

Orthopedic Manifestations and Management of Psoriatic Arthritis

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Abstract

Psoriatic arthritis is a complex, chronic inflammatory disease with both skin and joint involvement. Clinical presentation varies considerably among patients and during the course of the disease. Assessment of patients for psoriatic arthritis requires careful attention to patient history, a focused physical examination, and inspection for characteristic radiographic changes. Although this disease was once thought to be a rare and mild form of arthritis, recent studies have shown that patients with psoriatic arthritis may develop significant disability, with up to 20% of cases demonstrating a rapidly progressive, debilitating clinical course. Orthopedic manifestations of the disease can be severe and can cause significant physical disability. Although surgical intervention for psoriatic arthritis is relatively uncommon, having an understanding of the assessment, available treatment options, and surgical considerations allows for improved outcome in the management of this complex patient population.

Psoriatic arthritis, a complex clinical entity affecting both skin and joints, is characterized by chronic, immune-mediated, seronegative inflammatory arthritis associated with psoriasis.¹ Psoriasis is a common inflammatory skin disorder typically presenting as a papulosquamous disease with variable distribution, severity, and course.¹ There is a strong genetic component attributed to psoriasis, but ethnic and environmental factors are also involved, as evidenced by the variation seen in disease prevalence in different geographic populations.¹ In the United States, approximately 2% of the population has psoriasis. The disorder affects men and women equally and

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presents with a bimodal age of onset, with peaks at 15 to 20 years of age and 55 to 60 years of age.¹ In contrast to the abundance of information in dermatology and rheumatology texts, there is a relative paucity of literature on the orthopedic manifestations of psoriatic arthritis.

The presence of arthritis in a patient with psoriasis was first described in 1818. Initially thought to be a variant or subset of rheumatoid arthritis, psoriatic arthritis was recognized as a distinct entity after the discovery of rheumatoid factor (RF) in 1948.² In addition to a lack of

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RF in patients with psoriatic arthritis, the disorder is characterized by asymmetric distribution, distal interphalangeal (DIP) joint involvement, enthesitis, dactylitis, spinal and sacroiliac involvement, and association with various human leukocyte antigen (HLA) alleles.³ Currently, because of a lack of universally accepted diagnostic criteria, the exact prevalence of psoriatic arthritis is unknown.² Estimated prevalence rates have ranged from 0.04% to 1.2% of the general US population. Among patients with documented psoriasis, the prevalence of psoriatic arthritis has been reported to range from 6% to 42%.² Although this disease was once thought to be a rare and mild form of arthritis, recent studies have shown that affected patients may develop significant disability^{4,5} and that up to 20% of cases of psoriatic arthritis have a progressive, debilitating clinical course.³

The majority of patients with psoriatic arthritis can be managed effectively with nonoperative treatment modalities aimed at controlling the inflammatory process at the skin and joint level.³ However, patients with progressive joint involvement unresponsive to therapy with nonsteroidal anti-inflammatory drugs (NSAIDs) and disease-modifying antirheumatic drugs (DMARDs) may eventually require surgical intervention. One study found that approximately 7% of patients with psoriatic arthritis require orthopedic surgery.⁴ The literature on surgical intervention in psoriatic arthritis is small, with few data supporting the historical perception that such intervention increases the risk for infection and leads to poor outcome.⁶

In the present article, we review the orthopedic sequelae of psoriatic arthritis and focus on the clinical presentation, assessment, and surgical considerations involved in treating these complex cases.

PATHOGENESIS AND PATHOPHYSIOLOGY

The etiology of psoriatic arthritis is unknown, but epidemiologic and natural history studies suggest that a combination of genetic, environmental, and immunologic factors contribute to the disease process.⁷ Recently, investigators have attempted to identify specific genetic loci and T-cell and cytokine characteristics that may predispose patients to development of the inflammatory disease.^{8,9}

The genetic component of psoriatic arthritis is evidenced by the well-documented familial clustering of disease and by the high concordance rate in monozygotic and dizygotic twins (70% and 30%, respectively).^{3,8,10,11} Analysis of 100 families by Mease and Goffe¹⁰ and Moll and Wright¹² identified the risk for psoriatic arthritis to be 50 times higher in first-degree relatives of psoriatic arthritis patients than in patients from a control population. Monozygotic twins concordant for the disease have been shown to develop similar symptomatology, course, and severity.¹³ Genetic studies have suggested that several loci are involved in the pathogenesis of psoriatic arthritis, the majority in the major histocompatibility complex (MHC) on chromosome 6.^{8,9} Genome-wide linkage investigations have suggested that psoriatic arthritis is a multigenic disease with considerable genetic heterogeneity.¹³ Identification of specific HLA markers and their association with the progressive forms of psoriatic arthritis are the focus of ongoing investigations.^{8,10}

Environmental triggers such as traumatic events and infectious agents (bacterial and viral) have been implicated in the pathogenesis of psoriatic arthritis in the dermatology and rheumatology literature.^{8,10} The close temporal relationship between bacterial or viral infection and the development or flare of psoriasis and psoriatic arthritis suggests infectious involvement in the pathogenesis of disease.^{3,7} Initial observations of elevated levels of antibody to bacteria and bacterial components in guttate psoriasis patients suggested a relationship between hemolytic streptococcal infection and that condition.¹⁴ Muto and colleagues^{15,16} found elevated levels of monoclonal antibody against cell wall antigens of *Streptococcus pyogenes* in psoriatic arthritis patients. These antibodies were found to cross-react with the nuclei and cytoplasm of cells from the synovium and skin of control patients, suggesting that an antibacterial response may contribute to the disease process by provoking an exaggerated inflammatory cascade.^{8,15,16}

A link between viral infection and development of psoriasis and psoriatic arthritis was proposed subsequent to identification of an increased incidence of disease in patients with HIV infection.⁷ Early in the course of HIV infection, patients tend to develop a progressive, polyarticular lower limb arthritis. Similar correlations have been observed in patients with hepatitis C infection, leading authors to suggest that CD8 T cells have a significant role

in the pathogenesis of psoriatic arthritis.⁷ Traumatic events have been reported to lead to the development or progression of arthritic disease in patients with psoriasis. In a comparison of 25 posttraumatic psoriatic arthritis patients with 275 psoriatic arthritis patients without a history of trauma, Punzi and colleagues¹⁷ identified significantly higher erythrocyte sedimentation rates (ESRs) and C-reactive protein (CRP) levels in the posttrauma patients. The increase in the acute-phase reactants was transient, leading the authors to suggest that the development of trauma-induced arthritis in psoriatic arthritis patients represents a deep-level Koebner phenomenon.^{7,9,17}

