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Plasmodium Falciparum Malaria Complicated by Symmetrical Peripheral Gangrene - A Case Report

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ABSTRACT

Symmetrical Peripheral Gangrene (SPG) is a rare but severe complication associated with malaria, particularly caused by Plasmodium falciparum. We present the case of a 66-year-old male who presented with generalised malaise and mild SOB following recent travel to Sierra Leone. The patient was diagnosed with malaria caused by plasmodium falciparum and initial parasitaemia was 48%. He deteriorated clinically and required ICU admission with multi-organ failure. He was treated with artesunate and riamet, and unfortunately developed rapidly progressing bilateral gangrene of the upper and lower limbs on day 10 of his admission. The patient fully recovered and was discharged from ICU but required bilateral partial foot amputation. This paper reviews the pathophysiology, clinical presentation, and management strategies of SPG secondary to malaria.

Keywords: Symmetrical Peripheral Gangrene, Malaria, Plasmodium falciparum, Pathophysiology, Clinical Presentation

INTRODUCTION

Symmetrical Peripheral Gangrene (SPG) is an uncommon but potentially devastating complication associated with various infectious and non-infectious aetiologies. While SPG is most encountered in conditions such as sepsis, disseminated intravascular coagulation (DIC), and vasculitis, its occurrence secondary to malaria is particularly rare [1,2]. Plasmodium falciparum, one of the causative species of malaria, is associated with severe manifestations and complications, including SPG [3,4].

CASE REPORT

A 66-year-old male presented to the Emergency Department with one-week history of fatigue and feeling generally unwell, associated with nausea, poor oral intake, and mild shortness of breath. He reported recent travel to Sierra Leone, using mosquito nets but without completing malaria chemoprophylaxis course. There was a background history of hypertension and type 2 diabetes mellitus. He reported no known drug allergies, no history of smoking or alcohol consumption.

On initial assessment, the patient was lethargic. Cardiovascular, respiratory, abdominal, and neurological systems examination was unremarkable. A pulse rate of 72/min, blood pressure of 90/60mmHg, respiratory rate of 20/min and oxygen saturation of 94% on room air was recorded. Investigations revealed mixed diabetic ketoacidosis (DKA) and hyperosmolar hyperglycaemic state (HSS) picture, Hb 118g/L, platelets 17 10^9 /L, WBC 8.4 10^9 /L and CRP 274.3 mg/L. Coagulation profile

was within normal limits. The patient was in AKI Stage 3. The diagnosis of plasmodium falciparum malaria was rapidly made on the blood smear and parasitaemia was 48%. CT head on admission did not demonstrate acute pathology.

Initial management followed a multi-disciplinary approach including ICU review, advice from Haematology and Endocrinology teams, as well as from tertiary centre for Infectious Diseases. DKA protocol was initiated, anti-malarial treatment artesunate 2.4mg/kg and broad-spectrum antibiotics were also started. On the second day of admission, the patient clinically deteriorated with a marked drop in his GCS, worsening renal function, metabolic acidosis and liver impairment. Steady decline of parasitaemia from an initial 48 % to 7.2% was already evident (Table 1). He was transferred to the ICU, was intubated and ventilated, required vasopressor support with noradrenalin as well as CRRT (continuous renal replacement therapy) for refractory hyperkalaemia and metabolic acidosis. He developed acute liver impairment and haemolytic anaemia. Blood and platelet transfusions were administered following discussion with haematology team. After completing the 7-day course of artesunate, the patient was started on riamet as per Infectious Diseases team advice with daily blood films parasitaemia monitoring. He remained in isolation.

He showed progressive clinical improvement, vasopressors were stopped on the 4th day of his ICU admission, his liver and haematological profile stabilised and the peripheral blood smear became negative for Plasmodium Falciparum 7 days after initiating treatment. On sedation hold he was found to be inappropriate (not opening eyes, not following commands) and CT head was repeated and demonstrated bifrontal subdural hygromas. Neurosurgical team advised that no surgical intervention was required, and subsequent MRI head confirmed stable appearances.

