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Neurological Manifestations of Vitamin B12 Deficiency: Study of a Series of 43 Cases

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Abstract:
Background*: Vitamin B12 deficiency can affect the nervous system, with various neurological manifestations causing diagnostic difficulties, especially when the blood count and vitamin B12 dosage are normal.*
Materials and Methods*: We propose to analyze the epidemiological, clinical, para-clinical and evolutional profiles of 43 patients with neurological manifestations due to vitamin B12 deficiency, hospitalized at the department of neurology of Bab El Oued University Hospital Algiers.*
Results*: The study included 43 patient: Male:34, Female: 09 ; all the results will be shown in the tables below.*
Conclusion: *Our study shows the clinical polymorphism of neurological manifestations of vitamin B12 deficiency. The results should prompt us to early diagnosis and treat to improve the prognosis of this pathology*
Key Word*: Vitamin B 12 deficiency, Biermer's disease, neurological disorders.*

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I. Introduction

Vitamin B12 deficiency is frequently found in clinical practice (1). Its etiologies are multiple and are mainly represented by Biermer's disease and vitamin B12 non-dissociation syndrome in the elderly [2]. This deficiency is initially expressed by hematological anomalies; however, an inaugural or isolated neurological presentation is not uncommon [1]. These neurological manifestations respond well to parenteral vitamin B12 therapy, but the quality of recovery depends on the type of neurological damage and especially on the precocity of its establishment [2-4]. Very little work has studied the neurological manifestations of vitamin B12 deficiency in Algeria. We propose to analyze the epidemiological, clinical, para-clinical and evolutionary profiles of 43 patients hospitalized in the department of neurology of Bab El Oued University Hospital Algiers for neurological disorders related to vitamin B12 deficiency.

II. Material And Methods

Our work consisted in a retrospective study of 43 cases of patients with neurological manifestations due to vitamin B12 deficiency, hospitalized in the department of neurology of Bab El Oued University Hospital Algiers, over a period of 10 years between January 2010 and December 2019. Data collection was done through hospital records.

Inclusion criteria:

1. Presence of neurological signs and anomalies of the para-clinical examinations compatible with a vitamin B12 deficiency ;
2. Vitamin B12 level less than 200 mg/l ;
3. Patient having undergone an attack treatment followed by maintenance treatment based on hydroxocobalamin intramuscularly (IM) according to the standard protocol (5000 µg / day of hydroxocobalamin IM for one week, then 5000 µg / week for 1 month then a maintenance treatment with 5000 µg / month for life in the case of Biermer's disease and sequential for the other etiologies).

The parameters studied:

1. Demographic characteristics: age and sex;
2. Medical and surgical pathological history;
3. The data of the clinical examination, in particular the signs of anemia;
4. Neurological signs, their start dates and evolutionary data;
5. Hemogram data;
6. The serum dosage of vitamin B12;
7. Data of neurophysiological explorations;

8. Data of spinal and encephalic magnetic resonance imaging (MRI);
9. Data of eso-gastro-duodenal fibroscopy and the search for antibodies against intrinsic factors and anti parietal cells.

The results are expressed in absolute value and percentage for the qualitative variables, on average, standard deviation and extreme values for the quantitative variables.

III. Results

Table no 1: The average age of our patients was 49 ± 6 years with extremes ranging from 40 to 73 years. There is a male predominance with a sex / M / F ratio of 3.8.

Table no 1: Shows demographic characteristics

Characteristics	**Number of patients**	**Percentage**
Total patients	43	100%
Age		
Median	49±6 years	
Extreme values	40 – 73 years	
Sex		
Male	34	80%
Female	09	20%

Table no 2: With all our patients, vitamin B12 deficiency was revealed by neurological symptoms and hematological involvement was not previously known in any of our patients. In contrast, neurological involvement was only isolated in 8% of cases.

Table no2: Clinical features

Characteristics		**Number of patients**	**Percentage**	
Total of patients		43	100 %	
Diagnostic delay				
Average		3.8 ± 1.9 months		
Extreme values		10 days to 18 months		
Neurological signs				
Spinal cord damage		26	60 %	
Peripheral impairment		17	40 %	
Encephalic damage		03	07 %	
Optic neuropathy		05	12 %	
Association of damages (2 or more)		13	30 %	
Neurological investigations : (EPI, ENMG and brain MRI)				
Spinal cord damage (posterior cord +/- pyramidal)		26	60 %	
Peripheral impairment (sensory neuropathy)		28	65 %	
Encephalic damage		7	16 %	
Optic neuropathy		16	37 %	
Association of damages (2 or more)		35	81 %	
Macrocytic anemia (hemogram)				
Absent at diagnosis		06	14 %	
Present	discovered at diagnosis	37	86 %	86%
	Known	00	00 %	
Fundic atrophic gastritis with anti-intrinsic factor AC and / or anti-parietal cells		27	62 %	

Table no3: The most frequently etiology of vitamin B12 deficiency found in our series was Biermer's disease (29% of cases) followed by vitamin B12 deficiency with normal Schilling test.

Table no 3: Etiological data

Etiology	**Number of patients**	**percentage**
Total of patients	43	100 %
Biermer's disease	29	67 %
Vitamin B12 deficiency with normal Schilling test	09	21 %
Subtotal gastrectomy not supplemented	3	7 %
Unknown etiology	2	5 %

Table no4: Evolution data

Evolution	Number of patients	percentage
Total of patients	43	100 %
Full recovery	30	70 %
Partial recovery	13	30%

IV. Discussion

The demographic data in our series, concerning the average age and the sex ratio, join the literature data (4 and 7). Hematological involvement was not known before the onset of neurological impairment with all patients, but it was found at the time of diagnosis in the majority of cases (86%) and the neurological impairment was inaugural only in 13% of cases; which is consistent with the literature data (6) and should encourage us to evoke a vitamin B12 deficiency in front of an evocative neurological impairment, even in the absence of hematological involvement.

The diagnostic delay in our series was relatively short compared to the literature data (4 and 7). The nature of the neurological signs found clinically in our patients was consistent with the literature data. However, the use of investigations, in particular neurophysiological investigations allows to highlight a higher frequency of certain presentations and their overlaps (8, 13, 9, 12); which is also consistent with the literature data and underlines the interest of para-clinical explorations before any suspicion of a vitamin B12 deficiency to screen for sub-clinical damage, especially in the absence of hematological anomalies and in case of normal vitamin B12 levels (10).

Neurological recovery is partial in 34% of patients; which is most likely linked to the diagnostic and therapeutic delays which are relatively long with some of our patients. Nevertheless, the favorable evolution, in the majority of our cases, was guaranteed by the diagnostic earliness.

V. Conclusion

Our study shows the clinical polymorphism of neurological manifestations of vitamin B12 deficiency. It underlines the interest of complementary investigations, in particular neurophysiological, in the diagnosis especially in case of normality of the hemogram and the dosage of vitamin B12. The results, obtained in our patients, should prompt us to early diagnosis and treat to improve the prognosis of this pathology; vitamin B12 therapy should be recommended in any patient with neurological or psychiatric impairment whose cause has not been identified.

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