Case Report

Severe malaria associated acute respiratory distress syndrome: a case report

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Abstract

Background: Severe malaria is known to have multisystemic effects involving the brain, the kidneys, and occasionally the lungs, causing acute lung injury and, a part of the spectrum, acute respiratory distress syndrome.
Aim: To report a case of severe malaria associated with acute respiratory distress syndrome in a Nigerian teenager.
Methods: Electronic medical records at the emergency unit, the female ward, and the intensive care unit provided all the details about the patient's clinical course for this report.
Case Report: A 15-year-old girl presented to the A&E with a fever and vague abdominal pain. Malaria parasite test was negative. Urinary tract infection was suspected, empirical antibiotics were initiated, with little relief. The fever persisted and a repeat malaria test on the second day was positive. Oral antimalarial medication was administered on an outpatient basis. She represented later the same day with fever and respiratory distress. Laboratory test showed thrombocytopaenia. Chest radiograph showed bilateral lung infiltrates. Antibiotics were continued, and oxygen supplementation with no relief. The next day, dyspnoea worsened, with severe hypoxaemia. This necessitated intensive care unit admission. Non-invasive mechanical ventilation (continuous positive airway pressure ventilation) and parenteral artesunate were initiated and given for four days with full recovery.
Conclusion: Severe malaria from plasmodium falciparum with associated acute respiratory distress syndrome (ARDS) can occur with high morbidity and mortality. This necessitates prompt recognition, intervention, and early intensive care unit admission, initiation of continuous positive airway pressure ventilation (CPAP), and intravenous antimalarial treatment for a good outcome.

Keywords: Severe, malaria, acute, respiratory, distress, syndrome, adolescent

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INTRODUCTION

Malaria is endemic in Nigeria, with a heavy public health burden causing thousands of deaths each year.[1] Although there are several species of plasmodium causing the infestation, plasmodium falciparum species is known to cause severe disease, especially in the nonimmune, young, and pregnant women.[1-3] The systemic effects of severe malaria have been reported to affect the kidneys (AKI), the brain (cerebral malaria), and the lungs, leading to acute lung injury and the worst spectrum, acute respiratory distress syndrome (ARDS) with high mortality.[2-4] The pathogenesis of ARDS-associated malaria is linked to the activation of proinflammatory cytokines, leucocyte subclasses etc, to sequestrated infected red cells in the pulmonary capillaries, resulting in damage to the alveolar-capillary barrier to cause an influx of pulmonary exudates (infiltrates) and impairing gaseous exchange causing dyspnoea, tachypnoea, hypoxaemia and on radiography, bilateral pulmonary infiltrates. The management of acute respiratory distress syndrome includes early recognition of critical illness, early initiation of mechanical ventilation, CPAP and early initiation of antimalarial medication. [2-4]

We report the case of severe malaria with respiratory complication (diagnosis was made from the history, ancillary laboratory investigation and a positive malaria parasite test) in a 15-year-old girl with sudden respiratory distress, bilateral homogenous pulmonary opacity, hypoxaemia, severe thrombocytopaenia managed with antimalarial injections and ventilatory support with good outcome.

CASE REPORT

A 15-year-old girl presented to the paediatric outpatient unit with complaints of nonspecific abdominal pain, which initially started around the umbilicus and later became generalized. This was associated with high grade fever with a temperature of 39 degrees centigrade. There was no associated history of nausea or vomiting and no respiratory symptoms. The initial malaria test was negative, abdominal ultrasonography did not reveal any significant finding and urinalysis was normal. She was managed as a case of urinary tract infection with empirical antibiotics while awaiting the urine culture result which later was negative. The chest radiograph at presentation was a normal study (Figure 1). The patient was discharged home but the fever persisted. Blood culture did not yield any significant result. Two days after the presentation, she tested positive to plasmodium falciparum by rapid diagnostic test and optical microscopy. The parasite density was about 10,000parasites/µml, and her genotype was AA. She was commenced on oral antimalarial with some relief. A day after the commencement of antimalarial drug she developed sudden dyspnoea with oxygen saturation of 70- 88% on room air. The respiratory rate was 36 breaths/ min. There was no prior history of vomiting, no cough or catarrh to suggest an initial respiratory problem. Oxygen was administered with nasal prongs and later replaced with a non-rebreather mask at a flow rate of 10l/min with little effect, oxygen saturation improved occasionally to 90% and she was very dyspneic. Respiratory rate increased to 45 breaths per minute. Full blood count showed severe thrombocytopenia with a total platelet count of 58 x 10^3 cells/ µl. Total white cell count was 6.5x 10^3 cells/µl with neutrophil count of 67%. Chest radiograph showed bilateral lung infiltrates (Figure 2 and 3). In spite of the use of antibiotics for about four days, the fever persisted with CRP of 10mg/l. Clinically, she was fully conscious, calm and well oriented in time, person and place. She was immediately transferred to the intensive care unit. Non-invasive ventilation was instituted with mild sedation using fentanyl 50µg- 100µg as tolerated. Continuous positive airway pressure (CPAP) ventilation with an initial FIO_2 of 1.0 and PEEP 12cmH_20 were titrated. Range of PEEP was 12-15cmH_20, to generate $SP0_2$ to a minimum of 95%. Fentanyl injection helped to instill tolerance of the respiratory interface. She was ventilated (non-invasive) for four days, intravenous artesunate injection was continued till she was weaned off the ventilator before conversion to oral medications. Four days into the intervention, the fever stopped and she was weaned off non-invasive ventilation (NIV) and gradually recovered with no complications. Chest radiograph that was done two weeks after discharge was normal (Figure 4). The young girl was at a boarding school in Nigeria and

she visited home for a short holiday before the incident.

