RHEUMATOLOGY NURSE
PRACTICE
Accredited education for registered nurses and advanced practice providers

MANAGING COMMON COMORBIDITIES OF

PSORIATIC ARTHRITIS

Inside this Issue

VOLUME **5** / ISSUE **5**

- *What are the key comorbidities to be concerned about in patients diagnosed with psoriatic arthritis (PsA)?*
- *Why are patients with PsA at higher risk than the general population for developing cardiovascular disease, Type 2 diabetes mellitus, and other comorbidities?*
- *Which specific comorbidities may disqualify the use of commonly prescribed medications used in the treatment of PsA?*
- *What other medical specialties should be incorporated into the care team for specific PsA comorbidities?*

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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 will explore the prevalence of comorbidities in patients with psoriatic arthritis, review some of the key screening components of which clinicians should be mindful, and discuss treatment considerations in the presence of some of the more common and significant comorbidities.

LEARNING OBJECTIVES

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

- Identify the most common comorbidities among patients diagnosed with psoriatic arthritis (PsA)
- Analyze the clinical impact of expert guidance regarding the screening and treatment of these comorbidities
- Discuss the importance of mental health on overall patient outcomes in PsA
- Develop plans to incorporate or enhance screening practices for comorbidities among your patients with PsA

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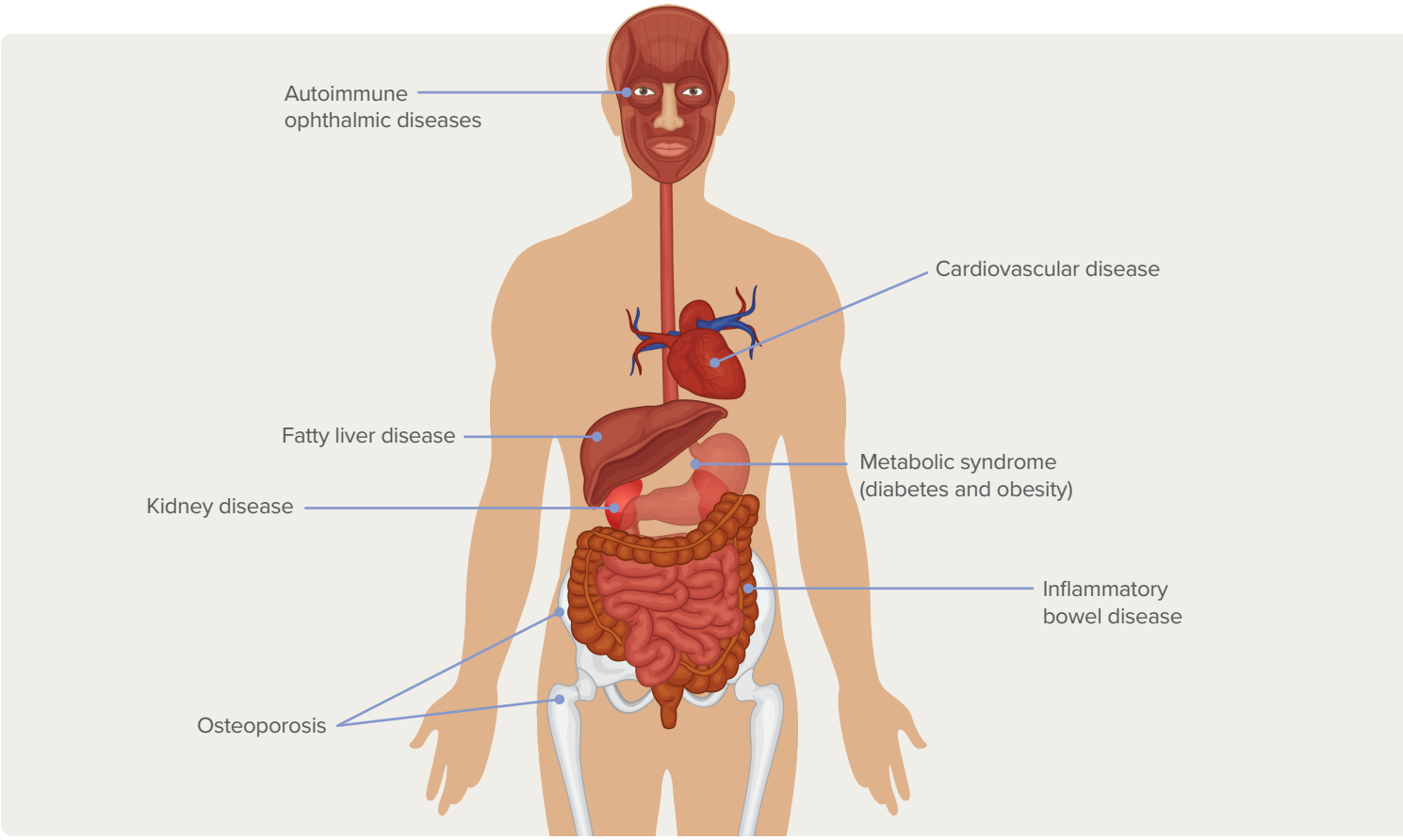
MANAGING COMMON COMORBIDITIES OF

PSORIATIC ARTHRITIS

Psoriatic arthritis (PsA) is an inflammatory musculoskeletal disease associated with psoriasis that involves six primary clinical domains: peripheral arthritis, axial disease, enthesitis, dactylitis, skin disease, and nail disease. As a systemic condition, PsA is linked to a range of potential comorbidities, including cardiovascular disease (CVD), metabolic syndrome, diabetes, depression, and inflammatory bowel disease (IBD) (Figure 1). More than 50% of PsA patients have at least one comorbidity and as many as 40% may have three or more.[1] This medical burden not only affects activities of daily living and quality of life but also impairs function and influences selection of therapy.[1]

Figure 1
Comorbidities Associated with PsA[1]



Considerations in Screening for and Managing Comorbidities

Screening for and recognizing comorbidities among patients with PsA is important to support comprehensive patient evaluation and management (Table 1). The presence of, or risk for, comorbidities also has a bearing on PsA treatment selection, although the efficacy evidence base for specific comorbidities is generally insubstantial. Clinicians should take patient preferences into consideration when selecting therapy for patients with active PsA and comorbidities. A Patient Panel with input into 2018 American College of Rheumatology/National Psoriasis Foundation (ACR/NPF) guideline recommendations for treating PsA emphasized the importance of selecting effective therapies that prevent further joint damage and improve quality of life, social participation, and function. The Patient Panel also advocated for therapies with limited adverse event profiles so as to avoid adverse events that have a negative impact on quality of life and social participation, such as fatigue and nausea.[2] Additionally, although a treat-to-target approach is increasingly recommended when considering therapy to improve patient outcomes, the ACR/NPF Patient Panel expressed concern that treat-to-target could increase patient costs and adverse events. As a result of this concern, 2018 ACR/NPF guidelines do not recommend specific targets in the context of therapy selection for PsA patients with comorbidities. Another expert consortium, the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA), published guidelines that recommend selecting therapies that target as many domains as possible for PsA patients with comorbidities, frequent evaluation of progress, and therapy modification as required.[3]

Polypharmacy in the setting of PsA comorbidities is an additional issue of which clinicians should be aware. Although not yet fully studied in PsA, emerging population-based data point to a high prevalence of polypharmacy and comedication in patients with psoriasis and PsA, including use of analgesics, drugs targeting osteoporosis, anti-inflammatory ophthalmic agents, anti-migraine medications, antihistamines, psychiatric therapies, antibiotics, vasodilators, and anti-arrhythmic therapies.[4]

Chronic viral infections also pose complications that can affect therapy selection in PsA. Viral reactivation is common in rheumatic disease patients with chronic hepatitis B virus (HBV) infection who are being treated with biologic therapies and should be considered as a possibility by clinicians treating patients with PsA.[5]

Table 1 *Screening for PsA Comorbidities*[1,17,64]

