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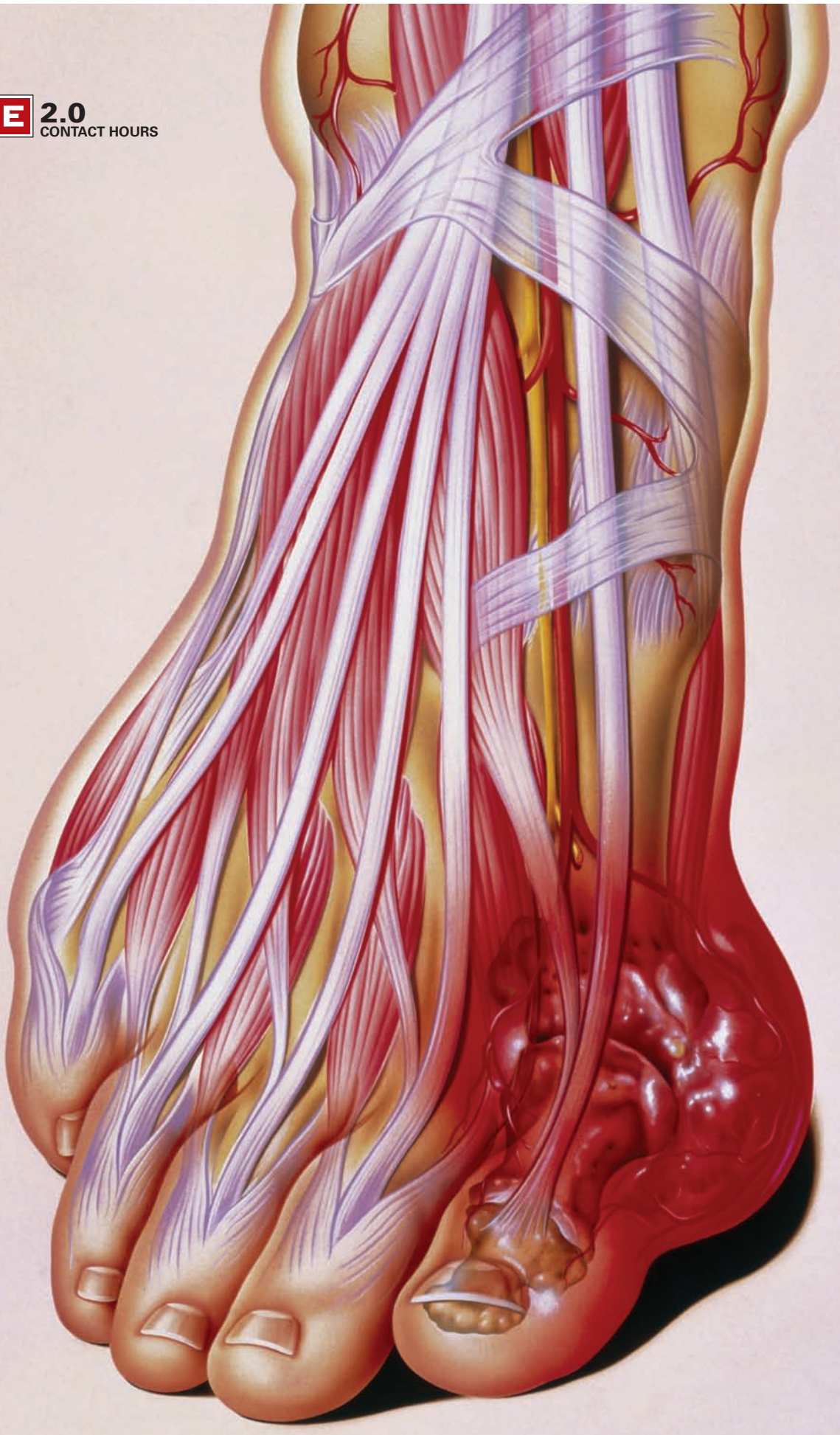


Treatment and prevention of gout

Abstract: Gout is a disorder of purine metabolism that primarily occurs in adult males. Elevated plasma uric acid concentrations (hyperuricemia) cause deposits of urate crystals in joint spaces causing severe, repeated attacks of arthritis.

