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Which Psoriasis Patients Develop Psoriatic Arthritis?

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Abstract

Psoriatic arthritis is a major comorbidity of psoriasis that significantly impairs quality of life and physical function. Because skin lesions classically precede joint symptoms, dermatologists are in a unique position to identify patients at risk for psoriatic arthritis before irreversible joint damage occurs. Here we review the literature to identify the clinical and genetic factors most highly associated with development of psoriatic arthritis, with the goal of assisting dermatologists in risk-stratifying their psoriasis patients.

Keywords

psoriatic arthritis; joint damage; genetic factors; clinical factors

INTRODUCTION

The prevalence of diagnosed psoriasis is reported as 3.15% of the U.S. population (95% CI 2.18–4.53%), or nearly five million adults.[1] An additional estimated 0.4% (95% CI 0.19–0.82) of U.S. adults have undiagnosed psoriasis.[1] Psoriasis is associated with many comorbidities, including cardiovascular disease, atherosclerosis, metabolic syndrome, nonalcoholic fatty liver disease, smoking, alcoholism, osteoporosis, depression, and psoriatic arthritis.[2–7]

Psoriatic arthritis is an inflammatory condition of the joints, entheses, tendon sheaths, and axial skeleton and most often occurs between the ages of 35 to 45 years.[8] A recent review of 20 epidemiologic studies found that the reported proportion of psoriatic arthritis among psoriasis patients ranges from 7% to 26%.[9] Two major obstacles to gathering precise prevalence data for psoriatic arthritis are that accurate case definitions for it are challenging given the heterogeneity of the disease[10] and that population data on both psoriasis and psoriatic arthritis are not readily available.

Nearly 60% of psoriasis patients and 40% of psoriatic arthritis patients report their disease as a large problem in their everyday life.[11,12] Psychosocial limitations of both diseases include enduring low self-esteem, feeling physically unattractive or sexually undesirable,

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and avoiding social activities.[13–15] Psoriatic arthritis patients may report embarrassment, helplessness, and depression.[15] Physical limitations of patients with this condition include mobility challenges, decreased sleep and energy, and difficulties with activities of daily living (ADLs), including climbing stairs, bathing, dressing, working, and exercising.[13–16]

Psoriatic arthritis is an inflammatory disease with the potential to cause irreversible joint damage, marked impairment on quality of life and physical function, and dissatisfaction with management. Approximately 2 years after diagnosis, psoriatic arthritis can result in radiologically identifiable joint damage in 47% of patients.[17] Skin lesions typically precede the joint symptoms.[18] One large phase IV trial found this to be true in 84% of 1,122 psoriatic arthritis patients.[19] The same study found that skin lesions were present for an average of 12 years before the onset of joint symptoms. Because psoriasis precedes psoriatic arthritis in the vast majority of cases, dermatologists are in a unique position to recognize psoriasis as a potential precursor to arthritic disease.

Despite the high prevalence of psoriasis, many dermatologists are faced with office constraints[19] that often limit the amount of time available for detailed joint assessment. Here we review potential clinical and genetic factors that can help facilitate identification of psoriasis patients at increased risk for psoriatic arthritis, with the goal of early detection and initiation of strategies aimed at joint preservation.

METHODS

We reviewed PubMed and MEDLINE articles appearing between 1950 and 2010 using the key words *psoriasis* and *psoriatic arthritis*. Additional data were obtained from the National Psoriasis Foundation website and medical textbooks.

RESULTS

Patient History

Psoriasis severity—Four large cohort studies have shown that psoriasis patients with more severe skin disease are at increased risk for psoriatic arthritis. First, a multicenter, cross-sectional, prospective cohort study of 1,511 adult psoriasis patients from Germany compared the clinical characteristics of patients with psoriatic arthritis versus those without psoriatic arthritis.[20] Factors significantly associated with increased psoriatic arthritis risk included several markers of psoriasis severity, including earlier age at diagnosis, increased Psoriasis Area and Severity Index and Dermatology Quality of Life Index scores, increased hospitalizations for psoriasis within the last 5 years, and more workdays lost in the past 12 months. Second, a longitudinal, retrospective, population-based cohort study of 1,633 psoriasis patients from Minnesota reported that having more than three body sites affected by psoriasis (i.e., compared with only one site) was associated with a 2.24-fold increased risk of psoriatic arthritis.[21] Third, a case series of 943 psoriasis subjects from Utah (26.5% of whom had psoriatic arthritis) reported that two clinical characteristics statistically associated with psoriatic arthritis were age of psoriasis onset and worst-ever body surface area (BSA) affected.[18] Finally, a study of 400 psoriasis patients from Singapore showed that

psoriatic arthritis was associated with maximum body surface involved, but not with sex, race, age of onset of psoriasis, smoking, or alcohol consumption.[22]

