Gout

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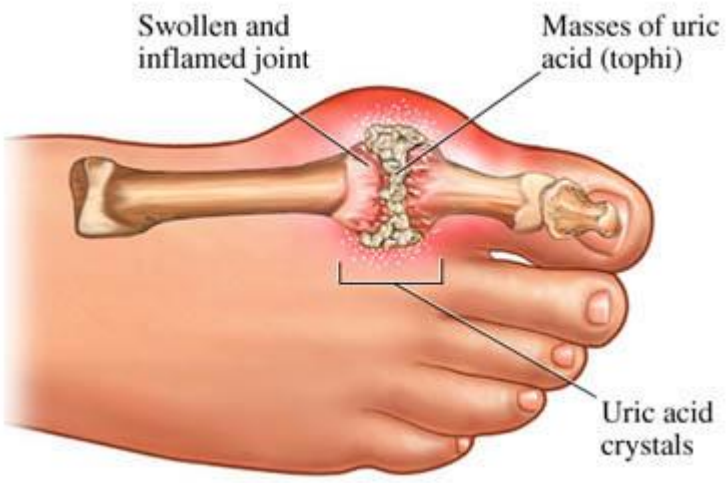
What is gout?

The term Gout is derived from the Latin Gutta, which means a drop. In the 13th century, it was thought that gout resulted from a drop of evil humor affecting a vulnerable joint.



It is a disease in which tissue deposition of monosodium urate (MSU) crystals occurs as a result of hyperuricemia (MSU supersaturation of extracellular fluids), resulting in one or more of the following manifestions: gouty arthritis (pain and swelling in the joints), tophi (aggregated deposits of MSU occurring in the joints and soft tissue (muscles), gouty nephropathy (impairment of kidney function due to gout), uric acid nephrolithiasis (uric acid deposition in the kidney).

Hyperuricemia (increased levels of uric acid in the blood) is defined as a serum uric acid concentration above what levels in males and females?

Seum uric acid concentrations are both age and sex dependent. Concentrations rise in association with the onset of puberty in males and menopause in females. Gout is rare in males under age 30 and premenopausal females. The peak age of onset of gout in males is 40-50 years and in females is after 60 years. Hyperuricemia is defines as a serum uric acid concentration >7 mg/dl in males and >6mg/dl in females.

Although only 15% of all patients with hyperuricemia develop gout, the risk increases to 30%-50% if the serum uric acid concentration is >10mg/dl.

What is uric acid?

Uric acid is the end product of the degradation of purines. Humans lack the enzyme uricase, which oxidizes uric acid to the highly soluble compound allantoin. The lack of this enzyme subjects humans to the potential risk of tissue deposition of uric acid crystals. Although humans possess the uricase gene, it is inactive. It is postulated that humans have acquired the propensity to become hyperuricemic because uric acid may have powerful antioxidant and free radical scavenger properties.

What pathogenic processes are responsible for the development of hyperuricemia (increased levels of uric acid in the blood)?

Overproduction of uric acid, underexcretion of uric acid and a combination of both processes. Most patients with hyperuricemia and gout (90%) are underexcreters of uric acid.

How do you determine if a patient with gout is an overproducer or underexcreter of uric acid?

A 24 hour urine collection is obtained for the determination of uric acid and creatinine excretion (to ensure an adequate 24 hour excretion). On a regular purine diet, a urate value >800mg/24hrs suggests overproduction of uric acid. A 24 hour urate value <800mg suggest under excretion.

Why does excessive alcohol consumption often lead to hyperuricemia and gout?

Alcohol consumption is associated with the production of lactic acid, which reduces the renal excretion of urate. In addition, it increases the synthesis of urate by accelerating the degradation of ATP. Beer contains a substantial amount of the purine gunaosine.

How do I know whether I am having a sudden attack of gout?