By Scott J. Saccomano, PhD, RN, GNP-BC, and Lucille R. Ferrara, EdD, MBA, RN, FNP-BC

Known as the "disease of kings," gout has been affecting man since 2460 BC. In ancient Egypt, the prevalence of gout was related to wealthy men overindulging with food and drink.[1,2] It is estimated that approximately 6 to 8 million people are affected by gout in the United States.[3] Gout is rarely found in children. Men are affected more than women; however, the disparity seems to narrow as women approach menopause and estrogen levels decrease.[4] Gout commonly develops between ages 40 to 60 after many years (10 to 20 years) of hyperuricemia.[2] In the United States, Black Americans have an increased risk for gout than White Americans.[5] The increase prevalence of gout in Black Americans is likely related to increased frequency of hypertension, kidney dysfunction, and diet.[6]

Gout, characterized by periods of remission and exacerbation, is usually not life threatening but can be extremely painful and disabling. Patients with recurrent episodes of gout are likely to develop kidney stones. According to the CDC, gout accounted for approximately 1.5% of the over 900,000 arthritis hospital admissions in 2004 and 2.3 million ambulatory care visits annually from 2001 to 2005.[7] Gout accounts for nearly two-thirds of primary care provider (PCP) visits.[8] Gout is considered a disease with low mortality.[7]

Pathophysiology

High serum uric acid levels (known as hyperuricemia) are the cause of gout. Hyperuricemia is caused by an increase of urate in blood plasma and body fluids. A monosodium urate crystal compound is deposited into joints, soft tissues, and cartilage as the urate crystals increase in blood plasma. These crystallized urate compounds formed during gout trigger the inflammatory response, causing the development of symptoms and clinical findings of gout. Gout can be triggered by many mechanisms; the most common triggers are surgery or trauma. In addition, prophylactic uricosuric agents (which typically lower uric acid levels) can trigger a gout attack.[2,9]

Uric acid levels continue to increase during male adolescence; however, the majority of men do not develop gout and remain asymptomatic. Younger women are protected from increased uric acid levels due to estrogen production; however, uric acid production is increased in postmenopausal women, increasing the risk of gout later in life for women. Patients, who abuse alcohol have more frequent gout attacks at lower uric acid, as urine production is decreased in patients who abuse alcohol.[9]

Microcrystals (microtophi) tend to form in cooler areas of the body, such as the great toe, ears, and the olecranon bursa. For reasons unknown, gout is more common in the

Keywords: arthritis, gout, gouty arthritis, hyperuricemia, uric acid

great toe (first metatarsal joint) known as podagra.[9,10] Pathophysiologic theories believe that the lower temperature in the distal peripheral joint allows for increased crystallization. In addition, continuous use of the joint (as with ambulation and weight bearing) impairs reabsorption of the intra-articular urate in the joint. Gout can extend to multiple joints.[2,9]

The development of urate crystals occurs at lower temperatures; typically, synovial fluid in joints is lower than body temperature. Crystallization of urate usually develops at a concentration of 7 mg/dL; however, it is more likely to develop at lower concentrations in articular joints. Individually elevated urate levels do not cause gout. Hyperuricemia triggers an inflammatory response where urate specific molecules coat the monosodium crystals that promote the development of urate crystallization. The urate crystals are deposited into several body tissues. Tophi (nodular deposits of urate crystals) develop after multiple gout attacks and can be deposited in heart valves, the kidneys, and the larynx, causing substantial pathology.[2,9]

Common risk factors for gout

Predisposing risk factors for gout are classified as primary and secondary risk factors. Primary risk factors for gout include enzyme deficits and decreased renal clearance of uric acid. Primary risk factors of gout include isolated renal tubular defects affecting the clearance of uric acid and inborn metabolic errors.[3,4] The most common metabolic enzymatic error is an inherited X linked genetic disorder known as Lesch-Nyhan syndrome (Hypoxanthine-guanine phosphoribosyltransferase [HPRT] deficiency). In general, the HPRT deficiency hastens purine synthesis, leading to overproduction of uric acid. Renal tubular defects occur primarily in the proximal tubule. Organic compound clearance is reduced when creatinine clearance is reduced simultaneously, leading to hyperuricemia.[11]

