Varied Presentation of Complicated Falciparum Malaria in a Family

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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 haemopoetic 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 Plasmodium 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% FiO2. 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 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% FiO2. 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 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 Plasmodium 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 46000 mm3, 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 artether and 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


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.

CASE REPORT

Case 1: A one year old female child presented to us with pancytopenia (hemoglobin 4.5g/dL, WBC count 2100/mm³, platelet count 13,000/mm³). A thorough evaluation