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Supporting young adults with psoriatic arthritis

BY CHRISTINE M. GALANTE, DNP, RN, ANP-BC, AE-C

Abstract: Psoriatic arthritis (PsA) is associated with psoriasis, a chronic inflammatory skin disease. About 30% of patients with psoriasis develop PsA, and some of these patients are children and young adults. Because onset can be gradual, PsA signs and symptoms are easily attributed to other causes, especially in younger patients. This article discusses the assessment, pathophysiology, and diagnosis of PsA and informs nurses how best to support patients with PsA.

Keywords: dactylitis, enthesitis, PsA, psoriasis, psoriatic arthritis, psoriatic plaques

PSORIASIS IS A CHRONIC inflammatory skin disease marked by demarcated patchy raised plaques on extensor surfaces of the body. Approximately 30% of patients with psoriasis develop the chronic disease known as psoriatic arthritis (PsA), an inflammatory autoimmune disorder affecting the joints and surrounding tendons and ligaments.[1-3] Occurring equally in women and men, it generally manifests in the third to fifth decade of life.[4] However, growing evidence suggests that PsA occurs earlier in 1% of young girls and boys, starting around age 10.[5,6]

In some patients, PsA is accompanied by few if any of the skin plaques associated with psoriasis: red, pink, or silvery scaly patches on the skin.[7] These plaques typically appear on the knees, elbows, and scalp, although they can develop anywhere on the body.[8] In the absence of visible plaques, healthcare professionals may not recognize PsA on initial encounter.[9,10] Using a case study as an example, this article discusses assessment, diagnosis, treatment, and nursing considerations for patients with PsA.

A case in point

AB, a 22-year-old college student and talented athlete, had been experiencing bilateral knee pain since he was 15. Initially both knees were reddened, swollen, and warm to touch. Over time, he reported to his coach that the pain and stiffness had progressed to other areas of the body.

As he approached his late teens, he began to experience early morning stiffness in his hands, feet, knees,

back, elbows, and neck. After playing a strenuous tournament, he developed swelling around his ankles as well. This was the complaint that first brought him to the primary care provider (PCP).

AB reported that over the years, his stiffness and joint pain had been attributed to the stress of athletic activity and treated with icing and over-the-counter (OTC) nonsteroidal anti-inflammatory drugs (NSAIDs).

AB's health history included a tonsillectomy at age 8 but was otherwise unremarkable. He denied smoking, vaping, alcohol consumption, or the use of recreational drugs. Neither of his parents had a history of autoimmune or joint disease.

The PCP's initial evaluation revealed a 1-cm plaque on the right knee; otherwise physical assessment findings were normal. The PCP ordered a complete blood cell count, a Lyme disease titer, erythrocyte sedimentation rate (ESR), and uric acid and rheumatoid factor (RF) levels. All were within normal limits except for the ESR, which was slightly elevated at 30 mm/h (normal for men: 0 to 22 mm/h).

The PCP diagnosed Achilles tendonitis, instructed AB to stop playing sports for 6 weeks, and prescribed treatment with ice packs and NSAIDs for 2 weeks followed by physical therapy for 8 weeks.

Over the years, the patient's stiffness and joint pain had been attributed to the stress of athletic activity.

Although the prescribed treatment initially eased AB's signs and symptoms, his joint pain and stiffness returned when he resumed playing sports.

Classic signs and symptoms

AB presented with many hallmark signs and symptoms of PsA, which is characterized by relapsing swelling of peripheral joints in the elbow, wrist, hand, and feet, or of the axial skeleton, primarily the hips, shoulder, and spine. Enthesitis, or inflammation of one or more entheses (areas where a tendon or ligament connects with a bone), is common in patients with PsA.[8] Although not experienced by all patients, changes in fingernails and toenails, such as pitting or separation from nailbeds, and dactylitis (inflammation of fingers or toes) are also characteristic of PsA.[7]

Onset of PsA is slow and insidious. Periods of self-limiting inflammation of tendons and joints can span up to 5 years before individuals seek treatment.[1,11,12]

PsA has been classified into five subtypes (see *Five subtypes of PsA*). Although PsA cannot be cured, early diagnosis and treatment can slow progression and reduce the risk of disability and other complications later in life.

