Acute Monoarthritis: Diagnosis in Adults

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Acute monoarthritis can be the initial manifestation of many joint disorders. The most common diagnoses in the primary care setting are osteoarthritis, gout, and trauma. It is important to understand the prevalence of specific etiologies and to use the appropriate diagnostic modalities. A delay in diagnosis and treatment, particularly in septic arthritis, can have catastrophic results including sepsis, bacteremia, joint destruction, or death. The history and physical examination can help guide the use of laboratory and imaging studies. The presence of focal bone pain or recent trauma requires radiography of the affected joint to rule out metabolic bone disease, tumor, or fracture. If there is a joint effusion in the absence of trauma or recent surgery, and signs of infection (e.g., fever, erythema, warmth) are present, subsequent arthrocentesis should be performed. Inflammatory synovial fluid containing monosodium urate crystals indicates a high probability of gout. Noninflammatory synovial fluid suggests osteoarthritis or internal derangement. Pitfalls in the diagnosis and early treatment of acute monoarthritis include failure to perform arthrocentesis, administering antibiotics before aspirating the joint when septic arthritis is suspected (or failing to start antibiotics after aspiration), and starting treatment based solely on laboratory data, such as an elevated uric acid level.
(*Am Fam Physician*. 2016;94(10):810-816.

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Author disclosure: No relevant financial affiliations.

Monoarthritis refers to the clinical presentation of pain or swelling in a single joint.[1] The diagnosis can pose a considerable challenge in the primary care setting because the pain may be limited to the joint, or it may represent early manifestation of a systemic disease.[2] Understanding the clinical clues associated with potential diagnoses and using an evidence-based, systematic clinical approach are of utmost importance.[3] A delay in diagnosis and treatment, particularly with septic arthritis, can have catastrophic results including sepsis, bacteremia, joint destruction, or death.[3-5] Pitfalls in the diagnosis and early treatment of acute monoarthritis include failure to perform arthrocentesis, administering antibiotics before aspirating the joint when septic arthritis is suspected (or failing to start antibiotics after aspiration), and starting treatment based solely on laboratory data, such as an elevated uric acid level.[3-5]

Etiology of Acute Monoarthritis

Any condition that may cause joint pathology can initially present as monoarthritis, resulting in a broad differential diagnosis[1] *(Table 1[6])*. Because of this, monoarthritis has no unifying etiology. The most common diagnoses in the primary care setting are osteoarthritis, gout, and trauma.[3]

Symptoms consistent with osteoarthritis include pain that tends to worsen with activity, morning stiffness lasting less than 30 minutes, and asymmetric joint pain.[7] The most commonly affected joints are the hands, knees, hips, and spine.[7] Although osteoarthritis often follows an insidious course, acute flare-ups are common and can be mistaken for other etiologies. The presence of focal bone pain or recent trauma requires radiography of the affected joint to rule out metabolic bone disease, tumor, or fracture.[3,4]

Gout is a common disorder with a 3% prevalence worldwide. It accounts for more than 7 million ambulatory visits in the United States annually.[8,9] Crystal-induced arthritis presents as a rapidly developing monoarthritis with swelling and erythema, and most commonly involves the first metatarsophalangeal joint.[8] Over time, the joint space can be irreversibly damaged with tophi formation.[8,10] The presence of monosodium urate crystals indicates gout; these crystals are identified by their needle-like appearance and strong negative birefringence[3,8] *(Figure 1[6])*. Calcium pyrophosphate dihydrate crystals are polymorphic, weakly positive under birefringent microscopy, and their presence indicates pseudogout.[3] Other crystal-induced arthritis etiologies include calcium oxalate and hydroxyapatite.[1]

Table 1. Causes of Acute Monoarthritis

Common	**Less common** *(continued)*
Avascular necrosis	Hemoglobinopathies
Crystals	Juvenile rheumatoid arthritis
Calcium oxalate	Loose body
Calcium pyrophosphate dihydrate (pseudogout)	Psoriatic arthritis
Hydroxyapatite	Reactive arthritis
Monosodium urate (gout)	Rheumatoid arthritis
Hemarthrosis	Sarcoidosis
Infectious arthritis	Systemic lupus erythematosus
Bacteria	**Rare**
Fungi	Amyloidosis
Lyme disease	Behçet syndrome
Mycobacteria	Familial Mediterranean fever
Virus	Foreign-body synovitis
Internal derangement	Hypertrophic pulmonary osteoarthropathy
Osteoarthritis	Intermittent hydrarthrosis
Osteomyelitis	Pigmented villonodular synovitis
Overuse	Relapsing polychondritis
Trauma	Synovial metastasis
Less common	Synovioma
Ankylosing spondylitis	Systemic onset juvenile idiopathic arthritis (Still disease)
Bone malignancies	Vasculitic syndromes
Bowel disease–associated arthritis	

