TABLE 24-2 Differential Diagnosis between Ankylosing Spondylitis and Sacroiliac Arthritis

Characteristic	Ankylosing Spondylitis	Sacroiliac Arthritis
History	Bilateral sacroiliac pain that may refer to posterior thigh Morning stiffness Male predominance	Bilateral sacroiliac pain referring to gluteal area (S1-S2 dermatomes) Morning stiffness (prolonged) Coughing painful
Observation	Stiff, controlled movement of pelvis	Controlled movement of pelvis
Active movement	Decreased	Side flexion and extension full Slight limitation of flexion
Passive movement	Decreased	Normal
Resisted isometric movement	Pain and weakness, especially if sacroiliac joints are stressed	Pain, especially if sacroiliac joints are stressed
Special tests	Sacral stress tests probably positive	Sacral stress tests probably positive
Sensation and reflexes	Normal	Normal
Palpation	Tender over sacroiliac joints	Tender over sacroiliac joints
Diagnostic imaging	X-rays diagnostic	X-rays diagnostic
Laboratory tests	Erythrocyte sedimentation rate increased HLA-B27 human leukocyte antigen present in 80%	Normal

From Magee DJ: *Orthopedic physical assessment*, ed 5, St Louis, 2008, Saunders.

peripheral symptoms associated with ankylosing spondylitis. It is reasonable to prescribe immunosuppressant drugs for severely affected patients who cannot participate in physical therapy.[25,27]

NONARTHRITIC RHEUMATIC DISEASES

GOUT

Gout is a metabolic disorder characterized by deposition of monosodium urate crystals in the joints, soft tissue, kidneys, and other connective tissue (Fig. 24-8).[9,19,27,58] When urate crystals are present, they can cause acute or chronic inflammation by stimulating inflammatory mediators.[9,19,27,44,58] The deposition of monosodium urate crystals is the outcome of hyperuricemia, which is the presence of high levels of uric acid in the blood. Hyperuricemia is due to the overproduction or underexcretion (or both) of uric acid.[9,19,27,44,58] Overproduction of uric acid is more common in individuals with a history of lymphoma, leukemia, or psoriasis.[19,27] Overproduction can also be due to enzyme abnormalities, hematologic malignancies, or other causes of rapid cell turnover.[58] Underexcretion of uric acid is more common with obesity, fasting, medication (e.g., diuretics, cyclosporine), renal insufficiency, hypertension, hypothyroidism, hyperparathyroidism, and acidosis.[19,27,58] Alcoholism increases risk of gout by increasing production and decreasing renal excretion of uric acid.[19,27,58]

Gout most commonly affects men older than 30 years; occasional cases occur in postmenopausal women.[9,19,27,58] Gout is characterized by acute monarticular onset of inflammatory arthritis that is worse at night. Although the metatarsophalangeal joint of the great toe is the most common site of pain, the ankle, knee, wrist, elbow, and fingers all can be affected.[19,27,58] The involved joints usually become tender, swollen, warm, and red.[19,27,58] A fever often occurs along with the joint complaints.[19,27] The early initial episodes last 3 to 10 days, and then a patient can go months to years with no symptoms.[19,27,58] Severe gouty attacks suddenly return with more frequency affecting more joints and lasting longer.[19,27,58] The gout can become chronic with multiple-joint damage leading to loss of function and disability.[19,27] Tophi, visible deposits of crystallized monosodium urate, usually occur several years after the first episode of gout.

Management of Gout

Management of acute gout focuses on the arthritis first and then addresses the hyperuricemia.[19,27] NSAIDs are most commonly used for pharmacologic management of acute gout and decrease inflammation and pain.[19,27,58] Corticosteroids are also used, but mainly when a patient has a contraindication for NSAIDs.[27] Another management option is to prescribe drugs to inhibit tubular reabsorption of uric acid to promote urinary excretion.[53] Rest, elevation, and joint protection are important during attacks of acute gout to promote decreased inflammation further.[19,58] Hyperuricemia is addressed between or after the acute attacks to help prevent future episodes.[19,27,58] Management during symptom-free periods includes changes in diet, avoidance of hyperuricemic medications, colchicine, and reduction of serum uric acid. Dietary changes that can decrease the risk of gout are weight loss, moderation of alcohol, and avoidance of high-purine foods.[19,27] Controlling hyperuricemia is important to prevent gout from becoming chronic.[19]

Important Concept

Gout is a nonarthritic rheumatic disease that is characterized by deposition of monosodium urate crystals in the joints, soft tissue, kidneys and other connective tissue. Management of gout focuses on arthritis and also addresses the hyperuriemia via medications.