Screening for PsA Comorbidities	
CVD	• Annual screening: blood pressure, lipid panel, smoking cessation counseling • CVD risk assessment at least once every 5 years and following major changes in anti-inflammatory therapy • Consider screening for asymptomatic atherosclerosis (e.g., carotid ultrasound) • Lifestyle modifications (diet, exercise, smoking cessation) • Cautious use of NSAIDs and corticosteroids • Advise patients on increased CV risk in PsA
Diabetes	• Check fasting glucose or hemoglobin A1c at least annually or if hyperglycemia symptoms occur
IBD	• Ask about bowel movements, diarrhea during review of symptoms • Refer to gastroenterologist in presence of IBD symptoms (e.g., hematochezia, chronic diarrhea)
Obesity	• Measure weight • Calculate BMI • Counsel patients on benefits of weight reduction for joint and CV health
Ophthalmic disease	• Ask about eye symptoms (e.g. dryness, redness, pain, vision loss) during review of symptoms • Refer to ophthalmologist for evaluation in presence of symptoms
Malignancy	• Screen as in general population • Annual skin check in patients with history of UV light therapy
Liver/kidney disease	• Check LFTs, Cr prior to therapy initiation • Check HBV/HCV serologies • Consider abdominal ultrasound in patients with diabetes or features of metabolic syndrome
Depression/ anxiety	• Ask about symptoms of depression and anxiety during review of symptoms • Refer to psychiatrist/therapist in presence of symptoms
Serious infections	• Consider screening for HBV, HCV, HIV, and tuberculosis according to local guidelines

CVD = cardiovascular disease; IBD = inflammatory bowel disease; LFTs = liver function tests; Cr = creatinine; HBV/HCV = hepatitis B virus/hepatitis C virus

Comorbidities in PsA: Prevalence, Screening, and Treatment

Cardiovascular Disease

Prevalence

In the last decade, it has become increasingly apparent that, as in rheumatoid arthritis (RA), patients with PsA have a significantly increased risk for the development of cardiovascular disease (CVD). This includes ischemic heart disease, congestive heart failure, cerebrovascular accident, and peripheral vascular disease.[6,7] CVD is among the leading causes of death among patients with PsA.[7] It is estimated that, compared with the general population, PsA patients have a 43% increased risk of having a cardiovascular (CV) event.[8] In one large population-based study in the United Kingdom, patients with PsA (n=8,706) who had not received disease-modifying anti-rheumatic drugs (DMARDs) had a significantly higher risk for major adverse CV events (cardiovascular death, myocardial infarction, and stroke) after adjustment for traditional cardiovascular risk factors compared with a control group.[9] Although the evidence for excess CV-related mortality is mixed, overall data appear to indicate that PsA is associated with increased CV-related mortality compared with the general population, and that increased CV-related mortality is associated with disease severity (ie, patients with higher levels of PsA disease activity are at higher risk of CV-related mortality).[7] Several factors contribute to increased CV risk in PsA that we'll detail in the next section.

Inflammatory Processes in PsA and Atherosclerosis

The atherosclerotic process and rheumatic diseases such as PsA share interrelated inflammatory mechanisms.[10] Monocytes, CD4 T lymphocytes, and proinflammatory cytokines that play a role in the pathology of PsA and other arthritides—including tumor necrosis factor (TNF) alpha, interleukin (IL)-1b, IL-6, and IL-18—are also involved in inducing or accelerating atherosclerosis.[11] For instance, the inflammatory burden of consistently elevated proinflammatory cytokines and immune cells in PsA can lead to insulin resistance, endothelial dysfunction, and atherosclerotic plaque formation (the so-called "psoriatic march").[7] At the same time, lipids that are active in host defense and tissue repair have been shown to modulate inflammation. High-density lipoprotein-cholesterol (HDL-C) in particular, which protects against atherosclerosis, also has an anti-inflammatory effect. However, lipids are also mediated by cytokines, and their anti-inflammatory mechanisms are disrupted in the presence of chronic inflammation, as in PsA. This disruption potentially contributes to subclinical atherosclerosis.[11] Dyslipidemia appears to be more prominent in PsA patients with active disease, which further points to a relationship between the degree of inflammation and lipid profile.

Traditional and Subclinical CV Risk Factors

Many studies report increased prevalence of traditional CV risk factors such as hypertension, hyperlipidemia, obesity, diabetes, and dyslipidemia among PsA patients compared with both the general population and patients with RA.[12,13] The prevalence of hypertension in PsA patients has been estimated at 37%,[14] compared with 29% in the general population.[15] Even after adjustment for antihypertensive therapy, hypertension is more prevalent in the PsA population compared with patients without PsA.[7,16] Lipid abnormalities such as low HDL-C and high triglycerides are also common in PsA patients, especially among those with active disease.[7] The presence of these risk factors increases the risk for major CV events such as myocardial infarction, stroke, and CV death.[9] It is important to note that these traditional risk factors are absent in between 30-50% of patients with atherosclerosis, further pointing to the potential role of chronic inflammation as an independent factor in elevating CV risk.[1] Studies have shown increased frequency of subclinical atherosclerosis in PsA patients without clinical evidence of CVD or presence of traditional CV risk factors.[11] Moreover, surrogate markers of subclinical atherosclerosis such as apoprotein B, C-reactive protein (CRP), arterial stiffness, and carotid intima media thickness are more prominent in PsA patients than in patients without PsA, and confer an unfavorable increase in CV risk.[7]

Screening for CV Risk

CV risk in patients with PsA is high, with systemic inflammation operating as an independent CV risk factor. However, CV risk factors are largely underdiagnosed and poorly controlled in this patient population.[12] As with RA, the increased CV risk in patients with PsA warrants multidisciplinary CV risk screening, evaluation, and management that is coordinated between primary care, rheumatology, and cardiology. Recent European League Against Rheumatism (EULAR) guidelines for CV risk management recommend that rheumatologists determine the CV risk profile for individual patients and discuss strategies for addressing modifiable risk factors as part of routine health maintenance.[17] Principles of primary CVD prevention established by the American College of Cardiology/American Heart Association and the European Society for Cardiology include using risk estimators to assess CV event risk; initiating lifestyle modifications that include diet and exercise with or without pharmacotherapy to control blood pressure, cholesterol and hyperglycemia; and advising patients about smoking cessation.[18,19] It is important to note that CV risk assessment scores do not account for the role of inflammation in CV risk calculation; therefore, EULAR guidelines recommend applying a multiplier of 1.5 to

Table 2 *Treatment Selection in the Presence of Comorbidities*[3,5,65-67]

Comorbidity	NSAIDs	Glucocorticoids	Hydroxychloroquine	Sulfasalazine	Methotrexate	Leflunomide	Cyclosporine	Etanercept	Adalimumab	Infliximab	Certolizumab	Golimumab	Ustekinumab	Apremilast	Secukinumab	Tofacitinib	Guselkumab
Cardiovascular Disease	C	ID	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	C	NI
Obesity	NI	NI	NI	NI	C	NI	NI	NI	NI	NI	NI	NI	NI	NI	E	M	NI
Metabolic Syndrome	NI	C	NI	NI	C	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	M	NI
Diabetes	NI	C	NI	NI	C	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	M	NI
Ulcerative colitis	ID	NI	NI	A/P	NI	NI	OL	NI	A/P	A/P	NI	A/P	A/P	NI	C	A/P	E
Crohn's disease	ID	NI	NI	A/P	OL	NI	NI	NI	A/P	A/P	A/P	NI	A/P	NI	C	NI	NI
Uveitis	NI	A/P	NI	NI	NI	NI	NI	ID	A/P	A/P	NI	NI	NI	NI	NI	NI	NI
Osteoporosis	ID	C	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI
Malignancy	NI	NI	NI	NI	NI	NI	NI	C	C	C	C	C	ID	NI	NI	M	NI
Fatty liver disease	C	NI	NI	C	C	C	NI	NI	NI	NI	NI	NI	NI	NI	NI	M	NI
Chronic HBV	C	NI	NI	NI	C	C	NI	A/P	M	M	M	M	ID	NI	NI	M	NI
Chronic HCV	C	NI	NI	NI	C	C	NI	ID	ID	ID	ID	ID	ID	NI	NI	M	NI
Chronic kidney disease	C	NI	NI	NI	C	ID	M	NI	NI	NI	NI	NI	NI	NI	NI	NI	NI
Depression	ID	ID	ID	ID	ID	ID	ID	ID	ID	ID	ID	ID	ID	C	C	C	NI

