Acute gouty arthritis is frequently misdiagnosed or diagnosed late in its clinical course, and therapy is often suboptimal. Because the treatment of gout as a chronic, progressive disease has not been standardized, optimal disease management remains a challenge.

My goal here is to help you improve your ability to accurately diagnose gout. I discuss when and how to make a presumptive diagnosis, indications for joint aspiration, and which diagnoses to consider in the differential. In a second article in a coming issue, I will outline the keys to effective treatment.

**CLINICAL EVALUATION**

Diagnosis guidelines published by the American College of Rheumatology (ACR) in 1977 are difficult to follow and thus are seldom used. In 2006 a consensus panel of experts from the European League Against Rheumatism (EULAR) published clearer key points for diagnosing gout (Table). The EULAR recommendations addressed the shortcomings of the ACR criteria and simplified the definition of gout (acute onset of severe pain in a joint that peaks at 6 to 12 hours, with overlying erythema and hyperuricemia). Nonetheless, some of the recommendations are challenging to apply in practice, and you must ultimately rely on clinical observations and judgments.

**Acute gout.** The overall prevalence of gout among men is 6/1000 and among women is 1/1000. The classic presentation of an initial flare is acute inflammatory monoarthritis in a middle-aged man. About 90% of initial attacks are monoarticular, and 50% occur in the first metatarsophalangeal joint of the great toe. Other sites commonly affected are the instep/forefoot, ankle, knee, wrist, and fingers.

**Symptoms.** Severe pain, redness, and swelling develop rapidly, and inflammation peaks within 1 day (Figure 1). When the attack occurs in the toe or foot, patients often cannot wear socks, and even the mere touch of a bed sheet may cause extreme pain. Systemic symptoms are usually absent, but a low-grade fever may be present. The initial attack is generally self-limited, lasting a few days to weeks.

**Risk factors.** Patients with a history of such attacks may recall that they were triggered by trauma or excesses of alcohol consumption or dietary intake. Risk factors for gout include:

- Family history.
- Older age.
- Renal insufficiency.
- Use of medications that reduce urate excretion, such as thiazide diuretics, cyclosporine, and low-dose salicylates.
- High intake of foods that increase urate production (beer, seafood, red meat, high-fructose beverages).
- Comorbidities such as obesity and metabolic syndrome.

Sample questions to elicit symptoms and risk factors are listed in the Box on page 1014.
Intercritical gout. After an acute gouty arthritis attack, patients usually enter an asymptomatic phase (intercritical gout). Joint damage may progress during these periods because monosodium urate crystals remain in the joints and provoke a continuing inflammatory reaction. Intercritical periods decrease over time as the disease advances and flares occur more frequently.

Chronic gout. Chronic gouty arthritis develops in patients who have had acute attacks of gout for years. Tophi, the hallmark of chronic gout, result from the subcutaneous deposition of sodium urate. They typically appear after 10 to 12 years of poorly treated chronic gout. The sites most commonly affected are the Achilles tendon, the prepatellar bursa, and the helix of the ear. Tophi may also occur over Heberden nodes and in the finger pads, particularly in elderly women. Although tophi are generally painless, they may ulcerate, become infected, and extrude chalky white material. They are also worrisome because the persistent crystal-induced inflammatory process can damage joints.

Atypical presentations. Gout in women occurs predominantly in those who are older than 60 years. Older women may exhibit atypical features, such as manifestations in the hands, polyarticular involvement, and nodules that resemble rheumatoid nodules. In elderly patients, gout may occur without swelling or inflammation.

Physical examination. Pay close attention to the joints, but also examine other areas of the body, particularly in patients with a history suggestive of gout. Examine the ears for tophi. Remove a patient’s shoes and socks and roll up pant legs to better see the feet and ankles. Expose the extensor surface of the forearms and inspect them for nodules.

LABORATORY AND IMAGING STUDIES
Proceed to a laboratory workup after the history taking and physical examination. In many cases, a patient with hyperuricemia and acute arthritis in a commonly affected joint can be treated empirically for acute gout.

Serum urate measurement. Because hyperuricemia is the underlying cause of gout, measurement of the serum urate level is warranted in patients at risk for gout or with suspected gout. The serum urate level may be normal during an acute attack; thus, the optimal time for measurement is 2 weeks after a flare resolves. Hyperuricemia is often associated with such comorbid disorders as diabetes, dyslipidemia, hypertension, and obesity. It may be good practice to measure serum urate in these patients and ask them about joint pain.

