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Psoriatic Arthritis

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Continuing Education Activity

Psoriatic arthritis (PsA) is a chronic inflammatory arthritis associated with psoriasis (PsO) and is found in about 20% of such patients with psoriasis. It shares many clinical features with other spondyloarthropathies and also rheumatoid arthritis (RA). It is usually seronegative, but a small percentage of patients may be positive for rheumatoid factor (RF) and anti-cyclic citrullinated peptide antibodies (anti-CCP antibodies). The clinical manifestations are varied and can change over time, evolving from one articular pattern to another. This activity reviews the important aspects related to the pathogenesis, epidemiology, diagnosis, and management of psoriatic arthritis and highlights the importance of an interprofessional approach to managing the different facets of psoriatic arthritis.

Objectives:

- Identify the etiology of psoriatic arthritis.
- Review the evaluation of a patient with psoriatic arthritis.
- Summarize correct interpretation and utilization of diagnostic modalities and the selection of appropriate treatment according to available guidelines and evidence-based medicine.
- Outline reasons why psoriatic arthritis patients are best managed with an interprofessional approach to treat articular disease, skin disease, other manifestations, and medical comorbidities.

Access free multiple choice questions on this topic.

Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory arthritis associated with psoriasis (PsO) and is found in about 20% of such patients.[1] It shares many clinical features with other spondyloarthropathies and rheumatoid arthritis (RA). It is usually seronegative, but a small percentage of patients may be positive for rheumatoid factor (RF) and anti-cyclic citrullinated peptide antibodies (anti-CCP). The clinical manifestations are varied and can change over time, evolving from one articular pattern to another. There is a considerable financial and psychological burden associated with this disease. There has been significant progress recently in understanding the disease pathogenesis, which has translated into new therapies.

Etiology

The etiology and pathogenesis of psoriatic arthritis are not fully understood, but it involves a complex interaction between genetic and environmental factors resulting in immune-mediated inflammation involving the skin and joints, and other organs. Approximately 33 to 50% of psoriatic arthritis patients have at least one first-degree relative who also has psoriasis or psoriatic arthritis.[2] Genes associated with psoriatic arthritis include those in the HLA region, which are involved in antigen presentation and immune recognition, and non-HLA genes involved in immune activation and inflammation, including intracellular signaling, cytokine expression, and signaling, and T cell effector function. The genetic associations between psoriatic arthritis and psoriasis are not identical, so some genes associated with psoriatic arthritis are not associated with psoriasis, and the same is true for psoriasis.[3][4] Also, certain genes are associated with specific phenotypes of psoriatic arthritis.[3][5][6]

Genetic associations in psoriatic arthritis include *HLA-B*08:01, HLA-B*27:05, HLA-B*38:01, HLA-B*39:01, HLA-B*44:02HLA-B*57:01, and HLA-C*06:02*.[6] HLA-B*08:01 is also associated with peripheral arthritis, joint damage, asymmetric sacroiliitis, and ankylosis. HLA-B*27:05 is associated with axial involvement with symmetric sacroiliitis, enthesitis, and dactylitis. *HLA-B38 and HLA-B39* genes are associated with polyarthritis.[5][3] HLA-B*44:02/03 is protective and associated with milder disease.[6] (See Table 1) The subset of patients with symmetric polyarthritis is associated with *HLA-DR4* (this gene is associated with RA).

Non-HLA genes associated with psoriatic arthritis include *IL-23R*.[4][6] These genes produce proteins involved in immune-mediated inflammation, including *TNFAIP3, TNF3IP2, REL, FBX19,* and *PTPN22*.[4][6] (See Table 2).

Psoriasis has a stronger association than psoriatic arthritis with *HLA-B*57:01* and *HLA-C*06:02* (referred to as the psoriasis susceptibility region 1 or *PSORS1*).[3][7] *IL-12B* gene is associated with psoriasis but not psoriatic arthritis.[4] Several other genes associated with psoriatic arthritis and/or psoriasis are not mentioned here.[8]

Axial disease on PsA is more complicated. *HLA-B27* gene is present in only 20% of patients with axial PsA (AxPsA) compared to 80 to 90% in patients with axial SpA (AxSpA), including AS (higher in White race.)[9][10] Axial disease in patients with PsA includes patients who are HLA-B27 positive and are clinically similar to AS with earlier age of onset, more back pain, and radiographically appear more like AS. AxPsA patients who are HLA-B27 negative have different clinical features with similar frequency in males and female, older age of onset, less back pain, more frequent cervical spine disease, with radiographic features including asymmetric sacroiliitis and non-marginal, bulky syndesmophytes which are often asymmetric.

