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CASE REPORT

Retiform purpura in the context of sepsis and autoimmunity

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Abstract

Retiform purpura is a reticular skin eruption resulting from involvement of cutaneous vessels, which may lead to tissue ischemia and necrosis. The differential diagnosis is broad and includes both inflammatory and non-inflammatory causes. We present a case of a young male with systemic lupus erythematosus and antiphospholipid syndrome who developed a reticular skin eruption during an episode of sepsis and was successfully managed despite several clinical constraints. This case highlights that multiple contributory mechanisms may coexist in a single presentation and emphasises the importance of early recognition and prompt treatment to improve outcomes.

Introduction

Retiform purpura is a net-like, non-blanching skin eruption due to involvement of the cutaneous vessels. Vessel involvement can be in the form of vessel wall thickening or obstruction of the lumen, which leads to ischemia and necrosis.[1] There is a wide range of differential diagnoses, which can be broadly categorized into inflammatory and non-inflammatory.[1] Early recognition of retiform purpura in acutely or critically ill patients is essential to guide prompt management and improve prognosis.

Keywords: retiform purpura; antiphospholipid syndrome; sepsis; cutaneous vasculopathy.

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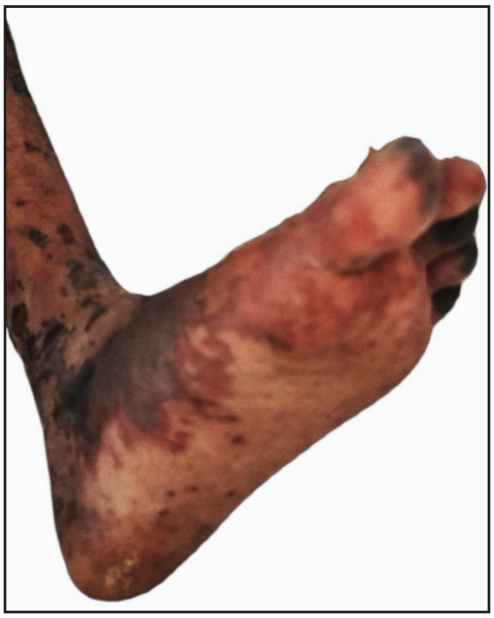
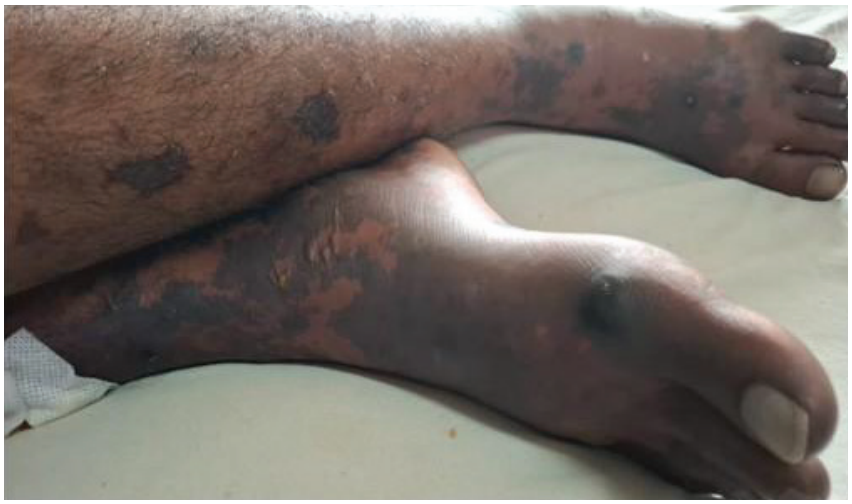
Case Presentation

A 21-year-old university student with systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APLS), who was on multiple immunosuppressants and had defaulted treatment for two weeks, presented with a febrile illness associated with loose stools for one day. He was admitted and treated at a private sector intensive care unit for septic shock with acute kidney injury. Subsequently he was transferred to a tertiary care hospital with a skin rash involving the lower limbs, upper limbs, and back, skin necrosis, and gangrenous toes over a two-day duration. He complained of severe pain and numbness in his lower limbs.

Examination revealed a non-blanching reticular skin eruption with areas of skin necrosis in the bilateral lower limbs, upper limbs, and the back of the chest. A few gangrenous toes and bilateral lower limb oedema were noted (Figure 1 A, B).



A



B

Figure 1. Retiform purpura. (A) Plantar aspect of the foot. (B) Lower limb with cyanosed toes.

