Diagnosis and Management of Gout

Case Study and Commentary, Lan X. Chen, MD, PhD, and H. Ralph Schumacher Jr, MD

Abstract
Acute gouty arthritis is a common problem in primary care practice. Although clinical features such as the acute onset of dramatic arthritis in a toe or other lower extremity joint can be suggestive, a definitive diagnosis depends on identification of monosodium urate crystals on analysis of fluid from a joint or tophus. In this article, a patient with gout and renal insufficiency is presented to review the general management of gout and additional concerns raised by the renal disease. The most common approach to acute drug management involves use of nonsteroidal anti-inflammatory drugs, but with renal insufficiency, corticosteroids may be safer. The indications for urate-lowering therapy are frequent gout attacks (>3 per year), presence of tophaceous nodules, and history of nephrolithiasis. Patient education can increase compliance with therapy, resulting in fewer attacks.

Gout, or monosodium urate crystal deposition disease, is caused by supersaturation of extracellular fluid with urate. The prevalence of gout in the United States is reported to be 8.4 cases per 1000 individuals, but this estimate is based on patient self-reporting and may overestimate or underestimate true prevalence by up to twofold [1].

The clinical manifestations of gout include recurrent attacks of acute inflammatory arthritis, accumulation of urate crystals in the joints and periarticular tissue (ie, tophaceous deposits), and uric acid nephrolithiasis. Monosodium urate crystals present in synovial fluid are clearly involved in the pathogenesis of gout [2]. Urate crystals in synovial fluid can be identified in clinical practice by use of polarized light microscopy, providing the means for a rapid, definitive diagnosis. Gout is most often first suspected when presenting with dramatic acute monarthritis in a joint in the lower extremity.

Effective therapies are available to treat the acute inflammatory attacks of gout and to reverse and prevent the chronic destructive manifestations of urate crystal deposition. Although these treatments have favorably changed the course of gout in the great majority of patients, the disease still may be difficult to treat in noncompliant or poorly instructed patients [3,4] and in patients with comorbid medical conditions, particularly those with chronic renal insufficiency.

CASE STUDY
Initial Presentation
A 57-year-old man recently hospitalized for congestive heart failure presents to his primary care physician with acute onset of right big toe, right ankle, and left elbow pain with swelling, redness, and warmth.

History
The patient has a past medical history of diabetes mellitus, hypertension, hyperlipidemia, diabetic cardiomyopathy, and renal insufficiency secondary to diabetes mellitus. He has been treated with diuretics for his congestive heart failure over the previous week. He reports having had 1 previous episode of a very swollen painful ankle 1 year before. The episode lasted 5 days and was self-treated with rest and over-the-counter...
nonsteroidal anti-inflammatory drugs (NSAIDs). He denies fever or chills, rashes, pleurisy, or recent infections.

Medications include furosemide, aspirin, amlodipine, carvedilol, digoxin, atorvastatin, and insulin. He denies alcohol intake, smoking, or recreational drug use. He denies a family history of gout or autoimmune disease.

**Physical Examination**

The patient is a mildly obese middle-aged man. Vital signs include a temperature of 37.8°C, pulse of 92 bpm, blood pressure of 120/74 mm Hg, and respiratory rate of 18 breaths/min. He has no icterus. No ear tophaceous nodules are appreciated. There is no palpable lymphadenopathy at the neck, axillary, or inguinal areas. He has symmetrical chest expansion and respiratory effort with scattered rales at both basilar lung fields. He has no heart murmurs or rubs on cardiac auscultation. His abdomen is not tender, and he has no hepatosplenomegaly. The neurologic examination is nonfocal with normal proximal and distal muscle strength and no sensory defects. Skin examination does not reveal rash, ulcers, or nodules. On musculoskeletal examination, there is swelling, warmth, erythema, and pain affecting the right first metatarsophalangeal joint, right ankle, and left elbow with limited passive and active motion. He has small, granular, nontender deposits suggestive of tophi on both olecranon areas.

