toparasite infection, suggest a humoral IgE response similar to that in many helminthic infections (12).

In the present study IgE antibodies specific to scabies mite were found in the sera of 2 of the five patients examined, wich gives further support to the involvement of an IgE-mediated reaction in scabies. Previously, positive intracutaneous skin test and Prausnitz-Küstner test reactions have been observed when using scabies mite antigen of human origin (7). The same authors reported a close correlation between skin test reactions to scabies and housedust mites in 35% of their patients. IgE antibodies to scabies mite were not examined with RAST in that study (7) but it was suggested that the scabies mite and house-dust mite antigens may cross-react. The present results, obtained with a smaller number of patients, do not support this view. The patients with IgE antibodies to scabies mite did not have antibodies to house-dust mite and, on the other hand, the patient and the control with IgE antibodies to house-dust mite did not show antibodies to scabies mite.

To sum up: we used the RAST to demonstrate the presence of IgE antibodies to scabies mite in patients with scabies. The present and previous results suggest that an IgE-mediated reaction plays a role in the manifestation of scabies in man.

REFERENCES

1. Araujo-Fontaine, A., Thierry, R. & Heid, E.: Les IgE sériques dans la gale. Ann Dermatol Venereol *104:* 203, 1977.
2. Bagnall, B. & Rook, A.: Arthropods and the skin. *In* Recent Advances in Dermatology, Vol. 4, p. 73. Churchill Livingstone, Edinburgh, 1977.
3. Ceska, M. & Lundkvist, U.: A new and simple radioimmunoassay method for the determination of IgE. Immunochem 9: 1021, 1972.
4. Fain, A.: Epidemiological problems in scabies. Int J Dermatol *17:* 20, 1978.
5. Falk, E. S.: Serum immunoglobulin values in patients with scabies. Br J Dermatol *102:* 57, 1980.
6. Falk, E. S. & Bolle, R.: IgE antibodies to house dust mite in patients with scabies. Br J Dermatol *102:* 283, 1980.
7. — In vitro demonstration of specific immunological hypersensitivity to scabies mite. Br J Dermatol *103:* 367, 1980.
8. Frenz, G., Veien, N. K. & Eriksen, K.: Immunofluorescence studies in scabies. J Cutan Pathol *4:* 191, 1977.
9. Hancock, B. W. & Ward, A. M.: Serum immunoglobulin in scabies. J Invest Dermatol *63:* 482, 1974.
10. Lundkvist, U.: Research and development of the RAST technology. *In* Advances in Diagnosis of Allergy: RAST (ed. R. Evans), pp. 85–99. Symposia Specialists, Miami, 1975.
11. Mellanby, K.: The development of symptoms, parasitic infection and immunity in human scabies. Parasitology *35:* 197, 1944.
12. Ogilvie, B. M.: Immunity to parasites (helminths and arthropods). *In* Progress in Immunology, Part II, vol. 4, p. 127. North-Holland Publishing Co., 1974.
13. Shealan, B. J.: Pathology of *Sarcoptes scabiei* infection in pigs. 1. Naturally occurring and experimentally induced lesions. J Comp Pathol *85:* 87, 1975.

Infestation of Scabies in the Scalp Area

Theodor Elmros and Rune Hörnqvist

Department of Dermatology, University Hospital, S-901 85 Umeå, Sweden

Received September 4, 1980

Abstract. A 25-year-old female patient with seborrheic dermatitis of the scalp developed a scabies infestation located chiefly in the scalp area. It is suggested that the treatment of the seborrheic dermatitis of the scalp with a local corticosteroid (hydrocortisone-17-α-butyrate) is the probable explanation for the atypical location of the scabies infestation in this patient.

The 'election of sites' of the scabies parasite on the human body is characteristic of the scabies disease (7). This localization to specific areas of the body is thus a valuable aid in the diagnosis of the disease. However, atypical location is sometimes observed (2, 7), though hitherto there have been no reports describing the localization of scabies parasites in the scalp area among adult patients. For instance, Orkin reported no cases with this location among 886 investigated patients with scabies disease (7). We therefore consider it a matter of interest to report on an adult patient with scabies parasites located chiefly in the scalp area.