Despite these improvements, on the 10th day of his admission, he developed rapidly progressing bilateral symmetrical gangrene of the upper and lower limbs accompanied by skin break down and blister formation, but there was no large vessel involvement (Fig 1-4). The patient was referred to vascular surgery team, line of demarcation was monitored, and prophylactic heparin treatment was given. The patient was extubated 15 days after his admission with liver, renal and neurological functions fully recovered. He was transferred to vascular surgery unit for bilateral partial foot amputation, and he remains under review for his upper limb lesions.

Figure 1&2: Day 10- Bilateral symmetrical gangrene of the upper limbs

Figure 3&4: Day 10- Bilateral symmetrical gangrene of the lower limbs

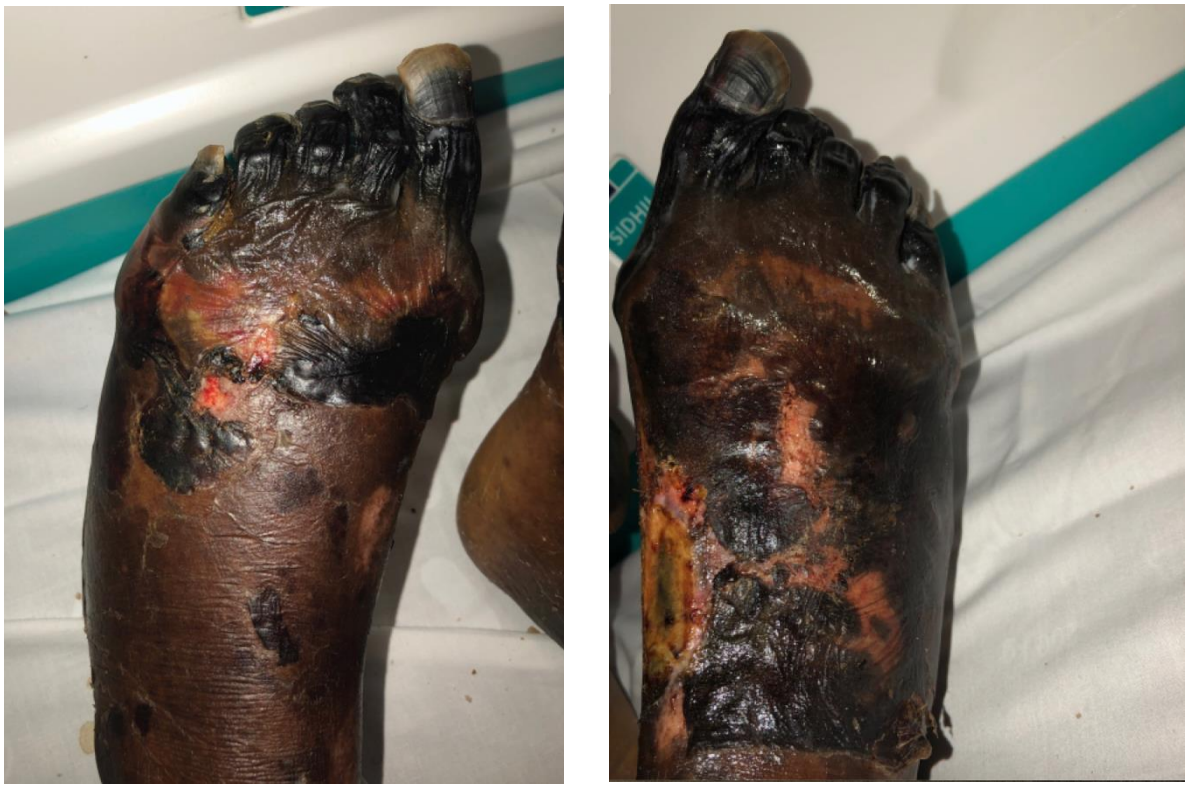

Table 1: Laboratory parameters

DAY OF ADMISSION	**1st day**	**2nd day (ICU)**	**5th day (ICU)**	**10th day (ICU)**
FULL BLOOD COUNT				
WBC (10^9/L)	8.4	9.4	9.9	6.4
Haemoglobin (g/L)	118	100	81	79
Platelets (10^9/L)	17	16	50	119
C-REACTIVE PROTEIN	274.3	222.6	142.9	59.3
COAGULATION SCREEN				
INR	1.1	1.1	1.4	1.0
Prothrombin time (sec)	12.8	13	17.4	12.1
APTT (sec)	23.8	23.2	25.5	87.6
APTT Ratio	0.8	0.8	0.9	2.9
LIVER FUNCTION TEST				
Total protein (g/L)	72	59	58	60
Albumin (g/L)	27	20	17	18
Total bilirubin (µmol/L)	53	67	64	63
ALP (U/L)	106	159	220	232
ALT (U/L)	48	1471	1055	189
UREA,CREATININE & ELECTROLYTES				
Sodium (mmol/L)	127	139	138	128
Potassium (mmol/L)	6.1		4.1	3.9
Urea (mmol/L)	34.3	34.7	12.5	31.7
Creatinine (µmol/L)	377	331	210	460
AKI Stage	3	2	1	3
MALARIA FILM	Trophozoites of plasmodium falciparum	plasmodium falciparum	n/a	n/a
Percentage parasitaemia	48	7.2	n/a	n/a

Table 2: Percentage parasitaemia

Day of admission	1	2	3	4	5	6	7	8	9	10
Percentage parasitaemia	48	7.2	0.9	-	-	0.1	0.1	n/a	n/a	n/a

DISCUSSION

The pathogenesis of SPG secondary to malaria involves a multifactorial process. Parasite-infected erythrocytes exhibit cyto-adhesive properties, leading to sequestration within the microcirculation and consequently microvascular obstruction [5,6]. Additionally, inflammatory cytokines released contribute to endothelial dysfunction and thrombus formation [7]. The alteration of erythrocyte membrane composition during the Plasmodium life cycle further exacerbates vascular occlusion [8,9]. These mechanisms collectively lead to ischemia and subsequent gangrene in distal extremities [10].