**Laboratory Testing**

White blood cell (WBC) count is 11,800/mm³ with 81% segmented neutrophils, 13% lymphocytes, 4% monocytes, and 2% band neutrophils. Hemoglobin is 11.2 g/dL, platelet count is 283,000/µL, serum creatinine is 2.5 mg/dL, and the erythrocyte sedimentation rate is 68 mm/hr. Urinalysis shows a protein concentration of 100 mg/dL and does not reveal WBCs, red blood cells, or casts. Serum uric acid is 11.8 mg/dL.

- **Which patients have an increased likelihood of developing gout?**

Gout most often affects middle-aged men who are obese, hypertensive, and frequent imbibers of alcohol. Other risk groups include patients with concurrent illnesses (eg, diabetes or chronic renal insufficiency), patients taking concurrent therapies that decrease uric acid excretion (diuretics, ethambutol), and those who have had an organ transplant and are on cyclosporine [4-6]. Transplant patients have an increased likelihood of developing gout because they often have antecedent medical conditions, such as renal insufficiency and heart failure, and are taking diuretics, in addition to taking cyclosporine for their transplants. Cyclosporine can lower the glomerular filtration rate (GFR), cause tubular damage, and impair fractional urate clearance, resulting in uric acid retention [7-9]. Common risk factors for development of gout and hyperuricemia are shown in Table. Risk factors for individual acute attacks include trauma or anything that suddenly changes uric acid levels, such as dieting, alcohol, surgery, or diuretics.

**Table. Some Common Risk Factors for Gout and Hyperuricemia**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Causal Mechanism</th>
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<tbody>
<tr>
<td>Male sex</td>
<td>Alcohol abuse</td>
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<tr>
<td>Postmenopausal status in women</td>
<td>Renal insufficiency</td>
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<tr>
<td>Obesity</td>
<td>Hypertension</td>
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<td>Metabolic syndrome (insulin resistance)</td>
<td>Dehydration</td>
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<tr>
<td>Diuretics</td>
<td>Lead poisoning</td>
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<tr>
<td>Ethambutol</td>
<td>Lymphoproliferative disorders</td>
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<td>Low-dose aspirin (&lt; 2.5 g)</td>
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<tr>
<td>Cyclosporine</td>
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</table>

- **How is gouty arthritis recognized and distinguished from other forms of arthritis?**

**Clinical Presentation and Diagnosis**

Gout most often will present to the primary care physician as an acute arthritis involving the first metatarsophalangeal joint or other lower extremity articulations. Clinical criteria for classification of acute gouty arthritis have been proposed [10]. Some of these clinical features suggestive of the diagnosis are the rapid progression of dramatic inflammation during the first day of the attack and cellulitis-like redness over the joint. The great majority of initial attacks are monarticular, but later bouts can involve multiple joints. Polyarticular gouty arthritis as seen in the case patient is uncommon as an initial manifestation of gout, occurring in less than 20% of cases, but tends to occur with increasing frequency in later recurrences. A polyarticular initial presentation of gouty arthritis may be more frequent in organ transplant recipients who are receiving cyclosporine and in patients with a myeloproliferative or lymphoproliferative disorder. Polyarticular symptoms are particularly common in patients with multiple recurrences, brief or absent symptom-free intervals, and tophaceous deposits. The polyarthritis in the case patient raises concern about other types of arthritis with involvement of multiple joints. The episodic nature in this patient and the asymmetry of joint involvement are evidence against rheumatoid arthritis. The history and physical examination were directed toward clues for reactive arthritis, spondylarthropathy, or collagen disease and no such clues were detected. Other crystal diseases could be considered and sought on synovial fluid examination.
Gouty arthritis can present a clinical picture indistinguishable from acute septic arthritis, including fever, leukocytosis, and an elevated erythrocyte sedimentation rate. Aspiration of synovial fluid from the affected joint and analysis of the fluid by Gram stain, culture, and polarized light microscopic examination distinguishes these disorders in most instances [11]. The presence of monosodium urate crystals in synovial fluid from the affected joint helps to confirm the diagnosis of gout. On polarized light microscopy, these crystals are needle-shaped and negatively birefringent. However, the presence of crystals does not totally exclude coexisting infection. Gram stain and cultures may be negative in septic arthritis if patients have received antibiotics before the aspiration was performed. Both gout and septic arthritis may have very elevated synovial fluid leukocyte counts and 90% or more polymorphonuclear neutrophils. Joint aspiration and synovial fluid analysis with polarized light examination is also helpful in differentiating acute gout from pseudogout (calcium pyrophosphate crystal deposition disease). In contrast, arthritis or periarthritis due to the deposition of basic calcium phosphate crystals usually cannot be diagnosed with certainty by polarized microscopy because the crystalline aggregates are below the resolution of standard light microscopy [12]. However, apatite crystals do clump together and may be seen as irregular, shiny, nonbirefringent chunks in joint fluid and are often associated with visible soft-tissue calcifications on radiographs [12,13].

