PATIENT INFORMATION | **RHEUMATOLOGY**

Understanding Gouty Arthritis

An evidence-based guide for patients in Pakistan — what causes it, how it is diagnosed, and how modern treatment can prevent attacks, dissolve crystals, and protect your joints.

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At a glance

1 – 4%	3 – 4 : 1	< 360 µmol/L	30.1%
Adult prevalence worldwide	Male-to-female ratio	Target serum urate level	Hyperuricaemia in Karachi adults

Gout is the **most common inflammatory arthritis** in adults. It is caused by the deposition of **monosodium urate (MSU) crystals** in joints and soft tissues, triggered by a sustained rise in blood uric acid (hyperuricaemia). Gout is now understood as a chronic, treatable metabolic disease — not simply a series of painful attacks. With modern urate-lowering therapy, gout can be effectively **cured** in the sense that crystal deposits dissolve and attacks stop entirely.

What is gout?

Uric acid is the end product of **purine** metabolism. In humans, purines come from two sources: dietary intake (especially red meat, organ meats, seafood, and alcohol) and from the natural breakdown of body cells. When blood urate exceeds its saturation point — approximately **404 µmol/L (6.8 mg/dL)** at body temperature — urate begins to crystallise in cooler, peripheral joints such as the great toe, ankle, and knee. These crystals trigger an explosive inflammatory response mediated by the **NLRP3 inflammasome** and **interleukin-1β**, producing the classical hot, red, exquisitely painful joint of an acute gout attack.

The four stages of gout

Stage	Description
1. Asymptomatic hyperuricaemia	Raised serum urate without symptoms. Most people with hyperuricaemia never develop gout, but the risk of a first attack rises sharply above 480 µmol/L (8 mg/dL).
2. Acute gout flare	Sudden, severe joint inflammation — most often the **first metatarsophalangeal joint** (podagra). Reaches peak intensity within 12–24 hours and resolves over 7–14 days even without treatment.
3. Intercritical gout	Symptom-free interval between attacks. Crystals continue to accumulate silently; without urate-lowering therapy, attacks recur with increasing frequency.
4. Chronic tophaceous gout	Visible deposits of urate crystals (**tophi**) in joints, tendons, and soft tissues (commonly elbows, fingers, helix of the ear). Joint destruction and disability ensue if left untreated.

Incidence and epidemiology

Gout affects approximately **1–4%** of adults worldwide and its prevalence has roughly doubled over the past three decades, driven by an ageing population, rising obesity, and the metabolic syndrome epidemic. Prevalence is highest in developed nations — **3.9% in the USA**, **5.2% in Australia**, **2.5% in the UK**, and over 6% in certain Pacific Island and indigenous populations. Men are affected three to four times more often than women, but the ratio narrows sharply after menopause, when the protective uricosuric effect of oestrogen is lost.

South Asian and Pakistani context

Published prevalence data on gout specifically in Pakistan remain limited, but the burden is substantial and almost certainly under-recognised:

• A cross-sectional study from **Karachi** (Mahmood *et al.*, *Cureus* 2019) of adult out-patients found **hyperuricaemia in 30.1%** — a high background population risk for future gout.
• Risk factors associated with raised urate in that cohort included **hypertension**, **diuretic use**, **frequent meat consumption**, **sedentary lifestyle**, and **elevated BMI**.
• A 2024 study at **Khyber Teaching Hospital, Peshawar** reported that **93%** of gout-cohort patients were aged 21–60 years, with **54%** reporting a positive family history.
• Regional comparative reviews place population gout prevalence in Bangladesh, India, and Pakistan at **under 0.5%** — but this almost certainly reflects under-diagnosis rather than true low burden, given the very high hyperuricaemia rates documented locally.

Pakistani diets rich in red meat, organ meats, lentils (daal), and sweetened beverages, combined with rising obesity and hypertension, create a population at high risk of gout. Early recognition and urate-lowering therapy can prevent decades of recurrent attacks and joint damage.

Who is at higher risk?

