RHEUMATOLOGY NURSE PRACTICE

Accredited education for registered nurses and advanced practice providers

PSORIATIC ARTHRITIS

PATHOPHYSIOLOGY 101

A NURSING PRIMER

Inside this Issue

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TARGET AUDIENCE

This activity has been designed to meet the educational needs of nurses, nurse practitioners, and physician assistants. Other healthcare providers may also participate.

ACTIVITY DESCRIPTION

In this issue of *Rheumatology Nurse Practice,* we'll examine the pathophysiology of psoriatic arthritis, focusing on epidemiology, pathogenesis, and common presenting features of the disease. We'll also review data reflecting the current diagnostic ability among rheumatology and dermatology providers.

LEARNING OBJECTIVES

After participating in the activity, learners should be better able to:

- List at least 3 reasons for delays in diagnosis of psoriatic arthritis (PsA)
- Describe key features that differentiate PsA from other forms of inflammatory arthritis
- Identify the five musculoskeletal domains of PsA
- Develop strategies to better identify and treat depression in patients with PsA

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PSORIATIC ARTHRITIS

PATHOPHYSIOLOGY 101

A NURSING PRIMER

Psoriatic arthritis (PsA) is a chronic, inflammatory spondyloarthropathy with a varied disease course whose type of tissue involvement can change over time.[1] In the past, PsA was classified as a variant of rheumatoid arthritis (RA); however, radiological, clinical, and laboratory data have established PsA as a distinct, progressive articular entity with the potential to impair function, reduce quality of life, and increase mortality.[2]

PsA is pathogenetically associated with psoriasis and contributes to deformities and joint damage in a majority of patients, with bone erosion often developing in the first 2 years of disease.[3] Early recognition and diagnosis is key to effective treatment. However, many clinicians in primary care and dermatology settings are unaware of the risk for musculoskeletal disease in patients with psoriasis and the need for regular screening for PsA in this patient population.[4]

Drug Names Included Within This Issue

GENERIC	BRAND
Etanercept	Enbrel
Adalimumab	Humira
Infliximab	Remicade
Golimumab	Simponi
Certolizumab pegol	Cimzia
Ustekinumab	Stelara
Secukinumab	Cosentyx
Brodalimumab	Siliq
Ixekizumab	Taltz
Apremilast	Otezla
Tofacitinib	Xeljanz
Abatacept	Orencia

Epidemiology of Psoriatic Arthritis

Prevalence and Incidence

The prevalence and incidence of PsA is challenging to quantify due to the heterogeneity of the disease and variations in diagnostic criteria. Depending on the criteria utilized and the populations studied, estimated prevalence in the general adult population of the United States ranges from 0.06–1.0%.[5–9] The prevalence of PsA among patients with cutaneous psoriasis, which affects approximately 3.2% of the population,[8] is higher than in the general population, ranging from 5–42%.[10] PsA prevalence is similar among both men and women.[5]

In the United States, the reported average annual incidence rate of PsA in patients with psoriasis ranges from approximately 2.7%–3.2%,[10] although time trend data suggest that the overall incidence may be rising.[11] The reported cumulative incidence of PsA is 1.7%, 3.1% and 5.1% at 5, 10, and 20 years following a diagnosis of psoriasis.[12] There is substantial variation in the global epidemiology of PsA, with higher prevalence and incidence rates in Europe and North America than in Asia.[3,13]

Clinical, Psychosocial, and Functional Burden

PsA confers considerable psychosocial and functional burden on patients, as well as impairing quality of life and impacting work disability. The visible skin and joint components of PsA are associated with poorer health-related quality of life for patients with PsA compared with both the general population and patients with RA, as well as with patients who have psoriasis alone.[14] Patients with PsA also commonly suffer from sleep disorders, fatigue, and low-level stress, as well as depression, mood changes, and anxiety about bodily symptoms and appearance.[14] This combination of pain, fatigue, and other quality of life changes can contribute to high rates of absenteeism from work, productivity loss, and unemployment, especially in the first 2–5 years of disease.[15] Globally, indirect costs such as loss of productivity account for up to 72% of total costs associated with the management of PsA, while the direct annual healthcare costs of PsA in the United States have been estimated at approximately $1.9 billion.[16]

The prevalence of comorbidities in patients with PsA is also high and further diminishes quality of life.[17] PsA is associated with systemic diseases such as hypertension, type 2 diabetes, obesity, metabolic syndrome, fatty liver disease, autoimmune disease, malignancies, and cardiovascular disease (Table 1).[18] Many of these comorbidities are exacerbated by persistent inflammation, further highlighting the need for early diagnosis.

Diagnostic Practice and Trends

Researchers have speculated that observed increases in the annual incidence of PsA may reflect improved awareness among clinicians, more systematic assessment of patients with psoriasis, and subsequent diagnosis of PsA.[11] However, several studies report that PsA remains under-recognized in dermatology and primary care practices, screening of psoriasis patients is limited, and patients with PsA tend to remain undiagnosed until they are seen by a rheumatologist.

Where Do Patients with PsA Present?

The prevalence of undiagnosed PsA in dermatology settings among patients with psoriasis is high. The Prevalence of Psoriatic Arthritis in Adults with Psoriasis: An Estimate from Dermatology Practice (PREPARE) study identified PsA in 30% of patients with psoriasis (n=949) following assessment by rheumatologists.[19] Patients had been evaluated by dermatologists, but had not previously received a diagnosis of PsA. Additionally, while patients with psoriasis typically present first to a dermatologist (although suspected arthritis is rarely noted as a reason for seeking care), many patients with PsA are diagnosed in primary care as opposed to dermatology settings. The Multinational Assessment of Psoriasis and Psoriatic (MAPP) Arthritis Survey

Table 1 *Comorbidities Associated with PsA*[9,41]

Atherosclerotic Disease	Cerebrovascular Disease	Malignancy
Type 2 Diabetes	Hyperlipidemia	Autoimmune Disease
Hypertension	Cardiac Arrhythmia	Parkinsonism
Metabolic Syndrome	Nonalcoholic Fatty Liver Disease	Chronic Obstructive Pulmonary Disease

is the largest population-based survey in North America and Europe involving more than 5,000 patients with psoriasis and PsA being treated by rheumatologists and/or dermatologists.[4,20] Data from this research show that more than half (55%) of patients with psoriasis or PsA are referred with a previous diagnosis from primary care providers.[22]

Can PsA be Diagnosed without Psoriasis?

PsA typically develops in patients with established psoriasis, although not all patients with psoriasis develop PsA.[3] This dichotomy has stimulated considerable debate on whether psoriasis and PsA are distinct entities or 'flip sides of the same coin'.[21] For a majority of patients with PsA, arthritic involvement and inflammatory arthritis typically develop approximately 7–10 years after the onset of psoriasis.[6,22,23] However, clinical and epidemiological studies show that for a minority of patients (~15%), psoriasis may develop after the onset of PsA, or arthropathies may coincide with the onset of psoriasis symptoms.[24] The risk of developing PsA is higher in patients with more severe psoriasis (measured by body surface area [BSA]).[9,25,26] The prevalence of PsA is also higher among patients seen in dermatology clinics, who tend to have more severe psoriasis.[9,19] These insights underscore the important role that clinicians in dermatology and primary care clinics play in screening patients with psoriasis for PsA.

Is Diagnosis of PsA Improving?

The timely diagnosis of PsA remains suboptimal, and PsA is often unrecognized until patients consult with a rheumatologist. Data from MAPP Arthritis surveys show average delays of 5–12 years between sign/symptom onset and diagnosis of PsA, with a diagnosis of PsA in only 26.9% of patients with psoriasis.[4,20] Reasons for these delays include not seeing a healthcare provider (HCP) or seeing an HCP who fails to screen for, or recognize, PsA. Although almost half (44%) of MAPP patients with psoriasis alone reported joint pain—including symptoms resembling dactylitis and/or enthesitis—48% had not seen a healthcare provider in the previous 12 months for their condition. Patients with psoriasis who had seen a healthcare provider in the prior 12 months most often saw a dermatologist (55%) or a primary care physician (34%). Other studies point to limited screening and under-recognition of PsA among dermatologists compared with rheumatologists. For instance, in one recent survey of dermatologists (n=391) and rheumatologists (n=390) in North America and Europe, dermatologists said they discussed the possibility of developing joint disease in less than half of their psoriasis patients. In addition, less than half (39.1%) of PsA diagnoses were made by dermatologists.[27] The same study noted that dermatologists classified 25.7% of their patients as having severe PsA versus 48% of rheumatologists.

New Models of PsA Care: Combined Dermatology/Rheumatology Clinics

The current evidence indicates that differentiating PsA from other arthritic diseases in dermatology and primary care remains a significant barrier to diagnosis, while delayed referral is one of the biggest challenges for rheumatologists in treating patients with PsA. Accordingly, combined rheumatology/dermatology clinics are emerging as an effective model for improving the quality of PsA patient care. There are currently 20 combined clinics in the United States affiliated with academic medical centers that prioritize collaborative care.[28] Although the processes of combined clinics can vary, they can include shared facilities, virtual consults, simultaneous patient assessments by a rheumatologist and dermatologist, or assessment by a single provider certified in both specialties.

