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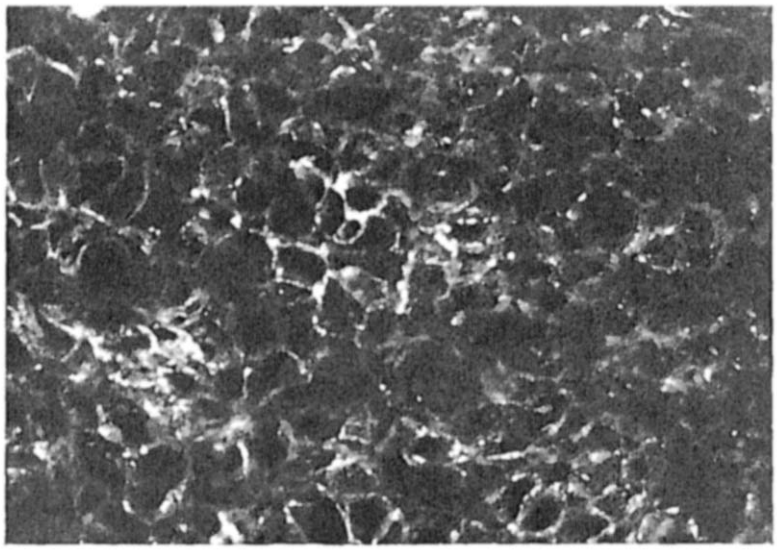

Fig. 3. Direct immunofluorescence staining of squamous cell carcinoma of patient shows anti-IgG at periphery of tumorous cells. (×500.)

seen at the periphery of the cells. These deposits were linear in some areas and granular in others. In control specimens (three squamous cell carcinomas and one undifferentiated squamous cell carcinoma of the lung) no immunoglobulin deposition was revealed by direct immunofluorescence studies.

Discussion. Previous publications have reported an association between pemphigus vulgaris and malignant proliferations such as lymphomas; Kaposi's sarcoma; thymoma; and tumors of the ovary, stomach, breast, and endometrium.[3] Squamous cell carcinoma of the esophagus and lung[1,2] and epidermoid carcinoma of the leg[4] have also been reported in association with pemphigus vulgaris.

To our knowledge this is the first study that has revealed IgG deposits in a squamous cell carcinoma associated with pemphigus vulgaris. In previous studies the binding of IgG to squamous cell carcinomas of the skin or other organs was not reported.[4-6]

The findings in our patient suggest that a relationship may exist between pemphigus vulgaris and squamous cell carcinoma of lung; both diseases appeared almost simultaneously, and IgG deposition was found in the carcinoma. The carcinoma in this patient may share some antigenic determinants with the antigen of pemphigus vulgaris. Cross-reaction between different antigenic determinants therefore cannot be ruled out.

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Benign lymphocytic infiltration (Jessner-Kanof): Another manifestation of borreliosis?

Donald C. Abele, MD,[a] Kenya H. Anders, MD,[a] and Francis W. Chandler, DVM, PhD[b]
Augusta and Atlanta, Georgia

Benign lymphocytic infiltration (BLI) was described in 1953 by Jessner and Kanof.[1] The lesions are asymptomatic, erythematous, discoid plaques that frequently clear centrally and are found most often on the head, neck, and upper trunk of young adults. The disorder has a prolonged course characterized by remissions and relapses. Although considered by some investigators to be a variant

From the Department of Dermatology, Medical College of Georgia, Augusta,[a] and the Division of Host Factors, Centers for Disease Control, Atlanta.[b]

Reprint requests: Donald C. Abele, MD, Department of Dermatology, Medical College of Georgia, Augusta, GA 30912-2900.

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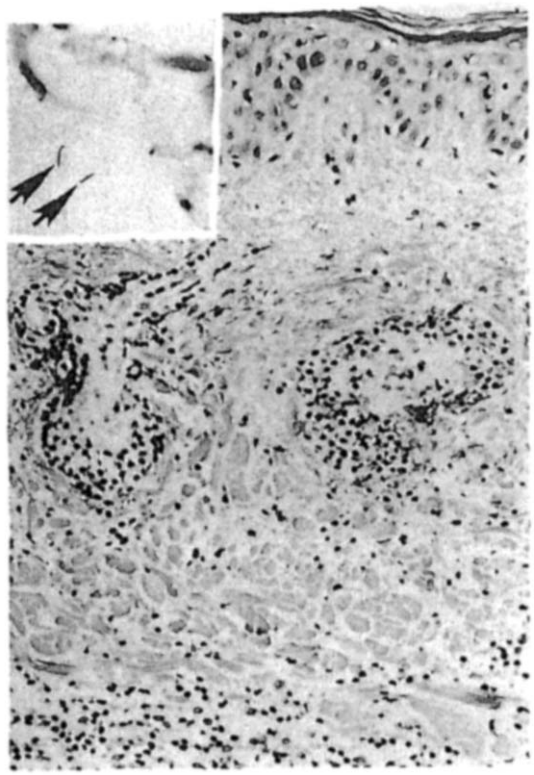

