Symmetrical peripheral gangrene with *Plasmodium falciparum* malaria

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Abstract

Symmetric peripheral gangrene is rare and relatively uncommon complication of malaria. We report a case of a 50-year-old male who survived *Plasmodium falciparum* infection with disseminated intravascular coagulation. Symmetric peripheral gangrene in our case, which ultimately required amputation of the toes, was most likely due to interaction between parasitic factors and host factors.

Key words: *Gangrene, heavy parasitemia, Plasmodium falciparum, severe malaria*

INTRODUCTION

Plasmodium falciparum is a protozoan parasite and one of the species of *Plasmodium* that cause malaria in humans. It is transmitted by the bite of female *Anopheles* mosquito. It causes the most dangerous form of malaria, with highest mortality and morbidity due to a distinct property called as sequestration. Symmetric peripheral gangrene (SPG)

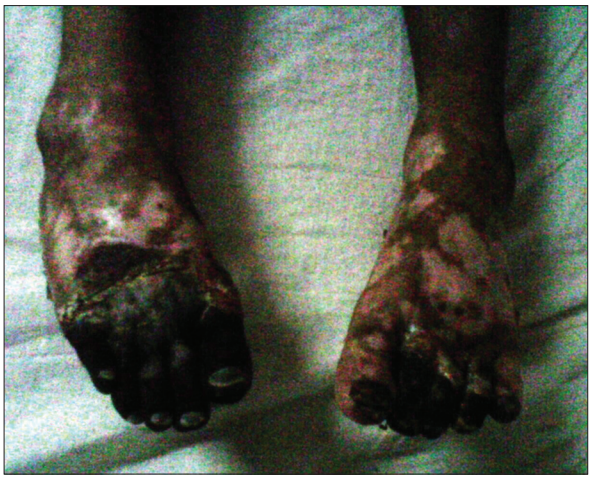

Figure 1: Gangrenous feet

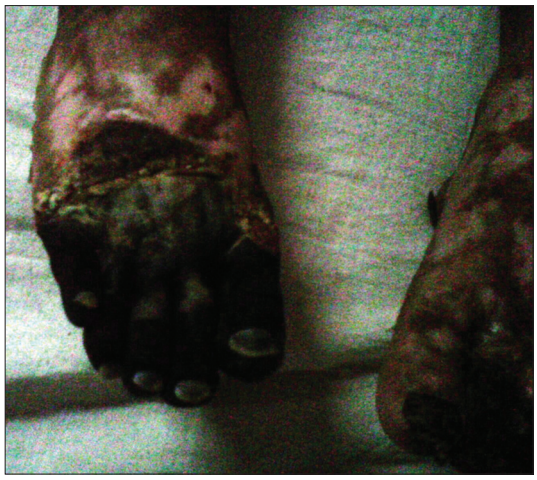

Figure 2: Symmetrical peripheral gangrene

Table 1: Investigation reports

Parameter	Patient values	Reference values
Hemoglobin	7 g/dL	11-13 g/dL
Total leukocyte count	17,900/mm^3	4000-11,000/mm^3
Platelets	146,000/mm^3	140,000-450,000/mm^3
Prothrombin time	16.5	11.5-15.5
SGOT	283.9	7-21 IU/L
SGPT	235.5	8-32 IU/L
INR	1.0	<1.5
D-dimer	1.8	0-0.5mg/l FEU (fibrinogen equivalent units)
Random blood sugar	84 g/dL	70-125 mg/dL

is defined as symmetrical distal ischemic damage at two or more sites in the absence of large vessel obstruction.[1] This syndrome has been reported in several conditions such as bacterial infections, Disseminated Intravascular Coagulation (DIC), low cardiac output states, drugs like ergot, vasopressin, noradrenaline, and rarely associated with *P. falciparum* malaria. We report a unique case of SPG with *falciparum* malaria infection.

CASE REPORT

A 50-year-old uneducated male of low socioeconomic status was scheduled for amputation of toes. To start with, he had complaint of fever associated with chills and rigors for 3 weeks. He consulted a local practitioner who prescribed some tablets about which patient could not give the details. He noticed blackening of toes of both the feet, which was slow and progressive over 1 week. As he was not responding to the treatment, he was rushed to our medical college. There was no history of trauma, significant drug ingestion particularly ergot, alcohol consumption or smoking. On physical examination, he was febrile, dehydrated, and had pallor; there was no icterus, cyanosis, edema, or significant lymphadenopathy. A pulse rate of 120/min, respiratory rate of 24/min, and blood pressure of 90/70 mmHg was recorded. All peripheral pulses were palpable. On systemic examination, mild hepatosplenomegaly was present and rest of the systemic examination was normal. On local examination, there was blackish discoloration of all toes, extending up to malleoli [Figures 1 and 2]. Intavenous line was secured. Ringer lactate was started and investigations were sent for. Reports were as follows [Table 1].

Peripheral blood smear showed ring forms of *P. falciparum* with occasional gametocytes. Three consecutive blood cultures for bacteria were negative. Color Doppler study of the lower limbs revealed normal flow in both the femorals and the popliteal, with slightly reduced flow in tibial and dorsalis pedis artery with hypoechogenic shadow in its lumen.

The patient was given i.v. quinine, loading dose of 20 mg/kg followed by a maintenance dose of 10 mg/kg thrice a day for 2 days. Then, the patient was shifted to oral quinine (dose 10 mg/kg) thrice a day and oral paracetamol 500 mg thrice a day for 5 days. Two units of packed cell transfusion were also given. Fever subsided within 5 days of treatment. His peripheral smear for malarial parasite became negative after 7 days of quinine course. Line of demarcation did not progress any further. For management of gangrene, amputation was planned. All repeated blood investigations were within normal limits except prothrombin time 16.5 s, fibrinogen level 2.9 g/L (normal value 1.5-4 g/L), and D-dimer 1.7. The surgery was done under total i.v. anesthesia.

DISCUSSION

The exact mechanism which may trigger intravascular coagulation in malaria infection is not known. But heavy parasitemia causing activation of the complement system,[2]

triggering the coagulation pathway,[3] and alteration in lipid distribution across the parasitized erythrocytes activating the intrinsic coagulation pathway have been proposed.[4] Parasitized erythrocytes get sequestrated in the microcirculation by molecular interaction with endothelial receptors, chiefly intercellular adhesion molecule (ICAM-I),[5] Vascular adhesion molecule (VCAM-I), thrombospondin, and histidine-rich protein.[6]

The presence of tight packing of parasitized erythrocytes due to decreased deformability of red cells or adherence of infected red cell to microvascular endothelium will initiate a microcirculatory obstruction in malaria[7] (Mac phasen *et al.* 1985).

In spite of the ever widening etiological spectrum of SPG, recent literature points to 100% association with DIC, a with high mortality rate up to 35% and the rate of amputation ranging from 70% to 90%.[8,9] However, in our case, DIC, parasitic factors (like cytoadherence tissue sequestration, rosette formation), and host factors like dehydration are sufficient to produce occlusion of peripheral vessels which may have predisposed to SPG.

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