Immunologic factors, in the form of both humoral and cell-mediated immune responses, also have a role in the pathogenesis of psoriatic arthritis, in agreement with the notion that a genetic predisposition to the disease results from the presence of specific alleles in the MHC. Studies of psoriatic arthritis patients have found elevated serum immunoglobulin (IgA, IgG) levels, immune complex deposition, and the presence of cellular infiltrates.¹⁰ Samples of synovial membrane from affected patients have been found to have a significantly larger number of plasma cells positive for IgA or IgG than those from nonaffected controls.¹⁰ Existence of a humoral component of the disease process is supported by the finding of circulating immune complexes and autoantibodies in patients with psoriatic arthritis.¹⁰ Recent investigations into the pathogenesis of psoriatic arthritis have focused on the role of T cells in the development and progression of joint inflammation and degeneration.^{9,13} As with other spondyloarthropathies, there is an increased number of CD8+ T cells in the synovial fluid of patients with psoriatic arthritis.^{9,13} Inflammatory cytokines produced by these activated T cells contribute to proliferation of synovial fibroblasts and degeneration of normal joint architecture.^{9,10} Support for the role of activated T cells in the pathogenesis of psoriatic arthritis is provided by recent studies showing clinical improvement after administration of agents designed to kill activated T cells.¹⁰

CLINICAL PRESENTATION

Psoriatic arthritis presents clinically as a systemic inflammatory disorder with articular and extra-articular features.^{3,7,10} The disease affects both the axial skeleton and the peripheral joints with 5 general patterns of involvement, as described by Moll and Wright¹⁸ and Wright¹⁹:

1. *Predominant distal interphalangeal degenerative arthritis* is typically considered the classic form of psoriatic arthritis, despite the fact that it accounts for only approximately 5% of cases. DIP joint disease may be symmetric or asymmetric, may affect a few joints or many, and commonly leads to progressive erosive bony lesions.^{3,9,10,20}

2. *Arthritis mutilans*, which accounts for 1% to 5% of cases, is a severe, destructive form of the disease with extensive erosive lesions of the phalanges, metacarpals, and metatarsals.^{7,18,20}

3. *Oligoarthritis*, the most common presenting pattern of psoriatic arthritis, is characterized by asymmetric involvement of fewer than 5 joints.^{3,20} This pattern accounts for more than 50% of cases and typically begins with symptoms localizing to a single knee.²⁰

4. *Symmetric polyarthritis*, the next most common presenting pattern, is characterized by involvement of 5 or more joints. This pattern is difficult to distinguish from rheumatoid arthritis, with prominent metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joint involvement.^{3,20}

5. *Predominant spondyloarthropathy* involves degenerative changes and inflammation of the sacroiliac joints and

Hand and wrist involvement in psoriatic arthritis typically consists of joint space narrowing at the wrist accompanied by significant reactive bone changes. Many psoriatic arthritis patients present with severe erosive changes, obliteration of the radiocarpal joint space, collapse of the proximal row, and/or spontaneous fusion.^{22,23} The wrist stiffness or ankylosis seen in psoriatic arthritis typically occurs in a functional position, obviating the need for operative intervention, though some patients require reconstructive wrist surgery to alleviate pain or correct deformity into a functional alignment.^{22,23} A high percentage of patients with polyarticular disease report stiffness at the MCP joints, with extension contractures occurring more often than

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the apophyseal joints of the spine and is seen in approximately 5% of cases.^{7,20} The lumbar spine tends to be the most common site of involvement in predominant spondyloarthritis associated with psoriatic arthritis.²⁰

Although these patterns of presentation may be useful in early identification and diagnosis of psoriatic arthritis, they are typically not permanent, as more than 60% of cases change pattern during the course of disease.⁵

Psoriatic arthritis may affect any and all peripheral joints. Affected joints generally present with pain and tenderness, with or without associated swelling. These inflamed joints typically have a erythematous/purplish discoloration superficially, a characteristic that is not commonly seen in rheumatoid patients.³ Joint effusions are often difficult to detect in psoriatic patients because of the tight capsule associated with their joint pathology. Psoriatic arthritis tends to affect peripheral joints in a “ray” pattern, in which all 3 joints of an affected digit are involved, with sparing of other digits.³ This is another distinction from rheumatoid arthritis, in which involvement tends to affect joints at the same level, leading to a symmetric joint distribution. Significant morning stiffness, a commonly reported symptom in inflammatory arthritides, is seen in only 50% of patients with psoriatic arthritis.³

Of the large joints, the hip and knee are the most commonly affected by psoriatic arthritis, often in the oligoarticular pattern of the disease.^{4,6} Michet and colleagues²¹ found that, though symptomatic hip disease occurs in a small percentage of the overall psoriatic arthritis population, affected patients tend to develop symptoms early, to have bilateral disease, and to experience symptoms that progress rapidly, causing significant physical disability. Patients with symptomatic large joint disease tend to experience significant morning stiffness, pain with activity, and limited range of motion (ROM) during flares of synovial inflammation.

flexion contractures. Extension contractures and flexion contractures of the PIP joints occur with equal incidence in psoriatic arthritis and often require surgical management. Spontaneous fusion of the DIP joints is relatively common over the course of psoriatic arthritis. Relief of pain may coincide with ankylosis, but functional deformity occurs frequently because of fusion malposition.^{22,23} Other manifestations of psoriatic arthritis affecting the hand and wrist include instability of the interphalangeal joint of the thumb, causing pain and loss of pinch strength, and the severe osteolysis and bony collapse seen in cases of arthritis mutilans.²³

Dactylitis, or sausage digit, is a typical feature of psoriatic arthritis, seen in up to 40% of affected patients.²⁴ Dactylitis is characterized by diffuse swelling of the entire digit secondary to inflammation involving the tendons and periosteum in addition to the degenerative arthritic changes seen in the DIP, PIP, and MCP or metatarsophalangeal (MTP) joints.^{3,10,20} Development of dactylitis may be triggered by traumatic events, a hypothesis supported by higher incidence in the feet, dominant hand, and index fingers of affected psoriatic arthritis patients.^{17,24} In their review of 260 psoriatic arthritis patients presenting with dactylitis, Brockbank and colleagues²⁴ found that erosive lesions were more likely to progress in digits affected by dactylitis than in digits not so affected.

