Inside this Issue

• What makes psoriatic arthritis (PsA) so difficult to accurately diagnose on initial presentation?
• How is the efficacy of treatment measured in patients with PsA?
• What pharmacologic and nonpharmacologic treatment options are currently available for patients with PsA?
• Why is folic acid often recommended for patients with moderate to severe PsA?

Learning Objectives

1. Identify common clinical, physical, and extraarticular features of PsA that differentiate the condition from other types of inflammatory arthritis

2. Appraise the relevance of various clinically validated PsA disease assessment and monitoring tools, and implement one or more into your practice as warranted within the next three months

3. Assess recent evidence on current and emerging agents used to treat mild, moderate, and severe PsA

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Psoriatic Arthritis: Pathophysiology, Treatment, and Patient Management

Psoriatic arthritis (PsA) is a form of inflammatory arthritis that occurs exclusively in patients with psoriasis. Historically, PsA was thought to have a mild prognosis, but clinicians now understand that the potential for structural damage and functional disability in PsA rivals that found in patients with rheumatoid arthritis (RA).1

PsA is often underdiagnosed in patients with psoriasis, leading to a delay in treatment.2,3 Symptoms of PsA can be severe and progressive, ultimately leading to significant deformity for many patients.4 In a study of patients with PsA, 39% reported that PsA was a “large problem” in their everyday life.5 Effective management of PsA requires treatment of skin and joint disease as well as psychological comorbidities, highlighting the need for multidisciplinary patient care.6

Prevalence of PsA
Psoriasis is a common skin disorder, affecting up to 7.5 million Americans.5 The average risk of PsA in patients with psoriasis is 30%, although risk estimates range from 6%-40% depending on the patient population and extent of skin involvement.4 Currently, PsA affects an estimated 320,000 individuals.3 Approximately half of these patients will develop a progressive form of the disease that can lead to structural joint damage. PsA typically develops 7-10 years after the onset of psoriasis, at an average age of 36 years.6 Patients with psoriasis should be screened for PsA and encouraged to report any changes in symptoms, particularly the appearance of nail disease and any pain or tenderness in the joints.

Clinical Features
By definition, all patients with PsA have psoriasis. However, not all patients with arthritis and psoriasis have PsA. Different types of inflammatory arthritis can coexist with psoriasis and mimic the presentation of PsA. Due to variable patterns of joint involvement in PsA, joint symptoms alone are not a reliable tool for distinguishing PsA from other types of inflammatory arthritis. PsA must be distinguished from other types of inflammatory arthritis on the basis of clinical features, radiologic findings, and laboratory tests (see Table 1 on following page).1
### RHEUMATOLOGY NURSE NEWSLETTER

#### VOLUME 4, ISSUE 2

### Table 1: Features of Types of Arthritis that Occur in Patients With Psoriasis or Mimic PsA

<table>
<thead>
<tr>
<th></th>
<th>PsA</th>
<th>RA</th>
<th>OA</th>
<th>ReA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CLINICAL FEATURES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>M=F</td>
<td>F&gt;M</td>
<td>M=F</td>
<td>M&gt;&gt;F</td>
</tr>
<tr>
<td>Average age at onset, years</td>
<td>35 to 45</td>
<td>30 to 50</td>
<td>&gt;50</td>
<td>20 to 40</td>
</tr>
<tr>
<td>Onset</td>
<td>Gradual</td>
<td>Acute/gradual</td>
<td>Gradual</td>
<td>Acute</td>
</tr>
<tr>
<td>Quality of joint symptoms</td>
<td>Discomfort/pain less than in RA</td>
<td>Pain</td>
<td>Discomfort</td>
<td>Pain</td>
</tr>
<tr>
<td>Morning stiffness, min</td>
<td>&gt;60, better with activity</td>
<td>&gt;60, better with activity</td>
<td>&lt;20, worse with activity</td>
<td>&gt;60, better with activity</td>
</tr>
<tr>
<td>Extraarticular features</td>
<td>Present</td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
</tr>
</tbody>
</table>

### PHYSICAL EXAMINATION

| Pattern of joint involvement | Oligoarthritis, polyarthritis | Polyarthritis | Monoarthritis, oligoarthritis | Oligoarthritis, polyarthritis |
| Peripheral joint involvement | Yes, including DIP            | Yes, including MCP | Yes, including DIP            | Yes, including DIP |
| Axial joint involvement      | Present                      | Absent        | Present                     | Present                     |
| Swelling and overlying erythema of joints | Present | Present | Absent | Present |
| Enthesitis                  | Present                      | Absent        | Absent                      | Present                     |
| Dactylitis                  | Present in 48%               | Absent        | Absent                      | Occasional                  |
| Synovitis                   | Present                      | Present       | Absent                      | Present                     |
| Typical deformity           | Opera glass (telescoping) digit | Boutonniere, swan neck, zig zag | Heberden and Bouchard nodes | Spinal ankylosis |

### LABORATORY TESTS

| Erythrocyte sedimentation rate | +/− | Elevated | Normal | +/− |
| C-reactive protein            | +/− | Elevated | Normal | +/− |
| Rheumatoid factor             | Usually absent               | Usually present | Usually absent | Usually absent |
| CCP antibodies                | Usually absent               | Present       | Absent | Usually absent |
| Presence of HLA* B27          | 15%-70%                      | 4%-8%         | 4%-8% | 50%-85%       |
| Synovial aspirate             | Inflammatory                | Inflammatory | Noninflammatory | Inflammatory |

