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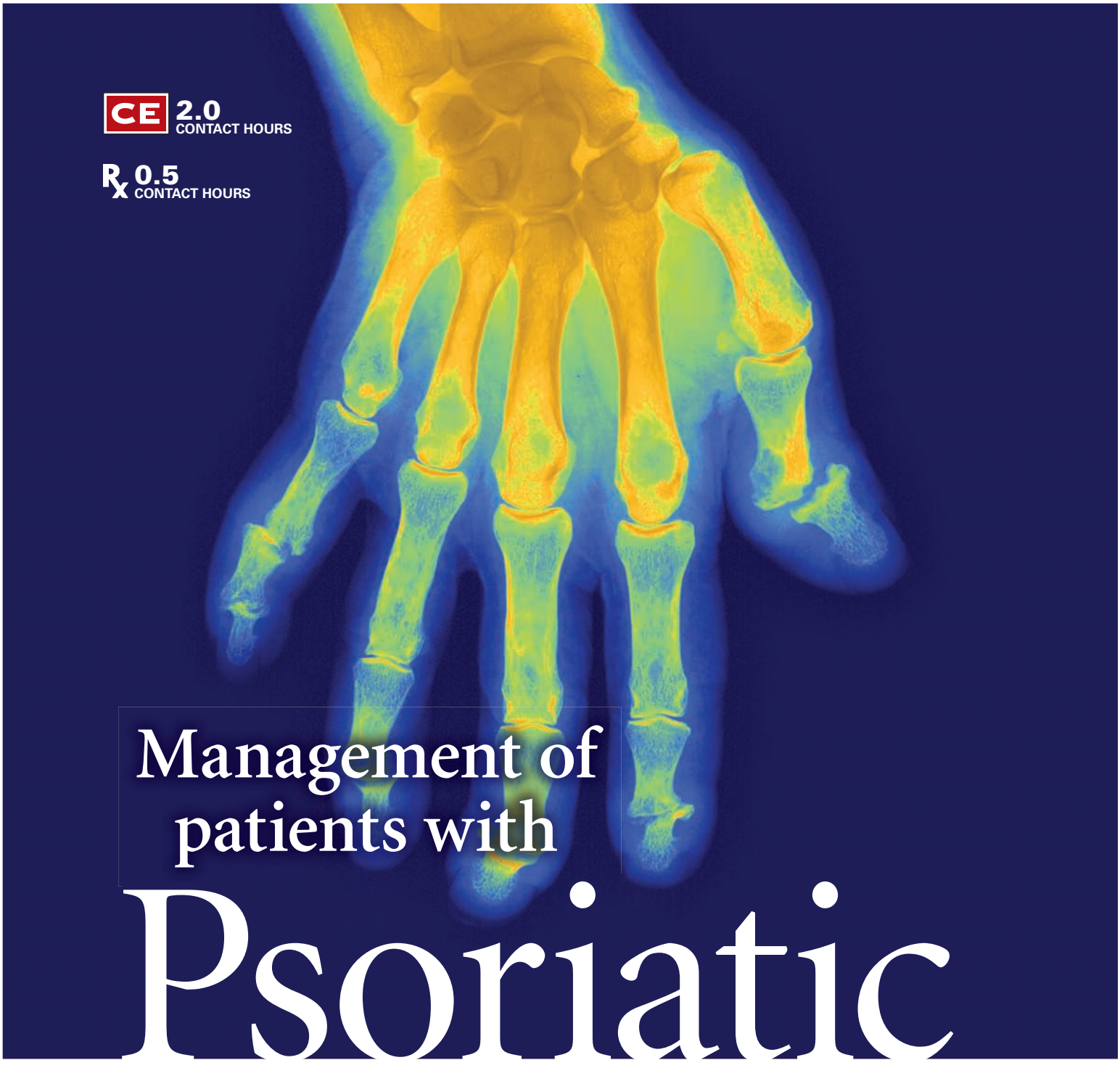
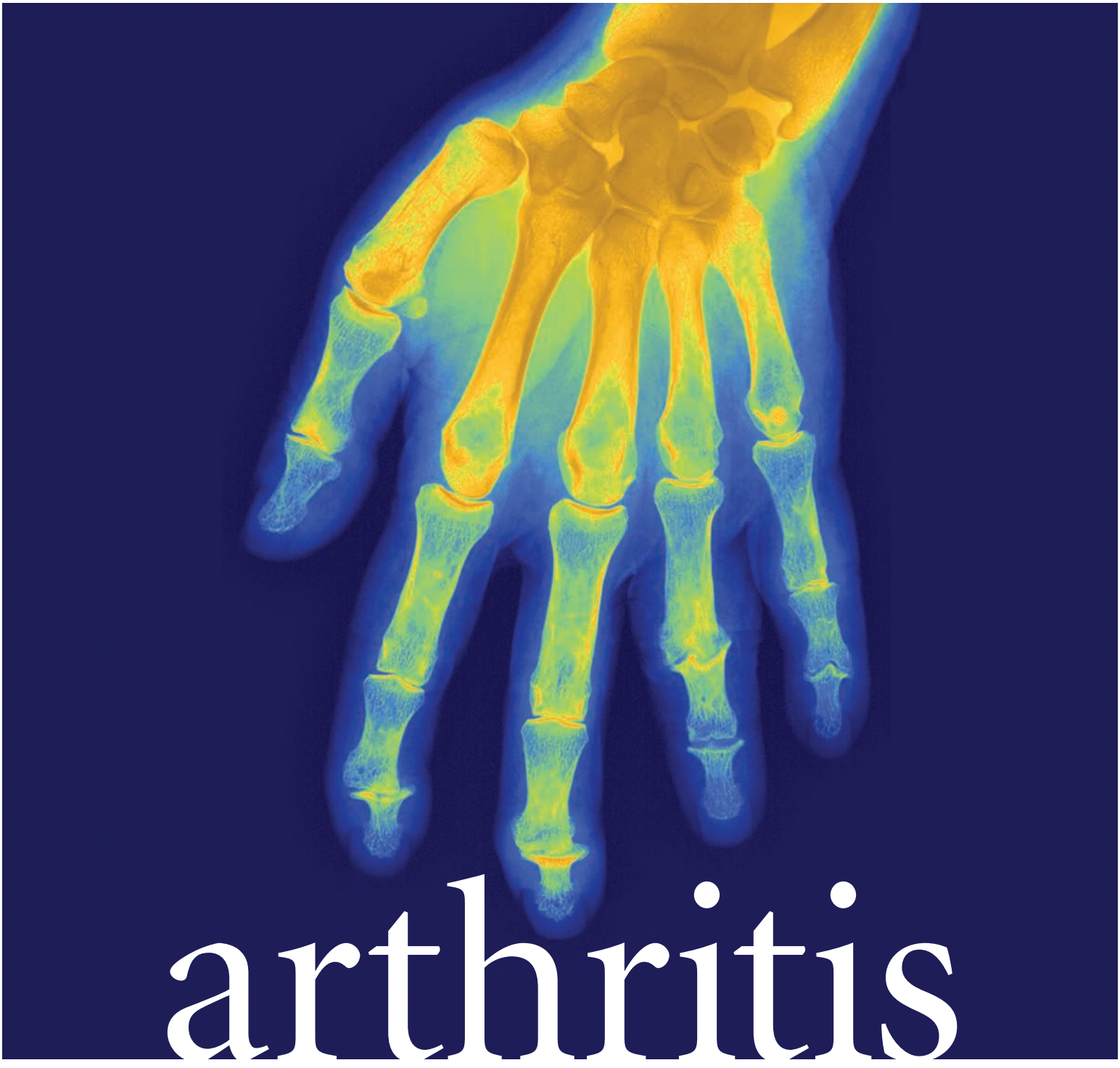


