Case Report

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Acquired Perforating Dermatosis Associated with Hodgkin Lymphoma: Rare Associated Manifestation

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Abstract:

Acquired reactive perforating collagenosis (ARPC) is a rare subtype of acquired perforating dermatosis (APD) that typically occurs in adults, often associated with diabetes mellitus or chronic renal failure. However, ARPC has also been reported in association with malignancies and other systemic disorders. We presented the case of a 21-year-old Thai woman presented with multiple erythematous, pruritic papules with central keratotic plugs on the trunk, back, and extremities for five months. With history of chronic cough and significant weight loss of 10 kg over three months. Punch biopsy consistent with ARPC. Further investigations revealed an anterior mediastinal mass, multiple enlarged cervical and intra-abdominal lymph nodes, and tiny pulmonary nodules with peribronchovascular consolidations at anterior segment of RUL; concerning for lymphatic or lung involvement. EBUS-guided biopsy showed atypical cells compatible with Classic Hodgkin lymphoma. The patient was diagnosed with ARPC with Hodgkin lymphoma stage IIIB with bulky disease, IPS 0. This case highlights the importance of recognizing ARPC as a potential cutaneous manifestation of underlying malignancy.

Keywords: acquired perforating dermatosis, central keratotic plug, significant weight loss, Hodgkin lymphoma

Introduction

Acquired reactive perforating collagenosis (ARPC) is a subtype of acquired perforating dermatosis (APD) that typically occurs in adulthood, often associated with diabetes mellitus (DM) or chronic renal failure (CRF).[1,2] Recently, ARPC has also been reported in patients with malignancies,[3] hepatic and endocrine disorders, acquired immunodeficiency syndrome, often including retiform purpura.[4] In severe cases, ischemic necrosis and purpura fulminans tuberculosis, pulmonary aspergillosis, neurodermatitis, atopic dermatitis, and scabies.[4] ARPC presents as umbilicated necrotic papules or nodules with central keratotic plugs.[5] Lesions can be localized or generalized,[6] commonly affecting the extensor surfaces of the limbs, while involvement of the trunk, face, and back is less frequent.[7]

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Case presentation

A 21-year-old Thai female presented with multiple erythematous pruritic papules with central keratotic plugs on trunk, back, and both extremities for five months. (Figure 1A, 1B, 1C) Three months before presentation, she developed a chronic cough accompanied by significant weight loss of 10 kilograms over a three-month period. Symptoms progressed, so she came to visit Dermatology department.

