PATIENT INFORMATION | **RHEUMATOLOGY**

Understanding Psoriatic Arthritis

An evidence-based guide for patients in Pakistan — what it is, how it is diagnosed, and how modern treatment can protect your joints, skin, and quality of life.

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At a glance

0.1 – 0.2%	~20 – 30%	30 – 50	1 : 1
Worldwide population prevalence	Of psoriasis patients develop PsA	Typical age at diagnosis (years)	Affects men and women roughly equally

Psoriatic arthritis (PsA) is a chronic, immune-mediated inflammatory disease that can damage joints, tendons, and the spine. In approximately 70% of patients, the skin condition **psoriasis** appears first; in about 15%, joint disease precedes skin disease; and in the remainder both arise together. Early recognition matters — sustained inflammation can cause permanent joint erosion within the first two years of disease.

What is psoriatic arthritis?

PsA belongs to a family of conditions called the **spondyloarthropathies**. Unlike rheumatoid arthritis, PsA is **seronegative** — meaning that rheumatoid factor and anti-CCP antibodies are typically absent. PsA is heterogeneous: the same diagnosis may present very differently from one patient to another. Five recognised clinical patterns (Moll & Wright classification) are:

- **Asymmetric oligoarthritis** — fewer than five joints, often in the hands and feet.
- **Symmetric polyarthritis** — resembling rheumatoid arthritis but seronegative.
- **Distal interphalangeal (DIP) joint predominant** — the joints nearest the fingertips, often with adjacent nail disease.
- **Spondylitis** — inflammation of the spine and sacroiliac joints, causing inflammatory back pain.
- **Arthritis mutilans** — a rare, severely destructive form with telescoping of the digits.

Incidence and epidemiology

Globally, PsA affects approximately 0.1 – 0.2% of the adult population, with incidence rates between 3 and 41 cases per 100,000 person-years. Prevalence is highest in Europe and North America, and lower in Asian and South American populations. The mean global prevalence has been estimated at 112 per 100,000 adults (Scotti *et al.*, 2018; updated 2024). Among individuals with established psoriasis, the lifetime risk of developing PsA is approximately 20 – 30%.

South Asian and Pakistani context

Robust population-level data on psoriatic arthritis in Pakistan remain limited. The available evidence is summarised below:

• A hospital-based descriptive study at **Bahawalpur Victoria Hospital** (Ejaz *et al.*, *J Pak Assoc Dermatol* 2013) examined 117 adult psoriasis patients and found a significantly raised serum uric acid level associated with psoriatic joint involvement, alongside lower mean haemoglobin (13.25 g/dL) and a raised ESR (mean 22.6 mm/h).
• Regional reviews of arthritis in Pakistan and India report a male-to-female ratio for PsA of approximately **3:1**, with peak incidence in the **20 – 39 year** age group — younger than the global average.
• Spondyloarthritis as a whole, of which PsA is a major subtype, shows a lower reported prevalence in South and South-East Asia (0 – 0.19%) compared with European and Middle-Eastern populations.

Under-diagnosis is widely recognised in Pakistan due to limited rheumatology services, low public awareness, and frequent misattribution of joint pain to age, occupation, or osteoarthritis. Patients with psoriasis should be screened for joint symptoms at every review.

Who is at higher risk?

- **Existing psoriasis**, particularly **scalp psoriasis**, **intergluteal/perianal psoriasis**, and severe (PASI > 10) skin disease.
- **Nail involvement** — pitting, onycholysis, or subungual hyperkeratosis (strongest single skin predictor).
- **Family history** of psoriasis or PsA in a first-degree relative (40 – 50% of patients).
- **Obesity** — body mass index above 30 doubles the risk and worsens treatment response.
- **Mechanical trauma** to joints or entheses (the *deep Koebner phenomenon*).
- **Smoking** and recurrent streptococcal infection have also been implicated.

How does it develop? — the science explained simply

PsA is driven by a misdirected immune response in genetically predisposed individuals. Several **HLA** alleles (notably HLA-B*27, HLA-B*38, HLA-B*39, HLA-Cw*06, and HLA-B*08) increase susceptibility. When an environmental trigger — typically mechanical stress, infection, or trauma — provokes the immune system, the following sequence is thought to unfold:

Step	What happens in your body
1. Innate trigger	Innate immune cells in the skin, entheses, and gut release **interleukin-23 (IL-23)** and **interleukin-12 (IL-12)**.
2. T-cell activation	These cytokines drive naïve T-cells to differentiate into **Th17** and **Th1** helper cells.
3. Inflammatory cascade	Activated T-cells secrete **IL-17, IL-22**, and **tumour necrosis factor alpha (TNF-α)**, amplifying inflammation.
4. Tissue damage	TNF-α and RANKL activate **osteoclasts**, causing bone erosion; concurrent bone formation leads to the characteristic mix of erosion and new bone (periostitis, enthesophytes, ankylosis).
5. Targeted therapy	Modern biologic drugs are designed to interrupt this cascade at **TNF-α**, **IL-17**, and **IL-23** — the same molecules driving disease.