Family history—Classically, the genetic heritability of a given disease can be estimated using twin studies. For psoriatic arthritis, one twin study from Denmark did not find a difference in concordance rates between monozygotic and dizygotic twins; however, this study was underpowered.[23] Moll and Wright's historical analysis found that the risk of psoriatic arthritis was 48.8 times higher among first-degree relatives of psoriatic arthritis patients compared with the general population.[24] Other studies have confirmed a strong genetic component to psoriatic arthritis.[10,24–28] Of note, psoriatic arthritis is thought to have an even stronger genetic heritability than psoriasis as measured by the sibling recurrence risk.[29] Although these studies show that a family history of psoriatic arthritis increases the risk of psoriatic arthritis, the question of whether a family history of psoriasis alone will increase psoriatic arthritis risk is not yet clear. Reich et al.[20] showed that a positive family history of psoriasis was associated with increased risk of psoriatic arthritis, and Tey et al.[22] identified a similar trend ($P = 0.09$), but these results were not replicated in another study.[18]

Symptoms—Evidence of psoriatic arthritis may be overlooked in routine dermatology office visits, when psoriasis patients are focused on describing their skin symptoms. Psoriatic arthritis is associated with pain, morning stiffness, fatigue, and difficulty performing ADLs. Pain manifests as tenderness over affected joints, with or without swelling. Because a large proportion of psoriatic arthritis patients have cervical spondylitis (60%) and sacroiliitis (30%–78%),[30,31] clinicians should inquire about neck or back pain, which is typically worse during rest and improved with activity. Morning stiffness for longer than 1 hour was found to differentiate psoriatic arthritis from osteoarthritis (OA).[32] Fatigue is a common symptom; Husted and colleagues reported in a study of 499 psoriatic arthritis patients that moderate fatigue was present in 49.5% of patients and severe fatigue in 28.7% of patients.[16] Psoriatic arthritis patients often have difficulty with ADLs.[13–15] Krueger et al. reported that 66% of psoriatic arthritis patients have difficulty using their hands, 64% report difficulty standing for long durations, and 63% have difficulty walking.[14]

Physical Examination

Skin assessment—Despite the overall association between severity of skin disease and presence of psoriatic arthritis, they may not be temporally related, as psoriasis flares do not always precede psoriatic arthritis flares.[33] The location of psoriasis, however, may help identify risk. One study identified a 3.89-fold increased psoriatic arthritis risk in psoriasis patients with scalp lesions and a 2.35-fold increased risk in those with intergluteal or perianal lesions.[21] The authors hypothesized that an abundance of microbial flora at these sites could trigger an immune reaction leading to psoriatic arthritis. The association of psoriatic arthritis with scalp psoriasis was also found to be significant in a retrospective cohort study of 162 psoriatic arthritis patients.[34]

Nail assessment—Psoriatic nail changes include pitting, onycholysis, onychoschizia, subungual hyperkeratosis, and pathognomonic oil spots. Multiple studies have confirmed that psoriatic nail involvement is a risk factor for psoriatic arthritis.[18,21,35–39] One large

study documented nail changes in 68.6% of psoriasis patients with psoriatic arthritis compared with only 40.5% of psoriasis patients without psoriatic arthritis.[20] Similarly, Ejaz et al.[40] reported in a study of 100 psoriasis patients that nail involvement was present in 74% of those with psoriatic arthritis and in only 29% of those without psoriatic arthritis. Whereas it remains unclear why psoriatic nail changes are associated with psoriatic arthritis, recent microanatomic studies suggest that one reason may be that the nail matrix is in close physical proximity to the extensor tendon insertion point to the bone of the distal phalanx. This enthesis site demonstrates inflammation in psoriatic arthritis and is subject to Koebner-like microdamage.[41]

Joint assessment—Peripheral joints affected by psoriatic arthritis may be tender to palpation and may display an overlying purplish hue. Because of the tight capsule, swelling may be present or absent. Psoriatic arthritis is often characterized by a ray pattern in which all the joints of a single digit are affected, in contrast to rheumatoid arthritis (RA), in which often the same joints on both sides are affected. Involvement of the distal interphalangeal (DIP) joints, though not always present, can also help distinguish psoriatic arthritis from RA. DIP involvement is also common in OA, but clinical signs of joint inflammation are less common in OA.

