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Acquired Reactive Perforating Collagenosis in Two Patients with Type 2 Diabetes: A Clinicopathological Correlation

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Abstract:

Acquired reactive perforating collagenosis (ARPC) is a rare perforating dermatosis characterized by transepidermal elimination of altered collagen fibers. It is most commonly associated with diabetes and chronic kidney disease and may be underrecognized in patients with chronic pruritus. We report two cases of ARPC in women aged 74 and 60 years with type 2 diabetes who presented with severe chronic pruritus and multiple hyperkeratotic umbilicated papules. In the first patient, dermoscopy demonstrated crateriform lesions with a central keratotic plug, and histopathology revealed transepidermal elimination of degenerated collagen fibers. In the second patient, the diagnosis was suspected clinically and confirmed histologically using special stains. Clinicopathological correlation established the diagnosis in both cases. Treatment with systemic and topical therapies resulted in symptomatic improvement. ARPC is an uncommon and probably underdiagnosed dermatosis strongly associated with metabolic disorders, particularly diabetes and chronic kidney disease. These cases emphasize the importance of integrating clinical, dermoscopic, and histopathological findings to distinguish ARPC from other causes of chronic pruritus. ARPC should be considered in diabetic patients presenting with chronic pruritus and crateriform hyperkeratotic papules. Early recognition may facilitate timely diagnosis and optimization of associated systemic diseases.

Keywords:

Reactive Perforating Collagenosis; Diabetes, Type 2; Pruritus; Perforating Dermatoses; Collagen

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Introduction

Acquired reactive perforating collagenosis (ARPC) is a rare perforating dermatosis characterized by the transepidermal elimination of altered dermal collagen [1,2]. Two clinical forms have been described: hereditary and acquired. The acquired form occurs predominantly in adults and is commonly associated with systemic diseases,

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particularly diabetes and chronic kidney disease [1,2]. Faver et al. proposed diagnostic criteria that include onset after 18 years of age, umbilicated papules or nodules with a central keratotic plug, and histopathological evidence of transepidermal elimination of necrotic collagen [1,2].

The epidemiology of ARPC remains incompletely defined because of its rarity and probable underdiagnosis [1,2]. It predominantly affects middle-aged and older adults, with a peak incidence between 50 and 59 years of age [2]. Several studies have shown a strong association with diabetes, chronic kidney disease, and hypertension [1,2,3]. Other reported associations include autoimmune diseases, malignancies, liver disorders, and thyroid diseases [2]. Although uncommon, ARPC is clinically relevant because its cutaneous manifestations may serve as a marker of underlying systemic disease and metabolic dysregulation.

The pathophysiology of ARPC has not been fully elucidated. Repeated superficial trauma and scratching secondary to pruritus may induce degeneration of papillary dermal collagen followed by transepidermal elimination [1,2,3]. In diabetic patients, oxidative stress, advanced glycation end products, and microangiopathy may contribute to connective tissue damage [1,2]. Overexpression of transforming growth factor beta-3 (TGF-β3) and extracellular matrix proteins has also been demonstrated in affected lesions [1].

Clinically, ARPC presents as hyperkeratotic, pruritic, umbilicated papules or nodules with a central keratotic plug, predominantly affecting the extremities and trunk [1,2,4]. Histopathology shows a cup-shaped epidermal invagination containing keratin, inflammatory debris, and vertically oriented degenerated collagen fibers, which can be highlighted using special stains [1,4,5].

The importance of accurately identifying ARPC lies in its association with prevalent systemic diseases, particularly type 2 diabetes and chronic kidney disease [2], while also considering less common associations such as rheumatoid arthritis, hypothyroidism, and hypertensive heart disease [6,7]. We present two cases of ARPC in patients with type 2 diabetes, highlighting their clinical, dermoscopic, and histopathological findings.

