

Confessions of a goutophile: Despite its treatability, gout remains a problem

In the spirit of full disclosure, I am a nonrecovering goutophile. Recent advances have even furthered my enthusiasm for the study of gout and boosted my optimism for improved management of patients with this disorder.

A curious clinical course that finally yields to insights

For years I have been fascinated by the clinical course of the gout—the explosive onset of attacks coupled with their spontaneous resolution. Attacks have been viewed as a relatively simple response to urate crystals, in contrast to the complex cascades that follow auto-antigen stimulation. Until recently, however, the nuances of the response to urate crystals were poorly defined. Several laboratories have contributed to our understanding of the mechanisms triggering the acute attack via activation by various cytokines of specific intracellular pathways involving inflammasomes. Models to explain the self-resolving nature of the attacks have also been developed.

For our patients, the concept that hyperuricemia plays a direct role in the development of hypertension and the progression of chronic kidney disease has been revitalized. Molecular studies have refined our insights into the renal handling of uric acid; we better understand how estrogen, diabetes, and certain medications affect renal uric acid reabsorption. Epidemiologic analyses, animal models of hyperuricemia, and human interventional studies have reintroduced urate as an etiologic agent in cardiovascular disease.

The development of new agents for the treatment of hyperuricemia (a less immunogenic uricase preparation and a nonpurine inhibitor of xanthine oxidase) has stoked interest in—and funding for—research related to patients with hyperuricemia and gout.

Effective diagnosis and treatment: Achievable but not widespread

Gout can be definitively diagnosed by documenting the presence of urate crystals in the synovial fluid from affected joints. Most attacks of gouty arthritis can be readily and safely treated, assuming that attention is paid to the patient's comorbid conditions. There is continuing discussion about which gouty patients need to have their urate levels reduced, but once the decision is made to treat, most patients can be effectively managed with urate-lowering therapies that will reduce the frequency of attacks and shrink tophi. So why does gout remain a problem?

Reviews of physician practice patterns and focus-

group discussions show that despite the high prevalence of hyperuricemia and gouty arthritis, we do a suboptimal job at managing patients with these conditions. Conversations with clinical rheumatologists reveal the shared perception that gout often is misdiagnosed (or goes undiagnosed) because of failure to examine synovial fluids for crystals. There is an overreliance on serum urate levels to diagnose gout, despite the well-recognized lack of sensitivity and specificity of this measure. Plus, interpretation of the serum urate level is complicated by the fact that laboratory “normal” ranges typically include serum urate values above the biological solubility of urate (~6.8 mg/dL).

It also seems that even when the decision is made to treat hyperuricemia, treatment is frequently suboptimal because of limited use of appropriate serum urate targets (ie, levels less than ~6.0 mg/dL), insufficient monitoring of the urate level, and overly conservative drug escalation.

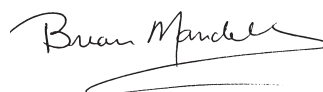
In other words, education in the management of gout and hyperuricemia is sorely needed.

A supplement conceived with nonspecialist feedback

In an effort to understand the difficulties faced by nonspecialists in managing patients with gout and hyperuricemia, I and several other rheumatologists with a special interest in clinical gout and continuing medical education took part in a symposium on this topic in Scottsdale, Ariz., on October 5, 2007. We made presentations to a group of invited internists and family practice physicians and gained feedback from them during breakout sessions that followed our talks.

This supplement is based on the series of formal talks presented at the symposium. The talks were transcribed and the authors developed their transcripts into the articles presented here, taking care to draw on questions and feedback from the breakout sessions to best address the educational needs of nonrheumatologists.

On behalf of my fellow authors, I hope we have succeeded in producing a readable and practical supplement that meets many of those needs and facilitates effective management of our patients with gout and hyperuricemia.



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