With regard to joint distribution, Moll and Wright[42] defined the classification of psoriatic arthritis based on presentation into five subtypes: (1) arthritis predominantly involving DIP joints; (2) arthritis mutilans; (3) a symmetric polyarthritis involving five or more joints; (4) an asymmetric oligoarthritis affecting fewer than five DIP joints, proximal interphalangeal joints, and metacarpophalangeal joints; and (5) arthritis with or without peripheral joint involvement where axial spine disease is the principal characteristic. An asymmetric oligoarthritis was described as the most common presentation.[42] Since this original report, a number of studies have investigated the most common presenting joint symptoms in psoriatic arthritis. In agreement with Moll and Wright, most studies identified oligoarthritis as the most common initial presentation, with polyarthritis found to be more common later in the disease course.[8,21,34,43–45] However, two studies, one from Germany and one from Sweden, found polyarthritis to be more common at initial presentation.[20,46] Discrepancies between oligoarticular and polyarticular initial presentation could arise from differences in the time to diagnosis from psoriatic arthritis onset or from population heterogeneity. An interesting finding was that polyarticular onset predicted more severe disease in some studies.[19,47,48]

Patients with psoriatic arthritis may also present with enthesitis, defined as inflammation of the insertion sites of tendons or ligaments onto bone. In one large study, the most common sites of enthesopathy were the plantar fascia (9%), finger flexor tendons (7%), and Achilles tendon (7%).[21] Dactylitis describes inflammation of the entire digit, commonly referred to as a “sausage digit.”[49] Dactylitis is highly specific for psoriatic arthritis and is rarely encountered in RA.[50,51] Notably, one study showed that dermatologists have poor interobserver agreement about the presence of dactylitis compared with rheumatologists,[52] suggesting that dermatologists may benefit from additional training to recognize the clinical signs of dactylitis.

Laboratory Evaluation

Characteristic laboratory abnormalities in psoriatic arthritis are few. Elevated erythrocyte sedimentation rate (ESR), complement levels, and C-reactive proteins (CRP) signify inflammation, whereas immunoglobulin levels are often normal even in active psoriatic arthritis.[53] Rheumatoid factor (RF) is detected in 5% to 9% of patients, with a high false-positive rate and low diagnostic significance when used alone.[31,54,55] Additionally, the absence of RF is 95% sensitive and 60% specific for psoriatic arthritis.[10] ESR may correlate to clinical joint severity based on the presence of inflammation.[31,54–56]

More recently, Gladman et al.[57] executed a post hoc analysis of the ADEPT (Adalimumab Effectiveness in Psoriatic Arthritis Trial vs placebo) trial to identify independent predictors for radiographic progression in psoriatic arthritis. Elevated baseline CRP and time-averaged CRP were strongly associated with radiographic progression for patients in the placebo group. Further multivariate analysis confirmed that elevated baseline CRP was the strongest independent risk factor for radiographic progression in psoriatic arthritis (OR 3.28, 95% CI 1.66–6.51, $P < 0.001$).

Imaging

Radiologic findings in psoriatic arthritis, primarily consisting of erosive changes and juxta-articular new bone formation, are seen in two thirds of patients consulting rheumatologists.[58] Early radiologic diagnosis of psoriatic arthritis is often hindered by features common to other arthritic conditions, such as soft tissue swelling, decreased cartilage space, bony erosions and ankylosis, subluxations, and subchondral cysts.[53] Devauchelle-Pensec et al.[59] reported a study of 258 patients with early arthritis and determined that baseline hand radiographs were useful in predicting all-cause arthritic diagnoses in only 12% of patients. Moreover, the radiographs identified 0% of patients with psoriatic arthritis. After a 2-year follow-up, 19.3% of patients were given the diagnosis of psoriatic arthritis (or other spondyloarthropathy).[59]