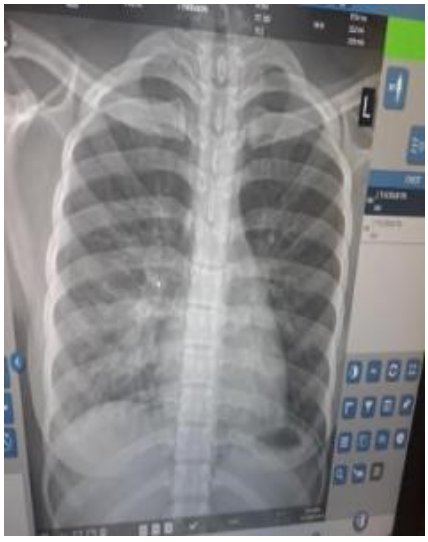

Figure 1: Chest radiograph - day 1 of admission

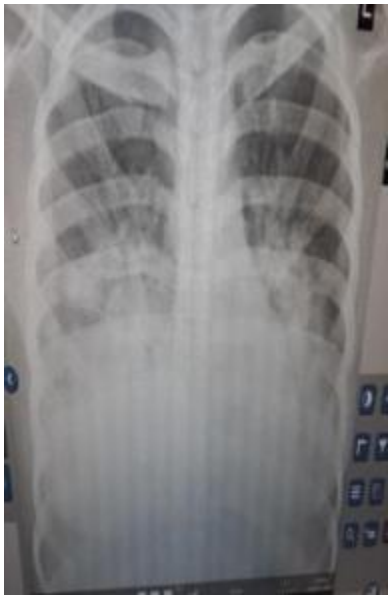

Figure 2: Chest radiograph - day 3 of admission showing bilateral infiltrates

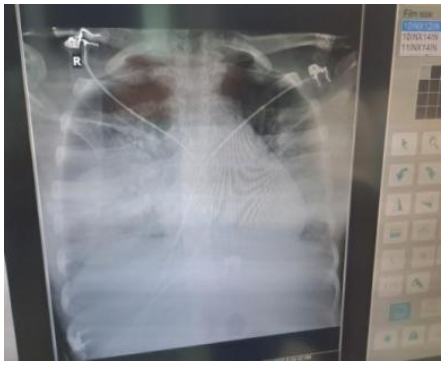

Figure 3: Chest radiograph - day 5 showing bilateral infiltrations

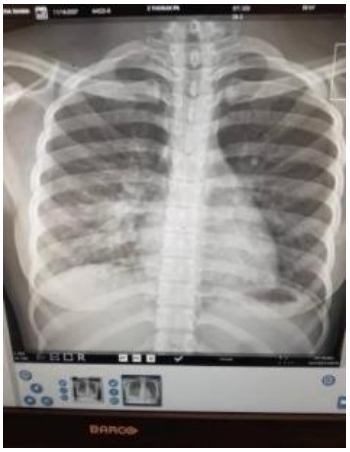

Figure 4: Chest radiograph - ten days after discharge

The parents consented to the use of the chest radiographs and other data for purposes of academic publication.

DISCUSSION

Nigeria contributes a significant share of global malaria deaths especially in the under-5 and severe malaria with its complications involving the brain or kidneys are known causes of morbidity and mortality.[1] There are reports that acute lung injury or its worst form acute respiratory distress syndrome, ARDS can be associated with severe malaria and necessitating a high index of suspicion in patients who show signs of respiratory compromise following onset of acute severe malaria.[2-4] Urban children like the index case are known to develop immunity to malaria later than semi or rural children[3] and can occasionally experience severe symptoms. The pulmonary involvement in severe malaria has long been reported and it could occur at any time in the course of the illness in addition to affecting other organs and systems and resulting in physiological deteriorations with subsequent organ-system failures which could be life threatening as experienced by this patient. The pathogenesis of malaria induced influx of plasma exudate into the interstitium and alveoli could be due to increased alveolocapillary permeability which is cytokine mediated, causing alveolar damage.[5] The resulting pulmonary oedema is non-cardiogenic, with evidence of inflammation and haemorrhages.[5,6] The epidemiology of ARDS in West Africa affects commonly

observed infections and infestations including malaria[3] with plasmodium falciparum species taking the lead in the causation of malaria associated ARDS[4] although other benign species had been reported to also induce ARDS.[7,8] The onset of respiratory distress in this child was sudden with rapid deterioration in the oxygen saturation to less than 90% with signs of respiratory distress and bilateral homogenous opacity on chest radiography (Figures 2 and 3) within two days of intensive care unit admission. This was also reported by Hoffmeister [9] even after the initiation of antimalarial treatment. Early recognition of the degree of severity and prompt diagnosis and management are key determinants for early recovery and satisfactory outcome. The initial presentation with non -specific symptoms was possible in children even with a negative laboratory result and this could enhance the progression to ARDS in a few days of presentation before the initiation of antimalarial treatment.[9] Treatment of severe malaria associated ARDS involved the use of parenteral artesunate [10,11] and early ICU admission with the initiation of continuous positive airway pressure (CPAP) using a tolerable facemask. Thrombocytopenia was observed as a surrogate of severity as other authors also reported.[3,12, 13]

CONCLUSION

Malaria is endemic in Nigeria and severe malaria associated acute respiratory distress syndrome (ARDS) can occur. It represents a serious complication of plasmodium falciparum species with high morbidity and mortality and necessitates prompt recognition, intervention. Early intensive care unit admission and the initiation of continuous positive airway pressure ventilation (CPAP) and intravenous antimalarial treatment have been found to improve outcome.

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Conflict of interest
There is no conflicts of interest

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