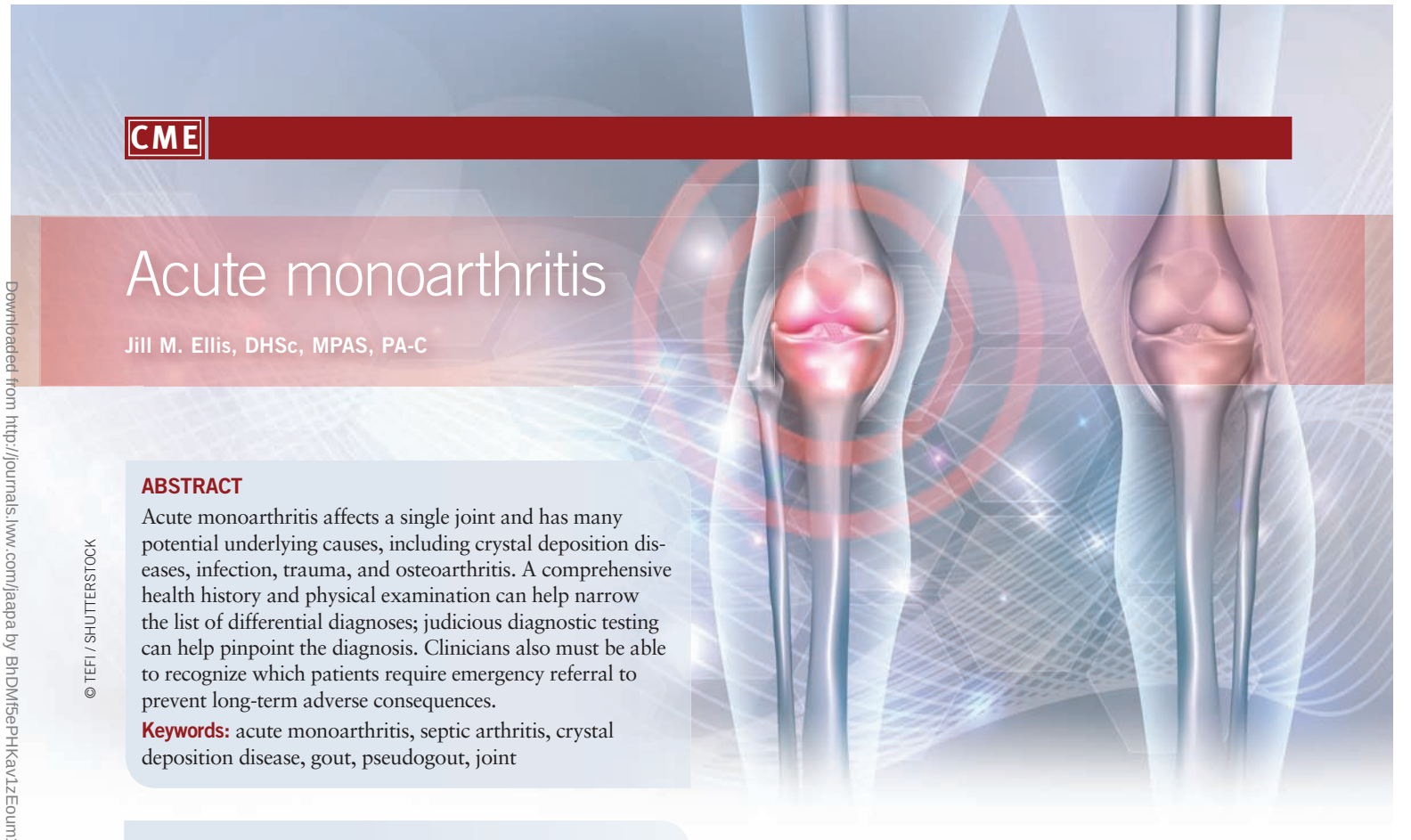

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CME

Acute monoarthritis

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ABSTRACT

Acute monoarthritis affects a single joint and has many potential underlying causes, including crystal deposition diseases, infection, trauma, and osteoarthritis. A comprehensive health history and physical examination can help narrow the list of differential diagnoses; judicious diagnostic testing can help pinpoint the diagnosis. Clinicians also must be able to recognize which patients require emergency referral to prevent long-term adverse consequences.