In psoriatic arthritis, enthesitis appears by magnetic resonance imaging (MRI) as extracapsular inflammation at the insertions of ligaments and tendons in addition to bone edema.[60,61] Marzo-Ortega and colleagues[62] described a study of 10 patients with RA and 10 patients with psoriatic arthritis and found that dynamic contrast-enhanced MRI was unable to differentiate between RA and psoriatic arthritis on the basis of entheseal-related disease, although 30% of psoriatic arthritis patients had diffuse extracapsular enhancement and 20% had diffuse bone edema. A scoring system based on MRI findings, the Rheumatoid Arthritis Magnetic Imaging Scoring (RAMRIS) system, is in development for evaluation of peripheral psoriatic arthritis, but standardization is not complete.[60]

A number of studies have shown that ultrasound can be used to detect entheseal abnormalities in psoriasis or psoriatic arthritis patients with no clinical signs of enthesitis.[63–66] However, the use of ultrasonography as a screening tool for psoriatic arthritis has not been demonstrated.

Genetic Markers of Psoriatic Arthritis

In the last 3 years, significant progress has been made toward identifying genetic markers associated with psoriatic arthritis. However, most of these markers are also associated with psoriasis, and thus a major remaining challenge is to identify genetic risk factors that are specific to psoriatic arthritis.

HLA locus—The HLA locus contains many genes responsible for immunologic function in humans. Psoriasis and psoriatic arthritis demonstrate human leukocyte antigen (HLA) associations, most frequently with HLA-Cw6.[29,67–69] However, the HLA region may potentially harbor multiple genes that are associated with psoriasis and psoriatic arthritis. For psoriasis, Feng et al.[70] recently identified three independent signals near the HLA locus that contribute to psoriasis risk: HLA-Cw6, c6orf10, and the region between HLA-B and MICA.

Although psoriatic arthritis is most strongly associated with HLA-Cw6, other HLA markers have also been associated with it. The frequency of HLA-B27 is reportedly higher among patients with psoriatic arthritis.[28,71,72] Gladmann and Farewell[71] performed a univariate analysis of psoriatic arthritis patients and showed that HLA-B27, HLA-B39, and HLA-DQw3 antigens were associated with psoriatic arthritis disease progression, whereas HLA-DR7 was protective. Additionally, the presence of HLA-B39 suggested early progression in psoriatic arthritis.

MICA locus—The HLA-Cw6 association is common among psoriasis patients whether or not they have concurrent arthritic disease. However, in 1999 the MICA-A9 polymorphism, corresponding to the MICA-002 allele, was introduced as an independent risk factor for the development of psoriatic arthritis in patients who carry Cw6.[73] The *MICA* gene is located on chromosome 6p between HLA-B and MICB. The association of *MICA-A9* with psoriatic arthritis was confirmed by several additional studies.[74–77]

Other genetic loci—Recent studies have demonstrated that variants within or near IL-12B, 1L-23R, TNIP1, IL-13, TRAF3IP2, NOS2, FBXL19, and NFKBIA are associated with psoriatic arthritis.[78–82] In all these cases, these variants were first found to be associated with psoriasis and then confirmed to be associated with psoriatic arthritis in a subgroup analysis. One study[80] found that polymorphisms within IL-13 were more strongly associated with psoriatic arthritis than with psoriasis; however, replication of this observation has not yet been reported. Interestingly, some of these gene associations cluster to specific biological pathways. TNIP1 and NFKBIA serve as regulators of the NF-KB pathway (downstream of tumor necrosis factor-alpha [TNF-α]) and IL-12B, IL-23R, and TRAF3IP2 are involved in activation of the Th17immune pathway. Biologics that inhibit TNF-α (etanercept, adalimumab, infliximab, golimumab) or IL12/23 (ustekinumab) are effective in the treatment of psoriatic arthritis.

DISCUSSION

Two overlapping but distinct challenges exist for rheumatologists and dermatologists with regard to the diagnosis of psoriatic arthritis. Rheumatologists are often charged with

assessing whether patients meet the criteria for definitive diagnosis of psoriatic arthritis (high specificity desired). Dermatologists, on the other hand, are charged with identifying those psoriasis patients at highest risk for psoriatic arthritis, with the goal of monitoring and early referral to their rheumatology colleagues (high sensitivity and moderate specificity desired).

