Subacute combined degeneration due to vitamin B12 deficiency in the absence of macrocytic anemia

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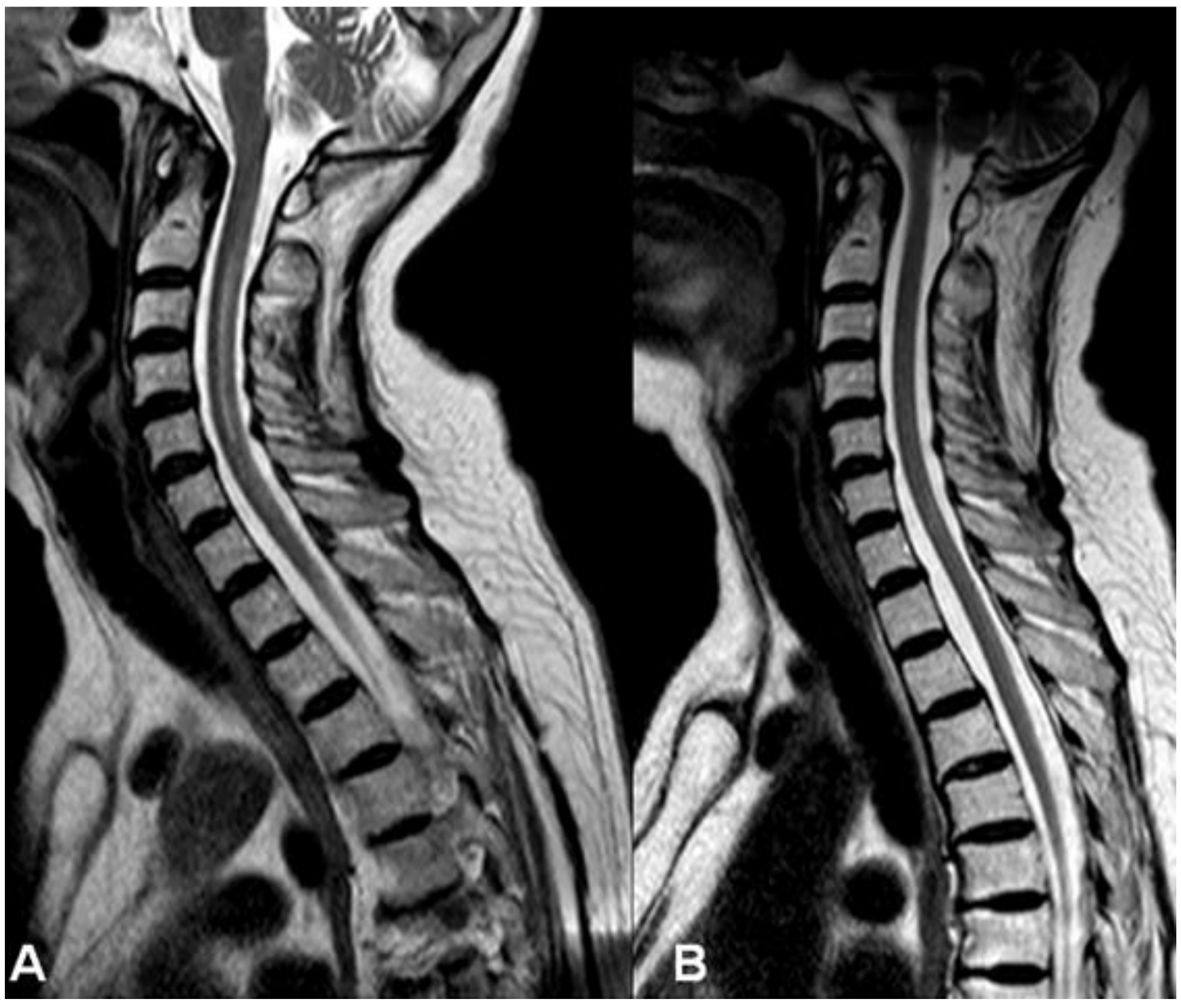
Background: Subacute combined degeneration (SACD) is the most frequent neurological manifestation of vitamin B12 deficiency. The radiological features of SACD could be misdiagnosed, if not supported by the presence of macrocytic anemia. We report the case of a patient with clinical manifestations of SACD, in absence of anemia, mimicking a myelitis.

Case Report: A 67-year-old woman was referred to our clinic as she became unable to walk. Neurological examination revealed upper limbs weakness and lower limbs paraplegia, absence of joint position and vibration sensation in the four limbs, decreased perception of touch, pain and temperature; deep tendon reflexes were brisk in the upper limbs and absent in the lower limbs, plantar response was in extension bilaterally. Laboratory investigations were unremarkable (Hb: 13.2 g/dl, RBC: 4.12x106/µL, MCV: 98.6 fl). Brain and spinal MRI were performed, depicting increased T2-weighted signal in the spinal cord from C2 trough C6 level without enhancement with gadolinium, interpreted as myelitis. Although the spinal lesion was interpreted of inflammatory origin and patient did not have features of anemia, the clinical picture was suspicious of SACD due to vitamin B12 deficiency. Hence, we proceeded with serum vitamin B12 dosage, which was found to be very low: 9 pg/ml. Anti-parietal-cell antibodies resulted positive (1/320) and homocysteine levels were 81.8 µmol/L. Gastric biopsy disclosed an atrophic gastritis. Patient was treated with cyanocobalamin 5000 µg daily for the first week and 1000 µg/week for the further 6 months, with clinical improvement.

Discussion: Neurological manifestations of vitamin B12 deficiency typically occur after the onset of hematological abnormalities. However, vitamin B12 deficiency rarely induces neurological signs without hematological alterations and when this clinical discrepancy is present, the most common manifestations are neuropsychiatric (1,2). Only Ralapanawa and colleagues described the case of a vegetarian patient presenting with SACD in the absence of hematological manifestations of vitamin B12 deficiency (3). However, in that case no MRI study of the spinal cord was mentioned to confirm SACD. In our case, cervical spinal cord lesion in the posterior and lateral columns fully recovered after 6 months of cyanocobalamin therapy. The neuroradiological resolution coincided with a meaningful improvement of patient's neurological status. In case of acute or subacute paralysis in association to sensitive impairment, especially if correlated with increased T2-weighted signal of spinal cord, vitamin B12 deficiency should be always suspected, even without hematological abnormalities, avoiding a delay of an appropriate treatment.

Figure. Spinal MRI T2-weighted image sagittal section shows high signal intensity lesion posteriorly within the cervical cord from C2 trough C6 (A); sagittal section of cervical spine at 6 months follow-up shows no evidence of the T2-weighted lesion previously detected (B).



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