

Gout

A Crystalline Deposit Disorder

Gout is a painful condition characterized by the deposition of uric acid crystals in the joints, which causes episodes of joint inflammation.

Uric acid is created as a byproduct of purine metabolism. If too much uric acid is created through increased cell destruction, or if the body's system for clearing uric acid is compromised (usually by defects in the kidneys' ability to clear uric acid), gout may result.

Gout causes acute attacks of inflamed, painful joints that subside gradually, though it can progress to a chronic condition if untreated. It often affects a joint in the first toe, but may occur in the ankles and knees as well. The first episode of acute gout often occurs at night, with the joints swelling and becoming painful. They may appear warm, red, and excruciatingly tender. The attack often subsides within 3 to 10 days, and no symptoms will be present until the next attack occurs.

Monosodium urate is the type of crystal that causes gout. Other kinds of crystals are associated with "pseudo gout" (so named because the symptoms can be similar to gout). These crystals include calcium pyrophosphate dihydrate, calcium hydroxyapatite, and calcium oxalate.

Gout affects men more often than women. It is the most common cause of inflammatory arthritis among men younger than 40, and is often encountered among middle-aged to elderly men and postmenopausal women (Roubenoff R 1990; Kasper DL et al 2005). Gout may be more common in men because of their higher levels of uric acid (Lawrence RC et al 1989; Meiner 2001). Less than 15 percent of gout cases in women occur before menopause (Puig JG et al 1991; Lally EV et al 1986). Overall, hyperuricemia, or elevated uric acid, affects between 2 percent and 13.2 percent of the general population, while the prevalence of gout is between 1.3 percent and 3.7 percent (Kasper DL et al 2005).

GOUT: CAUSES AND RISK FACTORS

Gout may be primary (occurring alone) or secondary (caused by other conditions). By definition, in primary gout, the cause of excess uric acid is usually not known; in secondary gout, the cause is usually known.

In most cases, primary gout is observed in patients as a result of either their inability to excrete uric acid in the kidney or their increased production of uric acid, or a combination of both. If high loads of dietary purines overwhelm the kidneys' ability to excrete uric acid, blood uric acid levels can rise. Some investigators believe this excretion flaw is genetic (Wortmann RL 2002; Wang WH 2004a).

- Secondary gout is caused by certain medications or health problems. These medications include diuretics, aspirin, niacin, levodopa, and cyclosporin. Medical problems related to gout include diabetes, alcoholism, obesity, anemia, leukemia, lung cancer, and heart failure (Saag KG et al 2005; Kasper DL et al 2005; Emmerson 1996; Fam AG 2002; Snaith ML 2001; Choi HK et al 2004b; Choi HK et al 2005a).

Elevated risk for gout attacks is related to lifestyle and dietary factors. Over the past 20 years, the rate of gout has increased in developing countries such as Thailand and other Southeast Asian nations, along with greater incidences of diseases such as obesity, diabetes, hypertension, insulin resistance, and cardiovascular diseases (Fam AG 2002).

Foods Associated with Gout

Among people with gout, attacks may be caused by diets with an abundance of foods containing purines. Purine-rich foods include:

- Beer
- Anchovies, sardines, herring
- Asparagus, mushrooms, spinach, cauliflower
- Meat broth and consommé, meat gravy
- Organ meats (kidney, liver)
- Yeast.

Gout is also closely associated with excessive alcohol intake (Fam AG 2002; Johnson RJ et al 2004). It is estimated that at least 50 percent of gout sufferers drink excessively (Sharpe CR 1984). Moderate wine consumption (two glasses a day) does not appear to increase the risk of gout, but two or more beers per day confer a greater risk of gout (Choi HK et al 2004a). The greater hyperuricemic effects of beer (compared to other alcoholic drinks) are attributed to its high purine content (Fam AG 2005; Choi HK et al. 2004a).

DIAGNOSING GOUT

The diagnosis of gout begins with its characteristic symptoms, including inflamed, painful joints in the extremities. The skin may be red and shiny above the affected area. The big toe is commonly affected. If a physician suspects gout, a number of tests should be ordered.

