http://jmscr.igmpublication.org/home/
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v10i10.19

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Ischemic stroke revealing Biermer's disease about a case: diagnosis, management and evolution

Authors
Ibrahima Sory Sidibe*, Abdelkarim Shimi, Mohamed Khatouf
Anesthesia and Intensive Care Unit A1, University Teaching Hospital Hassan II, Fez Morocco. Sidi Mohammed ben Abdallah University
*Corresponding Author
Ibrahima Sory Sidibé
Anesthesia and Intensive Care Unit A1, CHU Hassan II Fez, Morocco

Abstract
We report an observation of Biermer's disease revealed by a stroke. The originality of this case lies in the discovery of Biermer's disease in an 82-year-old male admitted to the emergency room for loss of consciousness and dysarthria. Emergency MRI showed a left fronto-parietal area cortico-subcortical hypersignal T2, FLAIR and ischemic vascular diffusion. This clinico-radiological presentation in no way made it possible to evoke Biermer's disease.
Keywords: *Ischemic stroke, Vitamin B12, Biermer's disease.*

Introduction

Biermer's disease is an autoimmune pathology characterized by the presence of chronic atrophic gastritis with the presence of anti intrinsic factor antibodies and anti parietal cells responsible for a deficiency in vitamin B12 by poor absorption, resulting in a hyperhomocysteinemia[1] [2]. The latter (hyperhomocysteinemia) is recognized as a risk factor that doubles the risk of ischemic stroke [3]. In this study, we report a case of ischemic stroke revealing Biermer's disease.

Patient and observation: This was an 82-year-old patient with a history of chronic gastritis and anemia, without any cardiovascular risk factor, admitted to the emergency room for dysarthria, sudden onset cognitive impairment with a Glasgow score of 13/15 and an anemic syndrome (pallor of the integuments, dyspnea on exertion, asthenia), all developing in a context of apyrexia and deterioration of the general condition. The remainder of the clinical examination revealed hypotension at 80/40mmhg, tachycardia at 110 beats per minute. A non-injected brain MRI was performed (Figure 1) showing a left fronto-parietal area cortico-sub cortical T2 hypersignal, FLAIR and ischemic like diffusion. The biological assessment suggested megaloblastic anemia on the myelogram, the complete blood count revealed: a macrocytic anemia with a hemoglobin level of 6.5 g / dl (13 - 18g/dl), an MCV of 113 fl (80 - 100fl). Hypovitaminosis B12 at 60 pg/mL, homocysteine blood levels were not assessed, the rest of the biological exams were unremarkable. He also underwent an eso-gastro-duodenal fibroscopy which revealed atrophic gastritis, followed by an echocardiogram of the heart and supra-aortic

trunks without abnormalities. Hence the diagnosis of ischemic stroke revealing Biermer's disease. Thus the treatment consisted of a transfusion with three globular concentrates, an administration of injectable cobalamin at a rate of 1000 µg per day intramuscularly for a week, then 1000 µg per week for a month, then 1000 µg per month continuously, and aspirin 100mg per day. The outcome under treatment was favorable with correction of the dysarthria anemia and resolution of the cognitive impairment.

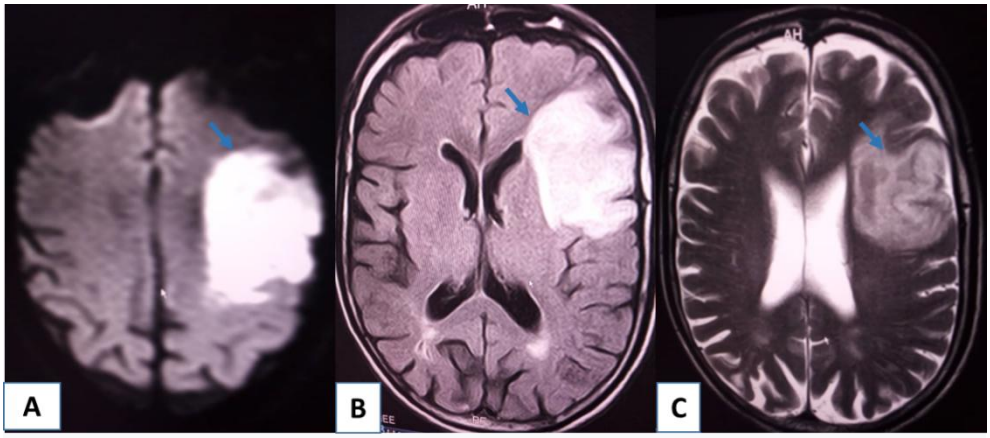

Figure 1: Cerebral MRI in axial slice in diffusion (A) FLAIR (B) and T2 sequence (C) showing left fronto-parietal range described in hypersignal Diffusion, Flair and T2 ischemic.

