

# Cerebral Malaria Causing Severe Sepsis, Acute Renal Failure, and Aspiration Pneumonia: A Case Report

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## Abstract

We report a case of severe malaria (cerebral malaria) developing aspiration pneumonia and acute renal failure in a 17-year-old student after returning from malarial hyper endemic zone. The infecting organism was *Plasmodium falciparum*. Treatment with intravenous quinine and haemodialysis for kidney failure and broad spectrum antibiotic for aspiration pneumonia caused complete recovery of the patient.

**Key words:** Acute renal failure; Aspiration pneumonia; Quinine; Malaria.

## INTRODUCTION

Malaria is one of world's most important infections and although it is almost has been eradicated from temperate zone. Travelers, travelling to endemic or hyper endemic zone are more prone to develop malarial infection. Recently there is a changing trend not only in the clinical manifestations but also in the pattern of complications in malaria. Over a decade ago, cerebral malaria was the predominant manifestation of severe malaria, whereas today the combination of jaundice and renal failure are more common.<sup>1</sup> Several factors including various chemical mediators, catecholamine release, dehydration, intravascular haemolysis, intravascular coagulation, sepsis, and hyperbilirubinaemia have been implicated in the pathogenesis of acute renal failure (ARF) in malaria.<sup>2</sup>

Acute tubular necrosis is the principal pathologic mechanism in malaria induced ARF.<sup>2</sup>

In this paper we report a case of a student who travelled in a hyper endemic zone and developed cerebral malaria and aspiration pneumonia and acute renal failure complicated the infection.

## CASE REPORT

A 17-year-old student from endemic zone of Eidgah, Cox's bazar travelled to Ramu hilly hyper endemic area for khatme quran in the holy Ramadan. Just after returning from Ramu on 22.08.12 he developed high grade fever which was initially intermittent and later on became remittent, used to come with chills and rigors. There was no convincing evidence of other cause of fever. Highest recorded temperature was 102°F. He took antipyretic from local pharmacy. After 3 days, on 25.08.12 he developed jaundice. Then he was hospitalized in a local Upazilla health complex and was treated with intravenous fluid. On 27.08.12 his condition gradually deteriorated and he became semiconscious and started

vomiting. Vomitus containing digested food particles, non-bilious, occurred 3–4 times per day. There were no features of meningeal irritation. He then developed respiratory distress. In later part of day he became unconscious and was admitted in Cox's Bazar hospital and treated as severe malaria with intravenous quinine and fluid empirically and after starting loading dose of quinine he was referred for tertiary level care in Chittagong city and admitted in a clinic. In that clinic peripheral blood film and ICT for malaria was done and found *falciparum* malaria. That time he was unconscious and febrile but haemodynamically stable. Investigation revealed anaemia (Hb - 9.5 gm/dl), thrombocytopenia (45000/cu mm), plenty of parasite in blood film, hyperbilirunaemia (6.0 mg/dl), SGPT (266.5 U/L), hyponatremia (129.6 mmol/L), and bilateral consolidation on chest imaging. As respiratory distress increased he was referred to another hospital for ICU support. On 28.08.12 at 11.00 pm he was admitted at National Hospital Chittagong ICU. Here he was found unconscious (GCS - 07/15), with axillary temperature 102°F, respiratory rate 35/min, BP - 100/70 mm Hg, Pulse - 130/min. His arterial blood gas revealed high anion gap metabolic acidosis (HCO<sub>3</sub>-9.1) with compensatory respiratory alkalosis (PCO<sub>2</sub>-14) with high anion gap (24.9 mmol/L). He was incubated at 1.30 am on 29.08.12 for hypoxia and hypoxemia and put on mechanical ventilator. His urine output was noted scanty which became anuric and serum creatinine was rising (2.8 mg/dl). Haemodialysis was given on that day after evaluated by nephrologist, and repeated on next day. Anaemia was increasing, white cell count was rising (18000/cumm), and his blood platelets were (60000/cumm). Blood culture showed no growth but tracheal aspirate culture revealed growth of *Escherichia coli*. After haemodialysis, his renal function regained and pneumonia was resolved by broad spectrum antibiotic (meropenem and metronidazole) and intravenous quinine was continued till gaining conscious level. Later, he was switched to oral form. This management of sepsis and aspiration pneumonia was given under supervision of internist Boksha.<sup>2</sup> On third day of admission in ICU (31.08.12) and intravenous quinine therapy his consciousness level regained and became febrile. Renal function, serum creatinine level became near normal. He was extubated that day and referred to cabin on 02.09.12 and discharged on 06.09.12. Seven days after returning to home

