# **Rheumatology Revealed 2.0: Advanced Insights and Evolving Best Practices for Family Physicians** Pearls for practice

From Skin to Joints: Mastering Psoriatic Arthritis in Primary Care Dr. Shafiq Akbar

#### Introduction

What is Psoriatic Arthritis (PsA)?

- It is a chronic inflammatory disease- skin and joints, as well as extraarticular features such as enthesitis and dactylitis.
- Historic: Moll and Wright criteria, which refer to "an inflammatory arthritis associated with psoriasis (Pso), which is usually negative for rheumatoid factor."
- Clinical Domains- eight domains peripheral arthritis, enthesitis, dactylitis, axial, skin, and nails, psoriasis and IBD.
- Comorbidities including depression, anxiety, uveitis, cardiovascular events, and cardiovascular risk factors such as hypertension, diabetes, and hyperlipidemia.

## Prevalence/Epidemiology

- Vary tremendously by countries suggesting geographic differences (Whites two times more common than other ethnic groups)
- Psoriasis 2-3 %
- Psoriatic arthritis 0.3% to 1% of the U.S. population
- 7% to 42% in patients with Psoriasis
- A Canadian population study from 2019 reported the cumulative prevalence estimate for PsA of 0.17 %, with an incidence of 15 per 100,000 population
- Historically, seronegativity for rheumatoid factor (RF) was required for the diagnosis; however, over 10 percent of patients with uncomplicated psoriasis and up to 15 percent of the normal population have RF present in their serum. Several reports also documented positive cyclic citrullinated peptide (CCP) antibodies in PsA patients.

# Prognostic factors associated to radiographic changes in PsA .

- Polyarthritis
- Structural damage
- Elevated acute phase reactants
- Dactylitis
- Nail involvement







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## **Domains of Psoriatic Arthritis**



## **Etiology and Pathogenesis**

- Imaging studies have demonstrated that Psoriasis (Pso) without clinical arthritis is strongly associated with subclinical abnormal signals in entheses and bone
- TNF (tumor necrosis factor) has a pivotal role in disease pathogenesis, Interleukin (IL)-23/IL-17, Phosphodiesterase 4
- JAK-STAT pathways are also effective for both skin and joint inflammation as well
- Both microscopic intestinal inflammation and intestinal dysbiosis may contribute to the initiation and persistence of skin and joint inflammation in PsA
- Evidence of trauma and infection play a role in PsA (deep Koebner phenomenon- 25% of people before onset of PsA







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# CASPAR, Classification Criteria for the Study of PsA

- Inflammatory articular disease (joint, spine, or enthesis)
- And at least three points from the following:
  - Current psoriasis (2 points), a personal history of psoriasis (1 point), or a family history of psoriasis (1 point)
  - Typical nail dystrophy (1 point): onycholysis, pitting, hyperkeratosis
  - Negative rheumatoid factor test (1 point)
  - Dactylitis (1 point): current dactylitis or a previous episode noted by a rheumatologist
  - Juxta-articular new bone formation (1 point) on hand or foot radiographs
- Three of more points have 99% specificity and 92% sensitivity for PsA

https://pubmed.ncbi.nlm.nih.gov/16871531/

## Psoriasis risk factors for PsA transition

- The specific mechanisms involved in the transition from psoriasis to PsA remain poorly understood.
- Risk factors for PsA development- Nail, scalp, and inverse psoriasis are associated with a greater risk of PsA
- Detailed examination of the skin, including nails and "hidden areas" such as the scalp, intergluteal cleft, behind the ears, and periumbilical area, is important to identify psoriasis

# Clinical Domains cont.

## Arthritis mutilans



#### Nail Psoriasis



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# Moll and Wright Classification of PsA

- Arthritis with predominant distal interphalangeal joint involvement
- Arthritis mutilans
- Symmetric polyarthritis—indistinguishable from rheumatoid arthritis
- Asymmetric oligoarticular arthritis
- Predominant spondylitis

Moll JMH, Wright V. Psoriatic arthritis. Semin Arthritis Rheum. 1973;3:55-78.

# **Clinical domains**

#### DIP joint involvement

Symmetric polyarthritis



Dactylitis



Enthesitis





- In contrast to skin disease there is a close association between nail and joint involvement .
- It's a risk factor for the future development of PsA
- 60% to 80% of patients with PsA have nail involvement
- Nail changes often occur only 1 to 2 years before the onset of joint involvement
- Nail psoriasis occurs in 15% to 50% of patients with psoriasis
- Pitting, ridging, hyperkeratosis, and onycholysis.





