Patient education: Gout (Beyond the Basics)

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GOUT OVERVIEW — Gout is a painful and potentially 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, but the symptoms of gout result from the body's reaction to deposits of urate crystals in tissues.

Although the joints are the most commonly affected part of the body, urate crystals can form in the kidney or other parts of the urinary tract (figure 1), where they can occasionally impair kidney function or cause urinary tract stones. Kidney stones caused by uric acid crystals occur in approximately 15 percent of people with gout. This compares with an 8 percent risk of kidney stones in people without gout.

Gout is different from another disease called calcium pyrophosphate crystal deposition disease (formerly called "pseudogout," which is discussed in a separate topic review). This develops in some people in response to the presence of a different type of crystal known as a calcium pyrophosphate (CPP) crystal. (See "Patient education: Pseudogout (Beyond the Basics)".)

GOUT RISK FACTORS — Gout usually develops in adulthood and is rare in children. It commonly develops earlier in adult men (often at ages 30 to 45 years) than in women (usually after age 55), and is particularly common in people older than 65 regardless of gender. It is estimated that gout affects nearly 4 percent of adults in the United States.

There are several medical conditions and lifestyle choices that increase the risk of developing gout, including:

- Obesity
- High blood pressure
- Chronic kidney disease
- Injury
- Fasting
- Consuming excessive amounts of alcohol (particularly beer, whiskey, gin, vodka, or rum) on a regular basis
- Overeating
- Consuming large amounts of meat, seafood, or beverages containing high fructose corn syrup (such as non-diet sodas)

- Taking medications that affect blood levels of urate (especially diuretics)

In people already diagnosed with gout, there are also certain characteristics that increase the risk of repeated attacks. These include:

- Injury or recent surgery
- Fasting
- Consuming excessive amounts of alcohol (wine may also be implicated as a risk for additional attacks of gout in people who have had prior attacks)
- Overeating
- Taking medications that affect blood levels of urate

GOUT SYMPTOMS — Gout attacks (also called flares) are sudden episodes of severe joint pain, usually 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. Attacks start more often overnight and in the early morning hours than during the day, but they can occur at any time. The pain and inflammation usually reach their peak intensity within 12 to 24 hours and generally improve completely within 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 develop when white blood cells and cells in the joint linings attempt to surround and digest urate crystal deposits. These cells recognize the crystal deposits as foreign material and release chemical signals that 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 — Initial gout flares usually involve a single joint, most often the big toe or knee. This attack is known as acute gouty arthritis. Over time, the attacks can begin to involve multiple joints at once and may be accompanied by fever. 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 an 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 and involve multiple joints.

Chronic tophaceous gout — People who have repeated attacks of gout or persistent hyperuricemia for many years can develop tophaceous gout. This designation describes the accumulation of large numbers of urate crystals in masses called tophi. People with this form of gout develop tophi in joints, bursae (the fluid-filled sacs that cushion and protect tissues), bones, and cartilage, or under the skin. Tophi may cause erosion of the bone and eventually joint damage and deformity (called gouty arthropathy).

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 can 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 or do not receive 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 for gout 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. The kidney stones composed of uric acid crystals that are part of gout often contain calcium crystals as well. The crystals can collect in the urinary tract and form a stone. If a stone is large enough, it can block one of the ureters (tubes that carry urine from the kidney to the bladder and out of the body) (figure 1). Medications that increase the amount of uric acid excreted by the kidneys may increase 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 in which 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 the fluid lining the joint (synovial fluid) from an affected joint under a microscope to look for urate crystals. 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 also be sampled with a needle to diagnose tophaceous gout.

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

- Rapidly developing pain and inflammation initially involving one joint at a time, especially the joint at the base of the large toe
- Complete resolution of symptoms between attacks
- A blood test showing high levels of urate (most accurate for diagnosis after an acute flare resolves)

TREATMENT OF GOUT ATTACKS — The goal of treatment of flares of gouty arthritis is to reduce pain, inflammation, and disability quickly and safely. Deciding which medication to use is based upon several factors, including a person's risk of bleeding, kidney health, and whether there is a past history of an ulcer in the stomach or small intestine. Antiinflammatory medications are the best treatment for acute gout attacks and are best started early in the course of an attack.

