

Cutaneous manifestations of malaria

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DESCRIPTION

Cutaneous findings, including skin necrosis, are a rare complication in patients with *Plasmodium vivax* malaria. Infected parasitised red cells can lead to complement activation, causing microvascular thrombosis and cutaneous complications. We present a woman in her third decade residing in a malaria-endemic area with no medical history who presented with fever associated with chills and rigors, abdominal pain, and decreased urine output for 7 days. She also gave a history of a painful, reddish black rash on the face for 4 days. Physical examination revealed tachycardia, with a pulse rate of 110 per minute, blood pressure of 150/90 mm Hg and necrotic black non-purpuric rash with perilesional erythema on the cheeks, tip of the nose and ear lobes (figure 1A,B). There was associated mild hepatosplenomegaly and pallor. The patient denied any history of joint pain, Raynaud phenomenon, photosensitivity, alopecia, over-the-counter drug use or exposure to extreme cold. There was no documented hypotension or vasopressor use. Laboratory evaluation revealed anaemia with haemoglobin of 78 g/L, thrombocytopenia with a platelet count of $76 \times 10^9/L$, serum creatinine of 636 $\mu\text{mol/L}$, unconjugated hyperbilirubinaemia with total and direct serum bilirubin of 3.5 mg/dL and 0.9 mg/dL, aspartate transaminase of 76 U/L, and alanine transaminase of 65 U/L. *P. vivax* was detected in peripheral blood as well as by rapid malarial card test. The remaining work-up including coagulogram, scrub typhus serology, dengue serology, antinuclear antibody, complement levels, antineutrophil cytoplasmic antibody and anticardiolipin antibody were negative. She also had evidence of Coombs-negative haemolysis in the form of raised lactate dehydrogenase (1939 IU/L), elevated plasma haemoglobin level (22 mg/dL) and low haptoglobin. Schistocytes were not seen on peripheral blood smear. A skin biopsy done from the edge of the necrotic areas revealed evidence of fibrin thrombi in dermal capillaries with endothelial swelling suggestive of thrombotic microangiopathy with no features of vasculitis. Immunofluorescence was negative. The patient was initiated on intravenous artesunate and primaquine with topical fluticasone for the skin lesions. She underwent two sessions of haemodialysis and blood transfusion. On day 4 of hospital stay, the lesions started healing, urine output improved and the patient became afebrile. On day 10 of illness, the lesions healed completely without scarring and serum creatinine had decreased to 61 $\mu\text{mol/L}$ (figure 1C,D).

Malaria remains a major cause of morbidity and mortality worldwide.¹ Cutaneous lesions reported in malaria include angio-oedema, urticaria, peripheral symmetrical gangrene and petechiae.²⁻⁴ It is postulated that heavy parasite burden leads to complement activation, thereby causing endothelial injury, swelling



Figure 1 (A, B) Image of the patient on admission showing black necrotic lesions on both cheeks, ear lobes and nose tip. (C, D) Image of the patient on day 10 showing complete resolution with minimal scarring.

Learning points

- Recognition of cutaneous complications is important for timely intervention and to prevent misdiagnosis of malaria as cutaneous vasculitis or frostbite.
- Skin necrosis can be a potential clinical manifestation of severe complicated vivax malaria.

and microvascular occlusion. In addition, inflammatory cytokines, disseminated intravascular coagulation and increased tissue factor expression lead to microvascular thrombosis. This pathophysiology is common for both *P. vivax* and *P. falciparum* malaria. Our patient's acute kidney injury could also be attributed to severe endothelial injury and resultant renal thrombotic microangiopathy.⁵ Management of such patients with malaria and microvascular thrombosis is controversial. Parasite eradication with antimalarials and occasional use of anticoagulants have been suggested. To conclude, we report a rare association of necrotic facial skin lesions with complicated vivax malaria mimicking frostbite and cutaneous vasculitis. Our case suggests that infection with *P. vivax* may rarely result in systemic endothelial injury, thrombotic microangiopathy and skin necrosis.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research.



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They should not be used in isolation to guide treatment choices or public health policy.

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