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Abstract:

Acquired Perforating Dermatitis (APD) is a perforating disease characterized by transepidermal elimination of dermal material [1,2]. This disease usually develops in adulthood. APD has been reported to occur in association with various diseases, but is most commonly associated with dialysis-dependent chronic renal failure (CRF) or diabetes mellitus (DM) [1,2,3,4]. Morton et al found that APD occurs in up to 10% of patients undergoing hemodialysis [5]. Additionally, Saray et al found that sixteen of twenty-two cases with APD were associated with CRF [3].

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Case Report

Acquired perforating dermatosis: a clinical and dermatoscopic correlation

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Abstract

Acquired perforating dermatosis (APD) is a perforating disease characterized by transepidermal elimination of dermal material. This disease usually develops in adulthood. APD has been most commonly associated with dialysis dependent chronic renal failure or diabetes mellitus. We present a case of a 65-year-old female with a 6-month history of multiple hyperpigmented, follicular keratotic papulonodular lesions located on the lower extremities. Histologic examination demonstrated perforating folliculitis, consistent with the diagnosis of perforating dermatosis. Dermatoscopic imaging with polarized light supported the histological findings. Clinical and histological presentation of APD may vary vastly from patient to patient. Lesions within the same patient may differ by the dermal component that is being transepidermally extruded. For these reasons, authors have suggested multiple biopsies. We have found the utility of dermatoscopy in visualizing gross histologic features. Dermatoscopy allows for improved in vivo visualization and may aide in distinguishing APD from simple prurigo nodularis.

Introduction

Acquired Perforating Dermatitis (APD) is a perforating disease characterized by transepidermal elimination of dermal material. This disease usually develops in adulthood. APD has been most commonly associated with dialysis dependent chronic renal failure or diabetes mellitus. We present a case of a 65-year-old woman with a 6-month history of multiple hyperpigmented, follicular, keratotic papules and nodules located on the lower extremities. Histologic examination demonstrated perforating folliculitis, consistent with the diagnosis of perforating dermatosis. Dermatoscopic imaging with polarized light supported the histological findings. Clinical and histological presentation of APD may vary vastly from patient to patient. Lesions within the same patient may differ by the dermal component that is being transepidermally extruded. For these reasons, authors have suggested multiple biopsies. We have found the utility of dermatoscopy in visualizing gross histologic features. Dermatoscopy allows for improved in vivo visualization and may aide in distinguishing APD from simple prurigo nodularis.

Case synopsis

A 65-year-old woman presented with multiple hyperpigmented, follicular, keratotic, papules and nodules (Figure 1AB), which were moderately pruritic. The majority of these lesions were located on the lower extremities and had persisted for 6 months prior to presentation. Her medical history included insulin-dependent diabetes, hypothyroidism, dyslipidemia, high blood pressure, and CRF for which she was receiving peritoneal dialysis.



Figure 1AB: Gross features four months after initial presentation. A. Hyperkeratotic, hyperpigmented papules with central keratinaceous crust (gross imaging) B. Hyperkeratotic, hyperpigmented papules with central keratinaceous crust (macro setting)

Biopsy of a representative lesion from the thigh revealed a distorted hair follicle that angled horizontally from the epidermis (Figure 1A). The lumen of the follicle was dilated and contained abundant parakeratotic keratinocytes and basophilic debris (Figure 1B). There was plugging of the follicular ostium by this same debris. Lichen simplex chronicus (LSC) was also present, including dense orthokeratosis, acanthosis, irregular elongated rete ridges, and vertical streaking of collagen (Figure 1A). The findings were those of perforating folliculitis, one of the characteristic lesions of perforating dermatosis.

The patient was initially treated with intralesional triamcinolone 10 mg/cc in addition to topical clobetasol ointment 0.05% and tacrolimus ointment 0.1%. The patient reported partial remission of the lesions but her pruritus persisted. Hydroxyzine 25 mg PO was prescribed as needed for the pruritus and the patient was advised to receive narrow-band ultraviolet B treatment three times a week.

Dermatoscopic imaging with polarized light demonstrated bright white clods, centered in a structureless grey area, surrounded by reticular brown lines. (Figure 2)[6].

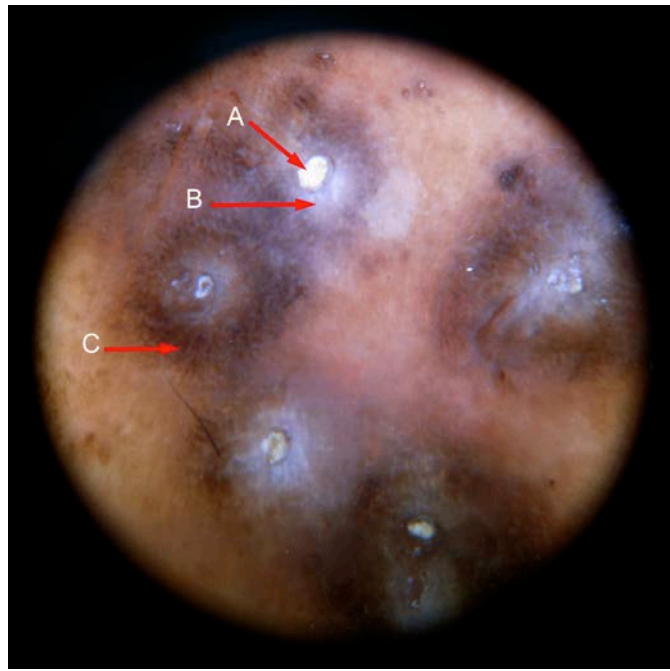


Figure 2: Dermoscopic features (polarizing dermatoscope with 10x magnification) four months after initial presentation. A. Bright white clods, centered in a B. structure-less grey area, C. surrounded by brown lines reticular.

