Varied Presentation of Complicated Falciparum Malaria in a Family

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Plasmodium falciparum is known for complications with a very high mortality. We report three cases in children of the same family, two of them developed ARDS, one of them died, the third child developed hemophagocytosis and one of them also had transient myocarditis, all unusual complications of falciparum malaria.

Key words: *ARDS, Child, Falciparum, Haemophagocytosis, Myocarditis*.

Malaria due to *Plasmodium falciparum* is responsible for significant morbidity and mortality amongst nonimmune patients. ARDS may develop as a severe complication of malaria and has a high mortality rate (80%) [1,2]. Hemophagocytosis characterized by proliferation of macrophages that exhibit phagocytosis of haemopoeitic elements is commonly associated with viral infections but rarely with malaria [3]. We describe 3 patients with complicated falciparum malaria from a single family.

CASE REPORT

The *first child* was a 2 year old male, who developed fever and vomiting since 2 days followed by sudden onset breathlessness. Peripheral smear was positive for *P.falciparum* with a parasite index of 80%. The chest *X*-ray showed bilateral fluffy infiltrates and the child was unable to maintain saturation even with 60% FiO_2. The child was intubated and put on ventilator but died within a few hours due to respiratory failure. Following the death of the first child, the family was referred to us for further management.

The *second child* was a 10 year old girl brought with complaints of fever with chills and vomiting of 7 days, respiratory distress, generalized edema and jaundice of 3 days. Peripheral smear was positive for *Plasmodium falciparum*. Vitals were suggestive of hypotensive shock, there was decreased air entry bilaterally and bilateral crepitations were present; along with hepatosplenomegaly. Investigations revealed 7.2g/dL hemoglobin, platelet-35000 mm^3, total bilirubin 8.8 mg/dL (direct 7.2 mg/dL). Child was not maintaining saturation with FiO_2 0f 60% and ABG was pH-7.23, pCO_2 67.6, pO_2 64.4, HCO_3 – 27.3, SO_2 88%. Chest roentgenogram revealed bilateral fluffy infiltrates. With bilateral pulmonary infiltrates, inability to maintain saturations with a very high FiO_2 and normal cardiac function on admission, a diagnosis of ARDS was made. The child was intubated and ventilated on volume control mode with low tidal volumes and high PEEP. Intravenous artesunate was continued and circulatory support was given with dopamine. In view of persistent tachycardia, a 2D-echocardiography was done, which was suggestive of myocardial dysfunction with serial ejection fractions of 45%, 35% and 25%, which gradually improved to 55% with fluid restriction and ACE inhibitors. As the child improved, settings were reduced and child was extubated. As soon as the child stabilized, she was administered a combination of artmether and halofantrine orally for 3 days.

The *third child* was a 12 year old girl brought with complaints of weakness and fatiguability of 6 days and fever of 3 days. The peripheral smear was positive for *P. falciparum*. Pallor, icterus and hepatosplenomegaly were present. There was no respiratory distress and the chest radiograph was normal. On admission her hemoglobin was 7.4 g/dL, platelet count was 46000mm^3, and total bilirubin was 4 mg/dL. She was started on intravenous artesunate and a packed cell transfusion was given. She improved over a period of 4 days after which there was a sudden drop of hemoglobin and platelet count. Fever spikes reappeared and were present every day. Blood culture was negative and ultrasound of the abdomen was normal. She was administered artemether plus halofantrine for 3 days followed by mefloquine since her fever persisted and her hemoglobin kept dropping. Bone marrow aspiration was suggestive of hemophagocytosis. Serum ferritin was 2136 ng/dL, serum triglyceride levels were 484 mg/dL and G6PD was normal. Investigations for other etiologies of hemophagocytosis such as EBV and HIV were negative. Child improved spontaneously with supportive treatment and was discharged with normal hemoglobin and platelet counts.

