CASE REPORT

Early treatment causes clinicoradiological reversal of myelopathy due to vitamin B₁₂ deficiency

Rajesh Verma,¹ Prakash Kori,² Tushar B Patil,¹ Heramba Narayan Praharaj¹

¹Department of Neurology,
King George Medical
University, Lucknow,
Uttar Pradesh, India
²King George Medical
University, Lucknow,
Uttar Pradesh, India

Correspondence to
Professor Rajesh Verma,
drrajeshverma32@yahoo.com

SUMMARY

Vitamin B₁₂ deficiency has a wide spectrum of clinical presentation with a variety of neurological symptoms and signs. As a result, many patients lack classic features of advanced severe deficiency. Early diagnosis and treatment are crucial in order to prevent the irreversible damage to the nervous system. We describe a 25-year-old man, who presented with predominant sensory symptoms without any signs on clinical evaluation. His serum vitamin B₁₂ levels were low and neuroimaging revealed myelopathy. The patient was treated promptly with cyanocobalamin injections, which lead to a rapid resolution of symptoms and radiological abnormalities.

BACKGROUND

Vitamin B₁₂ is an essential vitamin needed by the haematopoietic cells, epithelia and nervous tissue for maturation and growth. The deficiency of vitamin B₁₂, either due to malabsorption or dietary deficiency, results in various haematological, gastrointestinal, neurological and psychiatric manifestations. The neurological presentations include myelopathy, peripheral neuropathy, optic nerve atrophy and behavioural abnormality. Myelopathy, presenting as subacute combined degeneration (SCD) of the spinal cord, is the most frequent neurological syndrome of vitamin B₁₂ deficiency. The cord changes are initially reversible, and as the vitamin B₁₂ deficiency goes untreated for a longer time, the changes become more and more irreversible and the responsiveness to treatment decreases.

To highlight this fact, we present here a young patient who developed cervical myelopathy due to vitamin B₁₂ deficiency, manifesting as sensory complaints, with signal changes on MRI of cervical spinal cord. He received an early treatment with vitamin B₁₂, which resulted in a complete resolution of clinical symptoms as well as the neuroimaging changes.

CASE PRESENTATION

A 25-year-old man presented to our outpatient clinic with a 1-month history of a tingling sensation in both hands and which had spread up to both elbows over this duration. However, he did not complain of any tingling sensation or loss of sensation in his feet.

He did not have weakness in any of the limbs. But, on detailed questioning, he said that he had difficulty in walking in dark. He had no current or past exposure to heavy metals, toxins or drugs.

He had no history of high risk sexual behaviour. He was a strict vegetarian.

Clinical examination revealed a young, well-nourished man with mild pallor, without icterus or skin pigmentation. Liver and spleen were not palpable. On neurological examination, he was alert with intact higher mental functions. An examination of the cranial nerves, muscle strength, gait, tone and coordination did not reveal any abnormality. Deep tendon reflexes were hyperactive in all limbs with bilateral flexor plantar response. Sensory examination for pain, touch and temperature was normal. However, Romberg's sign was positive.

INVESTIGATIONS

Haematological investigations demonstrated a haemoglobin level of 8.2 g/dl, mean corpuscular volume (MCV) of 102 fl (normal range: 80–96 fl) and erythrocyte count of 3.2 million/ μ l. Peripheral blood smear revealed anisocytosis with a few megakaryocytes, normal leucocyte count and adequate platelets. Serum vitamin B₁₂ level was 34 pg/l (range 250–700 pg/l). Serum folate levels were normal. Microscopic and biochemical analyses of cerebrospinal fluid were normal.

On the basis of clinical presentation of upper limb sensory symptoms, positive Romberg's test, hyperactive deep tendon reflexes and laboratory features of megaloblastic anaemia due to vitamin B₁₂ deficiency, an initial diagnosis of myelopathy due to vitamin B₁₂ deficiency was made and the patient was subjected to an MRI of spine, which revealed an increased T2-weighted signal in the posterior columns of cervical spinal cord, extending from C2 to C5 level, without any gadolinium enhancement on T1-weighted contrast images (figure 1). Thoracic and lumbar spinal cord MRI was normal.

DIFFERENTIAL DIAGNOSIS

Treatment

In view of these clinical, haematological and radiological findings, the diagnosis of subacute combined degeneration (SCD) of cervical spinal cord due to vitamin B₁₂ was confirmed. The patient was started on replacement therapy with intramuscular vitamin B₁₂ 1000 μ g/day for 7 days, followed by maintenance dose of 1000 μ g/month. He also received iron and folic acid supplementation.

OUTCOME AND FOLLOW-UP

The symptoms totally disappeared 1 month after parenteral supplementation of vitamin B₁₂. Repeat MRI was performed 2 months later, which showed

To cite: Verma R, Kori P, Patil TB, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-009090

Figure 1 T2-weighted sagittal MRI of cervical spinal cord showing diffuse hyperintense signals in posterior cervical spinal cord.

the complete disappearance of signal changes in the cervical spinal cord (figure 2).

