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Beyond stasis dermatitis: a case report of pregnancy-associated acroangioidermatitis

Abstract

Acroangioidermatitis (AAD), or pseudo-Kaposi sarcoma, is a rare, benign vasoproliferative malady often complicating chronic venous insufficiency. While its pathophysiology is linked to venous hypertension, its occurrence during pregnancy remains poorly documented. We report a 32-year-old woman who presented with an erythematous-purpuric plaque on her right lower leg, which appeared during the 22nd week of gestation. Clinical and dermoscopic evaluations revealed characteristic vascular patterns, while venous Doppler ultrasound confirmed bilateral saphenous incompetence. Serological tests for HIV and Hepatitis C were negative. Histopathological analysis showed small capillary proliferation with regular endothelium and fibroblastic stroma, confirming acroangioidermatitis. Management focused on compression therapy and topical corticosteroids, resulting in clinical stabilization. This case highlights pregnancy-induced hemodynamic stress as a significant trigger for AAD. Distinguishing this reactive process from malignancy is crucial. Integrating clinical, dermoscopic, and Doppler findings allows for an accurate diagnosis, ensuring appropriate conservative management and alleviating patient anxiety during the gestational period.

Keywords: acroangioidermatitis, chronic venous insufficiency, Dermiteo creof Favre, pregnancy, dermoscopy, pseudo-Kaposi sarcoma

Introduction

Acroangiokeratosis (AAD), also known as pseudo-Kaposi sarcoma, is a rare, benign, reactive vasoproliferative dermatosis first described by Mali et al. in 1965[1]. Clinically characterized by red-violaceous to brown macules, papules, nodules, plaques, and occasionally ulcerations, predominantly on the extensor surfaces[2]. Due to its striking clinical resemblance to Kaposi sarcoma, accurate diagnosis requires careful clinicopathological correlation[3]. We report a case of acroangiokeratosis in a female patient with chronic venous insufficiency, highlighting clinical, dermoscopic, and histopathological features.

Case report

A 32-year-old woman was referred for evaluation of a skin lesion that had first appeared one year previously, during the 22nd week of amenorrhea in the course of her pregnancy. Clinical examination revealed an erythematous-purpuric plaque with irregular borders, located on the lower right leg, extending to the medial malleolus with varicose veins and varicosities on both lower extremities (Figure 1). Dermoscopy demonstrated violaceous background color, white shiny structures, peripheral coppery dots, superficial white scales, and red lacunae (Figure 2). Arterial Doppler ultrasound showed no abnormalities. Venous Doppler ultrasonography revealed bilateral saphenous vein incompetence with significant varicose networks and multiple perforator reflux on both sides. These findings confirmed chronic venous insufficiency. Serologic testing for HIV and hepatitis C was negative, which helped exclude key differential diagnoses, in particular Kaposi sarcoma. A punch biopsy was performed and showed orthokeratotic epidermal hyperkeratosis with a fibrous dermis containing a proliferation of small capillary structures with regular endothelial borders and focal erythrocyte extravasation (Figure 3). Based on the clinical presentation, dermoscopic findings, histopathological features, and Doppler results,

a diagnosis of acroangiodermatitis was established. Treatment included clobetasol propionate 0.05% ointment, along with supportive measures such as leg elevation and compression stockings. The patient was referred to vascular surgery for further management of her varicosities.

Figure 1. Erythematous-purpuric plaque with varicose veins and varicosities.

Figure 2. Dermoscopic view showing violaceous background color (yellow circle), white shiny structures (yellow arrow), peripheral coppery dots (black arrow), superficial white scales (green triangle), and red lacunae (white arrow).

Figure 3. Histopathology showing an epidermis with orthokeratotic hyperkeratosis and a proliferation of small capillary vessels in the dermis, associated with fibroplasia and a mild perivascular inflammatory infiltrate. Adnexal structures and subcutaneous adipose tissue are preserved (hematoxylin and eosin, $\times 40$).

Discussion

Acroangiodermatitis results from chronic circulatory disturbances leading to endothelial proliferation and neoangiogenesis, primarily mediated by vascular endothelial growth factor (VEGF) in response to tissue hypoxia[1]. Four clinical variants have been described in the literature: the Mali type, most frequently associated with chronic venous insufficiency. The type linked to congenital or acquired arteriovenous malformations (Klippel-Trenaunay syndrome, Stewart-Bluefarb syndrome). The type occurring in patients with chronic renal failure or iatrogenic arteriovenous fistulas[2]. The type associated with pregnancy, Dermiteocre of Favre, presents as purpuric macules and plaques over venous varicosities[4] as observed in our patient. This clinical presentation is primarily driven by the progressive increase in intra-abdominal pressure and blood volume during pregnancy, which exacerbates venous hypertension. The mechanical compression of the inferior vena cava by the gravid uterus, combined with hormone-induced vasodilation, leads to chronic

venous stasis and subsequent microvascular proliferation, triggering the reactive angiohistiocytic process characteristic of acroangiodermatitis[5,6]. Dermoscopy plays a valuable role in distinguishing acroangiodermatitis from clinically similar conditions, such as Kaposi sarcoma, lichen planus, or pigmented purpuric dermatitis[7]. Typical features include red or purple lacunae, hemorrhagic crusts, white scales, vascular lagoons, and a violaceous background showing polymorphic vessels, all reflecting the underlying vascular proliferation. Polychromatic color changes, rosettes, and white shiny structures may also be observed, the latter likely related to fibroblastic proliferation and the birefringence phenomenon caused by polarized light interacting with collagen. Peripheral coppery pigmentation is frequently present and is attributed to stasis dermatitis, with extravasation of erythrocytes and hemosiderin deposition[3,7]. On histological examination, it is characterized by lobular proliferation of small dermal blood vessels, with perivascular fibrosis. Erythrocyte extravasation and dermal siderophages are common features[8]. Management focuses on addressing the underlying vascular pathology. Compression therapy with elastic bandages and topical corticosteroid therapy are often beneficial[2,9]. Depending on the cause, additional options may include sclerotherapy, high ligation, valve grafting, and vein surgery. Erythromycin and dapsone have been reported as effective in the literature[1,10].

Conclusion

This case illustrates that pregnancy-related hemodynamic changes can trigger acroangiodermatitis. Early recognition through clinicopathological correlation and Doppler ultrasound is essential to justify conservative management with compression therapy, ensuring a favorable prognosis and alleviating patient anxiety.

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