

The Management of Gout: It Should Be Crystal Clear



The field of rheumatology is filled with mystery, poorly understood diseases, treatments that are often less than satisfying, and more questions than answers. We do not know what causes rheumatoid arthritis, lupus, spondyloarthritis, and most cases of vasculitis. The treatments for these diseases may not always be effective, and it is often impossible to predict which patients will respond.

But that is not the case with gout: we know what causes gout, we can diagnose it with absolute certainty, and excellent therapies exist. Will it not be wonderful when we can say the same about other rheumatic diseases! But why do not all patients with gout get appropriate management?

Although we understand the pathophysiology of gout a little better today, the ability to diagnose and the therapies available are essentially the same as they were 40 years ago. At a minimum the principles of gout management include:

1. Identification of urate crystals in synovial fluid or soft tissue aspirates establishes the diagnosis of gout
2. Acute gout attacks can be terminated with the use of colchicine, a nonsteroidal antiinflammatory drug (NSAID), corticosteroids, or corticotrophin
3. When recurrent attacks occur, a urate-lowering agent should be prescribed
4. Urate-lowering therapy should be life-long
5. Low-dose colchicine or NSAID should be prescribed in a prophylactic manner prior to initiating urate-lowering therapy and continued for some time thereafter.

Yes, there is debate whether colchicine should be used for treating acute attacks, and if so, what is the most appropriate dosing schedule. Opinions vary whether specific urate-lowering agents should be offered after the first, second, third, or fourth acute attack. Some recommend checking a 24-hour urinary uric acid level (or measuring uric acid and creatinine in a spot urine sample) to determine whether to prescribe a uricosuric agent or allopurinol; others do not. Vigorous discussions continue regarding how long one should take prophylactic colchicine (or NSAID) prior to initiating treatment with a

urate-lowering agent, and how long that prophylaxis should continue. Also, how frequently serum urate levels should be monitored is not precisely defined.

But there is no debate regarding the 5 principles outlined above. When those principles are followed and the dose of urate-lowering agent is sufficient to maintain the serum urate below 6.8 mg/dl (preferably below 5.0 to 6.0 mg/dl), gout attacks cease, and tophaceous deposits resolve. Why is this not crystal clear?

I believe patients continue to suffer the consequences of gout for 2 major reasons: poor patient compliance and incorrect prescribing practices by practitioners. Poor compliance is a problem in the management of any chronic disease, especially if the condition is asymptomatic. In addition, it may be difficult for some patients to fully comprehend how to appropriately take 3 different medications on 3 different dosing schedules. This may occur in part because we have not found a way to effectively educate our patients¹. Finally, the heavy alcohol consumption among many members of the gouty population also contributes to poor compliance.

In this issue of *The Journal*, Fang and colleagues report the results of a survey given to physicians in an academic medical center in Beijing designed to assess whether their management of gout was consistent with current evidence². In general, they found that management was often not consistent with recommended standards of care.

For example, although 78% thought aspiration of joint fluid was important for a definitive diagnosis of gout, this was rarely ever done. Whereas only 12% used any antiinflammatory prophylaxis when initiating a urate-lowering agent, 86% would initiate urate-lowering therapy in less than 2 weeks after onset of an attack, and only 20% would continue such therapy for over 5 years.

If only these management deficiencies were limited to China. Physicians in China may have an excuse, as gout was previously a very rare condition in that country. Fang reported the first 2 cases of gout in 1948, and only 25 patients were

See Management of gout at an academic healthcare center in Beijing, page 2041

identified in the following decade². We in North America have no excuse. Gout has been a common disease for centuries and we have had excellent therapies since the 1950s, when uricosurics became available, and 1964, when allopurinol was approved. There was great excitement with the advent of the urate-lowering agents. Patients with crippling recurrent attacks of arthritis and deforming, destructive tophi were "cured." Where did that excitement go? Perhaps we have taken gout for granted because it is so well understood and can be diagnosed with absolute certainty, and treatments for it are better than for any other noninfectious rheumatic disease.

Why can't the medical profession get it right? It should not be difficult to remember that the appropriate management of gout requires 3 different medications on 3 different schedules. Either we never learned the correct approach, have forgotten it, or don't believe it. Fang and colleagues concluded that high quality continuing medical education (CME) would be required to improve Chinese physicians' practice. But providing gout education at the CME stage may be too late. Since most gout is managed by primary care physicians, those of us in medical education need to take every opportunity to see that the principles of gout management are driven home to medical students and residents as well as rheumatology fellows.

Because the diagnosis of gout can be made with such certainty and treatments can be so effective, the management of gout should be very satisfying for the patient and gratifying for the physician. The only patients who should suffer the ravages of gout are those who are totally intolerant to all urate-lowering agents. The principles of management are clear. They are consistently outlined in textbook chapters and other medical literature and have not changed for 40 years. It should be crystal clear!

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