C	Reason for caution
ID	Insufficient data but concerns raised
NI	No information available
A/P	Approved or preferred therapy
OL	Used off-label
M	Special monitoring required
E	Emerging efficacy data

NSAIDs = nonsteroidal anti-inflammatory drugs

the CV risk calculation for all patients with rheumatic disease.[17]

In addition, psoriasis is a risk enhancer for CVD risk. The severity of skin lesions in PsA as determined by use of the Psoriasis Area and Severity Index score has been shown to have predictive value in identifying CV risk.[9]

PsA Therapy Selection and CV Risk

The effect of antirheumatic therapies, including DMARDs and anti-TNF agents, on CV risk factors is largely unknown, although it is hypothesized that controlling inflammation via pharmacotherapy will reduce CV risk in patients with PsA (Table 2). For instance, some data suggest that anti-TNF therapy may reduce the progression of surrogate markers of subclinical atherosclerosis such as aortic stiffness, C-reactive protein, and carotid intima media thickness.[20-22] Small case-control studies have shown short-term improvement in these surrogate markers with the administration of infliximab or etanercept.[7] Additionally, carotid plaque, obesity, and hepatic steatosis are considered negative predictors of achieving and maintaining MDA.[23] In the absence of therapy-specific evidence to guide CV risk attenuation in PsA, EULAR guidelines recommend reducing and maintaining low PsA disease activity to minimize CV risk and suggest following CVD treatment guidelines developed for the general population. EULAR guidelines recommend the use of TNF inhibitors after the failure of synthetic DMARDs, although anti-TNFs should be avoided in patients with New York Heart Association class III or IV heart failure.[3,11] At the same time, some anti-inflammatory medications may increase CV risk in PsA patients. Notably, glucocorticoids

are associated with the potential for hypertension, hyperglycemia, and dyslipidemia, while non-steroidal anti-inflammatory drugs (NSAIDs) are known culprits for hypertension, may promote thrombosis,[6] and increase the risk of coronary artery disease.[24] MTX can increase risk for atherosclerosis by affecting homocysteine levels.[6] Antihypertensive and statin therapies can be used in primary CVD prevention as in the general population.

Diabetes, Obesity, and Metabolic Syndrome

Prevalence

Although the relationship between insulin resistance and rheumatic diseases is supported by data from several studies, there are fewer studies on the relationship between rheumatic diseases and type 2 diabetes mellitus (T2DM). Nonetheless, available data from cohort and insurance database studies indicate that patients with PsA have a 33-45% increased risk for T2DM compared with both the general population and patients with RA.[12,16,25] One cohort study in the United Kingdom involving adult patients with RA (n=53,215) and PsA (n=12,548) reported T2DM prevalence in 16.6%, 14.3%, and 11.1% of PsA, RA, and control patients, respectively.[12,25] There are also signals that the risk of developing T2DM in PsA is higher for patients with severe skin disease.[26] Causative mechanisms for T2DM in PsA are unclear, but the high risk of T2DM in the PsA population is likely elevated by high inflammatory load from both skin and joint disease, which is, in turn, implicated in insulin resistance.[25] Differences in body composition or fat mass in the PsA population are also thought to contribute to T2DM.[13]

The high prevalence of T2DM in patients with PsA is associated with and attenuated by obesity and lifestyle factors.[25] Most patients with PsA are overweight (body mass index [BMI] >25 kg/m^2) or obese (BMI >30 kg/m^2). Clinical studies also show higher prevalence for obesity in patients with PsA compared with both the general population (37% vs 18%) and patients with RA (27%) or psoriasis (29%).[27]

As with diabetes and CV risk, obesity is associated with higher disease activity in PsA.[11] BMI, which is often higher in PsA patients than in both RA and the general population (29.6 kg/m^2 vs. 27.3 kg/m^2 vs. 26.1 kg/m^2, respectively), is also thought to be a factor.[27] A tight interrelation between obesity and PsA is suspected by which fat mass increases proinflammatory status via abnormal expression of adipokines such as TNF alpha, IL-6, and leptin, and stimulates the inflammatory cascade associated with diabetes.[28,29] This inflammatory cytokine cascade also drives insulin resistance (i.e., the reduced uptake of glucose by metabolically active cells when exposed to insulin), which may also be a feature of PsA.[11] A vicious cycle ensues in which obesity in patients with psoriasis may increase the risk for PsA by stimulating inflammation or by increasing joint damage, which in turn leads to more inflammation.[27] Obesity has been shown to reduce the efficacy of PsA therapy, lower the probability of achieving sustained remission, and limit the potential for achieving minimal disease activity (MDA).[28,30] Notably, patients with BMI >30 kg/m^2 have a 50% lower chance of responding to therapy or achieving remission (as measured by MDA) than PsA patients who are neither overweight nor obese.[17,28]

Metabolic syndrome is a systemic, proinflammatory state clustered around abdominal obesity, atherogenic dyslipidemia, hypertension, and insulin resistance. Although there are few studies on the impact of metabolic syndrome on patients with PsA, its prevalence is higher in patients with PsA (38%) than in the general population (25%), RA (20%), or ankylosing spondylitis (AS, 11%).[13,31] There is some evidence that metabolic syndrome may be independently related to underlying disease severity in PsA.[32] Controlling metabolic comorbidities may reduce both disease activity and the burden of CVD in patients with PsA.

Screening for Diabetes and Metabolic Syndrome

Clinicians should check fasting glucose or hemoglobin A1c levels at least annually or when hyperglycemia symptoms occur. They should also monitor patients for the presence of metabolic syndrome, which is diagnosed when at least three of the following five risk factors are present:[32,33]

- Fasting glucose ≥100 mg/dL or receiving pharmacotherapy for hyperglycemia
- Blood pressure ≥130/85 mmHg or receiving pharmacotherapy for hypertension
- Triglycerides >150 mg/dL or receiving pharmacotherapy for hypertriglyceridemia
- HDL-C <40 mg/dL in men or <50 mg/dL in women or receiving pharmacotherapy for reduced HDL-C
- Increased waist circumference with ethnic-specific circumference cut-points (i.e., ≥40 inches [≥102 cm] in Caucasian men/≥35 inches [88 cm] in Caucasian women)

PsA Therapy Selection in T2DM

There are limited data on the effect of antirheumatic therapies on the risk of diabetes in PsA patients. GRAPPA recommends caution concerning the use of glucocorticoids in PsA patients with concomitant T2DM.[3] ACR/NPF guidelines recommend use of an oral small molecule (OSM) other than MTX for treatment-naïve patients with active PsA and diabetes given the higher prevalence of fatty liver disease in this population and liver toxicity with

MTX use.[2] If diabetes is well controlled, a TNF inhibitor can be considered over an OSM.