Keywords: acute monoarthritis, septic arthritis, crystal deposition disease, gout, pseudogout, joint

Learning objectives

- Identify the key characteristics of common causes of monoarthritis.
- Create a comprehensive differential diagnosis for isolated joint pain.
- Recognize indications for urgent intervention and/or referral.

Arthritis is a nonspecific term referring to joint pain or disease associated with physical signs of articular inflammation or degenerative change. Arthralgia refers to joint pain that is not associated with abnormal findings on physical examination. Both arthritis and arthralgia can occur in one or more joints. When a patient presents with joint pain, identifying the presence or absence of physical findings and symptom distribution is key because the differential diagnosis varies substantially based on these criteria.

Acute monoarthritis is an arthritis that occurs in a single joint for less than 2 to 4 weeks, and is a common presenting symptom for patients in primary care and emergency settings.[1,2] Diagnosis of acute monoarthritis can be challenging and outcomes range from benign and self-limiting to chronically disabling and deadly.[3] Clinicians in primary and emergency care must be familiar with the clinical presentation and initial diagnostic workup for this condition so that they can provide appropriate initial treatment, provide timely referral for patients more likely to have emergency conditions such as septic arthritis, and identify patients at high risk for chronic disability or significant complications.

CAUSES

The most common causes of acute monoarthritis are crystal deposition diseases, including gout and pseudogout; septic arthritis; trauma; osteoarthritis; rheumatoid arthritis; undifferentiated arthritis; and other primary inflammatory arthridities, such as seronegative spondyloarthropathies.[1,4-8] A meta-analysis of four cohort studies analyzed the differential diagnoses for patients presenting with acute arthritis to an arthritis clinic, two EDs, and an acute care setting.[1] Two studies specified that arthritic symptoms needed to have been present for less than 2 to 4 weeks; the other two studies did not specify a time frame for symptom duration. Undifferentiated arthritis was the most common diagnosis in this patient population, followed by gout, septic arthritis, osteoarthritis, and rheumatoid arthritis.[1]

CLASSIFICATION SYSTEMS

Classification of acute arthritis is helpful to narrow the differential diagnosis during the diagnostic process. Common classification systems divide presentations according to

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Key points

- Acute monoarthritis, which affects a single joint, has many potential underlying causes.
- Certain patient history and physical examination findings can point to specific diagnoses.
- Some patients, including those with septic arthritis or compartment syndrome, need emergency referral to a specialist for care.

acuity, presence of inflammatory symptoms and signs, location of symptoms, and number of joints affected (**Table 1**).[2,9]

Acuity Extremely acute onset of joint symptoms, which can occur within seconds, suggests a process that disrupted the internal structure of the joint, such as traumatic injury to bone or soft-tissue structures.[9] Acute onset over hours to days is the most common time frame for acute inflammatory processes, such as septic or crystal-induced arthritis.[2,9] Gradual onset over days to weeks more commonly represents osteoarthritis, tumors, infiltrative disease, or more indolent infections.[2] Longstanding symptoms may occur due to a monoarticular process or to a monoarticular exacerbation of a polyarticular disease.[9]

Inflammatory symptoms and signs Inflammatory joint symptoms may be infectious, crystal-induced, immune-related, or reactive. The presence of synovitis (an acute inflammation of the membrane lining synovial joints) is the hallmark of inflammatory arthritis. Characteristic findings of synovitis include warmth over the joint, joint effusion, and pain with movement.[10] Inflammatory processes, especially systemic rheumatic diseases, commonly are associated with joint pain with inactivity and morning stiffness lasting more than 30 to 60 minutes.[10] Noninflammatory processes can be due to trauma, repetitive use, degeneration or ineffective repair, neoplasm, and pain amplification. Noninflammatory processes are associated with joint pain that increases with prolonged use and usually are not associated with morning stiffness.[10]