Joint Aspiration and Fluid Analysis

A consulting rheumatologist performs a right ankle aspiration. Synovial fluid analysis shows a WBC count of 33,000/mm³ with 79% segmented neutrophils, 11% lymphocytes, and 10% monocytes. Polarized light microscopic examination reveals many needle-shaped, negatively birefringent crystals (monosodium urate); no positively birefringent, rod-like calcium pyrophosphate crystals are found. The synovial fluid culture is negative. The physician makes a diagnosis of polyarticular gout. Because crystals were found, it is not necessary to test for rheumatoid factor.

- What is the general approach to management of acute gouty attacks?

Acute Gouty Arthritis

Colchicine

Oral colchicine is a traditional treatment for acute gout but now is used less commonly. It can be effective if it is used within 48 hours of the onset of the acute attack [14]. Colchicine has been shown to be effective compared with placebo at reducing symptoms and resolving attacks when used in the traditional American regimen of 0.5 mg orally every hour until the patient experiences relief or toxicity (eg, nausea, vomiting, or diarrhea) or has taken a total of 8 pills [15]. However, many patients (perhaps 80%) experience the nausea, vomiting, diarrhea, and abdominal pain after oral administration before they experience full clinical improvement. Doses must be decreased in the presence of liver or kidney disease, but safe doses in these situations have not been studied. Alternative regimens such as 1 mg 3 times daily have been used but have not been studied. Oral colchicine may be given for acute gouty arthritis or for prophylaxis of acute gout in a patient with renal insufficiency, but the dose should be adjusted to the degree of the patients’ renal insufficiency. Because of decreased renal function and decreased clearance of colchicine [16,17], this drug may induce the abrupt or insidious onset of a neuromyopathy: Myalgias, paresthesias, or weakness may be the first and only indications of colchicine toxicity. If patients develop such symptoms, colchicine must be discontinued or the dose reduced while the symptoms and findings are evaluated [17].

Intravenous colchicine is limited by its small benefit-to-risk ratio. Life-threatening side effects can occur with intravenous administration of colchicine, such as bone marrow suppression, renal failure, hepatic necrosis, disseminated intravascular coagulation, seizures, and death [18]. Administration of intravenous colchicine should be restricted to hospitalized patients who cannot take oral medications or are unable to use NSAIDs. A safe dose is 1 to 2 mg in an established intravenous line. In addition, these patients must be supervised by physicians experienced in the use of colchicine by this route of administration. Long-standing orders for intravenous colchicine should not be written in order to prevent serious side effects; no additional oral colchicine should be given for 1 week after use of intravenous colchicine as the drug is retained in the cells for this amount of time.