Secondary risk factors are related to other disease entities and medications that contribute to the overproduction and decreased excretion of uric acid. Common secondary risk factors include: excessive purine intake, obesity, alcohol use, and medications such as thiazide diuretics, cytotoxics, and salicylates. Additional secondary risk factors for gout include hypothyroidism, hyperparathyroidism, psoriasis, chronic kidney disease, hypertension, and diabetic ketoacidosis.[4]

Overproduction of uric acid occurs in the following secondary risk factors: psoriasis, diets high or rich in purine, and cytotoxics. Decreased excretion of uric acid occurs in these secondary risk factors: diuretics, low-dose salicylates, kidney failure, obesity, alcohol intake, diabetic ketoacidosis, hypothyroidism, hyperparathyroidism, and hypertension.[3,4]

Clinical stages of gout:[5,11,14]

- **Stage I**: Asymptomatic, characterized by hyperuricemia.
- **Stage II**: Acute phase or the inflammatory phase characterized by acute gouty arthritis with painful monoarticular or polyarticular attacks.
- **Stage III**: Intercritical gout, the patient is asymptomatic between acute attacks.
- **Stage IV**: Chronic tophaceous gout results after multiple attacks with urate deposits in periarticular and articular tissue. Joint mobility may be compromised with possible chronic pain and stiffness of affected joints.

Clinical presentation

The history and physical exam will vary according to the stage of gout. Generalized questions to assess the clinical stage of gout should include the following:

- Medication history–especially salicylates, diuretics, and urate-raising medications
- Alcohol use or abuse
- Diet and exercise
- Family history of gout–primarily in a first-degree relative
- History of polycystic kidney disease, dehydration, thyroid disease

Physical exam findings can begin with an exam of the skin, extremities, and joints. Symptoms can include a tender warm joint(s) with skin over affected joint(s), shiny and red with significant swelling. The skin over the joint(s) can be taut and can ulcerate easily. Fever and chills may be present during an acute attack. With the history of long-standing gout attacks, evidence of fibrous tophi may be present in multiple joints, including the ears, hands, elbows (olecranon bursa), and feet (metatarsophalangeal joint). The affected joint will have limited range of motion. A normal physical exam can be present if the patient is not currently in an acute gout attack. Physical exam findings will vary based on the clinical stage of gout.[10,12-14] (See *Clinical stages of gout*.)

A thorough patient history should be completed to include onset, characteristics, and causes of pain. Acute gouty arthritis is primarily triggered by an "event." Events that trigger an acute attack include trauma, surgery, alcohol use, and systemic infection. Specifically, during an acute attack, the patient reports sudden onset of rapid developing throbbing, crushing, or pulsating pain, often awakening the patient at night. The pain can be severe enough to cause limited motion in the affected joint, prevent weight bearing, and, if accompanied by an infection, fever, redness, and erythema. The initial episode is usually isolated to one joint or is monoarticular in men. The first metatarsophalangeal (MTP) joint (podagra) is affected in the majority of the patients. Other joints that can be affected include: fingers, wrists, elbows, knees, and midfoot. The acute phase can last up to 10 days whether treated or not.[15]

Chronic tophaceous gout develops after long-standing acute recurrent gout. Firm urate crystals or tophi develop

and appear as firm palpable moveable nodules; the tophi are composed of deposits of monosodium urate crystals. The deposition of the tophi initiates the inflammatory process. Common sites where tophi develop include fingers, feet, olecranon bursa, and even the helix of the ear.[12] Tophi can restrict movement to the affected joint and can cause chronic pain and joint malfunction. Chronic tophaceous gout develops in approximately half of the patients within 20 years after the first attack.[3,15]

Complications of gout

The recurrence of gout attacks can be controlled with the proper use of uric acid lowering agents, and adherence to treatment. Gout rarely produces a long-term health threat; however, some long-term complications of untreated gout include:

- **Renal insufficiency**—If left untreated, elevated uric acid levels have been associated with renal insufficiency. Individuals with uncontrolled serum uric acid levels have an increased risk of kidney disease.[16] There is also the possibility of developing kidney stones when uric acid levels remain elevated.[17,18]
- **Pain and disability**—Untreated gout can be severely disabling. Gout destroys bones and cartilage, leading to deformities and decreased motion in the affected joint. The gritty firm mass of uric acid buildup (tophi) can grow quite large, destroying bone and cartilage similar to rheumatoid arthritis. Individuals may have difficulty ambulating and wearing shoes, which can cause considerable pain. The development of spinal tophi can cause spinal compression, resulting in complete disability.[9,19]
- **Heart disease**—Information is conflicting regarding the rates of cardiovascular disease and hyperuricemia. Studies confirm that there is an association between elevated uric acid levels and cardiovascular disease.[18,20]