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Infection is a common etiology of joint pain. When infection is suspected in the presence of a joint effusion or inflammation, arthrocentesis should be performed, in addition to further laboratory testing as indicated *(Figure 2[6])*. (A video of arthrocentesis of the knee is available at https://www.youtube.com/watch?v=fZ2dcZhoGP8.) Gonococcal arthritis is the most common type of nontraumatic acute monoarthritis in young, sexually active persons in the United States.[11]

Septic arthritis is a key consideration in adults presenting with acute monoarthritis, particularly in the presence of joint pain, erythema, warmth, and immobility.[12] The most important risk factors for septic arthritis are a prosthetic joint, skin infection, joint surgery, rheumatoid arthritis, age older than 80 years, diabetes mellitus, and renal disease.[11] One study found that among persons presenting with acute joint pain and a predisposing condition, 10% had septic arthritis.[4] When septic arthritis is suspected, it is important to begin empiric antibiotics immediately following arthrocentesis, because failure to initiate prompt antibiotic therapy can lead to subchondral bone loss and permanent joint dysfunction.[5,11,12] The most common route of entry into the joint is hematogenous spread during bacteremia[4,12-16]; therefore, isolation of the causative agent through synovial fluid culture is essential for the diagnosis and guidance of antibiotic therapy.[12]

Less common causes of monoarthritis include systemic diseases such as spondyloarthropathies (e.g., psoriatic arthritis, reactive arthritis, ankylosing spondylitis), sarcoidosis, Behçet syndrome, systemic lupus erythematosus, and rheumatoid arthritis.[1] Rheumatic diseases and corticosteroid use can cause avascular necrosis of the bone.[1]

Diagnosing Acute Monoarthritis

The diagnosis of acute monoarthritis begins with a comprehensive history and physical examination to reveal potential diagnostic clues[3,6,17] *(Table 2[1,3,6,12,18,19])*. Key elements of the patient history include a review of systems, age, previous joint disease, recent trauma, medication use, family history of gout, concurrent illness, sexual history, diet, travel history, tick bites, alcohol use, intravenous drug use, and an occupational assessment.[3,6,17]

Symptoms that worsen with activity and improve with rest suggest a mechanical process, whereas symptoms with an inflammatory process often worsen with rest and present with morning stiffness.[1] Osteoarthritis often starts as mild joint inflammation that may initially arouse suspicion for new-onset inflammatory diseases,

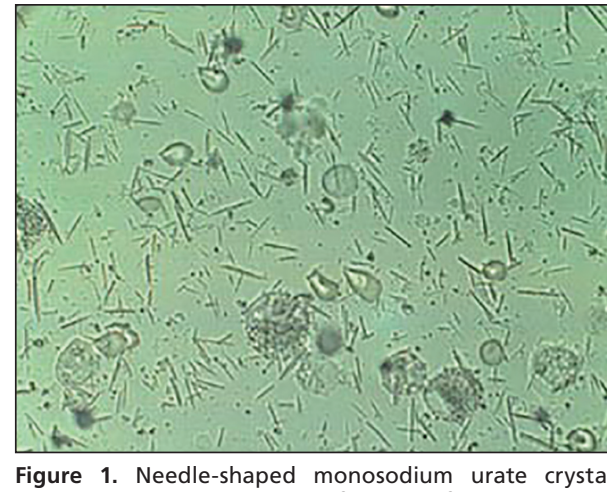

Figure 1. Needle-shaped monosodium urate crystals visible with light microscopy of synovial fluid in a patient with gout.

