normal or negative: tests for hepatitis B antigen, rheumatoid factor, antinuclear antibodies, cryoglobulin, level of complement components, and Coombs' test. A skin specimen from an urticarial lesion showed an infiltrate with numerous polymorphonuclear leukocytes around the dermal vessels. Direct immunofluorescence was negative. Bone x-ray film showed patchy condensation of the iliac crest, but a bone marrow biopsy specimen was normal.

The clinical symptoms were noticeably improved as a result of treatment. The attacks of urticaria became far less frequent and less intense and were not accompanied by fever.

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REFERENCE

Kaposi's sarcoma and Castleman's disease

To the Editor: I read with interest the study by Klapman (J AM ACAD DERMATOL. 1989;20:583-6) that discusses cutaneous diseases that precede lymphoreticular malignancies. In addition to the lesions mentioned by Klapman, Kaposi's sarcoma may also precede Castleman's disease (multicentric giant lymph node hyperplasia).

Castleman's disease is a heterogeneous lymphoproliferative disorder that may progress to malignant lymphoma. The aggressive form, the plasma cell variant, typically presents with generalized lymphadenopathy with associated polyclonal gammopathy, hypoalbuminemia, low serum iron levels, and splenomegaly. Histologically the lymph nodes demonstrate follicular hyperplasia with infiltration of mature plasma cells. The association of preceding or concurrent Castleman's disease with Kaposi's sarcoma is well documented.1

A current theory of the pathophysiology of Castleman's disease is dysregulation of the immune system with concurrent Epstein-Barr virus infection.2 Furthermore, Castleman's disease has been associated with numerous autoimmune disorders and, in fact, I am currently describing a case associated with pernicious anemia. As hypothesized by Klapman, a defect in immune surveillance may indeed predispose to both cutaneous malignancies and lymphoreticular disorders such as Castleman's disease.

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REFERENCES

Musculoskeletal symptoms and bone lesions in acne fulminans

To the Editor: We read with interest Jemec and Rasmussen's article, "Bone Lesions in Acne Fulminans: Case Report and Review of the Literature" (J AM ACAD DERMATOL. 1989;20:353-7). They described a case of acne fulminans with lesions in the clavicles and reviewed nine other cases of bone lesions reported in the literature. The associated musculoskeletal symptoms seem to appear in the most severe forms of acne fulminans, and a septic infection with osteomyelitis is often suspected. We recently reported five such cases of acne fulminans, four of which involved the development of bone lesions.1 We now have examined two additional cases,2 which suggest that such a condition is not as rare as previously thought.

The major clinical and laboratory findings of our patients are summarized in Table I. All the patients have been young men who had acute exacerbations of mild acne with ulcerative lesions and the simultaneous appearance of systemic symptoms such as septic fever, malaise, and various musculoskeletal complaints. The joint symptoms that appeared 2 weeks or more after the sudden exacerbation of acne included generalized pain in the chest or lower back, as well as local tenderness and swelling over the affected bones. X-ray films or bone scans in all six patients revealed osteolytic changes or increased uptake in one or several bones. Surgical exploration in three patients showed only chronic inflammatory material, and all bacterial cultures taken from the bones or blood remained negative. Because infection was suspected, all six patients received combined antibiotic treatment from 1 to several weeks with little improvement. Thus we agree with Jemec and Rasmussen that despite the clinical presentation, no evidence of a direct infectious mechanism causing the bone lesions in acne fulminans presently exists.

Acne fulminans responds to steroid treatment,3,4 and three of our patients (Table I, Nos. 4 to 6) received prednisolone (initial dosages 30 to 40 mg/day) combined with antibiotics. As in the case of Jemec and Rasmussen, our patients' condition improved quickly. All three patients, however, had minor flares of musculoskeletal symptoms when the steroid dosage was tapered. In one patient (No. 5) the steroid treatment had to be continued for 3½ months. Shortly thereafter, this patient had a sudden relapse with systemic symptoms and sternoclavicular joint swelling recurred. For this episode the patient received...
**Table I. Clinical and laboratory findings in six cases of acne fulminans with bone lesions**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)/ Sex</th>
<th>Fever* (°C)</th>
<th>ESR* (mm/hr)</th>
<th>Musculoskeletal symptoms</th>
<th>X-ray film or bone scan findings</th>
<th>Histologic findings</th>
<th>Flares or relapse of musculoskeletal symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>15/M</td>
<td>38.6</td>
<td>83</td>
<td>Pain in left shoulder; swollen knees</td>
<td>Osteolytic defect in left acromion</td>
<td>ND</td>
<td>Minor flares during 4 mo</td>
</tr>
<tr>
<td>2</td>
<td>16/M</td>
<td>38.0</td>
<td>98</td>
<td>Chest pain; swelling in sternum</td>
<td>Osteolytic defect; increased uptake in sternum</td>
<td>Tumorous mass in corpus sterni; chronic inflammation†</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>14/M</td>
<td>38.6</td>
<td>80</td>
<td>Pain in both legs, right hip</td>
<td>Osteolytic defect in left fibula; increased uptake in fibula, left clavicula</td>
<td>Chronic inflammatory material in clavicula†</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>16/M</td>
<td>38.0</td>
<td>70</td>
<td>Pain in chest, lower part of back</td>
<td>Osteolytic defect, increased uptake in sternum</td>
<td>ND</td>
<td>Minor flares during 2 mo</td>
</tr>
<tr>
<td>5</td>
<td>16/M</td>
<td>39.2</td>
<td>128</td>
<td>Pain in chest; swelling in left sternoclavicular joint</td>
<td>Osteolytic defect in left clavicula; increased uptake in clavicula, sternum</td>
<td>ND</td>
<td>Minor flares during 3½ mo; severe relapse after 4 mo with fever, swelling in sternoclavicular joint</td>
</tr>
<tr>
<td>6</td>
<td>15/M</td>
<td>39.0</td>
<td>94</td>
<td>Pain in chest, sacrum; swelling in right clavicula, wrist</td>
<td>Increased uptake in medial end of right clavicula, wrist</td>
<td>Nonpurulent material in clavicula†</td>
<td>Minor flares during 3½ mo</td>
</tr>
</tbody>
</table>

*Highest value measured during hospital stay.
†Negative cultures for bacteria.