his physical condition and biochemical parameters were evaluated and was found to be normal.

## DISCUSSION

Malaria is an acute and sometimes chronic infection caused by protozoan parasites of the genus *Plasmodium*. Malarial parasites undergo a sexual phase in Anopheles mosquitoes and an asexual phase in human. In the vertebrate host, release of merozoites from the ruptured hepatic schizonts causes blood stream infection, which is the clinical symptom of malaria. The attack is initiated by the synchronous rupture of erythrocytes with the release of new merozoites; therefore the recurrence of fever at 48 hourly intervals (*P. vivax* and *P. ovale*, sometimes *P. falciparum*) and at 72 hourly intervals (*P. malariae*) depends on the lifecycle of the parasite. Diagnosis is established by demonstrating parasites in thick and thin blood films. Species specific serological tests are useful for detection of organism. Malaria infections have repeatedly been reported to induce nephrotic syndrome and acute renal failure. People who are non-immune and live in a non-endemic area had a higher risk of developing acute renal failure when compared with semi-immune area. Acute renal failure is a life threatening complication of malaria infection caused by *P. falciparum*. In the majority of cases *P. falciparum* is the causative organism of acute renal failure, although malarial acute renal failure due to *P. vivax* has been occasionally reported.<sup>3</sup> Prevalence of malarial acute renal failure in endemic areas seems to be increasing.<sup>3</sup> The reported mortality of malarial acute renal failure is still high, ranging from 15% to 45%. The histological picture of malarial acute renal failure consists of a variable mixture of acute tubular necrosis, interstitial nephritis, and glomerulonephritis. In *P. falciparum* infection, malarial acute renal failure often occurs in association with signs of multi organ involvement and jaundice, anaemia and thrombocytopenia are present in more than 70% of cases.<sup>4-6</sup> In recent years, there have been an increasing number of reports favouring the existence of malarial hepatopathy from Asian countries, especially from India.<sup>7</sup> Aspiration pneumonia one of the complications in cerebral malaria feeding is started in unconscious or semiconscious patient or patient starts vomiting. This causes right sided or bilateral consolidation or abnormal chest imaging. Aspiration pneumonia causes high white cell count and tracheal aspirate or sputum culture

growth of organism. For this reason cautious starting of feeding in unconscious patient of cerebral malaria is one of the important part of management.<sup>8</sup> Anaemia in malaria is common mostly in *falciparum* malaria and white cell count rises when it is associated with infection like pneumonia which is present in this case. Thrombocytopenia is also present and platelet count may be as low as 50000/cumm and correlates with severity of infection. There may be mild rise in prothrombin time and hepatic enzymes like this case.<sup>9</sup> Quinine is still most effective drug for *falciparum* malaria. This case was also managed by quinine; loading

dose was started in primary care level hospital and maintenance dose was continued in our ICU due to the unavailability of artesunate.<sup>9</sup> Aspiration pneumonia and sepsis may also be present like this case and it was successfully managed by meropenem and metronidazole. Due to aspiration pneumonia and sepsis, respiratory distress and respiratory failure may occur and managed by endotracheal intubation and mechanical ventilator support. Renal failure may also occur in severe *falciparum* malaria which can be managed by haemodialysis and fluid management and improvement is evidence by diuretic phase of AKI.<sup>9</sup>

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