3, a vegetarian elderly male, had come for evaluation of degenerative spine with no specific neurologic complaints. MRI finding of posterior column [Figure 3a and b] hyperintensity was almost incidental, but serum B12 level (96 pg/mL) was low and he had vitiligo on examination.

Discussion

B12 deficiency in the Western world is rarely caused by an inadequate intake, which is most often seen in strict vegetarians. There, more commonly, B12 deficiency is the result of malabsorption syndromes such as bacterial overgrowth of the small bowel, pernicious anaemia, regional enteritis, tropical sprue or surgical procedures like gastric fundal or ileal resection.[1] Pernicious anaemia, the most common cause of B12 malabsorption in the United States, leads to achlorhydria, atrophic gastritis and decreased intrinsic factor; patients present clinically around 60 years and often have other associated autoimmune disorders like Graves' disease.[1]

Dietary Vitamin B12 deficiency has been shown to be a severe problem in the Indian subcontinent,[2-7] Mexico, Central and

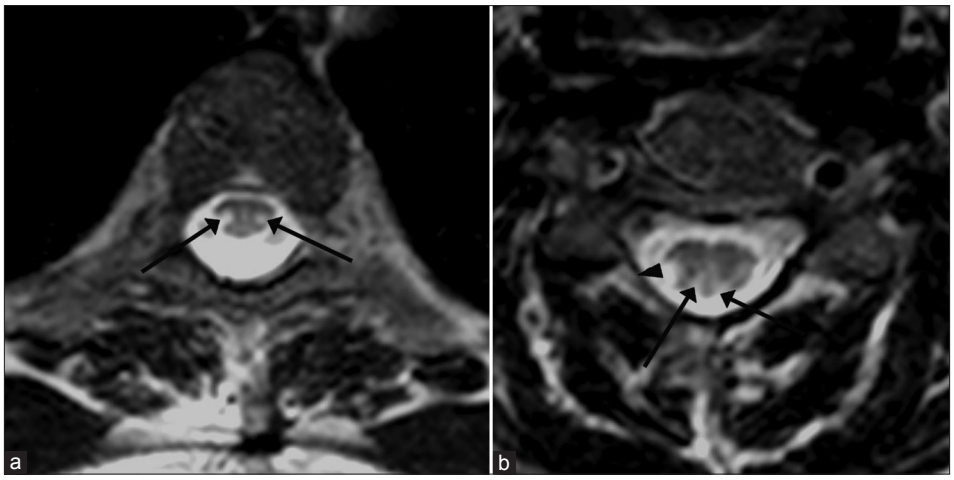

Figure 1: (a) Lateral column hyperintensity (arrows), (b) posterior column (arrows) and lateral column (arrowhead) hyperintensities

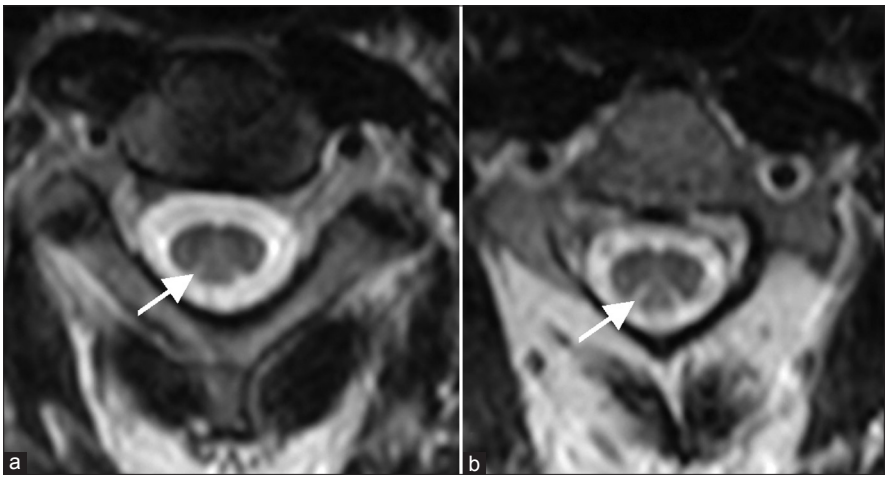

Figure 2: Posterior column hyperintensity seen faintly (a) and clearly (b)

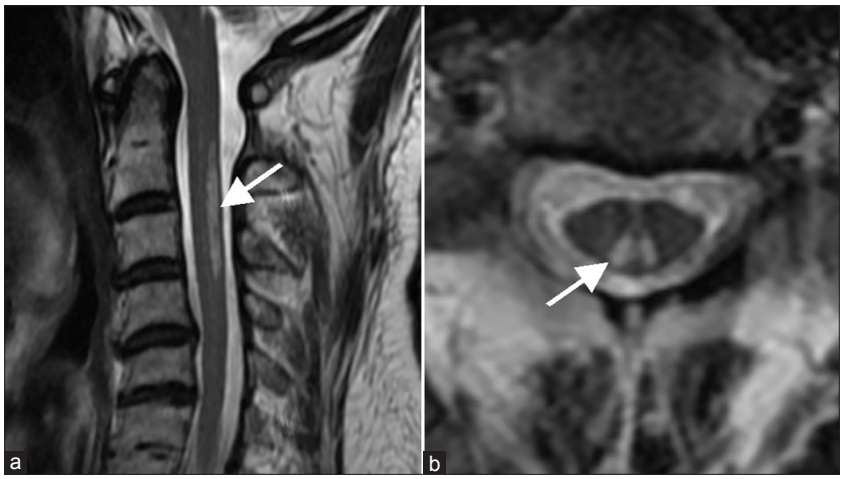

Figure 3: Posterior column hyperintensity on sagittal (a) and axial (b) sections

South America and selected areas in Africa.[8] It is also seen in immigrant populations[9,10] in the Western world, probably due to vegetarian diet. Among our three patients, one was a non-vegetarian, one was a strict vegetarian and another was a vegetarian with vitiligo suggestive of pernicious anaemia.