CASE REPORT

A 25-year-old female patient visited our dermatology department because of increasing pruritus in the scalp. For 5 years the patient had noted slight symptoms from

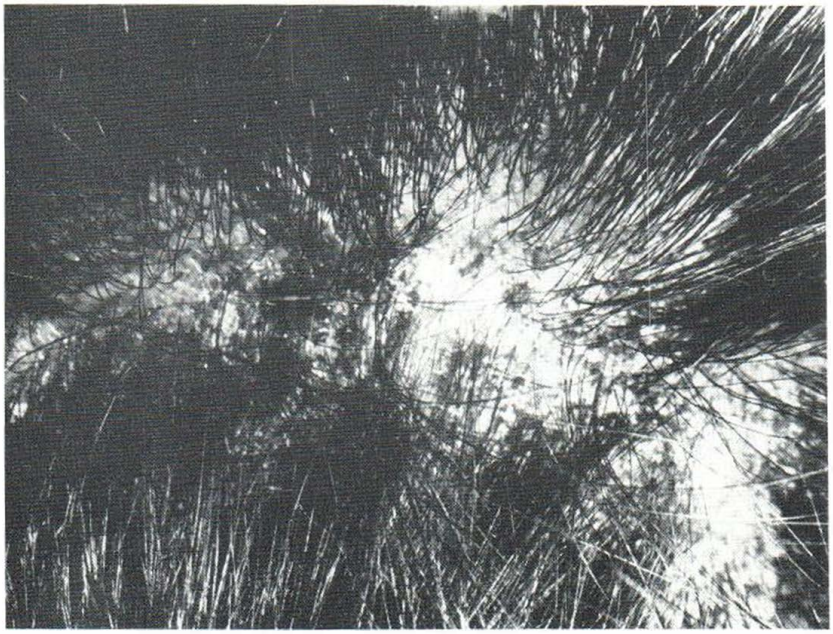

Fig. 1. The scaly and crusted area on the scalp in which an abundance of scabies parasites was found.

this area, but during the month prior to her visit the symptoms had grown worse. Two months previously she had observed red areas with vesicles between the fingers, on the volar aspect of the wrist and had experienced intense pruritus on these locations at night. The patient suspected a scabies infection and treated herself with Tenutex® (benzyl benzoate in combination with disulfiram). The symptoms on the body declined subsequently. On inspection a diffuse redness with scales was noted on the scalp. The patient was treated as having a seborrheic dermatitis of the scalp, requiring a local steroid (hydrocortisone-17-α-butyrate).

The symptoms from the scalp declined initially. However, after about a month they returned and grew worse than before. Two months after the first visit to the department the patient displayed on inspection a 3×5 cm large area on the scalp with red, crusted and partly ulcerated skin (Fig. 1). A diffuse redness was seen, together with signs of scratching. No burrows or vesicles could be found. Microscopic investigation of material taken from these crusts showed an abundance of scabies parasites. The symptoms on the scalp and on the body disappeared 3 weeks after treatment of both scalp and body with Tenutex®.

DISCUSSION

Among children, infestation by the scabies parasite is sometimes observed in the scalp area (5). In these patients the scabies mite has been found in crusts and cellular debris, as was the case in our patient. However, among adult patients the location of scabies on the scalp has not been described before.

In atypical forms, such as Norwegian scabies, a basic disturbance in the immune defence system is occasionally observed (4, 7). Hence, some of these patients are undergoing treatment with corticosteroids or cytostatics (1, 3, 6).

Prior to her scabies infestation, our patient was being treated with a local corticosteroid in the scalp for seborrheic dermatitis. The eczematous reaction could explain the atypical location of scabies mites in the scalp. However, the treatment with a local corticosteroid is a more likely explanation for the atypical location. We believe therefore that local corticosteroid treatment of an eczematous reaction can result in an atypical location of a concomitant infestation with scabies parasites.

REFERENCES

1. Clayton, R. & Farrow, S.: Norwegian scabies following topical steroid therapy. Postgrad Med J *51:* 657, 1975.
2. Dahl, O.: Scabies. Epidemiologi og klinikk. Tidskr Nor Laegeforen *96:* 548, 1976.
3. Espy, P. D. & Jolly, H. W.: Norwegian scabies. Occurrence in a patient undergoing immunosuppression. Arch Dermatol *112:* 193, 1976.
4. Hubler, W. R. & Clabaugh, W.: Epidemic Norwegian scabies. Arch Dermatol *112:* 176, 1976.
5. Hurwitz, S.: Scabies in babies. Am J Dis Child *126:* 226, 1973.
6. Millard, L. G.: Norweigan scabies developing during

treatment with fluorinated steroid therapy. Acta Dermatovener (Stockholm) *57*: 86, 1977.
7. Orkin, M.: Today's scabies. An editorial. Arch Dermatol *111*: 1431, 1975.
8. Orkin, M., Maibach, H. I., Parish, L. C. & Schwartzman, R. M.: Scabies and Pediculosis. J. B. Lippincott Comp., Philadelphia and Toronto, p. 14, 1977.