Discussion

The most common cause of B12 deficiency in the elderly is the inability to release cobalamin from food or transport proteins in atrophic gastritis[4]. Biermer's disease or pernicious anemia is another cause of vitamin B12 deficiency autoimmune disease with chronic atrophic gastritis and autoantibodies against intrinsic factor and against parietal cells. This leads to vitamin B12 deficiency through malabsorption[4]. Its clinical manifestations are often initially crude and they set in insidiously. Vitamin B12 deficiency very often goes unrecognized for a long time. However, the potential severity of its complications, in particular the neuropsychiatric and haematological manifestations, invites systematic research[5]. Its usual clinical signs are: megaloblastic macrocytic anemia, glossitis, digestive malabsorption, involvement of the central cerebral and medullary nervous system and the peripheral nervous system[4]. However, if the haematological abnormalities during this disease are most often modest, it appears that more than 10% of patients can present hematological manifestations which are life-threatening, including pancytopenia, severe or hemolytic anemia, and thrombotic pseudomicroangiopathy or thrombocytopenic thrombotic purpura[6]. In our patient the anemia was poorly tolerated and associated with neuropsychiatric disorders, hence the urgent diagnosis and treatment of Biermer's disease and ischemic stroke. Several studies report similar cases[1][4][7]. It has been established that this results from hyperhomocysteinemia linked to a vitamin B12 deficiency which would increase the cardiovascular risk by an atherothrombotic mechanism. Hyperhomocysteinemia is prothrombotic by interfering with the regulatory function of the endothelial cell on coagulation, by increasing platelet aggregation, by decreasing fibrinolysis, but also by acting on certain coagulation factors. This would explain the increased risk of venous thrombosis, but possibly also arterial thromboembolic disease[1].

The treatment of thrombosis secondary to vitamin B12 deficiency involves cobalamin supplementation and anticoagulation, and antiplatelet aggregation in ischemic strokes [5] [1]. Although vitamin B12, B6 or B9 supplementation decreases homocysteinemia, the effectiveness of

vitamin replacement therapy for cardiovascular disease has not yet been demonstrated, as has its effect on reducing the risk of recurrence of cerebrovascular accidents[1]. However, the following studies[1][4][7], found a complete cure of the patients taking vitamin B12.

The recommended therapeutic doses of injectable cobalamin for Biermer's disease are: 1000 mcg per day for one week, then 1000 mcg per week for one month and 1000 mcg every one to three months for life or until correction of the cause[6]. In our study, the evolution was favorable under treatment. Conclusion: Ischemic Stroke is a major complication of Biermer's disease, it is secondary to hyperhomocysteinemia. The combination of neurological and anemic syndrome should lead to vitamin and homocysteine serum dosing. Supplementation with cobalamin still retains its place in the management.

Conflict of Interest: The author and the co-authors declare no conflict of interest.

Contribution of the authors: all the authors took part in taking care of the patient in writing and correcting the manuscript. All authors have read and approved the final version of the manuscript.

Reference

1. Fall M, Gaye NM, Diop MS, Ndiaye M. Un cas de maladie de Biermer révélé par un accident vasculaire cérébral ischémique et revue de la littérature. Revintscméd 2017;19,2:149-51.
2. Loukili NH, Noel E, Blaison G, Goichot B, Kaltenbach G, Rondeau M *et al*. Données actuelles sur la maladie de Biermer: A propos d'une étude rétrospective de 49 observations. Rev médecine interne. 2004; 25(8):556-561. PubMed | Google Scholar
3. Rachid B, Zouhayr S, Chtaou N, Messouak O, Belahsen F. Accident vasculaire cérébral ischémique révélant une maladie coeliaque [Ischemic stroke revealingceliacdisease]. Pan Afr Med J. 2010;5:2. Published 2010 Apr 11. doi:10.4314/pamj.v5i1.56196
4. B. Leemann, N. Boughanem, A. Schnider. L'accident ischémique cérébral, une complication rare de la maladie de Biermer. Rev Neurol.2006; 162 : 10, 1007-10
5. Angelica Loup-Leuciuc, Pierre-Jean Loup, Tommaso Lombardi, Jacky Samson. Carence en vitamine B12 (1re partie) : mise au point. Med Buccale Chir Buccale 2011;17:211-224. DOI: 10.1051/mbcb/2011127
6. Federici L, Loukili Henoun N, Zimmer J, Affenberger S, Maloisel F, Andrès E. Manifestations hématologiques de la carence en vitamine B12: données personnelles et revue de la littérature. revmed, 2007;28(4),225-231. doi:10.1016/j.revmed.2006.10.319
7. Bougteba A., Basir A., Kissani N. Infarctus cérébral récidivant révélant une maladie de Biermer. RevNeurol 2009 ; 165(12), 1099–1102. doi:10.1016/j.neurol.2009.01.051.