Blood tests may be used to measure levels of uric acid, but are not always a dependable way to diagnose gout. Serum uric acid concentrations above 7 mg/100 ml in males and above 6 mg/100 ml in females are considered hyperuricemia (Scott JT 1983; DiPiro JT et al 1999; Snaith ML 1995; McGill NW 1997). However, uric acid may not be elevated during gout attacks, and elevated uric acid levels without symptoms are of questionable significance. Nevertheless, serum uric acid tests are almost always performed at some point during the diagnostic work-up, and the tests are frequently used to monitor treatment.

The most accurate test to diagnose gout is examination of the synovial (joint) fluid for crystals. This is accomplished by withdrawing a fluid sample with a needle (a technique known as needle aspiration) and examining the crystals under a microscope (Kasper DL et al 2005). X-rays can also be taken to view cystic changes and lesions around the joints. However, X-rays of gout-affected joints often show signs that are similar to those seen in other joint disorders, and thus may be of limited value in the diagnosis (Kasper DL et al 2005). Needle aspiration of the joint is also the best and most immediate form of treatment. Many patients whose gout cannot be controlled with diet and medication seek out needle aspiration to relieve the pain.

STAGES OF GOUT

The development and progression of gout often follows a typical pattern:

Asymptomatic Hyperuricemia: Uric acid levels are elevated, but no symptoms are present.

Acute or Recurrent Gout: An acute gout episode occurs in the joints of extremities in response to inflammation caused by the deposition of crystals in the joints or other soft tissues (Pascual E 1994; Weinberger A 1995). These inflammatory symptoms include sudden onset of severe pain, limited range of motion, and redness, swelling, and warmth in the affected joints (Perkins et al 1999).

Intercritical Gout: After recovery from an acute flare-up, the patients enter an asymptomatic phase (Pascual E et al 1999), which is known as intercritical gout. Some patients never have a second attack, but most will experience a second attack within six months to two years.

Chronic Tophaceous Gout: Tophi-chalky deposits of uric acid, located around tissues of joints affected by gout (Fam AG 1998), are present after 10 to 20 years of inadequately treated chronic gout (Agarwal AK 1993). The most common sites of tophaceous gout are the elbow and joints of the hand and feet (Meiner SE 2001).

CONVENTIONAL TREATMENT

Medical treatment of gout begins with drugs that reduce inflammation and pain, including nonsteroidal anti-inflammatory drugs (NSAIDs), COX-2 inhibitors, and corticosteroids.

In the United States, NSAIDs are considered the first choice in treating acute gout inflammation (Wortmann RL 2005). Indomethacin, naproxen, and sulindac have been approved by the U.S. Food and Drug Administration for the treatment of gout. Studies have shown that these drugs are effective in relieving pain and reducing inflammation in patients with acute gout, particularly if taken soon after the onset of the attack (Arnold MH et al 1988).

However, because of these drugs' gastrointestinal side effects, not all patients can tolerate NSAIDs. COX-2 inhibitors may be somewhat better tolerated, but drugs in this class have recently been linked to increased risk of heart attack and stroke, resulting in the removal of several COX-2 inhibitors from the market. Oral glucocorticoids, such as prednisone, are also effective, and injections of glucocorticoids directly into the affected joints may be given to elderly patients.

After the initial attack has subsided, patients may be placed on longer-term uric acid-reduction therapy. The most common drugs used to lower uric acid levels are allopurinol and probenecid. These drugs are considered safe over the long term, though their side effects include headache, nausea, skin rash, liver or kidney damage, and inflammation of the blood vessels. Allopurinol, especially, may be toxic in patients with kidney failure who are also on diuretics, as well as in patients who are allergic to penicillin and other antibiotics (Kasper DL et al 2005). Low doses of NSAIDs and/or colchicine may be used for several months after introducing uric acid-reduction treatment to prevent another attack.

NUTRIENTS AND SUPPLEMENTAL THERAPY

Gout is influenced by dietary and lifestyle factors, including obesity, alcohol abuse, dyslipidemia, and insulin resistance syndrome (Tikly M et al 1998; Emmerson BT 1998; Vuorinen-Markkola H et al 1994). Thus, patients with gout should:

- Avoid drinking alcohol
- Avoid foods high in purines (see sidebar)
- Lose weight (if overweight or obese)
- Increase exercise.

