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Neurological Manifestations of Vitamin B12 Deficiency: About a Case

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Abstract

The authors report a case of deficient sensory neuropathy secondary to vitamin B12 deficiency, diagnosed in the neurology department of the Sino-Central African Friendship University Hospital in Bangui. The diagnosis was made possible by electroneuromyography which showed subclinical neurological damage associated with hematological damage (anemia). Through this observation, we recall the diagnostic criteria of the disease in a context of difficult medical practice.

Keywords

Neurological Manifestations, Vitamins B12, Central African Republic

1. Introduction

Vitamin B12 deficiency is a common situation in adults; its prevalence is reported to be around 15% to 20% in the population. In general [1], it varies between 5% and 60% depending on the definition used, it is higher in elderly and/or institutionalized subjects (30% to 40%) [2]. Combined spinal cord sclerosis (CMS) being the main etiology reported neurologically that knowledge of its different clinical manifestations is necessary for practitioners in order to prevent possible serious complications.

2. Methodology

This was a descriptive cross-sectional study about a case of deficient sensory neuropathy secondary to vitamin B12 deficiency, diagnosed in the neurology department of the Sino-Central African Friendship University Hospital in Bangui.

3. Medical Observation

It was Mrs. W.F aged 49, civil servant, followed in the Neurology department of the Sino-Central African Friendship University Hospital in August 2022 for diffuse dysesthesia of the limbs and progressive physical asthenia. The onset of the symptomatology dates back to May 2022 with the progressive installation of isolated and symmetrical distal paresthesias of the four limbs like tingling. His history includes taking non-steroidal anti-inflammatory drugs (NSAIDs), and there is no evidence of metabolic or toxic disorders or familial peripheral neuropathy.

The clinical examination reveals a good state of consciousness with Glasgow score of 15/15, subpale conjunctivas with a skin recoloration time of less than 3 seconds, the BP is 130/85 mmHg, the temperature is 37.1˚C. There is painful hypoesthesia distal to the limbs, there is no trophic disorder and the exploration of myotatic and cutaneous reflexes is normal. The rest of the examination was unremarkable.

The paraclinical examination revealed:

- EMG carried out in Cameroon in October 2022 reveals a purely demyelinating neurogenic sensory damage to all four limbs compatible with a pure sensory polyneuropathy of deficiency or metabolic, inflammatory origin associated with a left L5 radiculopathy.
- EEG: Absence of paroxysmal abnormality.
- CBC: Normocytic normochromic anemia at 10 g/dl.
- Fasting blood sugar: 0.95 g/l.
- Blood ionogram: Within normal limits.
- Serologies: Lentivirus HIV, ASLO, BW, Hepatitis B, C are all negative.
- Homocysteine and vitamin B12 measurements could not be carried out.

Given these results, vitamin therapy with cobalamin (VITB12) and iron and folic acid supplementation were instituted. The clinical course is marked by a significant improvement in symptoms after ten days of medical treatment.

4. Discussion

In our series, it was a young subject unlike those reported in the majority of publications [3] [4]. This difference is explained by the small size of our sample.

The neurological signs were 100% initial in our patient while they were only 15.3% in the Maamar series [5]. This situation could result in preferential damage to the peripheral nervous system in our case.

The diagnostic delay for our patient was long and similar to other series [2] [3]. This is perhaps due in our case to diagnostic difficulties on the one hand, due to the rarity of the disease and to a delay in specialized medical guidance on the other hand.

The neurological clinical manifestations reported in our study were consistent with those usually described in vitamin B12 deficiency. However, carrying out additional examinations (electromyography, etc.) allowed the diagnosis of subclinical neurological damage but also of hematological damage (anemia).

This observation has been reported by several series [5] [6]; Mayer *et al.* diagnosed 9 asymptomatic disorders in 53 patients who underwent EMG [7]; Puri *et al.* demonstrated 31 myeloneuropathies, 5 isolated myelopathies and 4 isolated neuropathies in 40 patients using electrophysiological explorations [8].

On the paraclinical level: The appearance found on ENMG is a pure demyelinating neurogenic sensory damage of the four limbs compatible with a pure sensory polyneuropathy of inflammatory, metabolic or deficiency origin associated with an L5 radiculopathy. Cerebro-medullary MRI was not performed in our series. It is of interest in pure neurological presentations without hematological abnormalities or without vitamin deficiency.

