Clinical Practice Guideline

Psoriatic Arthritis (PsA)

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Abbreviations

ANA	Antinuclear antibodies
AS	Ankylosing spondylitis
BSA	Body Surface Assessment
CASPAR	ClASsification Criteria for Psoriatic Arthritis
CBC	Complete blood count
CMP	Comprehensive metabolic panel
CRP	C-reactive protein
CV	Cardiovascular
CVD	Cardiovascular disease
DMARDs	Disease-modifying antirheumatic drugs
ESR	Erythrocyte sedimentation rate
GI	Gastrointestinal
HBV	Hepatitis B virus
HCV	Hepatitis C virus
HIV	Human immunodeficiency virus
HLA-B27	Human leukocyte antigen-B27
IBD	Inflammatory bowel disease
IL	Interleukin
MDA	Minimal disease activity
MDG	Physician Global Assessment
MDHAQ	Multidimensional Health Assessment Questionnaire
NSAIDs	Nonsteroidal anti-inflammatory drugs
PASI	Psoriasis Area and Severity Index
PDE4i	Phosphodiesterase 4 inhibitor
PGA	Patient Global Assessment
PsA	Psoriatic arthritis
QoL	Quality of life
RA	Rheumatoid arthritis
RF	Rheumatoid factor
T2T	Treat to target
TNFi	Tumor necrosis factor inhibitor
VAS	Visual analog scale

Introduction

In the United States, approximately 3% of the general population (or 5 million adults) have been diagnosed with psoriasis. Between 0.4% and 2.28% of the population is reported to have undiagnosed disease.[1]

Psoriasis is a chronic inflammatory autoimmune disease of the skin, often presenting with patches of silvery scales on the skin and epidermal hyperplasia. Patients complain of dryness, itching, redness, soreness, and even pain in the affected areas. The skin overlying the elbows, knees, scalp, lower back, face, palms, and soles of the feet are the most commonly affected areas. Skin disease is often marked by unpredictable remissions and flares. Epidermal hyperplasia is a response to the activation of the immune system mediated by $CD8^+$ and $CD4^+$ T lymphocytes.[2]

Psoriatic arthritis (PsA) is an autoimmune inflammatory arthritis that affects up to 30% of the patients with skin psoriasis. It is most frequently diagnosed between the ages of 30 and 50 years and has a chronic progressive course. Found equally in men and women, PsA can be very disabling and negatively impact the quality of life (QoL) of those affected. In a 2005 report in the Journal of the American Academy of Dermatology, 39% of the patients with PsA indicated that the disease was a significant or large problem in their everyday lives and 38% that it was a problem; only 23% reported that it was a small problem or no problem in daily life.[3] Psoriatic nail lesions are seen in 80% to 87% of patients with PsA.[4, 5]

Data on the economics of caring for patients with PsA is poor. It is often included in the cost of caring for patients with psoriasis that include those with and without PsA. In addition, much of the data were generated prior to the introduction of effective but costly biologics. Brezinski et al.[6] published a systematic review of the overall costs of caring for patients with psoriasis and adjusted the base-year costs to 2013 dollars. They found that the direct costs were between $51.7 billion and $63.2 billion, with indirect costs ranging from $23.9 billion and $35.4 billion. Caring for comorbidities contributed another $36.4 billion. A paper published in 2010[7] looked at the costs of caring for patients with PsA and estimated them to be as high as $1.9 billion in 2000, before the widespread availability of effective biologic drugs. In addition, patients with PsA often had a decreased QoL, with limited ability to perform activities of daily living. They showed diminished productivity at work, had increased absenteeism, and were less likely to be employed than people without PsA. Patients with PsA with skin and/or nail psoriasis may also experience decreased self-esteem and self-consciousness, which can lead to depression.

