**Malarial hepatitis and renal failure: a study of two cases**

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**Abstract**

We report two cases of severe malaria who presented with non-specific clinical features. One of the cases is a 50 year old female who was brought to our hospital with high grade fever and severe abdominal pain, whereas the other case was a 20 year old male presented with high grade fever and headache. Both the cases presented with abnormal liver and renal function tests. *P. malariae* and *P. falciparum* are responsible for clinically important renal disease the former causes chronic progressive syndrome and the latter causes acute renal disease. Severe malaria may present with non specific clinical features making it difficult to distinguish from other febrile illness.

**Key words:** Plasmodium falciparum malaria, acute renal failure, fulminant hepatic failure
coat was positive and parasite index was 10%. Microscopy was positive for *Plasmodium falciparum* in the peripheral blood sample. The Ultrasonography confirmed the clinical findings of hepatosplenomegaly with no free fluid. Both chest x-ray and abdomen x-ray were normal. A diagnosis of severe falciparum malaria with renal and hepatic involvement was made. Patient received IV Ceftriaxone 2 gm BID, IV Artesunate 60 mg BID, IV Metranidazole 0.5gm TID, IV Pantoprazole 40 mg BID, IV Vitamin K 20 mg OD, Cap Doxycycline 100 mg BID and a Tablet of Primaquine 15 mg OD. The patient's condition improved and was subsequently discharged and advised to have a follow up investigation for all parameters within a week.

**Case report 2**

A 20 year old male arrived at KLES Dr Prabhakar Kore Charitable Hospital with high grade fever with chills, rigors and headache for the previous six days. On examination he was pale and jaundiced. Abdominal examination revealed splenomegaly. The following parameters were investigated and were as follows: Serum Creatinine 1.6 mg/dL, Blood Urea 75 mg/dL, Serum Sodium 133 mEq/L, Serum Potassium 4.6 mEq/L, Serum Total Bilirubin 10 mg/dL (Conjugated 8.3 mg/dL and Unconjugated 1.7 mg/dL), Serum Aspartate aminotransferase 138 IU/L, Alanine aminotransferase 97 IU/L, Total Protein 6 g/dL, Albumin 2.6 g/dL and A:G ratio 0.8. The Haematological report showed: Haemoglobin 6g/dL, Platelet count of 152 x 10³/mm3. Peripheral smear revealed normocytic hypochromic anaemia. Malaria parasite identification using quantitative buffy coat was positive. Microscopy was positive for *Plasmodium falciparum* in peripheral blood sample. A diagnosis of falciparum malaria with renal and hepatic complication was made. The Patient received IV Ceftriaxone 2 gm BID, IV Artesunate 60 mg BID, IV Metranidazole 0.5 gm TID, IV Ranitidine 50mg TID, Cap Doxycycline 100 mg BID and a Tablet of Primaquine 15 mg OD. Two pints of whole human blood was transfused to correct anaemia. The patient’s condition improved and was discharged subsequently and advised to have a follow up investigation for all parameters within a week.

**Discussion**

*P. malariae* and *P. falciparum* are responsible for clinically important renal disease. The former causes a chronic progressive syndrome and the latter cause an acute renal disease. The Incidence of Acute Renal Failure (ARF) is 1% - 4% and may be as high as 60% in high risk individuals [5]. ARF is one of the most common causes of death in severe malaria and a male preponderance has been observed in the literature [6,7]. Malarial ARF (MARF) is commonly found in non-immune adults and older children with *falciparum* malaria. It is quite common in Southeast Asia and in the Indian subcontinent where the intensity of malarial transmission is usually low, with occasional micro foci of intense transmission [8]. The pathogenesis of renal failure is uncertain. It may be related to red blood cell sequestration interfering with renal microcirculatory flow and metabolism. Clinically and pathologically, ARF manifests itself as an acute tubular necrosis [9]. MARF is most commonly associated with jaundice, seen in more than 75% of the cases [10]. Jaundice by itself is not considered as severe malaria. When serum bilirubin >3mg/dl is found, in association with other vital organ dysfunction, it indicates severe disease [4]. Jaundice is described as “biphasic” with a conjugated component resulting from cholestasis and an unconjugated component due to haemolysis [10].

Hepatitis in malaria occurs in around 20% of cases and it is characterised by elevated serum transaminases, particularly alanine aminotransferase [10]. Jaundice secondary to malarial hepatitis affects 5% to 20% of patients with severe *Plasmodium falciparum* malaria. Severe malaria due to *P. falciparum* may appear as fulminant hepatic failure (FHF), hepatomegaly with normal prothrombin time helps us to make the distinction between FHF due to malaria and viral FHF [11].

The description of the above 2 cases who presented non-specific clinical features had both renal and hepatic complications. It is thus difficult to distinguish between various febrile illnesses in a country like India where the burden of infectious disease is enormous. Severe *falciparum* malaria can mimic any of these febrile illnesses. Over the past 10-15 years, clinical presentation of severe malaria in India has slowly shifted from single to multiple complications. The Incidence of ARF and jaundice are on the rise, and development of multiple organ dysfunction results in increased mortality [8]. The measures most likely to decrease the mortality rate due to severe malaria are prevention and early diagnosis.
References


