Gout

Gout has the unique distinction of being one of the most frequently recorded medical illnesses throughout history. The term *gout* refers the disease that is caused by an overload of uric acid in the body, resulting in painful arthritic attacks and deposits of lumps of uric acid crystals in body tissues. Gout is one of the most common forms of arthritis.

A gout attack usually happens at night and within 12 or so hours there may be severe pain and swelling in the joint. Gout usually affects only one or two joints at the same time, typically in the feet and ankles. It often occurs in the big toe, and people can notice it at night when their bedding rubs on their toe and causes extreme pain. Gout happens when crystals derived from uric acid accumulate in the joints. These crystals cause inflammation.

Without treatment, an early attack of gout usually goes away in a week. It may be months or years until the next attack. As time goes on, more joints can become affected and the disease may cause disabilities or eventually cripple the patient. That is why it is important to get treated right away if you think you may have gout. Your doctor will order a uric acid test and may have to take fluid from the joint through a needle to look for the microscopic crystals.

Gout is nine times more common in men than in women. It predominantly attacks males after puberty, with a peak age of 75. In women, gout attacks usually occur after menopause.

A gout attack can be one of the most painful episodes that a person has to endure. They can come on unexpectedly, usually at night, and can leave the effected joint, red, swollen and the sufferer in absolute agony. If gout runs in your family or you have had a gout attack in the past, you are at risk of experiencing an attack or having a reoccurrence. As anyone who has had the misfortune of suffering

through a gout attack will tell you, the pain is extreme and can be pretty debilitating, often leaving you housebound and off work.

The easiest approach for relief is to look at and adjust your diet to a low uric acid diet. As the old saying goes; prevention is better than a cure – so making changes as soon as you realize you are at risk is very important.

The problem is that gout is caused by a build up of uric acid in the blood stream. When a specific saturation point is reached excess uric acid is deposited in the spaces and connective tissue of the joints. These deposits crystallize and the sharp, jagged crystals are the source of the swollen, painful joints.

Gout normally strikes in the big toe or one of the other foot or ankle joints, although it can affect any of the joints in the body. The reason for commonly affecting the feet is because they are the furthest points away from the heart and uric acid crystallizes in cooler temperatures.

When crystals form in the joints it causes recurring attacks of joint inflammation (arthritis). Chronic gout can also lead to deposits of hard lumps of uric acid in and around the joints and may cause joint destruction, decreased kidney function, and kidney stones.

It is often related to an inherited abnormality in the body's ability to process uric acid. Uric acid is a breakdown product of purines that are part of many foods we eat. An abnormality in handling uric acid can cause attacks of painful arthritis (gout attack), kidney stones, and blockage of the kidney-filtering tubules with uric acid crystals, leading to kidney failure.

On the other hand, some people may only develop elevated blood uric acid levels (hyperuricemia) without having arthritis or kidney problems.

Gouty arthritis is typically an extremely painful attack with a rapid onset of joint inflammation. The joint inflammation is precipitated by deposits of uric acid crystals in the joint fluid (synovial fluid) and joint lining (synovial lining). Intense joint inflammation occurs as white blood cells engulf the uric acid crystals and chemical messengers of inflammation are released, causing pain, heat, and redness of the joint tissues.

While an elevated blood level of uric acid may indicate an increased risk of gout, the relationship between hyperuricemia and gout is unclear. Many patients with hyperuricemia do not develop gout, while some patients with repeated gout attacks have normal or low blood uric acid levels. In fact, the blood level of uric acid often lowers during an acute attack of gout.

Risk factors

In addition to an inherited abnormality in handling uric acid, other risk factors for developing gout include obesity, excessive weight gain (especially in youth), and moderate to heavy alcohol intake, high blood pressure, and abnormal kidney function.

