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Original Research Paper

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BRAIN MALARIA: PRESENTATION OF A CASE AND REVIEW OF THE LITERATURE

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ABSTRACT Malaria is a zoonotic disease caused by protozoa of the genus Plasmodium, acquired through the bite of a female of the Anopheles mosquito genus. The initial symptoms of malaria are usually non-specific, presenting with fever, moderate to severe dehydration, tachycardia and tachypnea, with systolic blood pressure usually within normal ranges and in some cases with headache, nausea and vomiting. The clinical diagnosis can be confirmed by the presence of malarial retinopathy or the presence of parasites in at least 20% of the capillaries in the histopathological study of the brain. The drugs of choice are those derived from artemisinin, artesunate and quinine. We present a case of severe malaria with brain involvement.

KEYWORDS : Cerebral malaria, epidemiology, beta-amyloid protein.

INTRODUCTION

Malaria is a zoonotic disease caused by protozoa of the genus Plasmodium, acquired through the bite of a female of the Anopheles mosquito genus (1-3). Five species of Plasmodium are currently known to be capable of causing malaria in humans, with P. falciparum being the most frequently associated with mortality and complications, including cerebral malaria (1), a complication responsible for 90% of deaths from malaria (4).

We present a case of severe malaria with cerebral involvement and a narrative review of the literature.

Case report

A 25-year-old female patient, admitted with fever and headache, with a clinical picture of 10 days of evolution consisting of fever, headache, and vomiting that has been presenting intermittently. He comes to the emergency room due to deteriorating mental status and generalized tonic-clonic seizure. She was admitted to the emergency department stuporous, pale, jaundiced, dehydrated, with signs of respiratory distress. A complete blood count was performed showing severe normocytic anemia (4.5 gr/dL), with elevated bilirubins and LDH. Due to its origin from the jungle area, it is considered a thick smear taking evidence of Plasmodium falciparum trophozoites. A cranial CT scan revealed nonspecific changes, however, the brain magnetic resonance imaging showed diffuse and bilateral hyperintensity in the subcortical white matter (See figure 1). A

patient is considered in the context of severe malaria, starting treatment with intravenous artesunate for 3 days and then Artemether/Lumefantrine for 3 days. A thick smear is taken 7 days after starting treatment, with no evidence of parasites. A cured patient is considered, without complications at 12 months of follow-up.

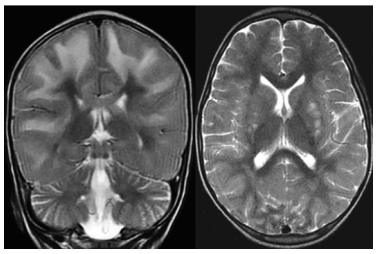

Figure 1. NMR. Diffuse and bilateral hyperintensity in the subcortical white matter, internal and external capsules, and corpus callosum; that correlates with magnetic susceptibility phenomenon in the same locations and that suggests micro-hemorrhages.

DISCUSSION

During the last two decades, a trend towards the reduction of Malaria has been observed worldwide with an approximate incidence of 2 million new cases each year (5). In 2020, 241 million cases of malaria and 602,000 deaths were estimated around the year. world, mainly in Africa(6), showing a slight increase compared to previous years, probably secondary to the disruption caused by the Covid-19 pandemic for the provision of health care services, and the implementation of public health programs aimed at to the prevention of vector-borne diseases, especially in sub-Saharan African countries (6).

In 2020, 81,363 cases of Malaria were reported in Colombia with a uniform distribution between the two species of Plasmodium circulating in the country (P. vivax 49.7%, P. falciparum 49.5%, mixed malaria 0.9%) of which 1,127 cases were recognized as complicated malaria and 23 of these presented with neurological complications. It is striking that only 10 of these cases were associated with P. falciparum infection, however, in the remaining cases no distinction was made between those with monoinfection and those that could have presented mixed malaria. In 2022, 5 cases of death from malaria were confirmed in the country, 80% of which were caused by P. falciparum (7).

Children and youth in endemic areas are at increased risk of malaria complicated by P. falciparum. Mothers in the second or third trimester of pregnancy, patients with HIV/AIDS, and those who have undergone splenectomy are also identified as high-risk populations (8).

The initial symptoms of malaria can be non-specific, presenting with fever, moderate to severe dehydration, tachycardia and tachypnea, with systolic blood pressure usually within normal ranges and in some cases with headache, nausea and vomiting (3,5). Jaundice, hepatomegaly, and splenomegaly may also be found in adults as the disease progresses. In case of suspected malaria infection due to the epidemiological link, the pathogen must be identified and treatment initiated quickly to prevent progression to severe malaria (9).

The neurological changes described in cerebral malaria include variable alterations in the state of consciousness, including superficial and deep coma. However, sometimes it is difficult to distinguish the cause of this neurological alteration between confounders such as the postictal state or the altered state of consciousness associated with medications. In these cases, cerebral malaria should be considered if the coma persists one hour after the convulsive event. In some cases, the eyes may remain open, so the determinants in the Glasgow scale for the clinical diagnosis should be the inappropriate or inferior verbal response and the motor response other than the location of the pain (5,9).

The clinical diagnosis can be confirmed by the presence of malarial retinopathy or the presence of parasites in at least 20% of the capillaries in the histopathological study of the brain (5,9,10). The lesions observed in the retina have a high correlation with the trapping of erythrocytes in the microcirculation of the central nervous system and the severity of the disease, being found in up to 41% of patients with cerebral malaria and 86% of fatal cases (11). The main findings include whitening of the retina and discoloration of the vessels (11,12), other possible findings, although not pathognomonic of cerebral malaria, include retinal hemorrhages, exudates and papilledema (12).

This entity is considered a neurological emergency and drug treatment should be started immediately. In an endemic area, treatment should be started without waiting for confirmation of the diagnosis. Treatment includes specific antimalarial therapy, supportive therapy for multiorgan dysfunction, and management of associated complications (13). The drugs of choice are those derived from artemisinin, artesunate and quinine. The latter, with a narrow therapeutic window and is only active against parasites in the last stages of the erythrocyte cycle, increases the probability of hypoglycemia by promoting insulin secretion, can cause QT interval prolongation and its rapid administration can generate hypotension, for which the patient should be monitored during its administration. Artesunate is the drug of choice because it is associated with greater efficacy, since it acts in all phases of the parasitic cycle and is associated with a 34.7% reduction in mortality compared to Quinine (14).

Cerebral malaria, without treatment, is inevitably fatal, but even with treatment the mortality rate is 15-20%, in some references up to 50%, most deaths occur within the first 24 hours of admission (15). There are 3 independent factors of treatment: advanced age, presence of coma and elevated parasitaemia. The prognosis also depends on the management of complications such as renal failure, severe jaundice, and metabolic acidosis. Metabolic acidosis is considered the best predictor of mortality in severe malaria (10,15).

In our case, the patient had brain involvement due to Plasmodium falciparum, with successful treatment.

Ethical standards and patient consent

We declare that the patient described in this study gave informed consent prior to inclusion in this study.

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