DISCUSSION

Vitamin B₁₂ deficiency usually presents with brain, spinal cord, optic nerve and peripheral nervous system manifestations. The most serious damage is demonstrated by the involvement of posterior and lateral columns of cervical and upper dorsal parts of spinal cord. This results due to the accumulation of methylmalonyl co A with the incorporation of abnormal fatty acids into neuronal lipids, which constitute the myelin sheath. This leads to the involvement of spinal cord, which is called as SCD.¹ Neuropathological studies show spongiform changes, with foci of myelin and axonal destruction not only in posterior and lateral columns but also involving anterior columns in advanced cases.²

Cases of complete resolution of clinical manifestations and radiological abnormalities indicate that early treatment can lead to the reversal of pathological changes. Paraesthesias, which

occurs due to peripheral nerve involvement or reversible impairment of central sensory conduction, usually resolves within weeks after treatment. However, objective signs of spinal cord dysfunction, including weakness, spasticity and proprioceptive deficits, may require months to improve.³ With early recognition and treatment, patients may resume a normal lifestyle with limited impairment of gait. Relapses may occur because of non-compliance with long-term supplementation. Our patient had only sensory symptoms without any sensory or motor signs, suggesting an early presentation of vitamin B₁₂ deficiency. Therefore, he had a complete clinical resolution on vitamin B₁₂ supplementation.

A review of neuroimaging features in patients with vitamin B₁₂ deficiency myelopathy revealed variable MRI abnormalities.⁴ The most common finding was the increased T2-weighted signal in the posterior columns of the cervical and/or thoracic spinal cord. Swelling of the cervical cord on T1-weighted imaging,⁵⁻⁶ enhancement of posterior columns and lateral columns on postcontrast T1-weighted Imaging⁷ and increased signal on T2-weighted imaging in the posterior columns with variable involvement of the lateral and anterior columns have also been described in vitamin B₁₂ deficiency. Differential diagnoses of abnormal signal lesions in the posterior columns of the spinal cord include infectious or postinfectious myelitis, peripheral neuropathy, lymphoma and other neoplasms, paraneoplastic myelopathy, cervical spondylosis, radiation myelitis, multiple sclerosis, sarcoidosis, arterial or venous ischaemia, traumatic cord injury, arterial or venous ischaemia, vascular malformations of the dura and spinal cord, syringomyelia and acute transverse myelitis.⁸ Treatment with hydroxycobalamin or cyanocobalamin delays disease progression, and to some degree of reversal of symptoms, with complete recovery in almost half of the patients. Treatment included intramuscular 1000 µg/day cyanocobalamin injections.

Vitamin B₁₂ is administered daily in the first 7–14 days, weekly in the subsequent month and monthly afterwards to replenish the stores of cyanocobalamin in the body.⁹ Although MRI findings in these diseases are non-specific, the findings of increased T2-signal intensity in the cervical cord, in conjunction with the clinical examination findings and laboratory testing, can help in the diagnosis of vitamin B₁₂ deficiency myelopathy. In addition, MRI can be used in conjunction with clinical and laboratory testing to assess response to treatment.

Figure 2 Follow-up T2-weighted MRI after treatment showing complete resolution of hyperintense signals in cervical spinal cord.

Learning points

- ▶ Subacute combined degeneration is clinically characterised by the predominant involvement of the posterior column.
- ▶ In some patients, MRI shows abnormalities of the spinal cord, indicating demyelination of the posterior column.
- ▶ Early diagnosis and treatment play an important role in the reversibility of neurological deficits; delayed treatment results in irreversible disabling neurological impairment.

Contributors RV conceptualised the idea of writing the case report and PK, TBP and HNP helped in drafting the manuscript.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Yamada K, Shrier DA, Tanaka H, *et al.* A case of subacute combined degeneration: MRI findings. *Neuroradiology* 1998;40:398–400.
- 2 Duchen LW, Jacobs JM. Nutritional deficiencies and metabolic disorders, vitamin B12. In: Adams JH, Duchen LW, eds. *Greenfield's neuropathology*. 5th edn. London: Edward Arnold, 1992:826–32.
- 3 Savage DG, Lindenbaum J. Neurological complications of acquired cobalamin deficiency: clinical aspects. *Baillieres Clin Haematol* 1995;8:657–78.
- 4 Locatelli ER, Laureno R, Ballard P, *et al.* MRI in vitamin B12 deficiency myelopathy. *Can J Neurol Sci* 1999;26:60–3.
- 5 Larner AJ, Zeman AZ, Allen CM, *et al.* MRI appearances in subacute combined degeneration of the spinal cord due to vitamin B12 deficiency. *J Neurol Neurosurg Psychiatry* 1997;62:99–100.
- 6 Ng VW, Gross M, Clifton A. Case report: MR appearances in vitamin B12 neuropathy. *Clin Radiol* 1997;52:394–6.
- 7 Kuker W, Hesselmann V, Thron A, *et al.* MRI demonstration of reversible impairment of the blood-CNS barrier function in subacute combined degeneration of the spinal cord. *J Neurol Neurosurg Psychiatry* 1997;62:298–9.
- 8 Vorgeer M, Tegenhoff M, Kuhne D, *et al.* Spinal MRI in progressive myeloneuropathy associated with vitamin E deficiency. *Neuroradiology* 1996;38(Suppl 1):111–13.
- 9 Bolaman Z, Kadikoylu G, Yukselen V, *et al.* Oral versus intramuscular cobalamin treatment in megaloblastic anemia: a single-center, prospective, randomized, open-label study. *Clin Ther* 2003;25:3124–34.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow