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Managing Gout in the Clinic: Pearls for Family Medicine Specialists

Shelly Dunne, MD

Gout is the most common form of inflammatory arthritis worldwide, often presenting diagnostic and management challenges. However, it can also be gratifying to treat, as successful treatment provides relief from an extremely painful arthritis, and proper preventive strategies can lead to a gout free future. We will begin with a case from my clinical practice.

Case

Meet Steve, a 49-year-old engineer with a 3-year history of gout, primarily affecting his left big toe. His medical history includes type 2 diabetes, hypertension (HTN), and obesity. He is currently taking metformin, telmisartan, hydrochlorothiazide, and he uses indomethacin as needed. There is a strong family history of gout—his father and brother are both affected. Steve was born in the Philippines, is a lifelong nonsmoker, and consumes beer, mostly on weekends, up to 12 per week. While his diet is generally healthy, he has a particular fondness for shellfish and red meat. He has become increasingly concerned because his gout attacks have grown more frequent and prolonged. His most recent attack lasted longer than previously and involved his knee and ankle as well as his big toe. Laboratory findings were normal apart from an elevated uric acid level of 531 µmol/L. Steve is seeking guidance on how to best manage his painful attacks, and importantly, how to prevent future attacks. He is particularly interested in dietary modifications and has expressed concern about potential side effects of the urate-lowering therapies (ULT).

Diagnosis	Characteristics
Bacterial cellulitis	Erythema over the surface of a joint can be confused for a gout flare-up; however, the joint is typically nontender without the presence of effusion
Basic calcium phosphate deposition disease	Milwaukee shoulder syndrome (rapidly progressing crystal arthropathy involving the shoulder and intra-articular deposition of hydroxyapatite crystals)
Calcium pyrophosphate dihydrate deposition disease ("pseudogout")	Calcium pyrophosphate dehydrate in fluid aspirate from joint
Osteoarthritis	Gradual typical onset, commonly in the hand, knee, hip, or first metatarsophalangeal joint
Psoriatic arthritis	Characteristic skin and nail changes
Reactive arthritis	Inflammatory polyarthritis in reaction to bacterial infection (commonly Chlamydia trachomatis genitourinary infections or gastrointestinal infections with Campylobacter, Salmonella, Shigella, or Yersinia)
Rheumatoid arthritis	Slow onset with symmetric joint involvement, commonly in hands
Sarcoidosis	Lofgren syndrome involving the ankles and erythema nodosum that can appear similar to gout; however, hilar adenopathy and lung involvement are not present in gout
Septic arthritis	Associated fever, elevated white blood cell count, elevated erythrocyte sedimentation rate

Table 1. Differential Diagnosis of Gout; *adapted from AFP: Gout: Rapid Evidence Review by Karl T. Clebak, MD; Ashley Morrison, MD; and Jason R. Croad, DO.*

Diagnosing Gout

Accurate diagnosis is key to effective gout management. When a patient presents with a red, hot, swollen big toe, chances are very high that gout is the correct diagnosis. While any joint can be involved, the ankle, foot, and knee are commonly affected sites. While joint aspiration and synovial fluid analysis for uric acid crystals remain the diagnostic gold standard, aspiration may not be necessary in patients with multiple risk factors and elevated serum uric acid. However, it is worth considering other diagnoses, specifically septic arthritis and other inflammatory arthritides (**Table 1**).

Risk Factors for Gout

Understanding the risk factors for gout is equally important. Steve, for example, illustrates several classic risk factors: male sex, type 2 diabetes, HTN, family history of gout, and a purine-rich diet. **Table 2** provides a more comprehensive list of risk factors.

Acute Flare Management: Treat the Acute Attack with the Safest Option Available

When treating an acute gout attack, the goal is to use the safest option available for the individual patient (**Table 3**). Nonsteroidal anti-inflammatory drugs (NSAIDs) are a good option in low risk patients without renal disease, HTN or a history of gastrointestinal ulcers. Indomethacin is traditionally considered highly effective, and in my clinical experience, it often delivers reliable results. For patients who cannot tolerate NSAIDs, colchicine is a reasonable alternative, especially when initiated early in the course of the attack. While several dosing regimens exist, I prefer to use 0.6 mg twice daily, as higher doses are well-known for causing gastrointestinal side effects such as severe diarrhea. Intra articular steroids are an excellent option when joint injection is feasible. For more widespread or stubborn attacks, or in cases where NSAIDs, colchicine, and injections are not suitable, oral prednisone is my go-to. I usually prescribe a short 5-day course of treatment, starting at 25 mg and reducing by 5 mg each day.

Risk Factors for Gout
Comorbidities
• Cardiovascular disease • Diabetes mellitus • Diuretic use (loop and thiazide) • Elevated triglyceride and cholesterol levels • Hyperuricemia • Menopause • Obesity • Renal disease, including renal insufficiency and chronic kidney disease
Demographic factors
• Certain ethnic groups, including indigenous Taiwanese, Pacific Islander, and New Zealand Maori • Living in high-income countries (specifically North America and western Europe) • Male sex (incidence 2 to 6 times higher than in females)
Dietary factors
• Alcohol consumption • Diet rich in meat • Diet rich in seafood • Fructose-rich food and drink consumption

Table 2. Risk Factors for Gout; *adapted from AFP: Gout: Rapid Evidence Review by Karl T. Clebak, MD; Ashley Morrison, MD; and Jason R. Croad, DO.*

