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PhotoDermDiagnosis

Sudden bilateral blackish discoloration of toes in middle-aged female

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A 30 years old non-diabetic, non-smoker, married female developed high-grade fever which was associated with palpitations. Fever was relieved by medications (paracetamol and aspirin). After 4 days, she developed moderate to severe pain in both feet. Two days later, she noticed erythema, which progressed to blackish discoloration bilaterally on toes within 24 hours. Pain was severe and constant in the affected areas. Systemic inquiry was insignificant.

On examination patient was pale, well oriented, and conscious and there was bilateral gangrene of toes of both feet up to metatarsophalangeal joints (**Figure 1** and **2**). All pulses were palpable. No other positive finding was recorded on examination. Investigations revealed Hb.9.8g/dl. ANA, anti-ds DNA antibodies, lupus anticoagulant, anti-cardiolipin antibodies and Coombs direct and indirect test were negative. Serum cryoglobulins levels were normal. Anti-HCV antibodies were positive. Coagulation profile was normal. Doppler studies for arterial and venous flow in both lower limbs, ultrasound abdomen, ECG and echocardiography were normal.

What is your diagnosis?

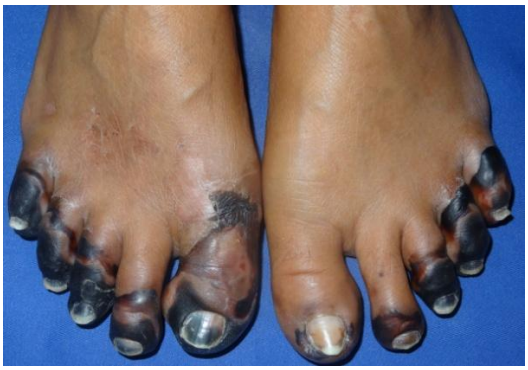

Figure 1 Symmetrical bilateral blackish discoloration of toes (dorsal view).

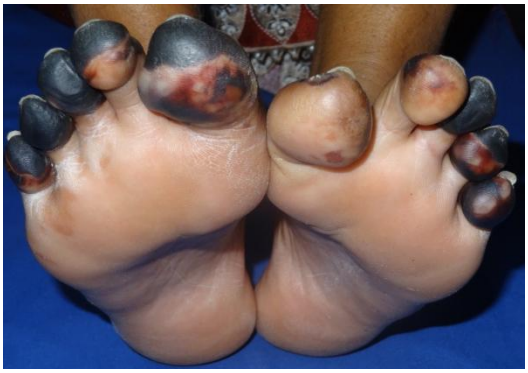

Figure 2 Symmetrical bilateral blackish discoloration of toes (ventral view).

Diagnosis

Symmetrical peripheral gangrene associated with hepatitis C.

Discussion

Symmetrical peripheral gangrene (SPG) is a rare clinical condition and is defined as symmetrical distal ischemic damage at two or more sites in the absence of large vessel obstruction. It was first described by Hutchinson (1891).[1]

Cutaneous vasculitis leading to symmetrical peripheral gangrene can be due to many factors including autoimmune disease, malignancy, drugs, and infections including streptococci, staphylococci, mycobacterium, hepatitis B and C etc.[2,3] (**Table 1**). The factors aggravating SPG include asplenia, immunosuppression, previous cold injury to extremities, diabetes mellitus, renal failure, increased sympathetic tone and use of vasopressors.[4]

Clinically SPG presents as palpable purpura which may progress to papules, nodules, vesicles, bullae, pustules, necrosis leading to gangrene.[5] The ischemic changes usually begin distally and may progress proximally. These changes are not generally preceded by vascular occlusion.

SPG should be suspected as the first sign of coldness, pallor, cyanosis, or pain in the extremity as the condition rapidly progress to frank gangrene.[6]

A postulated mechanism for necrotizing vasculitis in hepatitis C virus infection is a deposition of circulating immune complexes in postcapillary venules. Initial alterations in venular permeability due to the release of vasoactive amines from platelets, basophils, and mast cells facilitate the deposition of immune complexes and these may activate the complement system or may interact directly with Fc receptors on endothelial cell membranes. When the complement system is activated, the generation of anaphylatoxins C3a and C5a can degranulate mast cells. Also C5a can attract neutrophils that release lysosomal enzymes during phagocytosis of complexes and subsequently damage vascular tissue.[7]

Table 1 Causes of symmetrical acral gangrene of sudden onset.

Causes
Disseminated intravascular coagulation
Infections
Streptococci
Staphylococci
Hepatitis B
Hepatitis C
Sepsis
Low cardiac output state
Vasoactive drugs
Vasopressin
Dopamine
Frost bite
Collagen vascular disease
Cryoglobulinemia
Antiphospholipid syndrome
Malignancy

No treatment is universally effective. It should be individualized according to the underlying disease and the patient general condition.[8] In our patient the underlying disease was hepatitis C.

Patients with symmetrical peripheral gangrene require urgent assessment and treatment to prevent the spread of gangrene. Antibiotic and the removal of the cause has been proved effective.[9]

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