



Medical Professional Guide: Gout Diagnosis and Treatment



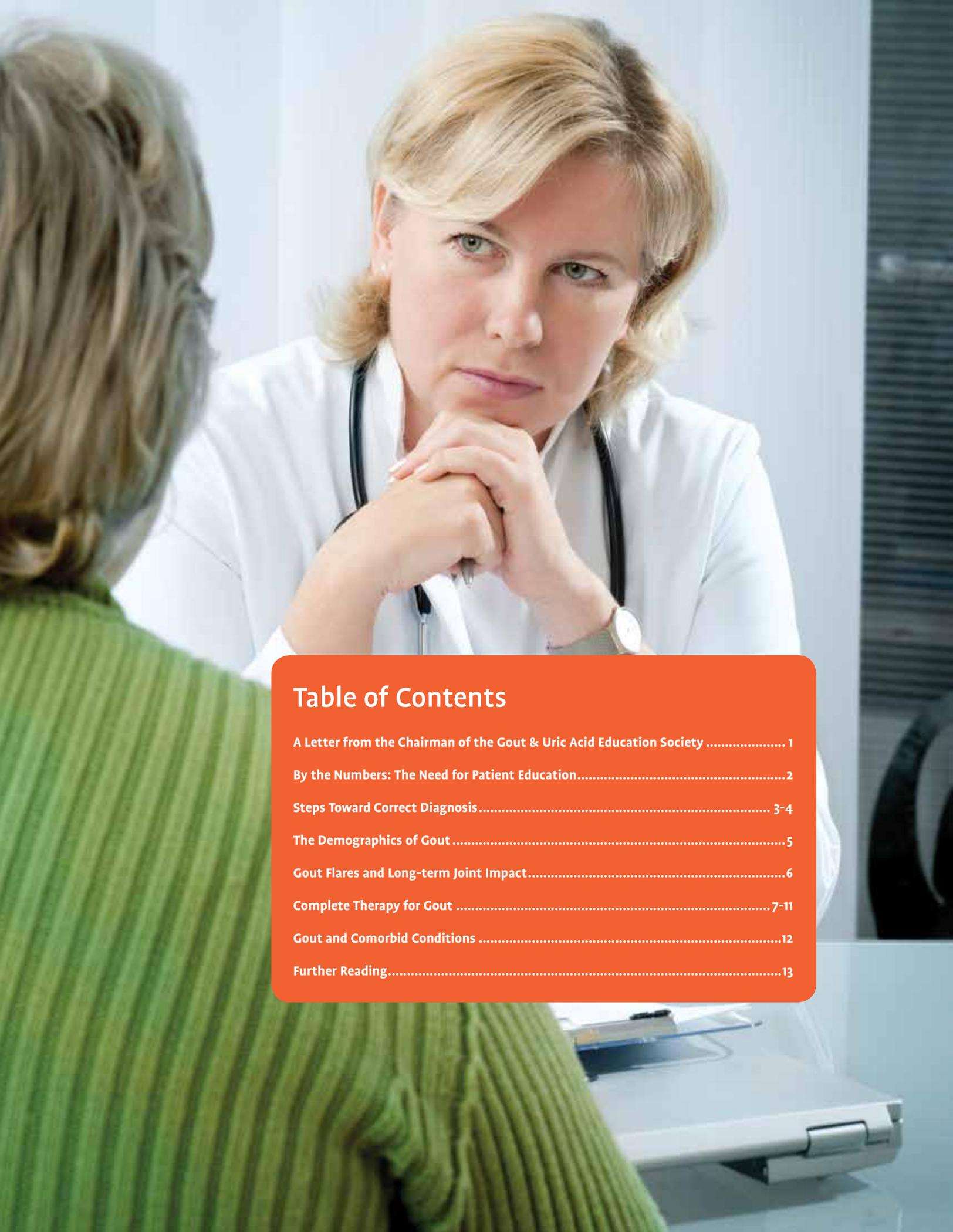
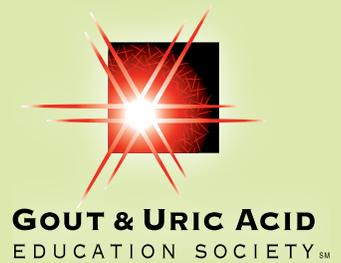


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A Letter from the Chairman of the Gout & Uric Acid Education Society



On behalf of the Board of Directors of the Gout & Uric Acid Education Society, I'm pleased to offer this new Medical Professional Guide on gout diagnosis and treatment. It was created to provide the latest perspective and information for all front-line medical professionals who see potential gout sufferers and new gout patients.



Gout incidence is on the rise. This extremely painful, inflammatory arthritis now affects more than 8.3 million Americans — about 8 percent of the population. Some of the rise can be associated with increased patients with obesity, high blood pressure, insulin resistance and blood lipid issues. Our research tells us, however, that only 10 percent of these gout sufferers are getting needed, ongoing treatment. This happens for a multitude of reasons — including sufferers relying on home remedies, patient non-adherence with treatment and misdiagnosis.