Other mechanisms of care delivery common to combined clinics include regular screening, well-defined referral pathways, and use of diagnostic tools.[29] Implementation studies of combined clinics in Europe and the United States have reported improved communication among clinicians, prompt and accurate PsA diagnosis, and improved disease control, as well as improved quality of life with use of this model.[29–31]

Pathogenesis of Psoriatic Arthritis

Genetic Factors

The pathogenesis of PsA is characterized by inflammatory responses at multiple sites that are triggered and/or modulated by an interplay of genetic, immunologic, and environmental factors.[32] PsA is a highly heritable disease with a recurrence risk ratio (RRR) of 30 vs. 7 in patients with psoriasis.[32] RRR reflects the ratio of disease in family members of an affected individual compared with prevalence in the general population, so this figure is indicative of the genetic influence on PsA. A range of genetic factors may contribute to this strong familial component, including frequencies of major histocompatibility complex (MCH) class II alleles such as human leukocyte antigens (HLA)-B*08, B*27, and B*38.[33,34]

The MCH association with PsA appears to lie close to the HLA-B region of the MCH compared with psoriasis, which is more strongly associated with the HLA-C region. Many of the alleles found in PsA differ from those found in patients with RA and are linked to specific PsA phenotypes. For instance, HLA-B27 is associated with symmetric sacroiliitis, enthesitis, and dactylitis, while HLA-B08 is associated with asymmetric sacroiliitis, joint fusion and deformities, and dactylitis.[33,34] Other HLA alleles (e.g., HLA-B*39) are predictors of disease progression.[33,34] Non-HLA genes inside and outside the MCH are also thought to increase susceptibility for PsA, including killer immunoglobulin receptor (KIR) genes, genes that regulate nuclear factor kappa B (NF κB), and tumor necrosis factor (TNF) promotor polymorphisms.[35]

Autoimmune and Inflammatory Pathways

Genome-wide association and other studies have also identified several loci containing genes associated with immune function in patients with PsA that involve interleukin. Markers that are specific to PsA include variants in the gene encoding the interleukin-23 receptor (IL-23R), interleukin-12A (IL-12A), and the (p40) subunit of interleukin-12B (IL-12B).[36,37] Many of these genes encode proteins that are important in innate and adaptive immune responses.

Although the pathogenic link between the inflammatory T cell responses of psoriasis and the joint inflammation of PsA has not yet been fully explained, it has been hypothesized that PsA is triggered by autoinflammatory cytokine networks that respond to microbiome and mechanical stress signals in psoriatic skin and synovial lesions (Figure 1).[33,34,38]

Figure 1
Mechanisms of Inflammation in Psoriasis and PsA[22]

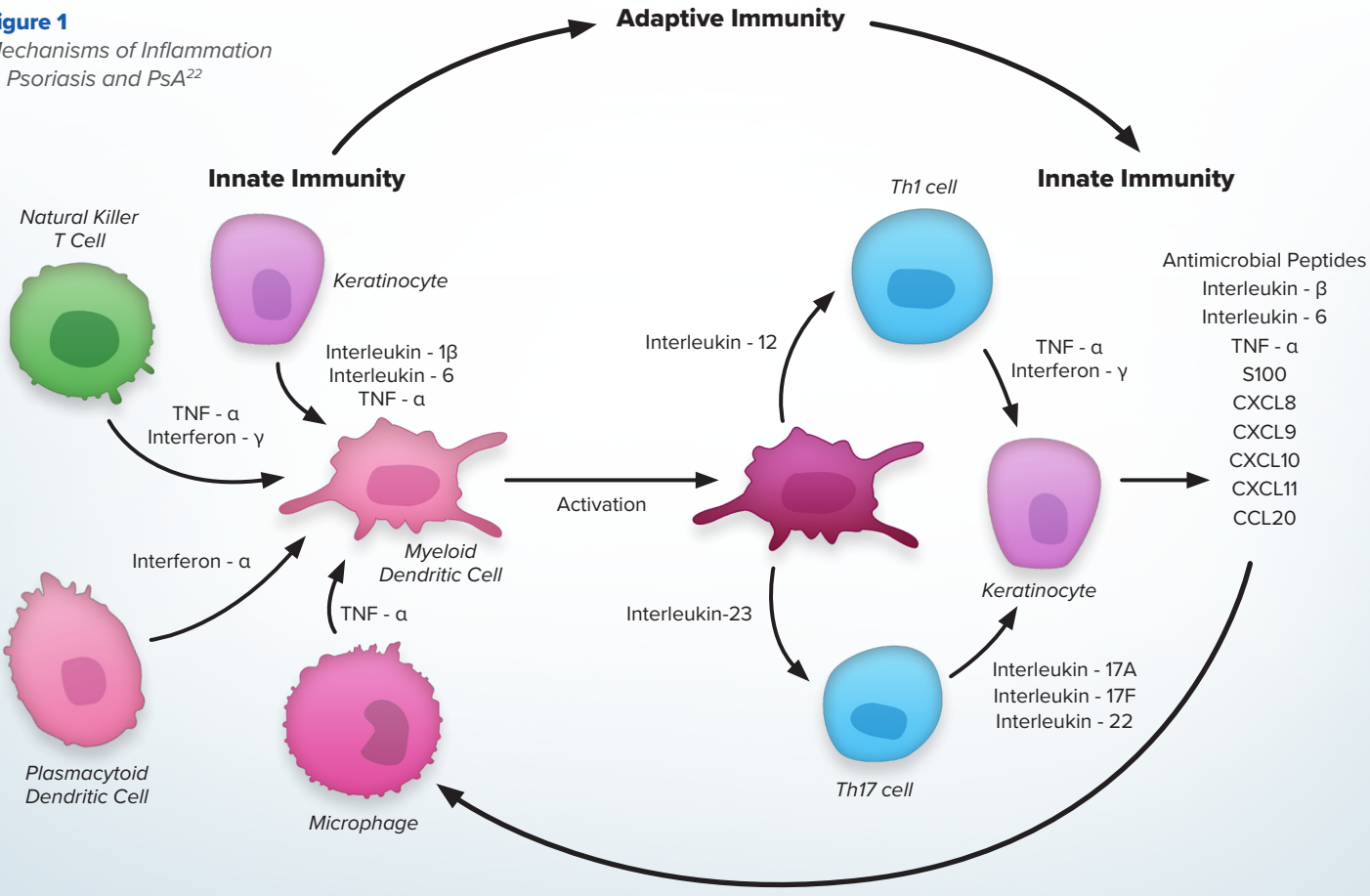


Table 2 *Environmental and Other Risk Factors for the Development of PsA*[3,26,27,34]

Risk Factor	How Risk Factors May Affect the Development of PsA
Obesity	• Systemic inflammation caused by adipose tissue • Increased mechanical loading on joints • Dyslipidemia
Nail Disease	• Associated with enthesitis • Precedes joint disease • Marker of immunoreactivity
Smoking	• Oxidative stress that stimulates inflammation • Nicotinic receptor activation that inhibits intracellular proinflammatory pathways
Alcohol	• Complex, undetermined
Trauma, Infection, and Stress	• Inflammation triggered by trauma in a genetically susceptible host - Biomechanical sheer stress, microtrauma - Streptococcal infection - Injury, heavy lifting
Microbiome	• Gut microbiota profile in PsA similar to that of inflammatory bowel disease • Dysbiosis may contribute to altered immune response by triggering IL-23 release and type 17 cells

Proinflammatory cytokines are major mediators of systemic and local inflammation. For instance, TNFα is produced by several types of immune cells and activates key effector cells involved in tissue inflammation. It was one of the earliest cytokines to be identified in the pathogenesis of PsA, as well as other inflammatory and autoimmune diseases.[33] Increased levels of circulating T helper 17 (Th17) cytokines have also been detected in the peripheral blood of patients with PsA, and IL-17 has been found in the skin, synovial tissue, and synovial fluid of patients with PsA.[34] Th17 pathways may stimulate release of inflammatory substances such as interleukin (IL)-12, IL-17, IL-22, IL-23, and TNF.[34] The exact mechanisms underlying these cytokine networks are unclear, but are thought to involve complex interactions between activated $CD4^+$ T-cells, $CD8^+$ T-cells, lymphocytes, and macrophages in the IL-23-IL-17 immune axis that result in osteolastogenesis, bone resorption, and cartilage destruction.[34,39]

Interleukin pathways that signal through the Janus-Kinase (JAK) family of receptor-associated tyrosine kinases are also implicated in the pathogenesis of PsA. JAKs activate signal transducer and activator of transcription (STATs) depending on the cytokine signal they receive and inhibit several pro-inflammatory cytokines, including IL-6, IL-12, and IL-23.[40]

Environmental Factors

Several environmental factors are thought to increase the risk of developing PsA by putting stress on joints. These factors include bacterial and viral infections, trauma (the Koebner phenomenon), and obesity (Table 2).

Clinical Features

Joint Inflammation and Extra-Articular Disease

In its simplest form, PsA represents an arthritis occurring in the presence of psoriasis and in the absence of a positive rheumatoid factor (RF).[7] However, PsA can present in myriad ways. Patients with PsA typically present with signs and symptoms of inflammation in peripheral joints (arthritis), the axial skeleton (spondylitis), sites where tendons, ligaments, and synovium attach to nails or bone (enthesitis, found in ~38% of patients), and extensive swelling of the soft tissues between the

metacarophalangeal and interphalangeal joints (dactylitis, found in ~30% of patients).[1,7] Approximately 15% of patients with PsA present with joint involvement only, while 15% present with both joint and skin involvement.[23] The Achilles and plantar fascia insertions are the most common entheseal sites; however, enthesitis can also occur at the insertions of the quadriceps and patellar tendons, iliac crest, rotator cuff, and epicondyles at the elbow.[7] Synovitis, tenosynovitis of the flexor tendons of the hands or the extensor carpi ulnaris, and morning stiffness lasting more than 30 minutes are also common presenting features.[39]

Extra-articular manifestations of PsA are common and appear to be associated with axial disease.[41] Up to 90% of patients with PsA have nail involvement that involves pitting, ridging, and distal onycholysis.[14] Other manifestations of extra-articular disease include iritis or uveitis (present in 8% of patients with PsA), oral ulceration, inflammatory bowel disease, and cardiovascular conditions (e.g., bundle branch block, increased arterial stiffness).[7,18,41,42] Distal limb edema or lymphedema has also been observed in approximately 20% of patients with PsA.[7]

Table 3 CASPAR Criteria[43]

*Psoriatic arthritis is considered to be present in patients with inflammatory articular disease (joint, spine, or enthesial) who have a score of **at least** 3 points from the 5 categories below.*

