

Gout

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THE STORY IN ONE SENTENCE

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“Gout happens when uric acid (a waste product from breaking down purines) builds up so high that it turns into monosodium urate crystals — tiny needle-shaped pieces that stab the inside of joints and trigger a furious inflammatory attack.”

Normal anatomy — the body's job

Your body constantly breaks down purines — building blocks found in red meat, organ meat, seafood, beer, and your own cells — into uric acid, a waste product. Normally, about two-thirds of uric acid leaves through your kidneys in urine, and one-third leaves through your intestines in stool. This keeps blood uric acid (serum urate) safely below 6–7 mg/dL. At that level, uric acid stays dissolved in blood like salt in water.

BODY'S JOB

The kidneys filter uric acid out of blood, and the gut secretes some into stool. This balance keeps uric acid low enough that it never turns solid. Think of it like sugar in iced tea — stir in a little and it dissolves; add too much or cool it down, and crystals form at the bottom.

1. Purines (from food like red meat, shellfish, alcohol, or from your own cells dying naturally) enter the body.
2. Cells break purines down into uric acid using an enzyme called xanthine oxidase.
3. Uric acid travels through the bloodstream dissolved in liquid.
4. The kidneys filter about two-thirds of uric acid out into urine and flush it out.
5. The intestines secrete the remaining one-third into stool.
6. Blood uric acid stays stable below 6–7 mg/dL, well under the solubility limit (the point where it would turn solid).

What breaks

Gout happens when uric acid in the blood climbs so high that it crosses the solubility threshold — the point where it can no longer stay dissolved — and crystallizes into needle-shaped monosodium urate crystals inside joints, especially the big toe.

CLINICAL TERM

Hyperuricemia (serum urate above 6.8 mg/dL, the saturation point) leads to monosodium urate crystal deposition in synovial joints, bursae, and soft tissue. Crystals trigger acute inflammatory arthritis via NLRP3 inflammasome activation in macrophages, releasing interleukin-1 β and driving neutrophil infiltration.

How the body compensates

The body has no effective compensatory mechanism to dissolve urate crystals once they form — the inflammatory response is an attempt to clear the foreign material but actually causes the pain and swelling. Vital signs often stay normal during an acute flare because gout is a local joint problem, not a systemic collapse, but infection must always be ruled out.

1. {'step': 'The immune system tries to clear crystals', 'plain': 'Macrophages (a type of white blood cell) swallow the needle-shaped crystals to remove them, but the sharp edges rupture the cell, spilling out inflammatory chemicals like interleukin-1 β that call in more immune cells and amplify swelling and pain.', 'why': 'This is the body's attempt to wall off and eliminate a foreign invader, but it backfires — the inflammatory cascade is what produces the classic red, hot, swollen, exquisitely tender joint.'}
2. {'step': 'Vital signs remain stable during acute flare', 'plain': 'Heart rate, blood pressure, and oxygen levels usually stay normal because gout affects a single joint, not the whole body's circulation or oxygen delivery.', 'why': 'This stability is deceptive — it can make the team underestimate the severity of pain and the urgent need to rule out septic arthritis, which can look identical but is life-threatening.'}
3. {'step': 'No true compensation for chronic hyperuricemia', 'plain': 'The body cannot increase uric acid excretion enough to catch up once production or retention is broken, so uric acid keeps climbing unless diet, medications, or kidney function changes.', 'why': 'Unlike glucose or sodium, there is no hormonal feedback loop to dump excess uric acid — the kidneys are already working at capacity, so the problem persists and crystals keep forming.'}

Domino effect

1. Uric acid climbs above the solubility threshold (6.8 mg/dL) — Either too much purine breakdown (from diet, alcohol, or cell death) or too little kidney excretion (from kidney disease, diuretics, or dehydration) pushes blood uric acid past the point where it can stay dissolved, like adding too much sugar to cold tea.
2. Needle-shaped monosodium urate crystals form in the joint space, especially the big toe — Uric acid solidifies into sharp crystals in cooler, more acidic areas like the first metatarsophalangeal joint (big toe), where lower temperature reduces solubility even further and minor trauma seeds crystal formation.
3. Crystals land on the synovial membrane (joint lining) and are eaten by macrophages — The immune system recognizes the crystals as foreign invaders, so macrophages (cleaning cells) try to swallow and break them down, but the sharp needle edges pierce and rupture the macrophage from the inside.
4. Ruptured macrophages spill interleukin-1 β and activate the NLRP3 inflammasome — When the macrophage bursts, it releases interleukin-1 β (a powerful alarm chemical) that triggers a massive inflammatory response — calling in neutrophils (another type of white blood cell) and flooding the joint with inflammatory proteins.
5. Neutrophils swarm the joint, releasing enzymes and cytokines that cause redness, heat, swelling, and severe pain — This is the acute gout flare — the joint becomes red, hot, swollen, and exquisitely tender (often can't even touch a bedsheet) because neutrophils are dumping tissue-damaging enzymes to kill what they think is an infection.
6. Flare resolves over days to weeks as the inflammatory response winds down, but crystals remain — The body eventually clears some crystals and the immune response exhausts itself, but if blood uric acid stays high, crystals keep forming and the cycle repeats, leading to more frequent flares.
7. Chronic high uric acid deposits form tophi (lumpy urate collections) in joints, tendons, soft tissue, and kidneys — Repeated crystal deposition over months to years builds visible chalky nodules that slowly destroy cartilage and bone, deform joints, and clog kidney tubules, forming uric acid kidney stones and causing chronic kidney disease (urate nephropathy).
8. Joint damage, chronic pain, and kidney impairment become permanent without urate-lowering therapy — None of this is random — each domino falls because of the one before, all tracing back to the upstream break: blood uric acid concentration staying above the point where the body can keep it dissolved.

