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Case Report

# 척수 앞뒤기둥을 모두 침범한 아급성척수연합변성

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# Co-Existence of Anterior and Posterior Column Lesions in Patients with Subacute Combined Degeneration

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**KEYWORDS**
Subacute combined degeneration, Vitamin B12, Spinal cord

Subacute combined degeneration (SCD) of the spinal cord, which is a result of vitamin B12 deficiency, may cause irreversible neurological deficits. The lesion in the spinal cord is typically localized to the posterior and lateral columns, as the name implies. In this paper, we report on two patients with SCD, whose lesions involved the bilateral anterior column as well as the posterior and lateral columns. This report illustrates the importance of awareness of clinical and radiologic variable characteristics of SCD.

Subacute combined degeneration (SCD) is a treatable myelopathy known complication of vitamin B12 (cobalamin) deficiency.[1] Cobalamin is essential for synthesis and maintenance of the myelin sheath. Several previous studies have demonstrated cobalamin deficiency causes white matter lesions or defective myelination in the nervous system.[2] It usually affects the posterior and lateral columns of spinal cord. So far, no large-scale studies of anterior column involving SCD have been reported. There are only a few cases that have been reported. We reported two patients with SCD whose lesion involved bilateral anterior columns and reviewed previous studies.

## Case

### 1. Patient 1

A 72-year-old man was admitted due to weakness and numbness of bilateral distal limbs for 3 months. He denied any previous medical or surgical diseases. On neurologic examination, the bilateral ankle dorsiflexion was impaired (Medical Research Council [MRC] scale of strength grade 4). Brisk bilateral patellar tendon reflexes were observed. Proprioception was not perceived the below bilateral proximal fingers and ankles. Mild sensorimotor polyneuropathy was observed on nerve conduction study. A complete blood count showed hemoglobin of 7.4 g/dL (normal, 13.0-17.0 g/dL) and mean corpuscular volume (MCV) of 117.6 fL (normal, 81-96

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fL). The screening test results for syphilis and human immunodeficiency virus (HIV) were negative. Vitamin B12 level was decreased to 32.02 pg/mL (normal, 197-894 pg/mL) and homocysteine level was elevated above 50 μmol/L (normal, 5.9-16 μmol/L). The bone marrow biopsy confirmed pernicious anemia. T2-weighted magnetic resonance imaging (MRI) of the spine revealed high signal intensity lesion in the posterior and lateral columns along the cervical cord (Fig. 1A), aconsistent finding in the SCD. Also, focal signal changes were noted in the bilateral anterior columns (Fig. 1B). No significant abnormal finding was observed on gastroscopy. Scheduled intramuscular injection of mecobalamin (1 mg/day of intramuscularly for a week, followed by 1 mg/week for 4 weeks and then maintenance treatment at 1 mg/month) was performed. He began walking without assistance within a month, and abnormal signal intensity was reduced in the 2 months follow up MRI scan. His distressing tingling sensation and weakness was disappeared after 18 months of treatment.

### 2. Patient 2

A 61-year-old man was admitted due to gait difficulty. He had complained about intermittent numbness and a tingling sense of distal limbs 10 years ago. A month before admission, he became unable to walk without a cane. He had a history of heavy alcohol drinking for 12 years. A neurologic examination demonstrated weakness of bilateral ankle dorsiflexion (MRC scale of strength grade 4) and finger flexion (MRC scale of strength grade 4). Vibration sense was bilaterally decreased below the bilateral wrists and iliac crests. Deep tendon reflexes were brisk. On neuropsychiatric test, he presented decreased function on visuospatial and language memory. Hemoglobin was 10.3 g/dL, and MCV was 109.8 fL Antibody to intrinsic factor and anti-parietal cell were negative. The screening test results for syphilis and HIV were negative, and the blood copper concentration was normal at 116.1 μg/dL. Serum vitamin B12 level was 40.87 pg/mL and homocysteine level increased above 50 μmol/L. T2-weighted MRI of the spinal cord depicted increased signal intensity within bilateral anterior, posterior, and lateral columns of cervicothoracic spinal cord (Fig. 1C, D). SCD and macrocytic anemia caused by alcohol abuse was diagnosed and cessation of alcohol drinking and scheduled mecobalamin injection (1 mg/day of intramuscularly for 1 week, followed by 1 mg/week for 4 weeks and then maintenance treatment at 1 mg/month) has been started. The tingling sensation was improved within the 2 months, but weakness of the legs has persisted during the 23 months of treatment period.

## Discussion

The posterior and lateral columns of the spinal cord are the most common sites of involvement in SCD. The reason that specific tracts are preferentially involved in SCD is not clearly known. By the pathologic studies, deficiency of vitamin B12 leads to the formation of intramyelinic vacuoles in the spinal cord, making the myelin lamellae separated. The changes begin in the posterior columns of the cervicothoracic spinal cord and spread longitudinally and transversely.[3] This might explain that signal change on MRI is more commonly