CASPAR Criteron	Points
1. Evidence of psoriasis (one of the following):	
a. Current psoriasis	2
b. Personal history of psoriasis	1
c. Family history of psoriasis	1
2. Psoriatic nail dystrophy (including onycholysis, pitting, hyperkeratosis)	1
3. Rheumatoid Factor negative	1
4. Dactylitis (one of the following):	
a. Swelling of entire digit	1
b. History of dactylitis	1
5. Radiographic evidence of juxtaarticular new bone formation	1

Classification of Clinical Features

Currently, there are no widely accepted diagnostic criteria for PsA. The Classification of Psoriatic Arthritis (CASPAR) criteria were developed in 2006 for use in the clinical trial setting, for which they have to be found to have high specificity and sensitivity.[7] These criteria recognize the potential variety in presentation of PsA and are based on the stem of inflammatory musculoskeletal disease. In clinical practice, providers may use these criteria to determine the presence of musculoskeletal inflammation. To meet the CASPAR criteria, a patient must have signs of inflammatory articular disease (joint, spine, or enthesial) with at least 3 points from the 5 categories (Table 3).[43]

The Group for Research and Assessment of Psoriasis and PsA (GRAPPA) has also developed practical criteria that clinicians in primary care and dermatology settings can use to identify inflammatory arthritis, enthesitis, dactylitis, and spondylitis (Table 4).[44]

PsA Screening Tools

Several tools with validated sensitivity and specificity exist to assist in screening patients with psoriasis for PsA, such as the Psoriatic Arthritis Screening and Evaluation (PASE) questionnaire, the Toronto Psoriatic Arthritis Screen (ToPAS), the Psoriatic Arthritis Screening Questionnaire (PASQ), and the Early Arthritis for Psoriatic Patients (EARP).[45] Because these tools can be time consuming to administer in primary care and dermatology clinics, researchers at Brigham and Women's Hospital have developed a screening tool that is currently being validated. The mnemonic "PSA" represents Pain, Swelling or Stiffness, and Axial disease, and can be used to identify patients with psoriasis and/or a strong family history of psoriasis who might benefit from a referral for a rheumatology work-up.[46]

Prognosis and Disease Progression

The median age of arthritis onset in patients with psoriasis is between 38 and 52 years.[7] While PsA is a progressive condition associated with loss of function in the first few years of the disease, a more severe course of PsA is associated with initial presentation at an earlier age, female gender, polyarticular involvement, genetic predisposition, and early radiographic signs of disease.[47] Dactylitis is considered an important marker for disease severity, as affected digits are more likely to show progressive radiographic change compared with nondactylitic digits.[2] Uveitis and nail disease (i.e., nail pits, nail bed hyperkeratosis, and splinter hemorrhages) are considered predictive of PsA,[10,26] and the severity of nail involvement may correlate with the severity and extent of joint disease.[48]

The risk of disease progression is higher for patients presenting with established PsA for more than 2 years than for those with a shorter history of joint involvement.[28] However, even delays of 6 months from diagnosis to

Table 4 *Definitions of PsA Patterns Defined by GRAPPA*[44]

Conditions	Descriptors
Inflammatory arthritis	• Age <40 years, 1 or more swollen joints, "boggy" swelling rather than bony swelling, tenderness, pain worse with rest, pain improves with activity, morning stiffness, nighttime pain, distribution of joints, daily symptoms, duration >6 weeks, decreased mobility, fatigue, weight loss, sweats • Extraarticular manifestations (psoriasis, nail disease, IBD, enthesitis, inflammatory back pain) • Family history of IBD, uveitis, or associated inflammatory disease • Prednisone responsive • Recurrent flares
Enthesitis	• Morning pain and stiffness, acute onset, persistent • Tenderness at tendon/ligament insertions • Multiple sites of involvement • Plantar fasciitis, Achilles tendonitis and patellar tendonitis • Rib or iliac crest pain • Possibly NSAID responsive • Diminished functional ability • Associated swelling, erythema, diffuse tenderness
Dactylitis	• Swelling of entire finger or toe, diffusely tender, erythema of entire digit • Asymmetric distribution of digits • Duration of a few weeks or more • Recurrent episodes • Chronic dactylitis may not be tender • Other manifestations including arthritis
Spondylitis	• Consider ASAS criteria* • Age <40 years, neck pain, back pain improved with activity/exercise, pain worse with rest, morning stiffness, nighttime pain, NSAID responsive, duration >3 months, alternating buttock pain, pain not affected by positioning, extraspinal symptoms (psoriasis, nail disease, enthesitis, arthritis, dactylitis, gastrointestinal symptoms, uveitis), family history of psoriatic arthritis or ankylosing spondylitis • Diminished chest wall expansion/lateral flexion, positive Patrick's test

**ASAS = Assessment in SpondyloArthritis International Society; IBD = inflammatory bowel disease; NSAID = nonsteroidal anti-inflammatory drug*

specialist consultation are associated with more erosions, sacroiliitis, and diminished quality of life.[49,50] Patients with elevated erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), anti-citrullinated protein antibody (ACPA)-positive status, and polyarticular disease are also at increased risk for disease progression.[51] ACPAs are present in up to 13% of patients with PsA and are also associated with erosive features and multiple joint involvement.[22] Longitudinal studies show that more than half of patients with PsA experience arthritis that is deforming, 17% have five or more deformed joints, 20–40% have spinal involvement, and 11–19% are disabled.[7]

Diagnostic Assessment and Evaluation

Physical Findings

In patients with suspected PsA, it is important to evaluate the severity and extent of psoriasis as well as the following five musculoskeletal domains of PsA (Figure 2):[3]

- Peripheral arthritis
- Axial disease
- Enthesitis
- Dactylitis
- Skin and nail disease

On examination, palpation of distal joints typically reveals soft swelling and tenderness due to inflammation. Effusions are typically present in the affected joints. The pattern of joint involvement in PsA varies and can change for individual patients over time.[1]

Polyarticular PsA affects approximately 63% of patients and involves ≥5 joints in a symmetric fashion that resembles RA, whereas mono- and asymmetric oligoarthritis, which affects approximately 13% of patients, involves <5 joints in an asymmetrical distribution.[47] The distal subtype exclusively affects distal interphalangeal (DIP) joints and occurs in approximately 5% of patients, while arthritis mutilans is rare and is characterized by telescoping and flail digits caused by marked bone resorption or osteolysis.[3] Although spondyloarthritis is

relatively uncommon, spinal involvement may occur in ~40% of patients with PsA.[3] Concomitant PsA and RA is possible, but rare.

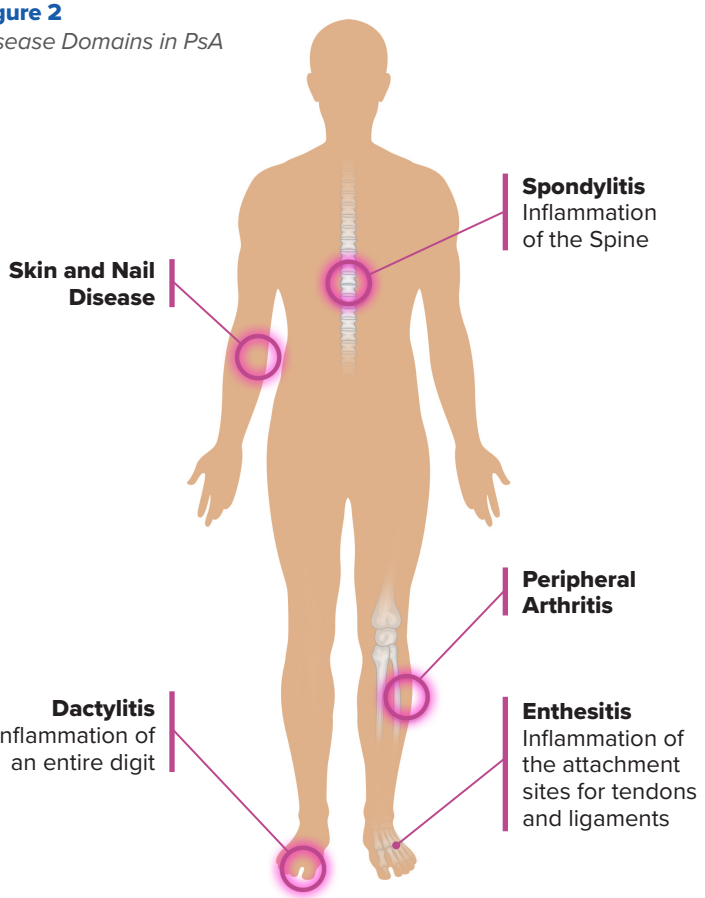
Early diagnosis can be especially challenging when arthritis is present without skin lesions. Patients with PsA often have a family history of psoriasis or PsA, but in patients with classic clinical features of PsA but no history of psoriasis, it is important to examine the patient's skin, scalp, and nails for signs of psoriasis.[7] Clinicians should ask their patients with psoriasis about signs and symptoms that might suggest PsA, such as morning stiffness lasting more than 30 minutes.[39]

Differential Diagnosis

Several arthropathies share some of the characteristics of PsA, including RA, erosive osteoarthritis (OA), crystal arthritis (including gout and pseudogout) and other seronegative spondyloarthropathies, including Reiter's syndrome, ankylosing spondylitis, and the arthritis of inflammatory bowel disease.[3] The monoarthritic subtype of PsA can be misdiagnosed as gout or pseudogout, especially when toes or dactylitis are involved, while the distal joint involvement associated with PsA is also observed in osteoarthritis (Table 5).[3]

A major differentiating feature of PsA compared with RA is that distal joints are affected in more half of all patients with PsA, with all joints of the same digit involved. In RA, the proximal joints of the hands and feet are typically affected in a symmetrical fashion. Other articular features that distinguish PsA from RA include dactylitis, DIP involvement, and entheseal inflammation. Inflammatory back pain or sacroiliitis on X-ray or MRI is a further distinguishing feature of PsA, since spinal involvement is uncommon in RA.[3]