In addition to lifestyle changes, a number of supplements have been studied for their ability to inhibit the formation of uric acid. These include:

Cherries and cherry extract

Cherries are rich in antioxidants such as anthocyanins, catechins, chlorogenic acid, flavonol glycosides, and melatonin. Studies have shown that cherry extract can reduce uric acid concentration among women, though the cause of this reduction is unknown (Jacob RA et al 2003). Cherries and their extracts traditionally have been used to treat gout (Fam AG 2005), and one small case series documented decreased duration and severity of gout attacks in three people on cherry-supplemented diets (Blau LW 1950).

Chinese herbs

Certain Chinese medicinal plants were tested for xanthine oxidase inhibitory activity (preventing the conversion of xanthine, a purine metabolite, to uric acid). The most active was the methanol extract of Chinese cinnamon (*Cinnamomum cassia*), followed by *Chrysanthemum indicum* (Asteraceae) and *Lycopus europaeus* (Labiatae). Among water extracts, the strongest inhibition was observed with *Polygonum cuspidatum* (Polygonaceae), (Kong LD et al 2000). These herbs have been used in China to suppress gout (Kong LD et al 2000).

Vitamin C

In a recent study, the effect of 500 mg of vitamin C daily on serum uric acid levels was compared to placebo in 184 healthy adults. The vitamin C increased the estimated glomerular filtration rate, a measure of kidney function. After two months, the test subjects had reduced serum uric acid compared to controls, suggesting that vitamin C might be beneficial in preventing and managing gout and other urate-related diseases (Huang HY et al 2005).

Grape seed procyanidins

Grape seed procyanidins were found to have uric acid-lowering effects in rats with hyperuricemia. The procyanidin-treated animals exhibited normal growth compared to animals treated with allopurinol, which exhibited some retarded growth (Wang Y et al 2004b).

Because of gout's close association with inflammation, gout patients should also consider Life Extension's anti-inflammatory recommendations. For more information on nutrients and supplements that help combat inflammation, please see the Inflammation protocol.

LIFE EXTENSION FOUNDATION RECOMMENDATIONS

Gout is a metabolic disorder that is often associated with controllable factors such as diet and weight. Researchers have shown that dietary modification is effective in reducing gout (Pascual E et al. 2004). Based on scientific studies, Life Extension suggests that gout patients:

- Avoid high-purine foods (Emmerson BT 1996).
- Avoid alcoholic beverages (Emmerson BT 1996).
- Lose excess weight (Choi HK et al 2005b).

- Increase consumption of foods from plant sources, especially fruits (such as cherries) and vegetables that reduce the risk of gout development (Lyu LC et al 2003). Because dietary fiber is beneficial for intestinal motility and has a potential role in binding uric acid in the gut for excretion, increasing one's fiber intake has been suggested to lower the risk of gout (Lyu LC et al. 2003). Soluble fiber supplements are also available.

Drink plenty of fluids and reduce salt intake (Benecke M 2003).

- **Vitamin C**—1000 milligrams (mg) daily.
- **Cherry extract**—follow label directions.
- **Grape seed extract**—100 mg daily.
- **Avoid the use of niacin** (Crouse JR 1996), vitamin A in high doses (Mawson AR et al 1991), and low-dose aspirin (Caspi D et al 2000) unless approved by a qualified physician.

GOUT SAFETY CAVEATS

An aggressive program of dietary supplementation should not be launched without the supervision of a qualified physician. Several of the nutrients suggested in this protocol may have adverse effects. These include:

Vitamin C

- Do not take vitamin C if you have a history of kidney stones or of kidney insufficiency (defined as having a serum creatine level greater than 2 milligrams per deciliter and/or a creatinine clearance less than 30 milliliters per minute).
- Consult your doctor before taking large amounts of vitamin C if you have hemochromatosis, thalassemia, sideroblastic anemia, sickle cell anemia, or erythrocyte glucose-6-phosphate dehydrogenase (G6PD) deficiency. You can experience iron overload if you have one of these conditions and use large amounts of vitamin C.

For more information see the Safety Appendix

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