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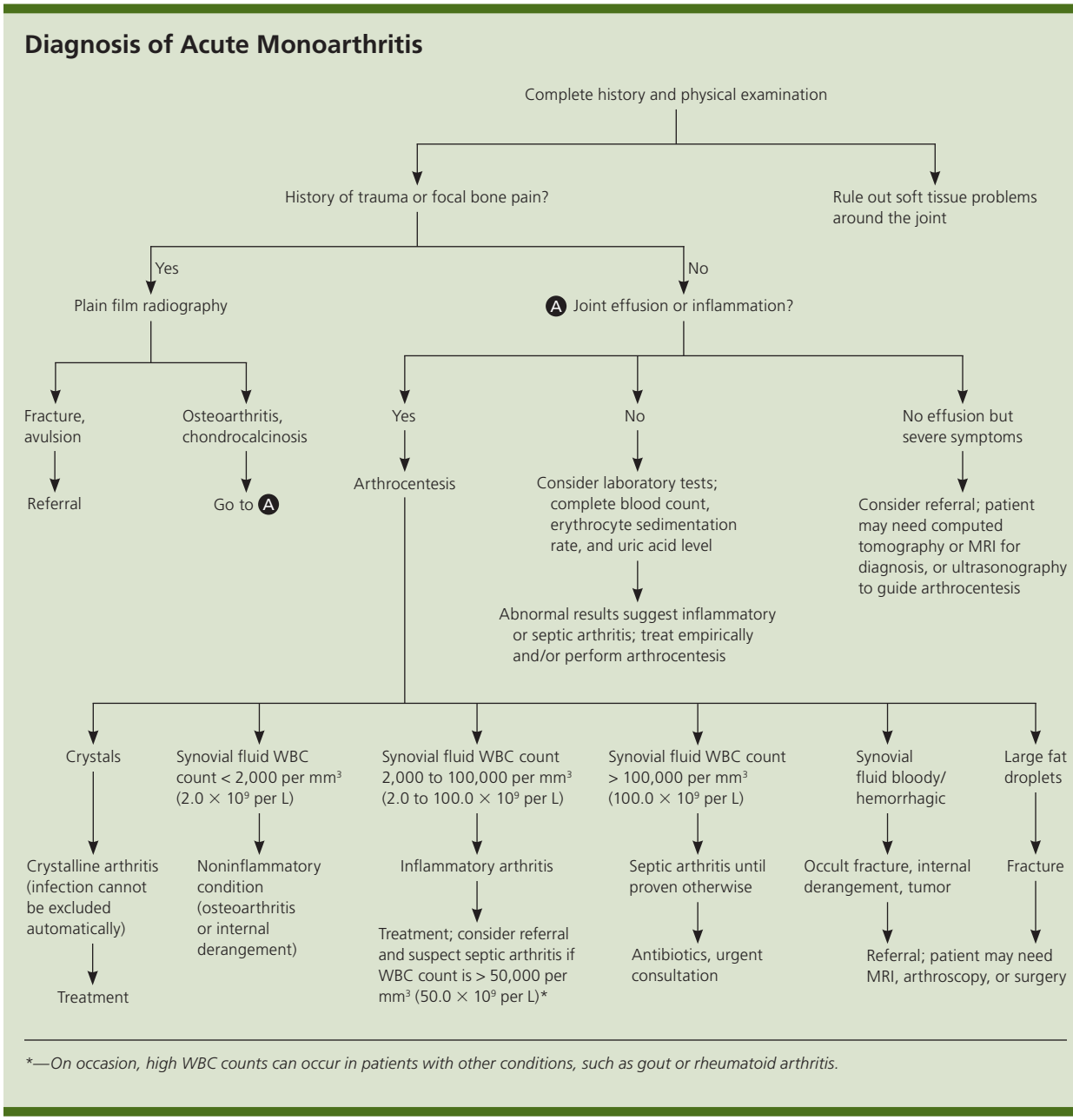

Figure 2. Algorithm for diagnosing acute monoarthritis. (MRI = magnetic resonance imaging; WBC = white blood cell.)