Location of symptoms Patients who present with joint pain may have pathology in intra-articular and/or periarticular structures. Intra-articular pathologic processes originate in the synovium, synovial fluid, articular cartilage, intra-articular ligaments, and/or joint capsule. Pathologic processes involving these structures include septic arthritis, crystal-induced arthritis, and systemic rheumatic diseases. Periarticular structures include supporting structures such as bursae, tendons, ligaments, muscle, fascia, bone, nerves, and overlying skin. Pathologic processes involving these structures include bursitis, tendonitis, sprains, strains, and others. Pain that originates from intra-articular structures typically is deep, worsens with movement of the joint and weight bearing, improves with joint rest, and is maximal at extremes of joint motion.[2,10] Associated swelling is common with intra-articular pathology. Pain that occurs with joint movement is more likely caused by pathology in periarticular structures.[2,10] Vaguely localized pain in the proximity of the joint may be due to referred pain from a distant source, such as shoulder pain associated with angina, or a lesion in the surrounding bone.[2,10] Another difference

TABLE 1. Characteristics of common causes of acute monoarthritis[2,9]

Condition	Onset	Inflammatory vs. noninflammatory	Location of symptoms
Gout	Acute (24 hours or less)	Inflammatory	Intra-articular and/or periarticular (inflammation may extend beyond intra-articular structures)
Pseudogout	Acute (hours to a couple of days)	Inflammatory	Intra-articular and/or periarticular (inflammation may extend beyond intra-articular structures)
Septic arthritis	Acute (hours to a couple of days)	Inflammatory	Articular
Trauma	Extremely acute (seconds to minutes)	Noninflammatory	Intra-articular and/or periarticular
Osteoarthritis	Gradual (weeks to months)	Noninflammatory (most common)	Articular
Rheumatoid arthritis	Gradual (days to weeks)	Inflammatory	Intra-articular and/or periarticular (associated soft tissue swelling common)
Seronegative spondyloarthropathies (includes ankylosing spondylitis, reactive arthritis, psoriatic arthritis, colitic arthritis)	Gradual (days to weeks)	Inflammatory	Intra-articular and/or periarticular (may have associated enthesitis, dactylitis, bursitis, tenosynovitis, myopathy)

between intra-articular and periarticular processes is that intra-articular processes are usually associated with reductions in both active and passive range of motion; periarticular processes are associated with reductions in active range of motion only.

The location of the joint also can provide diagnostic clues. Crystal-induced arthropathies typically affect the first metatarsophalangeal joint and the knee. Septic arthritis commonly involves the knee or hip.[5] Rheumatoid arthritis typically occurs in smaller, peripheral joints.

Number of joints involved Monoarthritis, by definition, involves only one joint. However, monoarthritis may be the initial presenting symptom for an oligoarthritis that involves two to four joints, or polyarthritis, which affects five or more joints. This most commonly occurs with rheumatoid arthritis and seronegative spondyloarthropathies.[2] Monoarticular or oligoarticular presentations occur in 5% to 20% of patients ultimately diagnosed with rheumatoid arthritis.[9] Additionally, a patient with an established polyarticular process may develop an acute monoarthritis, defined as pain in one joint that is out of proportion to pain in other joints.[2]