Nail disease is another common clinical component of psoriatic arthritis, affecting up to 80% of patients in natural history studies.²⁵ Patients with psoriatic arthritis have a higher incidence of nail pathology than patients with psoriasis alone.²⁵ Typical pathologic changes include pitting, subungual keratosis, dystrophy, discoloration, and onycholysis.²⁵ Jones and colleagues²⁶ found that nail disease in psoriatic arthritis is associated with adjacent DIP joint degeneration, suggesting a common local inflammatory mechanism. Cohen and colleagues²⁷ reported a similar



Figure 1. Plain x-rays of bilateral hands show “pencil-in-cup” deformity of marked lysis of the distal end of the third, fourth and fifth proximal phalanges with bony remodeling of the proximal end of the more distal phalanges.

significant relationship between DIP joint involvement and associated local nail disease. In an evaluation of 69 cases of psoriatic arthritis, Williamson and colleagues²⁵ found that patients with more severe nail disease tended to have worse skin disease and a higher rate of progressive degenerative arthritis and functional impairment than patients without nail involvement.

Symptomatic foot disease is common in psoriatic arthritis patients, occurring in 50% to 70% of cases, often early in the disease process.²⁸ The calcaneus and forefoot tend to be involved much more commonly than the ankle and subtalar joints. Patients typically complain of posterior heel pain, especially on awakening, and metatarsal pain.²⁸ Bauer toe, an erosive arthritic change in a DIP joint associated with periungual psoriasis and psoriatic nail changes, correlates with disease activity.²⁸

Spondylitis occurs in 20% to 40% of psoriatic arthritis patients, typically developing late in the disease course.⁵ The spondylitis of psoriatic arthritis most often involves inflammatory changes at the sacroiliac and apophyseal joints. Spinal involvement tends to be asymmetric and has been reported to be more severe in male patients than in females.²⁹ Clinically, patients complain of pain and stiffness in the cervical, thoracic, and lumbar regions, typically occurring at rest and improving with activity.³ Spondylitis in psoriatic arthritis may also be asymptomatic.³ Hanly and colleagues,³⁰ in an observational study of 52 patients with psoriatic spondylitis, reported that 65% of affected patients experienced neck and lower back pain and stiffness and that 17% showed clinical evidence of sacroiliitis based on eliciting of sacroiliac symptoms with Fabere or Gaenslen maneuvers. The spondylitis associated with psoriatic arthritis presents similarly to ankylosing spondylitis, but the disease course tends to be less severe, with psoriatic patients demonstrating better spinal ROM and a lower incidence of grade 4 sacroiliitis.^{3,29}

Enthesitis, an inflammation of tendons at points of bony insertions, is a common finding in psoriatic arthritis. Enthesitis can occur at any site but most commonly affects the Achilles tendon insertion at the calcaneus, the quadriceps insertion at the patella, the posterior tibial tendon, the flexor tendon insertions in the hands and feet, and the plantar fascia.³ Affected patients experience heel and knee pain with activity and on examination demonstrate tenderness localizing to the site of tendinous insertion.^{2,28} Ultrasound evaluation of patients with clinical evidence of peripheral enthesitis typically demonstrates effusions in the tendon sheath and adjacent joints.³¹ This finding has led some to hypothesize a causal relationship between inflammatory changes at the tendinous insertion site and subsequent adjacent large joint synovitis.^{32,33}

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The major extra-articular feature of psoriatic arthritis is the presence of psoriasis lesions.³ Most psoriatic arthritis patients exhibit the psoriasis vulgaris pattern of skin involvement, but associations have been made to the pustular, flexural, and guttate forms as well.^{2,3} There is no temporal relationship between the development of skin and joint manifestations of disease. Fifteen percent to 20% of psoriatic arthritis patients have joint disease before development of skin lesions. There is also no direct relationship between the severity of psoriasis lesions and the degree of joint inflammation and degeneration.²⁷ Other extra-articular manifestations of psoriatic arthritis include ocular involvement with iritis or conjunctivitis (7%-33% of patients); mitral valve prolapse (30%-50%); and aortic incompetence (~4%), reported to occur late in the disease process.³

Less common presentations of psoriatic arthritis, which do not fit into the original classification by Moll and Wright¹⁸ and Wright,¹⁹ have been reported in rheumatology and dermatology literature. These relatively rare arthropathies have clinical and radiographic features that overlap, with a common feature of bony changes (lysis or new bone formation) related to presence of enthesitis.³³ They include SAPHO (synovitis, acne, pustulosis, hyperostosis, and osteitis) syndrome,³³ palmar plantar pustulosis,³³ spondylodiscitis (noninfectious bony lysis of the spine adjacent to enthesopathic insertions),³⁴ onychopachydermoperiostitis,^{26,35} and chronic multifocal recurrent osteomyelitis.³⁶ The new bone formation and bony lysis seen in both classic psoriatic arthritis and these less common presentations occur adjacent to enthesal insertions, lending support to the importance of enthesitis in the pathogenesis of psoriatic arthritic disease.^{3,33}



Figure 2. Plain x-rays of right hand show distal interphalangeal joint erosive disease.

