Diagnosis at a Glance: Acquired Perforating Dermatosis

Hideyuki Kosumi, Hiroaki Iwata, Masumi Tsujiwaki, and Hiroshi Shimizu

Diabetes Care 2018;41:911–912 | https://doi.org/10.2337/dc17-2572

CASE SUMMARY

- Acquired perforating dermatosis (APD) is the most common perforating dermatosis, known to often occur in patients with chronic kidney disease or diabetes.
- We report a case of typical and aggressive APD in a 70-year-old man.
- Typical APD cases can be diagnosed at a glance, and clinicians should be aware of the characteristic eruptions of APD.

CASE NARRATIVE

A 70-year-old man receiving maintenance hemodialysis presented to the dermatology department with pruritic eruptions on the trunk and limbs. The lesions first appeared 30 months before his referral and had been gradually enlarging in size and increasing in number. His chronic kidney disease due to nephrosclerosis had been first diagnosed 20 years before his visit, and he had been receiving hemodialysis for 3 years. He also had well-controlled type 2 diabetes (HbA_{1c} 6.2%), and his other medical and family history was unremarkable. Physical examination revealed hundreds of crater-shaped pruritic nodules and plaques on the trunk and limbs. The lesions were umbilicated with thickened, greenish-brown, tree ring–like crusts (Fig. 1). The diagnosis of acquired perforating dermatosis (APD) was made. His itchiness improved with topical menthol lotion and oral antihistamines.

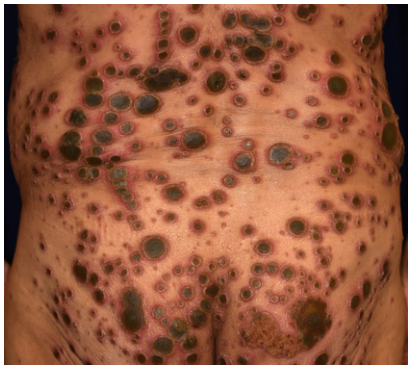

Figure 1—Crater-shaped nodules and plaques with thickened, greenish-brown, tree ring–like center crusts on the lower back.

Perforating dermatoses are skin disorders caused by the perforation of dermal connective tissue through the epidermis (1). They are classified into two general groups: primary perforating dermatoses and APD, the most common perforation dermatosis (1). APD is known to occur in middle-aged adults in association with underlying systemic disease, most commonly in patients with diabetes or chronic renal failure (1,2). Hemodialysis is the most significant risk factor for disease pathology; 3%–11% of patients receiving hemodialysis are reported to have APD (2). The pathogenesis of APD is poorly understood; however, it is postulated that intense scratching induces collagen necrosis and elimination of the necrotic collagens (1,3).

APD is diagnosed based on the visual or histological confirmation of transepidermal elimination of degenerated fibers. The typical eruption has a central yellow-to-greenish crust, which represents degenerated dermal collagens (1,3). Small eruptions can mimic folliculitis, prurigo, insect bites, and dermatofibroma; however, larger ones are easily distinguishable from them (1,4). Histologic analysis of the lesion is sometimes required for diagnosing small eruptions, and it also shows the elimination of degenerated collagens through the epidermis (also confirmed in the present case, data not shown). Treatment of the underlying diseases can improve the eruptions. Of note, renal transplantation has been reported to resolve the eruptions in many hemodialysis-associated APD cases (5). Topical corticosteroids and/or intralesional corticosteroid injections, antipruritic therapies such as antihistamines and menthol-based lotions, topical keratolytics such as salicylic acid, and ultraviolet B phototherapy are also reported to be efficient (1,4). APD is sometimes accompanied by severe pruritus and can impair the patient's quality of life; therefore, clinicians should be aware of these characteristic eruptions in patients with diabetes or chronic kidney disease receiving hemodialysis.

Duality of Interest. No potential conflicts of interest relevant to this article were reported.

Author Contributions. H.K., H.I., and M.T. mainly contributed to the patient care. H.K., H.I., and H.S. mainly drafted the manuscript and performed data interpretation. H.I. is the guarantor of this work and, as

Department of Dermatology, Faculty of Medicine and Graduate School of Medicine, Hokkaido University, Sapporo, Japan

Corresponding author: Hiroaki Iwata, hiroaki.iwata@med.hokudai.ac.jp.

Received 18 December 2017 and accepted 5 January 2018.

such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

References

1. Farrell AM. Acquired perforating dermatosis in renal and diabetic patients. Lancet 1997;349:895–896
2. Zelger B, Hintner H, Auböck J, Fritsch PO. Acquired perforating dermatosis. Transepidermal elimination of DNA material and possible role of leukocytes in pathogenesis. Arch Dermatol 1991;127:695–700
3. Thiele-Ochel S, Schneider LA, Reinhold K, Hunzelmann N, Krieg T, Scharffetter-Kochanek K. Acquired perforating collagenosis: is it due to damage by scratching? [published correction appears in Br J Dermatol 2001 Nov; 145(5):858] Br J Dermatol 2001;145:173–174
4. Karpouzis A, Giatromanolaki A, Sivridis E, Kouskoukis C. Acquired reactive perforating collagenosis: current status. J Dermatol 2010;37: 585–592
5. Chang P, Fernández V. Acquired perforating disease: report of nine cases. Int J Dermatol 1993;32:874–876