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Answer: CHRONIC TOPHACEOUS GOUT

This patient has numerous painless yellowish nodules in the hands, wrists, elbows and feet consistent with chronic tophaceous gout. His serum uric acid was within normal range and was continued on allopurinol and colchicine therapy.

Gout is a disorder of purine metabolism with resultant hyperuricemia. Intermittent precipitations of monosodium urate crystals in joints and soft tissues result in acute gout attacks. However, not all with hyperuricemia will experience acute gout. Chronic hyperuricemia can lead to chronic complications which include tophi depositions, nephron-urolithiasis and associated renal failure, and other complications (metabolic and cardiovascular).¹

Chronic tophaceous gout is now uncommon and typically occur in patients who are untreated or inadequately treated, often after 10 years or more of recurrent polyarticular gout.¹ Transplant recipients, women, and elderly people are particularly susceptible to developing tophi.² Continuous deposition of urate crystals leads to nodules formations involving the head and neck, skin, viscera, bones, tendons, ligaments, nerves, and axial skeleton. This can lead to joint deformities and destruction, pain, damage to surrounding

soft tissues and nerve compressions (i.e. carpal tunnel syndrome and spinal cord compressions).³ Areas frequently affected include helix of the ears, olecranon bursae, hands, knees, feet and fingers.³ However, any part of the body can be affected.

Gout can cause diagnostic dilemmas, as it can be a great mimicker of and can co-exist with infection, malignancy, and other connective tissue diseases. Plain radiography of affected areas often shows soft tissue densities of the nodules with punched out erosions of the bone adjacent to the nodules. For less common location biopsies may be required for diagnosis.

Treatment of tophaceous gout include management and prevention of acute attacks; control and termination of acute inflammatory and urate precipitations (steroids, colchicine, non-steroidal anti-inflammatory drugs and COX-II inhibitors) and for long-term control with uric acid reducing agents such as xanthine oxidase inhibitors (allopurinol, febuxostat) through reduction of production of uric acid and uricosuric agents (probenecid). Refractory case may need to be treated with Pegloticase, a recombinant pegylated uricase.⁴ Patients should also avoid food high in purine and be aware of medications that can precipitate attack. For some cases, surgery may be required to remove the nodules.

REFERENCES

- 1: Becker MA, Ruoff GE. What do I need to know about gout? *J Fam Pract.* 2010; 59(6 Suppl):S1-8.
 - 2: MacFarlane LA, Kim SC. Gout: a review of nonmodifiable and modifiable risk factors. *Rheum Dis Clin North Am.* 2014; 40:581-604.
 - 3: Forbess LJ, Fields TR. The broad spectrum of urate crystal deposition: unusual presentations of gouty tophi. *Semin Arthritis Rheum.* 2012; 42:146-54.
 - 4: Nuki G. An appraisal of the 2012 American College of Rheumatology Guidelines for the Management of Gout. *Curr Opin Rheumatol.* 2014; 26:152-61.
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