Figure 2
Disease Domains in PsA



Radiographic Findings

Erosive radiographic change is a diagnostic hallmark of PsA; therefore, plain radiographic imaging remains the gold standard for establishing baseline joint damage in peripheral joints in patients with PsA.[39,47]

Approximately half (47%) of patients with recent-onset PsA develop erosions within 2 years of disease onset, while 67% with established PsA have radiographic abnormalities.[52] Radiographic findings in PsA are distinct from patterns of change associated with other forms of inflammatory arthritis and typically include the following:[3,7]

- Asymmetric joint involvement
- Involvement of the interphalangeal joints of fingers and toes
- Joint space narrowing or involvement of entheseal sites
- New bone formation (e.g., periostitis and fusion) in the same joint or different joints in the same digit
- Soft tissue swelling
- Subluxation
- Bony erosion and resorption that results in 'pencil in cup' deformity (arthritis mutilans)
- Spinal involvement (less severe and asymmetric than in classic ankylosing spondylitis)
- Patients with spondylitis may also exhibit sacroiliitis and vertebral syndesmophytes that bridge adjacent vertebrae

MRI is more sensitive and specific than radiographs for identifying sacroiliitis and changes in the axial skeleton, and Doppler ultrasound may help to identify synovitis, enhanced blood flow, tenosynovitis, enthesophytes, and early erosions.[53]

Laboratory Tests

There are no specific serologic tests for PsA. Tests for RF, ACPA, or antinuclear antibodies (ANA) are typically negative, although they may be present in ~10% of patients with PsA. The following are also indicative of PsA:[3,7]

- HLA B*27 is present in ~8% of the general population but is present in ~25% of patients with axial inflammation and is considered a genetic marker for PsA among patients with psoriasis

Table 5 *Differentiating Features of Various Forms of Arthritis*[3]

Variable	Psoriatic Arthritis	Rheumatoid Arthritis	Osteoarthritis	Gout
Age at Onset	36-45	30-50	>50	30-50
Male to Female Ratio	1:1	F>M	1:1	M>F
Onset	Gradual	Acute/gradual	Gradual	Acute
Joint Distributiion at Onset	Asymmetric	Symmetric	Asymmetric	Asymmetric
Number of Affected Joints	Oligoarticular	Polyarticular	Monoarticular or oligoarticular	Monoarticular or oligoarticular
Sites of Hands/Feet Involved	Distal	Proximal	Distal	Distal
Areas Involved	All points of digit	Same joint across digits	Same joint across digits	Usually monoarticular
Purplish Discoloration	Yes	No	No	Yes
Spinal Involvement	Common	Uncommon	Noninflammatory	Absent
Sacroiliitis	Common	Absent	Absent	Absent
Key Radiographic Findings	• Joint space narrowing • Bone/cartilage destruction and bone proliferation	• Joint space narrowing • Bone and cartilage erosions in hands and feet	• Focal changes that reflect cartilage loss	• Subcortical bone cysts (tophi)
Key Laboratory Findings	• RF, ACPA generally negative • HLA-B27 positive in ~25% cases • Elevated ESR, CRP in ~40% cases	• Elevated ESR, CRP • RF is generally positive in 75%-80% patients • Positive ANA • Positive Anti-CCP in 60-80% cases	• Normal ESR or CRP	• Presence of monosodium urate crystals in synovial fluid • Elevated WBC

RF = rheumatoid factor; ACPA = anti-citrullinated protein antibody; HLA = human leukocyte antigens; ESR = erythrocyte sedimentation rate; CRP = C-reactive protein; ANA = antinuclear antibodies; WBC = white blood cells

- Arthrocentesis of active joints typically shows inflammatory synovial fluid and absence of crystals
- Elevation of acute phase reactants (ESR, CRP) is common in patients with RA, and may also be elevated in ~40% of PsA patients, especially those with polyarticular disease
- Hyperuricemia may be observed in association with metabolic abnormalities
- RF in PsA is generally negative but can be positive in ~10% of patients

Serum biomarkers to predict which patients with psoriasis are likely to develop PsA are currently being developed.[45]

Current Therapy for Psoriatic Arthritis

The long-term goals of treating patients with PsA are to maintain health-related quality of life, limit skin and joint symptoms and signs, prevent or slow structural damage, achieve the lowest possible level of disease activity in all domains of disease, and avoid or minimize complications.[51,54] The European League Against Rheumatism (EULAR) and the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) have devised evidence-based therapeutic schema that address specific clinical manifestations and target the five main disease domains noted earlier in this issue.[51,54]

Table 6 *Cytokines, Pathways, Current Therapies*[41,45,55]

Drug Class	Drug Name	Pathway	Approval Status
TNF-alpha (α) inhibitors	Etanercept	Dimeric p75 TNFα receptor Fc fragment fusion protein that binds TNF	• Approved 2002 to reduce signs and symptoms of active arthritis associated with PsA • Effective across disease domains
	Golimumab	Novel monoclonal antibody against TNFα	• Approved 2017 for adults with active PsA • Effective across disease domains
	Infliximab	Chimeric monoclonal antibody against TNFα	• Approved 1998 for adults with active PsA • Effective across disease domains
	Adalimumab	Human monoclonal antibody with a high affinity for TNFα	• Indicated for adults with PsA in whom conventional therapies have failed or are not tolerated • Effective for peripheral arthritis, nail involvement, skin psoriasis
	Certolizumab	PEGylated Fab' fragment of a humanized anti–TNFα monoclonal antibody	• Approved 2013 for adults with active PsA Effective across disease domains
Phosphodiesterase-4 inhibitor	Apremilast	Increases levels of cAMP, resulting in decreased levels of proinflammatory cytokines	• Approved 2014 for adults with active PsA • Effective for peripheral arthritis, enthesitis, nail involvement, skin psoriasis
Interleukin inhibitors	Ustekinumab	Human monoclonal antibody directed against the p40 subunit of IL-12/23	• Approved 2013 for adults with active PsA who have not responded to DMARDs • Effective for peripheral arthritis, sacroiliitis + spinal disease, enthesitis, dactylitis, skin psoriasis
	Secukinumab	IL17A monoclonal antibody	• Approved 2016 for adults with active PsA • Effective for peripheral arthritis, sacroiliitis + spinal disease, enthesitis, dactylitis
	Ixekizumab	Humanized monoclonal antibody against IL 17A	• Approved 2017 for adults with active PsA • Effective across disease domains
	Brodalumab	Monoclonal antibody that targets and blocks the signaling pathway of interleukin receptors (IL17A, IL17F and IL23)	• Has shown benefit for patients with PsA in clinical trials but has not yet received FDA approval for PsA • Effective for peripheral arthritis, sacroiliitis + spinal disease
JAK inhibitor	Tofacitinib	Oral inhibitor of JAK1, JAK3, and to a lesser extent, JAK2	• Approved 2017 for adults with active PsA who do not respond to DMARDs • Effective across disease domains
T cell modulator	Abatacept	CTLA4-Ig is a selective T-Cell costimulation modulator	• Approved 2017 for adults with active PsA • Effective for peripheral arthritis, skin psoriasis

NSAIDs may be used for treating mild symptoms or limited joint involvement, while intraarticular corticosteroid injections are recommended as adjunctive therapy for inflamed joints. Systemic therapy is recommended for patients with three or more inflamed joints despite conventional therapy, or in patients with persistent axial, entheseal, or dactylic disease. While there is little clinical data to support the use of synthetic disease-modifying anti-rheumatic drugs (DMARDs) in PsA (i.e., methotrexate [MTX], cyclopsporine, leflunomide), historically, oral MTX has been commonly prescribed as first-line therapy.[7] MTX reduces joint tenderness and swelling by increasing the release of adenosine (an anti-inflammatory agent) at the sites of inflammation, and early treatment with MTX is associated with mild inhibition of joint damage progression and improved health-related quality of life.[39] However, not all patients respond to MTX. Moreover, enthesis and spinal inflammation are more responsive to anti-TNF agents than to DMARDs.

Biologic Therapies

EULAR guidelines published in 2012 recommend treatment of PsA with a TNF inhibitor after the failure of one DMARD in patients with active disease (i.e., a high number of active joints, high inflammatory markers, and ongoing joint damage).[54] Five TNF inhibitors (etanercept, adalimumab, infliximab, golimumab, and certolizumab) are approved by the U.S. Food and Drug Administration (FDA) for the treatment of PsA and have demonstrated efficacy in treating skin and joint involvement as well as in preventing radiographic damage.[55] These five agents are also approved for the treatment of ankylosing spondylitis and are effective for the treatment of spondylitis in patients with PsA. However, TNF inhibitors are associated with a wide spectrum of hematologic and metabolic adverse events (e.g., opportunistic infections such as tuberculosis, cytopenias, lymphoma, heart failure, and hepatotoxicity), and as many as 45% of patients with PsA are dissatisfied with their use.[56] Indeed, adverse events are one of the principle reasons that patients report for discontinuing therapy with biologic agents.[56]

The range of biologic therapies available for treating PsA has rapidly expanded, with the development of several monoclonal antibodies that target interleukin pathways, modulate T cells, and inhibit JAK pathways.[57-60] These agents have demonstrated efficacy in patients with PsA naïve to anti-TNF therapy as well as in those who have had prior TNF inhibitor treatment (Table 6).

Ustekinumab is a monoclonal antibody that binds to the p40 subunit of interleukin (IL)-12 and IL-23 and inhibits the activity of these cytokines.[61] This agent has demonstrated efficacy in patients with active PsA despite use of a traditional DMARD or receipt of a prior TNF inhibitor. Ustekinumab can be administered as monotherapy or in combination with MTX.[58]

IL-17A blockade is also a therapeutic target in patients with PsA. Secukinumab is a fully human, high-affinity anti-IL-17A monoclonal antibody that is approved by the FDA for the treatment of patients with PsA. Ixekizumab is a second anti-IL-17 monoclonal antibody approved for treating patients with PsA. Apremilast, meanwhile, is an oral phosphodiesterase 4 (PDE 4) inhibitor that has demonstrated clinical benefit for patients with PsA and is now FDA-approved for patients with active PsA.[62] Abatacept is a T-cell modulator also approved for use in patients with active PsA. Finally, tofacitinib is a JAK inhibitor that was approved in 2017 for adults with active PsA who do not respond to DMARDs.