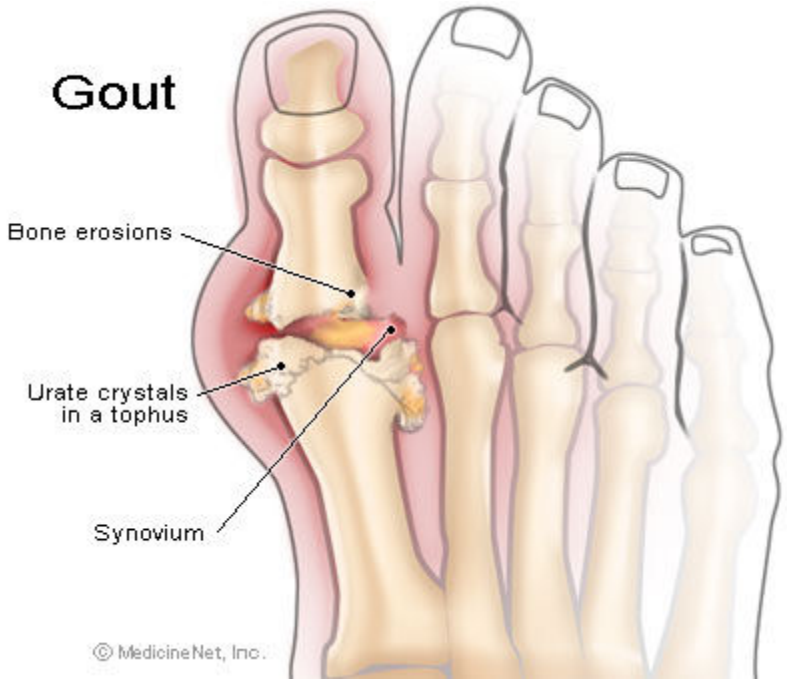
Certain drugs, such as thiazide diuretics (hydrochlorothiazide, low-dose aspirin, niacin, cyclosporine, tuberculosis medications (pyrazinamide and ethambutol), and others can also cause elevated uric acid levels in the blood and lead to gout. Furthermore, certain diseases lead to excessive production of uric acid in the body. Examples of these diseases include leukemias, lymphomas, and hemoglobin disorders. Interestingly, a recent study demonstrated an increased prevalence of abnormally low thyroid hormone levels (hypothyroidism) in patients with gout.

In patients at risk of developing gout, certain conditions can precipitate acute attacks of gout. These conditions include dehydration, injury to the joint, fever, excessive eating, heavy alcohol intake, and recent surgery. Gout attacks triggered by recent surgery are probably related to changes in the body-fluid balance as patients

temporarily discontinue normal oral fluid intake in preparation for and after their operation.

Symptoms and signs

The small joint at the base of the big toe is the most common site of an acute gout attack of arthritis. An acute attack of gouty arthritis at the base of the big toe is medically referred to as podagra. Other joints that are commonly affected include the ankles, knees, wrists, fingers, and elbows. Acute gout attacks are characterized by a rapid onset of pain in the affected joint followed by warmth, swelling, reddish discoloration, and marked tenderness. Tenderness can be intense so that even a blanket touching the skin over the affected joint can be unbearable. Patients can develop fever with the acute gout attacks. These painful attacks usually subside in hours to days, with or without medication. In rare instances, an attack can last for weeks. Most patients with gout will experience repeated attacks of arthritis over the years.



Uric acid crystals can deposit in tiny fluid-filled sacs (bursae) around the joints. These urate crystals can incite inflammation in the bursae,

leading to pain and swelling around the joints (a condition called bursitis). In rare instances, gout leads to a more chronic type of joint inflammation that mimics rheumatoid arthritis.

In chronic (tophaceous) gout, nodular masses of uric acid crystals (tophi) deposit in different soft-tissue areas of the body. Even though they are most commonly found as hard nodules around the fingers, at the tips of the elbows, in the ears, and around the big toe, tophi nodules can appear anywhere in the body. They have been reported in unexpected areas such as in the vocal cords or (rarely) even around the spinal cord!

Diagnosis

Gout is suspected when a patient reports a history of attacks of painful arthritis, particularly at the base of the toes. Ankles and knees are the next most commonly involved joints in gout. Gout usually attacks one joint at a time, while other arthritis conditions, such as systemic lupus and rheumatoid arthritis, usually attack multiple joints simultaneously.

The most reliable test for gout is finding uric acid crystals in a sample of the joint fluid obtained by joint aspiration (arthrocentesis). Arthrocentesis is a common office procedure performed under local anesthesia. Using sterile technique, fluid is withdrawn (aspirated) from the inflamed joint using a syringe and needle. The joint fluid is then analyzed for uric acid crystals and for infection. Shiny, needle-like uric acid crystals are best viewed with a special polarizing microscope. The diagnosis of gout can also be made by finding these urate crystals from material aspirated from tophi nodules and bursitis fluid.