Diagnostic testing

A complete and in-depth patient history and physical exam are vital to all diagnoses. Gout is attributed to many factors, including lifestyle (alcohol consumption, diet), underlying pathophysiology (decreased renal excretion of uric acid, comorbid states), and genetic predisposition (see *Lifestyle modifications*). The patient interview is an essential part of the diagnostic puzzle—especially when physical exam is not definitive and other differential diagnoses need to be considered. In addition to patient history and physical exam, the testing that is employed to support the diagnosis of gout is a combination of lab and radiologic studies.

Serum urate

Hyperuricemia or elevated serum urate levels are prevalent in 15% to 20% of populations studied that are at risk for gout due to lifestyle, underlying pathophysiology, or genetics.[21] It is recommended that serum urate levels be obtained for patients with an existing gout history or for those at risk for gout for baseline parameters. Hyperuricemia is often present in patients who are asymptomatic, but in some cases, serum urate levels may decrease during an acute attack.[22] It is because of this factor that fluid aspirated from the affected joint should be obtained (when possible) and evaluated for uric acid crystals in order to confirm diagnosis.[22]

Lifestyle modifications[3,13,30]

Activity

During a gout attack, limit activity, and maintain bed rest for 24 hours.

Immobilization

No weight bearing during acute attacks and joint immobilization.

Comfort

Ice packs 10 to 20 minutes during acute attack followed by warm compresses after acute pain. Warm compresses can be used 20 minutes twice daily to three times daily.

Long-term considerations

Weight loss, reduce alcohol consumption, reduce purine intake, and increase fluids.

Arthrocentesis

Joint fluid aspirate through arthrocentesis is recommended to confirm diagnosis of gout.[21-24] Some primary care settings do not support this procedure, and some providers are not skilled in performing joint aspiration; however, joint fluid aspiration should be performed if possible. Providers may opt to refer patients to a clinician who is skilled at performing joint aspiration. Using a sterile needle, fluid is aspirated from the affected joint and is then evaluated microscopically.

The aspirate is inspected for color; presence of bacteria; number of white blood cells and red blood cells; the presence and identification of crystals; and presence of protein and glucose. The fluid is then sent for culture and sensitivity to rule out infection. Joint fluid aspiration is most helpful for establishing a definitive diagnosis of gout and for assisting the clinician in choosing the appropriate pharmacologic therapy and planning for overall acute and chronic treatment.

Other lab testing

Additional blood testing includes a complete blood count (CBC), erythrocyte sedimentation rate (ESR), and white blood cell count (WBC). The CBC with differential WBC is performed to rule out or detect infection as in the case of septic arthritis, which, as mentioned previously, may coexist with gout and should be treated (however, it is rare).[21] The ESR provides essential information with regard to the level

and degree of inflammation, but ESR has also been found to be within normal range in some patients. Baseline lab studies that include blood chemistry, liver, and kidney function tests (such as creatinine, blood urea nitrogen, and electrolytes) should be performed in order to assess liver and renal clearance—especially when initiating pharmacologic therapy.

Radiologic testing

In addition to lab testing, radiologic testing can be performed. Plain film X-ray, computerized tomography (CT scan), magnetic resonance imaging (MRI), and ultrasound may be employed for the diagnosis of gout.

