A Rare Case: Enterococcal Septicaemia Complicating Severe Falciparum Malaria

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Abstract :

Falciparum malaria in severe cases is associated with many dreaded complications which might be even fatal. In the present case, the microcirculatory failure in intestine led to endothelial dysfunction and invasive enterococcal septic shock, even after the patient had responded to antimalarial therapy for severe malaria. Administration of a sensitive antibacterial, apart from the antimalarials, is essential for recovery from septic shock.

Keywords : Enterococcal septicaemia, Falciparum Malaria.

Introduction :

Plasmodium vivax and *Plasmodium falciparum* are two common species causing malarial infection in Asian subcontinent.[1] Severe falciparum malaria is associated with fatal complications, most common being cerebral malaria, renal failure, anemia, coagulopathy, and shock.[2] A rare complication of *P. falciparum* malaria is activation of an invasion by Enterococci, a normal inhabitant of intestinal flora, resulting in hyperpyrexia with septic shock.

Case Presentation :

A male patient aged 22 years was admitted with complaints of high grade fever for five days. On investigation, diagnosis of falciparum malaria was confirmed by *P. falciparum* grade 4 on peripheral smear examination. All other reports were normal except mild elevation of indirect bilirubin and insignificant rise in liver enzymes as shown in table 1. Patient was treated with injectable artesunate and intravenous fluid therapy for 48 hours. On third day of admission, patient suddenly developed hyperpyrexia with rigors (temp > 43° C), tachypnea, confusional state, hypotension, hypoxia and acidosis. On investigation, peripheral smear for malarial parasites was negative, but revealed polymorphoneucleocytosis with toxic granulations ,thrombocytopenia, a positive 'C' Reactive protein, mild elevation of CPK total and borderline elevation of Troponin; all parameters suggestive of bacterial infection. Blood culture was done & Enterococci were isolated; sensitive to vancomycin, linezolid and teicoplanin. Patient was treated with ventilatory support, vasopressure agents for 48 hours. Patient was also given complete antimalarial therapy as well as antimicrobial teicoplanin. As shown in the table 1, patient had recovered clinically as well as in terms of laboratory parameters and discharged from hospital on the 10th day of admission.

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Discussion :

Falciparum malaria is a mosquito transmitted protozoan disease associated with a spectrum of clinical manifestations with mild febrile illness on one end and complicated multiorgan dysfunction on the other; that may ultimately prove fatal. Patient with severe disease may develop sudden hypotension and shock known as 'Algid Malaria'.[3] The patient in this case was treated with antimalarial parenteral therapy as recommonded by WHO guideline for severe falciparum malaria and subsequently, peripheral smear turned negative for *P. falciparum*. On 3rd day of admission, patient developed sudden onset hyperpyrexia; temperature rising to 43° C with rigors, breathlessness and confusional state. Patient was shifted to intensive care unit and investigated and managed. As mentioned above, patient was in septic shock confirmed clinically by the presence of hypotension, hyperventilation, hyperpyrexia and laboratory data which showed WBC count rising from 5700/ $(mm)^3$ to 26500 $(mm)^3$,with toxic granular polymorphoneucleocytosis, high 'C' reactive protein, elevation of more than 3 times the normal values in liver enzymes, with moderate rise in Total creatinine phosphokinase and also troponin.[4] Blood culture isolated Enterococcus sensitive to the antimicrobials vancomycin, linezolid and teicoplanin. Patient was treated with teicoplanin and consequently he recovered from septic shock. Patient showed improvement in all clinical and laboratory data within

Table 1 : Clinical and laboratory parameters of the patient at different stages of the hospital stay.

DAY	Symptoms	Temperature	Hemogram	Biochemistry	Others
Day 1	Fever, Body ache, Throat pain	40^0 C	Hb 10.6 gm/DL, TC 5700/mm3, Platelet count 58000/mm3	S. Bilirubin Total 2.27mg./DL, indirect 1.25 mg/DL, SGPT60 I.U./ml	Ring form of P.Falciparum Grade 4 detected
Day 3	Fever with rigor, Breathlessness, confusion	45^0 C	Hb 8.8gm/DL, TC 21500/mm3, Platelet 37000/DL, Polymorph 79% with toxic granules.	S. Bilirubin Total2.82/DL, indirect 1.25 mg/DL.SGPT200 I.U./L S. Alkaline phosphatase112 IU/L,CPK Total 56 IU/ml. Troponin: 0.08 I.U.	Malaria parasite not detected, IgG for Leptospira negative. **Blood culture isolates Enterococci**
Day 6	No fever	38^0 C	Hb 10.2gm/DL TC 28500/mm3 Platelet 82000/DL, Polymorph 75% with toxic granules.	SGPT 79 I.U./L, S. creatinine 1.1 mg/DL	
Day 10	No complaint	37^0 C	Hb 10.8gm/DL TC 15500/mm3 Platelet 1 32000/DL' Polymorph 70% without toxic granules.		Discharge

seven days. *P. falciparum* infected erythrocytes get sequestered in microcirculation in vital organs interfering with microcirculatory flow and thereby with host tissue metabolism.[4] Sequestration of RBCs is thought to be a central pathophysiology of falciparum malaria, associated with cytoadherance to the microvasculature and rosette formation leading to obstructive anoxic damage to gut endothelium. Anoxic metabolism of endothelium results in increased permeability of gut bacteria and toxins. All these events lead to septic shock, as in our case, due to invasion by enterococci from gut endothelium. The poor prognostic signs observed in the case were acidosis, rise in creatinine, more than thrice the normal rise in liver enzymes, increase in C- Reactive protein levels, thrombocytopeniawith coagulopathy, rise in APTT, polymorphoneucleoleucocytosis with toxic granulations, grade 4 parasitemia, hypotension and convulsions. Enterococcal septic shock is a rare complication seen in 1% of severe malaria cases, which is also less frequent in falciparum malaria in general population.

Conclusion :

Falciparum malaria may be complicated by multiorgan dysfunction in severe cases. Gut endothelial dysfunction due to microvascular ischemia can lead to entry of gut inhabiting bacteria in circulation and result in septic shock even after recovery from primary malarial infection.

Acknowledgement :

Dr. Shaunak, Dr. Digant, & all staff of ICU, GCS Medical College, Hospital & Research Centre.

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