LETTERS

The "hollow scratch" sign

To the Editor:

Both polymyalgia rheumatica and malignancy are common afflictions of the elderly and may present with similar signs and symptoms. In many instances, the patient may provide a key bit of information during the review of systems, which may help with the diagnosis. I would like to report a case in which a patient with suspected polymyalgia rheumatica contributed a clue that led to the eventual diagnosis of metastatic renal cell carcinoma.

A 76-year-old woman with a tentative diagnosis of polymyalgia rheumatica was admitted to the hospital after a 3-month history of progressive fatigue, malaise, low-grade fever, and bilateral shoulder pain and weakness. She had been treated, by an orthopedic surgeon, with intrabursal steroid injections and nonsteroidal antiinflammatory drugs with some benefit. During the admission interview, the patient denied actual scalp tenderness but complained of a peculiar "hollow" feeling that was elicited by scratching the occipital area of her head.

The physical examination revealed the presence of bilateral shoulder pain on both passive and active abduction. Range of motion of the shoulders was very restricted because of pain. No abnormalities of the head were noted.

Pertinent laboratory data included a normochromic normocytic anemia, an elevated erythrocyte sedimentation rate of 86 mm/hour (Westergren), and normal blood chemistries. A skull radiograph was obtained because of the patient's peculiar complaints regarding her head. A 2-cm lytic lesion was noted in the left occipital region which corresponded to the "hollow" area described by the patient. Radiographs of the shoulders revealed lytic lesions in both humeral heads. These results prompted a search for an underlying tumor which eventually turned out to be an adenocarcinoma of the kidney.

Despite a review of the literature, I was unable to find a similar description of the "hollow scratch" sign described above. While the presence of a lytic lesion on the skull radiograph did not establish the source of the primary cancer, it certainly did alter the direction of the diagnostic process.

The clinical presentation of an elderly patient with a low-grade fever, malaise, and proximal muscle aching should obviously prompt a search for underlying malignancy. Often, the patient may contribute a clue that helps with the differential diagnosis. This case underscores the value of obtaining a detailed review of systems and of paying attention to "minor" complaints.

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Comment on report by Liberman et al

To the Editor:

In their excellent article (Liberman UA, Samuel R, Halabe A, Joshua H, Lubin E, Zoref-Shani E, Sperling O: Juvenile metabolic gout caused by chronic compensated hemolytic syndrome. Arthritis Rheum 25:1264–1266, 1982), Liberman et al do not mention measurement of red blood cell phosphofructokinase and its isozymes in their patients with compensated hemolytic anemia. Deficiency of this enzyme may be associated with isolated hemolysis and teenage onset of gout, an association we were unaware of until we read Dr. Liberman's paper in conjunction with Dr. Vora's review (Vora S: Isozymes of phosphofructokinase, Isozymes. Current Topics in Biological and Medical Research 6:119–167, 1982).

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Piroxicam (Feldene) predisposes to chemotherapy-induced acute uric acid nephropathy

To the Editor:

Nonsteroidal antiinflammatory agents are associated with a wide variety of renal disorders (1,2) including acute renal failure (3,4), acute interstitial nephritis (4), hyperchloremic acidosis (5), and edematous disorders (6). In addition to these overt renal abnormalities, a patient taking these compounds may be predisposed to renal failure from an otherwise insignificant insult. We here describe a patient receiving chemotherapy who developed acute uric acid nephropathy with the concomitant use of the nonsteroidal drug piroxicam (Feldene).

An 81-year-old man was admitted for confusion, weakness, and oliguria 5 days after his third chemotherapy cycle for clinical stage III, diffuse, poorly-differentiated lymphocytic lymphoma (null cell), which had been diagnosed 3 months before this admission. Chemotherapy with Cytoxan (1,500 mg), vincristine (1 mg), and prednisone (50 mg daily for 5 days) had been administered monthly on 2 previous occasions without complications. Allopurinol was not given before any of the chemotherapy treatments. No abnormal values of electrolytes, blood urea nitrogen (BUN), creatinine, or serum uric acid were noted during the previous chemotherapy cycles. Clinical response to the chemotherapy was judged appropriate.