The role of environmental factors is suspected but has been difficult to confirm. Epidemiological studies have shown an association between streptococcal infection and recent antibiotic exposure.[11][12][13][14] Skin trauma is known to induce flares of psoriatic skin lesions, which is known as the Koebner phenomenon. There is evidence the joint trauma may induce a flare of arthritis, referred to as the "internal" or "deep" Koebner phenomenon.[13][14][15] Tobacco, which is a recognized trigger for rheumatoid arthritis in patients with certain HLA-DR genes, appears to be protective against the development of psoriatic arthritis.[14][16][14]

Table 1 [6]

Table

Peripheral arthritis, joint damage, ankylosis, asymmetric sacroiliitis

Table 2 [6]

Table

Epidemiology

The epidemiology of psoriatic arthritis is heterogeneous and varies widely amongst various population groups. It has been estimated to have a prevalence of 0.05% to 0.25% in the general population and around 6% to 41% in psoriasis patients.[17] This variability of psoriatic arthritis in psoriasis is partially due to underdiagnosis. A meta-analysis showed the prevalence of undiagnosed psoriatic arthritis might be as high as 15.5%.[18] The onset of psoriatic arthritis is usually in the 30s and 40s and occurs about equally in males and females.[19]

In most patients, the onset of skin disease precedes that of arthritis (68%); in about 15% of patients, the arthritic manifestations coincide with the skin disease, and in 17% of patients, arthritis occurs before the skin manifestations making the diagnosis more difficult.[20] This latter group is more common in childhood psoriatic arthritis. When examining the occurrence of psoriatic arthritis over time in a population of patients with psoriasis, the annual incidence of psoriatic arthritis was 1.9 to 2.7% per 100 patients with psoriatic arthritis.[21][2] The cumulative prevalence of psoriatic arthritis in patients with psoriasis was 1.7% at five years, 3.1% at ten years, 5.1% at 20 years, and 20.5% at 30 years.[22][23] A severe psoriasis phenotype, scalp, intergluteal and perianal psoriasis, presence of nail pitting, low level of education, and uveitis are predictive of the development of psoriatic arthritis in patients with psoriasis.[22][2]

A literature review of the epidemiology of psoriatic arthritis showed a worldwide prevalence of PsA ranges from 0.1% to 1%.[24][1] There is great variability related to geographic and ethnic variation. The prevalence is higher in Europe (0.19%; Norway 0.67%, Sweden 0.02%) and North America (0.13%) and lower in the Middle East (0.01%), South Asia (0.06%), Sout East Asia (0.05%), East Asia (0.17%; China 0.002%, Taiwan 0.004%, Japan 0.001%) and South America (0.07%). There is evidence that the prevalence of PsA has been increasing over time.[1]

The pooled prevalence of PsA in patients with PsO is 19.7%, 21.6% in adults, and 3.3% in children.[25] The prevalence of PsA in patients with PsO was 23.8% using the CASPAR Criteria. PsA is more strongly associated with severe PsO than mild PsO (24.6% vs. 15.8%). The proportion of patients with PsO having psA varies depending on the geography and ethnicity of the target population, 22.7% in Europe, North America at 19.5%, South America at 21.5%, Africa at 15.5%, Asia at 14.0%.[25]

Pathophysiology

Various genetic risk factors predispose patients to develop psoriatic arthritis and psoriasis.[3][4][5][8] In these patients, an environmental trigger such as infection or mechanical stress initiates a chronic inflammatory process primarily involving the joints and skin, resulting in the production of IL-23, which is a central cytokine in the pathogenesis of psoriatic arthritis and psoriasis.[26][27] Macrophages and dendritic cells produce IL-23. In fact, the gastrointestinal tract may be the source of IL-23 due to disturbed barrier function or changes in the microbiota.[26][27][8] Enthesitis, which is inflammation at the site where ligaments, tendons, and joint capsules attach to the bone, is the prominent pathologic lesion in psoriatic arthritis in contrast to synovitis in rheumatoid arthritis.[28][26]

Distal interphalangeal (DIP) joints are frequently involved in psoriatic arthritis but not so in rheumatoid arthritis because these joints have many entheses but very little synovial tissue. In animal models of spondyloarthropathy, IL-23 stimulates resident T cells which are CD3+, CD4-, CD8-, IL-23R+, and ROR gamma-t+.[29] This stimulation leads to the production of IL-17, IL-22, and TNF-alpha, which promote inflammation, bone loss with erosions, and osteoproliferation.[30]

CD8+ T cells play a central role which is supported by the association of psoriatic arthritis with HLA Class I alleles, oligoclonal expansion of CD8+ T cells, and association with late-stage HIV infection.[27] Other immune cells involved in the pathogenesis include CD4+ type 17 helper (Th17) cells which produce IL-17 and IL-22, type 3 innate lymphoid (ILC3) cells which produce IL-17 and IL-22, and gamma-delta T cells which produce IL-17 and TNF-alpha.[27][26] These proinflammatory cytokines recruit neutrophils which enter the synovial fluid, activate synoviocytes, promote angiogenesis locally, active osteoclasts, which result in bone destruction, and osteoblasts, which result in new bone formation.[26][31][27][32]

This information about the basic pathophysiology has been used to develop therapies such as TNF inhibitors which were initially developed for rheumatoid arthritis and inflammatory bowel disease and are routinely used for psoriatic arthritis and psoriasis. IL-17 inhibitors are FDA approved for treating psoriatic arthritis and psoriasis but are not effective for rheumatoid arthritis. IL-12/23 inhibitors and IL-23 inhibitors are FDA approved for treating psoriatic arthritis and psoriasis.

