Acquired Reactive Perforating Collagenosis Triggered by Scabies Infection

Sir,

Reactive perforating collagenosis is a disease with unknown pathogenesis, often associated with systemic metabolic diseases such as diabetes mellitus and renal insufficiency. Superficial skin trauma is believed to act as a triggering factor. Here we report for the first time on the association with a cutaneous parasitic infection.

CASE REPORT

An 84-year-old man presented with a 6-month history of disseminated erythematous papules and small ulcers with central keratotic crusts mainly at the trunk. Lesions were extremely pruritic, especially during the night, and there were many excoriations. There was no known family history of similar disease, and no other persons in the patient's environment were suffering from similar symptoms.

Physical examination showed symmetrical disseminated papules up to 1 cm in diameter, the larger ones with a central, adherent plug of dark keratotic material embedded in a cup-shaped infiltrated erythematous epidermis.

Laboratory evaluation revealed a moderate hypercholesterolaemia and hypertriglyceridaemia. Type II diabetes mellitus had been diagnosed 5 years earlier and adequately controlled by oral antidiabetics.

A biopsy specimen was obtained from a keratotic papule of the right thigh (Fig. 1). Routine hematoxylin—eosin staining of the biopsy specimen showed the typical picture of a reactive perforating collagenosis: a cup-shaped plug within an epidermal invagination bordered by acanthotic hyperkeratotic epidermis. A dense inflammatory infiltrate surrounding basophilic fragments of collagen fibres was present. In the adjacent epidermis a subcorneally located mite could be seen.

After appropriate treatment of the scabies infection with hexachlorcyclohexan externally the symptoms of the perforating collagenosis subsided.

DISCUSSION

Reactive perforating collagenosis is characterized by the elimination of altered collagen bundles through the epidermis (1). This disease is classified into 2 types: an inherited or

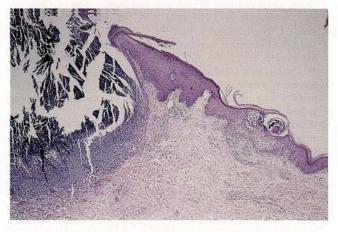


Fig. 1. Hematoxylin-eosin staining reveals a reactive perforating collagenosis with a cup-shaped plug within an epidermal invagination (*left*). A dense inflammatory infiltrate surrounds fragments of collagen fibres. On the right an adjacent subcorneally located mite can be seen.

childhood form and an acquired or adult form (2). Both are believed to be a cutaneous response to superficial trauma (3). The adult form is frequently associated with systemic metabolic diseases such as diabetes mellitus or renal insufficiency (4, 5), and occasionally with malignant lymphomas, hypothyroidism or acquired immunodeficiency syndrome (AIDS) (6).

The pathogenesis of reactive perforating collagenosis is unknown. Diabetic vasculopathy and increased glycosylation of basement membrane proteins are discussed as predisposing factors. The often observed linear distribution and reproducibility by scratching supports the role of a superficial trauma. Necrobiosis of collagen fibres in the dermal papillae with following epithelial reactions may lead to the transepithelial elimination of the necrobiotic connective tissue (7). This process is accompanied by an inflammatory reaction (8).

The prognosis of this disease is dependent on the therapy of the underlying metabolic disorder. For therapy topical steroids and both topical and systemic retinoids have been used successfully. There are also reports on the effectiveness of allopurinol and ultraviolet B light radiation (9, 10).

Our patient had a mild diabetes mellitus which was well controlled by oral antidiabetic medication. We believe that in addition to this predisposing factor the perforating collagenosis was triggered by intense scratching due to a chronic scabies infection. Accordingly, symptoms subsided after appropriate treatment of the scabies infection. To our knowledge, this is the first report of an association of a cutaneous parasitic infection and the development of a reactive perforating collagenosis. Therefore, patients with perforating collagenosis should be carefully investigated for diseases leading to intense pruritus such as scabies infection or insect bites.