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such as rheumatoid arthritis.[20] Morning stiffness and its duration in the affected joint, pain with activity or rest, recent history of trauma, history of previous joint symptoms, and family history of joint inflammation are all important factors that can help differentiate etiologies.[12] Osteoarthritis typically worsens with activity, particularly after a period of rest (gelling phenomenon).[18] Morning stiffness from osteoarthritis usually lasts for a shorter duration than that of rheumatoid arthritis, which typically lasts 45 minutes or more.[7]

A gout attack typically begins at night and peaks within 24 hours, causing pain, swelling, and erythema.[20] Common clues from the patient history include obesity, a high-calorie diet, alcohol intake, and the use of loop and thiazide diuretics.[8] Trauma may also precipitate an acute gout flare-up,[8,21] and the presentation can closely resemble septic arthritis.[20]

Determining whether a condition is truly monoarticular can prove beneficial, because prodromal arthralgias can suggest infection.[1] Gonococcal arthritis is often preceded by migratory arthritis and tenosynovitis before settling in a primary joint.[1] Conducting a sexual history is imperative, as is documenting any urinary problems, purulent discharge from the urethra, or other signs of

Table 2. Diagnostic Clues in Patients Presenting with Monoarthritis

Clues from history and physical examination	*Diagnoses to consider*	*Evaluation*
Active range of motion restricted more than passive range of motion	Periarticular pathology	Radiography and/or MRI if indicated
Back pain, eye inflammation	Ankylosing spondylitis	*HLA-B27* testing, radiography
Coagulopathy, use of anticoagulants	Hemarthrosis	CBC, ESR, arthrocentesis to confirm diagnosis and rule out infection
Diuretic medication, presence of tophi, renal stones	Gout	CBC, ESR, uric acid level, arthrocentesis with evaluation for crystals
Hilar adenopathy, erythema nodosum	Sarcoidosis	CBC, ESR, ACE level, chest radiography, pulmonary referral
Immunosuppression and/or intravenous drug abuse	Septic arthritis	CBC, ESR, arthrocentesis for cell count and cultures
Insidious onset of pain and swelling over days to weeks	Indolent infection, osteoarthritis, infiltrative disease, tumor	CBC, ESR, arthrocentesis for cell count and cultures, radiography and possible MRI if tumor is a consideration
Maximum pain at limits of range of motion (i.e., stress pain)	Osteoarthritis	Radiography
Normal joint examination	Referred pain	Consider alternative diagnosis
Onset of pain and swelling over several hours or one to two days	Infection, crystal deposition disease, other inflammatory arthritic condition	CBC, ESR, uric acid level, arthrocentesis with evaluation for crystals
Pain elicited by joint movements against resistance only	Tendinitis, bursitis	MRI or ultrasonography if diagnosis in question
Previous acute attacks in any joint with spontaneous resolution	Crystal deposition disease, other inflammatory arthritic condition	CBC, ESR, uric acid level, arthrocentesis with evaluation for crystals
Prolonged course of corticosteroid therapy	Infection, avascular necrosis	CBC, ESR, arthrocentesis for cell count and cultures if concern for infection, radiography and possible MRI if considering avascular necrosis
Psoriatic skin plaques, nail pitting, dactylitis	Psoriatic arthritis	CBC, ESR, arthrocentesis, *HLA-B27* testing, ANA testing; none of these tests are diagnostic but can exclude other conditions
Restricted active and passive range of motion	Intra-articular pathology	Radiography and/or MRI if indicated
Sudden onset of pain in seconds or minutes	Fracture, internal derangement, trauma, loose body	Radiography
Urethritis, conjunctivitis, diarrhea, rash, sacroiliitis	Reactive arthritis	CBC; ESR; arthrocentesis; *HLA-B27* testing; urine PCR testing for *Chlamydia trachomatis*; stool cultures for *Salmonella, Shigella,* and *Campylobacter*
Young adult, migratory polyarthralgias, tendons inflamed	Gonococcal arthritis	Urine PCR testing, blood culture, and synovial fluid analysis for *Neisseria gonorrhoeae*

ACE = angiotensin-converting enzyme; ANA = antinuclear antibodies; CBC = complete blood count; ESR = erythrocyte sedimentation rate; MRI = magnetic resonance imaging; PCR = polymerase chain reaction.