Comments

Clinical presentation of ADP may vary vastly from patient to patient. Different lesions in the same patient can also vary by the dermal component that is being eliminated transepidermally [2,3]. A single patient may have features of elastosis perforans serpiginosa, reactive perforating collagenosis, perforating folliculitis or Kyrle's disease [2,3,5]. Owing to the lack of uniformity among lesions, multiple biopsies are recommended in order to visualize the substance being eliminated (collagen, elastin, follicles) and for diagnostic certainty [2].

The pathogenesis of ADP appears to involve induction of epidermal hyperplasia as a dermal component transcends into the epidermis [1, 2]. Once the dermal substance has reached the epidermis it is eliminated through the surface by normal keratinocyte maturation [1, 2].

Dermoscopic imaging reveals several features that we believe give insight to both the underlying pathology and histology (Figure 2). The bright white clods (Figure 2A) in the center of the lesions correspond to a dilated infundibulum stuffed with keratin and extruded cell debris. The color signifies a complete reflection of the polarized light by follicular contents. Polarized light is regularly used in dermatoscopy to decrease reflection from the stratum corneum *in vivo*. Reflection is known to occur from intraepidermal nests of keratin as in seborrheic keratoses [7]. In the setting of epidermal hyperplasia it is possible that the reflection may be increased. The structure-less grey area (Figure 2B) surrounding the central white clod most probably is a consequence of the combination of epidermal changes (acanthosis, hypergranulosis, and hyperpigmentation of basal keratinocytes and pigmented inflammatory cells) and dermal changes (thickened bundles of collagen in vertical orientation). Grey and blue colors are known to be associated with melanin in the dermis and this color is caused by light scattering by dermal collagen inducing the Tyndall effect [8]. In this case, hair follicles are seen on the histology image (Figure 3) to plunge beneath the dermis at an angle to the surface of the skin so any debris in the follicles which contains melanin, could be reasonably expected to induce the Tyndall effect, giving a blue-grey color. Furthermore, the hyperkeratosis associated with the LSC would potentiate the Tyndall effect upon post-inflammatory epidermal pigment thus appearing as an overall structureless grey pattern under low power magnification in dermatoscopy.

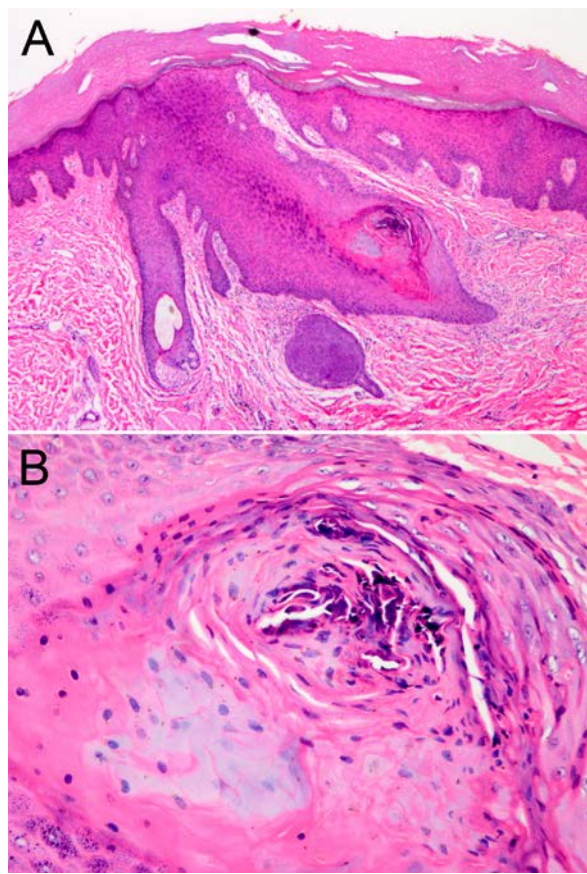


Figure 3. Histopathological features at initial presentation. A. A distorted follicle is seen angling horizontally from the epidermis. There is adjacent irregular acanthosis overlaid with dense orthokeratin (hematoxylin & eosin, 30x). B. The dilated lumen of the follicle was filled with parakeratotic keratinocytes and basophilic debris (hematoxylin & eosin, 200x).

This effect would be further increased by any extruded collagen at the level of the epidermis. It has been shown that reticular brown lines (network) are due to melanin pigment deposition along the rete ridges [9]. Elongated rete ridges with hyperpigmentation of basal keratinocytes are seen on either side of the follicle (Figure 3A). We speculate that the distribution of the lines clustered around follicles correlates to an increase in melanin pigment located in melanocytes and/or keratinocytes, following the initial inflammatory process similar to the findings in a dermatofibroma.

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