REFERENCES

- Mehregan AH, Schwartz OD, Livingood CS. Reactive perforating collagenosis. Arch Dermatol 1967; 96: 277-282.
- Poliak SC, Lebwohl MG, Parris A, Prioleau PG. Reactive perforating collagenosis associated with diabetes mellitus. N Engl J Med 1982; 306: 81–84.
- Bovenmyer DA. Reactive perforating collagenosis: experimental production of the lesion. Arch Dermatol 1970; 102: 313-317.
- Cochran RJ, Tucker SB, Wilkin JK. Reactive perforating collagenosis of diabetes mellitus and renal failure. Cutis 1983; 31: 55-58.
- Gupta AK, Gupta MA, Cardella CJ, Habermann HF. Cutaneous association of chronic renal failure and dialysis. Int J Dermatol 1986; 25: 498-504.
- Faver IR, Daoud MS, Su WPD. Acquired reactive perforating collagenosis. J Am Acad Dermatol 1994; 30: 575-580.
- Yanagihara M, Fujita T, Shirasaki A, Ishiguro K, Kawahara K, Ueda K. The pathogenesis of the transepithelial elimination of the collagen bundles in acquired reactive perforating collagenosis. J Cutan Pathol 1996; 23: 398–403.
- Zelger B, Hintner H, Auböck J, Fritsch PO. Acquired reactive perforating dermatosis – transepithelial elimination of DNA material and possible role of leukocytes in pathogenesis. Arch Dermatol 1991; 127: 695-700.
- Krüger K, Tebbe B, Krengel S, Gördt S, Orfanos CE. Erworbene reaktiv perforierende Dermatose-Erfolgreiche Behandlung mit Allopurinol in 2 Fällen. Hautarzt 1999; 50: 115-120.

 Vion B, Frenk E. Erworbene reaktive Kollagenose des Erwachsenen: Erfolgreiche Behandlung durch UV-B-Licht. Hautarzt 1989; 40: 448-450.

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Acyclovir Prophylaxis of Recurrent Herpes Labialis Before Electrolysis

Sir

Recurrent facial—oral herpes simplex infection is a common viral dermatosis which affects 25-40% of the population. Certain triggering factors such as exposure to ultraviolet light, emotional stress, menstruation and skin trauma cause the reactivation (1). Here, 2 patients with recurrent herpes labialis are presented, in whom reactivation occurred with electroepilation. The patient responded well to oral acyclovir prophylaxis.

CASE REPORTS

Case 1

A 24-year-old girl with facial hirsutism was treated with electroepilation applied monthly. Episodes of herpetic vesicules in the perioral area were observed after all 3 treatments. She informed that she had had no recurrence for years. Treatment with oral acyclovir (200 mg 3 times daily), given for 5 days beginning 2 days before the epilation, was successful and no recurrence was observed during 9 months.

Case 2

A 31-year-old woman who had been treated with electroepilation complained of recurrent herpetic lesions. Lesions occurred in the epilated area after 2 treatments. She had no history of recurrent herpes simplex in the last 10 years. Acyclovir (200 mg 3 times daily) was used for 5 days beginning 2 days before the electrolysis. At first, epilation was performed every 3 weeks, then the treatment intervals were increased up to 6-8 weeks. Herpes simplex did not occur within 1 year, while epilation was performed.

DISCUSSION

Skin trauma is one of the most important triggering factors of recurrent herpes simplex infections (2-4). Chemical peeling and dermabrasion have been shown to reactivate the lesions. Epilation was also mentioned as a possible triggering factor (5). In both patients reported here, herpes simplex in the perioral region recurred after epilation treatments and no recurrence was observed with prophylactic acyclovir use. The antiviral prophylaxis in such patients will prevent the reactivation of herpetic lesions in the treated area.

REFERENCES

- Spruance SL. The natural history of recurrent oral-facial herpes simplex virus infection. Semin Dermatol 1992; 11: 200-206.
- Hill TJ, Blyth WA, Harbour DA. Trauma to the skin causes recurrence of herpes simplex in the mouse. J Gen Virol 1978; 39: 21-28
- Hedner E, Vahlne A, Bergström T, Hirsch JM. Recrudescence of herpes simplex virus type 1 in latently infected rats after trauma to oral tissues. J Oral Pathol Med 1993; 22: 214-220.
- Bobrowski PJ, Capiola R, Centifanto YM. Latent herpes simplex virus reactivation in the guinea pig. An animal model for recurrent disease. Int J Dermatol 1991: 30: 29-35.
- Olsen EA. Methods of hair removal. J Am Acad Dermatol 1999; 40: 143-155.

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