### IMAGING TESTS

| Periostitis                  | Present | Absent | Absent | Absent |
| Osteopenia                   | Absent  | Present | Absent | Absent |
| Joint space narrowing        | Late manifestation | Present | Present | Late manifestation |
| Erosion                      | Marginal and central         | Marginal     | Absent | Marginal     |
| Sacroiliitis                 | Present | Absent | Absent | Present |

**Physical findings**

PsA is characterized by joint pain, swelling, and prolonged morning stiffness. In general, PsA affects fewer joints than RA. Although both PsA and RA can occur as polyarthritis (>5 joints involved), only PsA is also routinely seen as oligoarthritis (<5 joints involved). PsA is also seen more commonly with an asymmetrical distribution of affected joints compared with the symmetrical pattern of joint involvement seen in RA.1

Inflammation of the distal interphalangeal (DIP) joints is characteristic of PsA, while changes to the metacarpophalangeal (MCP) joints indicate RA. The pattern of DIP-predominant joint involvement is unique to PsA, although this pattern occurs in only 5%-10% of patients, and mostly in men. In advanced PsA, severe bone resorption results in the appearance of collapsed or “telescoped” digits, forming the characteristic deformity called “opera glass hand.”

Other common distinguishing features of PsA include skin involvement, nail disease, and tissue inflammation. Psoriatic skin lesions can occur anywhere on the body, but most often affect the scalp, nails, trunk, elbows, and knees. Although the prevalence of PsA tends to increase in patients with more extensive skin disease, cutaneous manifestations of psoriasis are not a prerequisite for PsA. Patients with little to no evidence of skin involvement can have severe PsA.4

Nail disease occurs in up to 83% of patients with PsA and may be the first sign of PsA in patients with psoriasis. Specific nail changes associated with psoriasis and PsA can include discoloration, pitting, fragmentation, and onycholysis, which occurs when the distal portion of the nail lifts from the nail bed. Nail psoriasis can be disfiguring, causing pain, functional impairment, and emotional distress.7 Although nail psoriasis is common in patients with PsA, there is little correlation between the severity of nail symptoms and the extent of joint involvement.5

General tissue inflammation and swelling often affects the fingers and toes, resulting in dactylitis (“sausage digit”) in patients with PsA. Enthesitis, or inflammation and swelling at the sites of tendon or ligament insertion into bone, is another characteristic feature of PsA that is not commonly observed in RA.1

CCP = citrullinated-containing proteins; DIP = distal interphalangeal; F = female; HLA = human leukocyte antigen; M = male; MCP = metacarpophalangeal; OA = osteoarthritis; PsA = psoriatic arthritis; RA = rheumatoid arthritis; ReA = reactive arthritis.
Imaging Studies
Radiographic images reveal the divergent patterns of altered bone remodeling and joint destruction occurring in patients with PsA, RA, and other forms of arthritis. The main identifying feature of PsA on plain radiographs is the appearance of new bony formation next to erosive changes at sites of bone and tendon attachment. This pattern is unique to PsA. In addition, deformities in PsA are characterized by the presence of lysis and ankylosis on different joints of the same digit. Joint space narrowing, a characteristic finding in RA, is present only as a late manifestation in patients with PsA.1

In general, clinical evaluation alone may underestimate joint damage in PsA. In addition to radiographs, ultrasound examination is beneficial for detecting very early joint and tendon abnormalities in the fingers and toes of patients with suspected PsA.1

Laboratory Findings
Traditional laboratory tests are unreliable in diagnosing patients with PsA. Acute-phase reactants such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) are elevated in only 50% of patients with clinically active PsA.1 Rheumatoid factor and anti-cyclic citrullinated protein antibodies (anti-CCP) antibodies are usually absent in patients with PsA.1

Extraarticular Features
Beyond skin and joint manifestations, patients with PsA can experience a variety of conditions related to underlying inflammatory disease. Generalized symptoms associated with PsA include fatigue, weakness, anorexia, and weight loss. Eye symptoms are also common: up to 33% of patients with PsA experience conjunctivitis or uveitis resulting in pain, light sensitivity, and excessive tear production.1 Patients with PsA also have a disproportionately high burden of cardiovascular risk factors, including atherosclerosis, hypertension, dyslipidemia, diabetes, and obesity.7

Psychosocial Burden of Psoriasis
Living with psoriasis and PsA is very distressing for some patients and may lead to psychological comorbidities such as depression, suicidal thoughts, and substance abuse. Patients with psoriasis also experience social stigmatization, high stress levels, and employment problems related to their disease. The psychosocial burden of psoriasis varies among patients and is not always proportional to other measures of disease severity such as plaque severity of body surface area involvement.11 The negative effects of disease activity on quality of life measures related to pain, general health, social functioning, and mental health can be just as severe for patients with PsA as for patients with RA.7 Rheumatology nurses should discuss the true burden of psoriasis with their

Why are you recommending that I take folic acid? How is that going to help me?
Folic acid is a water-soluble B vitamin that has been added to cereals, flour, breads, pasta, and bakery items since 1998 as required by federal law. Foods that are naturally high in folic acid include leafy vegetables such as spinach and lettuce, legumes, and egg yolks. Folic acid is essential to numerous bodily functions, including the production and maintenance of new cells for synthesis of DNA and RNA. It is also important as an aid to rapid cell division and growth, especially in infancy and during pregnancy.

For patients with PsA, folic acid is often recommended for patients while they are taking MTX. MTX may inhibit dihydrofolate reductase, resulting in a decreased supply of folates. The coadministration of folic acid may help minimize adverse effects often related to this inhibition (eg, stomatitis, gastrointestinal intolerance, bone marrow toxicity, and abnormal liver function tests).1

While evidence is not entirely conclusive, folic acid is thought to be a key vitamin to help prevent cardiovascular events.2

I find that I am always tired. Can you suggest anything to help fight this fatigue?
The key to managing fatigue in PsA is early and aggressive treatment that limits pain and skin and joint damage. Just ask golfer Phil Mickelson, who was diagnosed with PsA in 2010 and is doing great on biologic therapy.