It is widely believed that both psoriasis and PsA are complex genetic autoimmune disorders; however, the heritability of either disease is not clearly understood.[8]

Psoriatic arthritis usually presents with joint pain and swelling, erythema, and warmth around the affected joint(s). Patients may also complain about joint stiffness. In addition, painful swelling and tenderness at the enthesis (bony insertion of ligaments, tendons, or joint capsules) is common. Enthesitis (inflammation of the enthesis) most commonly occurs at the insertion of the plantar fascia, Achilles tendon, and around the elbow but can also be seen at the ligamentous attachments of the knees, ribs, spine, pelvis, and many other areas of the body.

Psoriatic arthritis can be oligoarticular (few joints) or polyarticular (many joints) and often involves the distal interphalangeal joints of the hands and feet. If these joints are involved, nail psoriasis is almost always present.[9] Dactylitis—a combination of enthesitis, tenosynovitis, and arthritis of all the joints of a single digit—is seen in up to 40% of patients with PsA. Clinically, there is diffuse swelling of a digit or digits. Psoriatic arthritis is often asymmetric in distribution helping to distinguish it from rheumatoid arthritis (RA), which is more commonly symmetric and less likely to involve the distal interphalangeal joints. The distinction between PsA and RA is based on clinical and laboratory data. In addition to the small joints of the hands and feet, large joints of the lower extremities, spine, sacroiliac joints, and pelvis may be affected by PsA. Approximately 40% of patients with PsA will have spinal involvement causing back pain and progressive ankylosis similar to ankylosing spondylitis (AS).[8] Sometimes, it is difficult to differentiate PsA from other rheumatic diseases, other types of arthritis and mechanical tendonitis and fibromyalgia. Therefore, it is essential for patients to have a complete evaluation by a rheumatologist, and if there is significant skin involvement, a dermatologist. All PsA domains should be evaluated; including signs and symptoms of peripheral arthritis, psoriasis, enthesitis, dactylitis, spondylitis (spine and sacroiliac joints), and nail disease. In addition, it has now become clear that patients with PsA have systemic inflammation with multiple organ manifestations leading to an increased risk of cardiovascular disease (CVD), inflammatory bowel disease (IBD), ocular inflammation, and others. When these are present co-management with physicians of other specialties is recommended.

Diagnosis

The CASPAR (ClASsification Criteria for Psoriatic Arthritis) criteria outlined below (Figure 1) should be used to establish the diagnosis of PsA. These criteria, published in 2006, are simple to use and have a sensitivity of 91.4% and specificity of 98.7%.[10]