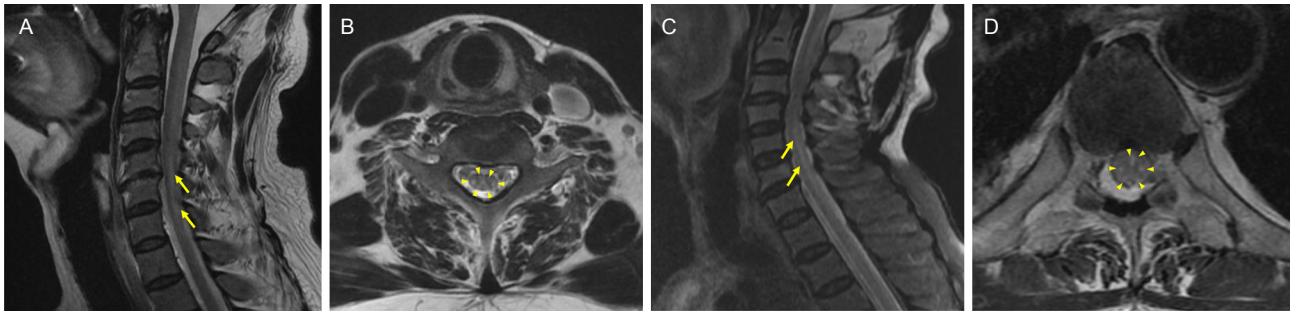


**Figure 1.** Magnetic resonance imaging images of the spinal cord lesions in two patients. Patient 1, T2-weighted imaging (T2WI) sagittal image of high-signal intensity lesion extends from C4 to C5 (A, arrows), T2WI axial image of bilateral anterior, lateral, and posterior column lesions at C5 level (B, arrowheads); patient 2, T2WI sagittal image of high-signal intensity lesion extends from C5 to C6 (C, arrows), T2WI axial image of bilateral anterior, lateral, and posterior column lesions at T3 level (D, arrowheads).

**Table 1.** Comparison of SCD patients with anterior column of spinal cord on previous reports

| | Patient 1 | Patient 2 | Wu et al.[4] (2020) | Paliwal et al.[5] (2009) | Puntambekar et al.[6] (2009) | Karantanas et al.[7] (2000) |
|---|---|---|---|---|---|---|
| Sex | Male | Male | Female | Female | Female | Male |
| Onset age (years) | 72 | 61 | 42 | 50 | 42 | 61 |
| Key clinical feature | Spastic ataxic paraparesis, hypoesthesia on distal limbs | Gait difficulty, spastic ataxic quadriparesis, hypoesthesia on distal limbs, memory loss | Spastic ataxic quadriparesis, symmetric hypoesthesia of bilateral distal limbs | Spastic ataxic quadriparesis | Numbness and weakness on distal lower limbs, urinary incontinence, memory loss | Gait difficulty, apathy, irritability |
| Related comorbidity | Pernicious anemia | Alcohol abuse | Pernicious anemia | Vegeterian | Pernicious anemia | Gastrectomy |
| Magnetic resonance imaging | Anterior, lateral, and posterior columns lesions on cervicothoracic spinal cord | Anterior, lateral, and posterior columns lesions on cervicothoracic spinal cord | Anterior and lateral columns lesions on cervicothoracic spinal cord | Anterior, lateral, and posterior columns lesions on cervicothoracic spinal cord | Anterior and posterior columns lesions on cervicothoracic spinal cord | Anterior and posterior columns lesions on thoracic spinal cord |
| Serum vitamin B12 level (pg/mL) | 32.02 | 40.87 | 50 | 78 | <150 | 65 |
| Treatment period | 18 months | 23 months | 1 month | 6 months | 12 days | 9 months |
| Treatment outcome | Complete recovery | Improved, weakness remained | Much improved, mild gait disability remained | Complete recovery | Much improved, mild distal weakness remained | Complete recovery |

observed in the posterior column than in the lateral column.

We summarized previous studies that have reported rare anterior column affected patients with SCD (Table 1). Most studies reported that co-existence of lesions in anterior and lateral and/or posterior columns of cervicothoracic spinal cord like our study.[4-6] Only one case was described to involve the anterior and posterior column of the lower spinal cord level, very unusual level in SCD.[7] The pattern of pathologic progress suggests that the anterior column involvement might mean longer disease duration and a more severe state. But the relationship among the extent of the MRI lesion, severity of the deficits, and the prognosis is uncertain. Some authors have suggested that MRI change represents early stage of SCD, so potentially reversible state of the disease.[8] Others have argued that MRI might have a clinical role in monitoring the efficacy of treatment.[9]

The above two patients had a different cause of SCD and a different outcome on mecobalamin treatment. On a previous study pernicious anemia may be related to a poor outcome.[9] Nevertheless in our report, patient 1 with pernicious anemia achieved complete recovery whereas the patient 2 who had been showing less severe anemia on initial evaluation, also presented poor recovery. The previous studies of anterior column SCD patients related to pernicious anemia also reported rapid recovery.[4-6] There is no certain study organizing the contributing factor of treatment outcome in SCD patients. In our assumption, the poor outcome of patient 2 might be related to a synergetic effect of metabolic blockade of vitamin and direct neurotoxicity caused by chronic alcohol exposure. Alcohol-related pathology is not limited to the spinal cord. Especially cortical degeneration due to alcohol abuse would be presenting the memory loss and behavioral change.[2] Patient 2 showed cognitive dysfunction on initial evaluation. Puntambekar et al.[6] and Karantanas et al.[7] also reported cortical and autonomic dysfunctions in patients with SCD, but each patient has different etiologies not related to alcohol abuse. Further evaluation of cerebral and autonomic dysfunctions in SCD patients will be needed for comprehensive treatment.

We reviewed patients with anterior column involved SCD and reviewed previous studies. In our opinion, anterior column can be involved in SCD, but more radiological studies should be performed to elucidate its clinical implication.

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