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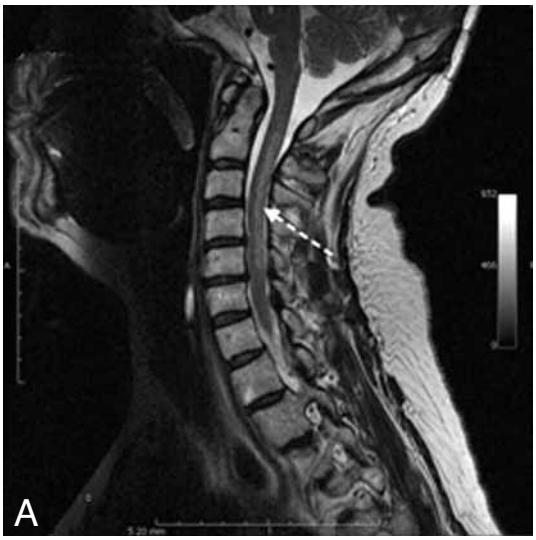
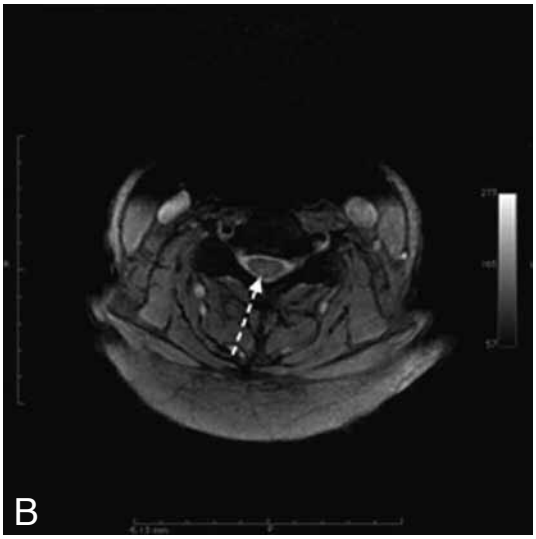
# IMAGES IN CLINICAL RADIOLOGY




## *Subacute combined spinal degeneration caused by cobalamin deficiency*

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A 51-year-old woman consulted the department of neurology with a 5 month-history of progressive numbness and tingling in a glove and stocking distribution. The problems started in both feet, resulting in unsteadiness and difficulty walking. Motor weakness and impairment of sensation of fine touch were also noticed.

Laboratory data revealed a vitamin B12 deficiency and nerve conduction studies showed limited polyneuropathy.

Magnetic resonance imaging of the cervical spinal cord demonstrated abnormal hyperintense signal changes on T2-weighted imaging of the posterior and lateral columns at the C2-C5 level (Fig. A,B). This is known as a typical finding compatible of SCD caused by a cobalamin deficiency.

*Comment*

Vitamin B12 deficiency is a systemic disease that affects many organs including the entire nervous system. It is caused by malabsorption in the gastrointestinal tract, insufficient nourishment by food, genetic deficiency of methylmalonyl-CoA mutase enzyme or medically induced (bariatric surgery, nitrous-oxide induced). The spinal cord is often affected first and exclusively although brain, optic nerves and peripheral nerves may also be affected. SCD denotes specifically the vitamin B12 deficiency as the etiologic cause of the spinal cord involvement. The diagnosis is based upon clinical features, laboratory findings of vitamin B12 deficiency, the presence of posterior and lateral column lesions on MRI and the improvement with vitamin B12 replacement.

Vitamin B12 plays a key role in the stimulation of myelin formation that speeds transmission of nerve signals. Damage of the myelin sheath causes degeneration of the sensory and motor nerve fibres from the spinal cord. Most affected are the posterior and lateral roots, with posterior column involvement being earlier in onset and more severe than that of the lateral columns and giving rise to the known clinical presentation.

MRI plays a key role in the diagnosis of SCD. Typical MRI finding of subacute combined degeneration are symmetrical abnormal hyperintense signal of the spinal cord on T2-weighted MRI of posterior and lateral columns of cervical and thoracal cords. This abnormal signal detected in advanced cases of vitamin B 12 deficiency reflects increased water content of the tissue (due to vacuole formation and swelling of the tissue). Similar sign is seen in multiple sclerosis or spinal cord infarction. The location and the relative symmetry of these lesions allow the differentiation of the disease from other intramedullary lesions rather than their signal characteristics. T1-weighted images may show decreased signal in the posterior columns and sometimes demonstrate reversible spinal cord swelling.

Contrast enhancement is also reported and is explained as the result of disruption of the blood-cord barrier that may occur in advanced stages.

There is a correlation between the normalization of the MRI signal and the overall improvement of the clinical picture, which makes MRI an important tool in the treatment follow-up. Therapy leads to remyelination of the affected white matter, the signal intensity returns to normal in parallel with clinical improvement. In severe cases of neuronal damage, total recovery is not always possible. Persisting high signal intensity and an irreversible neurological impairment can be seen even after aggressive treatment.

*Reference*

1. Ravina B., Loevner L.A., Bank W.: MR findings in subacute combined degeneration of the spinal cord: a case of reversible cervical myelopathy. *AJR*, 2000, 174: 863-865.

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