Case Presentation

Case 1

A 74-year-old woman with a medical history of hypertension and type 2 diabetes under treatment presented with a dermatosis of approximately four months' duration. The patient reported the progressive appearance of cutaneous lesions on the trunk and extremities associated with intense and persistent pruritus. She had not responded to multiple treatment regimens, including topical and systemic antihistamines, as well as topical clioquinol and fluocinolone. As a relevant clinical finding, the patient described the extrusion of a "small black particle" after scratching, accompanied by worsening of symptoms.

Physical examination revealed a disseminated dermatosis characterized by multiple excoriated erythematous papules distributed over the trunk and extremities. Some lesions on the dorsal region remained intact and exhibited a hyperkeratotic scaly surface with a firm consistency. Multiple excoriation marks secondary to scratching were also observed. Dermoscopic examination demonstrated crateriform papular lesions with a central keratotic plug (Figure 1). Initially, the differential diagnosis included metabolic prurigo and acquired perforating disease; therefore, a skin biopsy was performed, revealing findings consistent with acquired reactive perforating collagenosis (Figure 2).

Initial treatment consisted of oral prednisone in a tapering regimen combined with urea-based emollients, resulting in improvement of both the lesions and pruritus. The patient currently remains on low-dose prednisone and topical retinoic acid, with sustained symptomatic improvement.

Case 2

A 60-year-old woman with a history of type 2 diabetes treated with insulin presented with a disseminated dermatosis of approximately five months' duration associated with intense and persistent pruritus.

Physical examination revealed a dermatosis predominantly involving the upper and lower

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extremities, with greater involvement of extensor surfaces and areas readily accessible to scratching. The anterior and posterior trunk were spared; however, extensive involvement of the gluteal region was observed. Clinically, the dermatosis consisted of multiple dark-brown hyperpigmented papules, some excoriated or decapitated, while others displayed a central pustular appearance and crateriform morphology. The lesions were indurated and distributed either individually or with a tendency to coalesce in certain areas (Figure 3).

Based on the clinical characteristics of the lesions and their association with type 2 diabetes and severe pruritus, a clinical diagnosis of acquired perforating disease was established. Histopathological examination confirmed the diagnosis of reactive perforating collagenosis by demonstrating transepidermal elimination of collagen fibers (Figure 4). The patient was treated with topical corticosteroids and emollients, with partial improvement of pruritus and stabilization of skin lesions during follow-up.

Discussion

Since its first description by Mehregan et al. in 1967, acquired reactive perforating collagenosis has remained an uncommon and probably underdiagnosed condition [8]. Its close association with highly prevalent systemic diseases, such as type 2 diabetes, is reflected in the two cases presented herein. For internists, recognition of ARPC is particularly relevant because chronic pruritus is frequently attributed to diabetic xerosis, chronic kidney disease–associated pruritus, or drug-related causes, potentially delaying diagnosis and appropriate dermatologic evaluation.

Chronic pruritus is a common symptom in patients with diabetes and is frequently attributed to xerosis, neuropathy, or metabolic dysregulation. Consequently, uncommon entities such as acquired reactive perforating collagenosis may remain unrecognized for prolonged periods. Both of our patients experienced months of refractory pruritus before diagnosis, illustrating the importance of careful dermatologic examination and biopsy when characteristic lesions are present [2,3].

Both patients fulfilled the diagnostic criteria proposed by Faver et al. in 1994: (1) histopathological evidence of transepidermal elimination of necrotic collagen through a cup-shaped epidermal depression, (2) clinical presentation of umbilicated papules or nodules with an adherent central keratotic plug, and (3) onset of cutaneous lesions after 18 years of age [9]. The correlation between clinical morphology and histopathological findings was essential for establishing the diagnosis in both cases.

From an epidemiological perspective, both cases are consistent with the typical age profile of ARPC, occurring in patients aged 60 and 74 years [10]. Although some studies suggest a male predominance, sex distribution remains variable among published case series [10]. This variability suggests that ARPC is not restricted to either sex and that its development may be more closely related to the presence of systemic comorbidities than to patient sex.