Adapted with permission from Siva C, Velazquez C, Mody A, Brasington R. Diagnosing acute monoarthritis in adults: a practical approach for the family physician. Am Fam Physician. *2003;68(1):86, with additional information from references 1, 3, 12, 18, and 19.*

infection, such as pharyngitis.[11,12] Risk factors such as intravenous drug use, tick bites, and travel history can lead to a diagnosis of infectious or reactive arthritis.[1]

Axial skeleton inflammatory arthritis (e.g., sacroiliitis) in addition to symptoms in a single peripheral joint suggest a spondyloarthropathy.[1] Spondyloarthropathies can often present as monoarthritis and progress from joint to joint in a migratory or additive pattern.[20] It is important to ask patients about other symptoms, such as enthesopathy (tenderness at the muscle or fascia attachment sites) and dactylitis (sausage-like swelling of fingers or toes), because these are common.[20] Some patients also present with ocular inflammation (uveitis) and urethritis (Reiter syndrome).[20] A history of skin conditions and a positive family history of inflammatory arthritis suggest psoriatic arthritis, which can present as monoarthritis in the early stages.[20]

Physical Examination

The physical examination should focus on the involved and contralateral joints, the surrounding area, possible systemic manifestations, or polyarticular involvement.[3,6,17] The first step is to confirm that the joint pain is truly localized and not a periarticular process, such as tendinitis, bursitis, or cellulitis.[1] The presence of a joint effusion signifies intra-articular pathology, with patients typically reporting painful limitation of active and passive joint motion.[1]

A diagnosis of osteoarthritis can often be based on the history and physical examination.[3] The characteristic presentation includes painful and limited range of motion, crepitus, effusions, instability, or deformities.[18] Heberden and Bouchard nodes are pathognomonic for osteoarthritis. They result from hard, bony thickening that gradually forms around the distal and proximal interphalangeal joints of the fingers, respectively.[20] The first carpometacarpal joint is one of the most common sites of osteoarthritis.[20]

Inflammatory arthritis elicits painful range of motion and erythema that is typically confined to the affected joint. Gout can present with intense erythema of the skin. It is often confused with cellulitis because this finding may extend past the joint margin.[1,8]

There are many superficial examination findings that can suggest specific diagnoses. Subcutaneous nodules (tophi) and podagra (gouty arthritis of the first metatarsophalangeal joint) are highly specific for gout.[20] Erythema nodosum may be a manifestation of sarcoidosis or inflammatory bowel disease; psoriatic skin plaques are associated with psoriatic arthritis; and oral ulcers can indicate reactive arthritis or Behçet syndrome.[1]

Septic arthritis is most likely to seed within a larger joint,[1,22] and to be accompanied by erythema, warmth, and immobility.[12] Although clinical manifestations have low sensitivity,[12] acute monoarthritis with fever should be considered to have a bacterial etiology until proven otherwise because of the potential consequences of inadequate treatment.[20] For example, morbidity associated with septic arthritis includes functional deterioration, arthrodesis, and amputation[11,12]; the mortality rate is 10% to 20%.[12]

Diagnostic Tests

Because the causes of acute monoarthritis vary widely, a stepwise approach to diagnosis can aid decision making *(Figure 2[6])*. In the setting of trauma, radiography of the affected joint is required to rule out dislocation or fracture before performing active physical examination maneuvers.[1] Radiography can also show signs of osteoarthritis, such as joint space narrowing, osteophytes, and subchondral sclerosis.[1] If a joint effusion is present in the absence of traumatic injury, arthrocentesis should be performed, particularly when other inflammatory signs are present and there is a reasonable concern for infection (e.g., fever, erythema, warmth).[1]

Analysis of synovial fluid distinguishes infectious and inflammatory causes of acute monoarthritis (e.g., rheumatoid, septic, and crystal-induced arthritis) from noninflammatory causes (e.g., trauma, osteoarthritis).[3,4,11,14] Analysis should include cell count and differential, white blood cell count, Gram stain, cultures, and crystal evaluation.[1,4]

Many cases of acute gouty arthritis are diagnosed without synovial fluid analysis.[23] To improve the predictive value of clinical diagnosis, a gout calculator has been developed[24,25] *(Table 3[24])*. Seven predictive variables are included in this rule, and a scoring system has been developed to guide the decision to treat the patient for gout, pursue synovial fluid analysis, or search for other causes.[24,25] A complete blood count and uric acid level can also aid in the diagnosis, especially if synovial fluid cannot be successfully obtained.[3,19]

A synovial fluid white blood cell count greater than 50,000 per mm^3 (50.0×10^9 per L) with at least 90% neutrophils is the most useful laboratory finding for making an early diagnosis of septic arthritis.[3] This traditional cutoff lacks sensitivity because there can be wide overlap with inflammatory conditions, but higher white blood cell counts in the synovial fluid have a greater association with septic arthritis.[1,26] Staphylococci and streptococci are the most common bacterial causes at 40% and 28%, respectively, and their presence may suggest drug abuse.