History and Physical

The clinical presentation of psoriatic arthritis is varied. The earliest classification of psoriatic arthritis by Moll and Wright included five subtypes:

- Oligoarticular arthritis is asymmetric and involves less than five small or large joints
- Polyarticular arthritis is usually symmetric and presents similar to rheumatoid arthritis but may involve the DIP joints, rheumatoid factor negative
- Distal arthritis is signified by prominent involvement of the DIP joints
- Arthritis mutilans is characterized by severe destructive joint disease with deformities, especially in the hands and feet
- Spondyloarthritis pattern with sacroiliitis and spondylitis (this may occur with or without peripheral joint disease)

The asymmetric oligoarticular pattern is the most common form of presentation of psoriatic arthritis, accounting for at least 60% of cases of psoriatic arthritis. However, this is not true of all patient populations. Over time the majority of patients have polyarticular arthritis. In an analysis of 220 patients with psoriatic arthritis, the patterns of arthritis were as follows: oligoarticular arthritis - 14%, polyarthritis - 40%, distal arthritis - 12%, arthritis mutilans - 16%, sacroiliitis, and spondylitis - 30% (this adds up to more than 100% because axial and peripheral joint involvement may coexist.[20]

The Classification of Psoriatic Arthritis (CASPAR) study identified 63% of patients with polyarticular arthritis.[33] Axial disease is often associated with one of the patterns of peripheral arthritis. According to one prospective cross-sectional study, the frequency of radiological axial involvement in psoriatic arthritis has been found to be about 42.9%.[34] The distal pattern is less common, occurring in less than 20% of patients, and may be present along with axial disease. Arthritis mutilans prevalence can range from 2 to 21%, reflecting different definitions of this entity adopted in various studies.[35]

The classic definition referred to it as the most severe form, and its presentation was associated with osteolysis leading to digital telescoping, bone resorption, and sacroiliitis. GRAPPA initiative (Group for Research and Assessment of Psoriasis and Psoriatic Arthritis) in 2012 was able to achieve a consensus on the features related to arthritis mutilans, which involves consideration of specific features of the disease, including digital telescoping, digital shortening, “pencil-in-cup deformities,” osteolysis, and involvement of DIP joints and other small joints of the hands.[36] Features of psoriasis associated with an increased risk of axial disease include active skin lesions with induration, pustular psoriasis, nail involvement, and the Koebner phenomenon.[37]

The discrepancies seen in different studies can be explained by the heterogeneous patterns of the disease and the fact that many patients experience a transition from one pattern of arthritis to another over time; this is especially true for patients presenting with asymmetric oligoarthritis who often transition to symmetric polyarthritis. Other factors that may account for variations in patterns of psoriatic arthritis may be related to the fact that most of these studies are from referral centers for psoriatic arthritis and may have an overrepresentation of the more severe patterns of psoriatic arthritis, such as arthritis mutilans and distal arthritis. Axial involvement has generally been thought to occur mainly in patients who are HLA-B27 positive. However, this may be an oversimplification. AS with and without PsO has epidemiology similar to typical AS with a strong association with HLA-B27. Axial PsA (AxPsA) appears to be a distinct entity, and compared to AS; the prevalence is fairly equal among males and females, has an older age of onset, has a much lower association with HLA-B27 (20% vs. 80 to 90%), and is associated with HL-B*08 and HLA-B*38. Clinically, patients with AxPsA have different radiographic findings, more common C-spine involvement, improvement with TNFi and IL-17i, and possibly IL-23i.[9][10][16]

The clinical features of psoriatic arthritis are described in terms of articular and extra-articular manifestations.

Articular/Periarticular Manifestations of Psoriatic Arthritis

- Peripheral arthritis presents in an oligoarticular vs. polyarticular pattern.
- Periarticular disease includes enthesitis (inflammation around the insertion of ligaments, tendons, or joint capsules), dactylitis (swelling of the entire digit, finger, or toe, “sausage digit”), and tenosynovitis.
- Axial disease involving sacroiliac joints, usually asymmetric, and spondylitis with discontinuous involvement with bulky non-marginal syndesmophytes.

