Widely varying estimates of clinical outcome have been reported in PsA. At its worst, severe PsA with arthritis mutilans is potentially at least as crippling and ultimately fatal as severe RA. Unlike RA, however, many patients with PsA experience temporary remissions. Overall, erosive disease develops in the majority of patients, progressive disease with deformity and disability is common, and in some large published series, mortality was found to be significantly increased compared with the general population. There appears to be a greater incidence of cardiovascular death in psoriatic disease.

The psoriasis and associated arthropathy seen with HIV infection both tend to be severe and can occur in populations with very little psoriasis in noninfected individuals. Severe enthesopathy, dactylitis, and rapidly progressive joint destruction are seen, but axial involvement is very rare. This condition is prevented by or responds well to antiretroviral therapy.

LABORATORY AND RADIOGRAPHIC FINDINGS

There are no laboratory tests diagnostic of PsA. ESR and CRP are often elevated. A small percentage of patients may have low titers of rheumatoid factor or antinuclear antibodies. About 10% of patients have anti-CCP antibodies. Uric acid may be elevated in the presence of extensive psoriasis. HLA-B27 is found in 50–70% of patients with axial disease, but in ≤20% of patients with only peripheral joint involvement.

The peripheral and axial arthropathies in PsA show a number of radiographic features that distinguish them from RA and AS, respectively. Characteristics of peripheral PsA include DIP involvement, including the classic "pencil-in-cup" deformity; marginal erosions with adjacent bony proliferation ("whiskering"); small-joint ankylosis; osteolysis of phalangeal and metacarpal bone, with telescoping of digits; and periostitis and proliferative new bone at sites of enthesitis. Characteristics of axial PsA include asymmetric sacroiliitis. When compared with idiopathic AS, axial PsA manifests less zygapophyseal joint arthritis; nonmarginal, bulky, "comma"-shaped syndesmophytes that tend to be fewer and less symmetric and delicate than the marginal syndesmophytes of AS; fluffy hyperperiostosis on anterior vertebral bodies; severe cervical spine involvement, with a tendency to atlantoaxial subluxation but relative sparing of the thoracolumbar spine; and paravertebral ossification. Ultrasound and MRI both readily demonstrate enthesitis and tendon sheath effusions that can be difficult to assess on physical examination. A recent MRI study of 68 PsA patients found sacroiliitis in 35%, unrelated to B27 but correlated with restricted spinal movement.

DIAGNOSIS

Classification criteria for PsA were published in 2006 (Classification of Psoriatic Arthritis [CASPAR] criteria) that have been widely accepted (Table 384-2). The sensitivity and specificity of these criteria exceed 90%, and they are useful for early diagnosis. The criteria are based on the history, presence of psoriasis, characteristic peripheral or spinal joint symptoms, signs, and imaging. Diagnosis can be challenging when the arthritis precedes psoriasis, the psoriasis is undiagnosed or obscure, or the joint involvement closely resembles another form of arthritis. A high index of suspicion is needed in any patient with an undiagnosed inflammatory arthropathy. The history should include inquiry about psoriasis in the patient and family members. Patients should be asked to disrobe for the physical examination, and psoriasiform lesions should be sought in the scalp, ears, umbilicus, and gluteal folds in addition to more accessible sites; the finger and toe nails should also be carefully examined. Axial symptoms or signs, dactylitis, enthesitis, ankylosis, the pattern of joint involvement, and characteristic radiographic changes can be helpful clues. The differential diagnosis includes all other forms of arthritis, which can occur coincidentally in individuals with psoriasis. The differential diagnosis of isolated DIP involvement is short. Osteoarthritis (Heberden's nodes) is usually not inflammatory; gout involving more than one DIP joint often involves other sites and may be accompanied by tophi; the very rare entity multicentric reticulohistiocytosis involves other joints and has characteristic small pearly periungual skin nodules; and the uncommon entity inflammatory osteoarthritis, like the others, lacks the nail changes of PsA. Radiography can be helpful in all of these cases and in distinguishing between psoriatic spondylitis and idiopathic AS. A history of trauma to an affected joint preceding the onset of arthritis is said to occur more frequently in PsA than in other types of arthritis, perhaps reflecting the Koebner phenomenon in which psoriatic skin lesions can arise at sites of the skin trauma.

