Clinical and dermoscopic correlation of reactive perforating collagenosis

Correlação clínica e dermatoscópica da colagenose perfurante reativa

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ABSTRACT

The acquired perforating dermatosis is constituted by a group of diseases whose main characteristic is the transepithelial elimination of altered dermal material. It has the clinical appearance of crateriform papules or nodules, with central keratotic plug, which may suggest some kind of transepithelial purging; nevertheless the final diagnosis is histologic. The dermoscopic examination is a useful tool that helps to exclude similar dermatoses, such as nodular prurigo and hypertrophic lichen planus. The present article discusses the clinical and dermoscopic correlation of this dermatosis.

Keywords: dermoscopy; prurigo; diagnosis, differential

RESUMO

A dermatose perfurante adquirida é formada por um grupo de dermatoses que tem por característica a eliminação transepitelial de material dérmico alterado. Clinicamente se apresenta como pápulas ou nódulos crateriformes, com tampão queratótico central, o que pode sugerir algum tipo de eliminação transepitelial; porém o diagnóstico final é histopatológico. A realização da dermatoscopia é ferramenta útil, ajudando a afastar outras dermatoses similares, como o prurigo nodular e o líquen plano hipertrófico. Discute-se neste artigo a correlação clínica e dermatoscópica dessa dermatose.

Palavras-chave: dermoscopia; prurigo; diagnóstico diferencial

Diagnostic imaging

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INTRODUCTION

Acquired perforating dermatosis (APD) constitutes a group of dermatoses that are characterized by transepithelial elimination of altered dermal material (collagen, elastic fibers and/or hair follicle). ¹⁻³ It occurs in adulthood and is commonly associated with diabetes mellitus and chronic kidney disease (up to 10% of patients undergoing dialysis). It is also found in association with liver failure, hypothyroidism, hyperparathyroidism, pulmonary fibrosis, HIV/AIDS, scabies and malign neoplasias. ⁴

From the clinical point of view, there is emergence of papules or crateriform nodules with a central keratotic plug that may have follicular pattern. The most affected body sites are the extensor surface of the limbs and the trunk. Itching is common, with frequent excoriations and crusting. New lesions can arise through the Koebner phenomenon.²

In light of histologic findings, APD can be classified into: reactive perforating collagenosis, serpiginous perforating elastosis, perforating folliculitis and Kyrle's disease. There can be superposition of more than one type in the same patient, the coexistence being more common in dialysis patients. In this way, it is recommended that more than one biopsy be carried out.¹

There is no established standard treatment; the most frequently mentioned in the literature are: topical/intralesional corticosteroid, retinoid, allopurinol and phototherapy. Where there is an underlying disease, its control can lead to the improvement of APD. Pruritus can be controlled with anti-histamines.

In this paper, the authors describe two cases of APD, correlating clinical and dermoscopic aspects.

CASE REPORTS

Case 1: A 63 year-old male patient experienced the abrupt onset of intensely pruritic lesions on the dorsum, which quickly progressed to the cervical region and proximal members in two weeks. The dermatological examination revealed: multiple umbilicated papules and nodules, of brownish color and

varying sizes, some of follicular pattern, with central keratotic plug (Figure 1). Comorbidities: hypertension, diabetes mellitus, ischemic heart disease, peripheral vascular disease and chronic renal insufficiency/dialysis. The dermoscopic examination with polarized light allowed observing a peripheral brown-grayish amorphous area with a white keratotic center, with depressed appearance and presence of a yellowish area (Figures 2 and 3). The histology of the specimen (obtained through a fusiform excision on the dorsum) showed continuity in the papillary dermis, covered by a thick necrotic crust containing cellular debris, neutrophils, verticalized collagen fibers with transepithelial elimination, characterizing a reactive perforating collagenosis (RPC) (Figure 4). The patient was treated with 0.05% betamethasone dipropionate and topical 3% salicylic acid, in addition to 15mg/



FIGURE 2: Dermoscopy of the old lesion: brown-grayish amorphous area on the periphery with white keratotic center of depressed appearance and presence of a yellowish area



FIGURE 1: Multiple papules and crateriform nodules with central keratotic plug in the dorsum



FIGURE 3: Dermoscopy of the initial lesion: amorphous brown-grayish area on the periphery, discreet keratotic white center and yellowish area

Perforating collagenosis 63

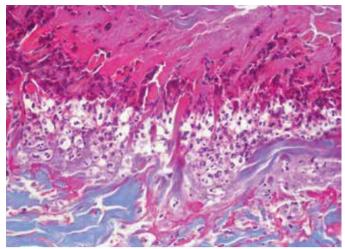


FIGURE 4: Segment with continuity solution of the dermis covered by fibrinleukocyte crust with cellular debris and elimination of fibers (verticalized) (Masson's trichrome staining, 200x)

day mirtazapine for controlling the pruritus. It showed improvement in 30 days.

Case 2: A 46 year-old male patient reported the appearance of pruritic lesions in the upper limbs six months before. The dermatological examination revealed multiple brownish papules with central keratotic plug (Figure 5). Background: hypertension, diabetes, chronic renal failure requiring dialysis. Dermoscopy with polarized light revealed a peripheral erythematous-grayish amorphous area, with white keratotic center, depressed appearance and a small yellow-brownish area. The histology showed similar findings to those of the previous case. The patient was treated with betamethasone valerate twice a day, with improvement of the picture and the absence of new lesions.



FIGURE 5: Multiple papules with central keratotic plug on the dorsum of the hand

DISCUSSION

Reactive perforating collagenosis is characterized by the transepidermal elimination of collagen. It can be primary (a possible autosomal recessive inheritance) or secondary (acquired), associated with systemic diseases. The clinical and epidemiological findings in the present report were consistent with the literature (adult diabetic and renal patient, requiring dialysis, with multiple keratotic, umbilicated, pruritic papules prevalent in the trunk).

The pathogenesis is not known and alterations in fibroelastic bundles can be partly explained by the diabetes. In addition, trauma would be another contributing factor, as the Koebner phenomenon is observed in sites with injuries, insect bites, herpes zoster and acne scars.³

The final diagnosis was confirmed by the histology, with the observation of a cupuliform depression filled with degenerated basophilic material, and the presence of foci of ulceration and vertically oriented collagen fibers crossing the dermal-epidermal junction that are better visualized using the Masson's trichrome staining (it stains the collagen in blue). In addition to the biopsy, performing dermoscopy is a useful tool, helping to exclude similar dermatoses, such as nodular prurigo and hypertrophic lichen planus. According to Errichete et al., the white starburst pattern (white lines arranged radially) on erythematous-brownish background is seen in nodular prurigo, while there is presence of whitish crossed lines (Wickham striae) in hypertrophic lichen planus.⁵ In the authors' dermoscopic analysis, it was possible to observe a central white area, yellow plugs of keratin with variable size (possibly corresponding to the area of fibrosis and extrusion of dermal material), a keratotic collarette with a brown-grayish amorphous or erythematous area surrounding it, possibly due to inflammatory reaction. The absence of other described patterns helped the authors to exclude those dermatoses.

With the two cases described in the present paper, the authors highlight the peculiar clinical presentation of perforating dermatosis and the importance of dermoscopy as an auxiliary method in the diagnosis.

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