

key points

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The prevalence of gout increases with age. Up to 7% of men >65 and 3% of women >85 have gout. Risk of gout increases significantly with increasing serum uric acid levels. Alcohol consumption and purine-rich foods such as red meat and seafood increase the risk of incident gout significantly. Loop and thiazide diuretics are also associated with increased risk. Gout is frequently associated with the metabolic syndrome. Dehydration, increasing creatinine levels, and surgery are also known to precipitate flares.

Acute gout manifests as severe joint pain, of rapid onset, reaching maximal intensity within a few hours. Gout has a predilection for lower extremity joints. It often starts at the first metatarsophalangeal joint, a condition termed podagra. Other common sites of gouty flares include: tarsal and subtalar joints; ankle; knee; wrist; small joints of the hands; Achilles tendon; and olecranon bursae. The joint affected is usually hot, red, swollen and very painful. This is commonly associated with skin erythema.

Identification of MSU crystals in the synovial fluid of an inflamed joint or from tophi allows a definite diagnosis of gout to be made. Hyperuricaemia does not confirm or exclude gout as most people with hyperuricaemia are asymptomatic, while serum uric acid levels tend to decrease during acute attacks.

Short-acting NSAIDs should be used at maximal dose as first drug of choice if not contraindicated. In patients at risk of GI complications, co-prescription of a proton pump inhibitor or the use of COX-2 selective agents should be considered. Colchicine can be particularly useful in patients with heart failure in whom NSAIDs are contraindicated but should be avoided in patients with severe renal impairment. Joint aspiration and injection of intra-articular steroids is one of the most effective ways of treating acute monoarthritic gout.

Uric acid lowering therapy is initiated if a patient suffers two or more attacks in one year. Many rheumatologists will start this therapy in hyperuricaemic patients whose first attack is very severe or in polyarticular gout. This treatment should also be initiated in patients with chronic gout arthropathy, tophi, radiological lesions, and uric acid renal calculi. It should be delayed for one to two weeks after an acute gout attack has settled and co-administration of low-dose colchicine or NSAIDs must be initiated to reduce the risk of acute gouty flares that may be triggered by the reduction of serum uric acid. Allopurinol, a competitive inhibitor of xanthine oxidase, is the main drug available to reduce serum uric acid.