Autoimmune atrophic gastritis presenting as myelopathy in a young patient

Mielopatia como apresentação de gastrite atrófica autoimune em um doente jovem

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Non-traumatic causes of extensive hyperintense spinal cord signal on T2-weighted images include a wide-ranging variety processes, such as tumors and associated disorders, inflammation, infection, vasculopathy, congenital disorders or metabolic diseases [namely vitamin B_{12} deficiency (VBD)].

We present a 36 year-old male patient complaining from progressive sensory loss on both hands and sensation of chest and abdominal tightness, lasting for five weeks. Neurological examination revealed a mild tetraparesis, glove and sock hyposthesia, C2 to T11 sensory impairment, vibratory anesthesia and proprioceptive errors of lower limbs. Cerebellar tests were normal. Romberg, Babinski and Lhermitte signs were absent. Laboratory tests revealed Hb 14.3 g/dL, mean corpuscular volume 104 fL. Renal and hepatic functions, iron study, copper levels and angiotensin conversion enzyme, and folic acid were normal. Vitamin B12 level was <82 pg/mL (189–883 pg/mL). Serum immunology, virology and bacteriology were negative. CSF study and brain MRI were unremarkable. Cervical spine MRI showed hyperintense spinal cord signal on T2-weighted images affecting mainly dorsal, but also lateral, columns between medulla oblongata and T11 levels, with subtle contrast enhancement (Fig A-C). Basing on clinical and laboratory examinations, the patient was evaluated for extensive myelopathy due to VBD, and it was performed an upper gastrointestinal examination which revealed an atrophic mucosa; histological examination showed chronic atrophic gastritis and intestinal metaplasia, excluding *Helicobacter pylori* infection. Anti-parietal cells were negative, but anti-intrinsic factor antibodies were positive. Treatment was performed with IM cyanocobalamin replacement, 1,000 μg IM daily for a week, then weekly for four weeks and monthly afterwards. Three months later, there was a significant clinical recover, only remaining hypoesthesia of hand fingers. MRI performed at this time showed an exuberant improvement (Fig D).

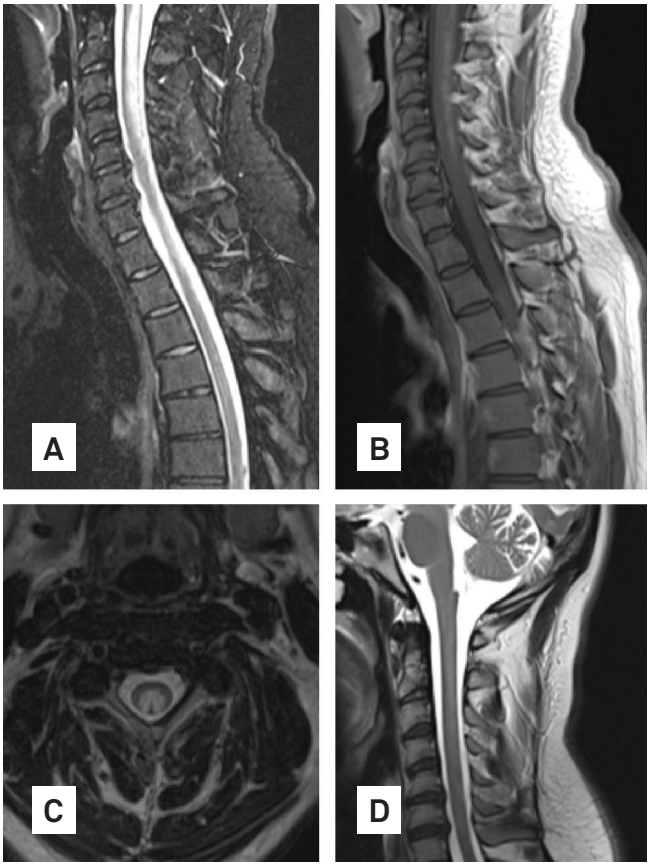

Fig. Spinal cord MRI: (A) extensive swelling of spinal cord, caused by vacuolization of myelin; (B) subtle contrast enhancement of posterior column; (C) cross-section images of spinal cord MRI: bilateral paired areas of hyperintensity affecting dorsal and lateral columns; (D) three months after treatment: significant improvement of lesions, with mild and no gadolinium enhancer T2 hyperintensity extending from bulbo-medular transition until C7 without spinal cord expansion.

Subacute combined degeneration (SCD) refers to degeneration of the posterior and sometimes lateral columns of the spinal cord usually as a result of VBD, which effects may not be appreciated until several years, since there is a significant body store of vitamin B_{12}[1,2]. Its absorption occurs via the ileal microvilli after binding to the intrinsic factor[3], and malabsorption

provoked by gastric atrophy is the commonest cause of VBD. Autoimmune atrophic gastritis (AAG) is a special type of gastric atrophy characterized by serum antibodies antiparietal cells and/or anti-intrinsic factor. AAG was an unexpected diagnosis since it is a relatively rare disease and the peak age of onset is 60 years, with only 10% of patients being <40 years of age. Myelopathy alone as clinical presentation is also a rare situation, occurring in about 12% of patients[1]. Concerning on imagiological findings, there was not only the involvement of the posterior column, but also of the lateral column, rarely involved in SCD[4]. Attention must be taken since AAG increases the risk of gastric carcinoid tumors and gastric carcinoma.

So, although being a rare condition, mainly in that age group, AAG needs to be remembered concerning its treatable nature and malignant potential that requires an appropriate follow-up.

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