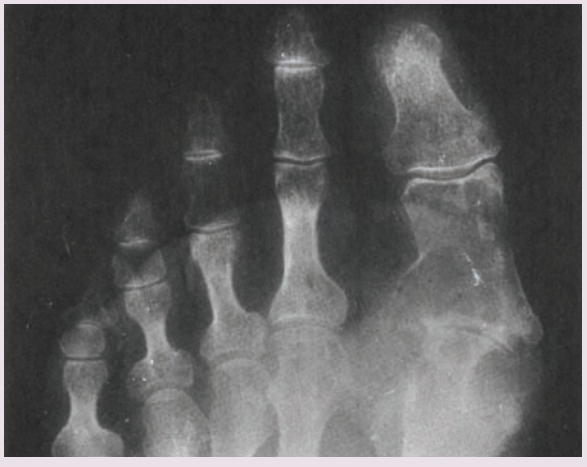
Plain films or X-ray are helpful when ruling out other orthopedic pathology but have not been found to be diagnostic of gout—especially in the early stages.[24] In later stages of gout, the typical findings include "punched out lesions," which are bone changes occurring outside of the MTP joint area.[25] As gout progresses, interosseous tophi become apparent with evidence of joint space narrowing.[25] (See *Gout changes on X-ray.*)

Both CT scan and MRI may be useful in providing an enhanced visualization of plain film findings as well as identifying soft tissue changes and other related orthopedic pathology. MRIs can be useful in detecting early tophic changes.[24,25]

Ultrasound has been found to be 96% sensitive and 73% specific in the diagnosis of gout.[25] The ultrasound is very helpful in the diagnosis of gout—especially when lab and radiologic findings are inconclusive.[25]

Gout changes on X-ray

The X-ray below of a patient with gout shows the first metatarsophalangeal joint with a lytic lesion that has destroyed the joint space. Additionally, there is an adjacent soft tissue tophus with surrounding edema.



Source: Rubin R, Strayer DS, Rubin E eds. *Rubin's Pathology: Clinicopathologic Foundations of Medicine.* 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012:1263.

Differential diagnosis

Differential diagnosis of gout includes the following:[12,13,26]

- Cellulitis—Acute gout with symptoms of redness, swelling, and pain can resemble cellulitis. Gout typically presents with a history of previous attacks, joint pain, and foot involvement.
- Septic arthritis—Can coexist with gout. Typical symptoms include joint pain in larger joints, swelling, erythema, fever, and chills; (+) Gram staining for bacteria.
- Rheumatoid arthritis–Results in a positive rheumatoid factor titer; rheumatoid arthritis has a symmetrical presentation.
- Psoriatic arthritis—In the early stages psoriatic arthritis, this can be similar to gout. Typically psoriatic arthritis presents with fusiform soft tissue swelling and early joint space narrowing.
- Pseudogout—Similar in presentation to gout; however, microscopic evaluation yields calcium pyrophosphate dihydrate crystal. Pseudogout is typically in older adults, is polyarticular, and is less severe than gout.

Pharmacologic treatment of gout

The main focus of pharmacologic treatment of acute and chronic gout is to relieve pain, reduce inflammation, and to lower serum urate levels to a target goal, which is often referred to as urate-lowering therapy (ULT). These outcomes can be achieved by initiating the various medications available for the treatment of gout either as a single agent or as a combination of two or more agents. Major considerations when initiating pharmacologic therapy include the presence of comorbidities that induce hyperuricemia, current medication use (diuretics, angiotensin-converting enzyme inhibitors), and hepatic/kidney function.[21,27,28] Providers should also consider eliminating unnecessary medications that may cause hyperuricemia.[27,28]

Acute and prophylactic therapy. Pharmacologic intervention typically occurs at two intervals: first during acute attacks and second for prophylaxis. For an acute attack of gout, nonsteroidal anti-inflammatory drugs (NSAIDs), intra-articular corticosteroid injection, and oral corticosteroid therapy are given and are the gold standards. Timing is the main focus for treatment during the acute phase.[29] To explain further, the sooner drug therapy is initiated during an acute attack of gout, the better the outcome for treatment success. Drug choice is based on contraindications of use, such as kidney disease, hypersensitivity, or peptic ulcer disease.[29]

The primary goal for prophylaxis is twofold. The first goal is to prevent bone, joint, and tendon disease, which are commonly seen with gout (arthritis, tophi). The second goal is to maintain low uric acid levels, which is well supported in the literature.[29,30] Allopurinol is the first-line drug of choice for achieving this goal. Febuxostat is also used especially

when allopurinol is contraindicated. According to the 2012 American College of Rheumatology Guidelines, the recommended target serum urate level should be a minimum of less than 6 mg/dL with further decrease to less than 5 mg/dL for overall improvement of symptoms.[27] Targets should be established based on an individual patient need. The current ULT agents used are allopurinol, febuxostat, and probenecid. Allopurinol and febuxostat are classified as Xanthine Oxidase Inhibitors (XOI). The mechanism of action of the XOI is to block or inhibit the activity of xanthane oxidase, which is an enzyme required for purine metabolism. The production of uric acid is decreased by blocking this enzymatic activity.

