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The Clinical Picture

The crystal, the gout, and the paradox



FIGURE 1

WE THINK we know what causes gout: chronic hyperuricemia causes synovial tissue supersaturation with urates, which leads to crystal formation and the acute gouty attack. We think that crystals are necessary and sufficient for the acute gouty attack to occur.

Things may not be that simple.

Although crystals are necessary for gout, they may not be sufficient by themselves. Urate crystals are often found in the synovial fluids from asymptomatic joints of patients who have had gouty arthritis, as well as in asymptomatic tophi. Experimental studies suggest that crystals cause less inflammation the longer they are in an inflammatory environment, perhaps because they become coated with proteins.

Also, in chronic gouty arthritis, although crystals can be found in the synovial fluid and synovium of the affected joints, and osseous tophi and erosions may be apparent on radio-graphs, the joints are far less tender and painful than in acute gouty arthritis.

FIGURE 1 shows the hands of a 68-year-old man with chronic gout. His complaint was chronic stiffness, mildly worse in the morning, and clumsiness due to loss of finger mobility. He had never been treated for gout.



FIGURE 2

Aspiration of a swollen but nonpainful joint revealed sodium urate crystals.

FIGURE 2 shows an intradermal tophus of the thumb in the same patient. These “bumps” would occasionally drain “pus,” according to the patient, but did not hurt. The pus most likely was chalky urate deposits. Tophi frequently appear in areas of trauma, and the finger pads are not spared.

FIGURE 3 shows “urate milk” aspirated from the distended but otherwise asymptomatic olecranon bursa of a 42-year-old man who had never previously been diagnosed with gout. On questioning, he recalled a few episodes of “painful red bunions” and “twisted ankles,” which in retrospect were likely attacks of gouty arthritis. The fluid revealed sheets of urate crystals, with only a scant number of leukocytes (60% neutrophils). No lipid crystals were observed.

Both patients were treated initially with colchicine (0.6 mg by mouth twice a day) and enough allopurinol to lower the serum uric acid level to approximately 5 mg/dL.



FIGURE 3

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