Much more about the treatment of PsA will be included in issues of *Rheumatology Nurse Practice* later this year.

Monitoring Treatment Response

A treat-to-target approach is recommended to assess treatment efficacy in patients with PsA. Until recently, approaches to assessing treatment response were largely derived from measures used in RA.[22] However, remission and disease activity criteria designed for RA are not considered feasible in clinical practice for the classification of PsA response and have proven to be limited in their capacity to evaluate all domains of PsA.[1] In recent years, several indices and composite measures for assessing remission and disease activity have been developed that show high sensitivity and specificity for PsA.[63] These measures include the Psoriatic Arthritis Disease Activity Score (PASDAS), the Composite Psoriatic Disease Activity Index (CPDAI), and minimal disease activity (MDA), which is defined by clinically significant improvement in five of seven response measures or domains.[63,64] These composite indices are considered more efficient in measuring disease activity and remission than unidimensional instruments and may be helpful in setting treatment goals.

Summary

PsA is a chronic and progressive inflammatory arthritis that confers significant morbidity on patients. Challenges clearly persist in the recognition and diagnosis of PsA, but clinicians in primary care are keenly poised to screen patients with psoriasis and, if necessary, refer patients for evaluation by a rheumatologist. Early recognition and diagnosis is key to ensure that patients with PsA have access to the rapidly expanding treatment options.

References

1. Ritchlin CT, Kavanaugh A, Gladman DD, et al. Treatment recommendations for psoriatic arthritis. *Ann Rheum Dis.* 2009;68(9):1387-1394.
2. Helliwell PS, Ruderman EM. Natural history, prognosis, and socioeconomic aspects of psoriatic arthritis. *Rheum Dis Clin North Am.* 2015;41(4):581-591.
3. Ritchlin CT, Colbert RA, Gladman DD. Psoriatic arthritis. *N Engl J Med.* 2017;376(10):957-970.
4. Lebwohl MG, Bachelez H, Barker J, et al. Patient perspectives in the management of psoriasis: results from the population-based Multinational Assessment of Psoriasis and Psoriatic Arthritis Survey. *J Am Acad Dermatol.* 2014;70(5):871-881.e871-830.
5. Ogdie A, Weiss P. The epidemiology of psoriatic arthritis. *Rheum Dis Clin North Am.* 2015;41(4):545-568.
6. Gladman DD, Antoni C, Mease P, Clegg DO, Nash P. Psoriatic arthritis: epidemiology, clinical features, course, and outcome. *Ann Rheum Dis.* 2005;64 Suppl 2:ii14-17.
7. FitzGerald O, Elmamoun M. Psoriatic Arthritis. In: *Kelley and Firestein's Textbook of Rheumatology.* 2017:1285-1308.e1284.
8. Rachakonda TD, Schupp CW, Armstrong AW. Psoriasis prevalence among adults in the United States. *J Am Acad Dermatol.* 2014;70(3):512-516.
9. Gelfand JM, Gladman DD, Mease PJ, et al. Epidemiology of psoriatic arthritis in the population of the United States. *J Am Acad Dermatol.* 2005;53(4):573.
10. Eder L, Haddad A, Rosen CF, et al. The Incidence and Risk Factors for Psoriatic Arthritis in Patients With Psoriasis: A Prospective Cohort Study. *Arthritis Rheum (Hoboken).* 2016;68(4):915-923.
11. Wilson FC, Icen M, Crowson CS, McEvoy MT, Gabriel SE, Kremers HM. Time trends in epidemiology and characteristics of psoriatic arthritis over 3 decades: a population-based study. *J Rheumatol.* 2009;36(2):361-367.
12. Wilson FC, Icen M, Crowson CS, McEvoy MT, Gabriel SE, Kremers HM. Incidence and clinical predictors of psoriatic arthritis in patients with psoriasis: a population-based study. *Arthritis Rheum.* 2009;61(2):233-239.
13. Liu JT, Yeh HM, Liu SY, Chen KT. Psoriatic arthritis: Epidemiology, diagnosis, and treatment. *World J Orthop.* 2014;5(4):537-543.
14. Husni ME, Merola JF, Davin S. The psychosocial burden of psoriatic arthritis. *Semin Arthritis Rheum.* 2017;47(3):351-360.
15. Tillett W, Shaddick G, Askari A, et al. Factors influencing work disability in psoriatic arthritis: first results from a large UK multicentre study. *Rheumatology (Oxford).* 2015;54(1):157-162.
16. Lee S, Mendelsohn A, Sarnes E. The burden of psoriatic arthritis: a literature review from a global health systems perspective. *P T.* 2010;35(12):680-689.
17. Truong B, Rich-Garg N, Ehst BD, et al. Demographics, clinical disease characteristics, and quality of life in a large cohort of psoriasis patients with and without psoriatic arthritis. *Clin Cosmet Investig Dermatol.* 2015;8:563-569.
18. Husted JA, Thavaneswaran A, Chandran V, Gladman DD. Incremental effects of comorbidity on quality of life in patients with psoriatic arthritis. *J Rheumatol.* 2013;40(8):1349-1356.
19. Mease PJ, Gladman DD, Papp KA, et al. Prevalence of rheumatologist-diagnosed psoriatic arthritis in patients with psoriasis in European/North American dermatology clinics. *J Am Acad Dermatol.* 2013;69(5):729-735.
20. Lebwohl MG, Kavanaugh A, Armstrong AW, Van Voorhees AS. US Perspectives in the Management of Psoriasis and Psoriatic Arthritis: Patient and Physician Results from the Population-Based Multinational Assessment of Psoriasis and Psoriatic Arthritis (MAPP) Survey. *Am J Clin Derm.* 2016;17(1):87-97.
21. Boehncke WH. Psoriasis and Psoriatic Arthritis: Flip Sides of the Coin? *Acta Derm Venereol.* 2016;96(4):436-441.
22. Mease PJ, Armstrong AW. Managing patients with psoriatic disease: the diagnosis and pharmacologic treatment of psoriatic arthritis in patients with psoriasis. *Drugs.* 2014;74(4):423-441.
23. Gladman DD, Shuckett R, Russell ML, Thorne JC, Schachter RK. Psoriatic arthritis (PSA)--an analysis of 220 patients. *Q J Med.* 1987;62(238):127-141.
24. Villani AP, Rouzaud M, Sevrain M, et al. Prevalence of undiagnosed psoriatic arthritis among psoriasis patients: Systematic review and meta-analysis. *J Am Acad Dermatol.* 2015;73(2):242-248.
25. Haroon M, Kirby B, FitzGerald O. High prevalence of psoriatic arthritis in patients with severe psoriasis with suboptimal performance of screening questionnaires. *Ann Rheum Dis.* 2013;72(5):736-740.
26. Ogdie A, Gelfand JM. Clinical Risk Factors for the Development of Psoriatic Arthritis Among Patients with Psoriasis: A Review of Available Evidence. *Curr Rheumatol Rep.* 2015;17(10):64.
27. van de Kerkhof PC, Reich K, Kavanaugh A, et al. Physician perspectives in the management of psoriasis and psoriatic arthritis: results from the population-based Multinational Assessment of Psoriasis and Psoriatic Arthritis survey. *J Eur Acad Dermatol Venereol.* 2015;29(10):2002-2010.
28. Soleymani T, Reddy SM, Cohen JM, Neimann AL. Early Recognition and Treatment Heralds Optimal Outcomes: the Benefits of Combined Rheumatology-Dermatology Clinics and Integrative Care of Psoriasis and Psoriatic Arthritis Patients. *Curr Rheumatol Rep.* 2017;20(1):1.
29. Okhovat JP, Ogdie A, Reddy SM, Rosen CF, Scher JU, Merola JF. Psoriasis and Psoriatic Arthritis Clinics Multicenter Advancement Network Consortium (PPACMAN) Survey: Benefits and Challenges of Combined Rheumatology-dermatology Clinics. *J Rheumatol.* 2017;44(5):693-694.
30. Favier G, Gladman DD, Merola JF, Armstrong AW, Boehncke WH, Helliwell PS. Benchmarking Care in Psoriatic Arthritis - The QUANTUM Report: A Report from the GRAPPA 2016 Annual Meeting. *J Rheumatol.* 2017;44(5):674-678.
31. Luelmo J, Gratacos J, Moreno Martinez-Losa M, et al. A report of 4 years of experience of a multidisciplinary unit of psoriasis and psoriatic arthritis. *Reumatol Clin.* 2014;10(3):141-146.
32. Chandran V, Schentag CT, Brockbank JE, et al. Familial aggregation of psoriatic arthritis. *Ann Rheum Dis.* 2009;68(5):664-667.
33. FitzGerald O, Haroon M, Giles JT, Winchester R. Concepts of pathogenesis in psoriatic arthritis: genotype determines clinical phenotype. *Arthritis Res Ther.* 2015;17:115.
34. Barnas JL, Ritchlin CT. Etiology and Pathogenesis of Psoriatic Arthritis. *Rheum Dis Clin North Am.* 2015;41(4):643-663.
35. Chandran V, Bull SB, Pellett FJ, Ayearst R, Pollock RA, Gladman DD. Killer-cell immunoglobulin-like receptor gene polymorphisms and susceptibility to psoriatic arthritis. *Rheumatology (Oxford).* 2014;53(2):233-239.
36. Rahman P, Inman RD, Gladman DD, Reeve JP, Peddle L, Maksymowych WP. Association of interleukin-23 receptor variants with ankylosing spondylitis. *Arthritis Rheum.* 2008;58(4):1020-1025.
37. Rahman P, Inman RD, Maksymowych WP, Reeve JP, Peddle L, Gladman DD. Association of interleukin 23 receptor variants with psoriatic arthritis. *J Rheumatol.* 2009;36(1):137-140.
38. Diani M, Altomare G, Reali E. T cell responses in psoriasis and psoriatic arthritis. *Autoimmun Rev.* 2015;14(4):286-292.
39. Boehncke WH QA, Merola JF, et al. Diagnosing and Treating Psoriatic Arthritis. *Brit J Derm.* 2012;170(4):772-786.
40. Gao W, McGarry T, Orr C, McCormick J, Veale DJ, Fearon U. Tofacitinib regulates synovial inflammation in psoriatic arthritis, inhibiting STAT activation and induction of negative feedback inhibitors. *Ann Rheum Dis.* 2016;75(1):311-315.
41. Kerschbaumer A, Fenzl KH, Erlacher L, Aletaha D. An overview of psoriatic arthritis - epidemiology, clinical features, pathophysiology and novel treatment targets. *Wien Klin Wochenschr.* 2016;128(21-22):791-795.
42. Ruiz DG, Azevedo MN, Santos OL. Psoriatic arthritis: a clinical entity distinct from psoriasis? *Rev Bras Reumatola.* 2012;52(4):630-638.
43. Taylor W GD, Helliwell P et al. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. *Arthritis Rheum.* 2006;54:2665–2673.
44. Mease PJ, Park JJ, Garg A, Gladman DD, Helliwell PS. Development of Simple Clinical Criteria for the Definition of Inflammatory Arthritis, Enthesitis, Dactylitis, and Spondylitis: A Report from the GRAPPA 2014 Annual Meeting. *J Rheumatol.* 2015;42(6):1041-1043.
45. Raychaudhuri SP, Wilken R, Sukhov AC, Raychaudhuri SK, Maverakis E. Management of psoriatic arthritis: Early diagnosis, monitoring of disease severity and cutting edge therapies. *J Autoimmun.* 2017;76:21-37.