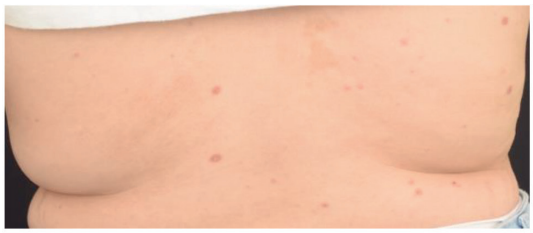


Figure 1A

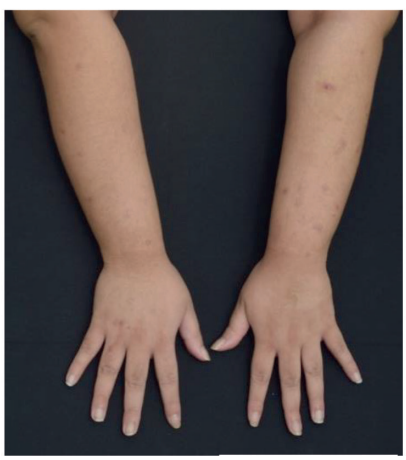


Figure 1B

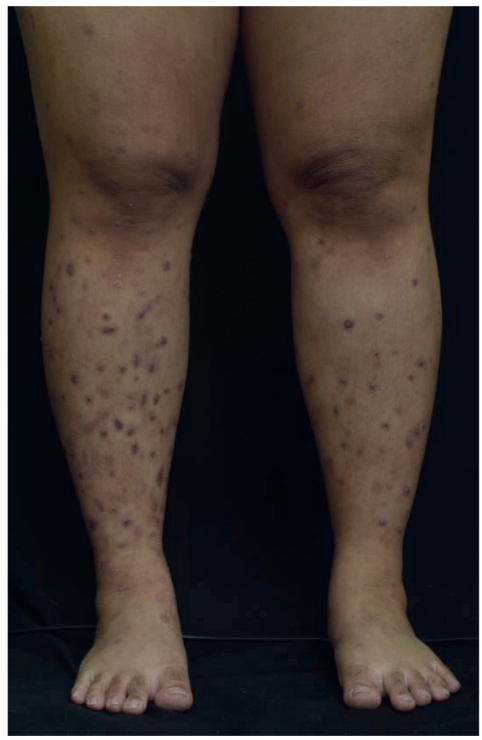
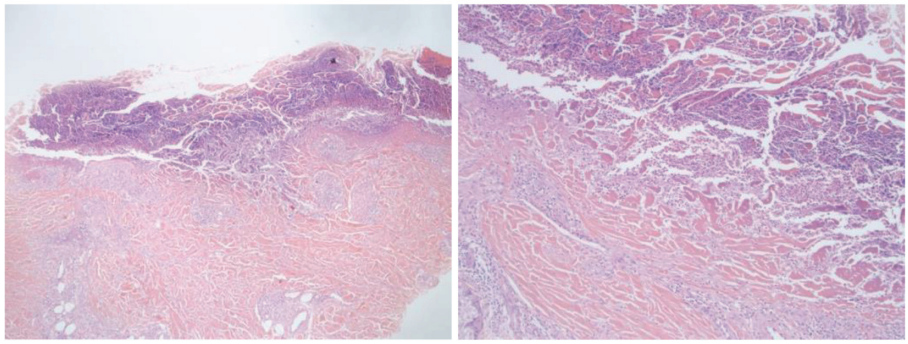


Figure 1C

Figure 1A, 1B, 1C Multiple well-defined border erythematous and hyperpigmented papules with central keratotic plug on trunk, back, and both extremities

Physical examination revealed multiple well-defined erythematous and hyper pigmented papules with central keratotic plugs distributed over the trunk, back, and both extremities. Bilateral cervical lymphadenopathy was noted, with multiple enlarged lymph nodes measuring up to 1.5 cm in diameter.

Punch biopsy from her left leg showed perforation of the epidermis forming a tunnel filled with cellular debris and was covered with scale-crust (Figure 2A). Thick collagen fibers perforated through this tunnel. The area of perforation was not connected to the hair follicle (Figure 2B). The dermis showed proliferation of small blood vessels and mid perivascular mixed inflammatory cell infiltration, comprised of lymphocytes and few neutrophils. There was no granuloma infiltration. The histopathological findings were consistent with acquired reactive perforating collagenosis.



H&E, x40
Figure 2A

H&E, x100
Figure 2B

Figure 2A(x40) Perforation of the epidermis forming a tunnel filled with cellular debris and was covered with scale-crust. **Figure 2B(x100)** Thick collagen fibers perforated through this tunnel. The area of perforation was not connected to the hair follicle.

Further investigations were performed to identify the underlying etiology of acquired reactive perforating collagenosis. Chest radiography revealed widening of the mediastinum. Computed tomography (CT) of the chest demonstrated a large anterior mediastinal mass, approximately 8x10x8 cm. The mass encased the SVC with bilateral brachiocephalic veins and right superior lobar pulmonary artery without significant compression. There were multiple bilateral cervical and right cardiophrenic lymphadenopathies. Perilymphatic distribution of multiple tiny pulmonary nodules with peribronchovascular and interlobar-septal at RUL and peribronchovascular consolidations at anterior segment of RUL; concerning for lymphatic or lung involvement from mediastinal mass, especially lymphoma. CT of the whole abdomen revealed several subcentimeter and enlarged nodes at right cardiophrenic, gastrohepatic, retrocaval, and paraaortic regions, size 0.9-1.3 cm; could be lymphomatous involvement. Endobronchial ultrasound-guided biopsy (EBUS) showed atypical lymphoproliferative lesion : rare large B cell proliferative for PAX5, MUM1(focal), and CD45 (focal), negative for CD3, CD15, CD20, CD23, AE1/AE3. ALK-1 and EBER. The lymphoid cells were mixture of predominant small T cell (CD3+) and few small B cells (CD20+). B cell: T cell around 1:5. The suspicious rare large bizarre cells were positive for CD30. The diagnosis was Classic Hodgkin lymphoma. Bone marrow aspiration and biopsy revealed normal cellular trilineage marrow with maturation. Flow cytometry findings were within normal limits. Laboratory investigations showed hemoglobin 13.6 g/dL, hematocrit 41.7%, white blood cell count 12,950/μL (68% neutrophils, 20% lymphocytes), and platelet count 496,000/μL. Renal function tests were normal (BUN 7.4 mg/dL, creatinine 0.9 mg/dL), and liver enzymes were within normal limits. Serologic tests for HIV, HBsAg, anti-HBc, anti-HCV, and Quantiferon-TB were negative. Thyroid function and HbA1c were also within normal range.