- **Male sex** and **age over 40**; in women, gout typically begins after the menopause.
- **Family history** — genetics influence urate transport (URAT1, GLUT9, ABCG2 variants) and account for around 60% of population variation in serum urate.
- **Obesity and metabolic syndrome** — central obesity, type 2 diabetes, hypertension, and dyslipidaemia cluster strongly with gout.
- **Chronic kidney disease** — even mild renal impairment reduces urate excretion.
- **Diet** — red meat, organ meats (liver, kidney, brain), shellfish, beer and spirits, and high-fructose sweetened beverages.
- **Medications** — thiazide and loop diuretics, low-dose aspirin, ciclosporin, tacrolimus, and pyrazinamide.
- **Dehydration**, fasting, crash diets, and surgical stress can all precipitate an attack.

How does gout develop? — the science explained simply

Gout is fundamentally a disorder of **urate handling**. In most patients (around 90%), the kidneys are **under-excreting** urate; in a minority, the body is **over-producing** it. The disease then unfolds in five linked steps:

Step	What happens in your body
1. Hyperuricaemia	Serum urate rises above the saturation threshold of about **404 µmol/L (6.8 mg/dL)**. This may be silent for many years.
2. Crystal formation	Monosodium urate crystallises in **cooler, peripheral joints** (great toe, foot, ankle) and around tendons. Crystals can also seed kidneys, causing urate stones.
3. Inflammasome activation	Resident macrophages engulf crystals; this activates the **NLRP3 inflammasome**, releasing **interleukin-1β (IL-1β)** and recruiting a flood of neutrophils.
4. Acute flare	Within hours, the affected joint becomes swollen, red, hot, and exquisitely painful — the classical acute gout attack.
5. Chronic damage	Repeated, untreated attacks lead to **tophus** formation, bone erosion, secondary osteoarthritis, urate kidney stones, and accelerated cardiovascular disease.

*Understanding this pathway explains the modern treatment strategy: **lower the urate to below saturation, and crystals dissolve.** This is the only way to truly cure gout.*

Symptoms — what to look out for

The presentation of gout is characteristic and is usually recognised on clinical grounds alone. Three patterns predominate:

Pattern	Typical features
Acute monoarthritis	Sudden onset — often overnight — of severe pain, swelling, redness, and warmth in a **single joint**. The **great toe** (podagra) is affected in approximately 50% of first attacks; the ankle, mid-foot, and knee are next most common. Pain peaks at 12–24 hours; even bedsheets may be intolerable.
Polyarticular gout	More than one joint affected simultaneously — seen in longstanding disease, post-menopausal women, transplant patients, and those on diuretics. May mimic rheumatoid or septic arthritis.
Chronic tophaceous gout	Firm, painless, chalky nodules (**tophi**) develop over years on the helix of the ear, olecranon bursa, Achilles tendon, finger pads, or in the fingers and toes. They may ulcerate and discharge white, toothpaste-like material.

Common triggers of an acute attack

Gout flares are often precipitated by a recognisable event. Common triggers include:
- A heavy meat- or seafood-based meal, particularly at weddings or feasts.
- Alcohol — especially beer and spirits; even moderate intake can trigger an attack.
- Dehydration, fasting (including Ramadan), or a sudden crash diet.
- Starting or stopping urate-lowering therapy without anti-inflammatory cover.
- Acute illness, infection, surgery, or trauma to a joint.
- Starting a thiazide diuretic, loop diuretic, or low-dose aspirin.

Gout is more than a joint disease

Patients with gout have a significantly increased long-term risk of **hypertension**, **chronic kidney disease**, **urate kidney stones**, **type 2 diabetes**, **non-alcoholic fatty liver disease**, and **cardiovascular events** — including myocardial infarction and stroke. Treating gout therefore protects not only joints but also the kidneys, heart, and life expectancy.

How is gout diagnosed?