As rheumatology specialists who specifically treat gout, our Board's goal is to raise awareness about the growing patient numbers, and to make sure more sufferers receive a proper diagnosis and the ongoing, necessary treatment — combining a healthy diet and exercise with the right medication.

Our Board believes this guide can be a helpful tool, because many medical professionals — whether PCPs, ER physicians, nurse practitioners, podiatrists or orthopedic surgeons — treat varying numbers of gout patients, therefore having varying levels of exposure to the disease. Our hope is this information will offer evidence-driven counsel on consistent disease diagnosis and treatment, which will result in better care and more optimal outcomes.

If we can provide specific assistance to you or answer questions, please reach out to us at www.gouteducation.org/contact-us.

We're here to take a stand on gout. We hope you will join this national effort to improve the lives of gout sufferers.

Sincerely,

N. Lawrence Edwards, M.D.



By the Numbers: The Need for Patient Education

Only 1 in 5 adults
thinks gout is a
“serious” condition,
and just 7% think it
is “very serious.”



A survey conducted on behalf of the Gout & Uric Acid Education Society suggests that today's population has little knowledge of gout – a disease shrouded in myths, ambiguity and misconceptions. Consider that:

- 65% of adults admit to being “not at all knowledgeable” about gout.
- 7 in 10 adults don't know that gout is a form of arthritis.
- 3 in 4 don't know the parts of the body that gout affects.
- Only 1 in 5 adults thinks gout is a “serious” condition, and just 7% think it is “very serious.”
- More than 8 in 10 do not know that, compared to 40 years ago, the number of Americans diagnosed with gout is growing.
- 1 in 3 adults believes gout is curable, as do more than half of gout sufferers.
- Half are not aware of the potential crippling effects of gout, such as needing to use a cane or walker, or having to stop working because of limited mobility.
- Only 1 in 5 is aware that family history can be a risk factor for gout.
- Only 1 in 10 recognizes cardiovascular disease, 1 in 3 recognizes obesity, and less than 1 in 5 recognizes kidney disease as risk factors for gout.

Unfortunately, even gout sufferers don't understand the severity of their disease, and what they should be doing to minimize their risk of serious debilitation and decreased quality of life. Findings from another survey conducted on behalf of the Gout & Uric Acid Education Society speak for themselves, reaffirming the need for consistent patient education on the part of the medical professionals who are responsible for their care.

- While 9 in 10 gout sufferers say gout has interfered with their daily life, 1 in 5 says he or she doesn't do anything to manage the disease.
- Only 1 in 3 gout sufferers associates excessive alcohol intake with gout.

More than 1 in 3 of those who have suffered from gout have not had their uric acid (UA) levels checked in the last five years, or do not know if they have.

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1 Harris Interactive® survey on behalf of the Gout & Uric Acid Education Society, May 5-7, 2009 via the QuickQuery online omnibus service, among 2,176 adults ages 18+.

2 Kelton Research online survey on behalf of the Gout & Uric Acid Education Society, May 2-9, 2011, among 1,000 nationally representative Americans ages 18+, of which 235 were gout sufferers.

Steps Toward Correct Diagnosis

Diagnosing Gout

Gouty arthritis is one of an estimated 107 types of arthritis and is the most common form of inflammatory arthritis. Rheumatoid arthritis and psoriatic arthritis are also in the inflammatory arthritis category, but incidence is far less than that of gout.

Aspiration of synovial fluid from an actively inflamed joint and identification of monosodium urate crystals through a polarizing microscope is the gold standard for making a correct diagnosis of gout. However, physicians and other health care providers rarely make a diagnosis this way, given the need for specialized equipment and training. Therefore, most physicians must make a probable diagnosis based on how closely symptoms align with the classic description of gout symptoms.

Typical clinical features associated with gout, which the American College of Rheumatology (ACR) Guidelines consider a good clinical diagnosis, include the following:

Sudden, Severe Pain

A classic sign of gout is the sudden onset of excruciating pain that escalates rapidly from a joint that otherwise has no symptoms to one that is very swollen, red, and inflamed over a 12-hour period. The pain stays at that level for several days and gradually wanes over a period of one week to 10 days. For patients who have experienced several of these episodes followed by an asymptomatic period, this is a fairly assured diagnosis.

Hyperuricemia: Can Lead to Gout

Another clinical feature for gout is hyperuricemia, a common biochemical abnormality. At least 25 percent of the population has an elevated UA level, which is defined as above 7 mg/dL. This is the level at which UA could potentially come out

of solution and form problematic crystals. Although it is the metabolic underpinning of gout, UA is often not elevated during an acute attack. UA levels while a patient is in significant pain can be lowered by 2-2.5 mg/dL from the baseline reading. This is a common cause of misdiagnosis, or a missed diagnosis. Furthermore, hyperuricemia alone does not make for a diagnosis. Most people with hyperuricemia do not ever develop gout, although the higher the UA level, the more likely someone is to develop it.