Understanding this pathway explains why some treatments work where others fail: PsA is not simply "wear and tear" — it is an immune disease, and it requires immune-directed treatment.

Symptoms — what to look out for

PsA may begin gradually and can be mistaken for an injury or wear-and-tear arthritis. The **five cardinal domains** are described below. You do not need to have all of them — many patients have only one or two.

Domain	Typical features
Peripheral arthritis	Pain, swelling, warmth, and stiffness in the joints of the hands, feet, wrists, knees, or ankles. Stiffness is typically **worse in the morning** and improves with movement (≥ 30 minutes).
Enthesitis	Inflammation where tendons or ligaments insert into bone. Common sites: **Achilles tendon, plantar fascia, lateral epicondyle (tennis elbow)**, and around the patella. Often misdiagnosed as sports injury.
Dactylitis ("sausage digit")	Uniform swelling of an entire finger or toe — a near-pathognomonic feature of PsA, present in 30 – 50% of patients at some point.
Axial disease (spondylitis)	Inflammatory back or buttock pain — **worse at rest, better with activity**, with prolonged early-morning stiffness. May involve the neck.
Skin and nail disease	Red, scaly plaques (psoriasis) — often on the scalp, elbows, knees, lower back, or in skin folds. Nail **pitting, onycholysis** (lifting), and ridging may be the only skin clue.

Beyond the joints — extra-articular features

PsA is a **systemic** disease. You should also be aware of:
- **Uveitis** (eye inflammation) — red, painful, light-sensitive eye, occurring in up to 25%.
- **Inflammatory bowel disease** — Crohn's disease and ulcerative colitis are more common.
- **Cardiovascular disease** — patients have a higher long-term risk of heart attack and stroke.
- **Metabolic syndrome** — obesity, type 2 diabetes, dyslipidaemia, and fatty liver disease.
- **Mental health** — depression, anxiety, and fatigue are common and deserve attention.

How is psoriatic arthritis diagnosed?

There is **no single diagnostic test** for PsA. Diagnosis is clinical, supported by blood tests and imaging. Your rheumatologist will take a detailed history, examine all your joints and skin, and apply the internationally accepted **CASPAR criteria**.

The CASPAR criteria (sensitivity 91.4%, specificity 98.7%)

You must have **inflammatory joint, spine, or entheseal disease**, plus at least **3 points** from the following:

Feature	Points
Current psoriasis (visible on examination)	**2**
Personal history of psoriasis (if no current skin disease)	1
Family history of psoriasis in a first- or second-degree relative	1
Typical psoriatic nail dystrophy (pitting, onycholysis, hyperkeratosis)	1
Negative rheumatoid factor	1
Current dactylitis, or history of dactylitis documented by a rheumatologist	1
Radiographic evidence of juxta-articular new bone formation (hands or feet)	1

Tests your rheumatologist may arrange

- **Blood tests:** full blood count, ESR, CRP (inflammation markers), rheumatoid factor and anti-CCP (typically negative in PsA), uric acid, urea and electrolytes, liver function, and HLA-B27 in selected cases.
- **X-rays** of the hands, feet, pelvis, and spine — looking for the characteristic combination of **erosion with new bone formation**, the "pencil-in-cup" deformity, and sacroiliitis.
- **Ultrasound** — sensitive for early synovitis, enthesitis, and dactylitis; increasingly used as a first-line modality in rheumatology clinics.
- **MRI** — particularly valuable for axial disease and early sacroiliitis when X-rays are normal.
- **Screening questionnaires** (PEST, EARP, ToPAS) are used in dermatology clinics to identify psoriasis patients who should be referred to rheumatology.

Treatment — a modern, target-driven approach

Treatment of PsA has been transformed over the past two decades. The current standard of care follows the **2023 EULAR** and **2021 GRAPPA** recommendations, using a **"treat-to-target"** strategy: the aim is **remission** or, if not achievable, **minimal disease activity**. Treatment is escalated stepwise until that target is reached, with regular monitoring every 1 – 3 months in active disease.

