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

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.

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, peptic ulcer, regional enteritis, tropical sprue or surgical procedures like gastric fundal or ileal resection. 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.

Dietary Vitamin B12 deficiency has been shown to be a severe problem in the Indian subcontinent, Mexico, Central and
It is also seen in immigrant populations 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.
Copper deficiency may present as sensory ataxia, anaemia. 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.\(^{[1],[2]}\)

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 and enhancement, are seen in a younger population and are associated with different clinical and laboratory findings.

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.

**References**

1. Fauci AS, Braunwald E. Harrison’s principles of internal medicine. 14th ed. New York: McGraw-Hill; 1998. p. 653-8.

2. Mehta BM, Rege DV, Satoskar RS. Serum vitamin B12 and folic acid activity in lactovegetarian and nonvegetarian healthy adult Indians. Am J Clin Nutr 1964;15:77-84.[Abstract]

3. Dhopeshwarkar GA, Trivedi JC, Kulkarni BS, Satoskar RS, Lewis RA. The effect of vegetarianism and antibiotics upon acid activity in lactovegetarian and nonvegetarian healthy adult Indians. Am J Clin Nutr 1964;15:77-84.[Abstract]

4. Banerjee DK, Chatterjea JB. Serum vitamin B12 in vegetarians. Br J Med 1960;2:992-4.

5. Jathar VS, Patrawalla SP, Doongaji DR, Rege DV, Satoskar RS. Serum vitamin B 12 levels in Indian psychiatric patients. Br J Psychiatry 1970;117:699-704.[Abstract/Free Full Text]

6. Jathar VS, Inamdar-Deshmukh AB, Rege DV, Satoskar RS. Vitamin B12 and vegetarianism in India. Acta Haematol 1975;53:90-7.[Medline]
7. Kumar J, Garg G, Sundaramoorthy E, Prasad PV, Karthikeyan G, Ramakrishnan L, et al. Vitamin B12 deficiency is associated with coronary artery disease in an Indian population. Clin Chem Lab Med 2009;47:334-8.
8. Stabler SP, Allen RH. Vitamin b12 deficiency as a worldwide problem. Annu Rev Nutr 2004;24:299-326.
9. Bindra GS, Gibson RS. Vitamin B12 and folate status of East Indian immigrants living in Canada. Nutr Res 1987;7:365–74.
10. Gupta AK, Damji A, Uppaluri A. Vitamin B12 deficiency. Prevalence among South Asians at a Toronto clinic. Can Fam Physician 2004; 50:743-7.
11. Holloway KL, Alberico AM. Postoperative myeloneuropathy: a preventable complication in patients with B12 deficiency. J Neurosurg 1990;72:732-6.
12. Hadzic A, Glab K, Sanvorn KV, Thys DM. Severe neurologic deficit after nitrous oxide anesthesia. Anesthesiology 1995;83:863-6.
13. Locatelli ER, Laureno R, Ballard P, Mark AS. MRI in vitamin B12 deficiency myelopathy. Can J Neurol Sci 1999;26:60-3
14. Petito C, Navia B, Cho E, Jordan B, George D, Price R. Vacuolar myelopathy pathologically resembling subacute combined degeneration in patients with the acquired immunodeficiency syndrome. N Engl J Med 1985;312:874-9.
15. Sartoretti-Schefer S, Blattler T, Wichmann W. Spinal MRI in vacuolar myelopathy, and correlation with histopathological findings. Neuroradiology 1997;39:865-9.
16. Santosh CG, Bell JE, Best JJ. Spinal tract pathology in AIDS: postmortem MRI correlation with neuropathology. Neuroradiology 1995;37:134-8.
17. Shimojima Y, Yazaki M, Kaneko K, Fukushima K, Morita H, Hashimoto T, et al. Characteristic Spinal MRI Findings of HIV-associated Myelopathy in an AIDS Patient. Intern Med 2005;44:763-4.
18. Goodman BP, Chong BW, Patel AC, Fletcher GP, Smith BE. Copper deficiency myeloneuropathy resembling B12 deficiency: Partial resolution of MR Imaging findings with copper supplementation. AJNR Am J Neuroradiol 2006;27:2112-4.
19. Goodman BP, Bosch EP, Ross MA, Hoffman-Snyder C, Dodick DD, Smith BE. Clinical and electrodiagnostic findings in copper deficiency myeloneuropathy. J Neurol Neurosurg Psychiatry 2009;80:524-7.
20. Mascalchi M, Salvi F, Piacentini S, Bartolozzi C. Friedreich's ataxia: MR findings involving the cervical portion of the spinal cord. AJNR Am J Roentgenol 1994;163:187-91.
21. Van der Knaap MS, van der Voorn P, Barkhof F, et al. A new leukoencephalopathy with brainstem and spinal cord involvement and high lactate. Ann Neurol 2003;53:252-8.
22. Sundblom J, Melberg A, Kalimo H, Smits A, Raininko R. MR imaging characteristics and neuropathology of the spinal cord in adult-onset autosomal dominant leukodystrophy with autonomic symptoms. AJNR Am J Neuroradiol 2009;30:328-35.
23. Vorgerd M, Tegenthoff M, Malin JP, Kühne D. Spinal MRI in progressive myeloneuropathy associated with vitamin E deficiency. Neuroradiology 1996;38:(Suppl 1):S111-3.

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