Available evidence indicates that most reported cases of ARPC occur in patients with underlying systemic diseases, whereas its development in otherwise healthy individuals is exceedingly rare [10]. Although diabetes is the most commonly reported associated condition, coexistence with malignancies, nephropathies, infections, autoimmune disorders, and thyroid diseases has also been documented. In our patients, the shared feature was type 2 diabetes and chronic pruritus.

Type 2 diabetes likely acted as the principal predisposing factor. Advanced glycation end products (AGEs) and overexpression of their receptors in dermal cells have been proposed to contribute to connective tissue damage and collagen degeneration, explaining the association between ARPC, diabetes, aging, and chronic kidney disease [10]. Persistent scratching may further promote epidermal disruption and transepidermal elimination of altered collagen fibers [10].

No standardized treatment for ARPC currently exists. Variable responses have been reported with corticosteroids, retinoids, phototherapy, keratolytic agents, and antipruritic therapies. Relapses are common, emphasizing the chronic nature of the disease and the importance of optimizing control of associated systemic conditions [2,10].

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Take-Home Messages

Acquired reactive perforating collagenosis should be considered in diabetic patients presenting with chronic refractory pruritus and hyperkeratotic crateriform papules.
Dermoscopy may facilitate recognition of characteristic lesions and guide biopsy.
Histopathological confirmation remains essential for diagnosis.
ARPC may serve as a cutaneous marker of underlying metabolic disease.
Early diagnosis may improve symptom control and reduce diagnostic delay.

Conclusion:

Acquired reactive perforating collagenosis is an uncommon and likely underrecognized dermatosis that should be considered in patients with type 2 diabetes who present with chronic refractory pruritus and characteristic hyperkeratotic umbilicated papules. The present cases highlight the diagnostic value of integrating clinical examination, dermoscopy, and histopathological evaluation to establish an accurate diagnosis and distinguish ARPC from other causes of chronic pruritus. Early recognition may facilitate appropriate dermatologic management and prompt evaluation and optimization of associated systemic diseases, particularly diabetes and chronic kidney disease.

Declarations

Declaration of Competing Interests: The authors declare no competing interests.

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Ethics Approval and Patient Consent:

Written informed consent was obtained from both patients for publication of this case report and accompanying images. According to the regulations of the IMSS General Zone Hospital with Family Medicine Unit No. 8, institutional ethics committee approval was not required for publication of this case report, provided that written informed consent was obtained.

Author Contributions:

Emanuel Chew: Conceptualization, data collection, literature review, manuscript drafting. Jose Castillo: Clinical management, manuscript review and editing. Paola Elizalde: Literature review, manuscript editing. Ana Dominguez: Dermatologic evaluation, histopathological correlation, supervision, manuscript review.

Data Availability:

The data supporting the findings of this report are available from the corresponding author upon reasonable request.

