Dermatology Practical & Conceptual

Granulomatous Dermatitis Characterized by the Manifestation of Tumor and Plaque Lesions Subsequent to Herpes Zoster: A Case Series

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Introduction

Herpes zoster (HZ) is a painful vesicular dermatomal exanthem caused by the reactivation of the latent varicella zoster virus in cutaneous sensory nerves. It is more common in immunocompromised patients and can lead to complications like postherpetic neuralgia and secondary bacterial infections [1]. Postherpetic isotopic response (PHIR) is a rare chronic cutaneous reaction that can cause new skin diseases independent of previous ones [2]. Herein, we report two cases of PHIR-granulomatous disease (GD) in patients with different hematological diseases.

Case Presentations

Case 1: An 87-year-old male patient with a history of chronic lymphocytic leukemia, benign prostatic hyperplasia, and hypertension presented with painless bruising and swelling on the inner side of his left thigh for nine months. The patient stated that he had an HZ infection 1.5 years ago, and the affected area matched the current lesion area. The lesion was a 10 x 10 cm tumor with a brown livid color and a pendulous appearance. A dermatological examination revealed a brown-purple flaccid tumor on the medial aspect of the left thigh along with keratotic plugs on a yellowish background

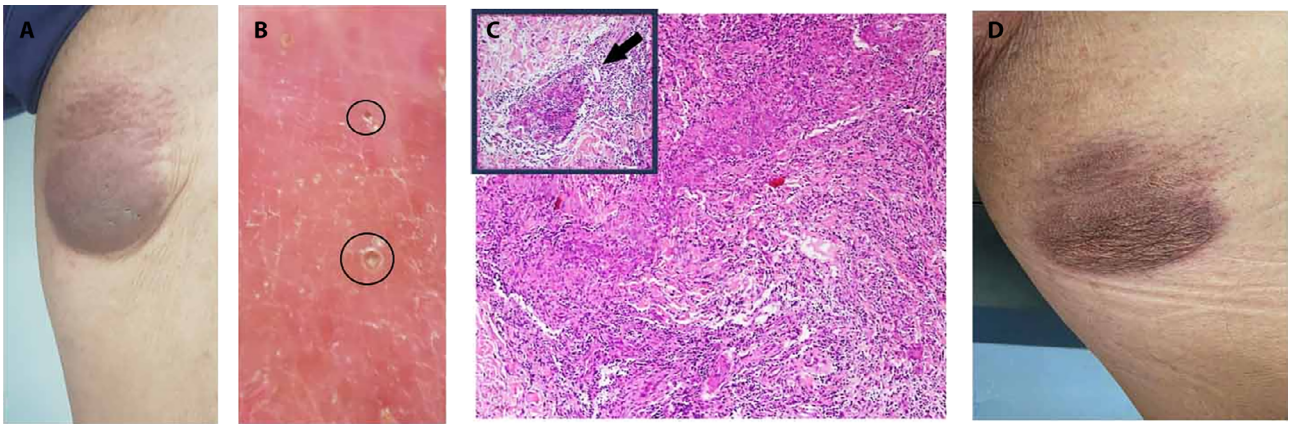

Figure 1. (A) A 10x10 cm brown-livid tumoral lesion on the left medial thigh of an 87-year-old male patient (Case 1). (B) Dermoscopy showed keratotic plugs on yellowish background (black rings). (C) Histopathological examination revealed mononuclear inflammatory cell infiltration with granuloma structures localized in the papillary dermis, where epithelioid histiocytes form, destroying the follicle, and multinucleated giant histiocytes scattered around (H&E stain, ×100; black arrow) and a granuloma structure formed by epithelioid histiocytes localized in the papillary dermis (H&E stain, ×200). (D) Regression of tumoral lesion after intralesional steroid therapy.