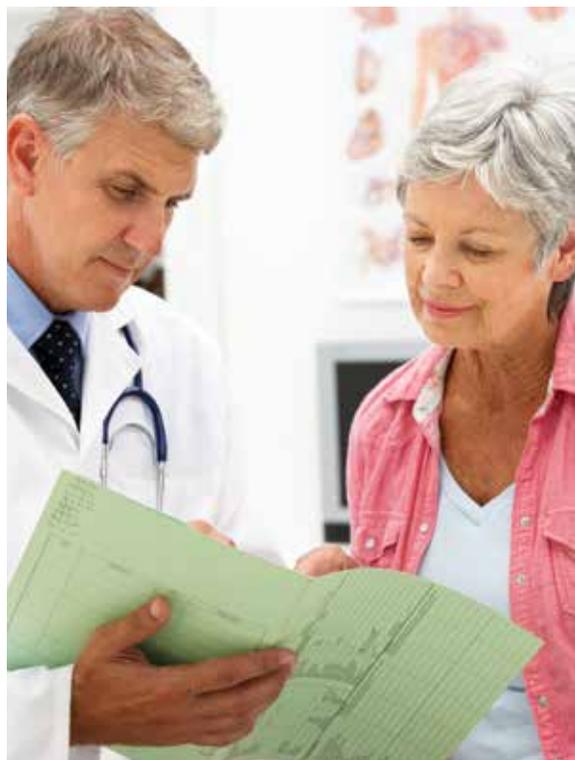
Hyperuricemia and the odds of gout:

- Only an estimated 1 in 5 people with UA above 7 mg/dL will ever experience symptoms of gout.
- Those whose UA level is above 9 mg/dL have a 1 in 2 chance of developing gout.
- If the UA level is around 12 mg/dL, the chance of developing gout increases to 3 in 4.

Distinguishing Gout from Other Conditions

Gout may be confused with infectious arthritis, which is also called septic joint and is a severe condition. Like gout, it is the only process that can cause rapid, very painful inflammation. Infectious arthritis can result from pneumonia, an abscess or a post-surgical infection elsewhere in the body. Drawing fluid from the joint and checking it for bacteria is important in these cases.

Another disease process that mimics and can be confused with gout is pseudogout. Clinically, it looks much like gout, but is caused by calcium pyrophosphate crystals. Typically, it presents with milder swelling and pain, but can be more severe and closely imitate gout. Pseudogout is usually found among people age 65 and older. Osteoarthritis, the most common non-inflammatory arthritis, is one of its risk factors.



Considering Family History

Another consideration is the genetic component. Anyone with a family history of gout may be at greater risk, although there is much more to learn about the role genetics play in the disease. Some known genetically inherited problems, such as some enzyme abnormalities and abnormalities in how kidneys transport and eliminate UA, can play a role in high UA levels.

Gout is a disease that “clusters” in families; those with a parent who had gout are more likely than the general population to get the disease. It is important to note that lifestyle issues, such as obesity and a diet rich in gout unfriendly foods, are risk factors often shared by family members. As with many lifestyle issues, some of these factors are modifiable and others are not.



What to Ask Patients

Review and confirm the health history. For patients with a number of comorbid medical conditions, such as heart disease, hypertension, hyperlipidemia, Type 2 diabetes, obesity, metabolic syndrome or chronic kidney disease, there is a greater suspicion that the joint symptoms may be gout.

Review the course of the pain during the flare. Typically, there are intermittent episodes of severe pain, with no pain experienced in between the episodes. As attacks continue over the years, painful episodes will last increasingly longer.

Ask the following questions to help in making an assessment:

1. Have you had any previous episodes that resemble the symptoms you are experiencing today?
2. Are there any events that may have triggered the symptoms, e.g., excessive consumption of alcohol or foods/beverages with high levels of purine?

3. Have you experienced any trauma to the inflamed joint?
4. Have there been any changes to the medications you take for other health conditions?
5. Have there been any changes to your diet or exercise routine?
6. Is there a family history of gout?

The bottom line for most primary care physicians in making a diagnosis is whether the patient has presented symptoms in typical fashion and if their UA is elevated during a time when they do not have severe symptoms. Once these are confirmed, a presumptive diagnosis can be made and treatment should begin. The more distant or atypical the case, the less assurance there is that gout is present. In such cases, referral to a rheumatologist to confirm the diagnosis may be appropriate.

Cautions

Medical professionals should be meticulous in making a diagnosis of gout unless they have witnessed symptoms firsthand and flares follow a classic history. Not everyone with elevated UA and joint pain has gout. There must be extensive evidence of inflammation not caused by infection, and the pain must be very severe. In men, the pain is usually in the lower extremities initially. Women usually experience initial pain in the upper-extremity joints.

Without witnessing a flare in a patient who has a classic history and hyperuricemia, or unless the gold standard diagnosis is made, medical professionals should be suspicious that gout is present. Infectious arthritis or even a stubbed toe or osteoarthritis could be the culprit.

Clinical Takeaways

- A definitive diagnosis of gout requires aspiration of synovial fluid and the presence of monosodium crystals.
- Hyperuricemia and a classic history of gout flares are sufficient for a presumed diagnosis of gout.
- Not everyone with elevated UA levels or joint pain has gout.