SPG secondary to malaria typically presents as distal ischaemic gangrene affecting two or more symmetrically distributed sites, such as the fingers, toes, or distal limbs [11]. Patients may experience pain, pallor, and oedema in the affected extremities. Diagnosis is based on clinical findings, along with a history of malaria infection. Laboratory investigations may reveal evidence of severe malaria, including hyperparasitaemia, anaemia, acidosis, and renal impairment [12,13].

Management of SPG secondary to malaria requires a multidisciplinary approach. Prompt initiation of antimalarial therapy is essential to eradicate the parasite and prevent further complications [14]. Anticoagulants, such as heparin, may be considered to counteract thrombosis, although their efficacy in this context is not well-established. Surgical intervention, including debridement or amputation, may be necessary in cases of extensive tissue necrosis or infection [15]. Optimisation of perfusion and avoidance of trauma to the affected limbs are essential components of supportive care [16].

CONCLUSION

SPG secondary to malaria is a rare but potentially devastating complication that requires prompt recognition and intervention. Understanding the underlying pathophysiology and clinical presentation and having a multi-disciplinary approach is crucial for healthcare providers treating malaria patients. Further research is needed to elucidate the optimal treatment approaches and outcomes associated with SPG in the context of malaria.

This case highlights the severe and multi-sided nature of P. Falciparum malaria, especially in patients with comorbidities. The rapid progression to multisystem failure (AKI, liver impairment and haemolytic anemia) proves the importance of early diagnosis, aggressive treatment, and multi-disciplinary approach. The development of peripheral gangrene, despite the cessation of vasopressors, points to the profound microvascular complications associated with severe malaria. The use of a multidisciplinary approach is critical to help recover major organ functions and manage the severe complications.

Declaration by Authors

Ethical Approval: This case report was made from patient admitted to Luton and Dunstable University Hospital and informed consent was obtained for sharing the anonymous case and necessary pictures for the purpose of educational publication and presentation.

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