Liver and Kidney Disease

Prevalence

Clinical studies show that patients with PsA tend to have higher levels of nonalcoholic fatty liver disease, which is, in turn, associated with metabolic syndrome, obesity, and dyslipidemia.[23] Both obesity and fatty liver disease negatively impact therapeutic responses in patients with PsA, and, in particular, reduce the efficacy of TNF inhibitors.[9,34] PsA is also an identified risk factor for renal damage and chronic kidney disease in patients with psoriasis. As in RA, reduced glomerular filtration rate has been identified as an emerging comorbidity in approximately 16% of patients with seronegative status (including PsA and oligoarthritis).[5]

Screening

There is no consensus regarding screening recommendations for nonalcoholic fatty liver disease in high risk populations such as PsA. Abdominal ultrasound and evaluation of liver enzymes are usually recommended in patients >50 years with diabetes and/or features of metabolic syndrome.[8] Creatinine clearance should be measured prior to initiation of therapy.[8]

Therapy Selection

Some anti-inflammatory medications (e.g., MTX and leflunomide) contribute to liver function test abnormalities and development of nonalcoholic steatohepatitis, nonalcoholic fatty liver disease, or cirrhosis, and should be used with caution in patients with liver disease.[8] The presence of fatty liver disease can complicate PsA therapy selection and reduce the likelihood of achieving MDA.[23] MTX should be avoided in patients with renal insufficiency, as renal impairment is a major risk factor for developing MTX toxicity. In addition, NSAIDs should be used with caution in PsA patients with renal damage.[8]

Autoimmune Ophthalmic Disease

Prevalence

The spectrum and prevalence of autoimmune ophthalmic disease is high among patients with PsA. Uveitis is especially common, affecting approximately 25% of patients with PsA compared with 33% of patients with AS; uveitis is often insidious in onset and unilateral in nature.[35] Other common eye conditions associated with PsA include keratonconjunctivitis sicca (dry eye), keratitis, blepharitis, conjunctivitis, episcleritis, and scleritis.[36]

Management Considerations in Obesity

Weight reduction has a substantial effect on response to therapy among patients with PsA since caloric restriction affects a variety of circulating inflammatory markers.[28] A 2014 study randomized PsA patients with obesity who were initiating anti-TNF therapy to either a hypocaloric diet (<1,500 kcal/day) or a free managed diet.[60] While the hypocaloric diet produced significantly greater weight loss than the free managed diet, patients in either arm who lost weight improved their response to therapy. Patients who lost 5-10% of their body weight were 3.75 times as likely to reach levels of minimal disease activity (MDA) compared with patients who lost <5% of their body weight. Patients who lost >10% of their body weight were 6.67 times more likely to reach MDA. More recent research shows that a low-calorie diet also contributes to significant weight reduction. A 2019 study showed that PsA patients treated with 600 calories/day not only lost weight but felt significantly better in terms of patient-reported outcomes and maintained the benefits of weight reduction at 12 months follow-up.[61]

It is recognized that regular physical activity can significantly reduce CV risk; however, the pain and inflammation associated with arthritic joints can lead to reduced physical activity, sedentary lifestyle, and obesity.[27] The role and benefits of physical activity in PsA is relatively understudied. One systematic review reported moderate evidence for the benefits of exercise therapy in spondylarthritis for improving physical function and disease activity, and low evidence for improving pain, stiffness, and cardiorespiratory function, although not CV risk factors.[62]

It is important for clinicians to encourage and support regular movement and physical activity in PsA patients. Clinicians should discuss physical activity goals with PsA patients on at least an annual basis and consider targeted interventions that are designed for patients with joint and functional mobility issues.[63] Although 2018 American College of Rheumatology/National Psoriasis Foundation (ACR/NPF) guidelines do not specify physical activity regimens for patients with comorbidities given the low evidence base for benefits of specific regimens, they do recommend exercise and physical therapy over no exercise for patients with active PsA.[2] Low-impact physical activities such as tai chi, yoga, and swimming are considered beneficial in patients with active PsA.

Screening

Directing questions to patients with PsA about ocular symptoms such as dryness, redness, pain, and vision loss can help to raise patient awareness about the potential for ophthalmic disease. Patients with suggestions of these symptoms should be referred to an ophthalmologist for evaluation.[8]

Therapy Selection

Current data support the use of systemic, periocular, and implantable corticosteroids, MTX, mycophenolate, cyclosporine, azathioprine, and the TNF inhibitors infliximab and adalimumab for patients with uveitis.[5] Local corticosteroid injections are considered options for treating sudden onset of severe, unilateral eye disease; prednisone or systemic immunosuppression are options for bilateral disease.[1] Infliximab, adalimumab, certolizumab, and golimumab have shown effectiveness in treating uveitis, although only adalimumab is approved for treating this condition in patients with PsA.[8] Studies are currently investigating the efficacy of IL-17, IL-12/23, and Janus kinase inhibitors for managing uveitis.

Depression and Anxiety

Prevalence

Patients with PsA are much more likely to be depressed than patients with psoriasis or the general population.[37] Although studies of depression in patients with psoriasis are more common than in PsA, a large prevalence study of 520,000 PsA patients reported that 39% considered PsA a large problem in their daily lives.[38] A study involving a Canadian cohort of patients with PsA reported a 36.6% prevalence for depression and 22.2% for anxiety. In this study, almost 18% of patients with PsA had both depression and anxiety.[39]

The consequences of depression and anxiety include lower health-related quality of life and a high probability of psychosocial problems, including major depressive disorder.[40,41] PsA patients also have a higher risk for attempted suicide compared with the general population.[42]

It remains challenging to decipher the multidimensional causal mechanisms between depression and PsA. The inflammatory responses involved in PsA are implicated in the pathophysiology of depression, as proinflammatory cytokines are raised in depression. Hypothalamic-pituitary-adrenal axis hyperactivity has also been linked to depression in patients with PsA.[41] Patients with PsA also have lower pain thresholds than those without PsA, which correlates inversely with depression.[1] PsA patients with comorbid depression often have low adherence to PsA medications and respond poorly to PsA therapy. For instance, a Danish registry study that included 1,750 patients with PsA reported that patients with depression and/or anxiety showed shorter persistence on TNF inhibitors than those without comorbidities.[43] Other studies show higher discontinuation rates for TNF inhibitors among PsA patients with comorbid depression or anxiety.[41]

> ***"Screening for depression and anxiety in patients with PsA is vital since depression and anxiety may affect pain perception, quality of life, and treatment outcomes.***

Screening

Screening for depression and anxiety in patients with PsA is vital since depression and anxiety may affect pain perception, quality of life, and treatment outcomes. Clinicians should screen for depression and anxiety and refer patients for management of debilitating symptoms. Questions about patient mood or sleep quality during history-taking may indirectly identify indicators for depression that can be further explored via standardized screening tools. There are several comprehensive, validated tools that clinicians can administer to screen PsA patients for dimensions of depression, including the Patient Health Questionnaire 9-item scale, the Hospital Anxiety and Depression Scale, and Becks Depression Inventory. Of note, the Patient Health Questionnaire-2 has been shown to enable rapid and efficient identification of depression and suicidal ideation behavior in patients with psoriasis by assessing anhedonia (diminished ability to experience pleasure) and depressed mood, which are two cardinal features of depression.[44] This tool might also be valuable in screening for depression in patients with PsA.

Therapy Selection

The mitigation of pain and disease activity with biologic DMARDs may reduce depression and anxiety. Additionally, concomitant treatment of depression and anxiety with

antidepressants may improve PsA symptoms. Compared with conventional therapy, biologic therapies are associated with a reduced risk for developing depressive symptoms in patients with psoriasis, including those with PsA.[37] Recent trials indicate that the use of some biologics in psoriasis and PsA (notably, adalimumab, etanercept, and ustekinumab) is associated with reduction in depression symptom scores.[41] Clinicians need to be aware of adverse psychiatric events associated with brodalumab and apremilast. Both of these biologic therapies carry a black box warning against their use in patients with a history of depression and suicidal ideation. These adverse events are not associated with other IL-17 inhibitors, including secukinumab and ixekizumab. Referral to support groups and/or counseling can also provide support to PsA patients being treated for depression.