Extra-articular Manifestations of Psoriatic Arthritis

- Psoriatic skin disease usually presents before the onset of arthritis but can occur simultaneously and even before the onset of joint disease. The severity of skin disease does not correlate well with the severity of the articular disease.[38]
- Nail disease is characterized by onycholysis, pitting, and splinter hemorrhages. The severity of nail disease correlates with the severity of both skin and joint disease.[39] It is present in 80 to 90% of patients with psoriatic arthritis and is associated with DIP joint involvement.
- Ocular disease in the form of uveitis, but unlike that associated with ankylosing spondylitis, it is often chronic, bilateral, and often involves posterior elements.

Evaluation

There are no laboratory tests that are specific for psoriatic arthritis. Acute phase reactants such as ESR (erythrocyte sedimentation rate) and CRP (C-reactive protein) may be elevated, as in most inflammatory diseases. However, a normal ESR and CRP should not be used to rule out a diagnosis of psoriatic arthritis as these levels are increased in only about 40% of patients.[40] RF and anti-CCP antibodies are classically considered absent in psoriatic arthritis, and a negative RF is considered a criterion for diagnosing psoriatic arthritis as per the CASPAR classification criteria. Various studies have shown positive rheumatoid factor in about 2 to 10% of patients diagnosed with psoriatic arthritis, and approximately 5% are positive for anti-CCP antibodies.[41][42] ANA may also be positive in these patients but usually at low titers. One study by Johnson et al. showed an ANA at a titer >1:80 in 14% of patients with psoriatic arthritis.[43]

Radiographic changes show some characteristic patterns in psoriatic arthritis, consisting of erosive changes, gross joint destruction, joint space narrowing, and "pencil-in-cup" deformity.[44][45] These findings are driven by bone destruction and pathologic new bone formation, often in the same digit or even the same joint, which is a characteristic feature of psoriatic arthritis; bone destruction with bone production. Despite treatment with DMARDs (disease-modifying anti-rheumatic drugs), psoriatic arthritis results in radiographic damage in about 47% of patients during the first two years of the disease.[46] The radiological features of peripheral arthritis in hands and feet include erosive changes (including the MCP, PIP, DIP joints, and wrists), new bone formation, bony ankylosis, and joint osteolysis.[47] Entheseal involvement, including erosions and new bone formation, is characteristic in all spondyloarthropathies.[48]

Axial features, including sacroiliitis and spondylitis, are characterized by the formation of syndesmophytes (ossification of the annulus fibrosis). The features which differentiate psoriatic arthritis from ankylosing spondylitis are the asymmetric and often unilateral presentation of sacroiliitis and syndesmophytes in psoriatic arthritis are often non-marginal, bulky, asymmetric, and discontinuous skipping vertebral levels. Plain radiography, CT scan, ultrasound, and MRI are all useful in assessing patients with psoriatic arthritis.[49] Imaging modalities such as MRI and ultrasound are more sensitive than plain radiography for detecting early joint inflammation and damage, and axial changes, including sacroiliitis.[50][51] However, they are not necessary to make a proper diagnosis of psoriatic arthritis.

Classification Criteria

The most accepted classification criteria for psoriatic arthritis is the CASPAR criteria (Classification of Psoriatic Arthritis) which have been in use since 2006.[33] Other classification criteria which clinicians have used include the original Moll and Wright (1973), Bennet (1979), and Vassey and Espinoza (1984), the modified ESSG (1991) criteria.[52]

Moll and Wright Criteria (1973)

- Inflammatory arthritis (peripheral arthritis and/or sacroiliitis or spondylitis)
- The presence of psoriasis
- The absence of serologic tests for rheumatoid factor[44]

CASPAR Criteria (2006)

Clinical Features/Characteristics/Points

- Skin psoriasis: present - 2 / previously present -1 / family history, patient not affected - 1
- Nail lesions: onycholysis, pitting, hyperkeratosis - 1
- Dactylitis: present or past, documented by a rheumatologist - 1
- Rheumatoid factor: negative by any method except for latex - 1
- Juxta-articular bone formation: distinct from osteophytes - 1

Per CASPAR criteria, psoriatic arthritis is considered present in patients with inflammatory arthritis who have at least 3 points; this has a specificity of 98.7% and a sensitivity of 91.4%.[33]

