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Case Report
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Acquired Reactive Perforating Collagenosis in Patient with Diabetic Chronic Kidney Disease: A Case Report

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Abstract

Perforating dermatoses are a group of skin diseases characterized by transepidermal elimination of dermal material. The disease is divided into two groups: the inherited group and the acquired group. Acquired reactive perforating dermatoses (ARPC) is an uncommon skin disorder seen in patients with diabetesmellitus, chronic kidney disease, or both together. We present the clinicopathological features of ARPC in a patient with diabetic kidney disease and discuss the recent therapy.

Keywords: Reactive perforating collagenosis; Chronic kidney disease; Diabetes mellitus

Introduction

The perforating dermatoses represent a group of skin disorders characterized by the perforation, or elimination, of dermal connective tissue through the epidermis. Although several classifications exist, there are four major primary perforating disorders grouped according to types of epidermal disruption, nature of eliminated material, and clinical features [1]. There are two types of reactive Perforan Collagenosis (RPC), Acquired RPC (ARPC) and Inherited RPC (IRPC). IRPC is more common in infants and children; while ARPC usually develops in adulthood in association with variety of systemic disorders, such as Type 2 Diabetes Mellitus (T2DM), Chronic Kidney Disease (CKD), hypertension, even malignant neoplasms [2]. Here, we reported a rare case of 57-year-old man who presented ARPC associated with three different systemic diseases including diabetes mellitus, chronic kidney disease and congestive heart failure.

Case Presentation

A 57-year-old male patient applied to emergency unit with shortness of breath, edema and generalized pruritus. He had a history of type 2 diabetes mellitus for 10 years, congestive heart failure and hypertension. His blood glucose was regulated with insulin at home. At the time of hospital admission her blood glucose was: 160 mg/dL, creatinine: 7.68 mg/dL, CRP: 73.69 mg/L, albumin: 2.84 mg/dL. His calcium and phosphorus results were detected 7.2 mg/dL and 6.6 mg/dL respectively. His ProBNP was >35000 pg/mL. The patient was admitted to nephrology clinic with pre-diagnoses of chronic kidney disease and diabetic nephropathy. The patient's proteinuria was 2.5 g/day and bilateral diabetic retinopathy was detected in fundus examination. The patient had multiple moderately itchy lesions for six years. Dermatologic examination showed disseminated red or brown umbilicated papules, plaques or nodules between 2 mm to 3 cm diameters on extremities and trunk. There were hemorrhagic and necrotic crusts or keratin plug on the centre of these lesions (Figure 1). A punch biopsy was performed by dermatologist from one of the lesions. Histopathologically; neutrophils, basophilic material consisting of keratin material and necrotic debris, and epidermal ulceration were seen. Masson trichromestaining showed transepidermal elimination of collagen fibers (Figure 2, 3). Based on the clinical and histopathological findings, the patient was diagnosed with acquired reactive perforating collagenosis. Narrow-band ultraviolet B three times a week, oral anti-histamine and topical steroid were started as treatment for the patient.

Discussion

Acquired Perforating Dermatosis (APD) is an all-encompassing term that describes the various types of perforating diseases that arise in adults, often related to an underlying systemic disease, most commonly diabetes mellitus and chronic renal failure [3,4]. APD encompasses acquired

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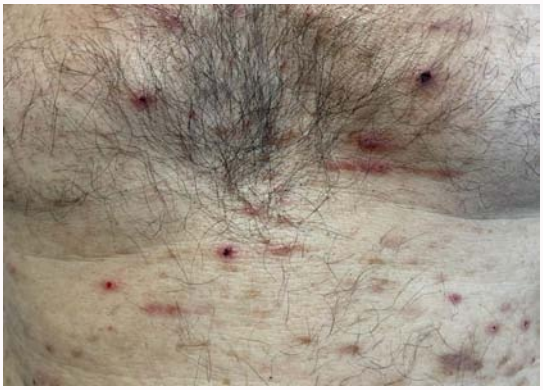

Figure 1A: Umbilicated papules and nodules with necrotic crusts on chest.

