

# Gout: A detailed discussion

## Gout

What is gout? Gout is a type of arthritis (inflammation of the joints) that mostly affects men age 40 and older. It is nearly always associated with an abnormally high concentration of uric acid in the blood. Uric acid is produced in the liver and enters the bloodstream. Under certain circumstances, the body produces too much uric acid or excretes too little. As uric acid concentrations increase, needlelike crystals of a salt called monosodium urate (MSU) form. In time, MSU crystals accumulate and cause inflammation and pain, symptoms typical of gout.

People with chronically high blood levels of urate (commonly referred to as uric acid) may develop a condition known as gout. These high blood levels of uric acid may cause crystals to deposit in the body's tissues, especially in joints. Deposition of the urate crystals and the attempts by white blood cells to consume the crystals appear to be a key factor in development of an acute gouty attack.

An attack of gout manifests itself by a suddenly painful and inflamed joint. It most commonly affects a single joint at a time and most frequently will involve the large toe on initial presentation. Urate crystals can be deposited in the kidney or urinary tract causing kidney stones and, occasionally, impairing kidney function.

"Tophaceous" gout, which can develop in a patient with chronic gout, occurs when large aggregates of urate crystals collect in joints, bones and cartilage. The urate causes a nodule called a "tophus" (plural "tophi") to form. The presence of a tophus can cause erosion of bone it is near. They often occur near the knuckles or small joints of the fingers and are usually not painful or tender. Occasionally, they can become inflamed and cause symptoms like those of an acute gouty attack.

Patients with gout are at increased risk for developing kidney stones. Uric acid crystals can collect in the urinary tract and form a stone. The most important risk factor for uric acid crystallization and stone formation is a low urine pH (below 5.5), rather than an increased urinary uric acid excretion. Causes of low urine pH include chronic diarrhea, severe dehydration and renal tubular disorders. A small collection of uric acid crystals can be a nidus for the formation of larger calcium containing urinary stones and ultimately affect kidney function.

Hyperuricemia (elevated blood uric acid levels) can be caused by genetic predisposition coupled with the wrong diet, or by either a) an increased production of uric acid such as seen in leukemia, hemolytic anemia, psoriasis, excessive exercise or b) decreased excretion of uric acid as seen in chronic renal disease, lead induced nephropathy (kidney damage) or diabetic ketoacidosis.

Nutrition plays a significant role in contributing to a gouty attack. Certain foods can cause an elevation of uric acid levels thereby provoking a gouty attack. They include excessive amounts of alcohol, coffee, soft drinks, anchovies, asparagus, legumes, mushrooms and meats, especially organ meats, and shellfish.

Some other instigators of gout are surgery, injury, use of antibiotics, vitamin B5 deficiency and chemotherapy. Any cause of potassium loss, such as surgery, fasting and diuretic use, may trigger gout as well.

Rapid weight loss or fasting can cause excess lactic acid buildup, which hinders uric acid excretion by the kidneys thereby triggering a gout attack. Dieting also may cause a loss of potassium, which can increase urate levels in the blood. Some

dieters also use diuretics which can deplete the body of potassium and other minerals, triggering a gout attack.

An elevated uric acid level as diagnosed by blood test only indicates that a patient is at risk for gout but does not necessarily have it. It can take 20-30 years for a patient with elevated uric acid levels to actually develop gout. The typical age of onset is 45.

If one has an acute attack of gout, the drug of choice is indomethacin, but other NSAIDs can be tried. Colchicine can be used in the event of an acute attack but is often times poorly tolerated due to diarrhea, abdominal pain and nausea. One treatment that has an immediate effect is intra-articular (into the joint) steroid injection. The physician, however, has to be certain that there is no element of infection in the joint as the steroid can exacerbate it.

Certain medications can decrease the blood level of uric acid. Probenecid and sulfinpyrazone are two such medications, called uricosuric agents, and cause the uric acid to be disposed of in the urine. These medications are not given to patients with impaired kidney function, because in order for the uric acid to pass in the urine, it must first pass through the kidneys. The majority of patients with gout who excrete less than 800 mg [4.76 mmol] of uric acid per day on a standard diet are potential candidates for uricosuric drug therapy

Another drug, allopurinol, prevents the formation of uric acid. It works by inhibiting an enzyme called xanthine oxidase and therefore decreasing the synthesis of uric acid. It is prescribed for the treatment of chronic gout and is not to be started in the event of acute gout, but continued if already on it. It is especially indicated for patients with overproduction of urate, kidney stones, tophaceous deposits or if the patient is older than 60.