Treatment / Management

General Principles

1. Treatment should be guided by disease severity, degree of joint damage, the extent of extra-articular disease, patient preference, and other comorbidities.
2. Non-pharmacological therapies, including physical therapy, occupation therapy, exercise program, and smoking cessation, should be strongly encouraged and incorporated into the treatment plan.[53]
3. **Treat-to-Target approach** is the most effective way to control disease activity and minimize joint damage.[54][55] A target of low remission or low disease activity should be employed depending on disease extent, chronicity, and other comorbidities.
4. Due to the heterogeneous presentation of psoriatic arthritis, the type of treatment initiated depends on the domains involved, including peripheral arthritis, enthesitis, dactylitis, axial disease, and skin/nail disease.
5. In treatment-naïve patients, NSAIDs (non-steroidal anti-inflammatory drugs) are generally useful for symptoms of mild peripheral arthritis.[56]
6. Mild to moderate peripheral arthritis may be treated with conventional synthetic DMARDs (disease-modifying antirheumatic drugs) such as methotrexate or occasionally sulfasalazine; the latter is ineffective for skin disease.[57]
7. Severe peripheral arthritis usually receives treatment with biologic DMARDs, especially TNF (tumor necrosis factor) inhibitors.
8. Axial disease and enthesitis are usually treated the same way except for the fact that there is a minimal role of conventional synthetic DMARDs. Patients who fail NSAIDs should automatically transition to biologic DMARDs.
9. A TNF inhibitor is usually recommended over an IL-17 inhibitor, IL-12/23 inhibitor, abatacept, or tofacitinib.
10. An IL-17 inhibitor is usually recommended over an IL-12/23 inhibitor, abatacept, or tofacitinib.
11. An IL-12/23 inhibitor is usually recommended over abatacept or tofacitinib.
12. In patients with severe psoriasis, an IL-12/23 inhibitor or an IL-17 inhibitor may be used instead of a TNF inhibitor.
13. Tofacitinib may be used instead of a TNF inhibitor in patients preferring oral medication and who do not have severe psoriasis.
14. ACR/NPF (American College of Rheumatology/National Psoriasis Foundation) 2018 guidelines recommend a TNF inhibitor over conventional synthetic DMARDs (labeled as OSM, oral small molecules) as a first-line treatment in treatment-naïve psoriatic arthritis patients.

A summary of various drugs used in treating psoriatic arthritis appears in a tabular form in Table 1.

Active Psoriatic Arthritis (as defined in ACR/NPF 2018 guidelines)

Defined as “disease-causing symptoms at an unacceptably bothersome level as reported by the patient, and judged by the examining clinician to be due to psoriatic arthritis” based on ≥1 of the following:

- Swollen joints
- Tender joints
- Dactylitis
- Enthesitis
- Axial disease
- Active skin and/or nail involvement
- Extra-articular inflammatory manifestations such as uveitis or inflammatory bowel disease[58]

ACR/NPF Guidelines for the Treatment of Psoriatic Arthritis: 2018. Initial Treatment

Oral small molecule (OSM): methotrexate (MTX), sulfasalazine (SSZ), leflunomide (LEF), cyclosporine (CSA), apremilast

- Treat with TNF inhibitor over oral small molecule (OSM): May consider OSM such as MTX in patients with mild psoriatic arthritis and psoriasis, patient preference, contraindication to TNF inhibitor.
- Treat with TNF inhibitor over IL-17 inhibitor: May consider IL-17 inhibitor in patients with severe psoriasis or contraindication to TNF inhibitor.
- Treat with TNF inhibitor over IL-12/23 inhibitor: May consider IL-12/23 inhibitor in patients with severe psoriasis of contraindication to TNF inhibitor.
- Treat with OSM over IL-17 inhibitor: May consider IL-17 inhibitor in patients with severe psoriasis and/or psoriatic arthritis.

- Treat with OSM over IL-12/23 inhibitor: May consider IL-12/23 inhibitor in patients with severe psoriasis and/or psoriatic arthritis or concomitant inflammatory bowel disease.
- Treat with Methotrexate over NSAIDs: May consider NSAIDs in patients with mild psoriatic arthritis and psoriasis.
- Treat with IL-17 inhibitor over IL-12/23 inhibitor: May consider IL-12/23 inhibitor in patients with concomitant inflammatory bowel disease.

EULAR Recommendations for the Management of Psoriatic Arthritis with Pharmacological Therapies: 2019 Update

Non-steroidal Anti-Inflammatory Drugs (NSAIDs), Disease-modifying antirheumatic drug (DMARD), Conventional synthetic DMARD (csDMARD), Biologic DMARD (bDMARD), Monoclonal antibody (mab) Targeted synthetic DMARD (tsDMARD), Janus kinase (JAK), Phosphodiesterase-4 (PDE4)[59]

Overarching principles

- Psoriatic arthritis is a heterogeneous and potentially severe disease that may require multidisciplinary treatment.
- Treatment of psoriatic arthritis patients should aim at the best care and must be based on a shared decision between the patient and rheumatologist, considering efficacy, safety, and costs.
- Rheumatologists are the specialists who should primarily care for the musculoskeletal manifestations of patients with psoriatic arthritis, with significant skin involvement work with a dermatologist.
- The primary goal of treating patients with psoriatic arthritis is to maximize the health-related quality of life through control of symptoms, prevention of structural damage, and normalizing physical and social function by controlling inflammation is an important component.
- When managing patients with psoriatic arthritis, non-musculoskeletal manifestations (skin, eye, and GI tract) and comorbidities (metabolic syndrome, cardiovascular disease, and depression) should be considered.

