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Case Report

Internal Medicine Section

Haematemesis: An Uncommon Presenting Symptom of Plasmodium Falciparum Malaria

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ABSTRACT

Plasmodium falciparum malaria with complications is a challenge for the clinicians worldwide, especially when it presents with rare manifestations. Haematological abnormalities and coagulopathy are the well documented complications in malaria. Rarely does malaria present with bleeding. We are reporting a 20 years old man who presented with haematemesis as the presenting symptom of falciparum malaria. In the review of literature, there are reports of haematemesis in malaria, but none of it as a presenting symptom.

Key Words: Plasmodium falciparum malaria, Haematemesis, Coagulopathy, Thrombocytopaenia

INTRODUCTION

Malaria, caused by the Plasmodium falciparum is known to develop fatal complications [1-3]. Haematological abnormalities and coagulopathy are the common complications which occur in malaria. Very rarely does malaria present with overt bleeding. In a 20 years old otherwise healthy male, whom we are reporting, haematemesis was an uncommon presenting symptom of Plasmodium falciparum malaria. The pathogenesis of bleeding in malaria is believed to result from platelet dysfunction and/ or coagulopathy. In endemic areas, a high index of suspicion helps in identifying and treating the most difficult cases of malaria and its complications, in spite of its wide presenting manifestations.

CASE REPORT

A 20 years old male with a history of fever (with chills and rigors), bodyache and headache of 3 days; presented to the Medical Outpatients Department with a bout of haematemesis (200 ml). He had taken paracetamol for a day, for his symptoms. There was no past history of upper gastrointestinal symptoms, bleeding tendencies or overt bleeding episodes. He was stable haemodynamically, with no pallor or jaundice. Except for a tender hepato-splenomegaly, his clinical examination was normal. There were no clinical markers of bleeding tendencies or coagulopathy. On investigations; his haemoglobin was 15.2 gm% with a PCV of 43%. His WBC counts were 6500cells/mm^3, with 76 % neutrophils and his platelets were 60,000/mm^3. His peripheral blood smear examination for malaria identified Plasmodium falciparum (++). The bleeding, clotting and prothrombin times; and the renal and liver biochemistries were normal. Blood culture, Widal test; and IgM for Dengue fever and Leptospirosis were negative. His chest X-Ray was normal; abdominal sonology showed mild hepato-splenomegaly and portal vein diameter was 11mm with no evidence of thrombosis.

He was carefully monitored for haemodynamic instability and was successfully managed with Artemesinin Combination Therapy (ACT) [4] and supportive drugs. He did not require fractionated blood products in the management; and he had an uneventful recovery without further bleeding episodes.

DISCUSSION

India accounts for approximately two thirds of the confirmed cases of malaria which are reported from south-east Asia. In spite of the advances in medical care, malaria is still associated with high mortality, especially among the underprivileged in the tropical and subtropical nations.

The P. falciparum infections are known to develop fatal complications as compared to the other forms of malaria [2]. The haematological abnormalities [5-11] such as anaemia, thrombocytopaenia, coagulopathy and disseminated intravascular coagulation are the common complications which occur in the P falciparum infections. Anaemia is the commonest among the haematologic abnormalities encountered in infections with P falciparum malaria. Our patient had thrombocytopenia normal haemoglobin and coagulation parameters, but he had thrombocytopenia.

Even though the complicated P falciparum infections develop bleeding manifestations, very rarely do they present with overt bleeding [5-7]. Kochhar R et al., [5] reported two cases of malaria from India which had gastrointestinal bleeding. Many clinical observational studies have reported active bleeding in the P falciparum infections. Our patient had about of haemetemesis prior to his admission and he did not have further bleeding episodes.