Sometimes patients with a classic history and symptoms of gout can be successfully treated and presumed to have gout without undergoing arthrocentesis. However, establishing a firm diagnosis is still preferable since other conditions can mimic gout. These include

another crystal-induced arthritis called pseudogout, psoriatic arthritis, rheumatoid arthritis, and even infection in the joint.

X-rays can sometimes be helpful and may show tophi-crystal deposits and bone damage as a result of repeated inflammations. X-rays can also be helpful for monitoring the effects of chronic gout on the joints.

Treatment

There are two key concepts essential to treating gout. First, it is critical to stop the acute inflammation of joints affected by gouty arthritis. Second, it is important to address the long-term management of the disease in order to prevent future gouty arthritis attacks and shrink gouty tophi crystal deposits.

The treatment of an acute attack of gouty arthritis involves measures and medications that reduce inflammation. Preventing future acute gout attacks is equally as important as treating the acute arthritis. Prevention of acute gout involves maintaining adequate fluid intake, weight reduction, dietary changes, reduction in alcohol consumption, and medications to lower the uric acid level in the blood (reduce hyperuricemia).

Maintaining adequate fluid intake helps prevent acute gout attacks. Adequate fluid intake also decreases the risk of kidney stone formation in patients with gout. Alcohol is known to have diuretic effects that can contribute to dehydration and precipitate acute gout attacks. Alcohol can also affect uric acid metabolism to cause hyperuricemia. Therefore, alcohol has two major effects that worsen gout by impeding (slowing down) the excretion of uric acid from the kidneys as well as by causing dehydration, both of which contribute to the precipitation of uric acid crystals in the joints.

Dietary changes can help reduce uric acid levels in the blood. Since purine chemicals are converted by the body into uric acid, purine-rich foods are avoided. Examples of foods rich in purines include

shellfish and organ meats such as liver, brains, kidneys, and sweetbreads. Researchers have reported, in general, that meat or seafood consumption increases the risk of gout attacks, while dairy food consumption seemed to reduce the risk. Protein intake or purine-rich vegetable consumption was not associated with an increased risk of gout. Total alcohol intake was strongly associated with an increased risk of gout (beer and liquor were particularly strong factors). Fructose in soft drinks also increases the risk of gout.

Weight reduction can be helpful in lowering the risk of recurrent attacks of gout. This is best accomplished by reducing dietary fat and calorie intake, combined with a regular aerobic exercise program.

There are three aspects to the treatment of gout with medications.

- Pain relievers such as acetaminophen (Tylenol) or other more potent analgesics are used to manage pain.
- Antiinflammatory agents such as nonsteroidal antiinflammatory drugs (NSAIDS), colchicine, and corticosteroids are used to decrease joint inflammation.
- Medications are considered for managing the chronic underlying metabolic derangement that causes hyperuricemia and gout. This means treating the elevated levels of uric acid in the blood with medications that reduce these levels.

NSAIDS such as indomethacin (microcid) and naproxen (Naprosyn) are effective antiinflammatory medications for acute gout. These medications are tapered after the arthritis resolves. Common side effects of NSAIDS include irritation of the gastrointestinal system, ulceration of the stomach and intestines, and even intestinal bleeding. Patients who have a history of allergy to aspirin or nasal polyps should avoid NSAIDS because of the risk of an intense allergic (anaphylactic) reaction. Colchicine for acute gout is administered by mouth to reduce inflammation as well as to prevent gouty arthritis attacks while correcting hyperuricemia with medications such as allopurinol (Zyloprim) or febuxostat (Uloric). For acute attacks, it is

given hourly or every two hours until there is significant improvement in pain or the patient develops gastrointestinal side effects such as severe diarrhea. For prevention, it is given once or twice daily. Other common side effects of colchicine include nausea and vomiting.

Corticosteroids such as prednisone, given in short courses, are powerful antiinflammatory agents for treating acute gout. They can be administered orally or injected directly into the inflamed joint. Corticosteroids can be prescribed to patients who have accompanying kidney, liver, or gastrointestinal problems. Long-term chronic use of corticosteroids is discouraged because of serious long-term side effects.