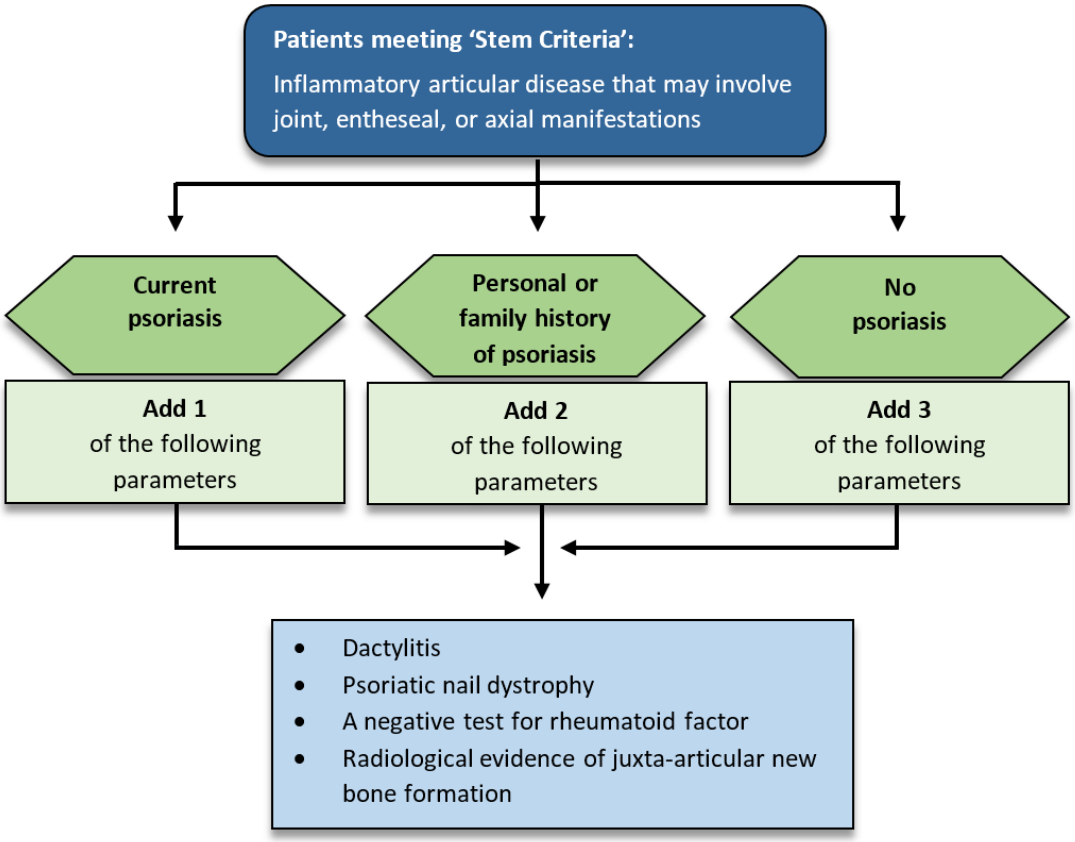

Figure 1. CASPAR criteria for the diagnosis of PsA

CASPAR, ClASsification criteria for Psoriatic Arthritis; PsA, psoriatic arthritis
Adapted from Taylor WJ, et al. Arthritis Rheum 2006;54(8):2665-2673.

Patient Assessment

Initial evaluation of a patient who meets the CASPAR criteria for PsA should include the following:

- Detailed medical history including a detailed vaccination history
- Laboratory tests
  - Complete blood count (CBC)
  - C-reactive protein (CRP)
  - Erythrocyte sedimentation rate (ESR)
  - Comprehensive metabolic panel (CMP)
    - Liver function
    - Renal function
    - Electrolytes
    - Glucose
  - Screening for hepatitis B (HBV) and hepatitis C (HCV)
  - Rheumatoid factor (RF)

- Screening for human immunodeficiency virus (HIV) in appropriate patients; baseline imaging is encouraged in areas of clinical involvement
- Patient Global Assessment (PGA)
- Patient pain VAS (visual analog scale)
- Multidimensional Health Assessment Questionnaire (MDHAQ; see Glossary).
- Physician Global Assessment (MDG)
- Number of tender joints
- Number of swollen joints
- If there is skin psoriasis, Psoriasis Area and Severity Index (PASI; see Glossary) or Body Surface Area (BSA) assessment

At this time, there is no confirmatory laboratory test to establish the diagnosis of PsA,[11] but many patients test positive for human leukocyte antigen-B27 (HLA-B27), and negative for RF (by any method other than latex fixation) and antinuclear antibodies (ANA).

Active disease is diagnosed in patients (who have met the CASPAR criteria above) with any of the following:[12]

- $\geq$ 1 tender and inflamed joint(s)
- $\geq$ 1 tender enthesis point(s)
- $\geq$ 1 dactylitic digit(s)
- Inflammatory back pain (IBP, see Glossary)

Patients are considered to have a **poor prognosis** if they have at least 1 of the following:[13]

- $\geq$ 5 actively inflamed joints
- Elevated acute-phase reactants such as ESR or CRP
- Imaging evidence of disease progression
- Poor response to nonsteroidal anti-inflammatory drug (NSAID) therapy or prior use of steroids
- Loss of function or diminished QoL

Management of Patients with PsA

The management of patients with PSA should be based on a treat-to-target (T2T) paradigm, with therapy aimed at achieving remission or minimal disease activity (MDA). Early diagnosis and treatment help reach these goals. Regular close monitoring and re-evaluation of therapy are essential to maintaining stability of the disease, avoiding medication toxicity, and identifying comorbid conditions early.