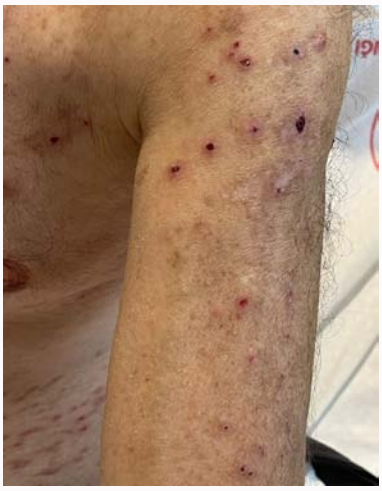

Figure 1B: Umbilicated papules and nodules with necrotic crusts on and upper extremity.

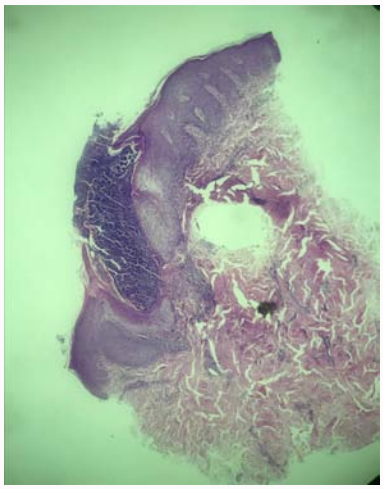

Figure 2: Mixed inflammatory cell predominantly neutrophils under the stratum corneum, basophilic material consisting of keratin material and necrotic debris, epidermal ulceration (H&E x4).

Reactive Perforating Collagenosis (RPC), Acquired Elastosis Perforans Serpiginosa (EPS), acquired Perforating Folliculits (PF), and classically described Kyrle disease, though among authors the classification is debated [1]. Reactive perforating collagenosis was first described in 1967, though the prevalence and incidence of RPC

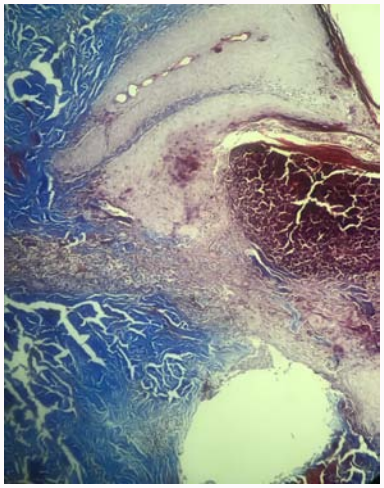

Figure 3: Transelimination of collagen fibers from the ulcerated epidermis (Masson trichrome x10).

are unknown, scattered cases have been reported [5,6].

Itching, which was also seen in our patient, was the most common symptom. The severity of pruritus was shown to be correlated with the severity of illness, response to therapy, and may be aggravated by coexisting secondary hyperparathyroidism [7,8].

Pathophysiology is still uncertain, but it is believed that chronic pruritus in predisposed patients may cause the rupture of collagen fibers with consequent elimination [9-11]. Diabetic microangiopathy may contribute to collagen damage and to the microdeposition of substances that are not removed by dialysis, causing local inflammatory reactions [9-11].

Extremities were the most commonly involved site, followed by the back. The lesions were distributed mostly on trauma-prone areas, with some unusual sites like the scalp and the nape of the neck [12]. As we saw in our patient, in many patients had evidence of co-existing microvascular and macrovascular disease [12].

The therapy involves measures to reduce itching like antihistaminics, topical retinoids, glucocorticoids, and keratolytic agents. Systemic therapy includes retinoids, glucocorticoids, and phototherapy. Narrow-band ultraviolet B phototherapy is effective because of the frequent concomitant uremic pruritus [13].

Conclusion

Acquired perforating dermatoses should be considered when papules, plaques or nodules with necrotic crusts or keratin plug are found in patients with predisposing diseases like diabetes and kidney disease, or both together. Multidisciplinary involvement is essential for early diagnosis by histopathology and immediate treatment.

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