Biermer’s Disease at the Donka National Hospital in Guinea

—Epidemio-Clinical, Therapeutic and Evolutionary Aspect in the Internal Medicine Department

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Abstract

Introduction: Biermer’s disease is an autoimmune disease characterized by a lack of absorption of vitamin B12 in connection with the production of antibodies (A) destroying the intrinsic factor (IF) which allows the absorption of vitamin B12 (cobalamin). These clinical manifestations are polymorphic and severe in our context. The objective of this work is to identify the epidemiological-clinical, therapeutic and evolutionary characteristics of Biermer’s disease in Guinean population. 

Materials and methods: This was a retrospective of patient files followed for Biermer’s disease at the internal medicine department of Donka National Hospital from January 2012 to December 2021.

Results: Eight patients were included including 5 women and 3 men. The average age of the patients was 48 years old. The diagnostic delay was 3.6 years on average. All our patients had bioclinical anemia (8 cases, i.e. 100%) followed by epigastralgia in 4 cases (50%), neurological damage such as sensitive polyneuropathy in 3 cases (37.5%). Four patients had acquired melano-derma (50%). Hypovitaminosis B12 was found in 4 patients. The myelogram performed in three patients (37.5%) found medullary megaloblastosis. One patient had Hashimoto’s disease associated with Biermer’s disease in endoscopy, (FOGD) found fundica trophy on macroscopy in 4 cases (50%). Treatment consisted of B12 vitamin therapy in all cases with a favorable clinical and biological outcome.

Conclusion: Biermer’s disease remains common in Africa and is characterized at a younger age in addition to the severity of clinical and biological manifestations. The care consists of taking vitamin B12 which remains accessible in our context.
Keywords
Biermer’s Disease, Donka, Aspects, Epidemiological-Clinical, Therapeutics

1. Introduction

Biermer’s Disease (MB) or pernicious anemia in the old name is an autoimmune atrophic gastritis mainly affecting the fundus responsible for a vitamin B12 deficiency by malabsorption [1]. It represents 20 to 50% of vitamin deficiencies B12 in adults according to the series [1] [2]. The clinical manifestations are numerous and variable and can be haematological, digestive or neurological for the most part [2] [3]. This clinical polymorphism often leads to diagnostic wandering in our countries. The etiologies of vitamin B12 deficiencies are dominated by three main causes: deficiencies of intake, the syndrome of non-dissociation of vitamin B12 and these carrier proteins and MB [2] [4] [5] [6]. With early diagnosis and appropriate management, this pathology can be potentially serious [2]. Given its pathogenesis, the diagnostic criteria as well as its management, MB is experiencing more and more renewed interest [7]. In the absence of data concerning this pathology in Guinea, we are studying the epidemiological-clinical, biological, therapeutic and evolutionary aspects of MB at the internal medicine department of the Donka national hospital.

2. Material and Methods

This was a retrospective, single-center study from January 2012 to December 2021 in the internal medicine department of the National Donka Hospital on all patient files followed for MB. The inclusion criteria were defined by the presence of the following elements:
- Vitamin B12 deficiency (level below 200 ng/l)
- Presence of auto-antibodies (anti-parietal cell antibodies and/or anti-intrinsic factor)
- Medullary megaloblastosis
- Acquired melanoderma apart from other causes found

The data is listed on an operating sheet including: age, sex, pathological history, clinical manifestations, blood count, myelogram, oesogastro-duodenal fibroscopy data, vitamin B12 assay, auto-antibodies and the treatments received as well as the evolution.

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

3. Results

During the study period eight patients were included including five women and three men with a sex ratio (F/M: 1.6). The average age of patients was 48 years
with extremes ranging from 30 to 62 years. The diagnostic delay was 3.6 years on average. Vitamin deficiency is revealed by an anemic syndrome in all our patients (8 cases or 100%) associated with gastric symptomatology such as epigastralgia in 4 cases (50%), neurological damage such as sensory polyneuropathy in 3 cases (37, 5%). One patient (12.5%) had symptoms of anxiety-depressive syndrome as a psychiatric manifestation. Four patients had acquired melanoderma (50%).