With regard to the first challenge, the original diagnostic criteria of Moll and Wright (inflammatory arthritis, presence of psoriasis, and negative RF) are the simplest and have been the most frequently used.[42,83] Recently, the CASPAR (Classification of Psoriatic Arthritis) study group[10] proposed more specific, evidence-based criteria for the diagnosis of psoriatic arthritis, defined as established inflammatory articular disease with at least 3 points from the following: current psoriasis (2 points), history of psoriasis without current psoriasis (1 point), family history of psoriasis without current psoriasis or history of psoriasis (1 point), dactylitis (1 point), juxta-articular new bone formation (1 point), RF negative (1 point), and nail dystrophy (1 point). This classification system yielded results that were 98.7% specific and 91.4% sensitive for psoriatic arthritis.[10] Recently, Congi and Roussou[84] compared the CASPAR criteria in a group of 69 psoriatic arthritis patients and determined that family history was the main advantage of the new criteria over Moll and Wright and other classification systems. The authors were encouraged that under these new criteria, it was possible to diagnose psoriatic arthritis accurately, even with positive RF and predominantly polyarticular symmetrical arthritis presentations. The CASPAR criteria were also applied in a small family medicine clinic setting with good sensitivity.[83] Further study of the validation and utility of the CASPAR criteria in recent-onset disease as a psoriatic arthritis screening tool in clinical practice is required.

With regard to the second challenge, stratification of high-risk psoriasis patients by dermatologists, this literature review shows that there are several aspects of the clinical encounter that may be useful in predicting risk of psoriatic arthritis. With respect to patient history, a history of severe psoriasis—as measured by affected BSA, hospitalizations, missed work, or low quality of life—is associated with development of psoriatic arthritis.[18,20–22] It should be noted that although overall psoriasis severity as measured longitudinally over time is associated with psoriatic arthritis risk, correlation between skin and joint severity at a specific time point may be low. For example, it is not uncommon for a patient to have mild psoriasis and severe arthritis and vice versa. The risk of psoriatic arthritis is increased with a positive family history of psoriatic arthritis[10,24–28] and possibly also a positive family of history of psoriasis,[20,22] although more confirmatory studies are needed for the latter. Additionally, a history of joint pain, neck or back pain, morning stiffness, fatigue, and difficulty with ADLs should be considered potential clues to psoriatic arthritis.

The physical examination is an important step when assessing for risk of psoriatic arthritis. Nail dystrophy and dactylitis are high-yield risk factors for psoriatic arthritis because the presence of either of these in a psoriasis patient with inflammatory arthritis qualifies the patient for a diagnosis of psoriatic arthritis according to the CASPAR criteria. It is intriguing that the location of a patient's psoriasis might also influence risk of psoriatic arthritis, with scalp, intergluteal, or perianal lesions conferring higher risk.[21,34] Although many

dermatologists may not have the time or experience to perform a detailed rheumatologic examination on their psoriasis patients, knowledge that inflammatory involvement of the DIP joints is somewhat specific for psoriatic arthritis and that asymmetric oligoarthritis and polyarthritis are the most common initial presentations may aid in early diagnosis.

Dermatologists should also realize that the presence of joint pain in a patient with psoriasis does not necessarily imply a diagnosis of psoriatic arthritis. Psoriatic arthritis must be distinguished from a broad differential that includes OA, RA, gout, ankylosing spondylitis, and reactive arthritis. Mody et al.[32] reviewed the records of 94 psoriasis patients seen in a dermatology-rheumatology clinic to determine the cause of musculoskeletal pain. Results were as follows: 41% of patients had psoriatic arthritis, 27% had OA, 15% had psoriatic arthritis + OA, 13% indeterminate, 2% gout, 1% psoriatic arthritis + gout, and 1% OA + gout.

Regarding laboratory evaluation, baseline CRP has emerged as an independent marker of risk of radiographic joint progression in cases of established psoriatic arthritis.[57] In patients with inflammatory joint disease, a negative RF can help exclude RA, and ESR may track psoriatic arthritis progression. The usefulness of either CRP or ESR as a psoriatic arthritis screening tool has not been established.