ASSESSMENT: RADIOGRAPHIC AND LABORATORY FEATURES

Radiographic features that are characteristic of psoriatic arthritis include joint space narrowing, joint erosions, osteolysis including resorption of the distal phalanges, bony proliferation including shaft and periarticular periostitis, spur formation, ankylosis, and spondylitis.^{37,38} The classic radiographic finding in the peripheral joint is the “pencil-in-cup” deformity of marked lysis of the distal end of a phalanx with bony remodeling of the proximal end of the more distal phalanx^{37,38} (Figure 1). Radiographic changes in psoriatic arthritis are often asymmetric and oligoarticular, most commonly involving the carpus, MCP, PIP, and DIP joints.³⁷ The hands tend to be involved more often than the feet, with the DIP joints typically being the first joints to show radiographic evidence of degenerative change³⁷ (Figure 2). The sternoclavicular, temporomandibular, and manubriosternal joints may also be involved during the course of psoriatic arthritis, requiring specific radiologic techniques for proper evaluation.³⁹ In contrast to rheumatoid arthritis, osteoporosis is atypical in psoriatic arthritis.³⁷



Figure 3. Plain x-rays of hand and elbow show periarticular erosive changes at margin of peripheral joints.

Evidence of erosive changes on x-ray early in the course of psoriatic arthritis is usually seen at the margin of the peripheral joints³⁸ (Figure 3). As the disease process progresses, the joints become ill-defined and irregular because of periosteal bone formation adjacent to the sites of periarticular erosion.^{10,37} In the phalanges, radiographic abnormalities are seen in the phalangeal tufts and at sites of tendinous insertion.³⁷ In the feet, degenerative changes caused by psoriatic arthritis typically affect the MTP and interphalangeal joints, with the interphalangeal joint of the great toe being most commonly involved^{37,38} (Figure 4). Endosteal and periosteal bone proliferation can lead to an “ivory” phalanx in which the radiodensity of the entire digit is increased, an uncommon yet specific radiographic finding for psoriatic arthritis.³⁷ Involvement of larger joints, such as the hip, is typically demonstrated by radiographic changes consistent with other inflammatory arthritides, such as axial or concentric joint space narrowing with associated marginal osteophytes²¹ (Figure 5).

Radiographic evidence of spondylitis is another characteristic of psoriatic arthritis.³⁷ Syndesmophyte formation



Figure 4. Anteroposterior and lateral x-rays of bilateral feet show disease affecting the metatarsophalangeal and interphalangeal joints of all digits. There is periarticular erosive disease plus subluxations of the metatarsophalangeal, proximal interphalangeal, and distal interphalangeal joints.

typically occurs paramarginally in psoriatic patients and is not found in consecutive vertebrae.³⁷ Bony erosions may be seen on the surface of the vertebral bodies, with syndesmophytes forming either at the sites of erosion or in the adjacent soft tissue.³⁷ Evidence of sacroiliitis is seen in 20% to 40% of psoriatic arthritis patients, although 78% of a series of 221 patients studied by Battistone and colleagues⁴⁰ exhibited radiographic changes consistent with sacroiliac joint inflammation. Sacroiliitis in psoriatic arthritis is typically unilateral and less severe than in cases of ankylosing spondylitis²⁹ (Figure 6). Other radiographic findings commonly reported in cases of psoriatic arthritis include apophyseal joint ankylosis, atlantoaxial subluxation, and ligamentous calcification.³⁷

There are no specific laboratory tests diagnostic for psoriatic arthritis. Historically, lack of RF was considered the most important laboratory feature of the disease, but recent studies indicated that 5% to 16% of psoriatic arthritis patients have low titers of RF and that 2% to 16% of patients have evidence of antinuclear antibodies.^{19,41} ESR is elevated in 40% to 60% of psoriatic arthritis cases, especially in the polyarticular form of the disease.⁴² CRP levels are also increased

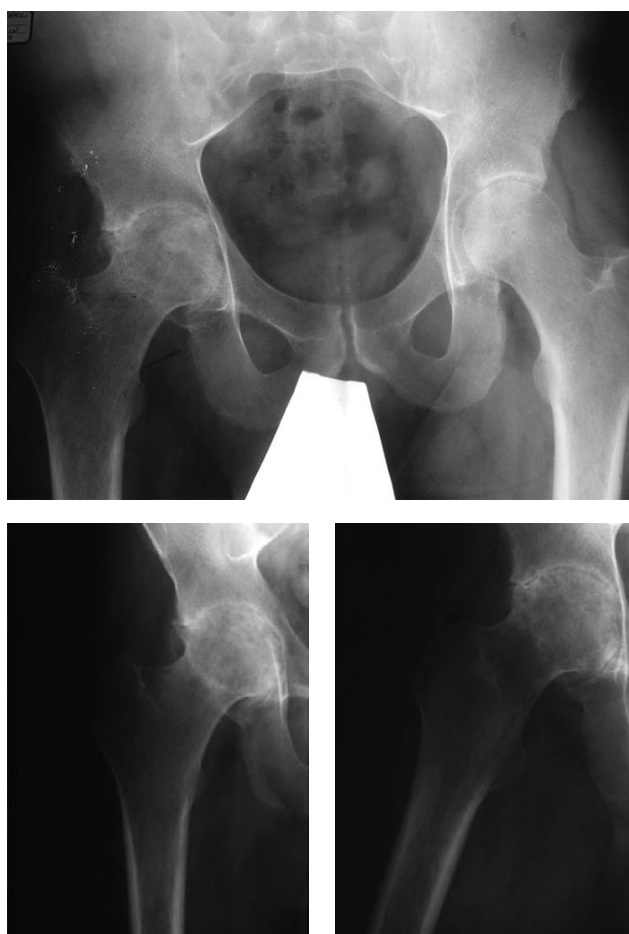


Figure 5. Anteroposterior x-ray of pelvis and anteroposterior and lateral x-rays of right hip show concentric degenerative changes consistent with inflammatory arthropathy plus secondary osteoarthritic changes at joint margins.

in 25% to 50% of patients.¹⁷ Other laboratory findings seen in psoriatic arthritis include increased complement activity, hypergammaglobulinemia, and elevated IgA levels.¹⁷

NATURAL HISTORY AND PROGNOSIS

Psoriatic arthritis is a pleomorphic disease that affects any joint and has an extremely variable course and severity. Based on the description of the disease by Moll and Wright,^{12,18} psoriatic arthritis was initially considered less severe than rheumatoid arthritis. Since the 1980s, however, evidence has mounted that the disease is considerably more aggressive than previously believed, with up to 20% of patients having a severe, debilitating form of degenerative arthritis.² Longitudinal observational studies have found that patients with psoriatic arthritis experience intermittent symptomatic flares, with between-attack remissions of variable duration.^{2,3} These studies have also found that the natural history of the disease varies by subtype of clinical and radiographic presentation.⁴³