DISCUSSION

ARDS is an uncommon complication in malaria but

carries a high mortality rate [4]. There is no precise data regarding the prevalence of ARDS during malaria infection; however, it is predicted nearly 20-30% of malaria patients admitted to ICU develop ARDS [5]. Proposed mechanism of development of ARDS is pulmonary vasculature dysfunction secondary to liberation of inflammatory mediators which increase vascular permeability, and parasitized RBCs' sequestration cause injury. Clinically, patients developing sudden onset tachypnea and dyspnea. Life threatening hypoxemia may develop within a few hours. Two of our patients developed ARDS, one died and other required mechanical ventilation.

Hemophagocytosis is associated with malignant, genetic, and autoimmune diseases. Viral infections as a cause are mainly limited to EBV infection. Malaria is a very rare cause and the mechanism of hemophagocytosis in malaria is unknown [3]. High levels of cytokines have been reported in malaria patients with hemophagocytosis which resolves soon after successful treatment of malaria [6-9]. Prolonged hemophagocytosis, has not been reported in patients with falciparum malaria. Once the cytokine cascade is triggered, hemophagocytosis may continue independent of the presence of the malarial parasite.

Thus, we had a family of three children, all with falciparum malaria with three unusual complications occurring in the same family.

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REFERENCES

1. Loser H, Schmid K, Wilfing A, Winkler S, Staudinger T, Kletzmayr J, *et al*. Experiences with severe *P. falciparum* malaria in the intensive care unit. Intensive Care Med. 2000;26:195-201.
2. Gachot B, Wolff M, Nissack G, Veber B, Vachon F. Acute lung injury complicating imported *Plasmodium falciparum* malaria. Chest. 1995;108:746-9.
3. Hamada A, Watanabe N, Tanaka H, Kobayashi A. Falciparum malaria with bone marrow abnormalities resembling malignant histiocytosis. Trans R Soc Trop Med Hyg. 1989;83:331.
4. Rowe AK, Rowe SY, Snow RW. The burden of malaria mortality among African children in the year 2000. Int J Epidemiol. 2006;35:691-704.
5. Taylor WR, Cañon V, White NJ. Pulmonary manifestations of malaria: recognition and management. Treat Respir Med. 2006;5:419-28.
6. Ohno T, Shirasaka A, Sugiyamma T, Furukawa H. Hemophagocytic syndrome induced by Plasmodium falciparum malaria infection. Int J Hematol. 1996;64:263-6.
7. Sermet-Gaudelus I, Abadie V, Stambouli F, Hennequin C, Lenoir G, Gendrel D. Haemophagocytic syndrome in Plasmodium falciparum malaria. Acta Pediatr. 2000;89:368-9.
8. Aouba A, Noguera ME, Clauvel JP, Quint L. Hemophagocytic syndrome associated with plasmodium vivax infection. Br J Haematol. 2000;103:832-3.
9. Zvulunov A, Tamary H, Gal N. Pancytopenia resulting from hemophagocytosis in malaria. Pediatr Infect Dis J. 2002;21:1086-8.

Acute Myeloid Leukemia Presenting as Obstructive Jaundice

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Jaundice as a presenting feature of pediatric acute myeloid leukemia is rare. We report two cases of AML who presented with obstructive jaundice, one with a malignant stricture at the common bile duct and other with a granulocytic sarcoma obstructing the bile duct. The prognosis is poor in these patients.

Key words: *Acute myeloid leukemia, Granulocytic sarcoma, obstructive jaundice.*

Obstructive jaundice as the presenting feature of acute myeloid leukemia (AML) is rare in children. It may be due to a stricture of the biliary tree or a granulocytic sarcoma compressing the biliary tree. We report two such cases.

CASE REPORT

Case 1: A one year old female child presented to us with pancytopenia (hemoglobin 4.5g/dL, WBC count 2100/mm^3, platelet count 13,000/mm^3). A thorough evaluation