# Gout

Timothy S.H. Kwok MD, Victoria Y.Y. Xu MD, Shirley L. Lake MD MSc

■ Cite as: *CMAJ* 2021 February 1;193:E171. doi: 10.1503/cmaj.201392

## 1 The prevalence of gout is rising, and many patients have common comorbidities

The prevalence of gout is about 4%, reflecting a 59% increase over the past decade.[1] More than 60% of patients with gout also have a metabolic syndrome, so patients should be assessed with this in mind. Risk factors for gout include male sex, increasing age, chronic kidney disease, dehydration (including diuretic use), hyperuricemia from rapid cell turnover (e.g., hemolysis, chemotherapy) and excessive purine consumption (Appendix 1A and B available at www.cmaj.ca/lookup/doi/10.1503/cmaj.201392/tab-related-content).[2]

## 2 Gout flares are typically monoarticular, reaching maximal intensity within hours

Flares often occur at night and usually target distal joints (e.g., first metatarsophalangeal joint). Joints with previous trauma and degenerative changes are predisposed to gout (Appendix 1C). Periarticular erythema, warmth and overlying desquamation may mimic infection.[2] The differential diagnosis for acute gout includes septic arthritis, traumatic arthritis and hemarthrosis, especially among patients on anticoagulants. In contrast to gout, osteoarthritis tends to present as a chronic condition, and inflammatory arthritis is typically of polyarticular distribution.[2]

## 3 Synovial fluid crystal analysis is the diagnostic gold standard

Microscopy shows needle-shaped monosodium urate crystals with negative birefringence. Gout and septic arthritis can co-occur; therefore, it is important to rule out infection with Gram staining and cultures. Radiographs may show tophi or overhanging erosions. Although serum uric acid can be elevated, normal levels do not preclude gout. A validated diagnostic rule can aid in diagnosis without arthrocentesis (Appendix 1A).[3]

## 4 Gout treatment within 24 hours of symptom onset reduces flare intensity and duration

Acute pharmacotherapy depends on patient comorbidities. Nonsteroidal anti-inflammatory drugs, colchicine and corticosteroids are first-line therapies (Appendix 1D). Pre-existing urate-lowering therapy (ULTs) should be continued during flares.[4]

## 5 ULTs should be started in patients with tophaceous disease, radiographic damage from gout or ≥ 2 gout attacks/year

ULTs (Appendix 1D) should be titrated to serum urate < 360 µmol/L by assessing levels monthly. Serum urate levels < 360 µmol/L promote crystal dissolution and reduce the chance of recurrent flare by about 80%.[5] Acute gout prophylaxis with colchicine, naproxen or prednisone should be prescribed during the initial 3–6 months of ULT (Appendix 1D). Losing weight (i.e., decreasing body mass index by > 5% ), limiting alcohol (< 1–2 units/day), and reducing fructose (< 1g/kg/day) and purine intake may help reduce urate levels.[4] Multidisciplinary approaches for comorbidity management and subspecialist referrals should be considered, if appropriate.

## References

1. Rai SK, Aviña-Zubieta JA, McCormick N, et al. The rising prevalence and incidence of gout in British Columbia, Canada: population-based trends from 2000–2012. *Semin Arthritis Rheum* 2017;46:451-6.
2. Dalbeth N, Merriman TR, Stamp LK. Gout. *Lancet* 2016;388:2039-52.
3. Janssens HJ, Fransen J, van de Lisdonk EH, et al. A diagnostic rule for acute gouty arthritis in primary care without joint fluid analysis. *Arch Intern Med* 2010;170:1120-6.
4. FitzGerald JD, Dalbeth N, Mikuls T, et al. 2020 American College of Rheumatology guideline for the management of gout. *Arthritis Care Res (Hoboken)* 2020;72:744-60.
5. Shoji A, Yamanaka H, Kamatani N. A retrospective study of the relationship between serum urate level and recurrent attacks of gouty arthritis: evidence for reduction of recurrent gouty arthritis with antihyperuricemic therapy. *Arthritis Rheum* 2004;51:321-5.

**Competing interests:** None declared.

This article has been peer reviewed.

**Affiliations:** Department of Medicine (Kwok, Xu, Lake), and Divisions of Geriatric Medicine (Xu) and Rheumatology (Lake), University of Toronto; Division of Rheumatology (Lake), Sunnybrook Health Sciences Centre, Toronto, Ont.

**Acknowledgements:** The authors would like to acknowledge Drs. Lucille Chan, Betty Lee and Mark Leung for their constructive comments on drafts of this manuscript.



**Correspondence to:** Shirley Lake, shirley.lake@sunnybrook.ca