

Subacute Combined Degeneration Of The Cord: Lateral Column Involvement Seen On Mri –An Uncommon Finding

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Citation

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Abstract

CASE DISCUSSION

A 46 year old man, a vegetarian, presented with acute onset of paresthesia (“pins and needles” sensation) involving both hands and feet of 15 days duration. He also complained of difficulty in walking and inability to feel the ground for the same period. There was no history of trauma, fever, vomiting, diarrhea, upper respiratory tract infection, visual disturbances, and bladder or bowel incontinence. On examination, the patient was restless and agitated but his higher mental functions were normal. There was impairment of sensation of fine touch, pin prick, joint position and vibration in both hands and feet bilaterally. All the deep tendon reflexes were exaggerated and symmetric, more so in the lower limbs; Plantars were extensor. Romberg's sign was positive. There was no evidence of motor weakness. His cranial nerves were normal. Spine was normal. Vision was normal. Other systemic examination did not reveal any abnormality.

The initial laboratory investigations revealed dimorphic anemia which was confirmed on bone marrow biopsy. CSF examination did not show any abnormality. Visual evoked potential and brain stem evoked potential studies were within normal limits. Gastric endoscopy and biopsy revealed changes of atrophic gastritis. Folic acid and vitamin B12 levels in the serum were 7 micrograms and 75 picograms respectively (normal range being 200 to 600 picograms /ml). Magnetic resonance imaging (MRI) of the spine showed intramedullary hyperintensity seen on T2-weighted images in the posterior column of the cervical spinal cord (fig-1). The image findings were consistent with earlier reports of SCD₁₂₃₄₅. The visualized upper dorsal cord was normal. The axial images revealed involvement of posterior and lateral columns bilaterally (Fig-2). There was no enhancement of the lesion on contrast studies. The image findings were

consistent with SCD. The patient was treated with parenteral administration of vitamin B12 and oral folic acid. At 10 months follow-up, there was significant symptomatic improvement.

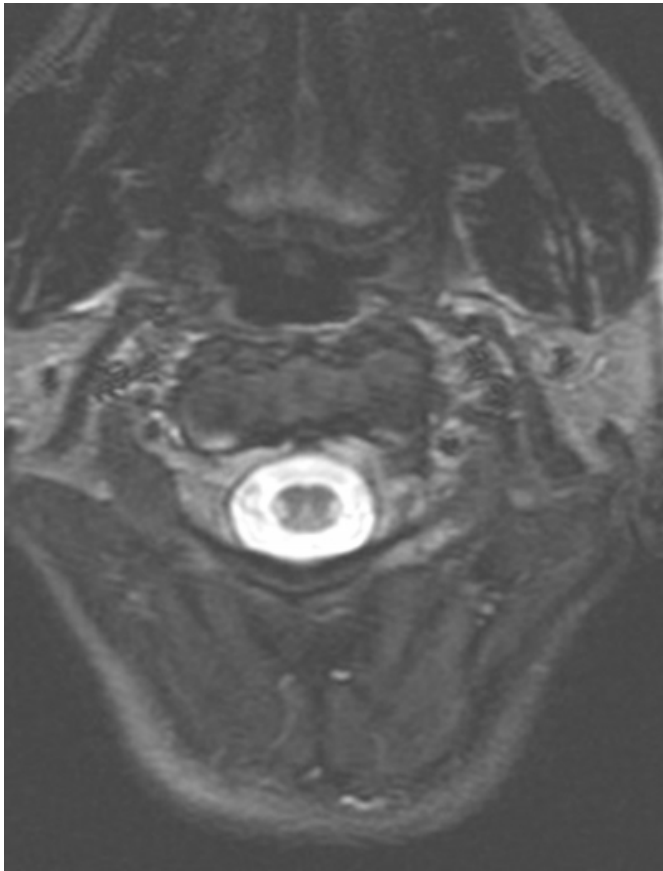
Figure 1

Figure 1: T2 weighted sagittal image of the cervical spine reveals intramedullary hyperintensity in the cervical cord posteriorly.



Figure 2

Figure 2: Axial T2 Fat Sat Image of the cervical spine reveals intramedullary hyperintensity in the cervical cord involving the posterior and the lateral columns.



