

Granuloma Annulare Induced by Scabies

Dagmar Wilsmann-Theis, Joerg Wenzel, Rainer Gerdson, Manfred Uerlich and Thomas Bieber

Department of Dermatology, University of Bonn, Sigmund-Freud-Strasse 25, DE-53105 Bonn, Germany.

E-mail: dagmar.wilsmann-theis@ukb.uni-bonn.de

Accepted March 24, 2003.

Sir,

Granuloma annulare (GA), a granulomatous disease of unknown aetiology, is characterized by small papules that are often fused into annular arrangements. Several variants are known: localized GA, generalized GA, subcutaneous GA, perforating GA and arcuate dermal erythema. Localized GA is the most common of the subtypes; 9–15% are afflicted with the generalized variant. The other subtypes are rare. Children and young adults are those predominantly affected – women twice as often as men. Human leucocyte antigen (HLA)-B8 has been reported to be increased in localized GA, while HLA-A31 and HLA-BW35 are reported to be increased in the generalized GA.

Proposed pathogenic mechanisms of GA include cell-mediated immunity (type IV), immune complex vasculitis and abnormalities of tissue monocytes, diabetes mellitus, thyroid disease, HIV and hepatitis C infection appear to be predisposing factors.

Aside from these predisposing factors, trigger factors seem to play an important role in the development of the disease. GA may appear following other disease at the same site: mycobacterial and fungal infections, herpes simplex infection, herpes zoster, erythema multiforme minor and insect bites can lead to the manifestation of GA. An isomorphic phenomenon (Koebner reaction) is the assumed pathophysiologic mechanism for these observations. However, to the best of our knowledge a case of GA induced by scabies infection has not been described previously.

CASE REPORT

A 50-year-old woman was referred to our department with strongly pruritic papules and plaques on her trunk, limbs, thigh and knee (Fig. 1). There was no evidence of diabetes mellitus. She had a 10-year history of relapsing GA. In the past, these lesions had been treated successfully with topical steroids. However, the present lesions had not responded to this treatment, nor to UVA1 therapy and fumaric acid. She



Fig. 1. Disseminated, erythematous, pruritic papules and plaques on the patient's thigh.

was now also suffering from pruritus that she had not observed in previous episodes of GA.

Histological examination of skin biopsies revealed formed palisaded granulomas containing mucin and degenerated collagen; moreover, in one biopsy a minor inflammatory infiltrate with some eosinophils. These findings were consistent with the diagnosis of GA. In some of the sections, scabies mites were found in the stratum corneum.

Afterwards the patient was treated with two 3-day courses of lindane topically, followed by topical steroids combined with phototherapy (UVA). This time the treatment led to a good clinical result.

DISCUSSION

GA is a benign, inflammatory skin disease of unknown aetiology. Several theories of the pathogenesis of GA include cellular immune defects, metabolic disorders and a primary disease of altered collagen and/or elastin. Scabies is a contagious infestation of the skin by the arachnoid mite *Sarcoptes scabiei*, variety *hominis*, with a wide spectrum of clinical manifestations. Like syphilis, scabies has come to be known as the great imitator (1). Next to the typical clinical picture with burrows and pruritic papules or vesicles, in last years scabies infection was shown to hide behind and to trigger diseases such as urticaria (2), lichen ruber (3), psoriasis (4), dermatitis herpetiformis, Darier's (5) and Hailey-Hailey disease (6). These diseases share the possible pathogenic factor of the Koebner phenomenon (isomorphic reaction), which appears to be responsible for our observation of GA in the same localization as the scabies mite.

In particular, the fact that previously ineffective treatment led to a convincing result after eradication of scabies mites underlines the possible meaning of scabies infection in the described case as well as the pruritus of GA lesions which appeared for the first time. Scabies should therefore be added to the list of possible trigger factors for exacerbation and atypical presentations of GA.

REFERENCES

1. Nowak MA, Miranda WS, Brodell RT. Crusted (Norwegian) scabies. A simple office test demystifies the 'great imitator'. *Postgrad Med* 1999; 105: 153–156.
2. Chapel TA, Krugel L, Chapel J, Segal A. Scabies presenting as urticaria. *JAMA* 1981; 246: 1440–1441.
3. Schlotmann K, Neumann NJ, Schuppe HC, Ruzicka T, Lehmann P. Scabies-provoked bullous lichen planus in a child. *Hautarzt* 2000; 49: 929–931.
4. Gach JE, Heagerty A. Crusted scabies looking like psoriasis. *Lancet* 2000; 356: 650.
5. Rudolph RI. Scabies resembling Darier's disease. *JAMA* 1982; 247: 1937.
6. Gerdson R, Hartl C, Christ S, Uerlich M, Bauer R, Bieber T. Hailey-Hailey disease: exacerbation by scabies. *Br J Dermatol* 2001; 144: 211–212.