Management of patients with Psoriatic

By Kori A. Dewing, DNP, ARNP

Abstract: Psoriatic arthritis is a chronic inflammatory arthritis condition. If left untreated, patients with psoriatic arthritis may suffer pain, reduced quality of life, joint damage, and disability. Understanding clinical presentation and comorbidities of the disease, as well as current guidelines for treatment, allows the nurse practitioner to provide comprehensive care for patients.

Keywords: inflammation, joint damage, psoriasis, psoriatic arthritis, rheumatology



arthritis

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Psoriatic arthritis is a systemic inflammatory arthritis characterized by pain, stiffness, and swelling of the joints, the surrounding tendons, and ligaments. It is frequently associated with low back pain, which presents in patients with psoriasis. It is a chronic and progressive disease that may cause joint pain and damage, leading to lost productivity and reduced quality of life. Presentation may range from fairly mild monoarthritis symptoms to a severe, widespread, destructive disease. The skin and nail changes associated with psoriasis precede joint or spinal symptoms in the vast majority of patients.[1] Up until the mid-20th century, psoriatic arthritis was believed to be a subset of rheumatoid arthritis, and in many cases, it may be difficult to distinguish between the diseases.[2]

Unlike rheumatoid arthritis, which affects proportionately more females than males, psoriatic arthritis affects men and women equally (typically those between the ages of 30 and 55).[3] Psoriatic arthritis is a common condition, and it has been estimated that between 0.25% and 1% of the U.S. population suffers from psoriatic arthritis.[4]

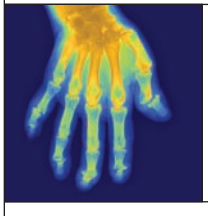
Etiology

Although the etiology of psoriatic arthritis remains unknown, it is thought that there are genetic, immunologic,

and environmental factors that may influence the onset of the disease. There is evidence of a strong genetic component to psoriatic arthritis. A large number of patients with psoriatic arthritis have a first-degree relative with psoriasis.[5] Additionally, research has shown a strong association with major histocompatibility alleles human leukocyte antigen (HLA) B18, C07, B27, B38, and B8. The highest positive predictive value is noted with HLA-B27, which is present in up to 50% of patients with psoriatic arthritis.[6]

Histologically, synovial inflammation in psoriatic arthritis has many similarities to rheumatoid arthritis. Both feature increased levels of activated T cells and inflammatory cytokines and mediators, such as tumor necrosis factor (TNF)-alpha, interleukin (IL)-17, IL-23, and other cytokines.[7]

Infection and trauma are possible environmental triggers to the development of psoriatic arthritis. Psoriatic lesions may serve as a port of entry for infectious organisms, such as group A *Streptococcus*, which may trigger the immune system in a genetically susceptible individual.[8] Development of psoriatic arthritis is also associated in patients with HIV or other immune system deficiencies.[9] The Koebner phenomenon, first described in 1872, refers to the appearance of psoriatic lesions after physical trauma to the surrounding tissue.[10]



The inflammatory arthritis presentation commonly affects the distal interphalangeal joints, but can affect any joint.

Pathophysiology

In a genetically susceptible individual in whom there has been a trigger (whether environmental or otherwise), the immune system receives a message that it should attack itself. This autoimmune response results in psoriatic arthritis. T cells become activated and drive the immune response that results in the development of inflammatory cytokines and mediators that ultimately cause erosive changes and reactive bony growth in the joints and surrounding tissue. This inflammatory cascade may occur in the joint synovium, in the enthesium (ligament or tendon attachment), or in the skin and nails. The pathogenesis of psoriatic arthritis is complex and poorly understood.[11]

Clinical presentation

Moll and Wright were the first to describe the five different patterns of psoriatic arthritis joint involvement:[12]

- distal arthritis, including distal interphalangeal (DIP) joints
- asymmetric oligoarthritis—less than 5 joints
- symmetric polyarthritis—similar presentation to rheumatoid arthritis
- arthritis mutilans—deforming and destructive
- spondyloarthritis.

Individual expression of disease varies but may include peripheral polyarthritis, skin and nail psoriatic lesions, sacroiliitis and/or spondylitis, enthesopathy, dactylitis, or ocular manifestations, such as uveitis.

The inflammatory arthritis presentation in psoriatic arthritis commonly affects the (distal interphalangeal) DIP joints but may affect any joint. Affected joints may become swollen and tender. Dactylitis is a related and common finding in psoriatic arthritis. A dactylitic digit is swollen throughout and has the appearance of a sausage digit (*see Dactylitis and onycholysis*). This pattern of swelling is due to concurrent synovitis and tenosynovitis affecting the entire finger or toe. X-rays of hands and feet in psoriatic arthritis patients, particularly those with long-standing, undertreated disease, may reveal joint space narrowing, bone erosions, and periarticular new bone formation most commonly at the joint margins.[13] Arthritis mutilans is a severe form of psoriatic arthritis that causes bone absorption of the digits, resulting in disfigurement and loss of function in the extremities. These digits are shortened and may have a "pencil-in-cup" appearance on X-ray.