Pathophysiology

PsA is associated with an excessive inflammatory response, but the specific cause is unknown. Genetic and environmental factors both play a part in disease development.[7]

The etiology of PsA is believed to be an innate immune biochemical response that targets skin and joint tissues. It is mediated by a cluster of differentiation (CD) 8 and T-cell response that leads to a release of inflammatory cytokines and mediators, producing inflammation of tendons and joints. This abnormal inflammatory response produces a lymphocytic proinflammatory response of cytokines, interleukins, interferons, and tumor necrosis factor (TNF) that attack the soft tissue of tendons, ligaments, and synovial membranes.[10] The inflammatory state causes joint pain, stiffness, and swelling

Five subtypes of PsA[7]

The most common subtypes of PsA are the symmetric polyarthritis and asymmetric oligoarticular forms.

- symmetrical polyarthritis: symmetrical bilateral joint involvement
- asymmetric oligoarthritic: unilateral; fewer than four large joints involved
- distal interphalangeal predominant: small joints of fingers and toes; associated with spotting, pitting, and separating of nail beds
- axial arthritis/spondylarthritis: inflammation of neck, spine, and sacroiliac joints; untreated, it can result in vertebrae fusion
- arthritis mulilans: bone loss resulting in permanent damage to hands, fingers, wrist, and feet (see *Arthritis mutilans*). Untreated, it leads to loss of mobility and joint deformities called "opera glass hands" and "telescoping fingers." This is the most severe but least common form of PsA

of the tendons and ligaments in finger joints and in the lower back, neck, hips, and spine. Without treatment, 40% to 70% of patients will have joint erosions within 2 years of symptom onset, leading to irreversible joint damage with functional impairment.[13-15] As part of the inflammatory process, patients can also experience uveitis, keratitis, blepharitis, conjunctivitis, episcleritis, and scleritis; these complications may threaten eyesight.[9]

PsA can be triggered by elements in the environment combined with genetic factors and human leukocyte antigen immunodeficiency.[16] Marked by remission and relapses, this progressive disease can be triggered by stress, physical exertion, environmental elements such as weather extremes, cutaneous injury, tobacco use, infection, and hypocalcemia.[10,17] Long-term use of certain medications including corticosteroids, beta-blockers, hydroxychloroquine, tetracyclines, lithium, and NSAIDs has been associated with triggering PsA.[18,19]

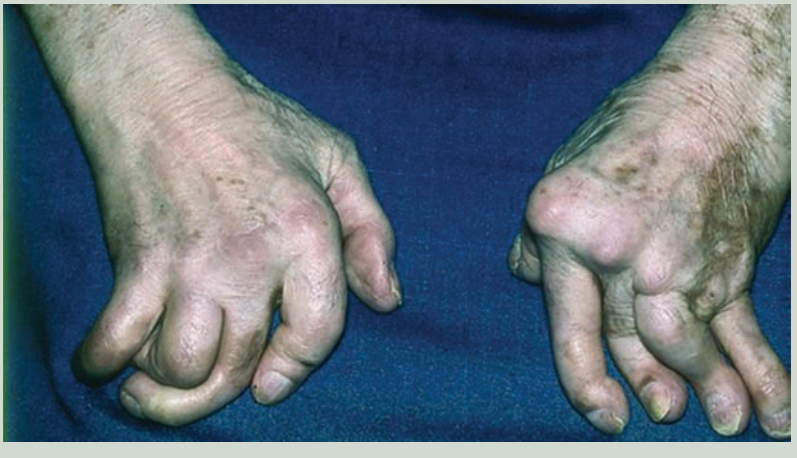
Factors that may predispose a pediatric patient to PsA include extreme emotional stress, increased body mass index (BMI), untreated streptococcal infections resulting in periodontitis and tonsillitis, and remitting Kawasaki disease.[20-24] Also called mucocutaneous lymph node syndrome, Kawasaki disease is characterized by vasculitis. Recent studies also implicate Koebner phenomenon, in which incidental injury, trauma, and release of endotoxins from B-hemolytic streptococci infections lead to psoriatic skin lesions and deeper tissue involvement, causing enthesitis and arthritis.[22,25-27]