The Demographics of Gout

Caucasian and African-American Men

Gout flares typically occur in men in their 40s or (more commonly) in their 50s, usually after years of high UA levels. Gout is more common in men because, while their UA levels are also in the 3-4 mg/dL range in childhood, they reach their adult UA level at puberty. Men can start accumulating extra UA decades before women. While the incidence of gout in both elderly men and women reaches some parity, the overall prevalence of gout skews male, because men reach their adult UA level much sooner.



Multicultural Patients

Gout is a global disease and is increasing in prevalence worldwide. The same types of factors leading to increasing incidence in the U.S., such as the obesity epidemic, are responsible for increasing incidence and prevalence of the disease elsewhere in the world. Since gout is also a disease of UA accrual, increased longevity in both developed and undeveloped countries means that the incidence will continue to rise.

However, while risk factors such as obesity and other comorbidities play a role, genetics supersedes them. Several ethnic groups have a higher predilection for developing the disease than U.S. Caucasian populations, including Asians, Hispanics, South Pacific Islanders and the Maoris of New Zealand.

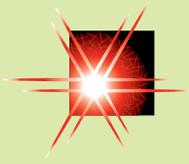
Flares may be seen approximately 10 percent more frequently among high-risk populations, and the disease progresses at a slightly accelerated pace and can be more destructive because UA levels are higher for longer. Otherwise, there is little difference in symptoms.

Post-menopausal Women and Gout

Women are at their greatest risk of gout in their post-menopausal years. At the same time, the risk for any particular woman is low. From a young age through their child-bearing years, women's UA levels remain at the low 3-4 mg/dL range established in childhood. Once they start to experience the gradual loss of estrogen over the next three decades, there is a gradual increase in their serum UA levels. Because they remain fairly protected from high UA even in menopause, women are at risk if their UA rises above 7 mg/dL. These are typically women who have kidney disease or are taking medications that raise UA, such as hydrochlorothiazide. Women at risk may not experience symptoms for a decade following menopause, unless they have another metabolic or medical problem. For those with a family history or other risk factors, or those taking a drug that can raise UA levels, testing their UA level is prudent.

Clinical Takeaways

- Be cautious of the stereotype that gout only afflicts overindulgent men.
- While gout is more prevalent in men, the decrease in estrogen in post-menopausal women can put them at risk.
- Everyone should be aware of the risk factors and symptoms. Gout is as serious as rheumatoid arthritis and requires lifelong care.
- Gout has a similar disease process across all ethnic groups, although it is more prevalent and more aggressive in some populations.
- Certain ethnic populations have a higher risk for comorbid conditions that can impact the development and progression of gout.



Gout Flares and Long-term Joint Impact

The ACR Guidelines for the Management of Gout confirm that therapy to lower UA is appropriate for patients who fit into one of several categories:

1. Any patient with relatively frequent attacks, defined as two or more attacks occurring in the same year.
2. Any patient with a history of kidney stones or chronic kidney disease of stage 3 or worse.

Location of Gout in the Body

For some patients, gout always stays in one of the big toes. But, as a general rule, arthritis moves around. For example, if the first affected joint is the right toe, the condition might move on to the left ankle, then to the mid left foot. In men, gout may initially skip between the lower extremities and stay there for years before moving on to upper-extremity joints, such as the wrist, elbow or small joints of the fingers. The more repeat flares a patient has in the early stage of gout, the more likely gout will affect several joints simultaneously. As time progresses and the patient is not placed on UA-lowering therapy, the frequency and duration of flares along with the number of joints affected will increase.

While women can present with similar symptoms as men, e.g., pain and inflammation in the great toe and intermittent flares, symptoms usually follow a different pattern. Women are more likely to experience early symptoms in the upper extremities such as the wrist, small finger joints and elbow. Nodal (lumpy) osteoarthritis is a common form of arthritis in older women. Sudden pain in the nodules could signal that there is gout in the joint. Women may also experience symptoms in other joints.

Onset and Progression of Gout Flares

Early on, attacks occur with a relatively short duration of five to seven days. After a first attack, it can be difficult to predict when the next one will strike – anytime from several months (11 is average) to several years later. Most patients will experience two attacks during their first year of symptoms; this is an important trigger that it is time to start a UA-lowering therapy.

Because attacks may occur infrequently in the beginning, gout patients often go untreated or may only experience one attack per year. The prevalence of gout attacks will grow more frequent, however. For example, flares may progress from one attack every 11 months to as frequently as once every seven to eight months. After a decade without proper treatment, patients may experience attacks accompanied by tremendous pain as frequently as once every three to four months. The duration of these attacks will also increase, from five to seven days up to two to three weeks at a time for up to a decade.

The bottom line: the frequency and duration of attacks will increase over time if a patient is not put on UA-lowering therapy.

Typically High Pain Level

The intensity of pain during gout flares will vary. Typically, patients will rate their pain as severe – at least a 7 on a 10-point scale; the average is 8.5. While these ratings may not change much over time, the location of the pain commonly will.