Inflammatory Bowel Disease

Prevalence

A spectrum of gastrointestinal/inflammatory bowel comorbidities is associated with PsA, including celiac disease, peptic ulcer disease, reflux esophagitis, Crohn's disease, and ulcerative colitis.[45] Data from the Nurses Health Study show that Crohn's disease is more prevalent than ulcerative colitis in patients with both PsA and psoriasis (risk ratio of 6.54 vs. 3.49) and is much higher for PsA patients than for the general population, affecting approximately 10% of PsA patients.[46]

Screening

Screening for IBD involves asking questions about bowel movements and diarrhea. Patients should be referred for a gastroenterology consult in the presence of such symptoms. Fecal calprotein testing is not disease-specific but is sometimes used as a functional quantitative measure of intestinal inflammation.[8]

Therapy Selection

Therapeutic options for managing IBD in PsA are similar to patients without PsA and include aminosalicylates, corticosteroids, metronidazole, MTX and several biologics, including golimumab for ulcerative colitis and certolizumab for Crohn's disease.[1,8] There are few published data to assess the appropriate therapy for concomitant PsA and IBD.[5] Current ACR/NPF guidelines recommend all monoclonal antibody TNF inhibitors over etanercept, which is a fusion molecule/soluble receptor biologic. These guidelines also recommend monoclonal antibody TNF inhibitors over OSM therapies, IL-12/23 inhibitors or IL-17 inhibitors for patients with active PsA and concomitant active IBD.[2] OSM therapies can be considered for patients with severe PsA and IBD who prefer oral therapy or have contraindications to TNF inhibitors. A monoclonal antibody TNF inhibitor or an IL-12/23 inhibitor can be considered for patients with active PsA and IBD who do not respond to an OSM. IL-17 agents (secukinumab, ixekimumab, brodalumab) are associated with IBD exacerbation; therefore, IL-12/23 inhibitors (e.g., ustekinumab) are preferred over these agents in patients with contraindications to TNF inhibitors. NSAIDs may exacerbate IBD symptoms and should be avoided or monitored closely in PsA patients with IBD.

Fatigue, Malignancy, and Osteoporosis

Prevalence

Fatigue is an acknowledged comorbidity in 50% of patients with PsA that contributes to considerable physical and psychosocial burden, undermines quality of life, and exacerbates the symptoms of PsA, including pain.[47,48] Although the mechanisms that cause fatigue in PsA are multidimensional, overproduction of pro-inflammatory cytokines and resultant inflammation are considered a primary causative factor.[49] Fatigue can exacerbate the effects of nonbiologic DMARDs such as methotrexate (MTX) and leflunomide.

The effect of PsA on malignancy is confounded by the use of immunosuppressive and ultraviolet therapies, although it remains unclear whether malignancy incidence in PsA differs from the general population. However, one meta-analysis of patient data from 74 randomized controlled trials (n=22,904) reported that risk for non-melanoma skin cancer was twice as high in patients treated with TNF inhibitors vs. controls.[50] A small number of cancers has also been reported in PsA patients treated with secukinumab and ixekinumab.[51-54]

Chronic inflammation is a known risk factor for bone loss, and osteoporosis is a well-documented comorbidity in patients with RA. Recent reports of osteoporosis in patients with PsA are somewhat conflicting but point to a higher prevalence than previously thought and raise warning signals about which clinicians should be aware, including higher levels of demineralization in PsA patients than in control groups.[1] A cross-sectional study in Brazil involving postmenopausal women showed that while PsA patients did not have lower bone mass density (BMD) than control patients, osteoporotic fractures and metabolic syndrome were more prevalent in women with PsA.[55] Another small study reported that the risk of osteoporosis increased with rising levels of serum osteoprotegrin and a greater number of affected joints.[56] In addition to inflammation, other possible causes of osteoporosis in PsA include joint pain-induced immobility, reduced physical activity, glucocorticoid use, and MTX use.[1]

Screening

Patients with PsA can be screened for cancer according to general population guideline recommendations. Unrecognized, untreated osteoporosis can contribute to fracture and other serious complications. Clinicians should maintain a high index of suspicion for osteopenia and osteoporosis in PsA patients with prolonged and extensive cutaneous disease and a high number of affected joints.[57] PsA patients should be screened for osteoporosis according to recommendations for the general population. Prophylactic therapy with calcium and vitamin D can also be considered.[56]

Therapy Selection

Pharmacologic treatment for fatigue relies on the successful treatment of a patient's underlying PsA; however, while current therapies effectively treat most or all of the current core PsA domains and reduce fatigue, none have been shown to eliminate fatigue.[49] Moreover, clinical trials in PsA are increasingly including patient-reported assessments of fatigue as part of the evaluation of therapeutic benefit to ensure that treatment is effective not only across physical domains but also results in quality-of-life improvements.[58] One recent addition to the PsA core domain set that includes fatigue evaluation in clinical studies endorses this strategy and points to the need for therapies that effectively treat a wider range of PsA domains, including fatigue.[59]

There are few data that examine the impact of osteoporosis medications on PsA disease activity and outcomes. In RA, there is some evidence that low-dose MTX, sulfasalazine, and TNF inhibitors are associated with improvements in bone density.[5] If using glucocorticoids, clinicians should follow ACR recommendations on preventing glucocorticoid-induced osteoporosis and consider concomitant use of a bisphosphonate based on level of risk.[57]

Conclusion

Extra-articular and extracutaneous manifestations of PsA impact patient quality of life and daily activities. The evaluation and management of these heterogenous comorbidities requires multidisciplinary attention by rheumatologists, dermatologists, ophthalmologists, gastroenterologists, psychologists, primary care providers, and other healthcare professionals. Effective treatment of skin and joint disease, as well as management of risk factors and comorbidities, can help to reduce excess mortality and improve quality of life and function in PsA patients. Early evaluation and management of comorbidities in PsA may also, over time, reduce the potential for polypharmacy in this population.

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RNS Chapters

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LOOKING BEYOND Our Patients' Physical Well-Being

by Carrie Beach, BSN, RN-BC

AUTHOR PROFILE:

Carrie Beach, BSN, RN-BC

Carrie Beach BSN, RN-BC, is a rheumatology nurse with the Columbus Arthritis Center in Columbus, OH, and the President-Elect of the Rheumatology Nurses Society

Depression is something we commonly see in many of our patients with rheumatic disease. With experience, the signs become obvious. Disengagement with family and friends, poor adherence to medication, missed appointments, spotty hygiene practices, and more. Given the physical manifestations of psoriasis as well as the pain, fatigue, and joint deformities associated with arthritis, patients with psoriatic arthritis (PsA) are among our most common who deal with depression. According to one recent study, approximately 20% of patients with PsA have comorbid depression.[1]

So then what do we do about this? How do we help our patients whose life is spiraling downward into the depth of depression? Let's take a look at a current patient of mine to hopefully spark some ideas.

When we first met Ken, he was a 54-year-old male who arrived with complaints of joint pain and stiffness along with daily fatigue. While he denied any history of psoriasis or rash, his joint symptoms had gradually gotten worse over the past year. Prior to our appointment with us, Ken had consulted a rheumatologist outside of our practice who ordered labs to be drawn, revealing negative anti-nuclear antibodies and rheumatoid factor. No formal diagnosis was given at that time.

Upon initial exam in our office, Ken had significant synovitis of the 2nd and 3rd metacarpophalangeal joints (MCP) on his right hand. He also had dactylitis of the left great toe and complained of bilateral heel pain, previously diagnosed as plantar fasciitis. X-rays of his hands were normal, although an MRI of the right hand showed tendonitis (but no erosive disease).

Based upon all of this information, we diagnosed Ken with PsA and started him on oral methotrexate (MTX) 12.5 mg weekly.

Ken's medical history included hypertension and cardiovascular disease that led to a cardiac bypass approximately 2 years prior. He was recently divorced and described feeling "very poor as far as mood" and added that he was experiencing "major fatigue."

