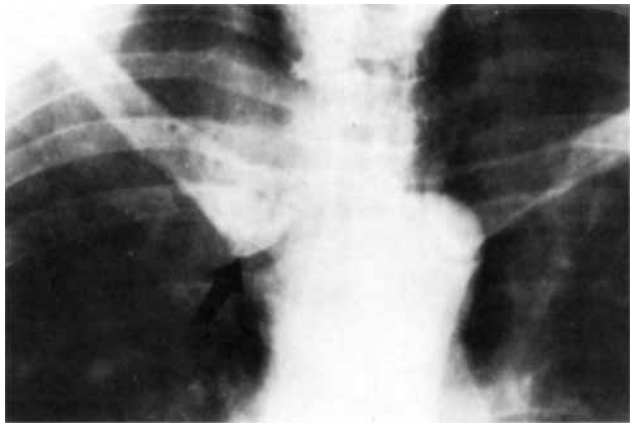

Figure 1. Roentgenograph showing hypercondensation of the head of the right clavicle, with erosive and lytic changes of adjacent manubrium sterni.

of malignant lymphoma. Although skeletal involvement frequently occurs in malignant lymphoma, an inflammatory lymphomatous arthropathy is uncommon and the symptoms are seldom prominent (2). Musculoskeletal symptoms usually exist when a pathologic fracture occurs, in cases of hypertrophic osteopathy associated with mediastinal involvement (3), or in cases of secondary gout (4) and synovial effusions (5). Interestingly, lymphatic and intercostal vessels and lymph nodes exist, but are not enlarged on physical examination of patients with lymphomas. Also, lymphomas have been associated with other connective tissue disorders, especially rheumatoid arthritis (6) and Sjögren's syndrome (7), but no causal relationship has been demonstrated.

In the case reported, the initial clinical diagnosis was of a seronegative rheumatic disease which was treated satisfactorily with antiinflammatory drugs, but the disease later proved to be malignant lymphoma. Thus, lymphoma should be considered as an uncommon cause of sternoclavicular joint arthritis.

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Griseofulvin for eosinophilic fasciitis

To the Editor:

Griseofulvin has been used for many years in the therapy of progressive systemic sclerosis (PSS) (1–3). Some clinical parameters, especially skin sclerosis, improve if long term treatment is used. Recently, it has been demonstrated (4) that griseofulvin produces a decrease of urinary hydroxyproline in scleroderma patients. This probably means it influences the metabolism of collagen, which is altered in PSS (5–7).

We have been treating a 25-year-old man suffering from eosinophilic fasciitis for about 8 months with griseofulvin only. The disease began 8 months before this treatment was started and the symptoms increased in spite of short treatment with azathioprine (50 mg/day) and corticosteroids (prednisone equivalent 20 mg/day).

Since treatment with griseofulvin was started, the clinical manifestations (especially hardening of the abdominal wall) have improved considerably. The eosinophils decreased from 1,411/mm^3 to 350/mm^3, gammaglobulins from 0.233 gm/liter to 0.182 gm/liter, erythrocyte sedimentation rate from 36 mm to 10 mm the first hour, and urinary hydroxyproline from 46 mg/24 hour/mm^2 to 30 mg/24 hour/mm^2 (our normal upper limit 20 mg/24 hour/mm^2 body surface area). The patient is still being treated because some symptoms are still present.

Corticosteroids, especially at high doses, are often useful in the therapy of eosinophilic fasciitis (8). This case shows that griseofulvin may represent an alternative to corticosteroid therapy. Griseofulvin may influence collagen metabolism; furthermore, the fact that griseofulvin is active in systemic sclerosis as well as in

eosinophilic fasciitis, but not in other connective tissue diseases, may mean that eosinophilic fasciitis is closer to systemic sclerosis than to other connective tissue diseases. In effect, collagen metabolism is altered in both these diseases. Thus, they could form a particular subgroup: "connective tissue diseases with collagen accumulation."

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Frostbite arthritis

To the Editor:

The recent article "Arthritis after Frostbite Injury in Children" by Carrera et al (Arthritis Rheum 22:1082–1087, 1979) states that frostbite arthritis may be differentiated by the absence of bony sclerosis and osteophytes and by frequent asymmetry. The following case is representative of a similar clinical problem but with different radiographic abnormalities.

A 22-year-old white woman presented to my office complaining of some discomfort in her hands. She stated that her fingers hurt her to a moderate degree "for as long as she could remember." The patient reported that she had developed lumps on her fingers at the age of 2 after an episode of severe exposure to cold followed by frostbite injury. Clinical examination showed evidence of Heberden's and Bouchard's nodes of several of her distal (DIP) and proximal interphalangeal (PIP) joints. There was no synovial proliferation, increased warmth, or swelling of any other peripheral joint.

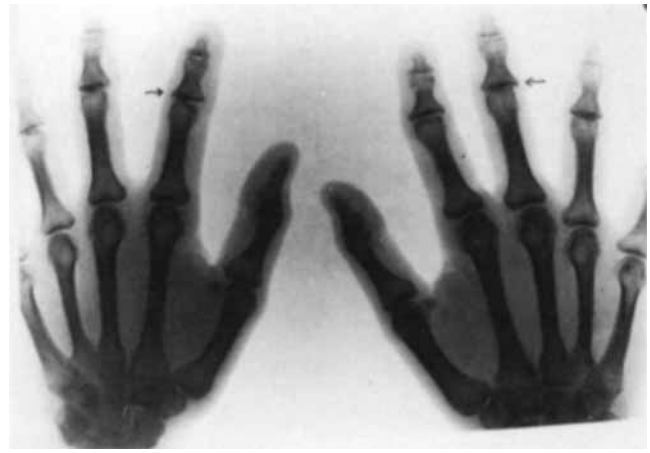

Figure 1. Osteophyte formation (**arrows**) resulting from frostbite injury.

Results of laboratory tests, including Westergren sedimentation rate and chemical profile, were within normal limits. There was nothing in the clinical examination, such as hyperlaxity syndrome or acromegaly, or laboratory testing to suggest other etiologies of premature osteoarthritis. Radiograph of her hands demonstrated osteophyte formation of several of the DIP and PIP joints (Figure 1).

This case is presented to demonstrate that there is no consistent radiographic finding to suggest frostbite arthritis, as suggested by Carrera et al. Their two patients demonstrated irregularity of the articular surfaces with some early subchondral lucencies resembling cysts, indicative of damage to articular cartilage and subchondral bones. The most dramatic finding in my patient is the moderate degree of osteophyte formation. I would suggest that the most important finding in diagnosing frostbite arthritis would be the history of such an injury and the exclusion of other causes of osteoarthritis.

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