DISCUSSION

Cobalamin (Vitamin B 12) is synthesized solely by microorganisms. The only source of cobalamin in humans is food of animal origin e.g., meat, fish and dairy products. Vegetables, fruits and other foods of non- animal origin are free from cobalamin unless they are contaminated by bacteria. Adult daily losses (mainly in the urine and feces) are between 1 and 3 µg (~0.1% of body stores) and, as the body does not have the ability to degrade cobalamin, daily requirements are also about 1 and 3 µg. Body stores are 2-3 mg, sufficient for 3-4 years if supplies are completely cut off.

The normal physiologic mechanism for the cobalamin absorption is active, occurs through the ileum and is mediated by the gastric intrinsic factor.

Cobalamin exists in a number of different chemical forms. The two major natural forms are (a) 2-deoxyadenosyl form (adococobalamin), which is located in the mitochondria; it is

the co-factor for the enzyme methylmalonyl CoA mutase, and (b) methylcobalamin, the form in human plasma and in cell cytoplasm; it is the co-factor for methionine synthase. Only two reactions in the body are known to require cobalamin. Methylmalonyl isomerization, which requires adococobalamin, and the methylation of homocysteine to methionine which requires both methylcobalamin and 5-Methyl tetrahydrofolate (5-MTHF).

Pernicious anemia, an antibody mediated intrinsic factor disorder, is the most common cause for vitamin B12 deficiency in western countries. However, in India, alcoholism, malnutrition and ileocecal tuberculosis are the common causes.

The most common manifestations of cobalamin deficiency are hematologic, gastrointestinal and neurologic. Cobalamin deficiency may cause a bilateral peripheral neuropathy or a progressive demyelination, which starts in the posterior columns and spreads anteriorly involving the lateral and anterior columns; the process begins in the lower cervical and upper thoracic cord, spreading up and down the cord as well. The same neuropathological pattern can involve the optic nerve and rarely the white matter of the brain.

The pathogenesis of neurological complication of vitamin B12 deficiency remains obscure. Deficiency state results in decreased activity of cobalamin-dependent methylmalonyl CoA mutase enzyme with resultant elevated levels of methylmalonic acid, which is toxic to myelin, causing destruction of both myelin sheaths and axons in the white matter of spinal cord₆. Based on animal studies, Scott et al suggested that inability to resynthesise methionine from homocysteine leads to SCD₇. In another study conducted to determine the biochemical pathogenesis of subacute combined degeneration of the spinal cord and brain it was suggested that deficiency of S-adenosylmethionine, synthesized from methionine, is critical to the development of demyelination in cobalamin deficiency₈.

Clinical features and MRI findings similar to SCD have been described in nitrous oxide toxicity₉. All these patients had undergone surgical procedures for various reasons with nitrous oxide as the anesthetic agent. Short term exposure to nitrous oxide in healthy patients seems to have no appreciable sequelae. The administration of nitrous oxide anaesthesia in patients with unsuspected vitamin B12 deficiency can induce neurological changes₁₀. It has been suggested that patients be screened and treated for cobalamin

deficiency before exposing them to nitrous oxide anaesthesia¹¹. Nitrous oxide irreversibly oxidizes methylcobalamin, the active form of vitamin B12, to an inactive precursor; this inactivates methionine synthase. Methylmalonic aciduria does not occur as adocobalamin is not inactivated by nitrous oxide. A neuropathy resembling cobalamin neuropathy has been described in dentists and anaesthetists who are repeatedly exposed to nitrous oxide.

Our patient had routine T1WI and T2WI in axial and sagittal planes. The T2WI demonstrated diffuse hyperintensity in the cervical cord involving the posterior column. Lateral column involvement is usually not demonstrated on MRI though clinically very well appreciated¹². In this case the involvement of lateral column is well demonstrated on MRI, as seen in the figure-2.

CONCLUSION

SCD is a known complication of vitamin B12 deficiency, which usually is reversible, if diagnosed and treated early. MRI demonstrated the involvement of the posterior and lateral columns of the cervical cord in our patient. Parenteral administration of vitamin B 12 produced significant symptomatic improvement. Demonstration of lateral column involvement on MRI as seen in this case is extremely rare and has been very infrequently reported in the literature.

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