There are many variables that may cause fatigue in patients with PsA. Pain, depression, immobility, embarrassment, social isolation, nonrestorative sleep, anger, frustration, medication induced anemia, and decreased self-esteem/self-worth are just a few. In general terms, these variables can be divided into mechanical/physiological and emotional/psychological categories.

The presence of pain alone often exacerbates fatigue. Research has demonstrated that high levels of pain drive up levels of catecholamine, which then increases heart rate and blood pressure.1 When a patient’s body is forced to work harder and faster over the course of several hours, it can lead to exhaustion and fatigue. This makes it vital to appropriately and aggressively treat PsA. Once a patient’s disease is under better control, pain and fatigue levels should both subside.

References

References
patients, including the degree to which it interferes with their physical and mental health and social activities, to ensure that appropriate therapy is provided.

Diagnosis and Classification of PsA
No single laboratory or radiographic test can confirm the diagnosis of PsA or distinguish PsA from other types of joint disease. Instead, PsA is diagnosed on the basis of clinical judgment. Although several classification criteria for PsA have been proposed, the optimal method for describing PsA is a topic of ongoing debate.

For decades, PsA was diagnosed according to the 1973 Moll and Wright criteria. To meet the Moll and Wright criteria, a patient must have psoriasis and inflammatory arthritis, must be seronegative for rheumatoid factor, and must present with at least 1 of the following clinical phenotypes:

- Symmetric polyarticular (≥5 joints) arthritis
- Asymmetric oligoarticular (<5 joints) arthritis
- Distal interphalangeal joint predominant
- Spondylitis predominant
- Arthritis mutilans

Although the Moll and Wright criteria marked major progress in the understanding of PsA, additional clinical experience revealed the need for new classification criteria. The Classification Criteria for Psoriatic Arthritis (CASPAR) classification algorithm, developed in 2006, reflects the heterogeneity of patients with PsA.

For instance, patients can meet the CASPAR criteria for PsA without being seronegative for rheumatoid factor or without having a current diagnosis of psoriasis. Although rare, these scenarios can arise. Under CASPAR criteria, such patients are no longer excluded from consideration for PsA as long as other typical features of PsA are present.

Patients with confirmed inflammatory articular disease — defined as the presence of swollen or tender joints and prolonged morning stiffness — can meet the CASPAR criteria for PsA by accumulating ≥3 points from 5 categories (Table 2). The CASPAR criteria have a high specificity (98.7%) and sensitivity (91.4%) for diagnosing PsA.

<table>
<thead>
<tr>
<th>DISEASE FEATURES</th>
<th>POINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Evidence of psoriasis or personal or family history of psoriasis</td>
<td>2</td>
</tr>
<tr>
<td>Current psoriatic skin or scalp disease</td>
<td>1</td>
</tr>
<tr>
<td>Personal history of psoriasis (if no current diagnosis)</td>
<td>1</td>
</tr>
<tr>
<td>Family history of psoriasis (if no current diagnosis or personal history)</td>
<td>1</td>
</tr>
<tr>
<td>2. Current dactylitis or history of dactylitis recorded by a rheumatologist</td>
<td>1</td>
</tr>
<tr>
<td>Juxtaarticular new bone formation</td>
<td>1</td>
</tr>
<tr>
<td>Rheumatoid factor negativity</td>
<td>1</td>
</tr>
<tr>
<td>Typical psoriatic nail dystrophy, including onycholysis, pitting, and hyperkeratosis</td>
<td>1</td>
</tr>
</tbody>
</table>

What is GRAPPA?
The Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) is an international collaboration of researchers and clinicians dedicated to improving the diagnosis and management of psoriasis and PsA. In 2009, GRAPPA published recommendations for the management of PsA. GRAPPA is also developing online training videos to help clinicians use standardized disease assessment tools, including the ACR response criteria, DAS-28, and PASI. In 2011, GRAPPA endorsed the use of the CPDAI score to assess and monitor patients with PsA in the research and clinical practice settings.

GRAPPA has partnered with the Outcome Measures in Rheumatoid Arthritis Clinical Trials (OMERACT) group to conduct a long-term observational study of PsA. Patients with PsA will be enrolled into the trial with no exclusion criteria, thereby representing a real-world PsA patient population. Once enrolled, patients will undergo baseline evaluation and follow-up assessment every 6 months. Study investigators will collect clinical data and biological samples to analyze molecular and genetic markers of disease and treatment response. Throughout the study, patients can receive any form of therapy at the direction of their treating physician. Future reports from GRAPPA may provide new insights into the optimal treatment of patients with PsA.
Disease Assessment and Monitoring

Disease activity measures can be used to evaluate patients with PsA and monitor response to therapy. Assessment should include objective measurement of joint involvement, including joint pain, tenderness, and swelling; the degree of radiographic joint destruction; and the effect of joint involvement on physical functioning. Other tools also incorporate measures of skin involvement and other manifestations of psoriatic disease.