Pathology is demyelination involving the dorsal columns, predominantly in the lower cervical and upper thoracic regions; it eventually involves the entire dorsal columns symmetrically,

with spread in the cranial and caudal directions and into the lateral columns.

Nitrous oxide irreversibly oxidizes active Vitamin B12 cobalamin to inactive cobalamin. As there is no reserve of cobalamin in patients with Vitamin B12 deficiency, nitrous oxide may bring about manifestations of Vitamin B12 deficiency in asymptomatic patients.[11,12]

Vitamin B12 deficiency may manifest with neurological features or megaloblastic anemia. Signs of dorsal column involvement (loss of position and vibration sense and ataxia), lateral column involvement (spasticity, hyperreflexia and positive Babinski sign) and spinothalamic tracts involvement (sensory level) should be looked for.

The diagnosis of B12 deficiency is made by a low serum B12 level or (if the B12 level is borderline) elevated levels of the metabolites homocysteine and methylmalonic acid. Haematological changes like megaloblastic anaemia are not reliable markers. Pernicious anaemia can be confirmed by positive findings on the Schilling test or by the presence of anti-intrinsic factor or antiparietal cell antibodies. Pernicious anaemia patients have a two-fold increased risk for gastric polyps and cancer, and additional examination may be required.

MRI Findings

On sagittal images, a vertical segment can be seen at the posterior aspect of the spinal cord. On axial images, bilateral paired areas of T2 hyperintensity are seen as an "inverted V" or "inverted rabbit ears" in the dorsal columns. Lateral column involvement[13] is seen in severe cases. Contrast enhancement is uncommon and if present, mild. After treatment for Vitamin B12 deficiency, there is interval improvement of signal abnormality. MRI of the brain may show confluent areas of abnormal signal intensity on T2-weighted images in the cerebral white matter; resolution of these changes is often seen within a few months of starting B12 therapy.

Differential Diagnosis on MRI

1. Acquired immunodeficiency syndrome (AIDS) presents with clinical symptoms, pathology[14-16] and MRI findings[15-17] similar to those of B12 deficiency. Bilaterally symmetrical continuous T2 and PD hyperintensity affecting white matter tracts (primarily gracile tract, followed by corticospinal and cuneate tracts) has been described. Other spinal cord lesions in HIV are accompanied by expansion of the cord and enhancement.
2. Copper deficiency has been reported to cause hyperintensity involving posterior columns of the cervical cord, with resolution on copper supplementation.[18,19] T2 hyperintensity may also involve both dorsal and central spinal cord, or the central spinal cord exclusively. No spinal cord contrast enhancement has been reported.
   Copper deficiency may present as sensory ataxia, anaemia and myelodysplastic syndrome. It has been attributed to excess zinc ingestion, malabsorption, gastric bypass surgery, nephrotic syndrome, parenteral nutrition, prematurity and malnourishment (in infants), or can be idiopathic. Menkes disease, an X-linked disorder of copper metabolism that develops in infancy, may show spinal cord demyelination. Serum copper and ceruloplasmin levels are usually markedly decreased.
3. Friedreich's ataxia shows thinning and intramedullary signal changes in the cervical portion of the spinal cord, involving posterior and lateral white matter tracts.[20] The most common hereditary ataxia, it is autosomal recessive and presents before 20–30 years of age.
4. Leukoencephalopathy with brain stem and spinal cord involvement and lactate elevation (LBSL) shows T2 hyperintensities in the dorsal and lateral columns of the spinal cord. A rare autosomal-recessive disorder with gradual onset usually in childhood or adolescence (occasionally in adulthood), it involves brainstem (pyramids in medulla) and the entire spinal cord,[21] as opposed to predominant involvement of the lower cervical and upper thoracic spinal cord in B12 deficiency.
5. Adult-onset autosomal-dominant leukodystrophy (ADLD) with autonomic symptoms has been reported to show dorsal column T2 hyperintensities.[22] A rare leukodystrophy seen in the American–Irish population with onset of symptoms in the fourth to sixth decades, it is associated with thinning of spinal cord and hyperintensities in the cerebral parenchyma, corpus callosum and cerebellar peduncles.
6. Vitamin E deficiency has been reported to cause symmetrical posterior column T2 hyperintensities.[23] In that patient, normalization of serum Vitamin E levels did not reverse MRI changes.
7. Multiple sclerosis plaques in dorsal column are not bilaterally symmetrical and longitudinal extent is less than two vertebral bodies. They may show contrast enhancement, are seen in a younger population and are associated with different clinical and laboratory findings.

Prognosis

B12 deficiency is treated with B12 intramuscular injections, continued monthly for life. Because the degree of resolution of the clinical symptoms is inversely proportional to their duration and severity, early detection is necessary for full clinical cure. Although not specific for B12 deficiency, MR findings have distinguishing features and should not be missed.

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