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Myelo-Myelo Neurological Complication Of Acquired Copper Deficiency.

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Abstract :
Copper deficiency myeloneuropathy is a rare condition. It is constantly associated with hematological manifestations. Some acquired etiologies are well individualized (gastric surgery, malabsorption, excessive zinc ingestion).
We report the observation of a 29-year-old woman with a history of unexplored intermittent pain, who presented with paraplegia, tingling and urinary incontinence, slowly progressively worsening.
The clinical examination found a growth retardation (a BMI of 18) and a spinal cord syndrome. Cerebrospinal MRI shows cervico-dorso-lumbar spinal cord atrophy without enhancement, low cuprumia and cupruria. The digestive fibroscopy followed by a biopsy is in favor of gastritis and erythematous bulbo-duodenitis with villous atrophy.
The diagnosis retained is that of copper deficiency myelodysplasia-myeloneuropathy secondary to diarrhea. chronicle.

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I. Introduction:

Myelo-myelo due to copper deficiency is a rare condition, described a few years ago (Kumar et al., 2004).(1). It constantly associates neurological impairment and hematological abnormalities.

The neurological picture is that of spinal cord injury with a pyramidal syndrome and proprioceptive abnormalities. Clinically, it combines spastic ataxia and predominantly sensitive axonal neuropathy.
Hematological involvement includes anemia, leukopenia or even pancytopenia.

Acquired copper deficiencies have a well individualized etiology (gastric surgery, malabsorption, excessive zinc ingestion).

We present a case of a patient with copper deficiency myelo-neuropathy associated with hematological manifestations.

II. Case report:

This is a 29-year-old woman with a history of unexplored intermittent diarrhea with predominantly crural muscle weakness in all four limbs, associated with tingling and a sensation of running hot water. She describes the progressive worsening of neurological signs that appeared 5 years earlier.

Clinical examination found mucocutaneous pallor, stature with a body mass index of 18. Neurological examination revealed spinal cord involvement manifested by a posterior cord syndrome, a spastic pyramidal syndrome in the four limbs and vesico-sphincter disorders of the incontinence type (urinary and fecal).

Spinal MRI (figure 1) shows cervico-dorso-lumbar spinal atrophy, without enhancement after injection of gadolinium. The brain MRI (figure 2) was normal
ENMG of 04 limbs finds sensory-motor axonal poly-neuropathy in the lower limbs.

Biological examinations revealed leuko-neutropenia (2000/Ul), with microcytic hypochromic anemia (hemoglobin 8g/dl). Cuprumia and cupruria have returned to low levels. The immunological assessment as well as the dosage of vitamin B 12 were normal.

Digestive fibroscopy was in favor of erythematous gastritis and bulbo-duodenitis with grade II villous atrophy on the anatomo-pathological analysis of the biopsy.

The patient was supplemented with copper orally with stabilization of the neurological syndrome (arrest of disease progression) and correction of anemia and leucopenia.

The diagnosis retained is myelo-myelo linked to a phenomenon of digestive malabsorption secondary to unexplained diarrhea.

