Patient information: Gout

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GOUT OVERVIEW

Gout is a painful and debilitating condition that develops in some people who have chronically high blood levels of urate (commonly referred to as uric acid). Not everyone with high blood urate levels (called hyperuricemia) develops gout; up to two-thirds of individuals with hyperuricemia never develop symptoms. It is unclear why some people with hyperuricemia develop gout while others do not.

Although the joints are the most commonly affected part of the body, other parts of the body can also be affected. Uric acid or urate crystals can be deposited in the kidney or urinary tract, causing kidney stones and occasionally impairing kidney function. Kidney stones caused by uric acid crystals occur in approximately 15 percent of people with gout. This compares to an 8 percent risk of kidney stones in people without gout [1].

Gout is different than pseudogout, which is discussed in a separate topic review. Pseudogout is a form of arthritis that develops in some people in response to the presence of calcium pyrophosphate dihydrate (CPPD) crystals. (See "Patient information: Pseudogout".)

GOUT RISK FACTORS

Gout is most common in men between 30 and 45, and in women between ages 55 and 70. It is estimated that gout affects approximately 2 percent of people in the United States.

The following characteristics increase the risk of developing gout:

- Obesity
- High blood pressure
- Injury or recent surgery
- Fasting
- Consuming excessive amounts of alcohol (particularly beer, whiskey, gin, vodka, and rum) on a regular basis
- Overeating
- Ingesting large amounts of meat and seafood
- Taking medications that affect blood levels of urate (especially diuretics)

GOUT SYMPTOMS

Gout attacks cause sudden severe joint pain, sometimes with redness, swelling, and tenderness of the joint. Although an attack typically affects a single joint, some people develop a few inflamed joints at the same time. The pain and inflammation are worst within several hours, and generally improve completely over a few days to several weeks, even if untreated. It is not clear how the body "turns off" a gout attack.

The characteristic pain and inflammation of gout develops when a type of white blood cell, called neutrophils, attempt to surround and digest urate crystal deposits. White blood cells are the body's first line of immune defense, and recognize the crystal deposits as foreign material. Chemical signals released by other types of white blood cells and cells in the joint contribute to the pain, swelling, and

redness associated with a gout attack.

PHASES OF GOUT

There are three main phases of gout: acute gouty arthritis, intercritical gout, and chronic tophaceous gout.

Acute gouty arthritis — Attacks of gout usually involve a single joint, most often the big toe or knee. This attack is known as acute gouty arthritis. People with osteoarthritis in the fingers may experience their first gout attacks in the fingers rather than the toes or knees.

Intercritical period — The time between gout attacks is known as the intercritical period. A second attack typically occurs within two years, and additional attacks may occur thereafter. If gout is untreated over a period of several years, the time between attacks may shorten and attacks may become increasingly severe and prolonged. Over time, the attacks can begin to involve multiple joints at once and may be accompanied by fever.

Chronic tophaceous gout — People who have repeated attacks of gout over many years can develop tophaceous gout. This causes large numbers of urate crystals to collect in joints, bones, and cartilage. The urate collection causes a nodule or mass called a tophus (plural tophi) to form. The tophus causes resorption and erosion of the bone, and can potentially cause deformity.

The presence of tophi near the knuckles or small joints of the fingers can be a distressing cosmetic problem. Tophi are usually not painful or tender. However, they can become inflamed and cause symptoms like those of an acute gouty attack (picture 1).

Tophaceous gout was more common in the past, when treatment for hyperuricemia was unavailable. Certain groups are still at risk for tophaceous gout, including:

- People who are treated with cyclosporine after organ transplantation
- Those who cannot tolerate adequate doses of medications to treat hyperuricemia (for example, due to kidney failure or drug allergy)
- Women who are postmenopausal, especially those taking a diuretic

The risk factors listed previously can also contribute to the development of tophaceous gout. (See 'Gout risk factors' above.)

GOUT COMPLICATIONS

People with gout are at increased risk of developing kidney stones. Uric acid crystals can collect in the urinary tract and form a stone. If a stone is large enough, it can block one of the tubes (ureters) that carry urine from the kidney to the bladder and out of the body (figure 1). (See "Patient information: Kidney stones in adults".)

Rarely, urate crystals collect in the kidney tissue itself, where they can cause inflammation and scar tissue, which reduce kidney function. Medications that increase the amount of uric acid excreted by the kidneys may help to reduce the risk of developing kidney stones.