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	*Evidence rating*	*References*
Radiography is not necessary for an accurate diagnosis of monoarthritis in the absence of trauma or focal bone pain.	C	3, 4
Analysis of synovial fluid distinguishes infectious and inflammatory causes of acute monoarthritis from noninflammatory causes.	C	3, 4, 11, 14
Gouty arthritis may be diagnosed without synovial fluid analysis using a diagnostic rule.	C	24
Disseminated gonococcal infections may not result in septicemia or positive synovial fluid cultures; therefore, cultures should be obtained from the potentially infected mucosal site.	C	31, 32
Inflammatory synovial fluid containing monosodium urate crystals, particularly in the presence of podagra, is highly suggestive of gout.	C	23, 24
Erythrocyte sedimentation rate and C-reactive protein level are more useful for following a disease course than discriminating the presence or absence of the disease in patients with monoarthritis.	C	19, 33

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to http://www.aafp.org/afpsort.

These organisms are also associated with cellulitis, endocarditis, and chronic osteomyelitis.[12,27,28]

Gram-stain results should guide initial antibiotic choice. Gram-negative organisms account for 10% to 21% of cases of septic arthritis.[1,12,29] Mycobacterial and fungal arthritis typically present in immunocompromised patients, and Lyme arthritis often presents as a late manifestation of Lyme disease.[1,12,30] Special consideration should be given to patients with prosthetic joint infection because the cutoff values for infection may be as low as 1,100 white blood cells per mm^3 (1.1×10^9 per L), with a neutrophil differentiation greater than 65%.[3] Gonococci are cultured from joints in fewer than 50% of cases of gonococcal arthritis[1,31,32]; therefore, it is often necessary to obtain cultures from appropriate mucosal sites of infection.[1,31,32] Noninflammatory synovial fluid (less than 2,000 cells per mm^3 [2.0×10^9 per L]) is suggestive of osteoarthritis or internal derangement.[1] Inflammatory synovial fluid containing monosodium urate crystals is highly suggestive of gout, particularly in the presence of podagra; however, the absence of crystals does not exclude crystal-induced arthritis, such as pseudogout.[23,24]

Erythrocyte sedimentation rate and C-reactive protein level are often elevated in inflammatory conditions.[19,33] Literature comparing these two values is limited; however, it has been determined that these tests are more useful for following a disease course than discriminating the presence or absence of the disease.[19,33]

Table 3. Diagnostic Rule for Gout When Synovial Fluid Analysis Is Unavailable

Patient with monoarthritis	
Male sex	2 points
Previous patient-reported arthritis attack	2 points
Onset within 1 day	0.5 point
Joint redness	1 point
Involvement of first metatarsophalangeal joint	2.5 points
Hypertension or ≥ 1 cardiovascular diseases*	1.5 points
Serum uric acid > 5.88 mg per dL (350 µmol per L)	3.5 points
Total score:	_____

≤ 4 points	*> 4 and < 8 points*	*≥ 8 points*
Non-gout in 95%	Uncertain diagnosis	Gout in 87%
Consider alternative diagnosis, such as CPPD arthritis, reactive arthritis, septic arthritis, rheumatoid arthritis, osteoarthritis, or psoriatic arthritis	Perform arthrocentesis and analysis with polarization microscopy for the presence of crystals; if not possible or available, then extensive follow-up of the patient	Manage the patient as having gout, including care for cardiovascular risk

CPPD = calcium pyrophosphate dihydrate deposition disease.

**—Indicates angina pectoris, myocardial infarction, heart failure, cerebrovascular accident, transient ischemic attack, or peripheral vascular disease.*

Adapted with permission from Kienhorst LB, Janssens HJ, Fransen J, Janssen M; British Society for Rheumatology. The validation of a diagnostic rule for gout without joint fluid analysis: a prospective study. Rheumatology (Oxford). *2015;54(4):612.*

This article updates a previous article on this topic by Siva, et al.[6]

Data Sources: PubMed and Medline searches were performed using the key terms arthritis, crystal, gonorrhea, gout, infectious, inflammatory, and synovial fluid. The search included reviews, clinical trials, and meta-analyses. Also searched were the Cochrane Database of

Systematic Reviews and Essential Evidence Plus. Search dates: September through November 2015, and September 2016.

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