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Figure Legends

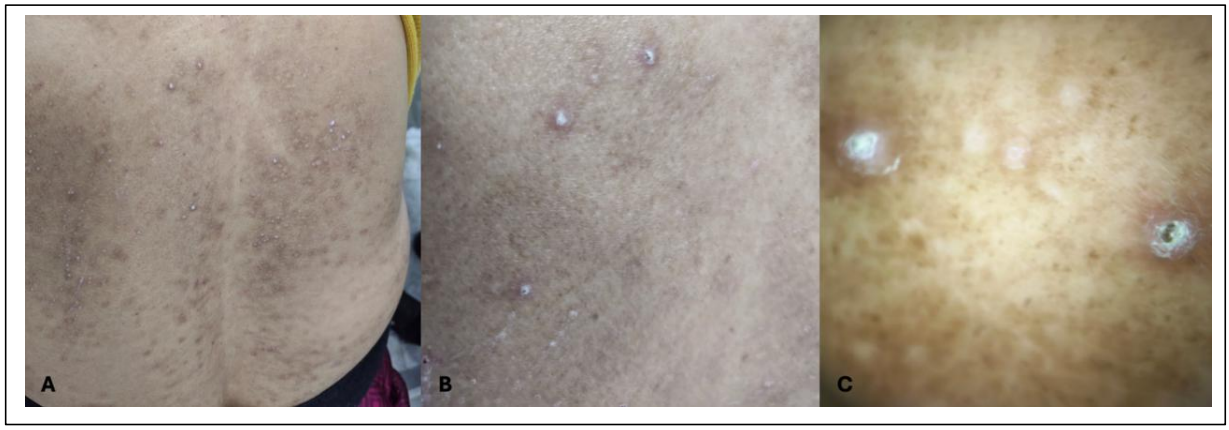

Figure 1. Clinical and dermoscopic manifestations. (A) Disseminated dermatosis on the dorsal region characterized by multiple hyperpigmented and excoriated papules. (B) Close-up view of umbilicated papular lesions with a keratotic center and crateriform morphology. (C) Dermoscopy showing a central keratotic plug surrounded by a hyperpigmented halo.

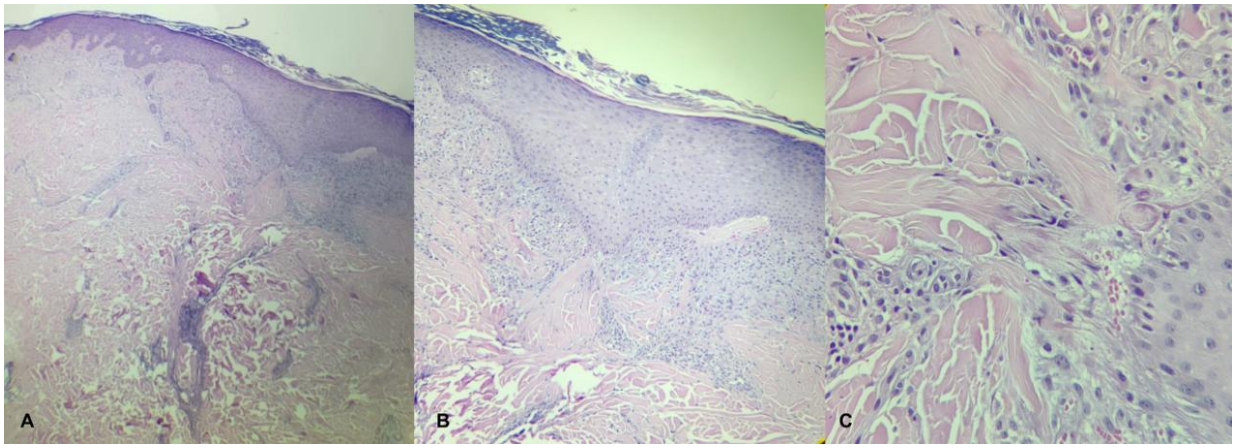

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Figure 2. Histopathological findings. (A) Low-power view showing reactive epidermal hyperplasia with a central depression and remnants of eliminated fibers within the stratum corneum. (B) Intraepidermal perforating channel through which degenerated collagen fibers are eliminated. (C) Vertically oriented collagen fibers extending toward the epidermis. Hematoxylin and eosin stain