GOUT DIAGNOSIS

There are many illnesses that can cause joint pain and inflammation. Gout is strongly suspected if a person has an acute attack of joint pain, followed by a period when there are no symptoms. It is important to confirm the diagnosis of gout to ensure that potentially harmful medications are not taken unnecessarily over a prolonged period of time.

The best way to diagnose gout is to examine synovial fluid from an affected joint, to look for urate crystals in the sample. To obtain the fluid, the provider uses a needle and syringe to withdraw a small amount of fluid from inside the joint. Tophi located just beneath the skin can be sampled with a needle

to diagnose tophaceous gout.

However, some people do not have urate crystals in their synovial fluid when symptoms are present. In this case, the diagnosis is based upon a person's symptoms and a physical examination. Criteria for diagnosing gout include:

- A history of pain and inflammation involving one joint at a time, especially the joint at the base of the large toe
- Complete resolution of symptoms between attacks
- Blood testing showing high levels of urate
- Rapid improvement in joint inflammation after treatment with colchicine

TREATMENT OF GOUT ATTACKS

The goal of treatment of flares of gouty arthritis is to reduce pain and inflammation quickly and safely. It may be necessary to use more than one drug to achieve this goal. Deciding which medication to use is based upon several factors, including a person's risk of bleeding, their kidney health, and whether there is a past history of an ulcer in the stomach. Anti-inflammatory medications are the best treatment for acute gout attacks, and are best started early in the course of an attack. (See "Treatment of acute gout".)

People with a history of gout should keep medication on hand to treat an attack because early treatment is an important factor in decreasing the pain and severity of an attack.

Nonsteroidal antiinflammatory drugs — Nonsteroidal antiinflammatory drugs (NSAIDs), work to reduce swelling in a joint, and include ibuprofen (Advil®, Motrin®) and indomethacin (Indocin®). NSAIDs are generally recommended for people who have no history of kidney or liver disease, no bleeding problems, do not use anticoagulant medications (blood thinners such as warfarin or Coumadin®), and who have no history of a stomach or duodenal ulcer. (See "Patient information: Nonsteroidal antiinflammatory drugs (NSAIDs)".)

NSAIDs are most effective in the treatment of a gout attack when they are started as soon as possible, before the attack is full blown. People who have had previous attacks may start taking an NSAID at the first signs of a recurrence.

Although aspirin is an NSAID, it is not usually recommended for the treatment of gout because it can raise or lower urate levels in the blood.

Colchicine — Colchicine may be prescribed instead of an NSAID. Colchicine does not increase the risk of ulcers, has no known interaction with anticoagulants, and in proper doses, does not affect kidney function. However, colchicine can have bothersome side effects, including diarrhea, nausea, vomiting, and crampy abdominal pain. For this reason, colchicine is generally reserved for patients who cannot tolerate NSAIDs. Some people have a great deal of success with colchicine and do not have side effects; colchicine might be used first for this group. Colchicine is generally taken as a pill.

Steroids — Steroids, also known as glucocorticoids, are effective anti-inflammatory agents. Commonly used oral steroids include prednisone, prednisolone, and methylprednisolone.

Steroids may be used if NSAIDs and colchicine cannot be used. They may be injected directly into the affected joint (called an intraarticular injection) or they can be given as pills or by intramuscular injection. People who have multiple affected joints or who cannot take NSAIDs or colchicine may be given oral steroids. However, there is an increased risk of recurrent gout attack (called rebound) in people who take oral steroids. For this reason, steroids should be tapered slowly over a period of seven to 10 days.

PROPHYLACTIC GOUT THERAPY

Prophylactic therapy aims to prevent or reduce the occurrence of acute flares of gouty arthritis. Colchicine is usually recommended as prophylactic therapy; it is taken daily at low doses to avoid

gastrointestinal side effects. Colchicine reduces the frequency of acute gout attacks, particularly while starting other drugs that lower urate levels.

Prophylactic colchicine is not usually used as a long-term (years) treatment, but is a helpful bridge as a person progresses from an acute flare to preventive therapy. Although not as well documented as colchicine, daily NSAIDs are sometimes used for prophylactic therapy, and may have an advantage (because of pain relieving properties) for people who also have osteoarthritis.

PREVENTIVE GOUT THERAPY

Preventive therapy includes medications and dietary changes that can be used long-term to lower urate levels and prevent the progression of gout. Progressive gout can cause bone destruction and deformity (gouty arthropathy), disability, kidney stone formation, and, possibly, kidney damage. People who have one or more of these complications are strongly encouraged to take a urate-lowering treatment. (See "Prevention of recurrent gout".)