The diagnostic **gold standard** remains the identification of **negatively birefringent, needle-shaped MSU crystals** in synovial fluid or tophus aspirate under polarised-light microscopy. In practice, however, many cases are diagnosed clinically using the **2015 ACR/EULAR classification criteria**, supported by laboratory and imaging investigations.

Investigations your rheumatologist may arrange

- **Serum uric acid** — should ideally be measured between attacks, as levels may transiently fall during an acute flare and give a misleading result.
- **Joint fluid aspiration** — the definitive test; also excludes septic arthritis, which can mimic gout and is a medical emergency.
- **Full blood count, ESR, CRP** — markers of systemic inflammation.
- **Renal function (urea, creatinine, eGFR)** — guides drug selection and dosing.
- **Fasting lipids, glucose, HbA1c, blood pressure** — to screen for the metabolic syndrome.
- **Plain X-rays** — late changes include "punched-out" peri-articular erosions with overhanging edges, characteristic of chronic gout.
- **Musculoskeletal ultrasound** — increasingly used; shows the highly specific **"double-contour sign"** (urate crystal deposition on cartilage) and tophi.
- **Dual-energy CT (DECT)** — when available, can directly visualise urate deposits and is particularly useful in atypical cases.

Treatment — a modern, two-pronged approach

Modern gout care follows the **2020 American College of Rheumatology** and **2016/2024 EULAR** guidelines. Treatment has **two distinct goals**: (i) settling the **acute flare** as quickly as possible, and (ii) achieving long-term **urate lowering** to dissolve crystal deposits and prevent recurrence. Both are essential — treating only the attack but not the underlying urate is the commonest reason gout becomes chronic and disabling.

Part 1 — Treating the acute attack

Option	Notes
NSAIDs (e.g. naproxen 500 mg twice daily, indometacin, etoricoxib)	First-line in patients without renal, gastric, or cardiovascular contraindications. Co-prescribe a proton-pump inhibitor.
Colchicine 1 mg followed by 0.5 mg one hour later, then 0.5 mg twice or three times daily until resolution	Most effective when started within 12–24 hours of onset. Reduce dose in renal impairment and avoid with strong CYP3A4 / P-glycoprotein inhibitors (e.g. clarithromycin, ciclosporin).
Oral glucocorticoids (prednisolone 30–40 mg daily, tapered over 5–10 days)	Excellent option in patients in whom NSAIDs and colchicine are contraindicated — particularly the elderly and those with CKD.
Intra-articular steroid injection	Highly effective for a single large joint once septic arthritis has been excluded.
IL-1 inhibitors (anakinra, canakinumab)	Reserved for severe, refractory flares or where all conventional options are contraindicated.

Part 2 — Long-term urate-lowering therapy (ULT)

Urate-lowering therapy is the only treatment that **changes the course** of gout. It is indicated for any patient with: **two or more flares per year**, **tophi**, evidence of joint damage on imaging, recurrent urate kidney

stones, or chronic kidney disease. The treatment target is a serum urate **below 360 µmol/L (6 mg/dL)**, or **below 300 µmol/L (5 mg/dL)** in severe or tophaceous disease.

Drug class	Examples & notes
Xanthine oxidase inhibitors (first-line)	**Allopurinol** — start 100 mg daily (50 mg in CKD), titrate by 100 mg every 2–4 weeks to target. Usual effective dose 300–600 mg/day. **Febuxostat** — start 40 mg daily, increase to 80–120 mg as needed. Useful in allopurinol intolerance or moderate-to-severe CKD.
Uricosuric agents	**Probenecid**, **benzbromarone**. Increase renal excretion of urate; require good hydration and adequate renal function (eGFR > 30 mL/min).
Recombinant uricase	**Pegloticase** — reserved for severe, refractory tophaceous gout when all other options have failed.
Flare prophylaxis	Low-dose colchicine 0.5 mg once or twice daily (or low-dose NSAID) is co-prescribed for the **first 3–6 months** of urate-lowering therapy to prevent paradoxical flares as crystals dissolve.

