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Case Report

Hyperreactive Malarial Splenomegaly: One Clinical-Case Splenomegalie Palustre Hyperimmune: A Propos D'un Cas

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Abstract

Introduction: The hyperimmune malarial splenomegaly is the tropical idiopathic splenomegaly (Charmot, 1959) and tropical splenomegaly syndrome (Pitney, 1968). It is linked to aberrant hyperimmune reaction to repeated malaria infections and prolonged. This article is aimed at reviewing the clinical aspects of HMS.

Observation: Here is a case of patient aged 43, Congolese, who was seen in consultation for splenomegaly. This splenomegaly was diagnosed several months before, fortuitously, on the occasion of an assessment for arthralgia and lumbago. The search of hamatozoon by sanguine smear-thick drop was positive with a parasitemy lesser than 1%. The patient has benefited of curative antimalarial treatment. After three months of treatment, splenomegaly was then measuring 9cm at ultrasound. 3 months later, the spleen was just palpable.

Conclusion: The HMS is the phase state of a specific hyper responsiveness syndrome of malaria infection. It follows the iterative Plasmodium infections for several years. This diagnosis should be evoked in patients who have lived for several years in a malaria-endemic country and present themselves with splenomegaly associated with a sharp increase in IgM. After usual antimalarial curative treatment, splenomegaly regressed in several months.

Introduction

Hyper-Reactive Malarial Splenomegaly (HMS) is the main cause of major splenomegaly in endemic malarial countries [1,2]. It is characterized by one remarkable excessive immune reaction, by hyper production of IgM antibodies, following multi-recurrent malarial infections. This article is aimed at reviewing the clinical aspects of HMS.

Observation

Here is a case of Patient aged 43, Congolese, who was seen in consultation for splenomegaly. This splenomegaly was diagnosed several months before, fortuitously, on the occasion of an assessment for arthralgia and lumbago. The patient was in good general condition, afebrile and without paleness and without jaundice. Splenomegaly was sensitive, measuring 18cm by ultrasound and associated with a discreet hepatomegaly. Biological examinations showed an hemoglobin at 13g/100ml (MCV: 84m3), leukocytes at 7, 5 G/l, neutrophiles at 70%, eosinophiles at 1%, lymphocytesat25%, monocytes at 45, and platelets at 162G/l. C-reactive protein at 65mg/l. The search of hamatozoon by sanguine smear- thick drop was positive with a parasitemy lesser than 1 %.

The patient has benefited of curative antimalarial treatment by Mefloquine (1,5g/day during one day), followed by Chloroquine (100mg/ a day during 30 days) and Doxycycline (100mg/day during 30days). After three months of treatment, splenomegaly was then measuring 9cm at ultrasound, and 3 months later, the spleen was just palpable.

Discussion

HMS is a clinical entity which explains itself by an excessive immune and specific reaction of plasmodium antigens; hence, we find a high rate of IgM which precedes, since several years ago, the apparition of Splenomegaly.

Description of HMS presents the patient as a young adult, living in an endemic malarial area since many years. Clinical major sign is voluminous splenomegaly which persists year after year. Splenomegaly can be held responsible for painful phenomenon as it is in our observation. With the development of major splenomegaly, occurs alteration of general state occurs, which can be punctuated by recurrent episodes of prostration, susceptible to last several weeks [1].

Spontaneous evolution can be enameled by acute hemolysis episodes that may account for the prostration of episodes and by frequent and often severe bacterial infections. Under appropriate antimalarial treatment, splenomegalia regresses in several months whereas immune abnormalities do that in a slow way as it appears in our observation.

Empirically, as it was the case in our observation, the duration of antimalarial treatment was guided by splenic volume, leading to treatment for several months. Prolonged use of Chloroquine was recommended on the basis of immune-modulatory effects of this molecule [3]. In malaria-endemic area, as splenomegaly is appearing in malaria-endemic area, during re-expositions, antimalarial maintenance therapy is poorly codified. Antimalarial treatment in the long term is unrealistic; malaria treatment courses could be suggested systematically, in plasmodium mosquito transmission period, like what is proposed in pregnant women.

Conclusion

The HMS is the phase state of a specific hyper responsiveness syndrome of malaria infection. It follows the iterative Plasmodium infections for several years. This diagnosis should be evoked in patients who have lived for several years in a malaria-endemic country and present themselves with splenomegaly associated with a sharp increase in IgM. After usual antimalarial curative treatment, splenomegaly regressed in several months.

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