Not everyone with gout will require preventive therapy; those who have rare or mild attacks are often able to manage their gout by treating the acute attacks alone. On the other hand, people with sporadic gout flares that are unusually prolonged, painful, and/or disabling are often encouraged to take preventive therapy.

Medications — Urate-lowering or antihyperuricemic medications lower urate levels by helping to eliminate or decrease production of uric acid. Antihyperuricemic therapy is usually started after a gout attack has resolved. People who take their medication regularly experience fewer attacks. At present, preventive therapy is recommended indefinitely because there is no benefit to taking a break from medication.

- Probenecid increases the efficiency of uric acid excretion by the kidney, and is called a uricosuric drug. Benzbromarone is a more potent uricosuric drug, but is not available in the United States. Both drugs can cause side effects, including rash, stomach upset, and kidney stone formation.
- Losartan is used to treat high blood pressure but also has a useful, though weak, urate-lowering effect, as does the lipid-lowering drug fenofibrate.
- Allopurinol (Alloprim®, Zyloprim®) works by preventing the formation of uric acid. It is the most commonly used drug for lowering urate levels in gout. Allopurinol can cause side effects, including rash, lowered white cell and platelet counts, diarrhea and fever, although these problems occur in a relatively small percentage of patients.

Lowering urate levels is a process that can take weeks or months. Very rapid urate lowering can cause acute flares of gout. Medications to lower urate are generally started at a low dose and increased slowly until the blood urate level is reduced and maintained at a level where urate crystal formation is unlikely. Increased fluids are recommended during this time (at least two liters per day are recommended).

The prophylactic therapy (colchicine or NSAIDs) (see 'Prophylactic gout therapy' above) may be discontinued when blood levels of urate are normal and have been stable for about six months. Longer prophylactic therapy may be needed, as in patients with tophi. Blood levels of urate are monitored periodically to ensure that the goal urate level is maintained.

- Febuxostat (Uloric®) works similarly to allopurinol to lower urate levels. Although it is a more expensive alternative, it may be useful for people who need treatment to prevent repeated attacks of gout but who cannot take probenecid, benzbromarone, or allopurinol. Periodic measurement of liver function is recommended during treatment with febuxostat.

Dietary changes — Changing your diet may reduce the frequency of gout attacks. Because obesity is a risk factor for gout, as well as for many other health conditions (heart disease, diabetes, high blood pressure), losing weight is an important goal. However, starvation or fad diets are not recommended

[2]. (See "Patient information: Weight loss treatments".)

Diet guidelines for patients with gout have changed over time, and it is not completely clear which combination of foods is best. The current recommendations include eating less (table 1):

- Red meat
- Seafood
- Beer and hard alcohol (eg, gin, vodka)
- Foods and drinks that contain high-fructose corn syrup (found in some non-diet sodas)

You are encouraged to eat and drink:

- Low fat dairy products
- Foods made with complex carbohydrates (whole grains, brown rice, oats, beans)
- A moderate amount of wine (one to two 5 ounce servings per day) is not likely to increase the risk of a gout attack.
- Coffee may decrease the risk of gout attacks
- Vitamin C (500 mg per day) has a mild urate-lowering effect and may be recommended.

Changes in diet are often recommended along with medications. Making changes in your diet, without taking a medicine, is not likely to make a big difference in your blood urate levels; following a very strict gout diet only lowers blood urate levels slightly (15 to 20 percent).

WHERE TO GET MORE INFORMATION

Your healthcare provider is the best source of information for questions and concerns related to your medical problem.

This article will be updated as needed every four months on our web site (www.uptodate.com/patients).

Related topics for patients, as well as selected articles written for healthcare professionals, are also available. Some of the most relevant are listed below.

Patient Level Information:

Patient information: Pseudogout
Patient information: Kidney stones in adults
Patient information: Nonsteroidal antiinflammatory drugs (NSAIDs)
Patient information: Weight loss treatments

Professional Level Information:

Asymptomatic hyperuricemia
Clinical manifestations and diagnosis of gout
Diuretic-induced hyperuricemia and gout
Hyperuricemia and gout in renal transplant recipients
Pathophysiology of gouty arthritis
Prevention of recurrent gout
Treatment of acute gout

The following organizations also provide reliable health information.