PsA has been associated with a 43% increased risk for cardiovascular disease.[27-29] Once believed to be limited to the skin and joints, PsA has recently been linked to a range of cardiometabolic disorders: hypertension, dyslipidemia, coronary artery disease, diabetes, and obesity.[30] Research has established an association between PsA and metabolic syndrome, a systemic disorder that includes hypertension, dyslipidemia, insulin resistance, and obesity.[31] In addition, researchers have linked excessive adipose tissue to an increased production of postinflammatory cytokines, interleukin (IL)-1, IL-6, and TNF—substances that trigger systemic inflammation. This proinflammatory state of elevated cytokines, interleukins, interferon-alpha, and TNF promotes endothelial vasoconstriction and atherosclerosis.[32]

Some evidence suggests that the proinflammatory state shares a genetic link, IL12B and IL23R, to an increased incidence of inflammatory bowel disease, Crohn disease, celiac disease, and irritable bowel syndrome (IBS).[33,34] Gene mapping to identify familial risk factors is still underway.

Arthritis mutilans

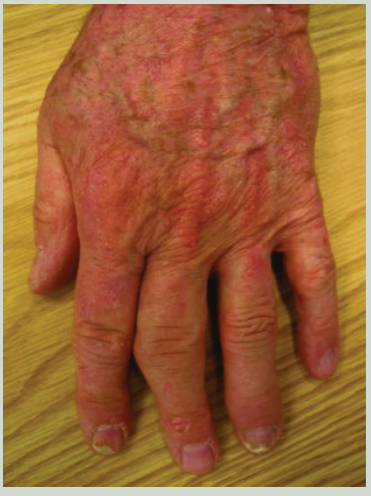
The most severe form of psoriatic arthritis, arthritis mutilans is also the most rare. It is characterized by marked deformities of the small bones in the hands and onycholysis.



Source: Goodheart H, Gonzalez M. *Goodheart's Photoguide to Common Pediatric and Adult Skin Disorders.* 4th ed. Philadelphia, PA: Wolters Kluwer Health; 2016.

Dactylitis of the fingers

This patient with severe psoriatic arthritis has marked finger deformities.



Source: *Stedman's Medical Dictionary.* 28th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.

Health history

PsA diagnosis is primarily based on a comprehensive health history and musculoskeletal exam.[35,36] When obtaining a patient's health history, the nurse should inquire about any personal or family history of psoriasis or other skin disease, as well as any personal or family history of arthritis or other joint disease. Ask the patient to describe signs and symptoms, including patterns of

pain and what typically exacerbates or relieves the pain; for example: Is pain worse in the mornings? Does it improve with activity?

Findings that should raise suspicions for PsA include a history of enthesitis (such as Achilles tendinopathy or plantar fasciitis), inflammatory back pain, dactylitis, eye disease, gout, nephrolithiasis, or chronic gastrointestinal disorders.[35] The nurse should also perform medication reconciliation and ask about the patient's use and response to both prescription and OTC medications and supplements.

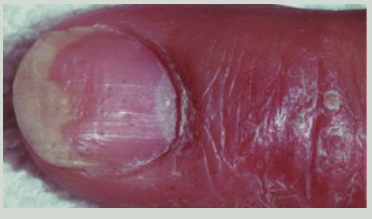
Physical assessment

On physical assessment, inspect the patient for edema in the hands, wrist, elbows, shoulders, knees, and ankles. Palpation is likely to reveal tenderness and edema over tendons. Dactylitis, known colloquially "sausage fingers," affects toes as well as fingers and can impair activities of daily living requiring fine motor control (see *Dactylitis of the fingers*).[35,37]

Due to the anatomical relationship of nail beds to the distal interphalangeal joint, some patients experience pitting of fingernails and toenails and separation of nails from nail beds (see *Nail pitting and onycholysis*). This sign is sometimes misdiagnosed as a fungal infection.

Nail pitting and onycholysis

In this patient, PsA has caused fingernail pitting and lifted the nail off the nail bed (onycholysis).



Source: *Stedman's Medical Dictionary*. 28th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.

Early, aggressive use of appropriate medications helps to slow disease progression and preserve mobility.