Destructive arthritis tends to occur in approximately 25% of patients who present with oligoarticular arthritis and in approximately 65% of patients who present with



Figure 6. Anteroposterior x-ray of pelvis and anteroposterior and lateral x-rays of lumbar spine show unilateral sacroiliitis and spondylitis with associated paramarginal syndesmophyte formation.

polyarticular disease.² Gladman and colleagues,⁴² reporting on a series of 220 psoriatic arthritis patients in which 67% had evidence of at least 1 component of erosive disease, observed that only 37 patients (17%) had erosions in 5 or more joints. This study and similar follow-up investigations have shown that, despite control of the inflammatory process of psoriatic arthritis, joint damage tends to progress such that, by 5 years of observation, the number of patients with 5 or more involved joints doubles.⁵ Longitudinal observational studies have reported that, by 10-year follow-up, the percentage of patients in which 5 or more joints are damaged is approximately 55%.⁵ Risk factors for severe, progressive destructive disease include female gender, polyarticular disease at presentation, younger age at symptom onset, and acute onset of arthritis.⁴³

In a prospective evaluation of 305 patients with fewer than 10 involved joints, seen at a psoriatic arthritis clinic, Gladman and colleagues⁴⁴ reported that presence of 5 or more swollen/tender joints and use of steroids for symptom relief on initial evaluation were predictive of disease progression during the follow-up period. In the same patient

cohort, ESR of less than 15 mm/h was found to be protective against disease progression.⁴⁴ HLA typing of 276 of the 305 patients demonstrated that HLA-B39 correlated with disease progression in early disease and that HLA-B27 in the presence of HLA-DR7 and HLA-DQw3 in the absence of HLA-DR7 also were associated with continued joint degeneration.⁴⁴

The mortality rate of patients with psoriatic arthritis appears to be higher than that of the general population. Wong and colleagues,⁴⁵ in an observational study of a patient cohort from a large psoriatic arthritis clinic, found a 62% increased risk for mortality associated with psoriatic arthritis. Mortality in this study was attributed to respiratory disease in 21% of cases, cardiovascular disease in 36%, and malignancy in 17%. Analysis of the patient data showed that ESR of more than 15 mm/h at presentation, radiographic evidence of erosive disease, high level of medication use at initial evaluation, and absence of nail lesions were all predictive of increased overall mortality.⁴⁵

Overall, the chronic, relapsing, destructive nature of psoriatic arthritis leads to significant functional disability and reduced quality of life. Functional evaluations of patients affected by psoriatic arthritis have demonstrated that, at time of diagnosis, up to one third of patients are either bedridden or have limited their activities of daily living to self-care.^{3,46} Progression of clinical damage is seen in the majority of patients, and only a small percentage achieve complete, prolonged remission without therapeutic intervention.⁴⁷

NONOPERATIVE MANAGEMENT

Management of psoriatic arthritis involves treatment of both skin and joint manifestations, regardless of the lack of correlation in severity and onset between the 2 main features of the disease. If the skin symptoms are the major problem and the joint manifestations are mild, the latter can be managed symptomatically with NSAIDs. It is nevertheless important to note that NSAIDs do not prevent the development or progression of the bony erosions of psoriatic arthritis, and they do not modify the disease course.³ The few controlled studies that have evaluated the efficacy of NSAIDs in psoriatic arthritis found that, although these medications are superior to placebos with regard to pain scores and swollen/tender joint counts, they have no appreciable effect on ESR or rashes, as assessed by Psoriatic Area and Severity Index (PASI) scores.⁴⁸

Intra-articular glucocorticoid injection is another symptomatic treatment of psoriatic arthritis that may be useful in managing patients with oligoarticular or polyarticular disease. Persistently inflamed joints may respond to periodic steroid injection with variable efficacy. This treatment should be used judiciously, however, as there can be a skin withdrawal reaction in the form of a flare of pustular psoriatic disease.⁴⁸

In severe progressive cases of articular disease not responsive to NSAIDs and cases in which NSAIDs are not well tolerated, DMARDs may be prescribed. This class of medication includes agents such as methotrexate, sulfasal-

zine, cyclosporine A, gold, antimalarials, and leflunomide. Although these drugs have been shown to help control the symptomatic manifestations of psoriatic arthritis, there is no evidence that they prevent progression of bone erosion and joint degeneration.⁴⁸ It is also important to note that the majority of efficacy and safety trials involving DMARDs have been performed in rheumatoid arthritis patient cohorts, with the results extrapolated for use in cases of psoriatic arthritis.⁴⁹

The principal target for therapeutic agents for psoriatic arthritis continues to be tumor necrosis factor (TNF- α). This pleiotropic proinflammatory cytokine has been implicated as a key factor in the pathogenesis of the disease. TNF- α inhibitors (eg, etanercept, infliximab, adalimumab, alefacept) have been shown to produce significant improvement in the signs, symptoms, and quality of life of patients with psoriatic arthritis, in addition to inhibiting progression of bone and joint degeneration.^{50,51} The important role that TNF- α has in the function of the immune system has raised concerns over use of anti-TNF agents in treating rheumatologic disease. Results from the infliximab and etanercept trials did not show any significant increase in infection rates.^{50,52}

OPERATIVE MANAGEMENT

There is a paucity of literature regarding the need for and outcomes of orthopedic surgical intervention in psoriatic arthritis. One review of 444 psoriatic arthritis patients reported that 7% required musculoskeletal surgery during the observation period.⁴ The incidence of surgical treatment in this study correlated with disease duration, and the number of actively inflamed joints and severe erosive changes on x-rays at presentation served as risk factors for need for operative intervention.⁴ The first surgery occurred a mean of 13 years after symptom onset (range, 1-49 years).⁴ Hip arthroplasty was the most common procedure performed in this patient cohort, followed by knee arthroplasty, hand and wrist joint fusions, and synovectomies of the wrist, knee, and elbow.⁴