Nail psoriasis is present in up to 90% of patients with psoriatic arthritis and may manifest as subungual hyperkeratosis, splinter hemorrhages, pitting of the nail, or onycholysis (separation of the nail from its bed). Nail changes may be difficult to differentiate from fungal infection upon observation alone. Psoriatic nail changes tend to be more common in patients with DIP arthritis involvement due to the proximity of the structures.[14,15]

Although back pain is a widespread problem in the general population, there are certain characteristics that may suggest back pain is inflammatory in nature, including young age at onset, improvement of pain with activity, and pain at night.[16] Radiographic psoriatic arthritis spine changes typically include asymmetrical sacroiliitis (sclerosis and loss of joint space or fusion) or spondylitis, which may affect any level of the spine and present as sclerosis or syndesmophytes (bone spurs).

Enthesopathy is a common symptom associated with psoriatic arthritis and manifests as pain and inflammation at the entheses, where the ligament or tendons attach to the bones. This presents as tenderness with palpation most commonly in the Achilles tendon, plantar fascia, or the tibial tuberosity regions.

In psoriatic arthritis, the inflammation is systemic and not limited only to the joint and surrounding tissues. Besides skin symptoms, the most frequent extra-articular manifestation of psoriatic arthritis is uveitis, which affects between 1.5% and 25% of patients.[17] Uveitis presents as an acute, painful red eye, accompanied by photophobia and blurred vision. If uveitis is suspected, immediate referral to an ophthalmologist is required because if not treated promptly, uveitis can lead to serious consequences, including vision loss. Other systemic symptoms may include fever or fatigue.

Diagnostic tests

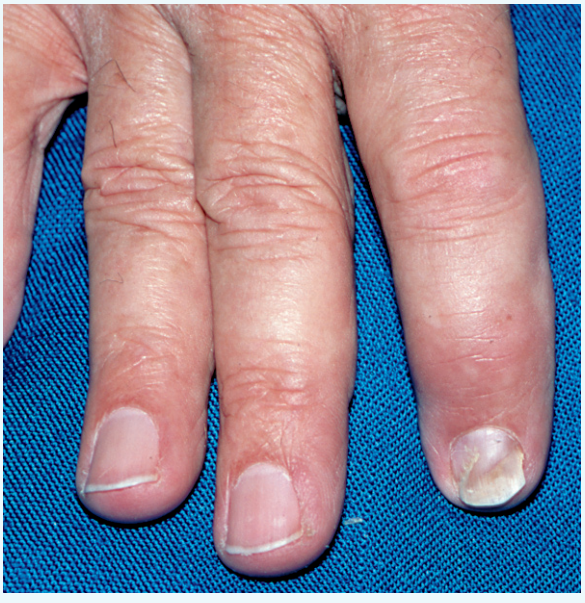
There are no autoantibodies specific to psoriatic arthritis, so there is no straightforward screening antibody test as there is for rheumatoid arthritis or other autoimmune diseases. As a result, diagnosing psoriatic arthritis—particularly early in the disease process—may be a challenge. Inflammatory arthritis in a patient with a history of psoriasis makes the diagnosis of psoriatic arthritis likely, especially if there are accompanying radiographic changes. However, other conditions may mimic psoriatic arthritis, such as rheumatoid arthritis, ankylosing spondylitis, osteoarthritis, and gout. There may be clues in the lab and imaging workup that can help guide the diagnosis.

Inflammatory markers, such as the erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP), may be elevated in patients with active psoriatic arthritis disease—particularly if the disease is polyarticular or widespread with a large burden of inflammation. However, the ESR and CRP may be normal in up to 50% of patients with active psoriatic arthritis who may not mount a significant systemic inflammatory burden.[18] There may be leukocytosis reflecting activation of the immune system or evidence of anemia of chronic disease with a mildly low hematocrit. A negative rheumatoid factor is one of the criteria of the Classification Criteria for Psoriatic Arthritis (CASPAR) criteria discussed below. It is included as a means to prevent patients with rheumatoid arthritis from being misclassified with psoriatic arthritis. However, between 2% and 16% of patients with psoriatic arthritis have a positive rheumatoid factor.[19]

The HLA-B27 genetic marker is positive in up to only 50% of patients with psoriatic spondylitis symptoms; therefore, it is not as useful in diagnosing these patients, as it is in the ankylosing spondylitis population, where up to 95% are positive. However, the HLA-B27 genetic marker is also common in the general population, affecting approximately 6.1% of the U.S. population.[20] A negative HLA-B27 may be misleading and could lead to a delay in diagnosis of psoriatic arthritis; it is therefore impractical as a screening test.