NSAIDs

NSAIDs are frequently used in the treatment of acute gout. The therapeutic success of NSAID therapy in gout treatment may be determined by how soon therapy is initiated rather than by the specific agent used. Prompt institution of full anti-inflammatory doses such as indomethacin 150 to 200 mg per day is most useful if there are no contraindications to the NSAID. A randomized, double-blind study found intramuscular ketorolac and oral indomethacin to be similar in the relief of gouty pain in the emergency department [19]. In a double-blind study with 59 patients, the ketoprofen and indomethacin groups showed a significant improvement over a period of 7 days from baseline in all variables [20]. NSAIDs should be avoided in patients with active or recent peptic ulcer disease and in patients on anticoagulants or platelet aggregation therapy. The use of a cyclooxygenase-2 (COX-2) inhibitor for the treatment of gout has been studied. Etoricoxib, which is not
yet available in the United States, was shown to have efficacy equal to indomethacin 150 mg/day with less toxicity [21]. A trial of a COX-2 inhibitor may be appropriate in patients with acute gout, especially in those with peptic ulcer disease.

NSAIDs or COX-2 inhibitors should be used cautiously in patients with renal insufficiency. Both may exacerbate renal insufficiency and cause hyperkalemia by inducing hyporeninemic hypoaldosteronism through inhibition of intrarenal prostaglandin formation. On rare occasions, NSAIDs may cause interstitial nephritis or papillary necrosis. NSAIDs are also contraindicated in patients with active congestive heart failure and known sensitivity to NSAIDs.

Corticosteroids
Corticosteroids are usually reserved for patients in whom colchicine or NSAIDs are contraindicated or ineffective. For acute gout, 20- to 40-mg doses of prednisone are administered daily for 3 to 4 days and then gradually tapered over 1 to 2 weeks [22]. Equivalent intravenous methylprednisolone can be used in hospitalized patients in whom oral administration is problematic. In patients with diabetes or in those prone to hyperglycemia, blood glucose levels should be monitored. Oral agents or insulin may need to be supplemented. For monarticular or oligoarticular acute gout attacks, local articular depot steroid injection can be given to minimize systemic side effects of steroids. In transplant patients already on steroids, temporary increases in the prednisone dose to 30 to 40 mg/day may be effective in acute gouty episodes. This is especially true with polyarticular gout attacks. Adrenocorticotropic hormone, although currently difficult to obtain, can also be useful.

Treatment of Acute Attack

Because NSAIDs are relatively contraindicated due to the patient’s active congestive heart failure and because renal insufficiency limits the use of both NSAIDs and colchicine, the physician decides to treat the patient with prednisone. A dose of 20 mg orally twice daily is prescribed. By 24 hours, the patient reports that the pain has decreased, and by 3 days the swelling has reduced dramatically. The prednisone is tapered to 30 mg after 5 days then by 10 mg increments every 3 days until it is withdrawn. The patient has no change in blood pressure, shortness of breath, or elevated creatinine levels throughout this treatment. After 10 days, all pain and inflammation have resolved.

• What is the approach to treatment of gout after resolution of an acute attack?

Interval Gout

For interval gout patients—those who are asymptomatic after 1 or more episodes—it is important to avoid factors that can exacerbate the disease. Numerous conditions can precipitate acute attacks of gouty arthritis, including trauma, surgery, starvation, alcohol ingestion, dietary overindulgence, and ingestion of drugs that increase serum urate concentrations (eg, diuretics). Each of these circumstances may promote gouty attacks by causing changes in extracellular urate concentrations. In some cases, such as trauma, surgery, or intake of antihyperuricemic medications, gout can occur with normal or even very low serum urate concentrations at the time of the acute event as sudden changes in uric acid levels may release crystals from deposits into joint fluids. In addition, inflammation itself can lower the serum uric acid level. It may be necessary to remeasure serum uric acid after the attack has resolved to establish the patient’s true uric acid level.