46. Cohen JM, Husni ME, Qureshi AA, Merola JF. Psoriatic arthritis: it's as easy as "PSA". *J Am Acad Dermatol.* 2015;72(5):905-906.

47. van de Kerkhof PCM NF. Psoriasis. In: JL Bologna JS, L. Cerroni ed. *Dermatology.* 4th ed. New York: Elsevier; 2018:138-160.

48. Williamson L, Dalbeth N, Dockerty JL, Gee BC, Weatherall R, Wordsworth BP. Extended report: nail disease in psoriatic arthritis--clinically important, potentially treatable and often overlooked. *Rheumatology (Oxford).* 2004;43(6):790-794.

49. Gladman DD, Thavaneswaran A, Chandran V, Cook RJ. Do patients with psoriatic arthritis who present early fare better than those presenting later in the disease? *Ann Rheum Dis.* 2011;70(12):2152-2154.

50. Haroon M, Gallagher P, FitzGerald O. Diagnostic delay of more than 6 months contributes to poor radiographic and functional outcome in psoriatic arthritis. *Ann Rheum Dis.* 2015;74(6):1045-1050.

51. Coates LC, Kavanaugh A, Mease PJ, et al. Group for Research and Assessment of Psoriasis and Psoriatic Arthritis 2015 Treatment Recommendations for Psoriatic Arthritis. *Arthritis Rheum (Hoboken).* 2016;68(5):1060-1071.

52. Kane D, Stafford L, Bresnihan B, FitzGerald O. A prospective, clinical and radiological study of early psoriatic arthritis: an early synovitis clinic experience. *Rheumatology (Oxford).* 2003;42(12):1460-1468.

53. Poggenborg RP, Ostergaard M, Terslev L. Imaging in Psoriatic Arthritis. *Rheum Dis Clin N Am.* 2015;41(4):593-613.

54. Gossec L, Smolen JS, Gaujoux-Viala C, et al. European League Against Rheumatism recommendations for the management of psoriatic arthritis with pharmacological therapies. *Ann Rheum Dis.* 2012;71(1):4-12.

55. Elyoussfi S, Thomas BJ, Ciurtin C. Tailored treatment options for patients with psoriatic arthritis and psoriasis: review of established and new biologic and small molecule therapies. *Rheum Int.* 2016;36(5):603-612.

56. Armstrong AW, Robertson AD, Wu J, Schupp C, Lebwohl MG. Undertreatment, treatment trends, and treatment dissatisfaction among patients with psoriasis and psoriatic arthritis in the United States: findings from the National Psoriasis Foundation surveys, 2003-2011. *JAMA Derm.* 2013;149(10):1180-1185.

57. Leonardi CL, Kimball AB, Papp KA, et al. Efficacy and safety of ustekinumab, a human interleukin-12/23 monoclonal antibody, in patients with psoriasis: 76-week results from a randomised, double-blind, placebo-controlled trial (PHOENIX 1). *Lancet.* 2008;371(9625):1665-1674.

58. Gottlieb A, Menter A, Mendelsohn A, et al. Ustekinumab, a human interleukin 12/23 monoclonal antibody, for psoriatic arthritis: randomised, double-blind, placebo-controlled, crossover trial. *Lancet.* 2009;373(9664):633-640.

59. Kavanaugh A, Ritchlin C, Rahman P, et al. Ustekinumab, an anti-IL-12/23 p40 monoclonal antibody, inhibits radiographic progression in patients with active psoriatic arthritis: results of an integrated analysis of radiographic data from the phase 3, multicentre, randomised, double-blind, placebo-controlled PSUMMIT-1 and PSUMMIT-2 trials. *Ann Rheum Dis.* 2014;73(6):1000-1006.

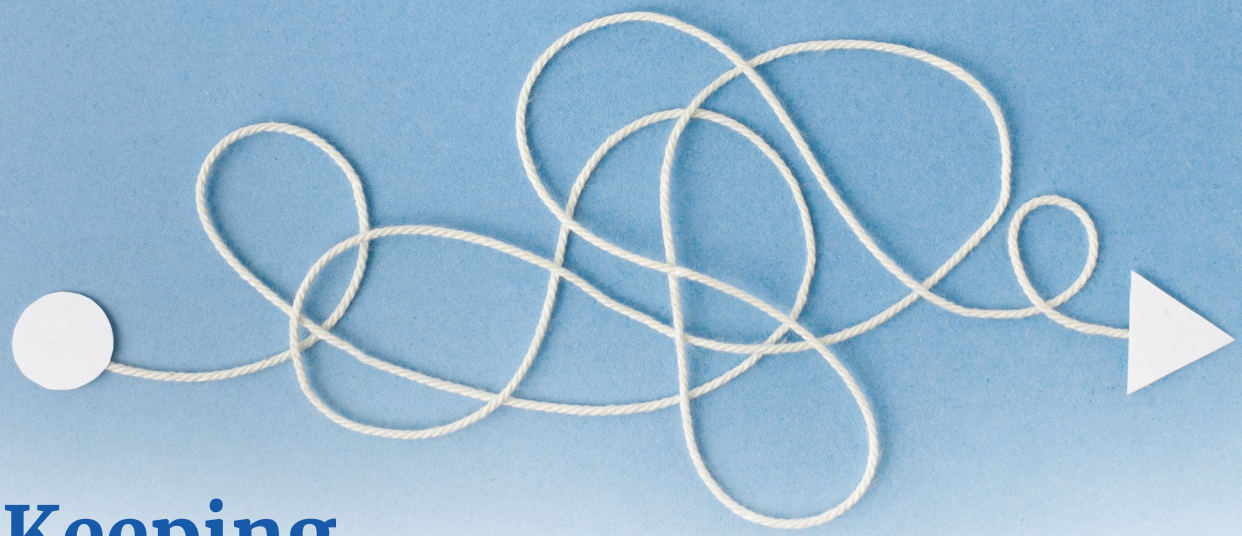
60. Ritchlin C, Rahman P, Kavanaugh A, et al. Efficacy and safety of the anti-IL-12/23 p40 monoclonal antibody, ustekinumab, in patients with active psoriatic arthritis despite conventional non-biological and biological anti-tumour necrosis factor therapy: 6-month and 1-year results of the phase 3, multicentre, double-blind, placebo-controlled, randomised PSUMMIT 2 trial. *Ann Rheum Dis.* 2014;73(6):990-999.

61. Mease PJ. Current and Emerging Treatments for Psoriatic Arthritis. In: Yamauchi PS, ed. *Biologic and Systemic Agents in Dermatology.* Cham: Springer International Publishing; 2018:175-185.

62. Apremilast prescribing information. Available at www.accessdata.fda.gov/drugsatfda_docs/label/2014/206088s000lbl.pdf. 2014. Accessed April 1, 2018.

63. Mease PJ, Coates LC. Considerations for the definition of remission criteria in psoriatic arthritis. *Semin Arthritis Rheum.* 2018;47(6):786-796.

64. Coates LC, Fransen J, Helliwell PS. Defining minimal disease activity in psoriatic arthritis: a proposed objective target for treatment. *Ann Rheum Dis.* 2010;69(1):48-53.



Keeping Complex Patients on the Right Path

Eileen J. Lydon, MA, RN, ANP-BC

The management of psoriatic arthritis (PsA) is often complex. It rarely involves the straightforward management of joint pain and psoriasis alone—there are frequently exacerbating comorbidities that need to be identified early and managed successfully in conjunction with the psoriatic disease to give our patients the best chances at success.

Three years ago, I was presented with a complex challenge when I first met JT, a 28-year-old female with a history of morbid obesity, insulin resistance, non-alcoholic fatty liver disease, and hypothyroidism. She had arthritis in her hands, wrist, knee, low back and bilateral shoulders. She also had a longstanding history of psoriasis (diagnosed at age 10) that affected her scalp, legs, elbows, ears, and gluteal cleft, as well as nail pitting.

During our initial meeting, JT said her lower back pain was her most troubling symptom, awakening her nearly every night and causing approximately 1 hour of daily morning stiffness. In addition to her other previously noted issues, JT also reported frequent loose stools and had had several recent bouts of bloody diarrhea.