Remission is defined as no evidence of active disease (see 'Patient Assessment', above). To achieve MDA, the patient must meet 5 out of the following 7 conditions, with each condition equal to 1 point:

1. Tender joints ≤1
2. Swollen joints ≤1
3. Pain VAS ≤15
4. Patient Global Assessment ≤20
5. MDHAQ
6. PASI <1 or BSA ≤3
7. Enthesitis ≤1

When selecting pharmacologic therapy, comorbidities must be considered. These include but are not limited to:[14]

- Uveitis
- IBD
- CVD
- Obesity, metabolic syndrome
- Diabetes
- Depression
- Chronic hepatitis
- Non-alcoholic fatty liver disease, cirrhosis
- Chronic alcohol abuse
- Renal disease
- Malignancies, including skin cancer
- Osteoporosis
- Central sensitization syndrome (fibromyalgia)
- Interstitial lung disease
- Recurrent or increased susceptibility to infections

Due to dysregulated immune function and exposure to immunomodulating medications, patients with PsA have an increased risk of infection. Prior to starting therapy, it is important to obtain a detailed vaccination history. All patients with PsA treated with biologics should have a pneumococcal vaccination according to the CDC-recommended schedule, and an annual flu vaccination. Psoriatic arthritis patients with risk factors for HBV infection being treated with disease-modifying antirheumatic drugs (DMARDs, see Glossary) or biologics should be vaccinated for HBV, if this has not been done previously. Herpes zoster vaccination (live virus) should not be given to patients who are currently being treated with biologics. However, if the patient has not yet started treatment with a biologic and is 50 years of age or older then a herpes zoster vaccination should be given. Biologics should not be administered for 2 to 4 weeks after the herpes zoster vaccination. The date and result of most recent tuberculosis (TB) evaluation should be documented and re-screening for TB in 1 year. A travel history to areas where certain fungal diseases are prevalent is also important.

Peripheral Arthritis

For DMARD-naïve patients with peripheral joint involvement, pharmacologic treatment should be started immediately upon confirmation of the diagnosis. Initial and ongoing symptom control may be attempted with NSAIDs at the lowest possible dose and for the shortest duration that is clinically appropriate, to avoid the well-known gastrointestinal (GI) and cardiovascular (CV) complications of these medications. Similarly, intra-articular injection of corticosteroids may be helpful initially or during the course of management if very few joints are involved. However, in all patients with active disease, DMARDs should be started. Methotrexate is the initial drug of choice for these patients if skin disease is present. For those with poor prognostic factors (see 'Patient Assessment', above) a tumor necrosis factor inhibitor (TNFi) should be prescribed as the initial drug or, if DMARDs fail to rapidly improve or stabilize the patient, rapid escalation to a TNFi is recommended (Table 1).[13-15]

Table 1. Pharmacologic treatment of peripheral PsA in DMARD-naïve patients

Drug*	Comments
NSAIDs **Oral or intra-articular steroids**	• Oral corticosteroids may be used at a low dose and for the shortest time possible to avoid toxicity and a potential psoriasis flare
DMARDs Methotrexate Leflunomide Sulfasalazine	• Methotrexate is preferred, especially if there is skin disease
PDE4i Apremilast	• Caution should be used in patients with depression
TNFi Infliximab Etanercept Adalimumab Golimumab Certolizumab pegol	• Direct use of TNFi in patients with poor prognostic factors should be considered

*If available, generic medications are preferred.

DMARDs, disease-modifying antirheumatic drugs; NSAIDs, nonsteroidal anti-inflammatory drugs; PDE4i, phosphodiesterase 4 inhibitor; PsA, psoriatic arthritis; TNFi, tumor necrosing factor inhibitor

Patients with peripheral PsA and either active disease or poor prognostic factors (see 'Patient Assessment', above) who have inadequately responded to DMARDs should receive a TNFi. If a patient has failed a TNFi, a different TNFi should be tried. In patients treated with infliximab, dose escalation can be considered prior to changing to another TNFi or changing to a biologic with a different mechanism of action. Another alternative for these patients are the interleukin inhibitors, ustekinumab and secukinumab. The phosphodiesterase 4 inhibitor (PDE4i) apremilast may be an alternative in patients who have inadequately responded to DMARDs but should be used only in appropriate situations (Table 2).[13-15]

