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Case Report

Thalassemia; a Protective Factor against Severe Malaria: A Report of a Case and Review of Literature

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Abstract

The severity of malaria depends on factors some of which are well known. They allow a better understanding of the conditions of the risk of severe, and adopt the terms of surveillance and prevention. We report a case of *Plasmodium falciparum* polycythemia protected against deadly cerebral malaria.

Introduction

Malaria is a major public health problem in tropical areas. It was originally, in 2013, to 584 000 deaths worldwide according to WHO [1]. Several *Plasmodium* species are responsible for human malaria; *Falciparum Plasmodium* (FP) is responsible for the vast majority of deaths. It is likely that mutations in the genes of α and β globin in thalassemic be a protective factor against the FP [2,3].

Observation

A man of 30, thalassemia known since childhood, during his stay in the Democratic Republic of Congo (DRC), at tested fever and abdominal pain. Clinical examination on admission find the patient is stable hemodynamically, fever to 40 degrees. The abdomen is painful, soft to the touch without the patosplenomegaly associated, are normally colored urine. Furthermore, the patient confirmed that he deliberately did not take chemoprophylaxis.

Biological examinations was objectified with hypochromic microcyticanemia (hemoglobin at 12 g / dl, MCV = 60.4 fL; MCHC = 30.1 g / dL); a wafer rate 201.103 /μl ; polycythemia with a rate of Red blood cells 85.8 H. glucose 0.74 g / dl; and CRP at 6 and creatinine at 12 g / l.

The blood smear made proved positive and thick films which objectified the presence of *Plasmodium falciparum* with a parasitemia of 4%.

The patient received anti-parasite treatment with 04 tablets of artemether / lumefantrine (20/120) single dose repeated eight hours apart once and then 04 tablets every 12 hours for a three-day treatment period.

A control count performed 24 hours after objectified a fall in hemoglobin 10.9 g / dL; a MCV at 59.4 fL; a rate of MCHC at 31.7 g / dL and a drop in platelet levels with thrombocytopenia at 86.103 /μl.

The outcome was favorable with disappearance of fever and abdominal pain in the third day of treatment. A blood smear and a thick drop test conducted in the fourth day did not show parasitaemia.

Discussion

The DRC is a region of central Africa where malaria is endemic in FP representing the leading cause of population mortality. In terms of chloroquine, the country is ranked area 3 [4-7]. Any stay in malarious area imposes chemoprophylaxis whose therapeutic choice depends on the *Plasmodium* strains present resistance profile in the region.

This antimalarial chemoprophylaxis should be reinforced by means of vector Prevention (body insecticide, mosquito net, long clothes, etc.). It is recommended that a limited number of medicines available in the long termantimalarial chemoprophylaxis. In 2001, doxycycline, mefloquine and atovaquoneproguanil Association were recommended by the World Health Organization (WHO) for chemoprophylaxis Long antimalarial chloroquine in areas [4,5,8,9].

The cases reported thalassemia is known since childhood who voluntarily did not take chemoprophylaxis during his stay in malarious area classified Zone 3 and presented a malaria

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parasitaemia with FP to 4 %; the strain involved in the vast majority of deaths. After transmission by a female Anopheles mosquito (the vector), the parasites migrate to the liver, hepatocytes envahient and proliferate. At the end of maturation, the hepatocyte membrane ruptures, releasing the parasites into the bloodstream. Parasites then invadered blood cells, where they proliferate. The rupture of infected erythrocytes causes fever, leading to a so-called single clinical form. Complications can arise, often associated with the cytoadherence of infected red blood cells in the microvessels in the deep organs. Blood stages are responsible for simple clinical forms (fever) and severe (severe anemia, cerebral malaria and severe respiratory distress).

During the blood invasion by the parasite stage, there is destruction of almost one third to one half of the red blood cells which can cause severe anemia below 5 g/dl which proves fatal during a severe illness [10].

An initially high rate of red blood cells in thalassemic prevents a severe drop in hemoglobin at the end of crisis. Indeed, the team Pr Karen *et al.* recently showed that polycythemia among thalassemia is the protective mechanism against a pernicious access to FP [10].

Thus, the simplest form of access malaria qui'a presented our patient without taking prior chemoprophylaxis in high-risk area; the conservation of its hemoglobin levels at admission and its moderate fall the second day of its access cannot be explained by the protective nature of its initial polycythemia its thalassemia.

Conclusion

The severity of malaria depends on the characteristics of the parasite; Topicnoisy and the environment that influences their relationships. A better study of these three partners will facilitate the recognition of those at high risk and will better ensure their protection; their supervision and the terms of their treatments.

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