On the biological level, there is a tendency towards the classic picture of vitamin B12 deficiency with associated hematological damage. In patients diagnosed early, it was the dosage of the precursors "homocysteine and methylmalonic acid" which allowed the diagnosis, in the absence of anemia or macrocytosis and even a vitamin B12 deficiency. Indeed, some authors have reported that 50% of patients with vitamin B12 deficiency can have a normal serum cobalamin level [6] [9].

Concerning the etiological diagnosis, the results of our series were similar to the data in the literature [2] [3].

The treatment of neurological damage does not differ from the treatment of forms without neurological damage. There are no recommendations regarding prescribing methods. In our series, the patient was subjected to a loading treatment based on cobalamin 1000 µg/day parenterally (intramuscular) for seven days and then 1000 µg/week, followed by a monthly maintenance treatment of 1000 µg orally. The effectiveness of oral treatment has been demonstrated in several studies, particularly on hematological damage. Only early replacement therapy guarantees full clinical recovery. Andrès *et al.* in 2010 confirmed the effectiveness and benefit of oral vitamin B12 through a review of all articles on oral vitamin B12 treatment, published between 1990 and 2007 [10].

From an evolutionary point of view, the improvement in neurological signs in our patient after ten days of treatment seems to be essentially linked to the precocity of the supplementation treatment. Under vitamin substitution, the development of neurological damage is assessed differently in the literature [10].

5. Conclusion

Our study revealed the polymorphic and non-specific nature of the neurological symptoms of vitamin B12 deficiency, which makes it difficult to establish a positive diagnosis and that there are also other extra-neurological manifestations. Knowledge of the symptoms of B12 deficiency is necessary in order to prevent possible serious complications.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Lindenbaum, J., Rosenberg, I., Wilson, P., *et al.* (1994) Prevalence of Cobalamin Deficiency in the Framingham Elderly Population. *The American Journal of Clinical Nutrition*, **60**, 2-11. https://doi.org/10.1093/ajcn/60.1.2

[2] Dali-Youcef, N. (2009) An Update on Cobalamin Deficiency in Adults. *QJM*, **102**, 17-28. https://doi.org/10.1093/qjmed/hcn138

[3] Loup-Leuciuc, A., Loup, P.J., Lombardi, T. and Samson, J. (2011) Vitamin B12 Deficiency (Part 1): Focust. *Médecine Buccale Chirurgie Buccale*, **17**, 211-224. https://doi.org/10.1051/mbcb/2011127

[4] Loukili, N.H., Noel, E., Blaison, G., Goichot, B., Kaltenbach, G., Rondeau, M., *et al.* (2004) Current Data on Biermer's Disease: About a Retrospective Study of 49 Observations. *Revue de Médecine Interne*, **25**, 556-561. https://doi.org/10.1016/j.revmed.2004.03.008

[5] Maamar, M., Tazi-Mezalek, Z., Harmouche, H., Ammouri, W., Zahlane, M., Adnaoui, M., *et al.* (2006) Neurological Disorders due to Vitamin B12 Deficiency: Retrospective Study of 26 Cases. *Revue de Médecine Interne*, **27**, 442-447. https://doi.org/10.1016/j.revmed.2006.01.012

[6] Brown, D.L. (2003) Vitamin B12 Deficiency. *American Family Physician*, **67**, 979-986.

[7] Mayer, R.F. (1965) Peripheral Nerve Function in Vitamin B12 Deficiency. *Archives of Neurology*, **13**, 355-362. https://doi.org/10.1001/archneur.1965.00470040021003

[8] Puri, V., Chaudhry, N., Goel, S., Gulati, P., Nehru, R. and Chowdhury, D. (2004) Vitamin B12 Deficiency: A Clinical and Electrophysiological Profile. *Electromyography and Clinical Neurophysiology*, **45**, 273-284.

[9] Saperstein, D.S., Wolfe, G.I., Gronseth, G.S., Nations, S.P., Herbelin, L.L., Bryan, W.W., *et al.* (2003) Challenges in the Identification of Cobalamin-Deficiency Polyneuropathy. *Archives of Neurology*, **60**, 1296-1301. https://doi.org/10.1001/archneur.60.9.1296

[10] Andrès, E., Mecili, M. and Ciobanu, E. (2010) Oral Vitamin B12 in the Era of Evidence-Based Medicine. *Médecineth Érapeutique*, **16**, 5-11.