Cobalamin Deficiency with Myelopathy: MRI Findings

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We described a 61-year-old man with cobalamin deficiency presenting with thoracic myelopathy for one month. Initial T2-weighted MR images revealed abnormal symmetrical hyperintense signal at posterior column of the spinal cord between T7 and T10 levels. The vertebral bone marrow depicted abnormal hypointense signal on both T1 and T2-weighted images. Subsequent serological test confirmed cobalamin deficiency. Although only few cases have been reported, abnormal MRI findings on the spinal cord and vertebral bone marrow should alert clinicians to consider Cobalamin deficiency. Laboratory tests should be performed to confirm the diagnosis. In our case, follow-up MRI findings correlated well with clinical outcome after treatment.

Key words: subacute combined degeneration, Vitamin B12 deficiency, Cobalamin deficiency, anemia, spinal cord, bone marrow, magnetic resonance imaging (MRI)

Patients with cobalamin deficiency may develop symmetrical sensory disturbance and loss of discriminative sensation of the lower limbs. These clinical manifestations commonly result from myelopathy of the posterior and lateral columns of the spinal cord. Cervical and upper thoracic segments are the most frequently involved areas. Findings on histology can reveal demyelination of the spinal cord [1,2]. Previous literature has had few case reports with magnetic resonance imaging (MRI) findings of the spinal cord [2-10]. Only two cases with vertebral bone marrow changes have been reported [8,9]. We herein report a patient with cobalamin deficiency presenting with myelopathy. MRI findings of the spinal cord and vertebral bone marrow changes before and after treatment are shown.

CASE REPORT

A 61-year-old man suffered from numbness and tingling pain of bilateral lower limbs for one month. Neurological examination revealed decreased proprioception and light touch sense around T10 level. The Romberg sign was positive. Under the impression of thoracic myelopathy, MRI (1.5-T, Horizon LX, General Electric, Milwaukee, Wis.) was performed with use of spine coil. On initial MRI study, T2-weighted images (TR=4200ms, TE=89.2ms) showed symmetrical abnormal hyperintense signal at posterior column of the spinal cord between T7 and T10 levels (Fig. 1a). No remarkable abnormality was detected on postcontrast T1-weighted images (TR=550ms, TE=13.6ms). The vertebral bone marrow depicted abnormal hypointense signal changes on both T1 and T2-weighted images (Figs. 1b and 1c). The MRI findings were possible compatible with cobalamin deficiency. Subsequent laboratory tests revealed macrocytic anemia (hemoglobulin, 9.7mg/dl; mean corpuscular volume, 128.4 fl; mean corpuscular hemoglobulin, 43.7 pg/cell). Serological tests revealed markedly decreased cobalamin level (1.0 pg/ml) as compared with the normal value (200-950 pg/ml). Endoscopy
showed a shallow ulcer and erosive gastritis at the gastric antrum. No evidence of atrophic gastritis was detected. Under the impression of cobalamin deficiency, the patient was treated with methylcobalamine (Methcoba) intramuscular injection. The patient’s clinical condition gradually improved after treatment.

After four months, follow-up MRI revealed no remarkable abnormal MR signal changes of the affected spinal cord and vertebral bone marrow (Fig. 2). The patient’s clinical condition was almost complete recovery.

**DISCUSSION**

Cobalamin deficiency causing spinal cord lesion is known as subacute combined degeneration [1,2]. Typical clinical manifestations are distal paresthesia and sensory ataxia of the lower limbs. Neurological impairment can progress to neuropathy, dementia and death in patients without treatment. Supplement treatment with vitamin B₁₂ can arrest the disease progression and relieve of symptoms to some degree. Approximately 50 percent of patients can completely recover after aggressive treatment [1].

Early pathological changes can occur in the center of posterior columns and, later on, the surface of the lateral columns of the spinal cord. The most severely involved areas are in the mid-thoracic segment but also can extend rostrally to involve cervical segments and medulla [1,2]. Microscopic findings are characterized by demyelination with eventual axon loss.

To our knowledge, few cases of subacute combined degeneration with MRI findings have been reported [3-10]. Typical findings are symmetrical abnormal hyperintense signal at the posterior columns of the spinal cord. In our case, hyperintense signal changes of the posterior columns were detected on T2-weighted images. No obvious abnormality was detected on T1-weighted and postcontrast images. No evidence of blood-nerve barrier (BNB) breakdown in the spinal cord could explain the MRI findings [10].

Another MRI finding was hypointense signal replacement of the vertebral bone marrow on both T1-weighted and T2-weighted images. The involved areas included vertebral bodies and posterior elements. Only one case report has mentioned this MRI findings [8]. Hematopoietic marrow hyperplasia may explain the hypointense signal replacement of the vertebral bone marrow on both T1-weighted and T2-weighted images. Our case did not show enhancement of vertebral bone marrow on postcontrast images. Yamada et al also reported a case with this finding [9].

Symmetrical involvement of the posterior column

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**Figure 1.** Initial MRI of the thoracic spine at T7 level. a. Fast spin-echo axial T2-weighted image (TR 4200/TE 89.2) shows symmetrical hyperintense signal at posterior column of the spinal cord (arrow), and abnormal hypointense signal at the bone marrow of the vertebral body. b. Sagittal T1-weighted (TR 550/TE13.6) and c. T2-weighted (TR 3600/TE 102) images show abnormal hypointense signal at vertebral bone marrow.
of the spinal cord is not specific for subacute combined degeneration. Other differential diagnoses may include multiple sclerosis, carcinomatous radiculopathy, myelitis and spinal cord injury. Although it is not common, hyperintense signal at the posterior column of the spinal cord on T2-weighted images and hypointense signal of vertebral bone marrow on both T1-weighted and T2-weighted images should alert clinicians to consider subacute combined degeneration. Laboratory tests should be performed to confirm the diagnosis. Findings of follow-up MRI may correlate well with clinical outcome after treatment.

**REFERENCES**


**Figure 2.** Follow-up MRI 4 months after treatment. **a.** Axial T2-weighted image (TR 4000/TE 88.8) of the thoracic spine at the same level as Fig. 1a shows no remarkable MR signal abnormality of the affected spinal cord and signal recovery of vertebral bone marrow. **b.** Sagittal T1-weighted (TR 550/TE13.6) image reveals normal signal intensity of the vertebral bone marrow.
維生素B₁₂缺乏症合併脊髓症狀：磁振造影之表現

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我們報告一名61歲男性因維生素B₁₂缺乏症合併脊髓症狀已一個月之病例。治療前胸椎（T7-T10）之T2加權磁振影像呈現脊髓後柱對稱性訊號增加，椎體骨髓在T₁與T₂加權影像均呈現低訊號，隨後之檢查證實維生素B₁₂缺乏。治療後，磁振影像所見之脊髓後柱高訊號消失，同時骨髓亦恢復正常訊號，與臨床病情改善表現一致。

關鍵詞：維生素B₁₂缺乏症、貧血、骨髓、脊髓、磁振造影。