ESR, Erythrocyte sedimentation rate; ND, not done.

only prednisolone (30 mg/day), and the symptoms were controlled within a week. These established responses to steroid treatment in acne fulminans with musculoskeletal symptoms seem to favor some underlying immunologic mechanism.

Assessment of complement levels and circulating immune complexes has not disclosed any common abnormality. Patients with acne fulminans can have depressed delayed skin test reactions during the acute phase of the disease, but it is unlikely that cell-mediated immunity is altered permanently in these patients. After the active phase of the disease, we have found only normal helper and suppressor T cell ratios and lymphocyte proliferation tests also have been normal. In contrast to our findings, Jemec and Rasmussen found depressed lymphocyte transformation and suppressor T cell function in their patient, but no data were given to indicate whether these tests were performed during or after the active phase of the disease.

In ordinary acne vulgaris the delayed skin reactivity to *Propionibacterium acnes* seems to correlate with the severity of the inflammation. In acne fulminans with bone involvement it has been proposed that a hypersensitivity reaction induced by skin bacteria may lead to the skeletal disease. We could not find any in vitro evidence, however, that the T lymphocyte responses to *P. acnes* were altered when we examined this function in one patient with acne fulminans; furthermore, the Langerhans cells and peripheral blood monocytes normally presented *P. acnes* antigen to T lymphocytes.

Immunogenetic factors are important in the pathogenesis of reactive arthritis. Thus we tissue typed two of our patients with acne fulminans and musculoskeletal symptoms. Interestingly, the first patient had human lymphocyte antigen (HLA)-B27, but the second patient and the two patients described in Jemec and Rasmussen's report were negative for this antigen. This suggests that the development of musculoskeletal symptoms in acne fulminans is not a HLA-B27-related phenomenon.

It seems evident that a causal relationship exists between the skin and bone lesions in acne fulminans, al-
though the reported cases have not disclosed any common bacterial or immunologic cause. This systemic disorder may cause diagnostic difficulties to the clinician because of the multiple symptoms and young age of the patient. In acne fulminans the preferred site for bone lesions seems to be the claviculae and sternum, but the long and small bones of the extremities may also be involved. As discussed by Jemec and Rasmussen, especially bacterial infection but also malignancy must be considered in the differential diagnosis. Moreover, radiologically identical bone lesions can occur in pustulosis palmoplantaris, another skin disease that is characterized by polymorphonuclear leukocyte inflammation but that has a milder course than acne fulminans.

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REFERENCES

Reply

To the Editor: We thank Reunala et al. for their kind comments and interesting observations.

The cause of the bone lesions, or acne fulminans itself, remains elusive. However, it seems reasonable to cease considering frank bacterial infection of the skin and bones as a possible etiologic factor. Immunity is frequently suggested to be of etiologic importance. Reunala et al., however, found no signs of altered immunity in their patients. Our patient had depressed lymphocyte transformation and suppressor T cell function in the active phase of the disease.

The observations of Reunala et al. and our review of the literature lead us to believe that bone lesions occur much more frequently in severe acne or acne fulminans than previously believed. In view of this, we find their concluding remarks to be of particular interest. Recent studies have shown that the incidence of skeletal manifestations in pustular psoriasis of the Barber type (pustulosis palmoplantaris) ranges from 9% to 36%, depending on the diagnostic method used. Clinically and radiologically the bone changes in the two diseases are very similar, and neither seems to be associated with the HLA-B27 tissue type. Although the morbidity in acne fulminans and pustular psoriasis of the Barber type is different, severe sterile pustulosis is a central element in both conditions. Comparative studies of the bone lesions in the two diseases might be rewarding.

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REFERENCES

Zidovudine treatment of psoriasis associated with acquired immunodeficiency syndrome

To the Editor: We read with interest the article by Kaplan et al. (J AM ACAD DERMATOL 1989;20:76-82) that described a marked antipsoriatic effect of zidovudine in psoriasis associated with acquired immunodeficiency syndrome (AIDS). We report two other cases of AIDS-associated psoriasis that were treated successfully with zidovudine.

Case reports

Case 1. Extensive psoriasis and Pneumocystis carinii pneumonia developed in a 34-year-old homosexual man in June 1988. In November 1988 the patient began treatment with zidovudine (200 mg every 4 hours). Within 2 weeks the psoriasis resolved. At the time of this writing he is in complete remission and has been taking zidovudine for 7 months.

Case 2. Psoriasis developed in a 36-year-old intravenous drug abuser in June 1988. During the next 3 months the patient became febrile and persistent diarrhea developed with weight loss. In November 1988 he presented with P. carinii pneumonia and psoriasis of the entire body. In December 1988 he began zidovudine treatment (200 mg every 4 hours). During the next 4 weeks the psoriasis cleared completely and the fingernails also improved. In March 1989 he stopped zidovudine treatment and