People with a history of gout should keep medication on hand to treat an attack because early treatment is an important factor in determining how long it takes to decrease the pain, severity, and duration of an attack.

Nonsteroidal antiinflammatory drugs — Nonsteroidal antiinflammatory drugs (NSAIDs) work to reduce swelling in a joint and include ibuprofen (sample brand names: Advil, Motrin), naproxen (sample brand names: Aleve, Anaprox), indomethacin (brand name: Indocin), and celecoxib (brand name: Celebrex). Among the NSAIDs, naproxen is considered one of the safer medications with regard to cardiovascular side effects and has documented efficacy in acute gout. NSAIDs are generally recommended for people who have no history of kidney or liver disease, who have no bleeding problems, who do not use anticoagulant medications such as warfarin (Coumadin), and who have no history of a stomach or duodenal ulcer. (See "Patient education: Nonsteroidal antiinflammatory drugs (NSAIDs) (Beyond the Basics)".)

NSAIDs are most effective in the treatment of a gout attack when they are started as early as possible in the attack and at the higher doses of these agents—at which they have antiinflammatory, not just pain-relieving

(analgesic), properties. People who have had previous attacks may start taking an NSAID at the first signs of a recurrence. NSAID treatment is withdrawn within a day or two of the resolution of the acute flare.

Although aspirin is an NSAID, it is not usually recommended for the treatment of gout because it can, depending upon the dose used, either 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 when given in excess, including diarrhea, nausea, vomiting, and crampy abdominal pain. Lower doses of colchicine than formerly used have been shown to be as effective for acute gout as the higher doses recommended in the past, and the gastrointestinal side effects have been much less of a problem. Colchicine seems to be most effective when given at the first symptoms of an acute attack. Colchicine should be taken only as a pill. Doses of colchicine should be reduced in people with impaired kidney function. The intravenous preparation of colchicine should be avoided because of potentially severe adverse effects.

Corticosteroids — Antiinflammatory steroids, also known more properly as glucocorticoids, are effective agents for treating acute gout flares. Commonly used oral corticosteroids include prednisone, prednisolone, and methylprednisolone.

Corticosteroids 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. There may be an increased risk of recurrent gout attack (called a rebound attack) in people taking oral corticosteroids for severe attacks but reducing the dose too quickly. For this reason, corticosteroid dosing should be reduced slowly over a period of at least 10 to 14 days.

PROPHYLACTIC ANTIINFLAMMATORY GOUT THERAPY — Prophylactic antiinflammatory 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 drugs that lower urate levels.

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

LONG-TERM URATE-LOWERING THERAPY — Therapy to prevent progression of gout may include medications and lifestyle changes that can be used long-term to lower urate levels and thus prevent or reverse the urate crystal deposits that cause worsening of gout. Progressive gout can cause severe gouty arthropathy, disability, kidney stone formation, and possibly kidney damage. People who have one or more of these complications are especially strongly encouraged to take a urate-lowering treatment.

Not everyone with gout will require urate-lowering therapy; those very fortunate few who have rare or mild attacks may be able to manage their gout by treating the acute attacks alone; but if progression to joint damage or tophus development occurs, even these individuals should receive urate-lowering medication. On the other hand, people with frequent gout flares or with flares that are unusually prolonged, painful, or disabling, or with gouty joint damage or tophi should always be encouraged to take urate-lowering therapy.

Medications — Urate-lowering (antihyperuricemic) medications lower urate levels in one of three ways: they increase uric acid elimination by the kidneys, they decrease production of urate, or they convert urate to the more readily excreted allantoin. Urate-lowering therapy is usually started after a gout attack has resolved. People

who take their medication regularly and maintain urate levels below a goal-range of 6 milligrams/deciliter (mg/dL) over months to years eventually experience fewer attacks. At present, it is recommended that preventive therapy be continued 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, upset stomach, and kidney stone formation. Effective probenecid use requires two or three doses daily. Probenecid is not effective in patients with advanced kidney disease.

- 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. These agents can be useful adjuncts to urate-lowering treatment with allopurinol or febuxostat or with lifestyle modifications in gout patients with high blood pressure or high blood lipid levels, respectively.