Despite her symptoms, JT was working long hours as a paralegal and finishing law school with plans to take the bar exam in a few months. She reported feeling fatigued and had difficulty motivating herself to be productive at work and school. All of the recent stress was exacerbating her overall anxiety.

A serological and radiographic evaluation led to a diagnosis of PsA with evidence of oligoarthritis, sacroillitis, and psoriasis. Given JT's history of non-alcoholic fatty liver disease and elevated liver function tests—which made methotrexate an unwise choice—we initially discussed biologic monotherapy as a treatment option. I explained the risks and benefits of biologic therapies and provided JT with both written and verbal material about PsA, including a discussion of her specific comorbidities (ie, obesity, insulin resistance, and non-alcoholic fatty liver disease).

JT seemed a bit nervous, but she agreed to give a biologic a try, and adalimumab every other week was initiated. JT was also referred to a gastroenterologist based upon our concerns of inflammatory bowel disease, with planned follow-up in our office 1 month later.

JT returned to our office with an additional diagnosis of Crohn's disease. Because adalimumab is indicated for Crohn's disease as well as PsA, we maintained our previous treatment regimen. After only two doses, JT enthusiastically reported less joint pain, as well as improvement in her psoriasis, diarrheal symptoms, and fatigue. However, JT also expressed anxiety over our previous conversations regarding potential medication side effects and seemed overwhelmed about how to manage her new diagnoses along with her stressful lifestyle. I reassured her during a comprehensive review of side effects and disease management approaches, but I also suggested that her recent nervousness, fatigue, and lack of productivity could be a sign of anxiety and depression, which are both associated with PsA.[1,2] JT agreed that she had been feeling "off," but she attributed that to just overall life stress and not her disease.

At this point, things began to slowly unravel. JT missed several scheduled follow-up appointments and didn't return to the office for 6 months. By the time I saw her again, she had discontinued adalimumab because she "kept forgetting" to take the medication and did not like self-administering the biweekly injections. Not surprisingly, her PsA

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began to flare and her fatigue intensified. There were some mornings, JT told me, where she was feeling so down that she couldn't even get motivated to get out of bed.

These are the times that challenge our nursing skills. We all know how important it is for our patients to remain adherent to their medication regimens, but if we don't take the time necessary to address the underlying reasons, their overall health and quality of life is at risk. JT was clearly at a tipping point if this visit was mishandled.

I patiently listened to JT as she told me about what had happened in the 6 months since her last visit. I did not want her to feel like she was an outlier with unusual feelings toward her medication and explained that many patients are "needle-phobic" and often forget to take their medications due to their busy lives. I also again sensed an underlying depression and used a screening tool to ask a few questions. Her scores suggested she would benefit from a psychological evaluation, and so I suggested several possible avenues of help such as cognitive behavioral therapy and medication. Together, we looked up some nearby psychologists included within her healthcare plan and found one who was convenient for her to visit. In addition, we discussed some new medication options for her PsA since JT was not interested in restarting with the self-injections.

After a discussion with her gastroenterologist, we decided the best option would be an infusible biologic—we chose infliximab, which also has a dual PsA/Crohn's indication—so that JT would not have to worry about self-injections and could be reminded by the infusion suite staff of scheduled appointments. Prior to her initial infusion, JT was introduced to the nurses and support staff in our infusion center to help ensure that she was comfortable with the new members of her treatment team. We set up periodic calls between appointments from the nursing team in the infusion clinic to make sure we stayed abreast of any changes in JT's mood or disease severity.

Six months and several rounds of treatment later, JT seems to be dramatically improved. She is adherent to the infusion schedule and is pleased with the regular support she is receiving from the nursing team. JT has even found a support group of sorts made up of other patients within the infusion center being treated at the same time as her. Even better, she is happy with her new therapist and responding to a newly prescribed antidepressant. JT now seems motivated to take better care of her overall health, losing 15 pounds thanks to help from a nutritionist and a regular workout schedule.

It is common when treating patients with rheumatic disease that we need to look beyond their actual diagnosed condition to also manage their comorbidities and underlying circumstances that prevent adherence to treatment. Only after we were able to help JT address her medication concerns and depression was she able to benefit from her treatment plan and begin to address other comorbidities. These comorbidities do not have to all be managed on our end—as in JT's case, several additional specialists have been brought in to help—but we often need to be the ones to help our patients identify the help they need to maximize the effectiveness of treatment and improve their quality of life.

References

1. Lewinson RT, Vallerand IA, Lowerison MW, et al. Depression Is associated with an increased risk of psoriatic arthritis among patients with psoriasis: a population-based study. *J Invest Dermatol.* 2017;137(4):828-835.

2. Bandinelli F, Prignano F, Bonciani D, et al. Clinical and demographic factors influence on anxiety and depression in early psoriatic arthritis (ePsA). *Clin Exp Rheumatol.* 2013;31(2):318-9.

Opening Our Eyes to the Side Effects of Biologic Therapy

by Amanda Mixon, PA-C

Thanks to a number of recent new introductions, we have more weapons than ever in our arsenal to combat psoriatic arthritis (PsA). One drug or drug combination doesn't work? Try something else. And then something else. And so on.

But it's not always lack of efficacy that stops us in our tracks when treating patients with PsA. Insidious and serious side effects can often waylay patients who are doing wonderfully on drug therapy and require rapid and sometimes drastic adjustments.

I started seeing KV, a 30-year-old female, approximately 3 years ago after her previous rheumatologist retired. Diagnosed with psoriatic arthritis (PsA) at the age of 24, KV's primary complaints when we first met were uncontrolled psoriasis and ongoing joint pain, especially in her back and knees. She also had considerable swelling in both knees.

KV expressed frustration with her joint pain but was even more sensitive to her severe psoriasis. She told me that she rarely went out during the summer months because she did not want anyone to think she was "contagious." As with a number of my PsA patients with severe psoriasis, KV showed clear signs of depression and so, based on her presentation, I knew that we needed to approach the treatment of her disease aggressively.

KV's medical history was notable for an intolerance to methotrexate as well as hydroxychloroquine, which exacerbated her psoriasis (a well-known medication side effect).[1] When I first met KV, she was being managed on prednisone 10 mg/d, which she had previously tried to taper down without success. The prednisone was likely the cause of KV's unwanted weight gain, another trigger of her depression.

We first tried sulfasalazine, which was only minimally effective, before moving onto adalimumab. The biologic led to rapid improvements in KV's symptoms, including near-complete resolution of her psoriasis, and allowed her to taper off of prednisone entirely.

There were several additional visible signs of improvement. A former competitive long-distance runner, KV had had to cut back on her training during the worst days of her PsA, but with clearance of her psoriasis, she was able to pick up running again. Her symptoms of depression improved, and she excitedly told me about her plans to get married in Mexico without fear or hesitation about how she looked.

Things continued smoothly for about a year until KV showed up in my office a few weeks after developing a high fever (~103 degrees F), axillary and cervical lymphadenopathy, and a rash on her legs that was inconsistent with psoriasis. She was also quite fatigued to the point where she was unable to work.

My initial concern was that this was either an infection, an underlying malignancy, or possibly drug-induced lupus. To help make the determination, I ordered extensive lab studies as well as a chest X-ray.

KV's lab report showed highly positive antinuclear antibody titers, dsDNA antibodies, and anti-Histone antibodies. Her compliment levels were low while markers of inflammation were significantly elevated. Based on the lab report and X-rays, I was able to rule out infection and malignancies as the cause of KV's current issues, leaving drug-induced lupus as the most likely diagnosis. A dermatology referral confirmed that her new rash was consistent with systemic lupus erythematosus (SLE).

Drug-induced lupus is a rare but known side effect associated with use of anti-TNFs. According to published studies, there are 15,000 to 30,000 new cases of drug-induced lupus each year in

"...it's always important to remember that serious medicines like biologics are often accompanied by potentially serious side effects."

the United States. Approximately 2 of every 1,000 patients treated with an anti-TNF will develop drug-induced lupus. Some patients will go on to develop clinical symptoms such as rash, fever, serositis, and arthritis. Without prompt diagnosis and treatment, these clinical symptoms can be severe and irreversible.[2] The mainstay in treatment of drug-induced lupus is to halt use of the offending drug. NSAIDs and/or corticosteroids can be used to speed the healing process, which often takes days or weeks.

Consequently, my first step after diagnosing KV with drug-induced lupus was to take her off of adalimumab. I initiated high-dose prednisone, which worked quickly to alleviate most of KV's pain and fatigue and helped to normalize her labs. Once her symptoms stabilized, I tapered the dose of prednisone back to 5 mg/d, knowing that many of KV's initial symptoms were likely to return, which they did. Her psoriasis resurfaced and her joint pain returned, which of course alarmed KV, especially with her wedding coming up in a few weeks.

Not wanting to wait, we agreed on a trial of ustekinumab due to its unique mechanism of action as an interleukin-12/23 inhibitor. Within 4 weeks, KV's psoriasis and joint pain resolved. It's now been approximately 3 months since KV started on ustekinumab, and her psoriasis and arthritis symptoms remain quiescent. At her most recent visit, KV brought pictures from her recent wedding, where she was a beautiful, psoriasis-clear bride.

Unquestionably, we are fortunate to have so many options for the treatment of PsA that prevent our patients from becoming deformed and potentially disabled. However, it's always important to remember that serious medicines like biologics are often accompanied by potentially serious side effects. As healthcare providers, we need to be vigilant in staying up to date on these possible side effects so that they can be identified and treated quickly and appropriately.

References

1. Herman SM, Shin MH, Holbrook A, Rosenthal D (2006) The role of antimalarials in the exacerbation of psoriasis: a systematic review. *Am J Clin Dermatol.* 2006;7:249-257.
2. Borchers AT, Keen CL, Gershwin ME. Drug-induced lupus. *Ann N Y Acad Sci.* 2007;1108:166-82.