Probenecid is a uricosuric**.** The mechanism of action for this classification of drug is to increase renal excretion of uric acid. As the release of uric acid from the tissue level increases, gout pain may be intensified. The need for analgesia and anti-inflammatory preparations is vital. The use of NSAIDs and corticosteroids is recommended as concomitant therapy for acute gouty arthritis flares.

NSAIDs. There are a wide variety of NSAIDs currently used for their analgesic and anti-inflammatory properties.[21,30] The most commonly used medications include ibuprofen, naproxen, indomethacin, and celecoxib. Gastrointestinal upset and bleeding are the most common adverse reactions associated with NSAID use; NSAIDs are therefore contraindicated in patients with a history of peptic ulcer disease. NSAIDs also increase the risk of cardiovascular thrombotic events. In addition, NSAIDs are not recommended for patients with liver or kidney disease; cardiovascular risk should be assessed when prescribing NSAIDs. Although the NSAIDs mentioned are effective in the treatment of gout, Naproxen and indomethacin are specifically FDA approved for the treatment of gout. Colchicine can be used as an alternative for patients who are unable to tolerate or have contraindications for NSAIDs. Colchicine is an alkaloid made from the seeds of the Colchicum Autumnale plant that has potent anti-inflammatory properties—especially during gouty arthritis flares.

The 2012 American College of Rheumatology guidelines recommends corticosteroids for the treatment of acute gouty arthritis flares—especially if joint involvement is moderate to severe.[30] Corticosteroid therapy is usually administered orally, but in some cases where more than one is joint involved, intra-articular corticosteroid injection may also be employed.[30]

Treatment outcomes

As stated, lowering serum urate levels is vital to the prevention of acute gout flares. Both positive and negative outcomes are directly related to patient adherence to therapy, provider initiation of drug therapy in a timely manner, and patient tolerance to the medications prescribed. The secondary benefit of gout prevention is the prevention of bone and joint deformities, which cause arthritis, tendon changes due to the formation of tophi, and most important, the prevention of patient immobility. Treatment success during an acute gout flare is dependent upon the timing of drug initiation, patient tolerance, and potential contraindications. Using a patient-centered approach to care in which a partnership is established between the patient and the provider markedly increases the overall success of therapy.

Dietary modifications

The dietary approach to managing gout includes limiting foods high in purines, maintaining adequate fluid intake, and limiting alcohol consumption. Consuming 1 to 2 drinks per day is associated with higher uric acid levels, increasing the risk of gout. Diets completely purine free or purine restricted are controversial; the current recommendation is to follow a modified purine diet. Foods high in purine include: seafood and red meats (especially organ meats), meat extracts, yeast, beer, beans, peas, oatmeal, lentils, spinach, asparagus, cauliflower, and mushrooms. Patients with gout who are overweight or obese would benefit from weight loss. Obesity and being overweight are associated with an increase in uric acid levels and the potential risk of gout. Patients should maintain an adequate fluid intake to produce an output of 2,000 mL/day—especially if patients are taking a uricosuric agent. Increased fluid intake can also prevent the development of uric acid kidney stones.[31-33]

Moving forward

A form of arthritis, gout has become more common recently as a result of changes in diet, lifestyle, and environmental factors. Gout symptoms are the result of the deposition of uric acid crystals or urate in tissues and joints that become crystallized. The treatment goals are to decrease serum uric acid levels to prevent crystallization. Patient cooperation with the treatment plan is essential for gout management. Patients will need education about the disease process, the importance of drug therapy to control symptoms and reduce attacks and to implement lifestyle changes to prevent gouty attacks. Lifestyle education should include following a low-purine diet, maintaining a healthy weight, drinking water and other non-alcoholic beverages, and avoiding joint surgery.

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The authors and planners have disclosed no potential conflicts of interest, financial or otherwise.

DOI-10.1097/01.NPR.0000469254.90496.ab

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