Neuropsychiatric Disorders Revealing Vitamin B12 Deficiency:About a case

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Introduction:

Vitamin B12 (cobalamin) deficiency is a common and potentially serious disorder. However, it is often underdiagnosed because of its insidious installation and its often unclear and sometimes atypical clinical manifestations.

Case report:

A 60-year-old patient, with no particular somatic history, having had a major depressive episode isolated in 1994 following incarceration. The patient was referred to our department in 2015 for irritability and character change. The examination revealed a depressive syndrome.

The patient was treated by fluoxetine (Serotyl *) and prazepam (Lysanxia *), but without improvement despite good compliance and regular monitoring. In July 2017, the patient consulted us with his wife who confirmed a worsening of the clinical symptoms with disabling memory disorders, sphincter incontinence, fine tremor in both hands with disinhibition and delirium against his wife. An etiological assessment was negative (biology and radiology). The dosage of vitamin B12 showed a vitamin deficiency evaluated at 146 Pmol / l.

Patient was referred to the internal medicine department for further follow-up.

The patient was treated with monthly injections (substitution treatment). The subsequent evolution was marked by an improvement in contact and mood, a more coherent speech with distancing delusional ideas, but memory problems persist to a lower degree.

Literature review:

Epidemiological studies indicate a prevalence of cobalamin deficiency close to 20% in the general population of industrialized countries; 15% in the Framingham study [1]. In the elderly and/or institutionalized subject, this prevalence seems higher: between 30 and 40% [2,3].

In adults, the etiologies of B12 deficiencies are mainly represented by the syndrome of non-dissociation of B12 from its carrier proteins and Biermer's disease, more rarely by intake or nutritional deficiencies and malabsorptions[2,4,5].

The main clinical manifestations of B12 deficiencies are extremely polymorphic and of varying severity, ranging from common sensory polyneuritis or isolated abnormalities of the blood count to macrocytosis or hypersegmentation of neutrophils, to extreme symptoms of combined sclerosis of ibthe marrow or hemolytic anemia or even pancytopenia and thrombotic pseudomicroangiopathy[6,7,8,9].

In practical terms, it should be noted that B12 deficiency can take several years to become apparent and that neuropsychiatric manifestations can be isolated (without abnormal blood count) and are frequently irreversible (aggravated by untimely folate intakes)[7].

In addition to hematological disorders, neurological signs are common during vitamin B12 deficiency. In the Healton[8,10] and Andres[11] series, neurological disorders are reported in two out of three patients. These disorders are dominated by the combined marrow sclerosis table. It clinically combines a pyramidal syndrome and a posterior cordonal syndrome where proprioceptive ataxia and paresthesias are in the foreground[8,10].

Paresthesias are described several months before deep-sensitivity disorders and the pyramidal syndrome is often reduced to a bilateral Babinski sign. Indeed, motor deficit is rare, it is especially the prerogative of advanced forms of late diagnosis. This combined sclerosis of the marrow is very suggestive of vitamin B12 deficiency because it accounts for 25 to 44% of neurological manifestations[8,10,12]. On the other hand, in series of combined sclerosis of the marrow, the deficiency origin of vitamin B12 is found in the majority of cases[13].

The contribution of bone marrow MRI is essential as a complement to clinical and biological studies, especially in pure neurological presentations without hematological modification or a decrease in vitamin B12. It classically shows a widening of the marrow with a T2 hypersignal of the posterior cords most often at the cervical level[14,15]. The reversibility of images after early adapted treatment confirms their relationship with vitamin deficiency. In addition, the location of MRI images is well correlated with neuropathological lesions found in post-mortem. The latter have a type of myelin thickening or degeneration, axonal destruction and gliosis that preferentially develop in the posterior and lateral cords of the cervicodorsal marrow[13,16,17]. Spinal cord MRI can also rule out other conditions that may be clinically responsible for a picture of combined sclerosis of the marrow.

Spinal cord MRI also has a prognostic value, since the persistence of spinal cord injuries at a distance from the beginning of treatment would indicate the presence of permanent clinical sequelae[11,14].

A few cases of polyradiculoneurocitis, dysautonomic disorders or regressive pain syndrome under vitamin treatment have also been reported[9].

Psychiatric disorders with mood instability, hallucination, aggressiveness, memory disorders or even dementia syndrome, improved with vitamin therapy, have been reported, especially in the elderly[18].

On the pathophysiological level, the neurological disorders observed during vitamin B12 deficiency are thought to be due to a methylation disorder of the myelin sheaths. Indeed, methylvitamin B12 is the cofactor of methionine synthetase allowing the methylation of homocysteine into methionine which participates in the formation of myelin basic protein[10]. The resulting hyperhomocysteinemia is an independent cerebrovascular risk factor and may be responsible for ischemic strokes and/or cognitive disorders in one of our patients[19]

Another mechanism mentioned in the genesis of these neurological disorders is the accumulation of methylmalonic acid, which is a fatty acid toxic to myelin and is normally converted to succinic acid by adenosyl B12[20]. The increase in homocysteinemia and methylmalonic acid is an early indicator of vitamin B12 deficiency[21].

The treatment of neurological impairment in vitamin B12 deficiency is no different from the treatment of forms without neurological impairment. [22],Cobalamin supplementation often leads to the almost complete disappearance of symptoms. However, some abnormalities only partially disappear, including some advanced neurological disorders secondary to old deficits, hence the need for early diagnosis and supplementation as soon as possible[22]

Conclusion :

Neurological disorders due to vitamin B12 deficiency are polymorphic. They can be isolated and occur outside of any hematological context and reveal the deficiency of which The etiologies are dominated by Biermer's disease and the non-dissociation syndrome of vitamin B12. The potential severity of some neuropsychiatric complications leads to to carry out a vitamin dosage before any neurological picture that does not prove its mechanism. A treatment early substitution is the only guarantee of the prognosis of these attacks.

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