Enthesitis, which can impact activity levels and endurance, can affect Achilles tendons, plantar fascia, or tibial tuberosities. Enthesitis can also be evident in the area of the ribs, pelvis, and spine.[35]

Diagnosis

No definitive serum biomarker exists for diagnosing PsA, but certain lab tests and imaging studies can help confirm the diagnosis and/or rule out other disorders such as gout, rheumatoid arthritis, and reactive arthritis.[36] In endemic areas, for example, a Lyme disease titer should be performed to rule out Lyme disease.[35,38] Testing may detect autoantibodies such as RF and antinuclear antibodies in some patients with PsA.[35,38] C-reactive protein (CRP) and ESR may be normal or elevated in acute flares.[35,39,40]

The value of imaging studies is limited, but radiographs may reveal bone erosion and resorption. Ultrasound is used to visualize peripheral joints and may offer evidence of enthesitis. MRI is sensitive for detecting structural damage of soft tissue and inflammation of tendons and ligaments in the hip and spinal areas.[41-44]

Management

Guidelines for managing PsA are published by the American College of Rheumatology/National Psoriasis Foundation (ACR/NPF), European League Against Rheumatism (EULAR), American Academy of Dermatology (AAD), British Society of Rheumatology (BSR), and Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA). Recommended treatments for PsA include both nonpharmacologic therapies and topical and systemic medications. Therapies specific to plaque psoriasis are beyond the scope of this article.

Nonpharmacologic therapies and treatment approaches for patients with PsA include the following.[35]

- **Complementary and alternative therapies**. Combined with medications, therapies such as yoga, acupuncture, meditation, and therapy with topical analgesics such as capsaicin, have been found to relieve pain and stiffness and improve the quality of life for those with PsA.[45]
- **Weight control.** Maintaining a healthy weight is important because excess weight, even a few pounds, increases joint pressure and exacerbates joint pain and stiffness.
- **Physical activity.** Regular exercise not only helps patients maintain a healthy weight, but it also protects the joints by building joint-protecting muscle, increasing endurance, and improving the ability to carry out activities of daily living. During acute flares, low-impact exercise such as walking, swimming, yoga, and even stretching are good options. Advise

For more information…

- **American Academy of Dermatology**
  www.aad.org
- **American College of Rheumatology**
  www.rheumatology.org
- **Arthritis Foundation**
  www.arthritis.org
- **British Society for Rheumatology**
  www.rheumatology.org.uk
- **European League Against Rheumatism**
  www.eular.org/index.cfm
- **National Psoriasis Foundation**
  www.psoriasis.org

patients to avoid strenuous lifting or straining.[46]

• **Physical and occupational therapy.** These two therapies are designed to strengthen muscles around affected joints, increase flexibility and range of motion, and protect joints from further damage.

• **Healthy eating.** A nutritious diet that is low in added sugar and sodium can ease inflammation. Fruits, vegetables, lean protein, fatty fish such as salmon, which is rich in inflammation-easing omega-3 fatty acids, nuts, olive oil, and whole grains are all good choices. Working with a nutritionist enhances the treatment plan. The use of probiotics to reduce inflammation in the GI tract is also recommended because many patients with PsA also have IBS.[47,48]

Pharmacologic therapy is individualized and targeted to relieve signs and symptoms, reduce systemic inflammation, limit structural damage, and achieve remission.[49] Early, aggressive use of appropriate medications helps to slow disease progression and preserve mobility.

• **NSAIDs.** Utilized for symptom relief, these are often the first oral medications recommended for patients with PsA to ease joint pain, stiffness, and inflammation. However, they do not alter disease progression. Examples include ibuprofen, naproxen, meloxicam, and diclofenac.[50]

• **Disease-modifying antirheumatic drugs (DMARDs)**. These immunosuppressive agents interfere with inflammatory pathways in the body. DMARD is an umbrella term for a large group of drugs with varying mechanisms of action. They are prescribed to reduce pain and slow the progression of various types of inflammatory arthritis including PsA, and may be administered orally, by injection, or by infusion.[51-53]

Biologic DMARDs target specific parts of the immune system to help ease or stop PsA symptoms and limit damage to the joints. They have five different modes of action: TNF inhibition, T-cell co-stimulation blockade, IL-6 receptor inhibition, B-cell depletion, and IL-1 inhibition. Examples include abatacept, adalimumab, etanercept, golimumab, and secukinumab.[54,55]

Nonbiologic or *conventional DMARDs*, which affect the immune system more broadly, include apremilast, cyclosporine, dimethyl fumarate, fumaric acid esters, leflunomide, methotrexate, and sulfasalazine.[53] Other medications that may be prescribed to manage PsA include the phosphodiesterase 4 inhibitor apremilast and acitretin, a systemic retinoid.