Prophylactic pharmacologic therapy should be given to patients starting a urate-lowering drug. Treatment includes small oral daily doses of colchicine, such as 0.6 mg once daily. A single, double-blind, placebo-controlled study demonstrated the prophylactic efficacy of one 0.5-mg colchicine tablet twice daily [23]. Prophylactic dosing of colchicine reduced the frequency of attacks by 75% to 85%. NSAIDs are also used by some for prophylaxis, but there are no studies on their value in this role.

Initiation of Prophylactic Therapy

The patient is given colchicine 0.6 mg orally 3 times per week as the prednisone is withdrawn for prevention of acute gout attacks. Although patients with normal renal function often use 0.6 to 1.2 mg/day, the colchicine dose is adjusted downward for his renal insufficiency. He will be monitored for signs of toxicity, including neurologic and muscle signs.

• Which patients should receiveurate-lowering drugs?

The indications for urate-lowering agents are frequent gout attacks (more than 3 attacks per year, based on a meta-analysis [24]), presence of tophaceous nodules, and a history of nephrolithiasis. When initiating therapy, an effort should be made to identify correctable factors that might be contributing to hyperuricemia, such as diet, alcohol use, obesity, and drugs. Compliance with therapy during long asymptomatic periods can be difficult [25]. Time used by physicians to explain to patients the need for maintaining urate-lowering therapy and providing educational material such as that available from the Arthritis Foundation (Atlanta, GA) is time well
spent. One study using a nurse educator showed that patient education can result in fewer attacks and lower serum uric acid levels [25]. Abuse of alcohol, in addition to its effects on uric acid handling, can also interfere with compliance.

- Which agents are used to lower serum urate levels?

**Xanthine Oxidase Inhibitors**

There are 2 main classes of urate-lowering drugs: xanthine oxidase blockers and uricosuric agents. Xanthine oxidase inhibitors, such as allopurinol and its active metabolite oxipurinol, decrease uric acid production by inhibiting xanthine oxidase. Allopurinol is the most commonly used urate-lowering drug [26,27]. The use of allopurinol is not prohibited by renal insufficiency, but the half-life of its metabolite oxipurinol is prolonged in renal insufficiency, requiring lower starting doses and close monitoring of its side effects. Doses of allopurinol are usually started at 100 mg/day and can be gradually increased to as much as 800 mg/day if needed and tolerated. Serum urate levels clearly begin to decrease within 2 days after initiation of allopurinol and reach stable levels in 1 week. One should monitor patients with measurement of serum uric acid levels once a month to ensure adequate lowering of uric acid. The uric acid level recommended to dissolve tophi and prevent gout attacks is below 6.0 mg/dL [28–30]. Allopurinol may not induce adequately low serum urate levels early in the course of therapy of patients with extensive tophaceous deposits. However, true refractoriness to the drug is uncommon. In most cases, refractoriness to allopurinol reflects a failure of patient compliance, which often can be due partly to a failure of physician-patient communication [25,27].

The therapeutic effectiveness of allopurinol has been documented for more than 30 years, but side effects and adverse reactions, sometimes severe, have been encountered. Allopurinol can precipitate acute gouty arthritis (especially if colchicine prophylaxis has been omitted) and can induce rash, leukopenia or thrombocytopenia, diarrhea, drug fever, gastrointestinal intolerance, vasculitis, and interstitial nephritis. Allopurinol also has caused a hypersensitivity syndrome, consisting of an erythematous skin rash, fever, hepatitis, eosinophilia, and renal failure; this is a rare but potentially life-threatening reaction [31]. Allopurinol is also associated with important drug interactions, including potentiation of the immunosuppressive and cytolytic effects of azathioprine and 6-mercaptopurine, bone marrow suppression in patients receiving alkylating agents such as cyclophosphamide [32], and an increase in the likelihood of an ampicillin-induced skin rash [33].

