Original Research Paper

Volume - 13 | Issue - 04 | April - 2023 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Internal Medicine

RARE PRESENTATION OF SYMMETRICAL PERIPHERAL GANGRENE IN SEVERE MIXED MALARIA.

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ABSTRACT Symmetrical peripheral gangrene is a known entity characterized by gangrene of two or more sites in absence of obvious large vessel obstruction. Such complications in common tropical infectious disease like Malaria is rare. Pathogenesis and treatment of symmetrical peripheral gangrene in malaria is unclear. However, can be attributable to cyto-adhesion, rosetting, and agglutination. Due to its rapid progression and sinister prognostic implications aggressive multidisciplinary team effort is essential. we present herewith a rare case of symmetrical peripheral gangrene in severe mixed Malaria.

KEYWORDS : Malaria, peripheral gangrene, symmetrical, SPG

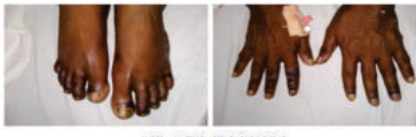
INTRODUCTION

Symmetrical peripheral gangrene (SPG) is a known entity characterized by gangrene of two or more sites in absence of obvious large vessel obstruction[1]. It has been reported with a wide gamut of causative factors both infective and noninfective[2]. The mortality rate with SPG can be up to 35% and rate of amputation may range from 70 % to 90%[3]. SPG in malaria is rare complication and has been been reported with plasmodium falciparum. We are presenting a unique case of SPG in severe mixed malaria.

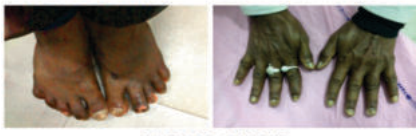
Case Report

A 55-year-old male presented with three days history of high-grade fever followed by breathlessness , and altered sensorium. Examination revealed tachycardia, hypotension, respiratory rate 20/min. Patient was drowsy, there were bilateral fine crepitations, per abdominal was unremarkable. There were no focal neurological deficits or signs of meningeal irritation.

Initial investigation revealed thrombocytopenia, pre-renal AKI, hepatopathy, hypoglycemia and metabolic acidosis .Rapid malaria dipstick test was positive for plasmodium vivax . On peripheral smear there were occasional ring forms of plasmodium falciparum and few gametocyte forms, ring forms and schizonts of plasmodium vivax. D-dimer was raised. PT/INR was normal. RTPCR for COVID-19 was negative. CT brain showed focal areas of subarachnoid haemorrhage. CT thorax was suggestive of pulmonary oedema. Patients was aggressively managed in ICU setup and received injectable artesunate, clindamycin, oral primaquine, vasopressors, adequate fluid resuscitation, and platelet and packed cells transfusion. On day three of admission, he developed blackish discoloration of fingers of both hands followed by involvement of toes on the next day. All peripheral pulses were well felt. Arterial doppler of bilateral upper limb and lower limbs was normal. 2D echo and ECG was normal. There was no progression of gangrene beyond fingers and toes. On 5th day of admission, once platelet counts improved, aspirin was initiated. Patient responded well to treatment and gangrenous changes did not progress. Antiplatelets were continued at discharge. At follow up gangrenous changes resolved completely. No amputation was required.



AT ADMISSION



AT FOLLOW UP

Image 1 . photographs showing gangrenous changes at admission and recovery at follow up.

Table 1 – various biochemical parameters at admission and follow up.

PARAMETERS	AT ADMISSION	AT DISCHARGE
Haemoglobin.	11.0 gm/dl	10.0 gm/dl
Total leukocyte count	7,480/ul	5,300/ul
Platelet count	11,000/mm3	2.8 lakh/mm3
Blood urea	123mg/dl	21.40mg/dl
Serum creatinine	3.02mg/dl	0.70 mg/dl
Total bilirubin	10.60mg/dl	2.10mg/dl
AST	52 U/L	47U/L
ALT	57 U/L	33 U/L
ALP	247 U/L	145 U/L
RBSL	LO	146mg/dl
Prothrombin time	11.0(control -10.3-11.3 sec)	10.9
INR	1.09(control -0.8- 1.2)	1.08
D-dimer	>12,800 ng/ml	922 ng/ml

DISCUSSION

Malaria is a vector born disease which can present with numerous complications, however SPG is rare. Few theories have been proposed to explain the mechanisms of SPG in malaria. Infected erythrocytes adhere to the endothelium via intercellular adhesion molecules-1(ICAM-1)[3]. Thus cytoadherence, Rosetting eventually block the microcirculation leading to thrombosis. Other mechanisms include heavy parasitemia causing activation of complement system and coagulation pathways, disseminated intravascular coagulation[4], decreased deformability of infected RBCs[5], and activation of intrinsic coagulation pathway. In our patient other causes of SPG were ruled out leaving malaria as the more likely cause.

Early occurrence and quick progression of SPG in this case report is comparable to findings in prior reports[5]. Management of severe malaria is well established yet the same for malaria with SPG can be challenging as there is no specific treatment[3]. Routine use of antiplatelets or anticoagulation is not recommended [3] however in prior case reports and reviews it has been tried with variable success rates [6].

Aggressive treatment of malaria, management of sepsis and DIC, timely use of vasopressors, fluid resuscitation and multidisciplinary team effort at early stages of SPG is lifesaving. In our patient antiplatelets were continued till follow up and he showed complete recovery of SPG .

CONCLUSION

SPG in malaria has rapid onset and quick progression practically giving very less time to intervene. Since malaria is a completely treatable infection, it is important to be aware of such a potentially life-threatening complication. Prompt team effort and aggressive management is the key learning lesson from this case report. Use of antiplatelets may be useful but needs to be confirmed by further research. Early anticipation of this complication can help to prevent the sinister prognostic implications in terms of loss of limb or life.

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