For treatment with either class of medications to be effective and lasting, patients generally must take them regularly and on a long-term basis. Among adverse reactions occurring in 3 to 5 percent of patients are rash, leukopenia or thrombocytopenia, diarrhea, and drug fever. Allopurinol can cause potentiation of the immunosuppressive effects of the medication 6-mercaptopurine and **azathioprine**, which are partially metabolized by xanthine oxidase.

**Losartan**, a drug used to treat high blood pressure, can actually reduce blood uric acid levels. It may provide a safe way of diminishing the diuretic-induced increase in uric acid levels and simultaneously potentiating the antihypertensive action of the diuretic.

**Fenofibrate**, a fibric acid derivative, used for treatment of high cholesterol, also has uricosuric activity.

#### **A nutritional approach to gout:**

There is much one can do nutritionally to prevent gout. First, one should limit intake of foods rich in purines, which are the information molecules in genes and are used in the process of converting genes to proteins. They act as messengers in cellular signaling processes, such as nerve conduction and muscle contraction. After metabolism, these purines break down into uric acid, which is elevated in gout.

Purine content is greater in red meats, so one should try to limit intake of organ meats and steaks, chops and corned beef. Poultry has a significantly lower intake and is a better source of protein for gout patients for this reason. Shellfish,

tofu, beans, peas and alcohol, especially beer and wine, also are high in purines.

Meats and dairy products also contain a fatty acid known as arachidonic acid, which further contributes to the inflammatory process. It is converted into prostaglandins and leukotrienes, inflammatory proteins that are high in different types of arthritis conditions. Anti-inflammatory medications aim to decrease these markers, thereby mitigating symptoms. Based on this theory, a diet low in saturated fats, and therefore low in arachidonic acid, may be beneficial. **Fish oils** may work to decrease these inflammatory markers and thus can be very beneficial in gout.

Cherries can lower plasma uric acid levels. They contain flavonoid compounds that may lower uric acid and reduce inflammation. Flavonoids called anthocyanins, often found in purple and blue colored berries, help to shut down the enzymes that cause tissue inflammation in the first place. Anthocyanins can therefore prevent and treat many kinds of pain. A study done at the University of California at Davis by Jacob et. al entitled "Consumption of cherries lowers plasma urate in healthy women" proved just that and also showed that there was a decrease in plasma urate after cherry consumption. **Bilberry**, hawthorn and blueberries also are high in anthocyanosides.

**Quercetin**, another flavonoid, can be added as a supplement to decrease uric acid levels. **Quercetin** can be taken with **bromelain** to improve its absorption. Bromelain, by itself, can act as an anti-inflammatory as well as contribute to the improved absorption of quercetin.

**Pantothenic acid**, a type of B vitamin (B5), is needed to help with the excretion process of uric acid. Stress often depletes the body of this vitamin which may explain why gout flares during times of stress. Therefore, incorporating pantothenic acid into the treatment plan may be crucial.

It is important to stay well-hydrated in order to prevent attacks of gout. This will prevent the uric acid from becoming too concentrated and inhibit uric acid crystallization.

Uric acid crystallizes and forms stones in an acidic environment. One way to prevent this is by alkalinizing the urine or making it more basic. Urine alkalinization can be achieved with administration of potassium citrate or sodium bicarbonate as directed by your physician. Lemon juice stimulates the formation of calcium carbonate, which neutralizes acids like uric acid. Therefore, drinking freshly squeezed lemon in a glass of water after meals can prevent a gouty attack. Baking soda also can help alkalinize the urine. **Black cohosh** also has been shown to moderate blood acidity, thereby making the urine more basic.

Cayenne pepper, which can affect the substance P, expressed in pain syndromes, can be boiled in vinegar and water and applied onto the painful joint. It can also be mixed with wintergreen oil to make a paste and subsequently applied to the affected area. Alternatively, capsaicin cream is available at the drugstore.