

necessary for psoriasis and hence might explain our patient's excellent clinical response.

There are no data to suggest that oral or subcutaneous methotrexate prevents progression to lymphoma in patients with lymphomatoid papulosis.⁷ Hence, it is even less clear whether a topical formulation of this drug would provide this benefit. Nonetheless, topical methotrexate might be an attractive alternative with few side effects for the symptomatic treatment of limited lymphomatoid papulosis.

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Beryllium dermatitis

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Chronic beryllium disease is a granulomatous disorder characterized by a cell-mediated immune response to beryllium. Most reports of chronic beryllium disease discuss pulmonary and noncutaneous immunologic findings. This report of occupational chronic beryllium disease emphasizes cutaneous findings and discusses the potential role of skin exposure in the disease. (*J Am Acad Dermatol* 2003;49:939-41.)

Cutaneous manifestations to beryllium exposure were first described in 1951.¹ Since this initial compilation of patients by Curtis, 5 classes of beryllium skin disease have been described: irritant contact dermatitis, allergic contact dermatitis, chemical ulcers, ulcerating granulomas, and allergic dermal granulomas. We report a case in which cutaneous beryllium disease was present before subjective pulmonary complaints.

CASE REPORT

A 29-year-old white man was referred for evaluation of a 4-month history of a papular eruption on his arms, left thigh, and right knee. The patient's

medical history was remarkable for infectious mononucleosis 1 month earlier, and his only medication was a multivitamin supplement. He had been employed at a factory for the past 3.5 years, where he operated a beryllium-alloy production furnace that melted beryllium, copper, cobalt, zirconium, and nickel. Treatment with a 2-week course of systemic corticosteroids and mid-potency topical steroids had been unsuccessful. Because he had chronic exposure to beryllium, the factory monitored pulmonary function tests on a routine basis. He had been referred to pulmonary medicine for further evaluation after a decline in his diffusion lung capacity from 133% predicted in March of 1998 to 97% predicted in March of 1999. A routine chest x-ray film and high-resolution computed tomography scan of the chest revealed no evidence of disease, bronchoscopy with bronchoalveolar lavage and transbronchial biopsies revealed no evidence of granulomas or other signs of disease, and both bronchoalveolar lavage lymphocyte proliferation test and blood lymphocytic proliferation test for beryllium were negative. In the fall of 1999, the patient's diffusion lung capacity was rechecked and had increased to 109% of predicted.

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Fig 1. Scattered violaceous papules on the forearm.

At the time of our evaluation in the summer of 2000, the patient had no pulmonary complaints but a recent blood lymphocytic proliferation test was positive.

Physical examination revealed scattered, violaceous papules on his forearms, left thigh, and right knee (Fig 1). A group of linear papules on the right forearm was consistent with the Koebner phenomenon. Biopsy specimens from the right wrist and left thigh revealed confluent granulomatous inflammation filling the superficial dermis (Fig 2). An occasional granuloma demonstrated central fibrinoid necrosis. The granulomas were composed chiefly of epithelioid histiocytes and multinucleated giant cells associated with a mild lymphocytic infiltrate. The granulomas lacked a well-developed cuff of lymphocytes. Stains with periodic acid-Schiff, Gomori methenamine silver, Twort, and acid-fast bacillus were negative for infectious organisms.

After the results of the biopsy and positive blood lymphocytic proliferation test, a follow-up pulmonary evaluation revealed pulmonary function tests within normal limits but decreased in value over the previous 13 months. A complete physical examination was remarkable for rhonchi at both lung bases. Further laboratory studies revealed a normal complete blood count and serum chemistries. A chest radiograph showed left hilar adenopathy with mild diffuse lung disease. A high-resolution computed tomography scan revealed patchy ground-glass changes on expiration with multiple pulmonary nodules seen. The changes on the chest radiograph and computed tomography scan are consistent with beryllium-induced pulmonary disease. Bronchoscopy showed no endobronchial abnormalities, but a transbronchial lung biopsy specimen revealed nu-

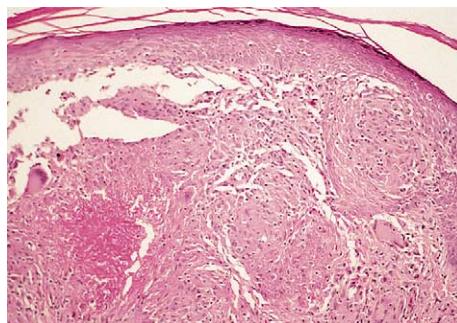


Fig 2. Photomicrograph of biopsy specimen shows confluent granulomatous inflammation filling the superficial dermis. (Hematoxylin-eosin stain; original magnification $\times 100$.)

merous noncaseating granulomas. Acid-fast stains were negative, and no organisms were isolated on fungal culture. A positive bronchoalveolar lavage lymphocyte proliferation test was identified. He was treated with 40 mg prednisone every other day, high-potency topical corticosteroids, and instructed to avoid further exposure to beryllium. This has resulted in marked improvement of his cutaneous lesions.