- Allopurinol (brand names: Alloprim, Zyloprim) and febuxostat (brand name: Uloric) work by preventing the formation of uric acid. Allopurinol is the most commonly used drug for lowering urate levels in gout. Allopurinol can cause side effects, including rash, lowered white blood cell and platelet counts, diarrhea, and fever, although these problems occur in a small percentage of patients. The starting dose of allopurinol needs to be reduced in people with impaired kidney function, but doses can usually be gradually increased to achieve the target urate level. No such dose-lowering concern is present with febuxostat when used at the approved doses. Periodic measurement of liver function is recommended during treatment with febuxostat and with allopurinol.

- Lesinurad (brand name: Zurampic) is a medication to help lower uric acid levels that was initially approved in late 2015. It is given in addition to either allopurinol or febuxostat if the person's uric acid levels are still too high after taking one of those drugs. Lesinurad should not be used alone.

- Pegloticase (brand name: Krystexxa) works by breaking down urate into allantoin, an end-product that is more easily excreted. Pegloticase is given by repeated intravenous infusions and can lower urate levels rapidly and profoundly. This biologic agent is expensive; may cause allergic-like infusion reactions, some of which can be severe; needs careful monitoring; and is effective in the long term in only about 50 percent of cases. For these reasons, it is recommended that pegloticase use be limited to patients with advanced gout that cannot be controlled with oral urate-lowering therapies.

Lowering urate levels to a goal range with oral medications is a process that should take a number weeks or months to achieve. During this period, doses of the uric acid-lowering medications should be gradually adjusted to meet the goal (usually a blood uric acid level <6 mg/dL). Very rapid urate lowering can cause more frequent acute flares of gout. Increased fluids are recommended during this time (at least two liters per day are recommended).

The antiinflammatory prophylactic therapy (colchicine or nonsteroidal antiinflammatory drugs [NSAIDs]) (see 'Prophylactic antiinflammatory gout therapy' above) can usually be safely discontinued when blood levels of urate are normal and have been in the goal range for about six months. Longer prophylactic therapy may be needed in some patients, especially those with tophi. Blood levels of urate should be monitored periodically to ensure that the goal urate level is maintained.

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, losing weight is an important goal. However, starvation or fad diets are not recommended. (See "Patient education: Weight loss treatments (Beyond the Basics)".)

Diet guidelines for patients with gout have changed over time, and it is not completely clear which combination of foods is best.

You are encouraged to eat and drink:

- Low-fat dairy products
- Foods made with complex carbohydrates (whole grains, brown rice, oats, beans)
- Only a moderate amount of wine (up to two 5-ounce servings per day [about 300 mL per day] may be acceptable, unless the individual patient has found that this increases their risk of a gout attack)
- Coffee (may decrease serum uric acid levels)
- Vitamin C (500 mg per day has a mild urate-lowering effect)

Changes in diet are often recommended along with medications. Diet change alone is unlikely to lower blood urate levels by more than about 15 percent, even if the diet is severely restricted. On the other hand, when diet control is accompanied by weight loss (often with increased exercise), improvements in urate control can be more impressive.

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 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 — UpToDate offers two types of patient education materials.

The Basics — The Basics patient education pieces answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials.

Patient education: Gout (The Basics)
Patient education: Calcium pyrophosphate deposition disease (pseudogout) (The Basics)
Patient education: Ganglion cyst (The Basics)

Beyond the Basics — Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are best for patients who want in-depth information and are comfortable with some medical jargon.

Patient education: Pseudogout (Beyond the Basics)
Patient education: Kidney stones in adults (Beyond the Basics)
Patient education: Nonsteroidal antiinflammatory drugs (NSAIDs) (Beyond the Basics)
Patient education: Weight loss treatments (Beyond the Basics)

Professional level information — Professional level articles are designed to keep doctors and other health professionals up-to-date on the latest medical findings. These articles are thorough, long, and complex, and they contain multiple references to the research on which they are based. Professional level articles are best for people who are comfortable with a lot of medical terminology and who want to read the same materials their doctors are reading.

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: Pharmacologic urate-lowering therapy and treatment of tophi
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.niams.nih.gov/)
- American College of Rheumatology
(404) 633-3777
(www.rheumatology.org)
- The Arthritis Foundation
(800) 283-7800
(www.arthritis.org)

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