In addition to medications for acute gout attacks, other drugs can be taken over prolonged periods to lower blood uric acid levels. Lowering blood uric acid levels reduces the risk of recurrent attacks of arthritis, kidney stones, and kidney disease, and also slowly dissolves hard tophi deposits. Medicines used to lower blood uric acid level work either by increasing the kidney's excretion of uric acid or by decreasing the body's production of uric acid from the purines in foods. These medicines are generally not started until after the inflammation from acute gouty arthritis has subsided because they can worsen the attack. If they are already being taken prior to the attack, they are continued and only adjusted after the attack has resolved.

Probenecid (Benemid) and sulfinpyrazone (Anturane) are medications that are commonly used to decrease uric acid blood levels by increasing the excretion of uric acid into the urine. Since these drugs can, in rare instances, cause kidney stones, they should be avoided by those patients with a history of kidney stones. These medications should be taken with plenty of fluid so as to promote the rapid passage of uric acid out of the urinary system in order to prevent kidney stone formation.

Allopurinol lowers the blood uric acid level by preventing- uric acid production. It actually blocks the metabolic conversion from purines in foods to uric acid. This medication is used with caution in patients with poor kidney function, as they are at a particular risk of developing side effects, including severe rash and liver damage.

Febuxostat was approved by the U.S. Food and Drug Administration (FDA) for the chronic management of hyperuricemia from gout in 2009. Febuxostat has been shown to be more effective than allopurinol in preventing acute attacks of gouty arthritis and is effective in shrinking tophi deposits of uric acid in the tissues such as the fingers, elbows, and ears. Because febuxostat is not significantly metabolized by the kidneys, it may have advantages over allopurinol in patients with underlying kidney disease. While taking febuxostat, patients have uric acid and liver function blood tests monitored regularly.

Again, uric acid-lowering medications such as allopurinol and febuxostat are generally not started in patients who are having acute attacks of gout. These medications, when started during an acute attack, actually can worsen the acute inflammation. Therefore, uric acid-lowering drugs are usually instituted only after complete resolution of the acute arthritis attacks, but if patients are already taking these medications, they are maintained at the same doses during the acute attacks. In some patients, increasing the dose of uric acid-lowering medications can precipitate gout attacks. In these patients, low doses of colchicine can be given to prevent the precipitation of acute gout.

Home remedies which can alleviate the symptoms of acute gout include resting and elevating the inflamed joint. Ice-pack applications can be helpful to reduce pain and decrease inflammation. Patients should avoid aspirin-containing medications, when possible, because aspirin prevents kidney excretion of uric acid.

Latest Trends

Active research is ongoing in a variety of fields related to gout and hyperuricemia. The management of the chronic gouty disease and its relationship to improving blood pressure and kidney function is becoming better defined.

Scientists recently reported that high animal protein intake slightly increased the risk for gout. Others found that dietary calcium intake may protect patients from getting gout attacks.

New medications to increase the elimination of uric acid in the urine (such as benzbromarone) and lower uric acid blood levels (such as PEG-uricase) are being evaluated in clinical trials. Researchers are also reporting on experimental drugs that can affect the chemical messengers involved in gouty inflammation.

The optimal regimens for the treatment of acute gout attacks and chronic gout conditions still require further long-term studies. Research scientists will continue to develop less toxic and more effective medications to battle this 'scourge of the ages.'

At a Glance

- Painful gouty arthritis is caused by uric acid crystal deposits in joint tissue.
- The tendency to develop gout and elevated blood uric acid level (hyperuricemia) is often inherited.
- Gout and hyperuricemia are aggravated by obesity, weight gain, alcohol intake, high blood pressure, abnormal kidney function, and certain medications.
- Gouty arthritis attacks can be precipitated by dehydration, injury, fever, heavy eating, heavy alcohol consumption, and recent trauma or surgery.
- The most reliable diagnostic test for gout is the identification of crystals in joints, body fluids, and tissues.

- The treatment of an attack of gouty arthritis is different than the treatment of hyperuricemia. There are two key concepts essential to treating gout. First, it is critical to stop acute inflammation of joints affected by gouty arthritis. Second, it is important to address the long-term management of the gout disease in order to prevent future gout arthritis attacks and shrink gouty tophi crystal deposits.