Important: HLA-B*5801 and allopurinol safety

A small proportion of South Asian patients carry the **HLA-B*5801** gene variant, which is associated with a rare but life-threatening hypersensitivity reaction to allopurinol (**SCAR/DRESS syndrome**). The American College of Rheumatology recommends that **HLA-B*5801 testing be considered before starting allopurinol** in patients of South Asian and African descent. Where testing is unavailable, **start at a low dose (50–100 mg daily)** and titrate slowly, and seek urgent medical advice at the first sign of rash, fever, or oral ulceration.

What you can do yourself

- **Drink plenty of water** — at least 2 litres daily, more in hot weather, to support renal urate excretion and prevent stones.
- **Lose weight gradually** if overweight — crash dieting can *precipitate* attacks, but steady weight loss meaningfully reduces serum urate.
- **Limit red meat, organ meats (liver, kidney, brain), and shellfish.**
- **Avoid alcohol**, particularly beer and spirits; wine in moderation is less problematic.
- **Avoid sugar-sweetened beverages and fruit juices** high in fructose, including many local soft drinks and energy drinks.
- **Low-fat dairy, cherries, coffee, and vitamin C** are associated with modest reductions in serum urate.
- **Review your medications** with your doctor — thiazides, loop diuretics, and low-dose aspirin can all raise urate and may be substitutable.
- **Take your urate-lowering treatment every day**, even when you feel well — stopping it is the commonest cause of attacks returning.

What is the outlook?

Gout is one of the few chronic rheumatic diseases that can be **completely controlled, and effectively cured**, by sustained treatment. With consistent urate-lowering therapy to target, crystal deposits gradually dissolve, attacks stop entirely, tophi shrink and disappear, and the joint damage stabilises. The challenge is one of **adherence**: gout is a lifelong condition and treatment must continue indefinitely. Patients who stop their medication almost always relapse. Recognising gout as a serious metabolic disease — not just a nuisance attack — and treating the urate aggressively also reduces the long-term risk of kidney disease,

hypertension, and cardiovascular events.

When to contact your rheumatologist promptly

- A new hot, swollen, very painful joint — especially with fever (to exclude infection).
- A flare lasting longer than 7–10 days despite treatment.
- Frequent attacks (more than 2 per year), or new tophi appearing.
- Severe loin pain or blood in the urine — possible urate kidney stone.
- A new rash, fever, mouth ulcers, or facial swelling after starting allopurinol — **stop the drug immediately and seek urgent medical care**.
- Side effects of colchicine: severe diarrhoea, vomiting, or muscle weakness.

Frequently asked questions

Question	Answer
Can gout be cured?	Yes — uniquely among inflammatory arthritides, sustained urate lowering can dissolve crystals and stop attacks entirely. However, the underlying tendency persists, so treatment must continue lifelong.
I feel fine between attacks. Do I still need daily medication?	Yes. Crystals continue to accumulate silently in your joints during symptom-free periods. Daily urate-lowering therapy is what prevents future attacks and joint damage.
Will diet alone control my gout?	For most patients, no. Diet typically reduces serum urate by only 60–90 µmol/L (1–1.5 mg/dL) — rarely enough to reach target on its own. Diet supports medication; it does not replace it.
Is allopurinol safe long-term?	Yes, for the vast majority of patients. The main risk is a rare hypersensitivity reaction in the first few weeks, particularly in HLA-B*5801 carriers. Starting low and titrating slowly minimises this risk.
Can I drink alcohol if I have gout?	Beer and spirits markedly increase the risk of attacks and should be avoided. Wine in moderation has a smaller effect. Best practice is abstinence during active disease and at most modest intake once urate is controlled.
Is fasting in Ramadan safe with gout?	Most stable patients on urate-lowering therapy can fast safely, but adequate hydration between iftar and suhoor is essential, and very high-purine iftar meals should be avoided. Discuss the timing of your medication with your rheumatologist.

Key references and further reading

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Speak to a specialist

Gout is treatable, and modern therapy can stop attacks for good — 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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