Two weeks before the third cycle of chemotherapy, Feldene was administered for nonspecific arthritis complaints. Laboratory test results were normal (BUN 10 mg/dl, creatinine 0.8 mg/dl, serum uric acid 4.5 mg/dl) 5 days after

this medication was initiated. Renal function was unchanged from previous values. The patient was not taking any medicine other than Feldene.

The patient was admitted 5 days after the third chemotherapy cycle. No change was made in the chemotherapy dosage. Physical examination revealed normal intravascular volume status. Laboratory tests showed a BUN of 144 mg/dl, calcium of 6.5 mg/dl, potassium of 9.0 mEq/liter, and a serum uric acid of 38.7 mg/dl. Urinalysis revealed numerous urate crystals. Renal sonography findings were normal.

Acute uric acid nephropathy was diagnosed and hemodialysis therapy was instituted. After 3 hemodialysis treatments the patient's serum uric acid was 9.7 mg/dl. Shortly thereafter, the urine output increased and renal recovery occurred uneventfully.

This patient's acute renal failure was caused by acute uric acid nephropathy secondary to tumor lysis. This is a well-known complication of cancer chemotherapy. When it occurs, it virtually always follows the first chemotherapy cycle (7). This patient, however, did not develop this syndrome until the third chemotherapy treatment; the addition of the nonsteroidal drug, Feldene, prior to the third cycle suggests that this agent was related to the development of the renal failure.

Although the nephrologic syndromes of the nonsteroidal antiinflammatory drugs are usually easily recognized (1,2), these agents may change renal homeostasis in a more occult fashion. With the inhibition of prostaglandin synthesis, these compounds decrease renal blood flow and glomerular filtration rate (8). Such alterations have previously been shown to predispose the patient to kidney failure resulting from a variety of insults. We suspect that such an alteration in renal hemodynamics predisposed our patient to acute renal failure due to a decrease in urate clearance during a time of elevated urate release from the chemotherapy.

Despite the increasing use and number of nonsteroidal antiinflammatory agents, we are unaware of previous reports linking these agents with acute uric acid nephropathy secondary to cancer chemotherapy. This case should alert physicians that piroxicam may increase the likelihood of acute renal failure developing with tumor lysis from cancer chemotherapy. Other nonsteroidal agents may have the same effect. If such agents must be given in addition to chemotherapy, the prophylactic use of allopurinol seems mandatory, as well as careful clinical followup after the chemotherapy has been given.

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Thymuline (FTS) in rheumatoid arthritis

To the Editor:

A beneficial effect of thymopoietin on the course of rheumatoid arthritis (RA) has been recently reported by Veys et al (1). Like thymopoietin, synthetic thymic serum factor (FTS), now called thymuline, is a pharmacologically active compound which can have various effects on T lymphocytes (2). We therefore undertook to evaluate it in RA. Two open trials were conducted using outpatients with active RA who gave oral consent. Eight female patients received 150 μg thymuline (Choay, Paris, France) subcutaneously each day for 2 weeks and then 3 times a week for 4 months. Eight other patients received 1 mg thymuline according to the same protocol. The age range of the subjects was 40–78 years, with means of 55.2 and 60.4 years in the two groups, respectively. The mean duration of disease was 15.1 ± 4.9 years and 16.5 ± 7.4 years, respectively. Rheumatoid factor was present in 6 of 8 patients in each group.

Patients had not been treated with basic slow acting antiinflammatory drugs in the preceding 3 months. RA was active despite the optimal use of other antiinflammatory drugs, and all patients continued to take the analgesic and/or antiinflammatory drugs they had been taking at the time of admission. Six patients in each group were receiving oral prednisone therapy for more than 3 months prior to the study.

Clinical and biologic statuses were evaluated monthly by the same investigator. Changes in these statuses were evaluated after 4 months of thymuline therapy. Statistical analysis was performed using the Student's *t*-test.

Table 1 summarizes clinical parameters evaluated at the beginning of the trials and the mean changes after 4 months of treatment. In the group of patients treated with