As the Disease Advances: Subcutaneous Tophi

Subcutaneous tophi are a calculus of monosodium urate crystals that develop in fibrous tissue around the joints as a result of gout. With the help of modern MRIs and ultrasounds, clinicians now believe that all stages of gout involve tophi. Even in early-stage gout, or before a patient develops symptoms, UA crystals begin depositing in and around the joint. This is characterized by a thin layer of crystals across the cartilage in the joint or small nodules of crystals deposited inside the joint. The nodules grow over time to be felt on physical examination and can become large and disfiguring. In some patients, they will erupt to the surface and drain the chalky UA crystals.

If a patient is receiving treatment, but their gout is progressing, the point at which a deformity begins to become apparent will vary significantly from person to person. The higher the UA level, the more rapidly the UA deposits will accumulate around the joints. A typical male patient who develops gout at age 50 will go through a decade or slightly more (10-12 years) experiencing these acute and intermittent flares. They will become more frequent and last longer, albeit with asymptomatic joints in between. After approximately a decade without appropriate treatment, the patient might enter into a stage of advanced gout in which symptoms are present consistently – either as flares or chronic aching and pain in the joint. While small UA deposits will be visible clinically at this advanced stage, the use of sophisticated imaging techniques can enable detection of tophi earlier.

Certain forms of gout will develop in patients who take drugs like cyclosporine or tacrolimus following organ transplantation of the heart, kidney or liver. In such cases, the time frame of elevated UA leading to gout symptoms and tophi may be significantly shorter. Tophi may begin to manifest within several years of beginning these drug regimens.

Complete Therapy for Gout

Before Choosing a Course of Treatment

There are a number of comorbid diseases associated with gout, such as heart disease, kidney disease and diabetes. Once a gout diagnosis is made, it is important to consider these comorbid diseases and how commonly used UA-lowering therapies may impact the patient's overall health. For example, avoid prescribing NSAIDs for patients with kidney disease and corticosteroids for patients with severe diabetes.

Physicians also need to be cognizant of what anti-inflammatory therapies patients can tolerate. For example, a patient who experienced gastrointestinal problems while on colchicine (nonsteroidal anti-inflammatory) should not receive it as treatment again. Always ask the simple question, "What have you taken before, and what was effective?"

Recommended Drug Therapy for Gout Flares

The treatment focus for patients experiencing gout flares should be on eliminating the pain as quickly as possible. Therefore, gout flares are generally treated with anti-inflammatory therapies to help appease intense pain and reduce swelling, although these do not address the underlying problem of UA elevation.

For acute gout flares, the standard therapies are either colchicine, some form of corticosteroid like prednisone or Medrol, or injections of corticosteroids such as triamcinolone or Depo-Medrol. Some patients will simply tolerate one form better than others. Most primary care physicians use NSAIDs such as naproxen (Naprosyn), ibuprofen or indomethacin (Indocin). High-dose ibuprofen at 800 mg three times per day for several days, followed by 600 mg, three times per day for another week is appropriate. Naproxen can



be given at 500 mg, two times per day and then cut down to 375 mg after several days, for a total of 10 days. See the chart on page 8 for more specific recommendations.

It is worth noting updated recommendations for the use of colchicine. Years ago, colchicine was administered in an IV formulation, which often had dangerous results. This led to an eventual ban on IV administration. Today, colchicine is available in tablet form. The recommended dosage to reach an effective level is much lower than in the past, yet just as effective; there are also fewer

side effects than the severe abdominal cramping and severe diarrhea patients experienced before. The appropriate dosing is two tablets at the onset of pain followed by a third tablet one hour later, with no more for the first 24 hours.

While all of these options provide temporary relief and help reduce inflammation, they are a temporary solution. Attacks will return until the underlying problem (elevated UA) is addressed. UA-lowering therapy is the cornerstone of preventing gout progression over time.



Therapies to Relieve Pain and Reduce Swelling of Acute Gout

Name	Dosage	Special Instructions	Possible Side Effects	Be Aware
Colchicine Colcrys	Two tablets (1.2 mg) immediately then one tablet (0.6 mg) after one hour. Then one tablet twice or three times daily for one week.	Take with food if stomach upset occurs. Drink plenty of fluids.	Diarrhea; nausea or vomiting; stomach pain.	High dose colchicine for acute flares is inappropriate. Colchicine should be used with caution in people with renal disease and those with bone marrow suppression.
Glucocorticosteroids Methylprednisolone (Medrol); Prednisone (Deltasone); Triamcinolone (Kenalog)	Kenalog 60 mg x1, followed by low dose steroids or oral prednisone given at 30 mg with a taper to 0 mg over 10 days		Retention of sodium (salt) and fluids; weight gain; high blood pressure; loss of potassium; poor glucose control; and headache.	Particularly useful for those with chronic kidney disease. Use with caution in diabetic patients.
Nonsteroidal anti-inflammatory drugs (NSAIDs) Celecoxib (Celebrex); Ibuprofen (Advil); Indomethacin (Indocin); Naproxen (Aleve, Naprosyn)	High dose of any non-steroidals given for first three days, followed by moderate doses for an additional seven days.		Nausea; stomach discomfort; retention of sodium and fluids; dyspepsia; gastric ulcers; and headache.	May interact with blood pressure and heart medications, especially in the elderly. Use caution in patients with a history of GI ulcers, kidney disease and the elderly.