DISCUSSION

Beryllium is one of the lightest metals (atomic weight, 9.015) and possesses a high melting point (2341°F) and dimensional stability. When alloyed with other metals such as copper, it will improve the properties of the parent metal. These characteristics have made it useful in a wide range of applications, including the manufacturing of electrical equipment, cathode ray tubes, fluorescent lights, and orthodontic devices.²

Curtis¹ reported the first compilation of patients with cutaneous hypersensitivity to beryllium in 1951. The morphology of the skin lesions ranges from eczematous, papulovesicular lesions during initial exposure to ulcerations and granulomas with prolonged contact. Epstein³ has described 5 groups of cutaneous disease: irritant contact dermatitis; allergic contact dermatitis; chemical ulcers; ulcerating granulomas; and allergic dermal granulomas, present in our patient.

Pulmonary disease through inhalation of beryllium particles has also been described. When beryllium particles are inhaled in high concentrations, acute chemical pneumonitis can develop.⁴ These patients are initially seen with dyspnea, chest pain, and cough. A chest radiograph will reveal diffuse or focal parenchymal infiltrate. As a result of manufacturing changes and industrial hygiene practices, acute beryllium disease almost never occurs. Chronic beryllium disease is characterized by non-

caseating granulomas.⁴ An insidious onset is the rule, and prolonged exposure to beryllium is usually required. If skin manifestations are present in these patients, a biopsy specimen will usually reveal non-caseating granulomas.⁵

As chronic beryllium disease can be difficult to diagnose early in its course, persons with known exposure should undergo a periodic beryllium blood lymphocyte proliferation test and pulmonary evaluation. Sensitization to beryllium can be detected by measuring the in vitro proliferative response of peripheral blood lymphocytes or bronchoalveolar lavage lymphocytes to beryllium sulfate. In patients who have repeatedly equivocal or uninterpretable test results, patch testing can be useful to clarify the diagnosis.⁶

Pure beryllium metal is insoluble on skin and cannot be used for patch testing.² In the initial article by Curtis, 1% and 2% beryllium fluoride was used for patch testing. These salts have subsequently been found to be a potent contact sensitizer.⁷ Currently, a 1% aqueous unbuffered solution of beryllium sulfate or chloride is recommended for patch testing.²

Cutaneous berylliosis eruptions should be treated with avoidance of beryllium exposure, mid-potency to high-potency topical corticosteroids, compresses, and antibiotics to prevent secondary infection. When beryllium nodules are present, surgical excision is the definitive treatment.⁵

In our patient chronic beryllium disease developed with positive blood beryllium lymphocyte proliferation test at about the same time that his cutaneous granulomas appeared. This was soon followed by abnormal chest radiographs and computed tomography scans, as well as granulomas on transbronchial biopsy. He has discontinued working with beryllium and might require prednisone for the foreseeable future to control his chronic beryllium disease of the lungs. Environmental sampling of beryllium is routinely performed in the workplace, which over recent years has led to increased precautions in the manufacturing plant to minimize cutaneous and respiratory exposure to beryllium.

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Primary mucinous carcinoma in a 54-year-old man

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Primary mucinous carcinoma is a rare malignant tumor that most frequently occurs in the periorbital area. This tumor originates from the deepest portion of the eccrine sweat duct. This normally asymptomatic and slow-growing tumor has demonstrated a local recurrence rate of 30% after excision with narrow surgical margins and can have local metastases. It is difficult to differentiate this tumor histologically from metastatic lesions. Immunohistochemical staining and cytokeratin profiles have been studied to aid in the differentiation between primary lesions and metastatic mucinous carcinomas. We present a case of a 54-year-old man with recurrent primary eccrine mucinous carcinoma and review the clinical, histologic, and immunohistochemical features of this tumor. (*J Am Acad Dermatol* 2003;49:941-3.)

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P primary mucinous carcinoma is a rare adnexal tumor that arises in the deep dermis and from the deepest portion of the eccrine secretory coil, the mucin-secreting dark cells. Snow et al¹ reviewed 73 cases of primary cutaneous mucinous carcinoma and reported a median age at onset of 63 years and a male predominance of 2:1. The ethnic