Until recently, Ken told us he had been very active. He was a certified scuba dive instructor and a world traveler. He worked full time as a produce manager of a local grocery store, a job that he had thoroughly enjoyed for more than three decades. While he was divorced,

“For some patients, like Ken, the pain and fatigue associated with the condition most significantly affects his well-being, while for others, the public embarrassment of psoriasis is far worse.”

Ken said he was interested in dating again, but with the deteriorations in his overall quality of life that caused him to be less active, he was worried that he wouldn’t be seen as a good potential partner. He was unhappy that he was unable to do the things that he had previously enjoyed most in his life. Our hope was that by improving his PsA-related symptoms, it would lift his overall mood and perspective on life as well.

At his 2-month follow-up, Ken said there was some improvement in his joint pain and stiffness, but he pointed out a deformity in one of his fingernails that he was concerned about. We reassured him that nail changes were common in patients with PsA and upped his dose of MTX to 20 mg weekly, with plans to see him back in 1 month.

Unfortunately, in just that short amount of time, things went swiftly downhill. Ken’s joint pain and swelling had both worsened, and he now complained of persistent lower back pain as well. We decided to add infliximab to his daily MTX, and he responded well after only a few infusions.

A few months went by before Ken came back for a routine follow-up with his latest bit of bad news. His disease had again progressed, but now in addition to increasing levels of joint pain and swelling, there was a breakout of psoriasis on several areas of his arms, legs, and trunk. It was a bit confusing for us to see how swiftly things had gone downhill after the initial success Ken had seen with infliximab. But then he told us the probable reason—his work hours had been curtailed and he could no longer afford the infusions. Embarrassed, he didn’t say anything to us or the staff at the infusion center and simply stopped showing up. Our team talked to Ken about the importance of open communication with our practice and reassured him that there would be no judgment of his economic situation. We would do everything we could, we told Ken, to get him back on the right track.

Over the next 15 months, Ken cycled through all of the approved tumor necrosis factor inhibitors—from infliximab to etanercept to adalimumab to golimumab—without much success. This predated the arrival of some of our more novel biologic and small molecule drugs with different targets, so our options were more limited than they would be today. We were unfortunately never able to find another biologic that replicated the initial improvements Ken experienced with infliximab.

As his disease progressed, Ken decided to undergo dual shoulder replacement surgeries. Following the second surgery, Ken’s orthopedic surgeon suggested that he file for disability. Before agreeing to do so, Ken called our office to get our opinion. He told me that really did not want to quit working because he still enjoyed his job but was concerned that he wouldn’t be able to perform his regular duties if he became more significantly impaired physically. We agreed that he should keep working for as long as he felt physically able to do so, as much for his mental well-being as anything. Ken mentioned that there were times he was embarrassed at being in the public eye at work when he had a psoriatic flair that some people, he felt, probably attributed to a hygiene issue as opposed to a medical problem.

We finally began making positive progress again once Ken started on certolizumab. His disease became better controlled, and things were getting better on the personal front as well—Ken remarried and was able to share his passion for scuba diving and traveling with his new wife. He seemed much happier when I saw him for most of his follow-up visits during the next two years.

Then tragedy struck when Ken's son died unexpectedly of a brain aneurysm. Not surprisingly, that tore Ken's world apart. He missed several scheduled appointments, and his disease began to spiral out of control. Both his mental and physical health were deteriorating quickly.

Once Ken finally came back to our practice 6 months ago, we were able to get him back onto infliximab. His out-of-pocket costs for the drug are now much less than they had been when he was first prescribed infliximab. As before, infliximab worked quickly, and Ken's joint pain and swelling have improved. In the meantime, Ken's primary care provider convinced him to start on duloxetine, which has improved both his mood and pain levels. Things are certainly looking up for now, though as with many of our patients, I know how fragile the situation is.

PsA affects our patients in so many different ways. For some patients, like Ken, the pain and fatigue associated with the condition most significantly affects his well-being, while for others, the public embarrassment of psoriasis is far worse. I have had many patients who refuse to wear short-sleeved shirts or shorts when leaving the house for fear of being mocked or questioned. While psoriasis was certainly an issue for Ken, especially as he re-entered the dating world, it was just one piece affecting his health.

It's always important to assess the mental health of our patients to understand how their disease is affecting them. While looking at a swollen joint, watching a patient walk, or seeing psoriatic plaques covering a patient's body are all important parts of our physical assessment, we have to remember to always go beyond those visual observations and ask our patients how they are really handling their disease. With Ken, I know that when he starts a conversation smiling and telling me about his latest dive, that he is doing OK. There were some dark days, especially after the death of his son, when Ken didn't flash his bright smile or talk about an upcoming trip with enthusiasm. As a rheumatology nurse, I feel blessed to have a connection with patients that enables me to recognize the importance of these differences right away. I have known some of my patients for more than 15 years, and I can usually tell within the first couple of minutes of a visit how that patient is really doing. This learned sense of mental acuity is a powerful tool as rheumatology nurses to help judge the mental health of those patients we know best.

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Giving Our Patients Their Lives Back

by Nancy Eisenberger, MSN, APRN, FNP-C

A diagnosis of any rheumatic disease, and especially psoriatic arthritis (PsA), can throw patients' lives off the rails. They are enjoying outdoor activities, time with the family, regular vacations, and then "Wham!" They have to start thinking in advance about every activity they have planned, often deciding whether or not it's worth the risk of another day or week in pain. It can be overwhelming and emotionally exhausting for our patients, and a difficult part of our job rebuilding their health and confidence so that they can once again embrace life instead of fearing it.

Anthony is a recent patient of mine whose life had unfortunately taken a turn for worse before I met him. He is an exquisitely kind gentleman who was diagnosed with PsA in his early 30s. I didn't meet him within our practice until his early 60s. Anthony has been married for over 30 years, had been a successful business owner before recently retiring, a father of 3 daughters, and a grandfather of 2 boys and a girl. When he first came to see us, he told us that he was preparing to marry off his last daughter within a year.

At our initial visit, Anthony demonstrated several of the hallmark features of PsA, including enthesitis, tendinitis, low back pain, nail pitting, and psoriasis of the scalp, elbows, and knees. He also had multiple accompanying comorbidities such as depression, uveitis, elevated liver function (ie, fatty liver disease), hypertension, hyperlipidemia, and cardiovascular disease. Due to his PsA, he had already undergone multiple joint replacement surgeries involving his right shoulder, hip, and knee. Clearly, this was not a patient in the best of health.

When I took over Anthony's care, his list of medications included etanercept 50 mg subQ once a week, prednisone 10 mg a day, prednisolone acetate ophthalmic 1% solution 1-2 drops in the right eye 2-4 times daily, metoprolol 50 mg daily, lisinopril 10 mg daily, atorvastatin 10 mg daily, daily low-dose aspirin, and daily vitamin D3 2000 units.

His history in rheumatology included failed trials of infliximab (infusion reaction), adalimumab (unsafe elevations in liver function), methotrexate (also related to liver function issues), certolizumab (lack of efficacy), and leflunomide (again, liver function issues).

Anthony told me at our initial meeting that he still suffered from flares related to uveitis despite his daily prednisone. He also told me had been taking rescue methylprednisolone dose packs several times a month. Anthony

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was currently taking etanercept as that was the only medication he claimed ever worked to help control his pain, although there was still significant swelling apparent. I aspirated 90 cc of inflammatory fluid from his left knee at this initial visit.

After hearing of his health-related issues, I asked Anthony about his activities of daily living. What did he enjoy about life? Where was he struggling? It's these personal connections that can often serve as the linchpin to building trust with our patients and are crucial to include in initial discussions.

Anthony told me he had not recently been seeing much of his grandchildren, which typically gave him so much joy. He feared that he wouldn't be able to walk his youngest daughter down the aisle at her wedding in a few months, much less join her on the dance floor. He was practically in tears by now. He also told me that he was arguing more and more frequently with his wife because she was "worried about his health," and he didn't want her to get so worked up over him. He added that he had gained 20 pounds in the last 3-4 months and really wasn't motivated to do much of anything.