Anti-inflammatory Prophylaxis for Prevention of Gout Flares

Name	Dosage	Special Instructions	Possible Side Effects	Be Aware
Colchicine Colcrys	One or two tablets (0.6 mg) per day. 1.2 mgs maximum per day. Patients with severe kidney disease may only need one tablet every other day or every third day, depending on creatinine clearance.	Take with food if stomach upset occurs. Drink plenty of fluids.	Diarrhea; nausea or vomiting; stomach pain.	Some people are very sensitive to colchicine. If diarrhea or abdominal pains occur, dosage should be reduced.
Nonsteroidal anti-inflammatory drugs (NSAIDs)	Low dose of any non-steroidals may be used prophylactically following the first six months of urate lowering therapy.		Nausea; stomach discomfort; retention of sodium and fluids; dyspepsia; gastric ulcers; and headache.	May interact with blood pressure and heart medications, especially in the elderly. Use caution in patients with a history of GI ulcers, kidney disease and the elderly. Ulcers may occur without any preceding symptoms.

Long-term UA Management

Most people who develop gout need lifelong therapy. It will take time to determine the correct UA-lowering therapy dosage that will enable a patient to reach and maintain the target range. Generally, once the target is reached, the patient stays on that dosage year after year. However, it may be necessary to increase the dosage if a patient adds or changes other medications that could interfere with the effectiveness of the UA therapy.

Typically UA-lowering therapy encompasses use of these drugs: allopurinol, febuxostat, pegloticase, probenecid and probenecid with colchicine. See the chart below for specific dosage recommendations, possible side effects and tips for each drug category.

Occasionally, a patient may make enough lifestyle modifications, such as losing weight or getting kidney disease under control, that it may be possible to use a lower dosage or go without UA-lowering

drugs to reach a UA level below 7 mg/dL. In that case, symptoms would subside and continued treatment would not be necessary. However, it is quite unusual. Most patients must remain on UA-lowering therapy for the rest of their lives to prevent further destruction and continued pain. It is important to monitor UA levels year over year, even after a diagnosis is made and even after UA-lowering therapy has achieved the target level.

Long-term Urate-lowering Therapies

Name	Dosage	Special Instructions	Possible Side Effects	Be Aware
Allopurinol Lopurin, Zyloprim	100 to 800 mg per day in a single dose. The dose is started and adjusted by 100 mg every two to four weeks to achieve a serum uric acid level lower than 6.0 mg/dL. Patients with severe renal impairment should be started with an initial dose of 50 mg per day with slower dose escalation to achieve target.	Stop taking medication at the first sign of a rash, which may indicate an allergic or hypersensitivity reaction. May need to give as BID in doses over 300 mg per day to avoid nausea.	Rash, hives or itching; nausea; transaminase elevation; rare severe cutaneous reactions occur in approximately 1 in 250-300 patient starts.	Never start or stop allopurinol during a gout attack. Minimize attacks by initially prescribing lower doses along with colchicine or NSAIDs until goal of a uric acid level of ≤ 6.0 mg/dL is reached. Caution with azathioprine, 6-mercaptopurine and theophylline.
Febuxostat Uloric	40 mg per day initially then increase to 80 mg per day in two weeks if serum uric acid level not lower than 6.0 mg.	Take any time of day without regard to food or antacid use.	Elevated liver enzymes (liver irritation); nausea; joint pain; rash.	Never start or stop febuxostat during a gout attack. Minimize attacks by prescribing colchicine or NSAIDs at the time of initiating treatment and until goal of a uric acid level of ≤ 6.0 mg/dL is reached. Contraindicated with azathioprine, 6-mercaptopurine and theophylline.
Pegloticase Krystexxa	8 mg given via IV every two weeks.	For use in difficult-to-control hyperuricemia and chronic gout.	Infusion reactions including fever, nausea and hypotension.	This drug should be given in a monitored infusion center.
Probenecid Benemid, Probalan	500 to 3,000 mg per day in two or three divided doses.	Take with food or an antacid. Drink plenty of fluids. Do not take with aspirin or other NSAIDs. Avoid alcohol.	Headache; loss of appetite; nausea or vomiting.	Ineffective in patients with glomerular filtration rate (GFR) less than 50. Should not be used with history of kidney stones.
Probenecid and colchicine Col-Benemid, Col-Probenecid, Proben-C	One tablet (contains 500 mg probenecid and 0.5 mg colchicine) twice per day.	Take with food or an antacid. Drink plenty of fluids. Do not take with aspirin or other NSAIDs. Avoid alcohol.	Diarrhea; headache; loss of appetite; nausea or vomiting; stomach pain; rash.	Ineffective in patients with GFR less than 50. Should not be used with history of kidney stones.



Diet and Lifestyle Tips

The new ACR Guidelines for the Management of Gout include lifestyle changes as part of the non-pharmacologic approach for patients to follow. General health recommendations from the ACR are weight loss – especially for patients who are obese – exercise and an overall healthy diet that eliminates certain foods that are particularly high in purines or very likely to elevate UA levels.