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Her current professional focus is in the treatment of inflammatory arthropathies such as psoriatic arthritis, rheumatoid arthritis, and ankylosing spondylitis. She also has an interest in systemic scleroderma and lupus.

SAYING IT OVER & OVER & OVER

by Monica Richey, NP, MSN, ANP-CP/GNP

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Ever had a patient who wouldn't listen to your recommendations (of course you have)? Has is happened to you this month? This week? Today?

The uncooperative patient is a challenge for all of us. Patients, of course, have the right to disagree with our recommendations, but that doesn't mean it's easy to have the suggestion door slammed in our face over and over again. Nonetheless, it's important to always keep trying as you never know when that breakthrough will happen.

When I met AL, a 32-year-old female, she weighed close to 300 pounds and had a very difficult personality, to say the least. Her primary complaint involved persistent left knee pain, but it quickly became very clear that there were a number of different issues at play.

It took 2 nurses and 3 physicians to convince AL that she needed to put on a gown so that we could look at her knee (there's the difficult personality)—AL finally conceded after we agreed that she could leave her long sleeve t-shirt on.

On examination, AL had so many psoriatic plaques on her left leg that it was hard to imagine it was limited to that one location, although she refused to allow us to conduct a more comprehensive examination of her other extremities. Overall, AL presented with many of the classic signs of psoriatic arthritis (PsA), including oligoarthritis, presence of psoriasis, and a negative workup for rheumatoid arthritis. Her left knee was red and extremely swollen, and we gave her a corticosteroid injection to help with immediate pain control.

Depending on the disease definition, the prevalence of PsA ranges from 6 to 25 cases per 10,000 individuals in the United States.[1] Previously thought to be somewhat rare—even in patients diagnosed with psoriasis—recent studies based on Classification of Psoriatic Arthritis (CASPAR) criteria have found that PsA occurs in up to 30% of psoriasis patients.[2]

While every sign pointed a diagnosis of PsA with AL, we were continually frustrated with her lack of cooperation. It wasn't until her fifth appointment that she finally let one of our rheumatologists complete a full physical examination. He found psoriatic plaques everywhere on her body, including her scalp, ears, buttocks, legs, arms, abdomen, and back.

It was clear that AL was embarrassed by her physical appearance. She told us that she rarely left the house and when she did, she

would cover herself no matter the weather so that people would not stare at her. While she used to swim regularly, AL said she had not been to a pool in many years and had gained more than 100 pounds in the last decade due to lack of exercise and a poor diet (she often overate to handle her severe depression). AL had stopped visiting friends and family and wouldn't even leave the house to buy groceries, instead having them delivered on a weekly basis. Our social worker administered a Patient Health Questionnaire (PHQ-9) and AL scored 18, an extremely high result indicative of severe depression. Unfortunately, AL refused a referral to a mental health specialist.

Depression among patients with psoriasis and PsA is common. A 2017 study of more than 1,000 patients with psoriasis and PsA showed that 64.1% and 28.4% of patients self-reported mild or moderate depressive symptoms, respectively. The frequency of participants with at least moderate depressive symptoms increased with greater psoriasis severity, from mild (23.7%) to moderate (27.5%) to severe (43.7%) psoriasis. Patients with both psoriasis and PsA were most likely to report moderate-to-severe depressive symptoms.[3]

AL continued to impede our best efforts at every turn. We suggested to her that a biologic might help improve her symptoms, but she would only agree to topical steroids, which did little to help. It took a full year to finally break down the wall and finally get AL to try something that might be more effective. We initially considered methotrexate, but when AL reported to us that she drank 3-4 beers daily (another way to deal with her depression), we ruled that out.

We felt that adalimumab was the next best option, even though AL expressed a fear of using needles. We had AL come into the office every 2 weeks for 2 months so that we could oversee the injections until she became comfortable enough to inject herself at home.

The biologic was immediately effective in improving AL's symptoms and reducing the spread of her psoriatic plaques. Approximately 4 months after she began adalimumab, AL showed up in our office after buying a swimsuit. She told us that she was finally ready to go back to the pool and was no longer embarrassed to be seen in public.

AL remained on adalimumab for nearly 5 years before developing resistance to the medication, at which time she was switched to apremilast. She responded nicely to that as well. Over the course of several years, AL lost about 30 pounds and continued to swim 3 times a week. She eventually agreed to see a mental health specialist and cut down significantly on her alcohol intake.

Unfortunately, AL eventually suffered a major stroke, perhaps due to the combination of obesity and hypertension. She was in a coma for nearly 2 months and lost both the ability to speak and movement over the right side of her body.

Two years later, AL now lives in a nursing home and comes to see us every 3 months. She is confined to a wheelchair, although she eventually regained her voice and remains in positive spirits.

AL is a constant reminder to me that it's never a waste of our time to sound like a broken record in front of our patients. You never know when they'll be ready to finally hear what you have to say. AL was frustrating for the providers in our practice for so many reasons, but she's managed to hang with us for more than a decade. Her life isn't a storybook tale, but few of our patients' journeys are. It's those incremental improvements that we need to find where and when we can.

References

1. Rachakonda TD, Schupp CW, Armstrong AW. Psoriasis prevalence among adults in the United States. *J Am Acad Dermatol.* 2014;70(3):512-516.
2. Ritchlin CT, Colbert RA, Gladman DD. Psoriatic Arthritis. *N Engl J Med.* 2017;376(10):957-970.
3. Armstrong AW, Edson-Heredia E, Zhu B, et al. Burden of psoriasis as reported in a cross-sectional study: association of psoriasis severity with health-related quality of life, depression, and work productivity. *J Psoriasis Psoriatic Arthritis.* 2017;2(4):106-112.

MY MOST *Memorable* PATIENT

by Linda Grinnell-Merrick, MS, NP-BC

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After more than 30 years as a healthcare provider, you'd think that writing about my most memorable patient would be easy. It's not. I have met and grown close to so many patients and their families over the years that I could pick out dozens of patients who had a lasting impact on my professional and personal life.

My nursing career started in chronic specialty care, working with patients who had end-stage renal failure and required dialysis. From there, I shifted to solid organ transplant before finally landing in rheumatology more than a decade ago. Yes, it seems that chronic disease has followed me from place to place, and I've always enjoyed the relationships that evolved over time when dealing with patients over many months and years. In fact, it was thanks to one of my patients that I met my current husband.

Still, there is one patient who has always held a special place in my heart—JD. He was a larger-than-life personality both in stature (he was 6-foot-3 and about 250 pounds) and personality. A local sheriff still working in law enforcement when I first met him in his early 50s, JD was a diabetic who went on to develop diabetic nephropathy and ultimately required dialysis. When I first met JD, he was just starting on hemodialysis, about which he constantly grumbled since it interrupted his independence and was a burden on his time schedule.

JD was extremely active, with a cottage he loved in upstate New York, a husband and father of grown children, and a full-time job. I met JD as my department was opening our peritoneal dialysis unit. Because it would offer him more independence and not take him away from his work or home life as much, JD was very interested in our new unit and became one of our first patients.

Our relationship developed over time as JD was trained on in-home dialysis. He would come into our office for periodic blood sugar checks and, while perhaps clichéd for someone in law enforcement (yet nonetheless true in this case), he would bring in coffee and donuts whenever he had an appointment. JD frequently shared stories about his work and family with me, and I learned a lot about his cottage, his boat, and his fishing tales.

A few years after we first met, I was faced with a personnel challenge of my own—my marriage was in crisis. While I didn't volunteer any information with my patients, there were many who were perceptive and could sense that there was something wrong with me. JD was one of those patients. Eventually, I opened up to him about my issues, and he shared with me the challenges he faced when his first marriage fell apart. He provided strength and re-assurance that both I and my two children would be OK.

During these months, JD came in more frequently than he needed to from a medical perspective to check on me and make sure I was OK. He was providing me with as much care as I was providing to him. As anyone who has gone through a divorce knows, there is always that initial worry of "things will never get better." JD assured me that that

was nonsense, and as new opportunities indeed did eventually open up for me with my new husband and our combined families, JD was always eager to say, "I told you so."

From a health perspective, JD's condition unfortunately deteriorated over the years. His diabetes grew progressively worse, and he eventually required amputation of his lower right leg due to a wound infection. JD was no longer able to easily get in and out of his beloved boat, so he bought a new boat and sold the old one to my new husband and I!

Not long after this, the hospital I was working at merged with another local facility, and the job cuts were expected to be significant. I decided to take a job at another nearby hospital before my position was phased out. Of course, saying goodbye to my beloved co-workers and patients was difficult.

JD and I continued, however, to cross paths. While on weekend call in my new role as a solid organ transplant nurse, I learned that a kidney had become available. JD was on the wait list for a new kidney at the time, and though I struggled to track him down (he was vacationing in a mobile home with his wife out of state), he quickly returned for the transplant when I finally reached him. Unfortunately, JD's body rejected the new organ, and he remained on dialysis.

Over the years, JD's health continued to decline as his heart weakened. One night while on call, I was asked to come in to drain the dialysate (peritoneal dialysis fluid) off a patient who was in cardiac arrest. It was JD. When I arrived, he was surrounded by his family while heading down for cardiac catherization.

He was not conscious. Having gotten to know them over the many years that he was my patient, JD's family was so happy to see me and to know I would be going down to the cardiac catheter lab with JD.

JD coded several times during the procedure. I begged for him to hang in there, but eventually the procedure was abandoned. JD's family was told that there was nothing more that could be done, and the decision was made to take JD off of life support. The family asked me to stay with them and JD during these final moments. It was simultaneously beautiful and tragic; I was honored to be asked to stay with JD and his family during this end-of-life experience.

To this day, our family still owns the boat that JD sold us those many years ago. Every time I hear the engine turn over, I get a click in my brain reminding me to hold every moment precious and to live life to the fullest. As nurses, we are there to serve our patients, but there are times when the script is flipped and they support us too. Even the most difficult experiences in life can bring about unforeseen opportunities.

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