Based on the clinical presentation and the results of laboratory and imaging investigations, the patient was diagnosed with acquired perforating dermatosis in association with Classic Hodgkin lymphoma Ann Arbor stage IIIB with bulky disease, International Prognostic Score (IPS) of 0.

Discussion

Perforating dermatoses are a group of papulonodular skin disorders characterized by transepidermal elimination of dermal material, they are divided into primary and secondary forms.[1] Primary perforating dermatoses are subdivided into Kyrle disease (KD), reactive perforating collagenosis (RPC), perforating serpiginous elastosis (PSE), and perforating folliculitis (PF).[1] The secondary form of the disease is known as acquired perforating dermatosis (APD).[1]

Clinically, ARPC presents as umbilicated necrotic papules or nodules with central keratotic plugs which are characteristic skin lesions of the condition. The papules gradually develop from pinpoint size to 5 to 6 mm in diameter, with a central concavity resembling a navel. The skin lesions would relieve spontaneously, with temporary hypochromic areas and atrophic scars left.[5] Lesions may be localized or generalized[6], usually distributing on the extremities and back, sometimes face and neck. Pruritus is common but Koebner phenomenon is rarely reported.[7]

Faver et al proposed the following diagnostic criteria for ARPC:

1. Transepidermal material elimination of dermal connective tissue
2. Keratotic plug at the center of umbilicated papules or nodules; and
3. The age of onset after 18 years old.[7]

Dermoscopy is a useful and rapid tool for diagnosing ARPC, characterized by a homogeneous yellowish-brown structureless area at the center of the lesion corresponding to the scale-crust; a whitish ring-shaped rim to the epidermal invagination (EI), the thickness of which varied; and the pink-white halo seemed to correspond to a combination of small blood vessels surrounding the lesion.[8]

Histopathological examination is diagnostic. Early lesions show epidermal hyperplasia, thickening of epidermis and widening of papillary dermis, with degenerated collagen bundles. Later lesions exhibit cup-shaped invagination filled with keratotic plug consisting of degenerated collagen, parakeratotic cells and cellular fragments can be seen. Necrotic basophilic collagen fibers are eliminated to the epidermis.[5] Collagen bundles can be highlighted by Masson's trichrome staining.[9]

Cases of Hodgkin lymphoma associated with ARPC have been reported in 1987, and 2016 encompassing three cases where manifestations of cancer and dermatosis were concurrent or where cancer preceded dermatosis.[3,10] Other hematologic malignancies reported in association with ARPC include non-Hodgkin lymphoma, mixed histiocytic lymphocytic lymphoma, myelodysplastic syndrome (MDS), acute myeloid leukemia (AML), and chronic lymphocytic leukemia (CLL).[11,12,13,6] Additional reported malignancies include hepatocellular carcinoma, prostate cancer, pancreatic cancer, undifferentiated adenocarcinoma, and renal cell carcinoma with liver metastasis.[14-18]

Several therapeutic options have been reported as first-line therapy in ARPC, including topical corticosteroids, local and systemic retinoids, and UVB phototherapy with variable outcomes. Photodynamic treatment, amitriptyline, and doxycycline have also been utilized.[19-21]

In this case, the patient received multimodal treatment. For the acquired perforating dermatosis, therapy included topical corticosteroids, oral prednisolone 15 mg/day for five days, antihistamines, acitretin 25 mg once daily for sixteen months, and anti-pruritic therapy with gabapentin 300 mg/day for one month, followed by pregabalin 150 mg/day for two months. Narrowband UVB (NBUVB) phototherapy was administered twice weekly for six months. The underlying Hodgkin lymphoma was treated with the ABVD (Adriamycin,

Bleomycin, Vinblastine, Dacarbazine) chemotherapy regimen. The patient demonstrated marked symptomatic improvement following treatment for Hodgkin lymphoma. Adjunctive and supportive dermatologic care resulted in reduced pruritus, with overall clinical improvement of the skin lesions observed within twelve months.

Conclusion

Acquired perforating diseases can be associated with malignancies. Early detected and prompt, thorough diagnostic workup is essential for identifying the underlying causes of these conditions.

Conflict of interest

The authors have no relevant conflicts of interest to disclose.

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