The Disease Activity Score with 28-joint count (DAS-28) was developed to assess disease activity in patients with RA, who have a different pattern of joint involvement. Given the potential involvement of the DIP joints in patients with PsA, a true total joint count should include 78 tender and 76 swollen joints. However, despite its origins in RA, the DAS-28 joint count is a reliable tool for evaluating peripheral joint involvement in PsA and differentiating mild vs. moderate/severe disease.14

The American College of Rheumatology (ACR20/50/70) scoring system was also developed for RA. When applied to patients with PsA, the ACR scoring system should include a 68/66 joint count for tenderness/swelling. The ACR20 criteria require a ≥20% reduction in the tender joint count, a ≥20% reduction in the swollen joint count, and a ≥20% reduction in 3 of 5 additional measures: patient assessment of pain, patient global assessment of disease activity, physician global assessment of disease activity, disability index of the Health Assessment Questionnaire (HAQ), and acute-phase reactants (eg, ESR and CRP). The ACR20 response has been used as the primary efficacy endpoint in most PsA clinical trials to date. ACR50 and ACR70 responses, which reflect corresponding reductions of at least 50% and 70%, respectively, also appear as secondary efficacy endpoints.15

The Psoriasis Area and Severity Index (PASI) score is used to grade the severity of psoriasis from 0 (no disease) to 72 (maximal disease). The PASI score is widely used to assess treatment response in psoriasis clinical trials, with a 75% reduction in baseline disease severity (PASI-75) a common efficacy endpoint.16 The PASI score is calculated by assessing skin involvement affecting 4 areas of the body (head, arms, trunk, and legs); percentage of area involved, scored from 0 (0%) to 6 (>90%); type of involvement (erythema, induration, and scaling); and severity of involvement, scored from 0 (none) to 4 (maximum). The complex formula tallies PASI scores in 0.1 increments and requires a calculator to measure. The simplified PASI (SPASI) score has been proposed for use in clinical practice.17 Although the PASI and SPASI scores do not account for joint involvement, both are relevant outcome measures for patients with psoriasis and PsA who prioritize skin improvement as a major treatment goal.

The PsA Response Criteria (PsARC) algorithm was developed for the specific purpose of assessing disease activity in patients with PsA. To achieve a PsARC response, patients must show improvement in at least 2 of 4 areas. One of the areas of improvement must be tender joint count or swollen joint count, and no areas should show evidence of worsening. The criteria include14:

- ≥20% improvement in physician global assessment of disease activity
- ≥20% improvement in patient global assessment of disease activity
- ≥30% improvement in tender joint count
- ≥30% improvement in swollen joint count

**If I get the psoriatic rash, does it mean that I will get erosive disease?**

Although the majority of patients develop skin lesions consistent with psoriasis long before they develop arthritis or enthesitis, it is important for nurses to raise awareness about the possibility of PsA in patients who are newly diagnosed with psoriasis. Current estimates state that approximately 20%-30% of patients with psoriasis will develop PsA, with the range of prevalence estimates between 6%–39%.1

Approximately two-thirds of patients with cutaneous manifestations present 5-10 years before joint complaints begin.2 The extent and severity of both skin and joint disease correlate closely with the severity of psoriasis.3

Unfortunately, there are no genetic or laboratory clinical biomarkers that can help identify which psoriasis patients will develop PsA, although members of Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) have worked on developing screening tools. It is imperative that the rheumatology office serves as the clinical coordinator with nutrition, orthopedics, psychosocial counseling, dermatologists, and physical and occupational therapy to drive appropriate therapy.

— Jacqueline Fritz, RN, MSN, CNS

**REFERENCES**

Disease Severity

Individual tools such as the DAS-28 and PASI scores can quantify specific aspects of disease activity in PsA, including joint and skin involvement. To assess overall disease severity, however, clinicians should also consider physical functioning, disability, pain, quality of life, and psychosocial factors. The American Academy of Dermatology (AAD) defines mild, moderate, and severe PsA according to the following parameters: 1) the effects of disease activity on quality of life; and 2) the patient’s need for and responsiveness to specific therapies (see Table 4).15

Table 3: Composite Psoriatic Disease Activity Index (CPDAI) Scoring18

<table>
<thead>
<tr>
<th>PsA Domain</th>
<th>Severity (score)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>None (0)</td>
</tr>
<tr>
<td>Peripheral arthritis</td>
<td>≤4 joints (swollen or tender); normal function (HAQ ≤0.5)*</td>
</tr>
<tr>
<td>Skin disease</td>
<td>PASI ≤10 and DLQI ≤10</td>
</tr>
<tr>
<td>Enthesitis</td>
<td>≤3 sites; normal function (HAQ ≤0.5)*</td>
</tr>
<tr>
<td>Dactylitis</td>
<td>≤3 digits; normal function (HAQ ≤0.5)*</td>
</tr>
<tr>
<td>Spinal disease</td>
<td>BASDAI ≤4; normal function (ASQoL ≤6)</td>
</tr>
</tbody>
</table>

*HAQ only counted if clinical involvement of domain (joint/enthesis/dactylitis) present. ASQoL = Ankylosing Spondylitis Quality of Life Index; BASDAI = Bath Ankylosing Spondylitis Disease Activity Index; DLQI = Dermatology Life Quality Index; HAQ = Health Assessment Questionnaire; PASI = Psoriasis Activity and Severity Index.

Table 4: Definitions of Disease Severity in PsA15

<table>
<thead>
<tr>
<th>Disease Severity</th>
<th>Impact on QOL</th>
<th>Response to Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>· Minimal</td>
<td>· Requires NSAIDs</td>
</tr>
<tr>
<td>Moderate</td>
<td>· Impacts daily tasks of living and physical/mental functions</td>
<td>· Lack of response to NSAIDs</td>
</tr>
<tr>
<td>Severe</td>
<td>· Large impact on physical/mental functions</td>
<td>· Cannot perform major daily tasks of living without pain or dysfunction</td>
</tr>
</tbody>
</table>

DMARDs = disease-modifying antirheumatic drugs; NSAIDs = nonsteroidal anti-inflammatory drugs; QOL = quality of life; TNF = tumor necrosis factor.