Fig. 1. Biopsy specimen of back lesion taken in 1989 shows chronic perivascular lymphocytic dermatitis. (Hematoxylin-eosin stain; ×190.) *Inset:* Spirochetes (*arrows*) morphologically consistent with *Borrelia* species between collagen fibers in deep dermis. (Steiner silver impregnation; ×750.)

of discoid lupus erythematosus, the lesions usually resolve without scarring and most consider the etiology to be unknown. We report a case of BLI in a patient in whom erythema chronicum migrans subsequently developed. *Borrelia* species were demonstrated in both types of lesions, which cleared with tetracycline therapy.

Case report. A 40-year-old white man was seen in 1981 with a six-month history of waxing and waning asymptomatic plaques on his face. Results of histologic examination confirmed the clinical suspicion of BLI of the skin (Jessner-Kanof type). In January 1989, he presented with a recurring polycyclic eruption on his back of 3 years' duration in addition to the original forehead and cheek lesions. He reported frequent exposure to ticks. Clinically, the eruption on the back was consistent with erythema chronicum migrans. A biopsy specimen revealed a chronic perivascular lymphocytic dermatitis (Fig. 1). Serum contained antibody to *Borrelia burgdorferi* by indirect fluorescence antibody (IgG) assay (Hillcrest Biologicals, Cypress, Calif.) at a titer of 1:128. Both the cheek and back lesions cleared with tetracycline therapy.

Steiner silver impregnation stains of tissue sections taken from the cheek lesion from 1981 revealed intensely argyrophilic spiral forms, fragmented spiral forms, and noncoiled filamentous forms morphologically suggestive of a *Borrelia* species (Fig. 2). Immunofluorescence staining of replicate tissue sections from this specimen with a species-specific monoclonal antibody against *B. burgdorferi* and with a monoclonal antibody specific for the *Borrelia* species (genus level only) revealed brightly fluorescent spiral and noncoiled filamentous forms. Steiner stains of the back lesion from 1989 (Fig. 1, *inset*) also showed spirochetal forms that were fluorescent with the genus-specific monoclonal antibody.

Discussion. Erythema chronicum migrans was described in 1910 by Afzelius,[2] who recognized an association of the disorder with tick bites. Since then several conditions have been associated with borrelial infections, including acrodermatitis chronica atrophicans, Lyme disease, lymphadenosis benigna cutis (Spiegler-Fendt), and some cases of morphea and lichen sclerosus.[3,4] There is a growing awareness that various species of *Borrelia* may produce prolonged disease with various cutaneous and systemic stages similar to the classic stages of syphilis.[5,6]

The case reported in this article confirms that erythema chronicum migrans, clearly a manifestation of *B. burgdorferi* infection, can run a protracted, relapsing course reminiscent of the various cutaneous manifestations of syphilis.[5] Of greater significance is the demonstration of borrelial microorganisms in a lesion of BLI (Jessner-Kanof) that resolved after tetracycline therapy. Hitherto considered a disease of unknown etiology, we propose that it may be another manifestation of borreliosis. The primary involvement of exposed areas of young men may relate to more exposure to tick bites while involved in outdoor activities; in addition, BLI frequently develops central clearing that gives an annular appearance similar to that of erythema chronicum migrans. It has histopathologic features of perivascular lymphocytic infiltration associated with cellular immune responses to an infectious agent and may run a course characterized by remissions and relapses similar to lues. Epidemiologic studies of patients with BLI and a history of tick exposure are needed; further attempts to demonstrate infection by identification of spirochetes in tissue or detection of serologic and T cell proliferative responses[7] may result in elucidation of its etiology.