Recommendations

- Treatment should be aimed at reaching the target of remission or, alternatively, low disease activity by regular disease activity assessment and appropriate adjustment of therapy.
- NSAIDs may be used to relieve musculoskeletal signs and symptoms.
- Local glucocorticoid injections should be considered adjunctive therapy in psoriatic arthritis; systemic glucocorticoids may be used with caution at the lowest effective dose.
- In patients with polyarthritis, a csDMARD should be initiated rapidly, with MTX preferred in those with relevant skin involvement.
- In patients with monoarthritis or oligoarthritis, particularly with poor prognostic factors such as structural damage, high ESR or CRP, dactylitis, or nail involvement, a csDMARD should be considered.
- In patients with peripheral arthritis and an inadequate response to at least one csDMARD, therapy with a bDMARD should be commenced; when relevant skin involvement, an IL-17 inhibitor or IL-12/23 inhibitor may be preferred.
- In patients with peripheral arthritis and an inadequate response to at least one csDMARD and at least one bDMARD, or when a bDMARD is not appropriate, a JAK inhibitor may be considered.
- In patients with mild disease and an inadequate response to at least one csDMARD, in whom neither a bDMARD nor a JAK inhibitor is appropriate, a PDE4 inhibitor may be considered.
- In patients with unequivocal enthesitis and insufficient response to NSAIDs or local glucocorticoid injections, therapy with a bDMARD should be considered.
- In patients with predominantly axial disease, which is active and has an insufficient response to NSAIDs, therapy with a bDAMRD should be considered, which according to current practice, is a TNF inhibitor; when there is relevant skin involvement, IL-17 inhibitor may be preferred.
- In patients who fail to respond adequately to or are intolerant of abDMARD, switching to another bDMARD or tsDMARD should be considered, including a switch within a class.
- In patients with sustained remission, cautious tapering of DMARDs may be considered.

Differential Diagnosis

Psoriatic arthritis shares some clinical features with other inflammatory arthritides, including rheumatoid arthritis (RA), reactive arthritis (ReA), and ankylosing spondylitis (AS). In some cases, it is difficult to make a precise diagnosis. Unlike psoriatic arthritis, rheumatoid arthritis tends to be symmetrical and generally spares the DIP joints. Ankylosing spondylitis has an earlier age of onset compared to psoriatic arthritis, and sacroiliac involvement is usually symmetric rather than asymmetric. The salient features of psoriatic arthritis compared to other arthritides are shown in Table 2.

Treatment Planning

Disease Monitoring

As with any other inflammatory arthritis, patients with psoriatic arthritis require regular disease activity monitoring and appropriate changes to therapy based on the measurement of disease activity. Evaluation of all the domains, including peripheral joints, entheses, digits, axial involvement, and skin and nails, is crucial. The following methods are used to assess disease activity in clinical practice and clinical trials.

Various Parameters are Used in Assessing Disease Activity in Psoriatic Arthritis

1. Tender and swollen joint counts of 68 joints and 66 joints, respectively, in peripheral arthritis
2. Axial disease activity is determined by BASDAI (Bath Ankylosing Spondylitis Disease Activity Index), also used in ankylosing spondylitis
3. Health-related QoL (Quality of Life) as measured by indices like PsAQOL

4. Fatigue assessment by FACIT (Functional Assessment of Chronic Illness Therapy)
5. Composite indices like DAPSA (Disease Activity Index for Psoriatic Arthritis), MDA (Minimal Disease Activity), America's College of Rheumatology criteria viz ACR 20/50/70 (Table 3), Psoriatic Arthritis Response Criteria (PsARC)(Table 4), and CDPAI (Composite Psoriatic Disease Activity Index)(Table 5)
6. The RAPID3 (Routine Assessment of Patient Index Data), which is useful for assessing disease activity in rheumatoid arthritis, is simple to administer and does not require any laboratory indices. It compares favorably to other, more complicated measures of disease activity in psoriatic arthritis and is more practical for routine clinical care.[60] The DAS28 (Disease Activity Score using 28 joints) is used frequently in the measurement of disease activity in rheumatoid arthritis, but it is inadequate as it focuses on peripheral arthritis.

A treat-to-target approach to attain remission or minimal disease activity (MDA) is a strong recommendation.[55] A patient is considered to have achieved MDA if they meet 5 of 7 following criteria.[61]

Minimal Disease Activity (MDA)

- Tender joint count of less than or equal to 1
- Swollen joint count less than or equal to 1
- Psoriasis Area and Severity Index of less than or equal to 1 or body surface area less than or equal to 3
- Patient pain visual analog scale (VAS) score less than or equal to 15 mm
- Patient global disease activity VAS score less than or equal to 20 mm
- Health Assessment Questionnaire score less than or equal to 0.5
- Tender entheseal points less than or equal to 1