Current Treatments for PsA

Recommendations for first-line therapy in patients with PsA depend upon disease severity. Mild PsA can be managed successfully with non-steroidal anti-inflammatory drug (NSAID) therapy alone. For patients with moderate to severe PsA, the AAD recommends first-line treatment with methotrexate (MTX), anti-tumor necrosis factor (anti-TNF) therapy, or a combination of MTX and anti-TNF therapy.8,15

Approximately 50% of patients with PsA have a progressive form of disease that will result in structural damage.9 For these patients, early intervention with disease-modifying anti-rheumatic drugs (DMARDs) is critical for slowing disease progression and preserving physical functioning and quality of life.19

Mild PsA

First-line NSAID therapy is recommended for patients with mild PsA. Local intra-articular corticosteroid injections are also an option when only a few joints are involved. While these options provide effective symptom control, NSAIDs and corticosteroids do not prevent structural joint damage in patients with progressive disease. If patients do not achieve an adequate response to NSAIDs after 2 to 3 months, treatment with MTX and/or anti-TNF therapy should be considered.8,15

As patients add or switch to more aggressive therapies throughout the duration of PsA treatment, adjunctive NSAIDs and low-dose prednisone (<10 mg/day) can be used for further symptom control.9 In general, full-dose systemic corticosteroids should be avoided in patients with psoriasis, due to the risk of cutaneous flares when treatment is discontinued.15
Moderate to Severe PsA

**METHOTREXATE**

Methotrexate (MTX) is recommended alone or in combination with anti-TNF therapy as first-line therapy for patients with moderate-to-severe PsA. Treatment with single-agent MTX is often tried before adding or switching to biologic DMARDs because of its significantly lower cost. In the Methotrexate in Psoriatic Arthritis (MIPA) trial, single-agent MTX improved skin and nail symptoms in patients with PsA. However, treatment with MTX did not change swollen/tender joint counts, ACR20, DAS-28, or PsARC scores, suggesting that MTX does not exert disease-modifying properties in patients with PsA.

Limited data support the use of other synthetic DMARDs in the management of PsA. Sulfasalazine and leflunomide are associated with modest benefits compared with placebo. Other standard DMARDs, including antimalarials, cyclosporine, and gold, are used even less frequently given their limited activity in patients with PsA. The 2011 AAD guideline specifies MTX as the preferred nonbiologic DMARD for the treatment of PsA.

**Table 5: TNF Inhibitors Approved for the Treatment of PsA**

<table>
<thead>
<tr>
<th>Agent</th>
<th>Indications</th>
<th>PsA Dosing</th>
<th>Toxicities</th>
<th>Monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adalimumab</td>
<td>Moderate to severe PsA; moderate to severe psoriasis; RA; AS</td>
<td>40 mg every other wk subcutaneously</td>
<td>Injection site reactions; rare cases of serious infection and malignancy. Pregnancy category B.</td>
<td>Baseline PPD, LFT, CBC, and hepatitis profile. Yearly PPD and periodic CBC and LFT.</td>
</tr>
<tr>
<td>Etanercept</td>
<td>Moderate to severe PsA; moderate to severe psoriasis; RA; AS</td>
<td>25 mg twice wk or 50 mg once wk subcutaneously</td>
<td>Injection site reactions; rare cases of serious infection and malignancy. Pregnancy category B.</td>
<td>Baseline PPD, LFT, CBC, and hepatitis profile. Yearly PPD and periodic CBC and LFT.</td>
</tr>
<tr>
<td>Infliximab</td>
<td>Moderate to severe PsA; severe psoriasis; RA; AS Contraindicated at doses &gt;5 mg/kg in patients with NYHA class III or IV CHF</td>
<td>5 mg/kg given intravenously at wk 0, 2, and 6, and then every 6-8 wk; dose and infusion interval may be adjusted as needed</td>
<td>Infusion reactions; rare cases of serious infection and malignancy. Pregnancy category B.</td>
<td>Baseline PPD, LFT, CBC, and hepatitis profile. Yearly PPD and periodic CBC and LFT.</td>
</tr>
<tr>
<td>Golimumab</td>
<td>Moderate to severe PsA, RA, AS Not indicated for psoriasis</td>
<td>50 mg every 4 wk subcutaneously</td>
<td>Injection-site reactions; rare cases of serious infection and malignancy. Pregnancy category B.</td>
<td>Baseline PPD, LFT, and CBC. Yearly PPD and periodic CBC and LFT.</td>
</tr>
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</table>

**BIOLOGIC DMARDs**

Biologic DMARDs target key molecules of the underlying immune response that drive the development and progression of PsA. Currently, four anti-TNF agents — adalimumab, etanercept, infliximab, and golimumab — are available for the treatment of moderate to severe PsA (see Table 5). The TNF inhibitors can be taken alone or in combination with MTX. Compared with anti-TNF inhibitors alone, adding low-dose MTX to anti-TNF therapy can lead to further improvement of joint symptoms. In clinical trials of anti-TNF agents in PsA, 40%-50% of patients were taking concomitant MTX.

When used as prescribed, all of the TNF inhibitors show similar efficacy in controlling the signs and symptoms of PsA and reducing the risk of radiographic progression. However, these agents have variable effects on cutaneous psoriasis. Infliximab appears to improve cutaneous psoriasis in the highest proportion of patients, followed by adalimumab and etanercept. Golimumab is the only of the four biologics approved for the treatment of PsA that is not also approved for the treatment of psoriasis.