Medication & Dosage	Dosing	Notes
NSAIDs (examples only) • Naproxen • Indomethacin • Celebrex	• 375–500 mg p.o. bid • 25–50 mg p.o. bid-tid • 200 mg p.o daily	Roughly 5–7 days until the acute attack subsides; Assuming no NSAID contraindication, variable dosing with lower doses preferred if effective
Colchicine	• 0.6mg p.o. bid-tid	Roughly 5–7 days until the acute attack subsides; Diarrhea a common side effect, so keep dosing to this lower dose regimen
Corticosteroids • Prednisone p.o. • Methylprednisolone (depomedrol) IM	• Prednisone 25mg po on day 1 and reduce by 5 mg per day to 0 • Methylprednisolone (depomedrol) 80mg IM	Intraarticular preferred if possible

Table 3. Acute management of gout; *courtesy of Shelly Dunne, MD.*

Medication	Mechanism of Action	Dosage
Allopurinol (first line option for all patients	Xanthine oxidase inhibitor	Start at ≤100 mg daily (or lower in ≥ stage 3 chronic kidney disease); dosages can be titrated to 800 mg daily
Febuxostat (Uloric)	Xanthine oxidase inhibitor	Start at ≤40 mg daily; maximum dosage is 80 mg

Table 4. Chronic management of gout using urate-lowering therapies; *adapted from AFP: Gout: Rapid Evidence Review by Karl T. Clebak, MD; Ashley Morrison, MD; and Jason R. Croad, DO.*

Initiating Urate-Lowering Therapy: Key Considerations

Allopurinol remains the first-line agent for ULT and should be considered in patients who experience frequent gout flares, defined as two or more per year, as well as those with tophi (nodules that form from a mass of uric acid crystals at the joints or in the soft tissues) or x-ray evidence of gouty erosions. However, in certain cases, such as after the first gout flare in patients with uric acid levels higher than 540 µmol/L, greater than stage 3 or greater chronic kidney disease, or a history of urolithiasis, early initiation of ULT may be appropriate. ULT is not recommended for asymptomatic hyperuricemia. Notably, recent guidelines now conditionally recommend starting ULT during an acute attack, rather than waiting for the episode to resolve. When initiating allopurinol, it is best to start with a low dose of 100 mg per day, especially in patients with chronic renal failure, as this has been shown to reduce the risk of allopurinol hypersensitivity syndrome. Prophylactic therapy with NSAIDS, colchicine, or low-dose prednisone is recommended during the first 3–6 months of ULT to prevent acute attacks triggered by urate mobilization.

The treatment goal is to reduce serum uric acid to 360 µmol/L or lower, with regular serum urate monitoring and dose adjustments as needed. Genetic screening for the *HLA B5801* allele is recommended in patients of Southeast Asian and African American descent, as this allele is more prevalent in these populations, which places them at an increased risk for severe hypersensitivity reactions to allopurinol (**Table 4**).

Role/Safety of Febuxostat

Febuxostat (Uloric) is a newer xanthine oxidase inhibitor that effectively lowers serum uric acid levels more rapidly than allopurinol. Earlier concerns about increased cardiovascular risk lead to a recommendation for it's use as a second-line agent after allopurinol. However, more recent evidence from the FAST trial has refuted these concerns, suggesting that febuxostat may be safer than initially believed. It remains a valid second-line option, though caution is still advised in patients with preexisting cardiovascular disease. When prescribing febuxostat, it is best to start at the lower dose of 40 mg/day. Ongoing studies are under way that may further inform its safety profile and could lead to updates in future guidelines (**Table 4**).

Lifestyle and Self-management

Patients should be encouraged to adopt lifestyle changes that support long-term gout control. Strategies such as achieving and maintaining a healthy weight, engaging in regular physical activity, and following a low purine diet (limiting intake of red meat, organ meats, shellfish, and sugary beverages). It is also important to reduce alcohol consumption, especially beer, and to avoid beverages sweetened with fructose or other added sugars.

Patient Education

It is important to help patients to understand that while dietary modifications can support gout management, they rarely are sufficient on their own to lower uric acid levels to target. Most patients will require ULT for effective long-term control. Emphasizing the chronic nature of gout is necessary. Patients should be encouraged to adhere consistently to their prescribed ULT regimen and to undergo regular monitoring of their serum uric acid levels to ensure treatment goals are being met.

When to Refer

Referral to rheumatology is appropriate for complex cases of gout, particularly those with refractory symptoms or severe comorbidities such as renal failure or poorly uncontrolled HTN. It is also advisable to consider referral in cases of diagnostic uncertainty or when a diagnostic joint aspiration is indicated for a joint that is difficult to access.

Follow-up: Back to Steve

After a thoughtful discussion regarding the potential side effects of allopurinol and the overall risks and benefits of treatment, Steve agreed to begin ULT. Before starting, he was given a laboratory requisition to test for the *HLA B5801* allele, which fortunately came back negative. He was then prescribed 100 mg of allopurinol daily, along with colchicine 0.6 mg twice daily as prophylaxis against acute attacks. Steve also received counselling on lifestyle modifications, particularly to reduce his intake of beer, shellfish, and red meat. A follow-up appointment was scheduled for 3 months later, with instructions to complete serum uric acid testing beforehand. The plan is to adjust the allopurinol dose if his uric acid level has not reached the target of 360 µmol/L by that time.

Conclusion

In summary, gout remains a common, yet challenging condition encountered in family practice. By applying these evidence-based tips, clinicians can significantly improve outcomes for patients living with gout in 2025.

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