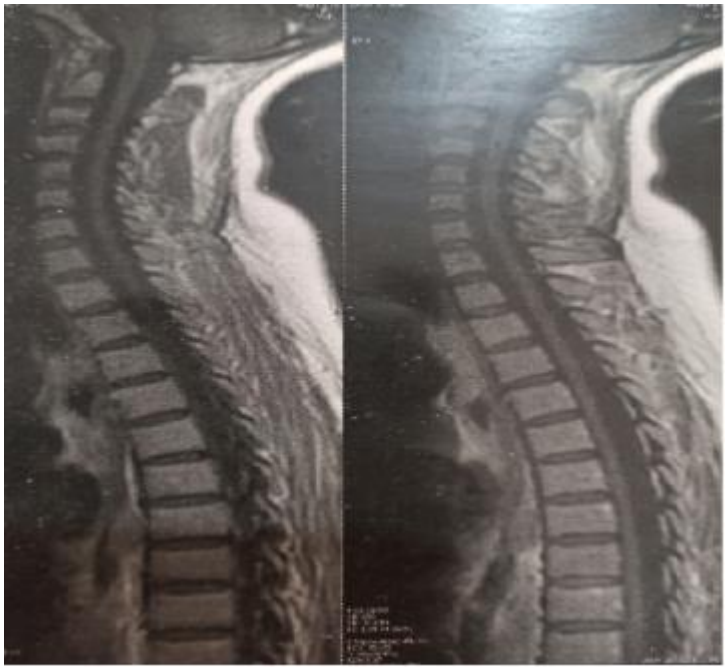

Figure 1: Spinal MRI (sagittal sections, T2 sequences): cervico-dorso-lumbar spinal atrophy.

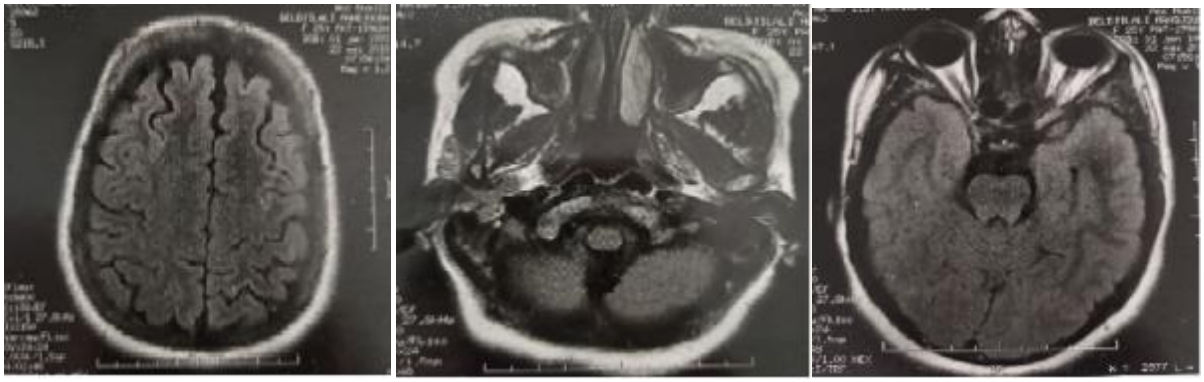

Figure 2: Brain MRI: (axial sections, T2 sequences): normal.

III. Discussion:

The origin of the neuro-hematological disorders in our patient was linked to a copper deficiency. Copper is a cofactor of many metallo enzymes including Cytochrome C.oxidase involved in the regulation of energy metabolism, the methylation of proteins including myelin, as well as hematopoiesis. This deficiency responsible for the physiopathological phenomena already described in the literature remains a rare cause but also underdiagnosed(2).

Copper deficiency is responsible for a clinical and paraclinical picture similar to that of vitamin B12 deficiency (combined sclerosis of the marrow). It can sometimes be manifested by damage to the peripheral motor neuron with amyotrophy, motor deficit, cramps and fasciculations (Madsen and Gitlin, 2007) (3), as well as subacute cerebellar ataxia (4), already described in animals. The best known is that of enzootic ataxia of lambs (5).

In a review of the literature published in the article by Jaiser and Winston (2010) (6); , the main etiologies of acquired copper deficiency were: upper gastrointestinal surgery (47% of cases), zinc intoxication (16% of cases) and malabsorption (15%) mainly in celiac disease. In 20% of cases, no etiology was found. The use of zinc-rich dental adhesive paste is also an unknown cause of copper deficiency myelopathy (7).

IV. Conclusion:

Acquired copper deficiency myeloneuropathy is a rare but probably underestimated pathology. Faced with a neurological picture with symmetrical involvement of the limbs, associated with hematological damage, a metabolic or mineral origin should be systematically sought and corrected, in particular a copper deficiency.

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