Table 2. Pharmacologic treatment of patients with peripheral PsA and an inadequate response to DMARDs

Drug*	Comments
TNFi Infliximab Etanercept Adalimumab Golimumab Certolizumab pegol	• Use in patients who have either active disease or poor prognostic factors and have failed DMARDs
DMARDs Methotrexate Leflunomide Sulfasalazine	• Consider in patients without poor prognostic factors • Methotrexate is preferred if skin disease is present
IL inhibitors Ustekinumab (IL-12, IL-23) Secukinumab (IL-17)	
PDE4i Apremilast	• Caution should be used in patients with depression • Use in patients with mild to moderate PsA and mild skin disease who have an inadequate response or contraindication to methotrexate • Consider use in patients with contraindications to biologic therapies

*If available, generic medications are preferred.

DMARD, disease-modifying antirheumatic drug; IL, interleukin; PDE4i, phosphodiesterase 4 inhibitor; PsA, psoriatic arthritis; TNFi, tumor necrosing factor inhibitor

Spondylitis and Sacroiliitis (axial PsA)

Axial disease rarely occurs alone; it is seen more frequently in combination with peripheral PsA. Inflammatory back pain or imaging findings of sacroiliitis establish the diagnosis of axial disease. Initial treatment for this domain should begin with NSAIDs and physical therapy. Sacroiliac joint injections can be considered in appropriate situations. Disease-modifying antirheumatic drugs are not indicated for the treatment of patients with poor response to NSAIDs. For patients failing to respond to NSAIDs, biologics are indicated (Table 3).[14, 16]. In patients treated with infliximab, dose escalation can be considered prior to changing to another TNFi or changing to a biologic with a different mechanism of action.

Table 3. Pharmacologic treatment of spondylitis

Drug*	Comments
NSAIDs	• Should be combined with physical therapy
TNFi Infliximab Etanercept Adalimumab Golimumab Certolizumab pegol	• Should be combined with physical therapy • DMARDs should not be used
IL Inhibitors Secukinumab Ustekinumab	

*If available, generic medications are preferred.

DMARDs, disease-modifying antirheumatic drugs; IL, interleukin; NSAIDs, nonsteroidal anti-inflammatory drugs; TNFi, tumor necrosing factor inhibitor

Enthesitis

Inflammation at the attachments of ligaments, tendons, and joint capsules to the bone is quite common in PsA. In fact, enthesitis and/or dactylitis may be the initial presenting complaint or symptom, or the only complaint or symptom in some patients.[17] According to expert consensus, NSAIDs should be started initially. These medications should be used at the lowest possible dose and for the shortest duration that is clinically appropriate, to avoid the well-known GI and CV complications of these medications. Physiotherapy should be tried initially as well. Local steroid injections should be used with extreme caution because data suggest potential structural damage and rupture of the enthesis.[18] Disease-modifying antirheumatic drugs are not recommended for patients whose main domain is enthesitis.[18] If there is an inadequate response to NSAIDs, the patient should be switched to a TNFi or to another biologic agent such as ustekinumab or secukinumab or to apremilast (Table 4).[18] In patients treated with infliximab, dose escalation can be considered prior to changing to another TNFi or changing to a biologic with a different mechanism of action.

Table 4. Pharmacologic treatment of enthesitis

Drug*	Comments
NSAIDs **Oral or intra-articular steroids**	• Oral corticosteroids may be used at a low dose and for the shortest time possible to avoid toxicity and potential psoriasis flare
TNFi Infliximab Golimumab Certolizumab pegol Etanercept Adalimumab	• DMARDs should not be used
IL inhibitors Ustekinumab (IL-12, IL-23) Secukinumab (IL-17)	
PDE4i Apremilast	

*If available, generic medications are preferred.