Zangger and colleagues⁶ described their 10-year results with 71 orthopedic surgical procedures performed in 43 patients with active psoriatic arthritis. Sixty-five percent of these patients had a polyarticular pattern of disease, and these patients underwent a range of operations on both small and large joints. Reconstructive procedures involving the hands and feet were the most common surgeries performed in polyarticular psoriatic arthritis patients.⁶ An oligoarticular pattern of disease was seen in 25% of cases; these patients typically required large joint surgery on the hip, knee, or ankle.⁶ Predominantly distal psoriatic arthritis was documented in 10% of cases, with isolated or combined PIP and DIP joint procedures being most commonly performed.⁶

Zangger and colleagues⁴ found hip arthroplasty to be the most common orthopedic operation performed in patients with psoriatic arthritis. Patients with disease onset before age 30 and patients with evidence of axial skeletal involvement appeared to be at highest risk for developing symp-

tomatic hip joint disease.⁴ In a review of hip joint disease in psoriatic arthritis, Michet and colleagues²¹ reported that, in a cohort of 504 patients with active disease, 6.3% developed significant, symptomatic hip arthropathy. Forty-one percent of these patients developed hip-related symptoms within 1 year after presenting with psoriatic arthritis, and 50% underwent total hip arthroplasty within 5 years of onset of hip pain.²¹ In this cohort, mean age at time of arthroplasty was 41 years (range, 25-63 years), and one third of patients underwent bilateral procedures.²¹ Outcomes after hip arthroplasty in psoriatic arthritis patients parallel those in rheumatoid arthritis patients and in ankylosing spondylitis patients. Potential complications include heterotopic bone formation limiting postoperative ROM and flares of psoriasis in the region of the surgical scar (Koebner phenomenon).⁵³

Indications for hand and wrist surgery in cases of psoriatic arthritis include significant joint pain and loss of function. In psoriatic arthritis patients, spontaneous fusion of the radiocarpal joint often occurs in a functional position, causing little pain or disability.²³ For patients who nevertheless experience persistent pain, progressive erosive change, and functional deformity, total wrist fusion, distal ulna resection, and arthroplasty are options for providing pain relief with maintenance of adequate function.²³ Patients with MCP extension contractures may benefit from manipulations or soft-tissue releases. Postoperative fibrosis, a common occurrence in psoriatic arthritis, may limit the postoperative ROM attainable. PIP joint pain and deformity (flexion and extension contractures) can typically be treated successfully with manipulations or arthrodesis.²³ In more mild cases, PIP arthroplasty can be considered, but postoperative fibrosis may limit postoperative ROM. Pain related to DIP joint disease is relatively uncommon secondary to the high incidence of spontaneous fusion, but for patients with persistent pain, progressive erosive disease, or malposition of the ankylosis, realignment and arthrodesis have proved to be a successful treatment option. Improvement of pinch strength and relief of pain related to instability of the thumb interphalangeal joint may be achieved by interphalangeal arthrodesis.

Belsky and colleagues²² retrospectively reviewed 25 patients who underwent hand and wrist surgery as treatment for active psoriatic arthritis. Of the 8 patients with persistent wrist pain, joint erosion, and deformity, 3 were managed with arthroplasty, 3 with distal ulna resections, and 2 with fusions. All 8 patients reported improvement in terms of relief of wrist pain, but ROM in the arthroplasty cases was limited.²² Seventeen of the 25 patients in the cohort underwent surgical intervention for PIP joint disease. Fifty PIP fusions, 11 arthroplasties, and 10 joint manipulations were performed during the study period.²² Every arthrodesis case achieved union without incident. Overall ROM after PIP arthroplasty was limited to 20°. Malposition of spontaneous DIP joint ankylosis was treated with realignment and arthrodesis in 8 patients, each of whom experienced pain relief and improved function.²²

Synovectomy, which has a well-established role in treating patients with rheumatoid arthritis, is also a treatment option for managing painful, chronically inflamed joints involved in psoriatic arthritis. Whereas a literature review found no controlled studies for this application, several case reports described pain relief, increased ROM, and improved function after synovectomy of the knee, elbow, and wrist.⁵⁴ Fiocco and colleagues⁵⁵ reported on 18 patients with active psoriatic arthritis treated with arthroscopic knee synovectomy. At follow-up, which ranged from 2 to 36 months, the patients experienced a significant reduction in signs of joint inflammation and a significant increase in ROM. At 3 years, 50.7% of patients maintained clinical remission of knee joint synovitis.⁵⁵

Knee arthroplasty was the second most common orthopedic procedure performed in patients with psoriatic arthritis, according to the review conducted by Zangger and colleagues.⁴ In their observational study of hip disease in psoriatic arthritis, Michet and colleagues²¹ found that approximately 25% of patients who underwent hip arthroplasty also had total knee arthroplasty (50% bilateral knee replacements) performed during the study period. Outcomes after knee arthroplasty appear to be similar to those for patients with rheumatoid arthritis. Potential complications associated with the procedure include psoriasis flares at the operative site and ROM limitation caused by postoperative fibrosis.⁵³

Historically, the literature regarding surgical intervention in cases of psoriatic arthritis highlights the possibility of an increased tendency for postoperative infection, both superficial and deep, secondary to the well-known phenomenon of bacterial colonization of psoriasis plaques.⁶ The majority of recent reports did not validate this concern but instead found no significant increase in incidence of postoperative infection in the presence of meticulous preoperative skin preparation.⁵⁶ Similarly, recent investigations regarding surgery in patients with active psoriasis did not show any significant wound-healing complications.⁵⁷

Postoperative complications that are more commonly encountered in psoriatic arthritis patients, and thus of greater concern, include postoperative stiffness and development of recurrent contractures, especially in the hand.⁵⁷ These are likely related to the diffuse involvement of the surrounding soft tissues in the chronic inflammatory process, leading to significant postoperative fibrosis. The fibrosis can be extensive, leading to postoperative ankylosis in extreme cases.⁵⁷ Rehabilitation focusing on ROM maintenance is thus essential in the postoperative management of psoriatic arthritis patients. Even then, it is not uncommon for patients to require repeated soft-tissue releases to maintain flexible, functional joint motion.