Associated symptoms The presence or absence of certain symptoms can narrow the differential diagnosis of monoarticular arthritis. For example, a sensation of "giving way" or "locking" of a joint in addition to joint pain and swelling suggests ligament or cartilage disruption or a loose body in the joint.[10] Weakness in association with joint pain and swelling suggests a neuromuscular cause for the symptoms. If the weakness is associated with altered sensation, pathology is likely present in an associated nerve root or peripheral nerve.[10] Fever can be an accompanying symptom for acute monoarthritis; however, it is not specific to a particular diagnosis. However, the presence of constitutional symptoms, such as fever and weight loss, in combination with systemic symptoms, such as dermatologic, ocular, and vascular manifestations, suggests the presence of systemic rheumatic disease.[10] The combination of arthritis, urethritis, diarrhea, conjunctivitis, and/or dermatitis suggests a reactive arthritis, a subtype of seronegative spondyloarthropathies.[2,9] The presence of psoriatic nail changes or plaques suggests psoriatic arthritis, another subtype of seronegative spondyloarthropathy.[2] Symptoms of ocular inflammation and back pain in addition to peripheral joint pain and swelling suggest ankylosing spondylitis, a form of seronegative spondyloarthropathy.[2]

CLINICAL PRESENTATION

History Obtain a complete history when evaluating a patient with suspected acute monoarthritis. Thoroughly document the onset, character, location, and presence of additional symptoms specifically related to the acute joint symptoms. Ask the patient about inciting events such as trauma and investigate comorbid conditions that might contribute to the clinical picture. For example, a patient with a history of hypertension, obesity, cardiovascular disease, chronic renal failure, or nephrolithiasis is at increased risk for gout.[11] Risk factors for septic arthritis include diabetes, rheumatoid arthritis, history of joint surgery, and current or recent skin infection.[2,12] Patients with a coagulopathy are at increased risk for hemarthrosis.[2]

The patient's medication list often provides additional diagnostic clues (**Table 2**). Certain medications cause musculoskeletal symptoms, such as statin-induced myopathy. A recent prolonged course of corticosteroids increases the patient's risk for infection and/or avascular necrosis.[2] Diuretic use increases the risk for gout and anticoagulant use increases the risk for hemarthrosis.[2,11]

A social history can contribute information helpful to a final diagnosis and can guide therapeutic options. Ask patients about risk factors such as:

- travel history—patients who have traveled to a country with endemic tuberculosis may be at increased risk for atypical infections.
- sexual history—sexually transmitted infections can be associated with the development of reactive arthritis.

> Joint effusion signals intra-articular pathology.

- exposure to environmental or occupational hazards—tick bites increase the risk for Lyme arthritis, and certain occupations, such as farming and mining, frequently are associated with overuse injuries and osteoarthritis.[2]
- dietary habits—patients with diets high in purine and those who consume increased levels of alcohol are at increased risk for gout.[2,11]
- use of alcohol or illicit substances. IV drug use increases the risk for septic arthritis, which may present in atypical locations.[2]

Obtaining a complete family history also is important because a patient's risk for certain conditions such as gout and rheumatoid arthritis is increased with a positive family history.[1,13]

Look for signs that indicate the need for speedy evaluation and referral: history of significant trauma; presence of a warm, swollen joint; presence of constitutional symptoms; weakness; neurogenic pain; and claudication pain.[10] These symptoms can indicate emergency conditions such as internal derangement of the joint, infection, or neurovascular compromise. Treatment decisions also may be influenced by information obtained in the health history. Certain treatments may be appropriate in young, otherwise healthy patients, but not in older adults with multiple comorbid conditions and reduced functional ability. Evaluate the patient's functional ability, strengths, and resources, and identify barriers to treatment.[10]

TABLE 2. Some causes of drug-induced musculoskeletal conditions[16]