TABLE 384-2 THE CASPAR (*CLASSIFICATION CRITERIA FOR PSORIATIC ARTHRITIS*) CRITERIA[a]

To meet the CASPAR criteria, a patient must have inflammatory articular disease (joint, spine, or entheseal) with ≥3 points from any of the following five categories:

1. Evidence of current psoriasis,[b,c] a personal history of psoriasis, or a family history of psoriasis[d]
2. Typical psoriatic nail dystrophy[e] observed on current physical examination
3. A negative test result for rheumatoid factor
4. Either current dactylitis[f] or a history of dactylitis recorded by a rheumatologist
5. Radiographic evidence of juxtaarticular new bone formation[g] in the hand or foot

[a]Specificity of 99% and sensitivity of 91%. [b]Current psoriasis is assigned 2 points; all other features are assigned 1 point. [c]Psoriatic skin or scalp disease present at the time of examination, as judged by a rheumatologist or dermatologist. [d]History of psoriasis in a first- or second-degree relative. [e]Onycholysis, pitting, or hyperkeratosis. [f]Swelling of an entire digit. [g]Ill-defined ossification near joint margins, excluding osteophyte formation.

Source: From W Taylor et al: Arthritis Rheum, 54:2665, 2006.

TREATMENT PSORIATIC ARTHRITIS

Ideally, coordinated therapy is directed at both the skin and joints in PsA. As described above for AS, use of the anti-TNF-α agents has revolutionized the treatment of PsA. Prompt and dramatic resolution of both arthritis and skin lesions has been observed in large, randomized controlled trials of etanercept, infliximab, adalimumab, and golimumab. Many of the responding patients had long-standing disease that was resistant to all previous therapy, as well as extensive skin disease. The clinical response is often more dramatic than in RA, and delay of disease progression has been demonstrated radiographically. The potential additive effect of methotrexate to anti-TNF-α agents in PsA remains uncertain. As noted above, anti-TNF therapy, paradoxically, has been reported to trigger exacerbation or de novo appearance of psoriasis, typically the palmoplantar pustular variety. In some cases, the therapy can nevertheless be continued.

Ustekinumab, a monoclonal antibody to the shared IL-23/IL-12p40 subunit, is an efficacious treatment for psoriasis and has shown promise in PsA in clinical trials. Other newer drugs that have shown efficacy for both psoriasis and PsA include the anti-IL-17 pathway agents, such as secukinumab and brodalumab, and an oral phosphodiesterase-4 inhibitor, apremilast. Data on the oral Jak inhibitor, tofacitinib, has been very limited but promising.

Other treatment for PsA has been based on drugs that have efficacy in RA and/or in psoriasis. Until recently, controlled clinical trial data on methotrexate in doses of 15–25 mg/week and sulfasalazine (usually given in doses of 2–3 g/d) suggesting clinical efficacy have been somewhat limited, but neither regimen effectively halts progression of erosive joint disease. A recent double-blind trial assessing methotrexate 15 mg weekly in PsA demonstrated no benefit to the joint-based inflammation, but improvement was seen in patient and assessor global scores and skin scores. Other agents with efficacy in psoriasis reported to benefit PsA are cyclosporine, retinoic acid derivatives, and psoralens plus ultraviolet A light (PUVA). There is controversy regarding the efficacy in PsA of gold and antimalarials, which have been widely used in RA. The pyrimidine synthetase inhibitor leflunomide has been shown in a randomized controlled trial to be beneficial in both psoriasis and PsA.