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#### Axial involvement

- Inflammation of the spine and/or sacroiliac (SI) joints
- Spondylarthritis is the dominant feature in only a minority of patients (around 5%)
- Clinical and radiologic involvement of the spine can be detected in approximately one-third of cases
- SI joints can be symmetric or asymmetric.
- Patients with bilateral sacroiliitis have a stronger association with HLA-B27
- Cervical spine involvement can be clinically silent, patients with PsA, particularly those with long-standing disease- must get C spine imaging before receiving general anesthesia

#### **Ocular Manifestations**

- Uveitis, keratoconjunctivitis sicca, keratitis, blepharitis, conjunctivitis, episcleritis, and scleritis are estimated to occur in 10% of patients with psoriasis and 31% of patients with PsA
- Uveitis is the most common ocular manifestation in PsA prevalence of up to 25%,
- The most common type of uveitis in spondyloarthropathies (SpA) is anterior uveitis
- However, while in AS and reactive arthritis uveitis is generally sudden, unilateral, confined to the anterior chamber, and completely resolves between episodes,
- The uveitis associated with IBD or PsA tends to be more insidious in onset, bilateral, chronic, and may be anterior, intermediate, posterior, or panuveitis.
- Axial pattern of PsA is associated with unilateral, anterior uveitis, while peripheral arthritis can be equally anterior or posterior.
- HLA B27 + tend to be males and have an axial pattern compared to HLA-B27 negative



Inflammatory Bowel Disease

- Patients with psoriasis and PsA present a higher prevalence of subclinical bowel inflammation and an increased risk for Crohn's disease
- Psoriasis, PsA, and IBD share pathogenic pathways, including TH1 and Th17 responses.
- Interleukin (IL)-12B and IL-23R are also common genes between psoriasis and Crohn's disease

#### Investigations

- No laboratory tests are diagnostic of PsA.
- ESR and C-reactive protein (CRP) are often elevated (but not always)
- Other typical inflammatory changes may be observed (e.g., anemia of chronic disease, hypoalbuminemia, increased fibrinogen levels)
- Usually, immunologic tests are negative.
- Hyperuricemia is present in up to 20% of patients with PsA
- Radiographic changes: X-Rays destruction and new bone formation, osteodestructive changes (erosions and resorption in mutilans)



## **Comorbidities-Liver Disease**

- Patients with psoriasis or PsA have a higher risk of nonalcoholic fatty liver disease (NAFLD)
- Pathologic mechanisms proposed- Insulin resistance may be a major link between the two conditions
- Methotrexate and leflunomide may cause liver enzymes abnormalities, and, in some cases, permanent liver damage and/or cirrhosis





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# Comorbidities as consequences of psoriatic disease

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#### **Comorbidities- Cardiovascular Disease**

- Cardiovascular disease is the leading cause of death among psoriatic patients.
  - Eular Recommendations for Cardiovascular Disease Risk Management in Patients with Rheumatoid Arthritis and Other Forms of Inflammatory Joint Disorders: 2015/2016 Update
  - Disease activity should be controlled optimally to reduce CVD risk in all patients with RA, AS, or PsA (Strength of recommendation B)
  - All patients with RA, AS, or PsA should be assessed for CVD at least once every 5 years and should be reevaluated after any major change in antirheumatic therapy (Strength of recommendation C)

## Role of Family Physician and when to refer

- Initial confirmation of diagnosis and management
- Monitoring for adverse effects
- Recognizing signs of inadequate control
- Persistent joint pain despite NSAIDs or csDMARDs
- Severe skin involvement or extra-articular manifestations
- Consideration of advanced biologic or targeted therapy

## **Goals of Therapy**

- Reduce joint inflammation and pain
- Prevent joint damage and deformity
- Improve quality of life
- Manage psoriasis symptoms

#### **PsA Management**

- Treat -to target approach optimal management of PsA
- Long-term remission appears possible in over 70% of patients with PsA
- Conventional synthetic DMRADs: methotrexate, leflunomide, sulfasalazine and Plaquenil (risk of Pso flare), apremilast
- TNF inhibitors (etanercept, infliximab, adalimumab, golimumab, certolizumab) efficacious and relatively safe for the treatment of peripheral arthritis and Pso and inhibit structural damage
- Targeted therapy against elements of the interleukin (IL)-17/IL-23 pathway appear to be efficacious in the management of Pso, peripheral arthritis, enthesitis, and dactylitis
- Janus Kinase (JAK) inhibitors by small molecules



## Conclusions

- Psoriatic arthritis is a complex disease
- Multiple inflammatory clinical domains, including peripheral joints, skin and nails, axial joints, entheses, eyes, and digits.
- Multiple therapeutic options including csDMARDs and bDMARDs such as anti TNFs, anti IL 17, IL 23
- Early assessment, diagnosis, and treat-to-target are key to the management of patients with PsA to facilitate the institution of appropriate therapy in a timely fashion.
- With the appropriate and timely use of current therapies and coordinated care with rheumatologists and dermatologists, it appears possible to prevent progressive structural damage in patients with PsA.
- Efforts are underway to further develop and validate biomarkers for diagnosis and outcome measures that will enable the appropriate use of increasingly effective novel therapies to control symptoms and disease activity, maintain functional ability, and improve QoL.

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