On the blood count, the average hemoglobin was 4.6 g/dl (3.4 - 7.5), the average MCV is 120 fl (75 - 130). Biological analysis objectified 4 cases of pancytopenia (50%), 2 cases of bi-cytopenia (25%) and 2 cases of isolated anemia (25%). The vitamin B12 level was done in four patients (50%) and found an average level of 82.5 ng/l (70 - 150) and a folate deficiency in one patient. The myelogram performed in three patients (37.5%) found medullary megaloblastosis. The dosage of auto-antibodies is carried out in 4 patients (50%) objectified anti-parietal cell antibodies in 1 patient, anti-intrinsic factor antibodies in 3 patients. FOGD) found fundic atrophy on macroscopy in 4 cases (50%). The search for Helicobacter pylori was not done.

An autoimmune disease was associated in 1 case with Hashimoto’s type thyroiditis. The diagnosis of Biermer’s disease was retained in all our 8 cases (100%). The treatment consisted of B12 vitamin therapy by intramuscular injection in 100% of cases in the attack phase and then orally for maintenance; associated with iron in 3 cases and vitamin B9 in 1 case. Four patients were transfused from one to two bags depending on anemia tolerance. Treatment with PPI (proton pump inhibitor) was initiated in 4 patients. A normalization of the blood count and improvement of the clinical symptoms was observed between 6 and 12 months of follow-up. We did not observe any deaths at the time of the follow-up but we note three lost to follow-up.

4. Discussion

Biermer’s disease is an autoimmune disease linked to a lack of absorption of vitamin B12 (Cobalamin) due to a lack of secretion of intrinsic factor by the parietal cells of the stomach [1] [8]. It is much more reported in the literature [9] [10] [11]. During our study period, we collected eight patients with a female predominance (5 women), which matches the data in the literature (Table 1).

This disease is diagnosed at a much higher age (after 60 years) in the West, particularly in Europe and Asia [7] [12] [13]. The average age of diagnosis in our series was 48 years, therefore younger, with extremes ranging from 30 to 62 years. The onset of MB at a young age is very characteristic in blacks, as reported in the sub-Saharan series [7] [10] [14] [15].

The diagnostic delay was very long, 3 years 6 months on average in our study with diagnostic error and a risk of morbidity. This can be explained by the low rate of specialist doctors or doctors aware of this pathology and access to care.
Clinically, the manifestations of BD are very polymorphic with variable modes of revelation [2] [3] [16]. Clinical anemia or anemic syndrome is the main clinical manifestation encountered with 61 to 100% of cases depending on the series [7] [13] [15]. All our patients presented a bioclinical anemia with an anemic syndrome corroborating with the data of the literature. Melanoderma was found in half of our patients with 50% (Figure 1).

This dermatological manifestation of BD is frequently found in black subjects and is characteristic as shown by Touré and all in Tivaouane as well as Berthé and All in Thiès in Senegal with high frequencies of 80 and 100% respectively [9] [16].

Sensory neuropathy is a frequent manifestation in case of MB which can sometimes be inaugural [16] [17] [18]. This sensory neuropathy was found in 3 of our patients, i.e. 37.5% taken as a neurological manifestation of BD. The realization of an EMG (electromyogram) would have made it possible to rule out other causes of neuropathies or associated. A patient entered the disease in the form of an anxiety-depressive syndrome with a long follow-up in psychiatry before correcting the diagnosis. The neuropsychiatric manifestations of BD are classic but are rarely inaugural [19]. Mrabet et al. in Tunisia reported 2 observations in 2014 of patients entering the disease in the form of neuropsychiatric manifestations, similarly Wun Chan et al in his series of 181 patients found 8.3% of inaugural cases [13] [19]. The fundic atrophy gastritis found in our patients who benefited from an upper endoscopy is very characteristic of MB. Concerning the anemia, it was significant and concerned all our patients (100%) with an average hemoglobin level of 4.6 g/dl. It is most often macrocytic but can be normochromic or even microcytic when there is an associated iron deficiency. This significant anemia found in our series is reported in several studies, particularly African [9] [10] [11] [14] [20].