Diagnosis and Management: Initial investigations showed very high inflammatory markers (ESR 105 mm/hr, CRP 285mg/L) with very high procalcitonin levels (>200ng/ml), depicting severe sepsis. Blood count showed leukocytosis (37×10^3cells/mm^3) with thrombocytopenia ($21\times.10^3$cell/mm^3). Normal coagulation profile (APTT-23.9s) and fibrinogen levels (1.7g/l) excluded disseminated intravascular coagulation. However, his complement levels, including C3 (50.3 mg/dl), were found to be low, indicating the possibility of SLE flare.

As there could be multiple contributory causes, we proceeded with a skin biopsy and started treating those possible causes, while awaiting histological confirmation. Sepsis was controlled with broad-spectrum antibiotics; high-dose systemic steroids were started considering SLE-related vasculitis and covered with subcutaneous enoxaparin for the possibility of catastrophic APLS.

Later, histology confirmed two foci of fibrin thrombi in dermal vessels without vasculitis.

Discussion

Retiform purpura is a special form of reticular skin eruption due to cutaneous vessel involvement in the form of pathology in the vessel wall or the luminal narrowing.[1,3] It can lead to progressive ischemia and skin necrosis.

Based on morphology, the clinician can arrive at a conclusion of the origin, whether it's inflammatory or non-inflammatory. Inflammatory lesions typically show surrounding erythema involving more than two-thirds of the lesion with a relatively smaller central area of impending necrosis (<1/3), which usually happens due to vessel wall involvement, like in vasculitis. In contrast, non-inflammatory lesions usually demonstrate a larger central necrotic area with less surrounding erythema. Here, the pathology is due to vaso-occlusion.1 However, there might be several overlaps in disease cause.

The distribution of the skin eruption can also provide diagnostic clues. Acral lesions are most commonly associated with embolic phenomena and cold-related disorders such as cryoglobulinaemia, whereas eruptions confined to the lower limbs are more often related to vasculitis. In our patient, both acral and non-acral areas were involved.

Given the broad range of clinically relevant differential diagnoses, categorizing causes according to vessel wall involvement or luminal occlusion can help narrow the diagnostic possibilities.

In autoimmune conditions, cutaneous vasculitis may occur in disorders such as IgA vasculitis (Henoch–Schönlein purpura), cryoglobulinaemia, polyarteritis nodosa, and other autoimmune vasculitides.

Luminal occlusion may result from emboli or thrombosis. Embolic causes include bacterial emboli, non-bacterial emboli as seen in Libman–Sacks endocarditis, and cholesterol emboli. Thrombotic causes include protein C or S deficiency, disseminated intravascular coagulation, and antiphospholipid syndrome.[1]

In our patient, sepsis was one possible contributor and may cause vascular compromise through direct bacterial invasion of vessels, septic emboli, reactive leukocytoclastic vasculitis, or disseminated intravascular coagulation. Both vessel wall inflammation and luminal occlusion may coexist. Furthermore, given the background of systemic lupus erythematosus and biochemical evidence of disease flare, SLE-related vasculitis involving the vessel wall was also considered, while antiphospholipid syndrome could contribute through thrombotic luminal occlusion.

Considering all the above possibilities, and after narrowing down the differential diagnosis, it's important to come to a diagnosis with the help of investigations. Septic screening, including blood cultures as well as a 2D echocardiogram, inflammatory markers, procalcitonin, blood pictures, and full blood count, will rule out sepsis. The coagulation profile will give clues on disseminated intravascular coagulopathy and possible APLS. Vasculitis-specific antibodies and complement levels rule out autoimmunity.

Skin biopsy will direct towards vasculitis or the occlusive disease.

Once a diagnosis is made, it is paramount to initiate treatment to prevent skin necrosis, ulceration, and digital gangrene. Underlying disease should be treated, as if sepsis is to be controlled with relevant measures, autoimmunity should be addressed with immunosuppression, and thrombosis with anticoagulation and antiplatelet.

Supportive measures, including pain management and wound care, are vital. Adequate pain relief may require opioids, gabapentin, ketamine, and benzodiazepines, among other agents. Wound debridement of wet necrotic tissue and prevention of infection are necessary. Hyperbaric oxygen may facilitate wound healing in some patients.[2]

Conclusion

Retiform purpura should be recognized early, especially in acutely and severely ill patients, and the causes can be multifactorial. Treatment should be administered promptly to prevent severe consequences.

Ethics statement

Informed written consent was obtained from the patient for the publication of this case report.

Authors' contributions

IH contributed to the case report by conceptualization, investigation, and writing the original draft. GK contributed to the case report by supervision, reviewing, and editing the draft and conceptualization.

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