**What makes you think I have psoriasis instead of another skin-related disorder?**

Skin conditions can be very difficult to definitively diagnose, although there are certain features of psoriasis that can help to identify the condition. Psoriatic lesions are found most commonly on the scalp, ears, elbows, knees, and the palms of the hands. These patches of skin overgrowth may appear red and flaky, or even silvery and scaly. They may itch. Psoriasis may also affect the fingernails and toenails, causing them to be pitted (separated from the nail bed) and resembling a fungal infection. Although a diagnosis of psoriasis is often made by visual appearance, a skin biopsy may be needed to rule out other conditions similar in appearance.

— Kori A. Dewing, DNP, ARNP

**REFERENCES**

EMERGING TREATMENTS FOR PsA

Several biologic DMARDs are under evaluation for the treatment of PsA. These include both investigational agents as well as agents that are currently approved by the U.S. Food and Drug Administration (FDA) for related indications such as psoriasis or RA.

AGENTS APPROVED FOR PSORIASIS

Ustekinumab is a monoclonal antibody that targets interleukin (IL)-12 and IL-23 to reduce the activity and further production of proinflammatory cytokines. Ustekinumab is currently approved for the treatment of moderate to severe plaque psoriasis, but not for PsA. In a phase II study, 42% of patients with PsA achieved an ACR20 response after treatment with ustekinumab compared to 14% of patients in the placebo arm.

Ustekinumab is undergoing further study as a potential treatment for PsA in phase III trials of patients with and without prior exposure to anti-TNF therapy. In addition, the ongoing Psoriasis Longitudinal Assessment and Registry (PSOLAR) will evaluate outcomes in 12,000 patients with psoriasis who are treated with ustekinumab (n=4000), infliximab (n=4000), and other standard therapies, including topical treatments, phototherapy, conventional systemic therapies, and other biologic therapies (n=4000). Launched in 2007, the 10-year PSOLAR study will track long-term psoriasis outcomes, including PsA.

Alefacept is a fusion protein that interferes with the inflammatory response by blocking T-cell activation. Alefacept is approved for the treatment of moderate to severe plaque psoriasis in patients who are candidates for phototherapy or systemic therapy. In a phase II study, patients with PsA who had previously taken DMARDs, including TNF inhibitors, achieved good ACR20 and PASI50 responses after treatment with alefacept in combination with MTX.

AGENTS APPROVED FOR RA

Certolizumab pegol is a pegylated monoclonal antibody directed against TNF. It is approved for the treatment of RA and Crohn’s disease, and is currently in phase III trials for the treatment of PsA. Abatacept, a fusion protein that inhibits the costimulation of T cells, is approved for the treatment of RA in patients who failed prior anti-TNF therapy. In a phase II study of patients with PsA who had previously taken DMARDs, including TNF inhibitors, abatacept 10 mg/kg significantly improved ACR20 response, PASI scores, imaging outcomes, and other endpoints compared with placebo.
INVESTIGATIONAL AGENTS

Apremilast (CC-10004) is an oral anti-inflammatory agent that inhibits type 4 phosphodiesterase (PDE4). In a phase II trial, 12 weeks of treatment with apremilast significantly improved ACR20 and ACR30 responses compared with placebo in patients with PsA. The ongoing phase III clinical trial program — PALACE-1, PALACE-2, PALACE-3, and PALACE-4 — will evaluate apremilast in patients with PsA.

Secukinumab (AIN457) is an investigational monoclonal antibody against IL-17a, a potent proinflammatory cytokine. Treatment with secukinumab shows promising activity in psoriasis, RA, and other inflammatory diseases. AIN457 is currently being evaluated in a phase II study of patients with PsA.

Summary

PsA is a clinically heterogeneous disease that can lead to severe joint deformity, functional impairment, and poor quality of life. Although PsA management has suffered from diagnostic uncertainty in the past, new classification criteria may improve the early detection of PsA. Treatment with disease-modifying therapy can slow structural damage and prevent the characteristic joint deforms in patients with progressive disease. Multidisciplinary care can also address the common psychosocial and quality of life issues associated with PsA.

Why was I misdiagnosed with RA initially? Are you sure you got it right this time?

While the association between psoriasis and arthritis was initially made in the mid-19th century, PsA was not distinguished from RA until the 1960s. PsA and RA share common features, especially early in the disease process, which can make differentiation of the two conditions difficult. Fortunately, treatment for PsA and RA is often similar, so an initial misdiagnosis is not necessarily cause for alarm among patients.

PsA should always be suspected when any inflammatory arthritis is apparent in the presence of psoriasis, although a patient does not need to have active subcutaneous psoriasis to be diagnosed with PsA. If a patient with psoriasis and inflammatory arthritis has rheumatoid nodules, he/she is likely to have coexistent RA with psoriasis, although this occurs rarely. Patients who are rheumatoid factor (RF) negative but have distal interphalangeal (DIP) joint disease and nail lesions are much more likely to have PsA than polyarticular arthritis, even if they present with a symmetric polyarthritis. The presence of spinal disease also tips the balance towards PsA.

Key clinical features that differentiate PsA from other polyarticular arthritic diseases include the following:

- Asymmetric joint involvement
- Absence of RF
- Significant nail pits
- Involvement of the DIPs in the absence of osteoarthritis
- “Sausage digits”
- Family history of psoriasis or PsA
- Axial radiographic evidence of sacroiliitis
- Paravertebral ossification
- Syndesmophytes
- Peripheral radiographic evidence of erosive arthritis with relative lack of osteopenia

— Joyce M. Kortan, RN

REFERENCES


Does having PsA mean that I have a higher risk of developing cancer?

