Three-point sign in subacute combined degeneration of the spinal cord
A case report

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Abstract

Rationale: Subacute combined degeneration (SCD) of the spinal cord has been reported to have distinctive characteristics on magnetic resonance imaging (MRI), such as an “inverted V sign”, a “pair of binoculars sign”, and a “dot sign”. We report a 3-point sign as a novel MRI characteristic, expanding the spectrum of imaging signs for SCD.

Patient concerns: A 64-year-old female vegetarian presented with a 3-month history of progressive numbness and weakness in the lower extremities.

Diagnosis: Laboratory examination showed a reduced serum vitamin B12 level. Spinal MRI showed hyperintensity within the posterior and lateral columns (appearing as a three-point sign) on T2-weighted imaging. Thus, the patient was diagnosed with SCD.

Interventions: The patient was treated with intravenous cyanocobalamin and oral vitamin B12.

Outcomes: After a follow-up period of 5 months, the symptoms were significantly improved.

Lessons: Clinicians should be aware of this atypical MRI pattern, which may facilitate an early diagnosis.

Abbreviations: MRI = magnetic resonance imaging, SCD = subacute combined degeneration.

Keywords: case report, magnetic resonance imaging, subacute combined degeneration, 3-point sign

1. Introduction

Subacute combined degeneration (SCD) of the spinal cord refers to a degenerative disease of the central and peripheral nervous system that is caused by vitamin B12 deficiency. SCD predominantly involves the white matter of the spinal cord (posterior and lateral columns) and peripheral nerves. Clinically, SCD manifests as paresthesia, ataxia, spastic paraplegia, and peripheral neuropathy, frequently accompanied by anemia. On magnetic resonance imaging (MRI), only 11.1% to 36.7% patients have shown remarkable abnormalities. The previously identified distinctive characteristics included an “inverted V sign”, a “pair of binoculars sign,” and a “dot sign.” Herein, we report a novel MRI pattern, the 3-point sign, in a case of SCD. The Ethics Committee of the First Hospital of Jilin University approved the study. The patient has provided informed consent for publication of the case.

2. Case report

A 64-year-old female vegetarian presented to us with a 3-month history of progressive numbness and weakness in the lower extremities. In the local hospital, a diagnosis of cerebral infarction was suspected, and antiplatelet and neurotrophic medications were prescribed but provided no benefit. Twenty days after the onset, the symptoms became aggravated, and the patient developed abasia and sphincter disturbance. Her previous medical history and family history were unremarkable. Notably, she had lost 5 kg over the past half year. After admission, physical examination revealed a loss of sensation below the cervical (C)3 dermatome and grade 2/5 weakness of bilateral extremities. Additionally, tendon hyperreflexia, positive Babinski sign, and ankle clonus were noted. Laboratory examination showed a reduced red blood cell count (2.23 × 10^{12}/L, normal range, 3.8 × 10^{12}/L – 5.1 × 10^{12}/L), a reduced hemoglobin level (95 g/L, normal range, 115–150 g/L), an elevated mean corpuscular volume (MCV; 120.2 fl, normal range, 82–100 fl), and a significantly reduced serum vitamin B12 level (9.94 pmol/L, normal range, 133–675 pmol/L). Bone marrow puncture showed an elevated myeloid-to-erythroid ratio and megaloblasts, indicating active hyperplasia. Electromyography as well as auditory- and visual-evoked potentials showed no abnormality. Spinal MRI demonstrated hyperintensity within the posterior and lateral columns (appearing as a 3-point sign) on T2-weighted imaging (Fig. 1). These findings suggested vitamin B12 deficiency and anemia, leading to a diagnosis of SCD.

The patient was treated with an intravenous drip of cyanocobalamin at a dosage of 0.5 mg/day for 14 days followed by oral vitamin B12 (0.5 mg, 3 times daily). Nutrition guidance and rehabilitation treatment were scheduled. After a follow-up period of 5 months, laboratory examination showed a normal serum vitamin B12 level. Also, the sensorimotor symptoms were
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sign,

thoracic segments are most commonly affected.[10,11] SCD often

due to the involvement of both the posterior and lateral columns of the spinal cord, which could

result in vitamin B12 deficiency. In the current case, the

patient was a vegetarian who ate very little meat and only

weighed 43kg. On MRI, abnormal hyperintensity was noted in

the posterior and lateral columns of the spinal cord, which could explain the sensorimotor disturbances in bilateral extremities. Further laboratory examination revealed an extremely low level of serum vitamin B12. These findings strongly suggested SCD induced by vitamin B12 deficiency. Following vitamin B12 replacement therapy, the patient’s symptoms were significantly relieved, confirming the diagnosis of SCD.

The differential diagnoses in this patient should include acquired copper deficiency myelopathy, multiple sclerosis, neuromyelitis optica, and spinal tumors. MRI is the mainstream neuroimaging modality for the differential diagnosis of SCD. Although the majority of patients with SCD may show no abnormality on MRI, visible signal abnormalities in the remaining patients are usually distinctive. SCD predominantly involves the posterior and/or lateral columns of the spinal cord, and the lower cervical and thoracic segments are most commonly affected.[10,11] SCD often appears hyperintense on T2-weighted imaging but normal on T1-weighted imaging, with unremarkable or slight enhancement. In the previous studies, the hyperintensity in the dorsal columns formed an “inverted V sign,” a “pair of binoculars sign,” or a “dot sign” on axial T2-weighted imaging.[3-6] The present case showed distinct characteristics on spinal MRI, appearing as a “3-point sign,” due to the involvement of both the posterior and lateral columns of the spinal cord.

In conclusion, we report a 3-point sign as a novel MRI characteristic of SCD, expanding its imaging spectrum. Clinicians should be aware of this atypical MRI pattern, which may facilitate early diagnosis and avoid irreversible neurological injuries.

3. Discussion

Vitamin B12 is an essential nutrient for biochemical metabolism and myelin formation. Deficiency in vitamin B12, caused by either intrinsic factor depletion or exposure to nitrous oxide, can lead to spinal demyelination and axonal degeneration.[3-9] Specifically, gastrectomy, excessive drinking, and vegetarianism have been identified as risk factors for vitamin B12 deficiency. The adult daily requirement of vitamin B12 is 2μg, and a poor diet can result in vitamin B12 deficiency. In the current case, the patient was a vegetarian who ate very little meat and only weighed 43kg. On MRI, abnormal hyperintensity was noted in the posterior and lateral columns of the spinal cord, which could explain the sensorimotor disturbances in bilateral extremities. Further laboratory examination revealed an extremely low level of serum vitamin B12. These findings strongly suggested SCD induced by vitamin B12 deficiency. Following vitamin B12 replacement therapy, the patient’s symptoms were significantly relieved, confirming the diagnosis of SCD.

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Author contributions

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