DMARDs, disease-modifying antirheumatic drugs; IL, interleukin; NSAIDs, nonsteroidal anti-inflammatory drugs; PDE4i, phosphodiesterase 4 inhibitor; TNFi, tumor necrosing factor inhibitor

Dactylitis

Dactylitis or so-called 'sausage digit' describes inflammation of an entire digit with diffuse swelling, pain, and tenderness of the digit and is frequently associated with progressive disease. Unfortunately, there is only weak evidence supporting the use of any specific drug. Initial therapy should begin with a trial of NSAIDs for a duration that is clinically appropriate to avoid the well-known GI and CV complications of these medications. Local injections of steroids (joint, tendon sheath) may also be tried as an initial treatment. Disease-modifying antirheumatic drugs can be used if the patient fails to respond to NSAIDs and/or local steroid injections, although data supporting the use of DMARDs for this indication are poor. Patients with severe dactylitis, involvement of multiple digits, or functional deficits due to dactylitis, or patients who failed to respond to DMARDs should receive a trial of TNFi such infliximab, certolizumab pegol, golimumab, or adalimumab. In patients treated with infliximab, dose escalation can be considered prior to changing to another TNFi or changing to a biologic with a different mechanism of action. If the patient fails to respond to TNFi therapy, IL inhibitors such as ustekinumab or secukinumab may be prescribed. Studies of apremilast are weak; therefore, TNFi agents are preferred (Table 5).[19]

Table 5. Pharmacologic treatment of dactylitis

Drug*	Comments
NSAIDs	
DMARDs Methotrexate Leflunomide Sulfasalazine	• There are weak data to support the use of DMARDs for dactylitis • TNFi agents are preferred
TNFi Infliximab Certolizumab pegol Golimumab Adalimumab	• Data to support the use of etanercept are weak • No recommendation for the use or avoidance of etanercept can be made in this domain
IL inhibitors Ustekinumab (IL-12, IL-23) Secukinumab (IL-17)	• Can be used as first- or second-line treatment for refractory dactylitis.
PDE4i Apremilast	• There are weak data to support the use of apremilast for dactylitis • TNFi agents are preferred

*If available, generic medications are preferred.

DMARDs, disease-modifying antirheumatic drugs; IL, interleukin; NSAIDs, nonsteroidal anti-inflammatory drugs; PDE4i, phosphodiesterase 4 inhibitor; TNFi, tumor necrosing factor inhibitor

Monitoring

When caring for a patient with PsA, clinicians must have a treatment target, which is often remission. In clinical practice, fewer patients achieve and maintain a remission than MDA.

Once patients have started therapy, they should be seen at 4- to 6-week intervals until the disease is stable. Once patients are stable on medication, they should be seen at least every 12 weeks. Minimal disease activity should be measured at every visit. Once patients have achieved MDA, they can be seen every 6 to 12 months, with disease measures obtained at each visit. Any patient experiencing a change in symptoms or functional ability should be seen as soon as possible. Monitoring of blood tests should be performed at regular intervals as clinically appropriate.

Glossary

Disease-modifying antirheumatic drugs (DMARDs)	There are conventional and biologic DMARDs. For simplicity in this document, the term 'DMARDs' refers to the conventional DMARDs—methotrexate, leflunomide, and sulfasalazine. Biologic DMARDs are termed 'biologics'.
Inflammatory back pain (IBP)	Chronic back pain for at least 3 months and 4 of the following 5 parameters:[20] 1. Age of onset <40 years 2. Insidious onset 3. Improvement with exercise 4. Lack of improvement with rest 5. Nocturnal pain that improves upon arising
Multidimensional Health Assessment Questionnaire (MDHAQ)	The MDHAQ is a practical patient self-report tool assessing that patients can complete in the waiting room. A form is available at (https://integrationacademy.ahrq.gov/sites/default/files/MDHAQ_0.pdf).
Psoriasis Area and Severity Index (PASI)	The PASI measures the severity of psoriasis of the skin, based on the body surface area involved. The affected body surface area is the percent of skin on the head, trunk, arms, and legs with erythema, induration, and scaling of the skin, with 1% body surface area equivalent to the surface area of the patient's palm (including the fingers). A free PASI online calculator can be found at http://pasi.corti.li/.

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Document Updates

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