As with many autoimmune diseases, the risk of developing lymphoma is increased in patients with PsA. An increased risk of developing certain types of skin cancer has also been identified in patients with psoriasis and PsA. However, it remains unclear whether this risk is due to the underlying condition or to treatments such as phototherapy that are used to reduce symptoms of the condition. There may also be an increased risk of some forms of solid tumors, but this increased prevalence may be due to the increased use of tobacco and alcohol in patients with PsA.

Because of these increased risks, all suspicious skin lesions should be evaluated promptly by a medical professional. Cancer screening, including a colonoscopy and mammogram, should be performed regularly. Prevention strategies, including smoking cessation and limiting alcohol use, may also help to reduce the risk of developing cancer.

— Kori A. Dewing, DNP, ARNP

REFERENCE


Activity Instructions & Criteria for Success

Continuing Nursing Education contact hours are offered to all activity participants. To successfully complete this activity and obtain a Certificate of Contact Hours awarded, the learner is required to read the entire newsletter, complete the post-test, and complete the activity evaluation form. Learners are required to correctly answer 80% of the learning assessment questions. Statements of Credit will be forwarded via email within 4 to 6 weeks. All forms must be received by October 1, 2012, to be eligible for credit.

1. Please fax both sides of this evaluation to the Institute at (215) 592-9085, OR
2. Please complete the evaluation online by going to www.iche.edu/content/rheumatology-nurse-newsletter.

NAME ______________________________________________________________________________    DEGREE/CERTIFICATION _____________________________

Activity Post-Test Questions

(Please circle the letter that matches the correct response to each question below)

1. Approximately what percentage of patients with PsA develop progressive disease that leads to structural joint damage?
   a. 25%
   b. 40%
   c. 50%
   d. 75%

2. JG is a 35-year-old female patient who you are seeing for the first time. She has significant pain, morning stiffness of approximately 70 minutes each morning that improves with activity, synovitis, elevated ESR, CRP and RF osteopenia, and marginal joint erosion. Based upon this limited information, which of the following inflammatory diseases does she most likely have?
   a. Psoriatic arthritis
   b. Rheumatoid arthritis
   c. Osteoarthritis
   d. Reactive arthritis

3. Which of the following statements is not true?
   a. Joint symptoms alone are not a reliable tool for distinguishing PsA from other types of inflammatory arthritis
   b. PsA typically develops within 7-10 years after the onset of psoriasis
   c. All patients with PsA have psoriasis
   d. All patients with psoriasis have PsA

4. How does the evidence supporting the use of phototherapy in patients with moderate to severe PsA compare to the evidence supporting the use of MTX and biologic DMARDs in these patients?
   a. There is a higher level of evidence supporting the use of phototherapy in these patients
   b. There is lower level of evidence supporting the use of phototherapy in these patients
   c. There is the same level of evidence supporting the use of phototherapy in these patients
   d. There is no evidence supporting the use of phototherapy in these patients

5. Evidence clearly shows that the increased risk of skin cancer among patients with PsA is due to which of the following factors?
   a. Underlying symptoms of the condition
   b. Phototherapy
   c. Use of biologic agents
   d. None of the above

6. TL, a 44-year-old male patient, presents with a family history of psoriasis but no current psoriatic skin or scalp disease, dactylitis, juxtaarticular new bone formation, rheumatoid factor positivity, and no psoriatic nail dystrophy. According to CASPAR criteria, should he be diagnosed with PsA?
   a. Yes
   b. No
   c. Not enough information is presented to make a clear determination

7. For which PsA patient group is the use of the PASI score most relevant?
   a. Those who prioritize skin improvement as a major treatment goal
   b. Those who prioritize reduction in pain as a major treatment goal
   c. Those with skin involvement that primarily affects the shoulders and neck
   d. Those whose activities of daily living are most severely affected by the disease

8. Which of the following is indicated as treatment for mild PsA?
   a. Full-dose systemic corticosteroids
   b. Phototherapy
   c. Methotrexate
   d. NSAIDs

9. Biologic agents indicated for use in both psoriasis and PsA include which of the following?
   a. Rituximab
   b. Etanercept
   c. Certolizumab pegol
   d. Golimumab

10. Folic acid is often recommended for patients with PsA prescribed which of the following agents?
    a. Infliximab
    b. Adalimumab
    c. Usteukinumab
    d. Methotrexate
<table>
<thead>
<tr>
<th>After participating in this activity, I am better able to:</th>
<th>Strongly disagree</th>
<th>Disagree</th>
<th>Neutral</th>
<th>Agree</th>
<th>Strongly agree</th>
<th>Relative to each objective, do you plan to change your practice? (Y-Yes, N-No, A-Already Doing):</th>
</tr>
</thead>
<tbody>
<tr>
<td>Identify common clinical, physical, and extraarticular features of PsA that differentiate the condition from other types of inflammatory arthritis</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>Y</td>
</tr>
<tr>
<td>Appraise the relevance of various clinically validated PsA disease assessment and monitoring tools, and implement one or more into your practice as warranted within the next three months</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>Y</td>
</tr>
<tr>
<td>Assess recent evidence on current and emerging agents used to treat mild, moderate, and severe PsA</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>Y</td>
</tr>
</tbody>
</table>

If you indicated “yes” in the table above, please give a specific example of what you will change:

____________________________________________________________________________________________________________________________________

____________________________________________________________________________________________________________________________________