SUMMARY

Psoriatic arthritis is a complex, chronic inflammatory disease with both skin and joint involvement. Clinical presentation varies considerably among patients and during the course of the disease. Assessment of patients for psori-

atic arthritis requires careful attention to patient history, a focused physical examination, and inspection for characteristic radiographic changes. Specialized assessment tools have been developed to help evaluate disease activity and to monitor patient response to therapeutic interventions. The prognosis for patients with psoriatic arthritis is as variable as the disease presentation. Currently, significant work is being conducted to try to identify biological markers for progressive disease. Nonoperative management continues to be the first line of treatment for patients affected by psoriatic arthritis, and new therapeutic agents are being developed to target specific etiologic factors in the pathogenesis of the disease. The progressive nature of the disease and the number of joints involved lead to significant orthopedic sequelae causing considerable physical morbidity and functional disability. Although recent reviews have shown that the incidence of musculoskeletal surgery in psoriatic arthritis cohorts to be relatively low, orthopedic surgery in these patients, when indicated, can successfully relieve pain and improve physical function.

AUTHORS' DISCLOSURE STATEMENT

The authors report no actual or potential conflict of interest in relation to this article.

REFERENCES

- Langley RG, Krueger GG, Griffiths CE. Psoriasis: epidemiology, clinical features, and quality of life. *Ann Rheum Dis*. 2005;64(suppl 2):ii18-ii23.
- Gladman DD, Antoni C, Mease P, Clegg DO, Nash P. Psoriatic arthritis: epidemiology, clinical features, course, and outcome. *Ann Rheum Dis*. 2005;64(suppl 2):ii14-ii17.
- Gladman DD. Psoriatic arthritis. *Dermatol Ther*. 2004;17(5):350-363.
- Zangger P, Gladman DD, Bogoch ER. Musculoskeletal surgery in psoriatic arthritis. *J Rheumatol*. 1998;25(4):725-729.
- Gladman DD. Natural history of psoriatic arthritis. *Baillieres Clin Rheumatol*. 1994;8(2):379-394.
- Zangger P, Esufali ZH, Gladman DD, Bogoch ER. Type and outcome of reconstructive surgery for different patterns of psoriatic arthritis. *J Rheumatol*. 2000;27(4):967-974.
- Veale D, FitzGerald O. Psoriatic arthritis. *Best Pract Res Clin Rheumatol*. 2002;16(4):523-535.
- Costello P, FitzGerald O. Disease mechanisms in psoriasis and psoriatic arthritis. *Curr Rheumatol Rep*. 2001;3(5):419-427.
- Gladman DD. Current concepts in psoriatic arthritis. *Curr Opin Rheumatol*. 2002;14(4):361-366.
- Mease P, Goffe BS. Diagnosis and treatment of psoriatic arthritis. *J Am Acad Dermatol*. 2005;52(1):1-19.
- Valdimarsson H, Karason A, Gudjonsson JE. Psoriasis: a complex clinical and genetic disorder. *Curr Rheumatol Rep*. 2004;6(4):314-316.
- Moll JM, Wright V. Familial occurrence of psoriatic arthritis. *Ann Rheum Dis*. 1973;32(3):181-201.
- Hohler T, Marker-Hermann E. Psoriatic arthritis: clinical aspects, genetics, and the role of T cells. *Curr Opin Rheumatol*. 2001;13(4):273-279.
- Rantakokko K, Rimpilainen M, Uksila J, Jansen C, Luukkainen R, Toivanen P. Antibodies to streptococcal cell wall in psoriatic arthritis and cutaneous psoriasis. *Clin Exp Rheumatol*. 1997;15(4):399-404.
- Muto M, Date Y, Ichimiya M, et al. Significance of antibodies to streptococcal M protein in psoriatic arthritis and their association with HLA-A*0207. *Tissue Antigens*. 1996;48(6):645-650.
- Muto M, Fujikura Y, Hamamoto Y, et al. Immune response to *Streptococcus pyogenes* and the susceptibility to psoriasis. *Australas J Dermatol*. 1996;37(suppl 1):S54-S55.
- Punzi L, Pianon M, Bertazzolo N, et al. Clinical, laboratory and immunogenetic aspects of post-traumatic psoriatic arthritis: a study of 25 patients. *Clin Exp Rheumatol*. 1998;16(3):277-281.
- Moll JM, Wright V. Psoriatic arthritis. *Semin Arthritis Rheum*. 1973;3(1):55-78.
- Wright V. Psoriasis and arthritis. *Ann Rheum Dis*. 1956;15:348.
- Krueger GG. Clinical features of psoriatic arthritis. *Am J Manag Care*. 2002;8(6 suppl):S160-S170.
- Michet CJ, Mason TG, Mazlumzadeh M. Hip joint disease in psoriatic arthritis: risk factors and natural history. *Ann Rheum Dis*. 2005;64(7):1068-1070.