Condition	Well-established causative agents	Less well-established causative agents
Nonspecific inflammatory arthritis	• Immunizations (BCG, hepatitis B, rubella) • Interferon-alfa	• Bisphosphonates • Bupropion • Cefaclor • Cardiovascular agents (ACE inhibitors, beta-blockers, clopidogrel, hydralazine, streptokinase, ticlopidine) • Cytokines • Minocycline • Other immunizations
Nonerosive arthropathy/ arthralgia	• Immunizations (BCG, hepatitis B, rubella) • Interferon-alfa	• Antimicrobials (fluoroquinolones, pyrazinamide) • Cytokines • Granulocyte and granulocyte-macrophage colony stimulating factors • Other immunizations
Crystal deposition disease (gout, pseudogout)	Diuretics	• Antimicrobials (ethambutol, pyrazinamide) • Cardiovascular agents (gemfibrozil, low dose salicylate, nicotinic acid) • Gastrointestinal agents (H2 receptor antagonists, proton pump inhibitors) • Immunosuppressants (corticosteroids, cyclosporine, tacrolimus) • Cyanocobalamin, retinoids, thiamine
Septic arthritis	Corticosteroids	
Hemarthrosis	Anticoagulants	
Avascular necrosis	• Bisphosphonates • Corticosteroids	
Myalgia/ myopathies	• Cardiovascular agents (fibrates, nicotinic acid, statins) • Corticosteroids	• Anti-inflammatory agents (chloroquinoline, colchicine, hydroxychloroquine) • Antimicrobials (fluoroquinolones, zidovudine) • Granulocyte and granulocyte-macrophage colony stimulating factors
Nonspecific bone pain	• Bisphosphonates • Granulocyte and granulocyte-macrophage colony stimulating factors • Immunosuppressants (cyclosporine, tacrolimus)	Cardiovascular agents (fibrates, statins) Retinoids
Tendinopathies	• Corticosteroids • Fluoroquinolones	
Systemic rheumatologic disorders (lupus, polymyositis, dermatomyositis, scleroderma)	• Antimicrobials (isoniazid, minocycline) • Cardiovascular agents (hydralazine, methyldopa, procainamide, quinidine) • Chlorpromazine • Chelating agents (penicillamine) • Tryptophan	• Antiepileptic drugs • Antimicrobials • Antithyroid drugs • Cardiovascular agents • Chelating agents • Hormonal therapy • Immunosuppressants • Psychotropic agents

Physical examination Perform a thorough examination of the symptomatic joint and surrounding area, the contralateral joint, and a general screening examination to identify additional affected joints and/or systemic manifestations. Include inspection, palpation, range-of-motion testing, assessment of stability, and strength and sensory testing in the examination. On joint inspection and palpation, the patient may have soft-tissue swelling secondary to edema in periarticular structures, synovial thickening, and/or joint effusion. Joint effusion may be difficult to appreciate on examination but is important to detect because it signals intra-articular pathology. Special techniques can be used to detect effusion in various joints (**Table 3**).[2]

The physical examination should localize the source of pain to periarticular or intra-articular structures. Point tenderness and/or tenderness over periarticular structures can occur with pathology in tendons or bursae, such as tendinitis or bursitis. Tenderness located over the entire joint can indicate inflammatory arthritic processes such as gout or rheumatic disorders. Noninflammatory joint disorders, such as osteoarthritis, tend to be nontender to palpation.[10] Reduced active and passive range of motion suggests synovitis, structural joint abnormality, or soft-tissue contracture.[10] Reduced active range of motion and preserved passive range of motion suggests a periarticular soft-tissue disorder such as bursitis or tendinitis.[10] Tenderness to palpation along the course of a tendon and/or pain produced when the tendon is stretched or stressed during range of motion and strength testing suggests pathology in the tendon.[10] The presence of instability suggests ligamentous injury or tear.[10] The feeling of crepitus with joint motion in association with joint pain can signify articular surface abnormalities or synovitis; however, crepitus without associated joint pain often is a benign finding.[10]

The presence of extra-articular findings, such as dermatologic abnormalities, pericardial or pulmonary rubs, hepatomegaly, splenomegaly, lymphadenopathy, and/or neurologic abnormalities, suggests the presence of systemic disease as the cause of the arthritis.[10]

Certain physical examination findings are specific to various diagnoses. For example, tophi have high clinical diagnostic value for gout.[2] Patients with associated rash, pustules, and hemorrhagic bullae likely have gonococcal infection.[2] Ocular inflammation, hilar adenopathy, and erythema nodosum suggest the presence of arthritis associated with sarcoidosis.[2]

TABLE 3. Physical examination techniques to detect joint effusion[2]