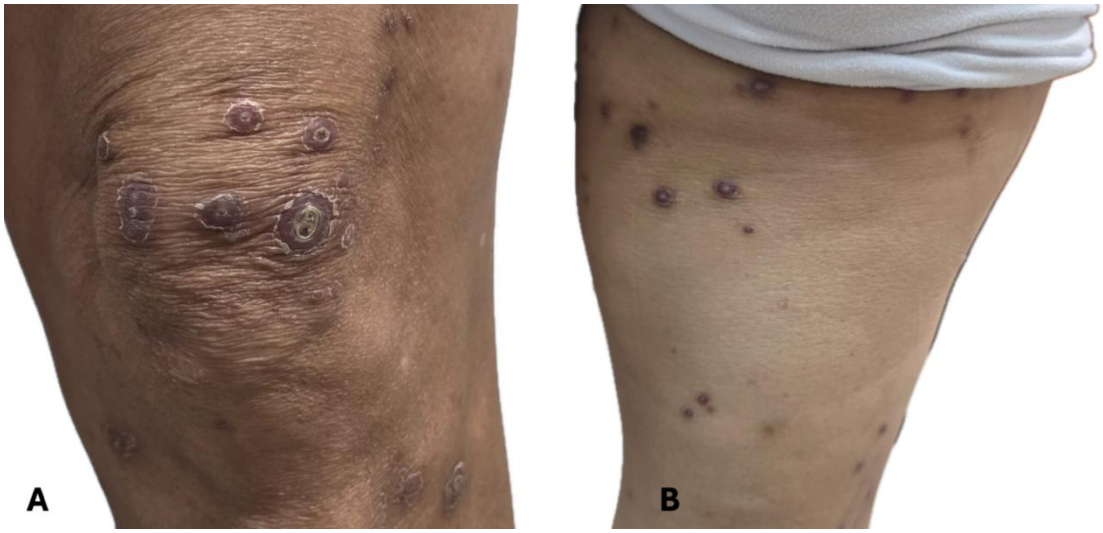

Figure 3. Clinical manifestations. (A) Multiple hyperpigmented crateriform papules with a central keratotic plug on the extensor surface of the knee, some showing a tendency to coalesce. (B) Hyperpigmented papular lesions distributed over the gluteal region, exhibiting umbilicated morphology and a central keratotic core.

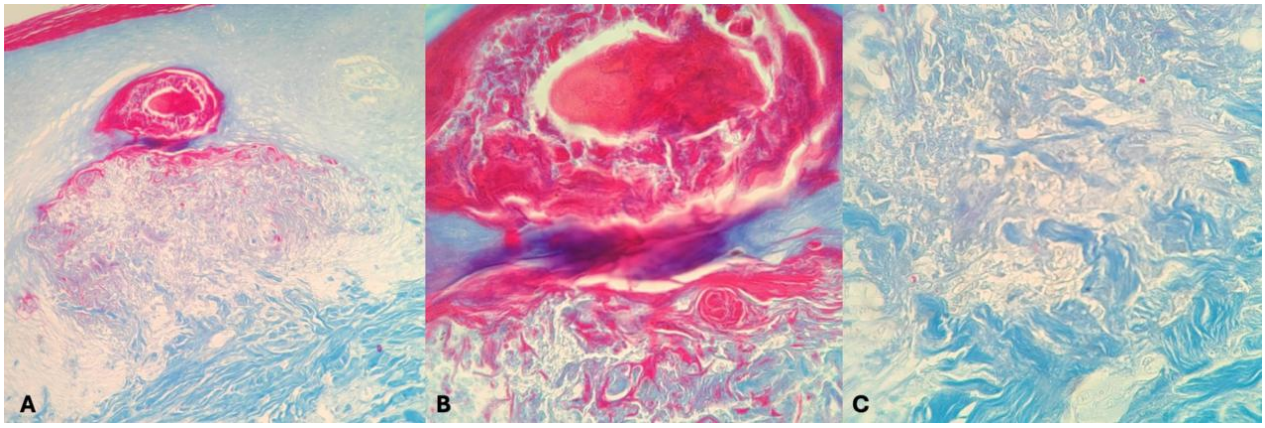

Figure 4. Histopathological findings. (A) Low-power view showing vertically oriented collagen fibers penetrating the epidermis, with debris accumulating within a central channel. (B) Higher magnification demonstrating degenerated collagen remnants and accumulated debris within the channel. (C) Vertically oriented collagen fibers. Masson's trichrome stain.