22. Belsky MR, Feldon P, Millender LH, Nalebuff EA, Phillips C. Hand involvement in psoriatic arthritis. *J Hand Surg Am.* 1982;7(2):203-207.
23. Rose JH, Belsky MR. Psoriatic arthritis in the hand. *Hand Clin.* 1989;5(2):137-144.
24. Brockbank JE, Stein M, Schentag CT, Gladman DD. Dactylitis in psoriatic arthritis: a marker for disease severity? *Ann Rheum Dis.* 2005;64(2):188-190.
25. Williamson L, Dalbeth N, Dockerty JL, Gee BC, Weatherall R, Wordsworth BP. Extended report: nail disease in psoriatic arthritis—clinically important, potentially treatable and often overlooked. *Rheumatology (Oxford).* 2004;43(6):790-794.
26. Jones SM, Armas JB, Cohen MG, Lovell CR, Evison G, McHugh NJ. Psoriatic arthritis: outcome of disease subsets and relationship of joint disease to nail and skin disease. *Br J Rheumatol.* 1994;33(9): 834-839.
27. Cohen MR, Reda DJ, Clegg DO. Baseline relationships between psoriasis and psoriatic arthritis: analysis of 221 patients with active psoriatic arthritis. Department of Veterans Affairs Cooperative Study Group on Seronegative Spondyloarthropathies. *J Rheumatol.* 1999;26(8):1752-1756.
28. Bezza A, Niamane R, Amine B, El Maghraoui A, Bensabbah R, Hajjaj-Hassouni N. Involvement of the foot in patients with psoriatic arthritis. A review of 26 cases. *Joint Bone Spine.* 2004;71(6):546-549.
29. Gladman DD, Brubacher B, Buskila D, Langevitz P, Farewell VT. Differences in the expression of spondyloarthropathy: a comparison between ankylosing spondylitis and psoriatic arthritis. *Clin Invest Med.* 1993;16(1):1-7.
30. Hanly JG, Russell ML, Gladman DD. Psoriatic spondyloarthropathy: a long term prospective study. *Ann Rheum Dis.* 1988;47(5):386-393.
31. Lehtinen A, Taavitsainen M, Leirisalo-Repo M. Sonographic analysis of enthesopathy in the lower extremities of patients with spondylarthropathy. *Clin Exp Rheumatol.* 1994;12(2):143-148.
32. Taylor PW, Stoecker W. Enthesitis of the elbow in psoriatic arthritis. *J Rheumatol.* 1997;24(11):2268-2269.
33. McGonagle D, Conaghan PG, Emery P. Psoriatic arthritis: a unified concept twenty years on. *Arthritis Rheum.* 1999;42(6):1080-1086.
34. Toussrot E, Dupond JL, Wendling D. Spondylodiscitis in SAPHO syndrome. A series of eight cases. *Ann Rheum Dis.* 1997;56(1):52-58.
35. Boisseau-Garsaud AM, Beylot-Barry M, Doutre MS, Beylot C, Baran R. Psoriatic onycho-pachydermo-periostitis. A variant of psoriatic distal interphalangeal arthritis? *Arch Dermatol.* 1996;132(2):176-180.
36. Helliwell P, Marchesoni A, Peters M, Barker M, Wright V. A re-evaluation of the osteoarticular manifestations of psoriasis. *Br J Rheumatol.* 1991;30(5):339-345.
37. Ory PA, Gladman DD, Mease PJ. Psoriatic arthritis and imaging. *Ann Rheum Dis.* 2005;64(suppl 2):ii55-ii57.
38. Niamane R, Bezza A, El Hassani S, Bensabbah R, Hajjaj-Hassouni N. Value of the radiographic criteria "fingers and toes" in the early diagnosis of psoriatic arthritis [in French]. *J Radiol.* 2005;86(3):321-324.
39. Kononen M, Kilpinen E. Comparison of three radiographic methods in screening of temporomandibular joint involvement in patients with psoriatic arthritis. *Acta Odontol Scand.* 1990;48(4):271-277.
40. Battistone MJ, Manaster BJ, Reda DJ, Clegg DO. The prevalence of sacroiliitis in psoriatic arthritis: new perspectives from a large, multicenter cohort. A Department of Veterans Affairs Cooperative Study. *Skeletal Radiol.* 1999;28(4):196-201.
41. Veale D, Rogers S, Fitzgerald O. Classification of clinical subsets in psoriatic arthritis. *Br J Rheumatol.* 1994;33(2):133-138.
42. Gladman DD, Shuckett R, Russell ML, Thorne JC, Schachter RK. Psoriatic arthritis (PSA)—an analysis of 220 patients. *Q J Med.* 1987;62(238):127-141.
43. Kammer GM, Soter NA, Gibson DJ, Schur PH. Psoriatic arthritis: a clinical, immunologic and HLA study of 100 patients. *Semin Arthritis Rheum.* 1979;9(2):75-97.
44. Gladman DD, Farewell VT, Nadeau C. Clinical indicators of progression in psoriatic arthritis: multivariate relative risk model. *J Rheumatol.* 1995;22(4):675-679.
45. Wong K, Gladman DD, Husted J, Long JA, Farewell VT. Mortality studies in psoriatic arthritis: results from a single outpatient clinic. I. Causes and risk of death. *Arthritis Rheum.* 1997;40(10):1868-1872.
46. Kane D, Stafford L, Bresnihan B, Fitzgerald O. A prospective, clinical and radiological study of early psoriatic arthritis: an early synovitis clinic experience. *Rheumatology (Oxford).* 2003;42(12):1460-1468.
47. Queiro-Silva R, Torre-Alonso JC, Tinture-Eguren T, Lopez-Lagunas I. A polyarticular onset predicts erosive and deforming disease in psoriatic arthritis. *Ann Rheum Dis.* 2003;62(1):68-70.
48. Nash P, Clegg DO. Psoriatic arthritis therapy: NSAIDs and traditional DMARDs. *Ann Rheum Dis.* 2005;64(suppl 2):ii74-ii77.
49. Pipitone N, Kingsley GH, Manzo A, Scott DL, Pitzalis C. Current concepts and new developments in the treatment of psoriatic arthritis. *Rheumatology (Oxford).* 2003;42(10):1138-1148.
50. Mease P. Psoriatic arthritis: the role of TNF inhibition and the effect of its inhibition with etanercept. *Clin Exp Rheumatol.* 2002;20(6 suppl 28):S116-S121.
51. Kraan MC, van Kuijk AW, Dinant HJ, et al. Alefacept treatment in psoriatic arthritis: reduction of the effector T cell population in peripheral blood and synovial tissue is associated with improvement of clinical signs of arthritis. *Arthritis Rheum.* 2002;46(10):2776-2784.
52. Mease PJ. Current treatment of psoriatic arthritis. *Rheum Dis Clin North Am.* 2003;29(3):495-511.
53. Lambert JR, Wright V. Surgery in patients with psoriasis and arthritis. *Rheumatol Rehabil.* 1979;18(1):35-37.
54. Linschoten NJ, Krackow KA. Psoriatic arthritis of the knee treated with synovectomy. *Orthopedics.* 1993;16(11):1268-1270.
55. Fiocco U, Cozzi L, Rigon C, et al. Arthroscopic synovectomy in rheumatoid and psoriatic knee joint synovitis: long-term outcome. *Br J Rheumatol.* 1996;35(5):463-470.
56. Stern SH, Insall JN, Windsor RE, Inglis AE, Dines DM. Total knee arthroplasty in patients with psoriasis. *Clin Orthop.* 1989;(248):108-110.
57. Piro MH, Cash JM. Treatment of refractory psoriatic arthritis. *Rheum Dis Clin North Am.* 1995;21(1):129-149.

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