

may be formed and released from macrophages. Parallels to the pathogenesis of silica-induced scleroderma are given, as observed in 111 patients in our department (14).

As silica cannot be removed from the body, its precipitating effect cannot be stopped. This indicates that the clinical course is that of classical autoimmune diseases, with progressions and remissions. The therapy has to be in line with these facts and has to be adjusted according to the criteria of the clinical activity of the disease. The best way to prevent this type of SLE is to minimize the exposure to silica.

#### REFERENCES

1. Kilburn KH, Warshaw RH. Prevalence of symptoms of systemic lupus erythematosus (SLE) and of fluorescent antinuclear antibodies associated with chronic exposure to trichlorethylene and other chemicals in well water. *Environmental Res* 1992; 57: 1–9.
2. Hatron PY, Plouvier B, Francois M. Assoziation von systemischem Lupus erythematosus und Silikose. *Kasuistik (5 Pat.)*, *Rev Med Interne* 1982; 3: 245–246, ref. in: *Zbl Haut-u Geschl-Krkh* 1983/84; 149(1): 403.
3. Ebihara I, Kawami M. Health hazards of mineral dust with special reference to the causation of immuno-pathologic systemic diseases. *J Science Labour* 1985; 61: 1–31.
4. Mehlhorn J, Gerlach Ch. Gemeinsames Auftreten von Silikose und Lupus erythematosus. *Z Erkr Atm-Org* 1990; 175: 38–41.
5. Ziegler V, Pfeil B, Hausteil U-F. Berufliche Quarzstaubexposition – Progressive Sklerodermie und Lupus erythematosus. *Z Hautkr H + G* 1991; 66: 968–970.
6. Özoran K, Uçan H, Tutkak H, Caner N, Yücel M. Systemic lupus erythematosus arising in a patient with chronic silicosis. *Rheumatol Int* 1997; 16: 217–218.
7. Siebels M, Schulz V, Andrassy K. Systemischer Lupus erythematosus und Silikose. *Dtsch Med Wochenschr* 1995; 120: 214–218.
8. Silicosis and Silicate Disease Committee. Diseases associated with exposure to silica and nonfibrous silicate minerals. *Arch Pathol Lab Med* 1988; 112: 673–720.
9. Doll JN, Stankus RP, Hughes J, Weill H, Gupta RC, Rodriguez M, et al. Immune complexes and autoantibodies in silicosis. *J Allergy* 1981; 68: 281–285.
10. Jones RN, Turner-Warwick M, Ziskind M, Weill H. High prevalence of antinuclear antibodies in sandblaster's silicosis. *Am Rev Respir Dis* 1976; 113: 393–395.
11. Frank R, Giese T, Dummer R, Walther T, Rytter M, Ziegler V, et al. Silica-induced cytokine release in human monocyte cultures and its possible involvement in the pathophysiology of silica-associated scleroderma. *Eur J Dermatol* 1993; 3: 304–309.
12. Anderegg U, Vorberg S, Herrmann K, Hausteil U-F. Silica directly induces intercellular adhesion molecule 1 (ICAM-1) expression in cultured endothelial cells. *Eur J Dermatol* 1997; 7: 27–31.
13. Anderegg U, Vorberg S, Herrmann K, Hausteil U-F. Increased expression of interstitial collagenase in silica-treated fibroblasts. *Eur J Dermatol* 1996; 6/1: 51–55.
14. Hausteil U-F, Herrmann K. Environmental scleroderma. *Clin Dermatol* 1994; 12: 467–473.

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## Centroblastic-centrocytic Lymphoma Arising at the Site of Previous Herpes Zoster Eruption

Sir,

The development of a skin disorder at the site of an unrelated, already healed disease is known as isotopic response.

We describe a patient in whom a centrocytic-centroblastic lymphoma developed at the site of a previous herpes zoster.