Dactylitis and onycholysis

Clinical findings of dactylitis and onycholysis in a patient with psoriatic arthritis.



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Given the complexity of the psoriatic arthritis diagnosis due to the heterogenic patient population and absence of validated and widely accepted diagnostic criteria, the CASPAR classification criteria for psoriatic arthritis were developed. The CASPAR criteria have a specificity of 98.7% and sensitivity of 91.4%.[21] To meet CASPAR criteria, a patient must have inflammatory arthritis (joint, spine, or entheses) with greater than or equal to 3 points from the following categories:

- Current psoriasis (2 points) or
  - Psoriasis present by history (1 point) or
  - Family history of psoriasis, if the patient is not affected (1 point)
- Psoriatic nail changes (nail dystrophy such as onycholysis, pitting) (1 point)
- Dactylitis (1 point)
- Negative rheumatoid factor (1 point)
- X-ray showing new bone formation near a joint (hands or feet) (1 point)[21]

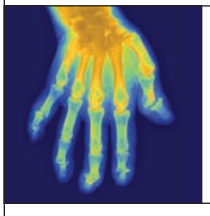
Treatment/management

Treatment of psoriatic arthritis is variable depending on severity and manifestation of disease. The treatment involves the use of a variety of medications to control the inflammation and pain as well as extra-articular manifestations of the disease. The goal of therapy is remission or

at least low disease activity if remission cannot be achieved.[22,23]

Early identification and treatment of psoriatic arthritis is important. Left untreated, irreversible joint damage and disability may occur. Initially thought of as a mild form of inflammatory arthritis, research has shown otherwise. In a large prospective 2003 clinical trial before the widespread use of biologic therapy for psoriatic arthritis, 47% of patients with early disease treated with disease-modifying antirheumatic drugs (DMARDs) developed radiographic damage within the first 2 years of disease onset.[24]

Early psoriatic arthritis treatment guidelines produced by GRAPPA (Group for Research and Assessment of Psoriasis and Psoriatic Arthritis) focused on basing therapy choice on disease manifestation and quality of evidence supporting the choice.[25] More recently, the European League Against Rheumatism (EULAR) released respected and more widely adopted recommendations that provide evidence-based guidance for the management of psoriatic arthritis.[26]



Glucocorticoid injections may be a useful adjunctive therapy in patients with single joint flares or limited dactylitis or enthesitis.

The EULAR guidelines recognized that nonsteroidal anti-inflammatory drugs (NSAIDs) should be used as first-line treatment for joint symptoms.[26] NSAIDs have been found to be helpful for spondylitis-associated back pain and joint symptoms but do not affect the skin. Cardiovascular (CV), renal, and gastrointestinal risk must be taken into consideration when prescribing or recommending NSAID therapy. Gastro-protective agents may be considered particularly in patients who develop dyspepsia symptoms or who are over the age of 65. COX-2-specific agents may also be better tolerated in some patients with gastrointestinal sensitivity. Patients taking chronic NSAID therapy should receive BP monitoring as well as routine blood tests, including serum creatinine to monitor for renal adverse reactions. NSAIDs are avoided in patients with known CV disease, as there is a dose-related increased risk of CV events.[26]

DMARD therapy should be initiated in patients with active disease despite NSAID therapy (or for those in whom NSAID therapy is contraindicated) and potential poor prognosis. Methotrexate is the most commonly used DMARD in psoriatic arthritis.[26] Serious risks include liver toxicity, interstitial lung disease, and bone marrow suppression. Leflunomide and sulfasalazine are also frequently prescribed. Benefit is seen primarily in skin symptoms and peripheral arthritis and little benefit in axial symptoms.[27] Furthermore, it is unclear whether DMARD therapy is able to prevent progression of joint damage. Therefore, DMARD monotherapy may be insufficient in patients with aggressive disease or baseline damage.

The EULAR recommendations suggest that glucocorticoid injections may be a useful adjunctive therapy, particularly in patients with single joint flares or limited dactylitis or enthesitis.[26] The use of oral or systemic glucocorticoids may trigger a flare of pustular psoriasis lesions (which may be severe) upon discontinuation and are therefore recommended with caution. Additionally, long-term use of glucocorticoids may lead to serious adverse events, such as diabetes or osteoporosis; patients should be tapered to the lowest effective dose or off when possible.

