

CLINICAL PRACTICE

Clinical Images

Digit-Threatening Severe Tophaceous Gout

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CLINICAL DESCRIPTION

A 54-year-old Tongan man with a 20-year history of gout, complicated by bilateral below knee amputations 2 years prior for severe foot infections, presented with





Figure 1 Panel A: Photograph demonstrating extent of bilateral tophaceous disfigurement of digits. Panel B: Radiograph depicting tophi eroding joint spaces and dislocation of digits 1–3 at the metacarpal-phalangeal joints.

bilateral hand pain. He had taken xanthine oxidase inhibitors previously, but had been without medications or primary medical care for over a year. The patient's hands ((Fig. 1, Panel A) were disfigured by tophi and had an overlying cellulitis with weeping serosanguinous as well as thick, milky-white fluid. Radiographs ((Fig. 1, Panel B) demonstrated diffuse demineralization of the bone and calcification of tophi eroding and destroying joint spaces. Labs were notable for a uric acid level of 11.9 mg/dL, well above the level at which urate precipitates out of solution and can begin to form tophi—a process directly correlated with the duration and severity of hyperuricemia. If left untreated, up to 75 % of patients with gout will eventually develop tophi, while only 5 % do so if adequately treated.²⁻⁴ While the incidence of gout has increased to 3.9 % as of 2008, the incidence of tophi continues to decline due to wider availability of effective urate-lowering treatment.⁵ Our patient underwent wound debridement without amputation of digits and was discharged home on a xanthine oxidase inhibitor.

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REFERENCES

- Choi H, Mount D, Reginato A. Pathogenesis of gout. Ann Intern Med. 2005;143:499–516.
- Gutman AB. The past four decades of progress in the knowledge of gout, with an assessment of the present status. Arthritis Rheumatol. 1973:16:431–45.
- O'Duffy JD, Hunder GG, Kelly PJ. Decreasing prevalence of tophaceous gout. Mayo Clin Proc. 1975:50:227–8.
- Harris MD, Siegel LB, Alloway JA. Gout and hyperuricemia. Am Fam Physician. 1999:59(4):925–34.
- Zhu Y, Pandya BJ, Choi HK. Prevalence of gout and hyperuricemia in the US general population: The National Health and Nutrition Examination Survey 2007–2008. Arthritis Rheum. 2011;63(10):3136-41.