This depth of anemia is explained by the fact that it gradually sets in, allowing the body to adapt, but also problems related to access to care. The B12 hypovitaminosis found in our four patients with The dosage is classic in the case of MB and this has been shown by Diallo, Touré and Segbena [7] [9] [14]. Auto-antibodies (anti-intrinsic factor antibodies and anti-parietal cell antibodies)

Table 1. Sociodemographic and clinical characteristics of patients.

<table>
<thead>
<tr>
<th>characteristics</th>
<th>Number</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female sex</td>
<td>5</td>
<td>62.5</td>
</tr>
<tr>
<td>Male sex</td>
<td>3</td>
<td>37.5</td>
</tr>
<tr>
<td>Anemia</td>
<td>8</td>
<td>100</td>
</tr>
<tr>
<td>Epigastralgia</td>
<td>4</td>
<td>50</td>
</tr>
<tr>
<td>Paresthesia</td>
<td>3</td>
<td>37.5</td>
</tr>
<tr>
<td>Melanoderma</td>
<td>4</td>
<td>50</td>
</tr>
<tr>
<td>Depression</td>
<td>1</td>
<td>12.5</td>
</tr>
</tbody>
</table>
were sought and found in half of our patients. we have enough elements (atrophic gastritis and or melanoderma) in favor of MB or transfused before sampling for the assay of vitamin B12 (Table 2).

These antibodies are specific but may be absent in 30 to 50% of cases in certain patients, however suggestive of MB [21].

MB being an autoimmune disease, do not ignore and look for another autoimmune disease that may be associated, such as the case of one of our patients who had Hashimoto’s thyroiditis prior to MB (Table 3).

Management consisted essentially of intramuscular administration of vitamin B12 in the initial phase while taking into account the degree of thrombocytopenia in view of the risk of bleeding. A relay by the oral route was done as validated in the literature and effective [10] [22]. This allowed a gradual regression of

![Figure 1. Patient with foot melanodermia.](image)

**Table 2.** Biology caracteristic of patients.

<table>
<thead>
<tr>
<th>Biology</th>
<th>Number of patient</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia</td>
<td>8</td>
<td>100</td>
</tr>
<tr>
<td>Pancytopenia</td>
<td>4</td>
<td>50</td>
</tr>
<tr>
<td>Bicytopenia</td>
<td>2</td>
<td>25</td>
</tr>
<tr>
<td>Vitamine B12 defiency</td>
<td>4/4</td>
<td>100</td>
</tr>
<tr>
<td>Folate defiency</td>
<td>1/1</td>
<td>100</td>
</tr>
<tr>
<td>Anti Intrinsic Factor Ac</td>
<td>3/4</td>
<td>75</td>
</tr>
<tr>
<td>Ab Anti Parietal Cell</td>
<td>1/4</td>
<td>25</td>
</tr>
<tr>
<td>Medullary megaloblastosis</td>
<td>3/3</td>
<td>100</td>
</tr>
<tr>
<td>Fundal Atrophic Gastritis</td>
<td>4/4</td>
<td>100</td>
</tr>
</tbody>
</table>

**Table 3.** Distribution of patients according to medical history.

<table>
<thead>
<tr>
<th>Background</th>
<th>Number of patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hashimoto thyroidis</td>
<td>1</td>
</tr>
<tr>
<td>Diabète</td>
<td>2</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
</tr>
</tbody>
</table>
clinical and biological manifestations. No deaths observed but we deplore three lost to follow-up.

Due to the limitations of our study in terms of epidemiological (retrospective, recruitment only hospital), prospective studies involving a greater number of patients are needed.

5. Conclusion

MB is increasingly diagnosed in our countries with the awareness of health professionals to this pathology as well as the improvement of the technical platform. In Africa, it occurs at a much younger age and the clinical-biological picture is more severe. Early diagnosis and well-conducted management can limit morbidity.

Conflicts of Interest

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

References