Nursing considerations

Keeping cardiovascular risks in mind, nurses should monitor patients' BP, heart rate and rhythm, and BMI and assess lab results for elevated fasting glucose levels, liver function, total cholesterol, and both low-density and high-density lipoprotein levels. Patient education should include encouraging healthy lifestyle choices, including weight control, a nutritious diet, and regular exercise.

Engage patients in dialogue about their long-term treatment goals for PsA to maximize quality of life, prevent joint destruction, and preserve mobility. Engaging in dialogue and shared decision-making promotes self-management, improves patient outcomes, and promotes adherence to the treatment regimen.[56]

While every person with PsA is different, treatment plans typically include an individualized medication regimen, regular checkups, and lifestyle changes. Guidelines published by EULAR, ACR/NPF, AAD, BSR, and GRAPPA vary in approach and management of PsA. For example, both GRAPPA and EULAR use step-up approaches, but the GRAPPA recommendations allow for "skipping ahead" based on the patient's clinical condition.[51]

Because PsA can involve various body systems, nursing assessment and patient teaching should cover the following points.

• Ask the patient about bowel function. If the patient reports abnormal bowel function, such as alternating diarrhea and constipation with mucus or blood in the stool, the PCP may order more testing, including a fecal calprotectin test, fecal occult blood testing, and colonoscopy.

• Assess the patient for vision and ocular problems such as dry eye, eye inflammation or pain, or vision loss. As part to the inflammatory process, the patient may experience uveitis, keratitis, blepharitis, conjunctivitis, episcleritis, and scleritis, so encourage regular checkups with an eye care provider.[9]

• Inquire about disturbed sleep patterns and fatigue, which have been correlated with a higher incidence of depression and mortality.[57,58]

AB's case progression

During his period away from sports, AB's joint pain and stiffness progressed rather than improved. He also began to have gastrointestinal symptoms including frequent bowel movements, abdominal cramping,

and diarrhea alternating with constipation. He noticed larger thick scaly red skin plaques, and his fingers and toes were continuously swollen and stiff. As his symptoms progressed, he became depressed and began to withdraw from social activities.

Encouraged by family and friends, he sought treatment for depression and consulted another PCP for a second opinion. Suspecting PsA, the second PCP referred AB to a rheumatologist who confirmed the PsA diagnosis and prescribed treatment with subcutaneous etanercept weekly.

At week 12, AB reported only minimal improvements in joint mobility and pain. The rheumatologist instructed him to continue etanercept and added oral methotrexate weekly to his treatment plan. On week 20, repeat lab work revealed his ESR and CRP remained elevated. The PCP noted swelling and tenderness on range of motion of his axial joints. The methotrexate dosage was increased.

Returning for follow-up at week 50, AB was asymptomatic, with no joint deformities or pain on full range of motion. Since then, he has been able to resume his athletic activities and follows up with the PCP every 4 months. ■

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Supporting young adults with psoriatic arthritis

TEST INSTRUCTIONS

- Read the article. The test for this CE activity is to be taken online at **www.nursingcenter.com/CE/nursing.**
- You'll need to create (it's free!) and log in to your personal CE Planner account before taking online tests. Your planner will keep track of all your Lippincott Professional Development online CE activities for you.
- There's only one correct answer for each question. A passing score for this test is 14 correct answers. If you pass, you can print your certificate of earned contact hours and access the answer key. If you fail, you have the option of taking the test again at no additional cost.
- For questions, contact Lippincott Professional Development: 1-800-787-8985.
- Registration deadline is **September 2, 2022.**

PROVIDER ACCREDITATION

Lippincott Professional Development will award 1.5 contact hours for this continuing nursing education activity. Lippincott Professional Development is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation. This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 1.5 contact hours, and the District of Columbia, Georgia, and Florida CE Broker #50-1223.

Payment: The registration fee for this test is $17.95.