Asymptomatic hyperuricemia generally should not be treated. The risk of reactions to allopurinol has been estimated to be greater than the risk of an attack of gout. However, asymptomatic hyperuricemic patients with 24-hr urinary uric acids levels greater than 1 g are sometimes treated with allopurinol to decrease the risk of stones. There has been speculation that hyperuricemia might actually worsen hypertension [34]. If an association between high uric acid levels and hypertension were proven, it might be a rationale for urate-lowering treatment in asymptomatic patients.

Allpurinol can be given in patients with renal insufficiency and interval gout; however, the initial dose should be adjusted down based on the creatinine level [35,36]. The dose may need to be further adjusted in transplant patients being treated with azathioprine, who are typically managed by specialists. Allopurinol interferes with the metabolism of 6-mercaptopurine, the active metabolite of azathioprine, which in part involves xanthine oxidase [37]. Thus, 6-mercaptopurine accumulation and possibly severe bone marrow toxicity may ensue [38]. If the patient has severe gout and allopurinol must be used, azathioprine can also be changed to mycophenolate, which does not interact with allopurinol.

**Uricosuric Drugs**

Probenecid and sulfinpyrazone are the uricosuric drugs used in United States, while other agents such as benzbromarone have been used in European countries. Uricosurics have fewer life-threatening risks and should be the safest drugs to use in patients with normal renal function who are not overexcretors of uric acid (24-hr levels > 800 mg). Probenecid and sulfinpyrazone are likely to be ineffective in patients with renal insufficiency (serum creatinine > 2.0 mg/dL), while benzbromarone is an effective uricosuric agent in patients with creatinine clearance as low as 20 mL/min [39]. Measurement of 24-hr urine excretion of uric acid should be performed before institution of a uricosuric. In general, a uricosuric would not be used if the 24-hr urine uric acid level were over 800 mg as these drugs act by increasing urate excretion and this would further raise the urate concentration in urine and increase the risk of stones. The risk of renal stones can be decreased by maintaining urine volume with daily intake of at least 8 glasses of water. Uricosuric agents may be used for the prevention of gout in transplant patients if renal function is adequate and there is no history of renal calculi. However, if using sulfinpyrazone, levels of cyclosporine must be closely monitored and the dose adjusted accordingly because sulfinpyrazone lowers trough cyclosporine concentrations.

The angiotensin II receptor antagonist losartan also has uricosuric effects [40–42]. In a double-blind crossover study of 10 hypertensive heart transplant recipients, individuals were randomized to losartan (50 mg once daily) and enalapril (20 mg once daily) [43]. Significantly lower levels of
plasma uric acid were observed with losartan (P < 0.05). When given with hydrochlorothiazide, losartan may also blunt hydrochlorothiazide’s effect of increasing the serum uric acid level and potentiate the antihypertensive effect of the diuretic [40–42].

**Addition of Urate-Lowering Agent**

Because the case patient already has tophi, urate-lowering therapy is needed. He is started on 100 mg allopurinol once daily 6 weeks after the acute attack, and his allopurinol dose is eventually increased to 300 mg once daily in order to lower his uric acid level to below 6.0 mg/dL.

**References**


30. Li-Yu J, Clayburne G, Sieck M, et al. Treatment of chronic urate-lowering therapy is needed. He is started on 100 mg allopurinol once daily 6 weeks after the acute attack, and his allopurinol dose is eventually increased to 300 mg once daily in order to lower his uric acid level to below 6.0 mg/dL.
MGAWMENAGEMENT OF GOUT


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### Part 1. Please respond to each statement.

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<tr>
<th>Statement</th>
<th>Strongly Agree</th>
<th>Strongly Disagree</th>
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<tr>
<td>I was provided with new information pertinent to my practice.</td>
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<td>This article will help with clinical decision making.</td>
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<td>The case is communicated in a manner that kept my interest.</td>
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<td>My attitude about this topic changed in some way.</td>
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- see no need to change my practice.
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