Specific Agents [62]

csDMARDs

- Methotrexate (MTX): Useful in patients with mild peripheral joint and skin disease; Dose is 7.5 to 25 mg every week, PO or SC usually given with folic acid 1 mg every day; Toxicities include oral ulcers, nausea, cytopenias, increased risk of infections, pulmonary toxicity (pneumonitis), teratogenic; Avoid excess alcohol, be careful with CKD; Check CBC and CMP every 2 to 4 months.
- Sulfasalazine (SSZ): Useful in patients with mild peripheral joint disease, not helpful for skin disease; Dose is 1000 to 1500 mg bid; Toxicities include rash, nausea, diarrhea, elevated LFTs, rarely leukopenia/neutropenia; Check CBC and CMP every 2 to 4 months.
- Leflunomide (LEF): Useful in patients with mild peripheral joint and skin disease; Dose is 10 to 20 mg every day; Toxicities include diarrhea, hair loss, skin rash, leukopenia, elevated LFTs, weight loss; Check CBC and CMP every 2 to 4 months.
- Cyclosporine (CSA): Useful in patients with active skin disease but less so for peripheral joint disease; Dose is 2.5 to 5 mg/kg/day in bid doses (usually 3 mg/kg/day); Toxicities include decreased kidney function, hypertension, headache, elevated cholesterol, excessive hair growth, gum hypertrophy.

bDMARDs

- TNF inhibitors (TNFi): Useful for patients with moderate-severe active PsA with peripheral and axial joint disease and skin disease; check TB test before starting, check CBC and CMP every 3-4 months; all TNFi are also approved for RA and AS.
- Monoclonal antibody TNFi is effective for uveitis and IBD.
    - Etanercept: p75 TNF-a receptor:IgG Fc fusion protein; Dose is 50 mg SC every week; FDA approved for PsA and PsO.
    - Infliximab: chimeric mab to TNF-alpha; Dose is 5 mg/kg IV every six weeks after loading; FDA approved for PsA and PsO.
    - Adalimumab: human mab to TNF-alpha; Dose is 40 mg SC every 2 weeks; FDA approved for PsA and PsO.
    - Golimumab: human mab to TNF-alpha; Dose is 50 mg SC every month or 2 mg/kg IV every 2 months; FDA approved for PsA and PsO.
    - Certolizumab pegol: humanized Fab' fragment specific for TNF-a conjugated to 40kDa PEG; Dose is 200 mg SC every 2 weeks after loading; FDA approved for PsA, effective for PsO.
- Toxicities include administration reactions, increased risk of infections including mycobacterial and fungal infections, demyelination, CHF, drug-induced lupus, and paradoxically psoriasis.
- IL-17 inhibitors (IL-17i): Useful for patients with moderate-severe active PsA with peripheral and axial joint disease and skin disease; check TB test before starting, check CBC and CMP every 3-4 months.
- IL-17i are not effective for uveitis or IBD.
    - Secukinumab: human mab to IL-17A; Dose is 150 to 300 mg SC every month after loading; FDA approved for PsA, PsO, and AS. Secukinumab at doses of 300 mg every month was effective in patients with PsA and axial disease in a double-blind, randomized trial. [63]
    - Ixekizumab: humanized mab to IL-17A; Dose is 80 mg SC every month after loading; FDA approved for PsA, PsO, and AS.
    - Brodaulmab: human mab to IL-17R: Dose is 210 mg SC every 2 weeks after loading; FDA approved for PsO, not for PsA.
- Toxicities include an increased risk of infections, including mycobacterial and fungal infections and candida infections.
- IL-12/23 and IL-23 inhibitors (IL12/23i, IL-23i): Useful for patients with moderate-severe active PsA with peripheral joint disease and skin disease; check TB test before starting, check CBC and CMP every 3-4 months.
- IL-12/23i are not effection for axial disease of AS or nr-AxSpA by may be effective for axial PsA.

- Ustekinumab (IL12/23i): human mab to IL-12/23 p40 subunit; Dose is 45 mg SC every 3 months after loading (90 mg if weight >100 kg); FDA approved for PsA and PsO, also approved for Crohn's disease.
- Guselkumab (IL-23i): human mab to IL-23 p19 subunit; Dose is 100 mg SC every 2 months after loading; FDA approved for PsA and PsO.
- Tildrakizumab (IL-23i): human mab to p19 subunit of IL-23; Dose is 100 mg SC every 3 months after loading; FDA approved for PsA and PsO.