The stepped-care ladder

Tier	Class & examples	Role
1	**Lifestyle & non-drug:** weight reduction, smoking cessation, physiotherapy, occupational therapy, regular low-impact exercise, vaccination review.	Foundation of every treatment plan.
2	**NSAIDs:** e.g. naproxen, etoricoxib. **Local corticosteroid injections** for single inflamed joints or entheses.	Short-term symptom relief (≤ 4 weeks for NSAIDs in line with EULAR 2023).
3	**Conventional synthetic DMARDs (csDMARDs): Methotrexate** (first line), sulfasalazine, leflunomide, ciclosporin.	First-line for peripheral arthritis; methotrexate is also useful for skin disease. **Not effective** for spine or pure enthesitis.
4	**Biologic DMARDs (bDMARDs):** • **TNF-α inhibitors:** adalimumab, etanercept, infliximab, golimumab, certolizumab • **IL-17 inhibitors:** secukinumab, ixekizumab, bimekizumab • **IL-23 (p19) inhibitors:** guselkumab, risankizumab • **IL-12/23 inhibitor:** ustekinumab	Used when csDMARDs fail or are contraindicated. Choice is tailored to dominant disease domain (skin, axial, IBD, uveitis).
5	**Targeted synthetic DMARDs (tsDMARDs):** • **JAK inhibitors:** tofacitinib, upadacitinib • **PDE-4 inhibitor:** apremilast	Considered after biologic failure or where biologics are unsuitable. JAK inhibitors require careful assessment of cardiovascular, thrombotic, and malignancy risk.

What you can do yourself

- **Achieve and maintain a healthy weight** — even a 5 – 10% reduction improves joint symptoms, skin clearance, and the response to medication.
- **Stop smoking** — smoking reduces the efficacy of TNF inhibitors and accelerates joint damage.
- **Exercise regularly** — combining aerobic activity, strength work, and stretching protects joints; swimming and cycling are ideal.
- **Eat a Mediterranean-pattern diet** — rich in vegetables, fish, olive oil, and whole grains; evidence supports anti-inflammatory benefits.
- **Vaccinate appropriately** — annual influenza, pneumococcal, and (where indicated) COVID-19 and shingles vaccines, ideally before starting biologics.
- **Mental health matters** — depression and chronic pain are linked; do not hesitate to ask for help.

What is the outlook?

PsA is a **life-long condition**, but the outlook today is very different from a generation ago. With early diagnosis and modern biologic therapy, most patients can achieve sustained remission or low disease activity, preserve joint function, and lead an active life. Untreated or under-treated PsA, by contrast, can lead to permanent joint deformity, disability, and increased cardiovascular risk. **Time matters**: a diagnostic delay of more than 6 months from symptom onset is associated with worse long-term joint outcomes.

When to contact your rheumatologist promptly

- A new hot, swollen, or very painful joint.
- An entire finger or toe swelling like a sausage.
- A red, painful eye with blurred vision or sensitivity to light (possible uveitis — seek same-day care).
- Severe, persistent back or buttock pain that wakes you at night.
- Signs of infection while taking biologic therapy: fever, productive cough, persistent diarrhoea.
- Unintended weight loss, persistent fatigue, or new skin lesions.

Frequently asked questions

Question	Answer
Can psoriatic arthritis be cured?	No, but it can be very effectively **controlled**. Sustained remission is now an achievable goal for most patients.
Is it hereditary?	Genetics play a role — around 40% of patients have a relative with psoriasis or PsA — but environment and chance also contribute. Carrying a risk gene does not guarantee disease.
Will I need treatment forever?	Most patients require long-term treatment to prevent joint damage. In some cases of sustained remission, dose reduction may be considered under specialist supervision.
Are biologic injections safe?	When prescribed and monitored appropriately, biologics have an excellent safety record. Pre-treatment screening (TB, hepatitis B and C, full blood count) and ongoing review are essential.
Can I become pregnant?	Yes. PsA does not reduce fertility, and several treatments — including certolizumab — are considered safe in pregnancy. Plan ahead with your rheumatologist; methotrexate must be stopped well before conception.
Will diet alone control my disease?	A healthy weight and Mediterranean-pattern diet help, but they do not replace medication. Inflammation in PsA is driven by the immune system and almost always needs targeted treatment.

Key references and further reading

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Speak to a specialist

Early diagnosis changes the course of psoriatic arthritis — book a consultation today.

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Important disclaimer. This leaflet provides general information for patients and does not replace individual medical advice from a qualified rheumatologist. Treatment decisions must take into account your full medical history, examination findings, and investigations. Please discuss any concerns directly with Dr Saira Khan or your treating physician.

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