Joint	Physical examination technique	Comments
Knee	Inspection and palpation	Effusions may lead to a full appearance in medial, lateral, and suprapatellar joint spaces
	Bulge sign	Milk fluid to lateral aspect of knee. Compression of this area can cause appearance of a fluid bulge medial to the patella.
	Ballottement sign	Milk synovial fluid into center of knee from all four quadrants. Push the patella firmly into the trochlear groove and release. Moderate effusion will cause clicking or tapping sensation.
Elbow	Inspection and palpation	Inspect and palpate area in center of triangle bound by lateral epicondyle of humerus, tip of olecranon, and radial head

DIAGNOSTIC EVALUATION

Diagnostic testing should refine and confirm suspected diagnoses based on information obtained during the health history and physical examination. Synovial fluid analysis is the key diagnostic test to evaluate acute monoarthritis and is required if septic arthritis is a diagnostic possibility. Arthrocentesis, the procedure performed to remove synovial fluid for analysis, can be both diagnostic and therapeutic in some patients because removing excess fluid can relieve symptoms and control infection.[2]

Synovial fluid analysis should include gross analysis of fluid appearance, white blood cell (WBC) count with differential, Gram stain, culture, and polarized light microscopy to evaluation for septic and crystal-induced arthritis.[2,10,12] Normal synovial fluid is clear. Inflammatory changes cause synovial fluid to become cloudy and opaque; noninflammatory pathologic processes in synovial fluid can result in a clear or cloudy appearance.[2] WBC counts in inflammatory synovial fluid are significantly increased, with associated increased percentages of polymorphonuclear cells.[10,12] Isolation of infectious agents is more likely to occur in patients with nongonococcal septic arthritis compared with those with gonococcal septic arthritis.[2] If clinical suspicion of infection remains high and initial cultures fail to isolate a pathogen, repeat and confirm the cultures.[2]

Identifying crystals is important for the diagnosis of crystal-induced arthritis. However, crystals can be difficult to identify depending on the skill of the observer.[11] Polarized light microscopy may not be available in certain clinical settings and tentative diagnosis can be made if crystals are identified using an ordinary light microscope. The needle-shaped monosodium urate crystals associated with arthritis caused by gout are more easily identified with an ordinary light microscope than are the calcium pyrophosphate dihydrate crystals associated with pseudogout.[2,14] Remember that crystal-induced arthritis can exist in association with other arthritic processes, including septic arthritis, so identification of crystals does not rule out concurrent infection.

Additional laboratory testing may aid the diagnostic approach. For example, elevated serum uric acid levels suggest a possible diagnosis of gouty arthritis; however, gouty arthritis can develop with normal or elevated serum uric acid levels.[2,10,12]

Routine blood and urine tests, such as metabolic panels, complete blood cell count, and urinalysis, should be performed when systemic disease is suspected based on the patient's clinical presentation.[10] Leukocytosis may be

found in patients with septic arthritis but is a nonspecific finding.

Obtain blood cultures in patients with suspected septic arthritis. Additional cultures of the pharynx, urethra, cervix, or rectum may be indicated for suspected cases of gonococcal arthritis.[2]

Rheumatic laboratory tests, such as erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), rheumatoid factor, anticitrullinated peptide antibody (anti-CCP), and antinuclear antibody (ANA), should only be ordered after other acute inflammatory and noninflammatory processes have been excluded based on patient history, physical examination, and/or synovial fluid analysis. ESR and CRP are nonspecific and can be elevated in patients with infection, inflammation, or malignancy. Inflammatory markers, such as ESR and CRP, can be helpful to distinguish between inflammatory and noninflammatory causes for acute monoarthritis when this distinction cannot be determined clinically.