By now, warning bells were going off in my head. All of these issues were clear signs of depression. I knew from his medical record that Anthony had a history of depression, so it wasn't as delicate a conversation as it might otherwise have been. I suggested to Anthony that he might want to see a local therapist, and he agreed it would be worthwhile. He soon started on duloxetine 30 mg daily, upped within a week to 60 mg. I thought this would help both his mood and pain.

That was one initial hurdle overcome. Now it was time to address his arthritis. As noted earlier, Anthony's disease was very poorly controlled, with multiple areas of enthesitis and tendinitis. His psoriasis, however, was fairly well controlled. Because of his medication history and pattern of repeated biologic failures, it was a tricky call. It was obvious that etanercept wasn't the answer, but what was the best choice now? He had not had any better success with a previous trial of secukinumab, an interleukin-17 inhibitor, than previous tumor necrosis factor (TNF) inhibitors, so I decided to suggest a switch to golimumab, the only TNF inhibitor Anthony had not previously tried. Anthony was skeptical, having been down this road many times where he had gotten his hopes up only to see them quickly dashed. While we waited for his lab work to return, Anthony continued on etanercept but said he would consider a switch to golimumab.

Two weeks later, Anthony was back for his initial follow up. His mood had improved, and he said he recently spent several hours with his grandchildren over the preceding weekend. His lab work showed elevated inflammatory markers and mildly elevated alanine transaminase and aspartate transaminase levels. Hepatitis B/C and QuantiFERON Gold screens were all negative. Anthony received his first golimumab infusion later that day with plans to return for follow-up after his third infusion. He was instructed to call if he had any problems or concerns prior to that scheduled visit.

I heard nothing from Anthony so assumed everything was going well as his appointment date neared. Fortunately, it was. He virtually danced into my office he was so happy. The golimumab infusions and duloxetine had had a rapid, remarkable effect on his physical function and emotional stability. He told me that he was now confident he would be able to dance at his daughter's wedding. Anthony's lab results showed improvement as well, although his inflammatory markers were still mildly elevated. His liver function, however, had normalized. There was mild enthesitis upon physical exam, but this had improved greatly from Anthony's previous visit. Based upon these findings, I added sulfasalazine 500 mg twice daily to his regimen and decreased the prednisone to 5 mg daily, with plans to hopefully increase the sulfasalazine to 1000 mg daily and stop the prednisone entirely. I asked Anthony to get a new set of labs in a month, and every 2-3 months thereafter.

At our next follow-up visit, Anthony was weeks away from his daughter's wedding. Happily, things continued to progress well. His most recent labs were completely normal, and he told me he was pain free. There had been no recent flares of his uveitis. Basically, his life was back on track. He was so grateful that he would be able to give his daughter away at her wedding and celebrate this important family milestone.

Of course, due to the chronicity of PsA, it is likely that we'll need to continue to tweak Anthony's treatment at some point in the future, but it was so rewarding to be able to see him looking forward to one of the big milestones in his life. On the day of his daughter's wedding, while Anthony danced the night away, I lifted a glass from my home as congratulations. For him and for me.

STAYING ON OUR TOES

For Our Patients' Healthy Hearts

by Teri Puhalsky, BSN, RN, CRNI

When I think of cardiovascular risk factors, the usual ones initially come to mind. Things like genetics, unhealthy lifestyle, smoking, obesity, minimal exercise, a low-quality diet, uncontrolled hypertension, and depression.[1] For the patient with psoriasis and psoriatic arthritis, the risk of developing cardiovascular disease is increased due to excess systemic inflammation, something which is central both to psoriatic and cardiovascular disease.[2] Additional, less common risk factors for the development of cardiovascular disease among our patients with psoriasis and psoriatic arthritis include insulin resistance, dyslipidemia, angiogenesis, oxidative stress, and endothelial dysfunction.[3]

The presence of systemic inflammation in conjunction with the metabolic syndrome—which includes issues such as obesity, hypertension, dyslipidemia, and diabetes—puts our patients at high risk of developing cardiovascular issues. Patients with psoriasis and PsA often have difficulty controlling their hypertension and require multiple medications to help. New-onset diabetes is also common, which again adds additional medications to the mix.

Although PsA patients tend in general to have lower levels of inflammatory biomarkers such as C-reactive protein (CRP) and erythrocyte sedimentation rates (ESR) than individuals with other rheumatic diseases, any elevations in these levels tend to indicate greater disease activity. In fact, persistent CRP elevations have been associated with an increased risk of cardiovascular events and mortality, while higher levels of ESR are associated with a greater burden of atherosclerosis and clinical cardiovascular events.[1]

Rodney is one of my recent patients whose disease management required our practice to be on its toes to prevent dangerous and damaging health outcomes. He entered our practice in 2010 at the age of 58 after a referral from orthopedics due to the presence of uncontrolled, whole body plaque psoriasis. He had been diagnosed by his primary care physician with a host of other conditions, including type 2 diabetes mellitus, gout, hypertension, and hypercholesterolemia, but PsA didn't enter the picture until our practice became involved. His medical history included a short trial of methotrexate approximately 7 years ago, but because of his high level of liver enzymes, we

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"Yet while his PsA was clearly under better control, we knew about the dangers of cardiovascular disease lurking below the surface due to his Rodney's history."

knew that wasn't going to be an option anymore. According to Rodney, he had seen a rheumatologist "about 10 years ago," but was told at the time that he did not have arthritis, so he chalked up his joint pain to the natural aging process. Additional prior medical history included hyperlipidemia, daily tobacco use, bipolar disorder, degenerative lumbar disc disease, and parental cardiovascular disease.

At his initial visit to our practice, Rodney presented with severe pain in his bilateral ankles that kept him awake for most of every night. He also had severe pain in the small joints of both hands. He had stopped driving several months ago due to the pain he was suffering. In addition to these issues, Rodney also had severe plaque psoriasis covering most of his body. Plain film x-rays showed degenerative arthritis of his knees and hands. While waiting for his insurance to approve a trial of infliximab, we started Rodney on the smallest possible dose of methotrexate (2.5 mg daily) as well as a topical NSAID cream.

I first met Rodney a year later after he had been on infliximab 5 mg/kg every 6 weeks for approximately 6 months. There was significant clearing of his psoriasis, with only small patches on his arms and legs. His knees, hands, and ankles showed only intermittent minor swelling and tenderness. There was also moderate improvement of pain in Rodney's proximal and distal interphalangeal joints in his hands. Yet while his PsA was clearly under better control, we knew about the dangers of cardiovascular disease lurking below the surface due to his Rodney's history. Patients with psoriasis and PsA are at a significantly greater risk of a major adverse cardiovascular event following myocardial infarction, and often see the pain and swelling associated with their disease increase as well. Some new studies have shown a 43% increased risk of cardiovascular disease and 68% increased risk of myocardial infarction in patients with severe psoriatic disease and high levels of systemic inflammation.[2] For these and other reasons, we were always careful about talking to Rodney about his cardiovascular risk factors at his regular follow-up appointments and encouraged him to embrace a more healthy lifestyle.

Despite our efforts, in 2016, Rodney suffered a heart attack and was diagnosed with a non-ST segment elevation of myocardial infarction. Fortunately, he recovered within a few weeks and was able to resume his infliximab infusions.

His disease remained under control for the next several years, with no additional serious cardiovascular events. Degenerative pain in his right knee led to a knee replacement in early 2019. We administered his scheduled infliximab injection a week before his surgery. Surgical complications required hospitalization, at which time Rodney became septic. He was diagnosed with Methicillin-resistant *Staphylococcus aureus* in his right knee, which fortunately resolved with appropriate antibiotic treatment.