#### CASE REPORT

In 1994, a 58-year-old Caucasian man had a herpes zoster eruption on the left antero-lateral thoracic region. Three months later, asymptomatic, firm, brown-red papulonodular lesions, 0.5–2.0 cm wide, began to develop on the zoster site, where only a pigmented macule had remained (Fig. 1). Axillary lymph nodes and spleen were not palpable.

Laboratory tests, including complete blood cell counts, liver and renal function, were within normal ranges. No concurrent extracutaneous disease findings were detected by chest radiography, computerized abdominal tomography and echography.

Histopathology of a nodular lesion showed a lymphoid infiltrate in the papillary and reticular dermis without epidermotropism, mainly composed of a mixed centrocytic/centroblastic population with a conspicuous follicular pattern. Collagen bundles were in part dissociated by the infiltrate. Immunohistochemically, most of the lymphoid cells stained with anti-CD20 monoclonal antibodies and a few of them with anti-CD3 monoclonal antibodies. Immunocytochemistry showed a monoclonal proliferation of cells staining for mu and kappa chains.

Treatment with natural interferon alpha (3 MU three times a week IM) was initiated, and the lesions cleared completely in 8 months. The drug was then stopped and, 3 months later, the tumour relapsed

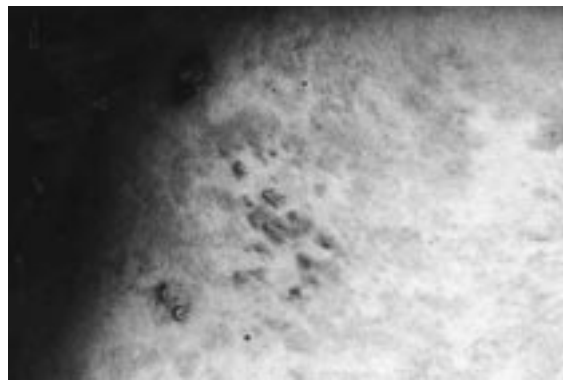


Fig. 1. Papulo-nodular lesions surrounded by annular erythema on the left antero-lateral thoracic region.

in the same area. The lesions partly cleared after a second course of interferon therapy at the same dosage.

#### DISCUSSION

The term isotopic response refers the occurrence of a new skin disorder at the site of another one, already healed and unre-

lated (1). The interval between the first and the second disease is variable. The preceding disease is frequently a herpesvirus infection and herpes zoster is by far the most common of them. Post-zoster diseases include granulomatous vasculitis (2), granulomatous lesions (3), granuloma annulare (4), sarcoid granuloma (5), pseudolymphoma (6), lymphoplasmacytoid lymphoma (7), Kaposi's sarcoma (8), basal cell carcinoma (9) and squamous cell carcinoma (9, 10).

In literature, only one case of cutaneous B cell lymphoma has been reported arising in a site of a previous zoster eruption (7). In that patient, however, the B cell lymphoma had already been diagnosed when the zoster erupted. In our case, instead, the isotopic response was the first manifestation of the cutaneous lymphoma.

The pathogenesis of isotopic response is unclear (1). Either the virus is directly responsible for the second disease or the latter is the result of an immunologic hypersensitivity to viral antigens or to the tissue damage. Evidence is the detection of varicella-zoster virus DNA in post-zoster granulomatous lesions and of herpes simplex DNA in a squamous cell carcinoma (3, 10). On the other hand, in a case of post-zoster granulomatous vasculitis no viral DNA was detected in the lesional skin (2).

Centroblastic-centrocytic lymphoma is a low-grade malignant B cell lymphoma, involving the skin and confined to it for many years without any lymph node or visceral involvement (11). The back and the thorax are zones of preference and the possibility of a fortuitous coincidence cannot be excluded.