- National Library of Medicine

  (www.nlm.nih.gov/medlineplus/healthtopics.html)

- National Institute of Arthritis and Musculoskeletal and Skin Diseases

  (301) 496-8188
  (www.nih.gov/niams/)

- American College of Rheumatology

(404) 633-3777
(www.rheumatology.org)

- The Arthritis Foundation

(800) 283-7800
(www.arthritis.org)

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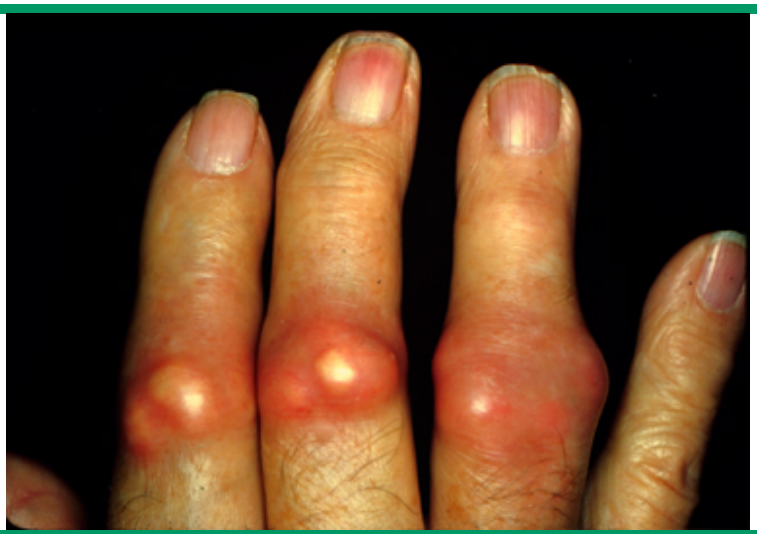
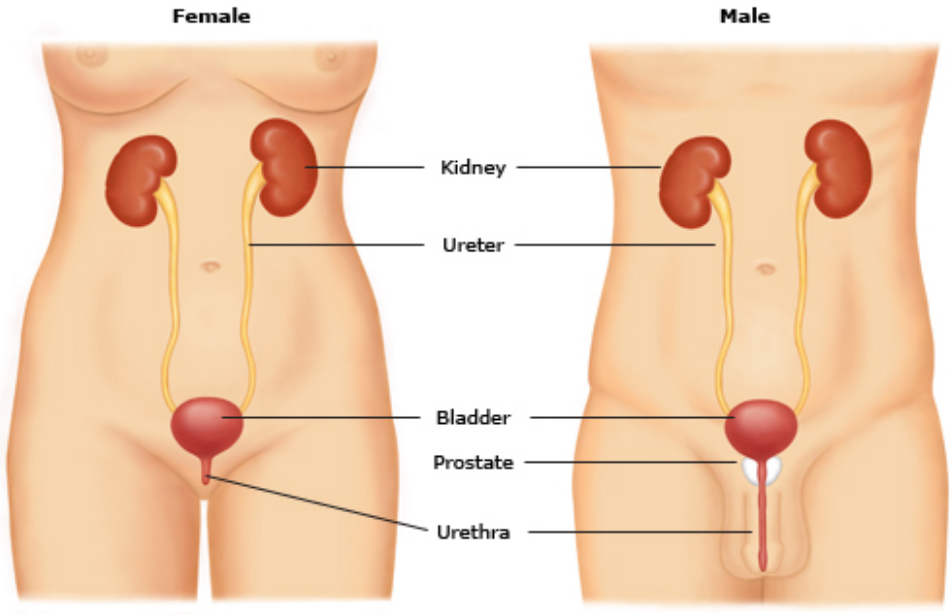
GRAPHICS

Inflamed tophaceous gout



Three inflamed tophi over the proximal interphalangeal joints in a patient with chronic tophaceous gout. Several of the lesions ruptured spontaneously over the next three days, exuding a pasty material composed of urate crystals and inflammatory cells but no organisms. The inflammation largely subsided over one week after the administration of a nonsteroidal antiinflammatory drug. *Courtesy of Michael A Becker, MD.*

Urinary tract



Urine is made by the kidneys. It passes from the kidneys into the bladder through two tubes called the ureters. Then it leaves the bladder through another tube, called the urethra.

Diet recommendations for gout

The current recommendations for lowering levels of urate in your blood include eating less:
• Red meat
• Seafood
• Beer and hard alcohol (eg, gin, vodka)
• Foods and drinks that contain high-fructose corn syrup (found in sweets and non-diet sodas)
You are encouraged to eat and drink:
• Low-fat dairy products
• Foods made with complex carbohydrates (whole grains, brown rice, oats, beans)
• A moderate amount of wine (up to two 5 ounce servings per day) is not likely to increase the risk of a gout attack
• Coffee may decrease the risk of gout attacks
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Dietary guidelines for patients with gout have changed over time, and it is not completely clear which combination of foods is best. The list above includes a few suggestions to lower your urate level.