Spinal abnormalities in vitamin B12 deficiency

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A 25 year-old woman was hospitalized in psychiatry with progressive weight loss and diagnosis of anorexia. Her past medical history mentioned hyperthyroidism.

For 3 months, she had been complaining of paresthesia of the lower limbs and ataxia without vertigo or lomboischialgia. Neurological examination revealed considerable ataxia without motor deficit neither abnormality of the cranial nerves.

Diffuse tactile hyopaesthesia of lower limbs bilaterally (most on the left side) was found.

There was a vibration anesthesia and with lost position sense of lower limbs but with normal thermalgesia.

Evoked potentials showed a central origin, medullar bilateral (most on the left).

No lesions were found on CT-scan of the brain.

Magnetic resonance imaging (MRI) of the cervical spine, revealed focal hyperintense lesions into the dorsal spinal cord, most evident at the C4-C5 level. Hyperintense lesions on the sagittal T2 weighted/TSE (Fig. A) and STIR images (Fig. B) in the dorsal spinal cord with also a combined alteration of the bone marrow (hypo-intense signal of bone marrow on T1 and T2 weighted/TSE) were seen. These hyperintense lesions were found in the dorsal spinal cord columns, more evident on the left side, on axial T2 gradient echo weighted images (Fig. C). The combination of lesions involving both the dorsal spinal cord columns and also the bone marrow, together with the clinical history of anorexia, was suggestive of deficiency alterations. Blood analysis revealed a megaloblastic anemia with vitamin B12 deficiency. Folic acid level was normal. Endoscopy demonstrated chronic atrophic gastritis with intestinal metaplasia. The patient was treated with intramuscularly injections of cobalamin with a rapid clinical recuperation and also a normalization of the signal abnormality in both cord and bone marrow (Fig. D).

Comment

On MRI, the vitamin B12 deficiency, which is causing a subacute combined sclerosis or degeneration, will show most typically hyperintense lesions to the dorsal spinal cord columns. Also the alteration of the bone marrow is considered as a result of the anemia with an augmentation in hematopoesis. In the differential diagnosis of the spinal cord lesions, a large list of etiologies must be considered. Among them, inflammatory demyelization (multiple sclerosis or disseminates encephalomyelitis) is the most frequent cause. Also infectious lesion (herpes, HIV,...), miscellaneous (sarcoïdosis,...), amyotrophic lateral sclerosis, iatrogenic (radiation myelitis), traumatic or degenerative compressive myelopathy, vascular (ischemic – vascular malformations) and metabolic (deficiency syndrome B12, Cu, Vit E, N³O,...), are potentially reasons for spinal cord abnormalities. However, the selective dorsolateral spinal cord degeneration is typically for this subacute combined sclerosis, due to the vitamin B12 deficiency, with to pernicious anemia a as result. Other etiologies of vitamin B12 deficiency, includes other intestinal diseases (Crohn's disease, celiac disease, bacterial overgrowth in intestinal blind loops) and also strict vegetarian diet. Nevertheless, preferential involving of the posterolateral columns of the spinal cord and also the changes of the bone marrow are highly suggestive of vitamin B12 deficiency. The treatment is life-long parenteral vitamin B12 administration and an early diagnosis is important, because the treatment will stop the degenerative myelin sheath degeneration, but in will not restore the destroyed nerve fibers. In our case, the rapid administration of cobalamin, was resulting in a good clinical recuperation and disappearing of the lesions in both spinal cord and bone marrow.