#### REFERENCES

1. Wolf R, Brenner S, Ruocco V, Filioli FG. Isotopic response. *Int J Dermatol* 1995; 34: 341–348.
2. Langenberg A, Yen BTS, LeBoit PE. Granulomatous vasculitis occurring after cutaneous herpes zoster despite absence of viral genome. *J Am Acad Dermatol* 1991; 24: 429–433.
3. Serfling U, Penneys NS, Zhu W-Y, Sisto M, Leonardi C. Varicella-zoster virus DNA in granulomatous skin lesions following herpes zoster. *J Cutan Pathol* 1993; 20: 28–33.
4. Friedman SJ, Fox BJ, Albert HL. Granuloma annulare arising in herpes zoster scars. Report of two cases and review of the literature. *J Am Acad Dermatol* 1986; 14: 764–770.
5. Bisaccia E, Scarborough DA, Carr RD. Cutaneous sarcoid granuloma formation in herpes zoster scars. *Arch Dermatol* 1983; 119: 788–789.
6. Wolff HH, Wendt V, Winzer M. Cutaneous pseudolymphoma at the site of prior herpes zoster eruption. *Arch Dermatol Res* 1987; 279 (Suppl): 852–854.
7. Aloï FG, Appino A, Puiatti P. Lymphoplasmacytoid lymphoma arising in herpes zoster scars. *J Am Acad Dermatol* 1990; 22: 130–131.
8. Niedt GW, Prioleau PG. Kaposi's sarcoma occurring in a dermatome previously involved by herpes zoster. *J Am Acad Dermatol* 1988; 18: 448–451.
9. Wyburn-Mason R. Malignant change arising in tissues affected by herpes. *BMJ* 1955; 2: 1106–1109.
10. Claudy AL, Chignol MC, Chardonnet Y. Detection of herpes simplex virus DNA in a cutaneous squamous cell carcinoma by in situ hybridization. *Arch Dermatol Res* 1989; 281: 333–335.
11. National Cancer Institute. Sponsored study of classifications of non-Hodgkin's lymphoma: summary and description of a working formulation for clinical usage. The non-Hodgkin's Lymphoma Pathologic Classification Project. *Cancer* 1982; 49: 2112–2135.

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## Surgical Treatment of Pemphigus Vulgaris Localized to the Genital Mucosa

Sir,

Cases of pemphigus vulgaris located on a single mucosa, sometimes preceding by months or even years a more diffuse development of the disease, are not unusual but the oral mucosa is nearly exclusively involved in such observations.

We here report an original observation of an immunologically typical, unilesional pemphigus vulgaris located on the foreskin in a middle-aged man. The patient was completely cured by a limited surgery, without relapse after a 3-year follow-up.

#### CASE REPORT

A 47-year-old man was first referred to our institution in 1993 for chronic, bullous and erosive lesions of the foreskin of 2 years' duration. Clinically, a limited area of the balano-prepuccial fold and of the free foreskin mucosa was involved, with a number of small erosions ranging from 1–2 mm in diameter. The patient denied taking any long-duration medication and no other mucous membrane manifestations had ever been present. The mucocutaneous and general examination were otherwise normal and no Nikolski's sign was present. ESR, full blood count, comprehensive chemical panel, liver tests and renal

function were all normal. Two different biopsy specimens taken on the margin of the erosions revealed a suprabasal cleavage with a typical acantholytic picture, along with an important mononucleated infiltrate of the upper chorion (Fig. 1). Direct immunofluorescence showed the deposition of intercellular IgG and C3c in the epithelium;

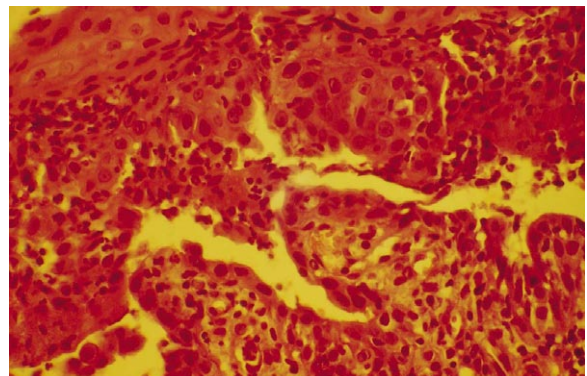


Fig. 1. Typical suprabasal acantholysis.