- Toxicities include an increased risk of infections, including mycobacterial and fungal infections.
- T cell costimulatory inhibitors: Useful for patients with mild-moderate PsA with peripheral joint disease, minimally effective for PsO, check TB test before starting, check CBC and CMP every 3-4 months.
  - Abatacept: CTLA4:IgG Fc fusion protein, Dose is 125 mg SC every week or 500 to 1000 mg every month after loading (depending on weight); FDA approved for PsA and RA.
- Toxicities include an increased risk of infections, including mycobacterial and fungal infections.
- In the randomized, placebo-controlled trials of bDMARs, the use of csDMARDs such as MTX did not improve outcomes for PsA or PsO. Analyses of large cohorts of patients with PsA and csDMARDs such as MTX improved clinical outcomes; using csDMARDs improved TNFi drug survival[64] and remission rates, especially with MTX.[65]
- Some retrospective studies suggest that patients with PsO treated with bDMARDs have a lower risk of developing PsA.[66][67][68]

tsDMARDs

- PDE-4 inhibitors: Useful for patients with mild PsA with peripheral joint disease and skin disease; no laboratory testing is needed to monitor therapy.
  - Apremilast: Dose is 30 mg bid after titration.
- Toxicities include GI intolerance, nausea, diarrhea, weight loss, and depression.
- Janus kinase inhibitors (JAKi): Useful for moderate-severely active PsA with peripheral and axial disease, modestly active for PsA, check TB test before starting, check CBC, Neutrophil count, and CMP every three months, lipid panel.
  - Tocilizumab: Dose is 5 mg PO bid or XR, 11 mg PO every day; FDA approved for PsA, and also AS, RA, and ulcerative colitis.
  - Upadicitinib: Dose is 215 mg daily: FDA approved for PsA, AS, RA, and ulcerative colitis.
- Toxicities include an increased risk of infections including herpes zoster, TB, fungal, neutropenia, elevated LFTs, elevated cholesterol, and GI perforations. There is evidence of an increased risk of cardiovascular disease and cancer (RA trial).[69]

Prognosis

Psoriatic arthritis is considered an aggressive disease with the potential for significant morbidity and poor quality of life in patients. Some features are harbingers of a severe disease course and poor prognosis. These include a large number of actively inflamed joints or polyarticular presentation, elevated ESR, clinical or radiographic damage, loss of function, and diminished quality of life.[70]

Complications

Once considered a mild disease, psoriatic arthritis is now considered a debilitating disease requiring targeted treatment with frequent monitoring and follow-up care. Complete symptomatic relief is achievable, but a significant majority of patients continue to have persistent inflammatory disease.[71] Patients with uveitis will require evaluation and treatment by an ophthalmologist. Patients with PsA have an increased prevalence of comorbidities, including metabolic syndrome; obesity, diabetes mellitus, hyperlipidemia, hypertension, and cardiovascular disease.[72]

Consultations

Patients are best managed by rheumatologists but in collaboration with dermatologists if they have significant psoriasis.

Deterrence and Patient Education

Patients should be extensively educated and counseled with regards to the chronic nature of psoriatic arthritis and the importance of non-pharmacological measures, including exercise, smoking cessation, weight loss, physical therapy, and occupational therapy. They should be made aware of the fluctuating nature of this disease, requiring very close monitoring by the multi-disciplinary treatment team. The side effects related to immunosuppressive medications require a detailed explanation, and an attempt should be made to educate the patient family as well.

Enhancing Healthcare Team Outcomes

Patients with psoriatic arthritis have a heterogeneous clinical presentation with the involvement of various domains and are best managed with an interprofessional team approach to treating articular disease, skin disease, other manifestations, and medical comorbidities. Patient education is vital to ensure that the symptoms are under control. The physical therapist should encourage exercises to restore joint function. The pharmacist should educate the patient on different medications, their benefits, and adverse reactions, as well as monitor agent selection, dosing and checking for potential drug-drug interactions. Nurses should educate patients on the importance of abstaining from alcohol and discontinuing tobacco, answer questions, and help monitor treatment progress. The dietitian should encourage a healthy diet and weight. A mental health nurse and psychiatrist should be involved, as many patients develop severe anxiety and depression. Patients should be encouraged to seek stress relief. The social worker should assess the home to ensure it can accommodate the patient's lifestyle. All of these various disciplines need to chart and share their perspectives with the rest of the team so that all healthcare team members are operating from the same information base and corrective actions can be taken when necessary.

It merits noting that psoriatic arthritis patients are also at increased risk of death compared to the general population from cardiovascular diseases such as coronary artery disease leading to angina and myocardial infarction.[73] Thus, reversing the risk factors for ischemic heart disease is vital. Effective interprofessional coordination and communication between rheumatology, dermatology, and primary care, as well as nursing staff,

pharmacy, and other ancillary healthcare team members mentioned above, are required to attain the best clinical outcome in patients with psoriatic arthritis. [Level 5]

Review Questions

- Access free multiple choice questions on this topic.
- Comment on this article.

Figure

Psoriatic Arthritis Tables. Contributed by Vivekanand Tiwari, MD

Figure

Psoriatic Arthritis: Arthritis Mutilans in patient with Psoriatic Arthrits, showing destruction of both hand joints. A "pencil in cup" change of the metacarpophalangeal joints is charcterisitic. Contributed by Pol J Radiol. 2013 Jan-Mar; 78(1): 7–17. (more...)

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