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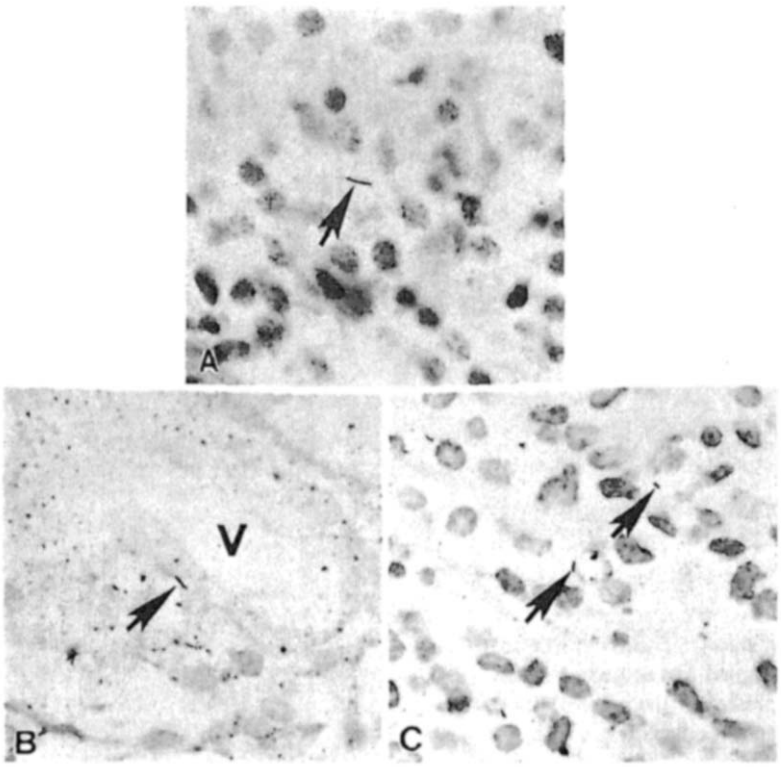

Fig. 2. Morphologic diversity of *Borrelia* species in biopsy specimen of cheek lesion taken in 1981 and examined retrospectively. **A,** Noncoiled filamentous form *(arrow).* **B,** Tightly coiled spirochetal form *(arrow)* within wall of dermal blood vessel *(V).* **C,** Apparent intracellular fragmented forms *(arrows)* that were positive for *Borrelia* species by immunofluorescence staining. (Steiner silver impregnation; ×750.)

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Skin lesions simulating chronic dermatomyositis during long-term hydroxyurea therapy

Marie Richard, MD,[a] François Truchetet, MD,[b] Jean Friedel, MD,[b] Christian Leclech, MD,[b] and Ernest Heid, MD[b] *Montreal, Quebec, Canada, and Strasbourg, France*

Hydroxyurea is a cytotoxic drug used mainly to treat chronic myelogenous leukemia and, occasionally, extensive psoriasis. This drug inhibits DNA synthesis through its action on ribonucleoside diphosphate reductase, which catalyzes the reduction of ribonucleotides.[1] In 1975 Kennedy et al.[2] first described cutaneous reactions during long-term maintenance therapy with hydroxyurea. Their observations and those of others[3,4] indicate the existence of unusual cutaneous eruptions encountered with long-term daily hydroxyurea therapy. We report a case with unusual clinical and histopathologic findings.

Case report. A 55-year-old woman was referred to us in June 1984 for evaluation of skin lesions present for 3 months. Her clinical history included a hemicolectomy for sigmoid carcinoma in 1972 without evidence of recurrence. Since 1980 she had been treated for concurrent chronic myelogenous leukemia and thrombocythemia with hydroxyurea, 500 mg/day to 3 gm/day. The patient was also taking allopurinol.

Examination disclosed a telangiectatic erythema on the face, mostly on the cheeks, associated with edema of the eyelids. There was a linear erythematous scaling and atrophic eruption on the dorsa of the hands. Previous bullae and erosions produced sclerosis and atrophy of the hands (Figs. 1 and 2). There was also a dry, erythematous palmar keratoderma; leukokeratotic and erosive lesions of the buccal mucosa and lips; and an ulceration of the left internal malleolus.

Examination of two skin biopsy specimens from the lateral border of the hand disclosed alternating slight atrophy and

From the Department of Dermatology, Saint-Luc Hospital,[a] Montreal, and the Clinic of Dermatology (Pr Grosshans) Hospices Civils de Strasbourg.[b]

Reprint requests: M. Richard, MD, 831, rue Stuart No. 3, Outremont, Quebec, Canada, H2V 3H7.

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