In patients who have developed an adverse event associated with DMARD therapy or who have had an inadequate response after a sufficient trial (usually 3 to 6 months), the EULAR recommendations suggest biologic TNF inhibitor therapy be considered (adalimumab, certolizumab pegol, etanercept, golimumab, and infliximab). Anti-TNF therapy has been shown to reduce pain and inflammation associated with axial and peripheral arthritis, dactylitis, enthesitis, slow joint destruction, and reduce psoriatic skin lesions by inhibiting TNF alpha, a potent inflammatory cytokine involved in the inflammatory pathway.[28-33]

Since the 2012 release of the EULAR treatment guidelines, there have been two additional medications approved by the FDA for the treatment of psoriatic arthritis. Ustekinumab is an IL-12 and IL-23 inhibitor that has been shown to reduce both the psoriatic skin lesions as well as reduce joint pain and swelling. As is the case with other biologic therapies (including anti-TNF therapies), ustekinumab is an immunosuppressant therapy that has been found to increase the risk of infection (including opportunistic infection, such as tuberculosis).[34] Apremilast is an oral therapy that inhibits phosphodiesterase 4, or PDE4, and has been found to reduce joint and skin symptoms associated with psoriatic arthritis. Patients and caregivers should monitor for the emergence or worsening depression, and there have been reports of unexplained weight loss. Additionally, apremilast should not be used concurrently with P450 enzyme inducers.[35] Although not discussed in the EULAR guidelines, joint replacement surgery remains a valid treatment option for patients with end-stage joint damage, which can help to alleviate pain and improve function.[36]

Implications for the advanced practice nurse

Effective management of patients with psoriatic arthritis requires a team approach that includes coordinated care with primary care. Early recognition and referral to a rheumatologist or other clinician skilled in the treatment of psoriatic arthritis paired with aggressive therapeutic management helps prevent irreversible damage and disability. Patients with psoriatic arthritis commonly have several comorbid conditions affecting their general health and limiting therapeutic options. Husted and colleagues performed a prospective study of 631 patients with psoriatic arthritis and found that 42% had three or more comorbid conditions.[37] Monitoring and management of comorbidities are essential aspects of care.

Studies have found an increased risk of CV disease associated with psoriasis and psoriatic arthritis, independent of established CV risk factors.[38] In a retrospective cohort study of 3,603 patients with psoriasis compared with a control group of 14,330 patients with no history of psoriasis, Abuabara and colleagues found that patients with psoriasis have a reduced life expectancy of 6 years and that CV disease was the most frequent cause of death.[39] Psoriatic arthritis and CV disease share common immunologic and inflammatory pathways, and it is believed that the underlying chronic systemic inflammation is to blame for the increased CV risk. Additionally, patients with psoriatic arthritis have (higher than observed in the general population) increased independent risk factors for CV disease, which include metabolic syndrome, tobacco use, hypertension, diabetes, insulin resistance, and dyslipidemia. Efforts by all members of the healthcare team to focus on CV risk reduction are therefore essential.

Patients with psoriatic arthritis are also at higher risk for depression, even more so than in psoriasis, rheumatoid arthritis, or ankylosing spondylitis. This is felt to be due to the psychosocial difficulties in living with a disfiguring skin condition, reduced quality of life, as well as alterations in neurotransmitters related to chronic pain.[37]

Obesity is an independent risk factor for the development of psoriatic arthritis. Additionally, it has been found that obese patients are less likely to achieve remission or near remission and are less likely to respond to anti-TNF agents. It is thought that excess body fat has a proinflammatory effect.[40] Obesity is modifiable, and weight/abdominal fat reduction is important in psoriatic arthritis patients and should be encouraged with every patient encounter.

Patients with psoriatic arthritis have an increased risk of skin cancer believed to be due to treatment-related UV light exposure. It is recommended that patients with psoriatic arthritis wear protective equipment during UV light treatment, use sunscreen when outdoors to reduce this risk, and undergo a full-skin examination annually.[41]

Improving quality of life

Psoriatic arthritis is a common chronic autoimmune condition that causes visible psoriatic skin lesions, joint swelling, surrounding tissue pain and swelling, and chronic low back pain that may lead to irreversible joint damage and disability. Recognition and aggressive management can help to restore function and quality of life. Treatment of comorbidities, especially those that contribute to CV risk, can improve quality of life. NP

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