Specifically, patients should avoid organ meats (liver, kidney, sweetbreads and tongue) and sweetened beverages with high-fructose corn syrup. Alcohol overuse – which for men is more than two alcoholic drinks per day and for women is more than one drink per day – should also be discouraged. In addition, there are several foods that should be limited: red meat such as beef, lamb or pork, and shellfish. The guidelines encourage the use of non-fat or low-fat dairy products. Dairy products not only provide a good source of protein, especially the non-fat or low-fat varieties, but also contain casein, which can help lower UA levels.

Patients should also stop tobacco use, if applicable, and stay well hydrated.

Barriers to Treatment

The most pervasive barrier standing between the proper management of gout arguably remains the ability of health care providers to correctly diagnose and appropriately treat the disease, and their success convincing patients to embrace a treatment regimen for life.

Also, distrust in the health care system and a propensity to bypass traditional therapies in favor of natural remedies can deter proper treatment among certain populations. Medical folklore is another issue; the mythology surrounding gout, patient profiles and treatment must be overcome.

The result of health care disparities in rural areas and other countries have contributed to increased prevalence in certain populations. This limits access to therapies, especially newer therapies, making it more difficult for patients to see a doctor and receive treatment.

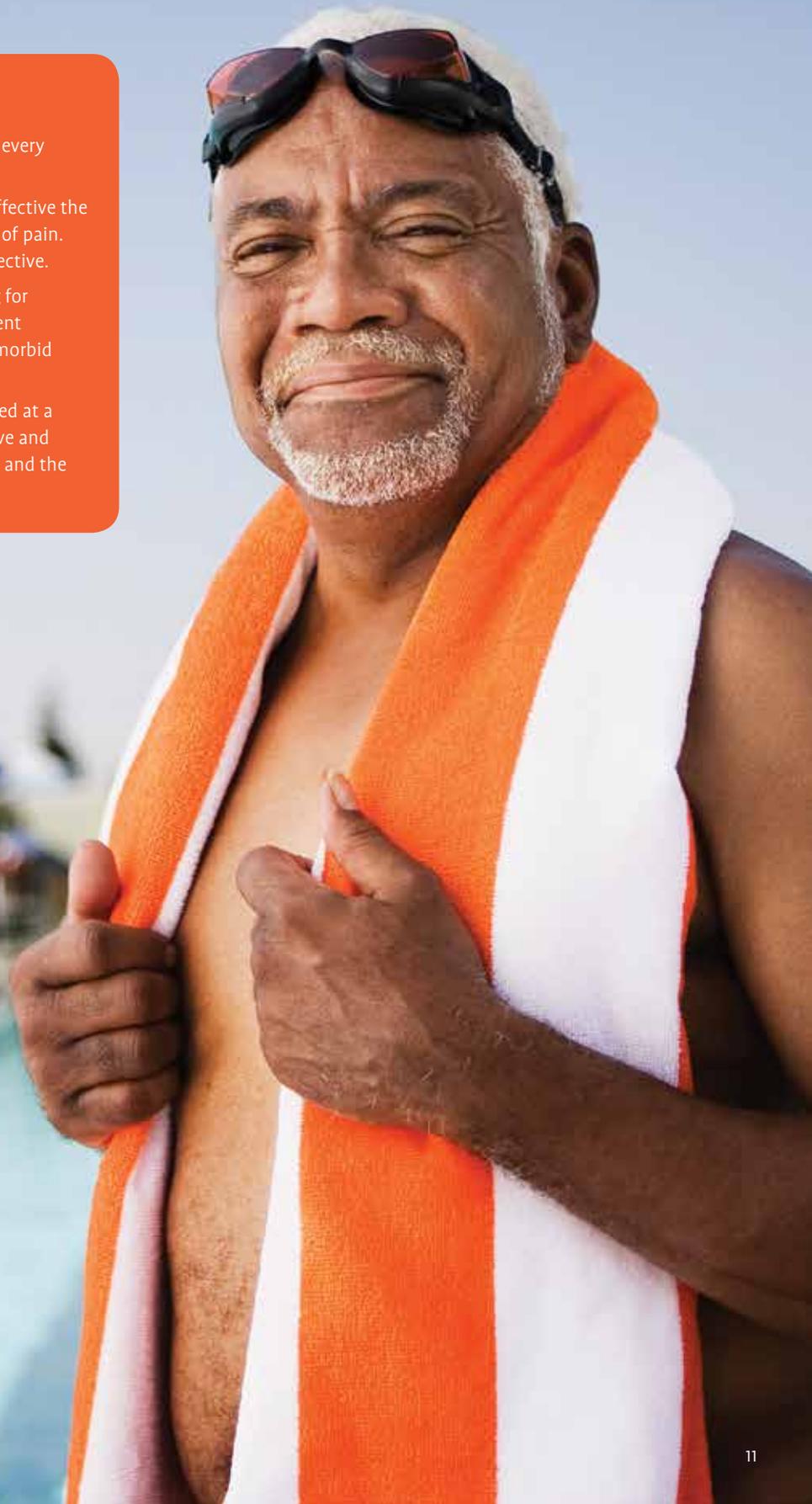
Tips for All Patients

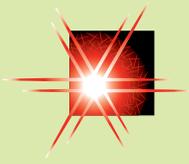
Clinicians should make sure gout patients understand the following:

