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Latest evidence on gout diagnosis, management and prevention: what the clinician needs to know

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Gout is characterized by painful joint inflammation, most commonly in the first metatarsophalangeal joint, resulting from precipitation of monosodium urate crystals in a joint space. Definitive diagnosis of gout requires synovial fluid or tophus aspiration to identify negatively birefringent monosodium urate crystals under polarizing microscopy, but crystal evaluation is not routinely performed in clinical practice. Hyperuricemia may not be present during acute gout attacks and therefore may not be useful for diagnosis. Differential diagnosis of acute gout includes calcium pyrophosphate dehydrate or other crystal-induced arthritides and a septic joint. If a septic joint is suspected, joint aspiration with Gram staining and culture must be performed.

The aim of this study was to review the medical literature devoted to the standards of diagnosis and treatment of patients with gout and subsequent review by students and young scientists with the basic provisions of the most effective and rational schemes of diagnosis and treatment of gouty arthritis.

Materials and methods. We were conducted selection, referencing and the following synthesis of more than 30 medical articles devoted to modern methods of diagnosis and treatment of gout.

Results and discussion. Gout is typically diagnosed using clinical criteria from the American College of Rheumatology. Diagnosis may be confirmed by identification of monosodium urate crystals in synovial fluid of the affected joint. Microscopy of joint fluid is used less often, primarily in equivocal cases. In these situations, the diagnosis is established by aspiration of a joint or tophus and identification of needle-shaped monosodium urate crystals, preferably intracellular, with bright, negative birefringence on compensated polarized light microscopy. The differential diagnosis for acute monoarticular joint swelling includes pseudogout, infection, and trauma.

Acute gout may be treated with nonsteroidal anti-inflammatory drugs, corticosteroids, or colchicine. To reduce the likelihood of recurrent flares, patients should limit their consumption of certain purine-rich foods (e.g., organ meats, shellfish) and avoid alcoholic drinks (especially beer) and beverages sweetened with high-fructose corn syrup. Consumption of vegetables and low-fat or nonfat dairy products should be encouraged. The use of loop and thiazide diuretics can increase uric acid levels, whereas the use of the angiotensin receptor blocker losartan increases urinary excretion of uric acid. Reduction of uric acid levels is key to avoiding gout flares. Allopurinol and febuxostat are first-line medications for the prevention of recurrent gout, and colchicine and/or probenecid are reserved for patients who cannot tolerate first-line agents or in whom first-line agents are ineffective. Oral corticosteroids,intravenous corticosteroids, NSAIDs, and colchicines are equally effective in treating acute flares of gout. Patients receiving urate-lowering medications should be treated concurrently with nonsteroidal anti-inflammatory drugs, colchicine, or low-dose corticosteroids to prevent flares. Treatment should continue for at least three months after uric acid levels fall below the target goal in those without tophi, and for six months in those with a history of tophi.

Conclusions. Historically, urate-lowering medication was thought to worsen acute gout flares, but recent evidence suggests that allopurinol (Zyloprim) can be started during an acute flare if it is used in conjunction with an NSAID and colchicine. Patients receiving a urate-lowering medication should be treated concurrently with an NSAID,colchicine, or low-dose corticosteroid to prevent a flare.