Approximately, how many patients with PsA do you treat and/or manage on a monthly basis?
- ☐ Less than 3 patients
- ☐ 3-5 patients
- ☐ 6-10 patients
- ☐ More than 10 patients

What is your primary practice setting?
- ☐ Hospital
- ☐ University/academic medical center
- ☐ Clinic
- ☐ Private practice
- ☐ Group or staff model health maintenance organization
- ☐ Other: ______________________________________________________

How many years have you been in practice as a rheumatology nurse?
- ☐ 0-5 years
- ☐ 6-10 years
- ☐ 11-15 years
- ☐ 16 or more years

Was disclosure information provided to you at the beginning of this activity?
- ☐ Yes ☐ No

Did this activity promote a particular company or product?
- ☐ Yes ☐ No

If yes, please explain in detail: ____________________________________________

_______________________________________________________________________

_______________________________________________________________________

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First Name ________________________________________________ Middle Initial _____ Last Name __________________________________________________________

Degree (please check all that apply) ☐ RN ☐ NP ☐ CNS ☐ CRNA ☐ CNM ☐ LPN ☐ Other ______________________________________________________

Your certificate will be emailed to the address you list below.

Address _____________________________________________________________________________________________________________________________________

City __________________________________________ State ___________ ZIP _________________________

Telephone ___________________________________ E-mail Address _____________________________________________________________________________________

I certify that I have participated in the above-named continuing education activity.

Signature _________________________________________________________________________________________ Date ___________________________
Establishing Effective Communication with Other Specialties

BY VICKY RUFFING, RN

While reviewing Mrs. G’s routine lab work, you see a significant drop in her hematocrit and hemoglobin levels. You have her stop her NSAIDs and send her for a stool guaiac test. After receiving a positive result for fecal blood, you immediately refer her to a gastroenterologist for evaluation.

Because of the complex nature of rheumatic diseases, scenarios such as these take place daily in rheumatology practices. Referrals to and from other specialists are common, and adding multiple layers of care from specialties such as dermatology, hematology, orthopedics, endocrinology, or infectious disease is almost inevitable.

In practices with an established network, such as an HMO, referrals may flow smoothly back and forth, although in other circumstances, the coordination of care can often go awry. This can happen for many reasons.

Many times, the primary care provider (PCP) chooses to serve as the quarterback of a patient’s care while sometimes he/she relinquishes this role to the primary specialist managing the patient, seeing the patient for only minor illnesses or comorbidities. Without the development of a consistent working relationship, it’s hard to know which camp individual PCPs will fall into.

Other hiccups occur when results or feedback from specialists don’t arrive in a timely manner (or at all). There could also be a change in treatment plan or medications that are out of sync with the plan the rheumatologist had previously established with the patient.

So what is the role of the rheumatology nurse in this conundrum? How can we help ease the communication pathway between offices? There are several ways I believe this can be accomplished, depending on the situation.

**WHEN REFERRING TO ANOTHER SPECIALIST**

- Most practices have a standard referral form that accompanies a request for a specialist visit. Include the urgency of the problem when you fill out the form, along with a cover sheet that includes a polite note and a simple “thank you” to establish a collegial relationship. Make sure to highlight where any results, notes, or findings should be sent. In more urgent situations, a nurse-to-nurse call can make a huge difference in getting your patient a timely appointment. Sharing with your nursing colleague information about your experience with the patient and any relevant background information can make a name on a paper become a real person.

- If, in your practice, referrals are the office coordinator’s responsibility, ensure that there is a polite relationship established between offices. Make sure that a copy of the specialist referral goes not only to the specialist but also to the PCP and the patient. A patient can be your ally in making sure that all relevant parties are kept in the loop. The better you arm them with information, the easier it will be for them to advocate for themselves.

- Once a collegial relationship is established with specific offices, there is nothing wrong with using them over and over for issues in their specialty area. For example, in our case scenario above, we would choose from among two or three gastroenterologists to whom we typically refer these sorts of patients. Having templates preprinted with their information available to send a quick thank you for seeing a particular patient can be a nice added touch.

**WHEN RECEIVING A REFERRAL FROM ANOTHER SPECIALIST**

- Expect the same information from a referring specialist that you would provide to them. If you don’t get all of the necessary information on the initial referral, consider sending a letter, fax, or email that explains the information you require. Depending on how often you work with the referring office and how solid your relationship is with them, phone calls may work too.

**WHEN RECEIVING A PCP REFERRAL**

- In our office, we have specific information we want from the PCP that allows us to prioritize the urgency of new patients and helps us assign patients to the right rheumatologist for their diagnosis. Lab values, x-ray and MRI results, patient history, physical findings, and prior therapies used are all included in the checklist we request from PCPs.

**POTENTIAL PROBLEM AREAS**

- There are times when a specialist may change or discontinue a patient’s medications without a discussion with the rheumatologist. This is where your patients can help — emphasize to them the importance of informing you if another doctor wants them to stop or add to their rheumatology medications. For example, orthopedic surgeons often request that a patient stop methotrexate 6-8 weeks before a joint replacement, even though there is no evidence that methotrexate, at the doses we use, interferes with healing.

- Track outstanding referrals in a table to assure the ball has not been dropped.

- Communication back to the PCP is important. Fax back any abnormal lab values with a note explaining their importance. Do not assume the PCP will read your note suggesting that he/she perform a thyroid workup. That should be requested in a separate note.

As we slowly move toward the patient-centered medical home model in which a clear referral system serves as the crux of care, communication between sub-specialties and the medical home should improve. Until that evolution occurs, nurses should still serve at the forefront to ensure that communication among specialists runs smoothly and has a positive impact on patient outcomes.

**REFERENCES**


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