Rheumatoid factor and anti-CCP antibody should only be ordered when there is at least a moderate suspicion for the diagnosis of rheumatoid arthritis.[6] Rheumatoid factor is nonspecific and can be identified in low levels in up to 15% of healthy patients.[15] Additionally, the presence of rheumatoid factor can be associated with other diseases, including Sjögren syndrome, systemic lupus erythematosus, pulmonary diseases, and infectious diseases.[15] Anti-CCP is more specific but less sensitive than rheumatoid factor.[15]

Avoid ANA testing in patients who present with localized joint pain that is not associated with systemic symptoms; up to 30% of healthy patients may have positive ANA at very low titers.[10,15] The presence of high ANA titers can reduce the risk of false positives; however, these antibodies also are seen in patients with other systemic and organ-specific autoimmune disorders, infectious diseases, and malignancies. The American College of Rheumatology recommends that clinicians avoid ordering bundled arthritis laboratory panels because of the high frequency of false-positive test results.[10]

Diagnostic imaging Plain radiographs, bone scintigraphy, CT, ultrasound, and MRI are common imaging tools to evaluate acute musculoskeletal complaints.[9]

Plain radiography often is the initial imaging study of choice and is indicated after significant trauma, when pain is not relieved following conservative therapy, and when the patient has a history of malignancy.[2,10] Obtain radiographs of the affected and contralateral joints. Despite the frequency with which plain radiographs are used in the clinical setting, they are nondiagnostic in most cases. Identification of erosive disease on early radiographs is a poor prognostic factor and indicative of future disability.[3] Chondrocalcinosis may be seen on plain radiographs and is suggestive, but not diagnostic, of pseudogout.[2,10]

Radiographs should be performed as a baseline study for patients with confirmed and suspected septic arthritis; however, they have low sensitivity and abnormal radiographic findings are nonspecific for septic arthritis. Arthrocentesis is the only way to definitively diagnose septic arthritis; however, radiographs provide supplemental information about disease severity and complications. Initial changes that may be seen on radiograph in patients with septic arthritis are soft-tissue swelling, followed by joint space narrowing. Chronic infection often leads to more destructive changes.[9] CT and MRI scanning are more sensitive and specific imaging modalities for this condition and may be required.

CT, ultrasound, and MRI improve visualization of intra-articular and periarticular soft-tissue structures.[9] Bone scintigraphy is optimal for assessing bone turnover.[10] These advanced imaging studies can help refine the diagnosis of monoarthritis in patients who are difficult to diagnose, and frequently are used in follow-up evaluation of patients with acute monoarthritis but are rarely indicated at initial workup.

TREATMENT

Most patients who present with acute monoarthritis can be treated conservatively with activity modification, local care, and appropriate analgesia. Recognizing patients who require emergency care is critical: septic arthritis, fracture, compartment syndrome, osteomyelitis, necrotizing fasciitis, tumors, and systemic vasculidities can cause acute joint symptomatology.[10] Patients who may have any of these conditions should be referred immediately to an appropriate specialist. Urgently refer patients with internal derangement associated with severe pain, poor function, or instability, and patients with suspected acute tendon or muscle rupture.[10] The American College of Rheumatology recommends timely referral for patients with

- undiagnosed multisystem or systemic rheumatic disease
- undiagnosed synovitis, in patients whom repeat arthrocentesis or synovial biopsy may be needed
- musculoskeletal pain that is undiagnosed after 6 weeks
- unexplained immunochemical test abnormalities suggestive of underlying rheumatic disease
- musculoskeletal pain not adequately controlled with therapy
- musculoskeletal pain associated with severe or progressive loss of function or work productivity
- conditions for which treatment with corticosteroids or immunosuppressive drugs is being considered
- dysfunction out of proportion to objective findings.[10]

CONCLUSION

Acute monoarthritis is a common condition for patients and has a wide range of causes, from benign to life-threatening. Clinicians in primary and acute settings need to be comfortable obtaining a complete patient history and physical examination in order to select appropriate

diagnostic tests to narrow the differential diagnosis to a single, working diagnosis. Clinicians also need to recognize which patients can be treated conservatively and which require emergency or timely referral to specialists to minimize adverse consequences and future disability. **JAAPA**

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