Radiographic features such as pencil-in-cup deformities or asymmetric erosive changes in the small joints of the hands and feet are typically seen in established psoriatic arthritis. The presence of juxta-articular bone formation is an important criterion for the diagnosis of psoriatic arthritis under the CASPAR system because it differentiates psoriatic arthritis from RA. Although MRI and ultrasound may detect early evidence of enthesitis-associated pathology, this is not sufficient for diagnosis. Standardization of MRI scoring systems for psoriatic arthritis is still in progress. Radiographs may be potentially helpful for confirmation of psoriatic arthritis, but currently they are minimally helpful as screening tools.[59]

Because HLA-Cw6, IL12B, IL23R, and other newly discovered genetic variants are associated with both psoriasis and psoriatic arthritis, they cannot be used as a marker to predict which psoriasis patients are likely to develop psoriatic arthritis. The demonstration in several studies[73–77] that the MICA-A9 allele may serve as an independent marker of psoriatic arthritis is intriguing and deserves further study. Just as genome-wide association studies have greatly furthered the identification of psoriasis risk variants,[29,85,86] genome-wide association studies currently under way in psoriatic arthritis (where psoriatic arthritis “cases” are compared with psoriasis-only “controls”) will likely identify additional genetic risk factors specific for psoriatic arthritis.

Often the demand for dermatologic care exceeds the available supply of physicians.[87] Office screening tools and questionnaires may assist dermatologists with psoriatic arthritis risk stratification. The 2007 annual Group for Research and Assessment of Psoriasis and Psoriatic arthritis (GRAPPA) suggested that the most rigorous methods for validation of psoriatic arthritis include the Toronto Psoriatic Arthritis Screening (ToPAS) tool[88] and the

Psoriatic Arthritis Screening and Evaluation (PASE) tool,[89] which has demonstrated good sensitivity.[90]

Because of time constraints, dermatologists may benefit from distributing these screening questionnaires to patients in the waiting room. Patients who score high on these instruments may be given a targeted assessment for risk of psoriatic arthritis (Table 1). A targeted assessment for psoriatic arthritis should consist of the following: categorization of overall psoriasis severity; notation of positive family history of psoriasis or psoriatic arthritis; review of psoriatic arthritis symptoms, including joint/neck/back pain, morning stiffness, fatigue, and difficulty with ADLs; inspection of the scalp, gluteal, and perianal areas for psoriasis; examination for nail dystrophy, dactylitis, or swollen/tender joints; and for patients with evidence of an inflammatory arthritis, laboratory request for CRP and RF. Based on the results of this targeted assessment, referral to a rheumatologist may be initiated. An ongoing relationship with a rheumatologist is vital to ensuring optimal care and judicious follow-up.[91,92]

CONCLUSION

Early identification of psoriatic arthritis is necessary for preservation of quality of life and physical function among psoriasis patients. Available literature suggests that the highest yield clinical features indicating increased risk for psoriatic arthritis include the following: increased psoriasis severity; positive family history for psoriasis or psoriatic arthritis; patient history of musculoskeletal pain, morning stiffness, fatigue, and difficulty with ADLs; presence of scalp, intergluteal, or perianal psoriasis; nail dystrophy; and dactylitis. Based on the individual patient's circumstances, further management options include close dermatologic monitoring or referral to a rheumatologist for further examination, laboratory testing, and radiologic assessment.

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Table 1

Recommendations for Targeted Assessment of Psoriatic Arthritis

Targeted Assessment to Assess Risk of Psoriatic Arthritis
Patient History • Severe psoriasis (high BSA, hospitalizations, missed work, low quality of life) • Positive family history of psoriasis * or psoriatic arthritis • Joint, neck, or back pain • Morning stiffness, especially lasting >1 h • Fatigue • Difficulty with ADLs (e.g., getting dressed, brushing teeth, getting into car)
Physical Examination • Current psoriasis * • Presence of psoriasis in scalp, gluteal, or perianal regions • Psoriatic nail changes * • Dactylitis * • Inflamed, swollen, or tender joints * (especially DIP)
Laboratory Tests (if evidence of inflammatory arthritis) • C-reactive protein (CRP) • Rheumatoid factor (RF) *

ADLs = activities of daily living; BSA = body surface area; DIP = distal interphalangeal (joint)

* Indicates component of CASPAR criteria for diagnosis of psoriatic arthritis