A year later, Rodney was back in our office with the latest bit of bad news. He told us that he had been suffering from periodic chest pain in the spring and had been having difficulty breathing, especially at night. A referral to cardiology for a nuclear stress test demonstrated abnormal results. He then underwent a cardiac catheterization for placement of a drug-eluting stent in the proximal left anterior descending artery. In December 2019, Rodney was again hospitalized after a mild heart attack, though he was discharged a day later after adjustments were made to his medication regimen.

In the last few months, there have been further complications. Rodney began complaining of angina with increased fatigue and shortness of breath over the summer, and a coronary angioplasty was performed on the left anterior descending artery due to stenosis. He is currently receiving injections for his pain, and we have had to pause his infliximab for a few months until his condition stabilizes.

Managing Rodney's constellation of health issues is undoubtedly going to continue to be a challenge. PsA should be recognized as a systemic inflammatory disease, with our treatment strategies based on the goal of reducing the effects of the inflammation. We desperately need improvement in the ability to screen our patients for potential cardiovascular disease, particularly those whose diagnosis has been delayed and have greater pathological changes. These are the patients, like Rodney, most likely to have ups and downs to their health that require us to remain on our toes at every visit.

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AUTHOR BIO

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Leanne Donaldson writes about her experiences as a mom and parent with a chronic illness on her blog, ***Smiles and Sundays****.*

Mental Health Comorbidities of PsA: Pieces of a Much Larger Puzzle

by Leanne Donaldson

Receiving a diagnosis of psoriatic arthritis (PsA), or any chronic illness, requires acceptance of a new reality. The struggle to accept and cope with this new reality can have a major impact on an individual's psychological and emotional health. They are intertwined in a way that makes it difficult to know for certain whether depression is in fact a symptom or a side effect of the disease. Regardless of the answer, both patients and providers need to be aware of the role that mental health plays in the larger PsA picture.

When I was diagnosed with PsA approximately a decade ago, I was a mom to three children aged 4 years and under. I was also a third-grade teacher, dealing with lots of little personalities and quirks in the classroom. And yet, I was able to care for my own needs as well as the needs of others pretty well, from my students in the classroom to my tiny humans that I tucked into bed every night. I was reliable and dependable. My disease was reasonably well controlled, and what pain and stiffness I had didn't slow me down much. Even after my diagnosis of PsA when my symptoms began to worsen, I continued to push through increasing levels of fatigue, pain, and muscle weakness with a smile on my face, optimistic about the future.

I remember the precise moment when everything changed. It was a typical morning where I found myself getting glasses of milk together for my kids' breakfast. As I hefted the gallon of milk to pour into bottles and sippy cups, it fell from my weak and throbbing hands, splattering and splashing milk everywhere. Under the watchful eyes of my children, the weight of the physical pain combined with the psychological awareness of an uncontrollable, uncertain future showered down on me. It took every single ounce of strength I had in that moment not to join the milk on the floor in a puddle of my own tears, frustration, and anger.

Physical pain does not exist within its own boundaries. It is not an independent phenomenon. Rather, it is interconnected with the psychological reality that is typically altered in those of us with a chronic disease. They are but two of the pieces of the larger puzzle that connect our lives.

What I experienced that morning was both a psychological and emotional reaction to physical pain. It showed me how my mental health is inextricably interwoven with my physical health. When I am—no matter how temporarily—physically unable to meet the immediate needs of my children, I feel absolutely helpless and utterly useless. After reliably caring for their every need for the first several years of their lives, as my disease made every-day activities more and more challenging from a physical perspective, my psychological and emotional state started breaking down as well.

"It is easy for providers to see the joint swelling and rash, but they can't so easily see the toll that PsA takes on our mental health and wellbeing."

When I spilled that milk on the kitchen floor, my mind knew that the joint pain and weakness that is a part of having PsA caused the problem, but living with the daily reality of my condition felt mentally and emotionally like an overwhelming burden. In my experience, this is the crux of what many medical professionals—through no fault of their own—struggle to fully grasp. It is easy for providers to see the joint swelling and rash, but they can't so easily see the toll that PsA takes on our mental health and wellbeing.

A patient doesn't need to have a history of mental health issues such as depression or anxiety to find themselves battling with them after their diagnosis of PsA. Prior to life with PsA, I never had mental health problems. In fact, my husband even lovingly gave me the nickname, Rainbows and Sunshine. But now, I struggle daily with the demons of depression and anxiety. The physical pain and fatigue, the unpredictability, and the isolation that exists with my disease combines to wreak havoc on my mental health.

Talking about mental health with anyone, even a trained mental health professional, often makes for an uncomfortable conversation. I have a pretty good relationship with my current rheumatologist, but even I know that if I were brave enough to bring up the topic of PsA-related anxiety with her, she would likely tell me to make an appointment to see my PCP or find a psychologist to talk to about it. Would I do it? Probably not. Why? Because the last thing I need are more doctor appointments when I barely have the energy to get through each day.

"How would you rate your level of fatigue each day?" This is always such a loaded question I am asked at every one of my rheumatology visits. Separating the fatigue that accompanies PsA from fatigue that is related to depression or anxiety is next to impossible. I never know quite what to say. "Ok?" "Fine?" Is there a difference between the two? I think so. But I'm not sure my rheumatologist feels the same way.

What can make identifying mental health issues so exceptionally difficult for rheumatology providers is that they often don't meet patients before they got their diagnosis to see what their life used to be like. Many of us, like me, were told by other providers that the pain and fatigue we are feeling is "all in your head" or perhaps is "simply part of being a new mom." Having heard that so many times, it's difficult to open up and trust anyone with feelings of depression or anxiety.

I know this all sounds terribly pessimistic, but there is hope—I promise. Once providers understand that depression and anxiety can be linked to the diagnosis of a chronic disease, there are things they can do to put a patient more at ease and help them feel comfortable talking about their emotional and mental health. Here are a few things I'd suggest:

1. **Ask direct mental health questions at EVERY appointment.**

   "Have you been depressed lately?" "Do you sometimes feel you have trouble getting through the day?" Pay particular attention to evasive answers, lack of eye contact, and attempts to change the subject.

2. **Leave out articles that discuss the links between chronic conditions and mental health struggles in every patient examination room and waiting area.**

   Those wait times to see the doctor can get long and, contrary to popular opinion, many of us actually do read all the posted signs and literature in the room while we're waiting. Include articles that go beyond scientific jargon and focus more on personal patient experiences (like this one!) to take some of the stigma out of mental health labels.

3. **Remind patients that just because they "fail" a medication, it doesn't make them a failure.**

   I know this seems like it should be obvious, but you'd be surprised how many patients in the PsA community take medication failures very personally. Even though we're told over and over that we have no control over whether a certain medication works for us or not, it can be a huge personal letdown when we start seeing our old friends—pain and stiffness—coming back around. It is a frustrating ride, especially if you find yourself in an endless cycle of hope, waiting, and letdown as you try one medication after another.

4. **View and treat each patient as a whole person rather than an assortment of swollen joints and skin rashes.**

   There isn't a single part of my body or my life that PsA hasn't impacted. True to the nature of the disease, PsA can attack joints, tendons, organs… everything. Why then would our mental health be untouchable? Just because you can't see it or touch it doesn't mean that it isn't part of the disease.

5. **Create a peer counselor community for interested patients.**

   I am well aware that the rheumatology provider community is severely overtaxed. Nurses and doctors are stretched entirely too thin and their patients' mental health is often the piece of the puzzle that falls on the floor and gets chewed on by the dog. It would make sense from a patient perspective to offer peer mental health counseling because patients are often much more open with their peers about their emotional and psychological well-being than they are with their providers.

Without having each piece in place—physical, emotional, and psychological—the life of a patient with PsA can crumble. It's so important to step back and look at the bigger picture. Help and support your patients' mental health. Give them the tools—all the tools—they needed to navigate life with their chronic disease. Everyone's puzzle is going to look a little bit different, but with a little guidance and understanding, the pieces can indeed come together to paint one amazing picture. At least one without any spilled milk.

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