Most of the literature reviews have suggested platelet dysfunction and/or coagulopathy to be the pathogenesis for bleeding in falciparum malaria [8-11]. Srichaikul T et al., [12] who studied the platelet functions in malaria, correlated the suppression of platelet aggregation and thrombocytopaenia to cause the bleeding. Mohanty D et al., inferred that fibrinolysis and the monocyte derived coagulant activity contributed to coagulopathy in acute malaria [13]. Prasad R et al., [14] studied the coagulation status and the platelet functions in 40 children with severe falciparum malaria and they observed bleeding among 6 patients. They observed reduced platelet aggregation in majority of the patients with low platelet factor-3 availability. The liver dependent coagulation factors V, VII, and IX were the most sensitive parameters in the expression of the coagulation defects and these were affected in malaria, with liver involvement [10].

In the literature review, no specific upper gastrointestinal endoscopic features which occurred in malaria, have been described. We believe that the gastric erosions or tears which were caused by retching were the sources and that thrombocytopaenia with dysfunctional platelets could have been the cause of haematemesis in our patient. Our patient did not develop any bleeding after his admission to the hospital. He was given Artemesinin Combination Therapy and he did not require fractionated blood products in the management [4]. In endemic areas, a high index of suspicion identifies and treats the most difficult cases of malaria and its complications, in spite of its wide presenting manifestations.

CONCLUSION

Early uncomplicated malaria is an easily and completely treatable disease and an appropriate management can prevent its complications and thereby, the cost of the therapy. But, complicated falciparum malaria is a challenge for the clinicians worldwide, especially when it presents with rare manifestations.

REFERENCES

[1] White NJ, Breman JG. Malaria. In: Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL. (eds.) Harrison's Principles of Internal Medicine. 17th ed. 2008; 203:1280-93.

[2] World Health Organisation. World malaria report 2010. http://www.who.int/malaria/world_malaria_report_2010/world_malaria_report_2010.pdf.

[3] World Health Organization. The guidelines for the treatment of malaria. 2nd ed. Geneva: WHO; 2010.

[4] Kochhar R, Goenka MK, Mehta S, Mehta SK. Gastrointestinal bleeding in malaria. *Indian J Gastroenterol.* 1990; 9: 295-96.

[5] Corne P, Landreau L, Moulaire V, Jonquet O. Intra-alveolar hemorrhage during Plasmodium falciparum malarial crisis. *Presse Med.* 2001; 30: 1499.

[6] Gall C., Spuler A., Fraunberger P. Subarachnoid hemorrhage in a patient with cerebral malaria. *N Engl J Med.* 1999; 341: 611-13.

[7] Kueh YK, Yeo KL. Haematological alterations in acute malaria. *Scand J Haematol.* 1982; 29: 147-52.

[8] Pukrittaykamme S, White NJ, Clemens Chiltamas S, Karges HE, Desakamll. Activation of the coagulation Cascade in falciparum malaria. *Trans Royal Soc Trop Med Hyg.* 1989; 83: 762-66.

[9] Sharma SK, Das RK, Das BK, Das PK. Haematological and coagulation profile in acute falciparum malaria. *J Assoc Physicians India.* 1992; 40: 581-83.

[10] Rojanasthien S, Surakamolleart V, Boonpucknavig S, Isarangkura P. Hematological and coagulation studies in malaria. *J Med Assoc Thai.* 1992; 75:190-94.

[11] SrichaiKul T. Hemostatic alterations in malaria. South East Asian, *J Trop Med Public Health.* 1993; 24: 86-91.

[12] Srichaikul T, Pulket C, Sirisatepisarn T, Prayoonwiwat W. Platelet dysfunction in malaria. *Southeast Asian J Trop Med Public Health*. 1988; 19:225-33.

[13] Mohanty D, Ghosh K, Nandwani SK Fibrinolysis, inhibitors of blood coagulation, and monocyte derived coagulant activity in acute malaria. *Am J Hematol*. 1997; 54: 23–29.

[14] Prasad R, Das BK, Pengoria R, Mishra OP, Shukla J, Singh TB. Coagulation status and platelet functions in children with severe falciparum malaria and their correlation of outcome. *J Trop Pediatr.* 2009; 55: 374-78.

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