Synovial fluid analysis. The gold standard for a definitive diagnosis of gout is the detection of urate crystals in synovial fluid. However, many primary care physicians are reluctant to perform the procedure because of time constraints, inexperience with the technique, or lack of access to polarizing microscopes for analysis. A reasonably accurate diagnosis of gout can be made without joint aspiration when typical symptoms are evident and serum urate levels are elevated. Some factors, however, exclude a presumptive diagnosis of gout: if the patient has a high fever or does not respond to empiric
therapy, refer to the emergency department or a rheumatologist for joint aspiration.

**Radiographs.** In most patients with acute gout, radiographs appear normal and may show only nonspecific soft tissue swelling. In patients with chronic tophaceous gout, radiographs may reveal punched-out joint erosions with well-defined sclerotic margins and an edge protruding over the erosion.

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**Key Questions for Patients With Suspected Gout**

- Is this the first time you have experienced this type of pain? How quickly did it begin and how long has it lasted?
- Which joints are affected (fingers, great toes, etc)? Do they become red, swollen, and painful?
- Have you ever received a diagnosis of gout?
- Is there a history of gout in your family? Is there a history of rheumatoid arthritis or other arthritic conditions?
- Have you ever received a diagnosis of renal insufficiency or been told that your kidneys are not working properly?
- Have you been told you have other diseases, such as metabolic syndrome, hypertension, or diabetes?
- Do you take thiazide diuretics, sometimes called “water pills”?
- Have you had any recent tests that included the injection of dye?
- Have you had any recent infections? If so, please describe them.
- Have you ever been told your serum urate level was high?
- What kind of diet do you usually eat?
- How often do you drink beer, colas, or other sugary drinks?
- How often do you eat canned fruits and juices?
- How often do you eat seafood or red meat?
- Do you consider yourself overweight? Have you always been overweight? If not, how old were you when you became overweight?

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**DIFFERENTIAL DIAGNOSIS**

Gout is sometimes confused with other conditions that present with similar signs and symptoms.

**Rheumatoid arthritis.** Gout is usually monoarticular, and tophi generally occur in the olecranon bursa; in contrast, rheumatoid arthritis (RA) is typically polyarticular and rheumatoid nodules occur more commonly on the extensor surface of the forearm. As gout progresses, however, it can become polyarticular, with symmetric small joint involvement and tophaceous deposits resembling rheumatoid nodules on the extensor tendon surfaces. Up to 30% of patients with tophaceous gout also have low titers of rheumatoid factor, adding to the diagnostic confusion. Moreover, hyperuricemia may be present in patients with RA who do not have gout.

**Pseudogout.** Patients with pseudogout are usually older than 60 years and have no family history of gout and no personal history of hyperuricemia with gouty attacks. Both sexes are affected equally. The patient may have had recent trauma to the affected joint, or an infection that was associated with dehydration. The upper extremities are affected more often than the lower extremities, and there may be pain in several joints and fever. Suggestive features also include soft tissue swelling and radiographic evidence of thick linear punctate meniscal calcifications or thin curvilinear densities with superimposed osteoarthritis.

**Septic arthritis.** This serious disease can cause rapid joint destruction, and it is associated with significant morbidity and mortality. Suspected septic arthritis calls for joint aspiration for cultures...
and Gram staining of the synovial fluid.\textsuperscript{2} The rigors, shaking chills, and high fever associated with septic arthritis are usually absent in patients with gout.\textsuperscript{3} A low-grade fever suggests an inflammatory process rather than septic arthritis. However, patients who take immunosuppressant drugs or glucocorticoids will have a suppressed fever response. Always suspect septic arthritis in patients with joint pain who have a high fever or who are at high risk for infection (eg, because of dialysis or immunocompromised status).

Septic arthritis may affect any joint, but most commonly the knee is involved. Joint effusion may be present. In early septic arthritis, radiographic findings are often normal.\textsuperscript{5} The white blood cell count (greater than 50,000/μL) is much more elevated than it is in gout (2000 to 50,000/μL).\textsuperscript{5}

If you suspect septic arthritis, consult immediately with an infectious diseases specialist or a rheumatologist who can perform joint aspiration, make the appropriate diagnosis based on synovial fluid examination, and admit the patient to the hospital for intravenous antibiotic therapy if infection is found.

References:

REFERENCES:

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