Early episodes of acute (sudden) gouty arthritis typically involve only one joint(85%) and begin suddenly, often during the night or early morning. The affected joint becomes exquisitely painful, warm,red and swollen. A low grade fever may be present. The redness and swelling around the joint may progress to resemble a noninfectious process termed gouty cellulitis. Acute gout may occur in sites other than joints like bursae (fluid pockets around the joints) and tendon (a structure that attaches muscle to a bone). Early attacks often spontaneously resolve over 3-10 days. Peeling of the skin overlying the affected joint may occur with the resolution of the inflammation. Subsequent attacks of gout can occur more frequently, can involve more number of joints, and persist longer.

Which joints are most commonly involved in gout?

The joints of the lower limbs (legs) are typically involved more often than those of upper limb joints (hands). The first metatarsophalangeal (MTP) joint of the great toe is involved in >50% of initial attacks and over time is affected in >90% of patients. Acute gout of the first MTP is called as podagra. In order of frequency of involvement after the MTP joints are the ankles, heels, knees, wrists, fingers and elbows.

Spinal involvement in acute attacks of gout is rare. Gout and tophi have a predilection for cooler, peripheral joints, where the solubility of MSU crystals may be decreased as a result of cooler temperature.

What are the events that may trigger an acute attack of gout?

Alcohol ingestion, excess intake of purines in the food, bleeding, acute medical illness including infection, exercise, trauma, drugs, radiation therapy. Surgery (typically during postoperative days 3-5).

Can gout be managed by diet alone?

Unfortunately, it is often difficult to manage gout by diet alone because the purine content of the diet typically contributes only 1mg/dl to the serum uric acid concentration. Patients with gout should avoid red meat (mutton, beef, and pork) and organ meat (liver, kidney). Also, they should avoid seafood (sardines, shellfish, and anchovies), vegetables and legumes (cauliflower, spinach, beans, peas and mushroom). Beer should be avoided and red wine is recommended for patients with gout.

Since strict diet restriction is not practical in day to day life, I usually don't recommend strict diet restriction for my patients. I don't mind even if my patients have some beer or red meat occasionally.

How is the diagnosis of gout established?

Fresh synovial fluid from the affected joints must be evaluated for the presence of MSU crystals. The synovial fluid is inflammatory (typically 20,000-100,000 cells/cumm) with a predominance of neutrophils (a group of white cells in the blood and other body fluids). Seum uric acid levels will be elevated at some time in almost all patients with gout, but the levels can be normal at the time of an acute gouty attack in as many as 30% of patients.

How long it is from the initial attack of gout until the appearance of tophi (aggregated deposits of MSU)?

In untreated patients, tophi develop on average 10 years after the initial attack of gout. Tophi can ulcerate through the skin and extrude a white, chalky material consisting of MSU crystals.

How do women with gout differ from male patients with regards to disease onset and clinical features?

Female patients develop gout at an older age (typically after menopause). At the onset more than one joint is involved in the acute attack. Tophi are particularly common in previously damaged joints.

When should treatment of asymptomatic hyperuricemia considered?

Asymptomatic hyperuricemia should only be treated in situations where there may be an acute over production of uric acid (acute tumor lysis syndrome, a complication which occurs as a result of treatment of certain tumors) , or where severe hyperuricemia exists (serum uric acid >12mg/dl or 24-hr urinary uric acid > 1100mg). The prevalence of uric acid nephrolithiasis is 50% in patients with severe hyperuricemia.

How is an attack of acute gout treated?

NSAIDs (pain killers) and colchicines are effective in the treatment of acute gout. In patients with contraindications to these medications or in patients in whom these drugs don't work, corticosteroids may be used to suppress the inflammatory response. Drugs that alter serum uric levels (allopurinal, probenecid) should never be started until after complete resolution of the acute gouty attack. Also, these drugs should not be stopped if an acute gouty attack occurs while the patient is on these medications.

When one should undergo chronic treatment for gout?

Lifelong therapy with drugs that lower uric acid levels in the blood is indicated in following situations: >2 or 3 acute attacks of gout within 1-2 years, presence of renal stones, tophaceous gout, chronic gouty arthritis with destruction of the bones, asymptomatic hyperuricemia with serum uric acid >12mg/dl or 24 urinary excretion> 1100mg to decrease the risk of uric acid stones in the kidney.

I thank Robert. W. Janson, MD for contributing valuable information required for this article.

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