Tripolar Mitosis in a Keratoacanthoma

Larry I. Giltman

Department of Pathology, USPHS Hospital, Norfolk, Virgina 23508, USA

Received December 19, 1980

Abstract. This report demonstrates a case of kertoacanthoma presenting with classical clinical, gross and microscopic features. Histopathologically, however, one cell is seen undergoing tripolar mitosis, which is often not accepted as a feature in benign lesions.

Key words: Keratoacanthoma; Tripolar Mitosis; Actinic Changes; Squamous cell carcinoma

Keratoacanthoma is a well known entity, with many classically described gross and microscopic features. On occasion, the differential diagnosis between keratoacanthoma and squamous cell carcinoma may be difficult, especially when only a portion of the lesion is submitted for histopathologic examination. Cellular atypia is an accepted feature in keratoacanthoma, but atypical mitosis often is not. Here we present a case of keratoacanthoma showing classic clinical, gross and microscopic features of the lesion. In addition, a tripolar mitosis is clearly demonstrated.

CASE REPORT

A healthy, asymptomatic 61-year-old white male developed a 2 cm painless, non-pruritic nodular lesion over the left pre-auricular area. The lesion grew rapidly over the next 2 months from apparently normal skin. There were no other significant dermatologic abnormalities and the patient's past medical history was essentially unremarkable.

Physical examination revealed a tan-colored, nodular lesion with a central crater filled with keratinaceous material.

The nodule was well defined and firm, but he margins were not indurated. There was no regional lymphadenopathy. An excisional biopsy was performed.

Microscopic examination revealed a craterform lesion filled with a plug of keratinaceous material surrounded by nests of proliferating squamous epithelium extending into the lower dermis. The borders of the lesions extend in a lip-like manner over the crater (Fig. 1). A granular layer is evident but associated actinic changes are not noted. The penetrating nests of squamous epithelium are surrounded by a dense chronic inflammatory cell infiltrate (Fig. 2). Some squamous epithelial cells show atypia. An isolated cell was observed, undergoing tripolar mitosis (Fig. 3).

Comment. Keratoacanthoma, first described in 1888 (3), is a well known entity arising from the upper portion

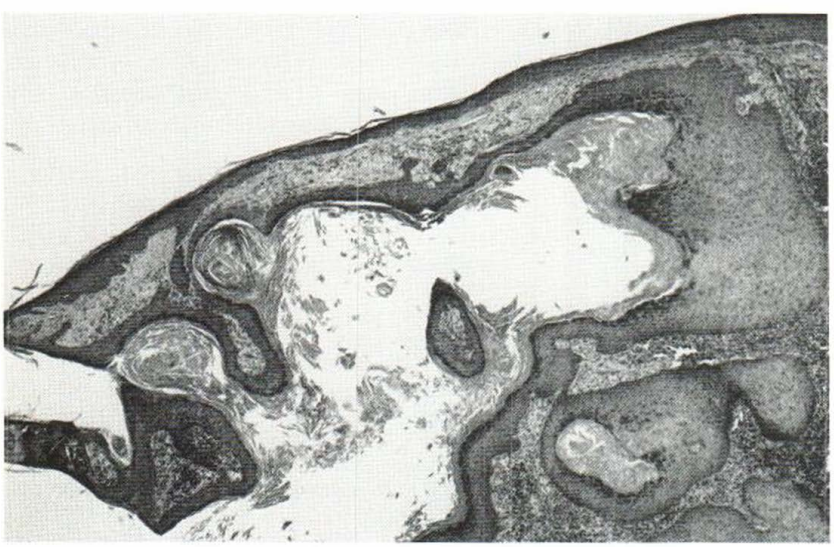

Fig. 1. Low-power photomicrograph showing characteristic "lipped" margin of keratoacanthoma. Also note the proliferating nests of squamous epithelium mixed with chronic inflammatory cells (*lower right*). Hematoxylin-eosin, ×40.