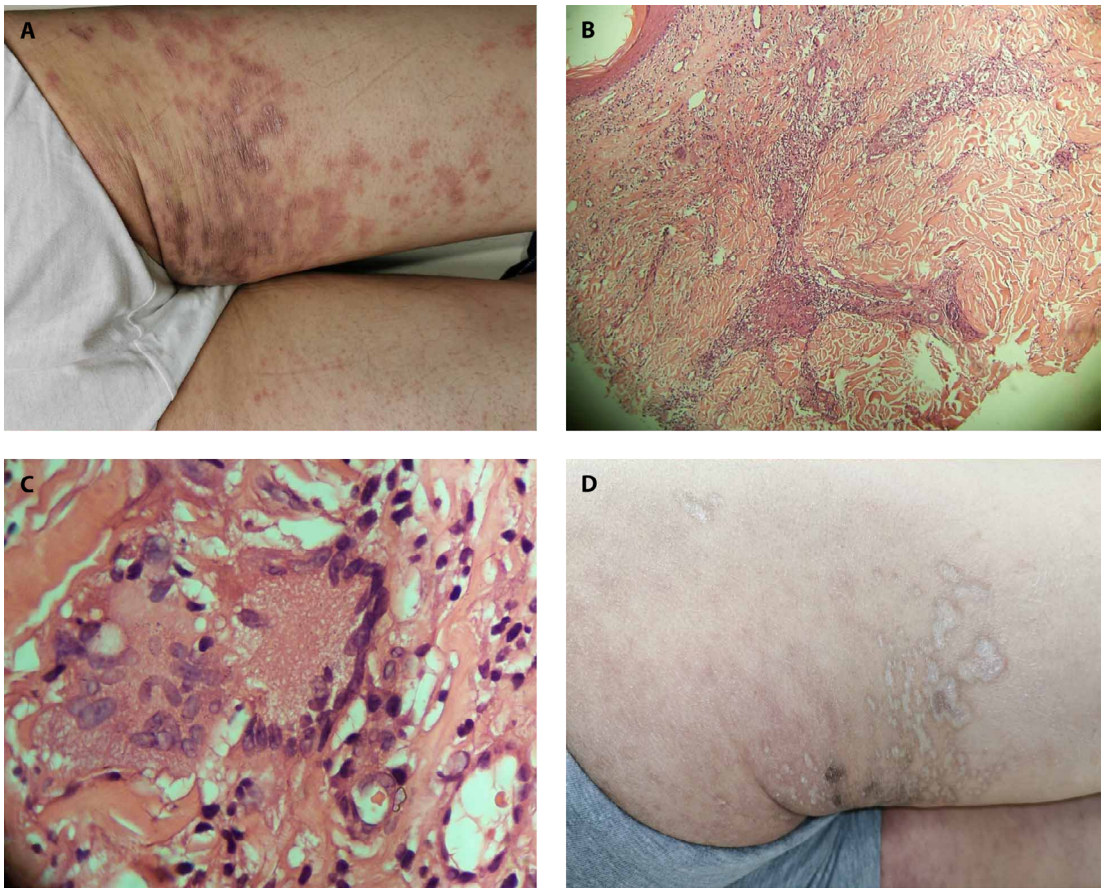

Figure 2. (A) Erythematous papules and plaques on the posterior aspect of the right thigh of a 68-year-old male patient (Case 2). (B) Histopathological examination showed granuloma structure in the dermis and (C) multinuclear giant cells phagocytosing fragmented elastic fibers. (D) Improvement of granulomatous lesions with atrophic scar after topical corticosteroid therapy. (F) H&E stain, ×100. (G) H&E stain, ×1000.

(Figure 1, A and B). The differential diagnosis included B-cell lymphoma, anaplastic large cell lymphoma, granulomatous loose skin syndrome, and tuberculosis based on the clinical presentation. Histopathological examination of the lesion revealed well-formed granuloma structures, mononuclear inflammatory cell infiltration, and fragmented elastic fibers (Figure 1C). Immunohistochemical studies showed CD20+, CD2, CD3, CD5, CD7, CD8, and CD4+. The patient's PPD

was 0 mm, and the Quantiferon test was negative, as were the tuberculosis polymerase chain reaction and deep fungal culture. PHIR-GD was diagnosed. The patient received intralesional steroid injections once a month for four months, and after significant regression of the lesions, topical steroids were continued (Figure 1D).

Case 2: A 68-year-old male patient with myelodysplastic syndrome presented with a painless rash on his right buttock and thigh for six months. He had had an HZ infection two years prior, which matched the current lesion area. Dermatological examination revealed erythematous papules and plaques on the right gluteal area and posterior aspect of the thigh (Figure 2A). Histopathological examination revealed a compact hyperkeratosis layer, acanthosis in the epidermis, flattening of rete projections, scattered eosinophil exocytosis, vacuolization, focal subepidermal separation in the basal layer, and granulomatous lymphohistiocytic infiltration in the dermis. While collagen degeneration and mucin deposition, which support interstitial granulomatous drug-related dermatitis, were not observed in this case, multinucleated giant cells that phagocytosed elastotic fibers were detected (Figure 2A and B). The patient was diagnosed with PHIR-GD and started on clobetasol propionate ointment, which resolved within two months with atrophic scars (Figure 2D).

Conclusions

Wolf's isotopic response is a phenomenon where a healed skin condition reappears at the same site, causing a new condition. The cause is unknown, but it is suggested to be an immunosuppressed region [1-3]. This area becomes susceptible to infections, tumors, and immune disorders. Chronic lymphedema, herpetic infections, vaccinations, and physical injuries can damage the cutaneous site, making it vulnerable. The most common isotopic response is PHIR, which is characterized by papules or plaques, patchy or nodular lesions, and hyperpigmentation [1-4]. Most cases have immunosuppressive and hematological malignancies, and both cases have hematological malignancies [1-5]. Topical steroids are commonly used in treating PHIR-GD cases [1-5], but no case has been treated with intralesional steroids. In conclusion, PHIR-GD can cause tumoral lesions, and intralesional corticosteroid therapy can be used for tumor management.

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