

PRIMER ON DIAGNOSIS AND TREATMENT OF GOUT

Gout, the most painful of all the arthritides, is common, and its incidence is increasing. It is caused by inflammation induced by monosodium urate (MSU) crystals that precipitate in joint fluid. When these microscopic crystals lose their protective protein coatings, they trigger intense macrophage and neutrophil-induced inflammation in joints, tendon sheaths, and even in subcutaneous cellulite.

DX:

Proper diagnosis is key. Care providers should realize that not all podagra is gout, and not all gout presents as podagra! Pseudogout, rheumatoid arthritis, and most commonly, osteoarthritis, can all cause podagra. Conversely, gout can occur in literally any joint, so it is incorrect to conclude that a patient with podagra definitely has gout. There are well-established diagnostic criteria, the most reliable of which is identification of MSU crystals in aspirated synovial fluid, or demonstration of MSU crystals in material exuded from a tophus. Rapid response to oral colchicine is also characteristic of gout, but less specific than crystal identification. Analysis of synovial fluid for crystal identification should always be pursued if the diagnosis has not yet been definitively established. Most practitioners do not have a polarizing microscope readily available, but joint fluid refrigerated and sent to a lab or rheumatology office within 24-48 retains visible crystals.

RX:

Successful treatment of acute attacks differs in important ways from prophylaxis of attacks. Acute gouty attacks can be treated with oral colchicine, oral NSAID's in relatively high doses, oral "burst" prednisone regimens, intraarticular injection of steroids (most rapid and most effective), or other less commonly used methods such as ACTH or IV colchicine. Allopurinol has NO PLACE in the treatment of acute gouty arthritis, and is used only for prophylaxis, or for indications listed below. In fact, due to its induction of rapid changes in serum uric acid, allopurinol can actually make the acute attack worse, and/or prolong it.

Patients who experience frequent recurrent attacks are candidates for prophylaxis. It should be emphasized that allopurinol is NOT needed in all gout patients, and good prophylaxis can be accomplished in many patients by use of daily colchicine, adjusted for renal function. Colchicine does not damage kidney function, but doses must be reduced in the face of renal insufficiency to avoid elevated blood colchicine levels and resultant toxicity to marrow or muscle. Contrary to common belief, allopurinol does not adversely affect renal function either, but it is eliminated via the kidneys, and higher blood levels of allopurinol increase the risk of side effects such as cutaneous rashes, and serious Stevens-Johnson syndrome. Regardless of renal function, the dose of allopurinol should be that dose necessary to reduce serum uric acid below 6.0 mg/dl, the approximate saturation point of MSU in body fluids at pH 7.4. Thus, the issue is balance of allopurinol dose versus side effects. Asymptomatic hyperuricemia of less than 9.0 mg/dl in females and less than 11.0 mg/dl in males, need not be treated.

Indications for use of allopurinol:

- failure of colchicine or NSAIDs prophylaxis alone to prevent recurrent attacks
- presence of tophi either in the skin or in bone
- uric acid greater than 9 in females and 11 in males
- history of uric acid nephrolithiasis
- proven urate nephropathy

Initiation of oral allopurinol lowers serum uric acid very rapidly, and likewise, sudden discontinuation of allopurinol results in rapid re-elevation of serum uric acid to pre-treatment levels. Either of these rapid changes often precipitates acute gout. Thus, initiation of allopurinol treatment must be preceded by daily oral colchicine for at least two weeks to prevent induction of an acute attack. Furthermore, most experts recommend continuation of colchicine for several weeks or months after initiation of allopurinol. In patients with tophi, it is probably wisest to continue colchicine prophylaxis until all tophi have dissolved, which often takes years. Patients should be counseled never to run out of their allopurinol, since the sudden increase of serum uric acid will precipitate attacks, and sudden restarting of the medication will cause sudden decrease. Either way, worsening an ongoing attack, or precipitation of a new acute attack can result.

About the Author

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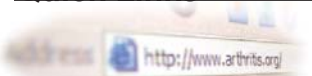


Attended Cornell University, graduated with bachelor's degree in Physics. Medical School at University of Illinois, Chicago campus 1972-1976. Medical Residency, University of Illinois, Chicago 1976-1979. Chief Medical Resident University of Illinois, Chicago 1979-1980. Clinical Rheumatology Fellow University of Illinois Chicago, 1980-1981. Research Fellow, National Institutes of Health, Bethesda Maryland, USA 1981-1987.

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Enjoy football (soccer-especially English, Spanish and Italian Leagues), attempting to play football.

Academic interests: musculo-skeletal ultrasound and its use in rheumatology, Gout, rheumatoid arthritis and biological therapies.

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