1. Gout develops due to an accumulation of UA over time. By the time patients experience symptoms and see a doctor, accumulation of excess UA in their bodies has been going on for years – or even decades. Even if they begin UA-lowering therapy, it can take many years to achieve a UA level in the normal range.
2. Patients should always know their serum UA level, and it should always be below their target. At a minimum, this means below 6 mg/dL. If they already have evidence of tophi, then this target level should be 5 mg/dL, or even below 4 mg/dL. If their UA level is not within these targets, patients need to discuss the reason with their health care provider. Patients are not going to improve until they achieve the target level.
3. After initiating UA-lowering therapy, there may be a period of three to six months when patients could potentially have more flares, unless the clinician ensures that the patient is on suppressive, anti-inflammatory therapies. The ACR Guidelines recommend beginning either daily low-dose colchicine or low-dose NSAIDs two weeks prior to the initiation of any UA-lowering therapy. Caution patients that even though flares may become more frequent during the effort to lower their UA level, it is critical to stay on the treatment.
4. If a flare develops while on UA-lowering therapy, patients who have had previous flares and know which anti-inflammatory medication works for them should always have a filled prescription on hand. They should begin using it at the onset of symptoms.
5. Heat tends to worsen symptoms. Research suggests that using ice on the affected joints helps to decrease the pain for some patients. Immobilizing the joint can also help to minimize pain.
6. Frequent monitoring of UA level is important when initiating UA-lowering therapy and during the medication adjustment period. Once at their personal target, patients should have their UA level tested twice per year. This is important because changes in diet, medications or other diseases could cause a fluctuation in UA level.
7. Multicultural patients in groups with a high incidence of gout should avoid all lifestyle factors that contribute to the disease. This means being vigilant to stay close to their ideal body weight and eating a healthy diet. In particular, the consumption of alcohol (especially beer), fructose-sweetened beverages (including those with high-fructose corn syrup) and high-purine foods (such as red meats and shellfish) should be limited or avoided altogether.

Clinical Takeaways

- Emphasize diet and lifestyle changes for every patient at every office visit.
- Therapy for an acute flare will be more effective the sooner treatment begins after the onset of pain. After 48 hours, intervention will be ineffective.
- The choice of an anti-inflammatory drug for an acute flare depends on what the patient has responded to in the past and the comorbid diseases they may have.
- Anti-inflammatory therapy should be used at a dosage that is high enough to be effective and for as long as it takes for the flare to end and the symptoms to subside.





Gout and Comorbid Conditions

Gout is closely linked to a number of other serious health conditions. This makes prompt diagnosis of gout and ongoing management of comorbidities important. The most common comorbid diseases are coronary artery disease, hypertension, Type 2 diabetes, chronic kidney disease, hyperlipidemia and metabolic syndrome. All are associated with obesity and elevated UA. In fact, an obese patient is four times more likely to develop gout than someone with a normal body weight.

Heart Disease

Gout is often associated with cardiac problems including high blood pressure, coronary artery disease and congestive heart failure. Hyperuricemia is associated with a higher risk of death from these conditions. Gout may contribute to unhealthy cholesterol and lipid levels; conversely, patients with high lipid levels and high triglycerides commonly have gout.

Diabetes

Preliminary research suggests that insulin resistance may play a role in the development of gout and that hyperuricemia may worsen insulin resistance. Controlling blood sugar will help diabetic patients manage their UA level and make their gout easier to control. Poor blood circulation in the limbs may cause a buildup of UA in the joints that may contribute to diabetes in gout patients.

Kidney Disease

Kidney disease is common among patients with gout because kidney stones are often composed of UA. In fact, approximately one in five gout sufferers will develop kidney stones. For patients with kidney disease, it is important to monitor their kidney function throughout the treatment phase and avoid potentially dangerous anti-inflammatories.

Ethnicity and Comorbidities

The incidence of comorbid conditions that can impact gout also varies by ethnicity. For instance, obesity is a particular problem in both Hispanic and African-American populations. African Americans are also more likely to develop hypertension, while Hispanics are more likely to develop Type 2 diabetes. Treatment of these conditions and the fact that both can lead to kidney disease can worsen the course of gout in these patients.

Medications and Comorbidities

Managing medications for patients with comorbid diseases requires ongoing vigilance. Some of the more common treatments can increase UA levels and adversely affect gout. For example, treating patients who have high cholesterol with niacin to lower triglycerides can increase UA levels. Encourage these patients to gain control of their diet and use fenofibrate, which helps lower UA, instead of niacin. Some hypertension medicines, such as HCTZ, can significantly elevate UA levels. They can cause more frequent flares or make it more difficult to control UA levels. Switching to a different blood pressure medication may help resolve this problem.



Clinical Takeaways

- Gout patients frequently have serious comorbid conditions, requiring a holistic approach to treatment.
- Regular monitoring of UA level is especially important for patients with comorbidities.
- Some medications prescribed for comorbidities can increase UA levels and an alternative may be needed.

Further Reading: Seminal Research Studies on Gout Diagnosis and Management

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