Symptoms · what the patient feels, and why

- Sudden, severe pain in one joint — often the big toe, ankle, or knee — that comes on at night or early morning: Uric acid crystals (sharp, needle-like pieces) have settled into the joint fluid. The body's immune cells rush in to attack them, releasing chemicals that cause swelling and pain. At night, body temperature drops slightly and fluid drains from joints, making the uric acid more likely to form crystals.
- The joint becomes hot, red, and swollen; the skin may look shiny and tight: The immune system sends white blood cells and fluid to the joint to fight the crystals, causing inflammation (the body's alarm response). Blood vessels widen to bring more defenders, which makes the skin red and warm. Fluid leaks into the joint, stretching the skin and making it look shiny.
- The joint is so tender that even a bedsheet touching it can hurt: Nerve endings in the inflamed joint are extremely sensitive. The inflammation chemicals (like prostaglandins and bradykinin) turn the pain dial way up, so even light touch feels unbearable.
- Pain and swelling usually peak within 12 to 24 hours: The immune attack reaches full force as more white blood cells arrive and release more inflammatory chemicals. After a day or so, the body starts to clean up the crystals and the attack begins to calm down.
- Attacks are triggered by alcohol (especially beer), red meat, shellfish, sugary drinks, water pills (diuretics), hospital stay, surgery, or starting uric-acid-lowering medicine: These triggers either raise uric acid levels quickly (alcohol, red meat, shellfish, sugary drinks block the kidneys from clearing uric acid; surgery and illness cause cells to break down, releasing purines that turn into uric acid) or cause sudden shifts in uric acid that shake crystals loose (starting uric-acid medicine drops levels fast, destabilizing old crystals). Dehydration during hospital stays concentrates uric acid in the blood.
- Firm, chalky lumps (tophi) under the skin near joints or on the ears: When uric acid stays high for years, crystals clump together into lumps called tophi (TOE-fie). The body walls them off with scar tissue. Tophi show that gout has been untreated for a long time.
- Fever and feeling generally unwell during a severe attack: The immune system's inflammatory chemicals (cytokines like interleukin-1 and tumor tissue death (necrosis) factor) signal the brain's hypothalamus (the body's thermostat) to raise body temperature and trigger fatigue, part of the body's defense response.
- Multiple joints flaring at once, or an attack that won't go away: This can mean gout is very advanced with widespread crystal deposits, or it may signal a dangerous infection (septic arthritis) or another inflammatory disease. The immune system is overreacting or fighting something more serious than crystals.

Focused exam

- Red, hot, swollen joint — usually the big toe (first MTP joint, where the big toe meets the foot): This is the hallmark of gout. When needle-shaped uric acid crystals land inside the joint, the body sees them as invaders and rushes white blood cells to attack them. That causes huge inflammation — redness, heat, and swelling all at once. If you see one joint suddenly blow up overnight in the lower leg or foot, think gout first.
- Joint is so tender the patient cannot bear weight or let bedsheets touch it: Gout pain is dramatic and out of proportion to what you would expect from a simple sprain or bump. The crystals trigger an alarm in the nerves and the immune system pours in chemicals that amplify pain. This extreme tenderness tells you the inflammation inside is severe.
- Tophi — hard, bumpy deposits under the skin on ears, fingers, elbows, or feet (if chronic gout): These are piles of uric acid crystals that built up over years because blood urate stayed high. Tophi tell you the patient has had poorly controlled gout for a long time and the crystals have started depositing in soft tissue, not just joints.
- History of triggers: alcohol binge (especially beer), red meat or organ meat meal, high-fructose drinks, diuretic use, or recent surgery or illness: All of these raise uric acid or lower how much the kidneys can flush out. Alcohol and fructose both push the liver to make more urate. Diuretics make the kidneys hold onto urate instead of peeing it out. Surgery and illness cause cell breakdown, which releases purines (the building blocks of urate). Gout flares are not random — they follow a cause. Asking about triggers helps you trace back to what tipped the balance.
- Kidney function (serum creatinine, estimated GFR) and history of kidney stones: Too much uric acid damages kidneys in two ways: crystals can form stones that block the tubes that drain pee, and chronic crystal deposits can scar the kidney tissue itself (called urate nephropathy). This is why gout is not just about the toe — it is a whole-body urate problem. Knowing kidney function also tells you which urate-lowering drugs are safe to use.

Diagnostics · every test answers a question

- Arthrocentesis (joint aspiration) with synovial fluid analysis under polarized microscopy → Is this truly gout, and is there infection hiding inside the joint?
- Serum uric acid (urate) level → Is the patient's baseline urate high enough to form crystals?
- Serum creatinine and estimated glomerular filtration rate (eGFR) → Are the kidneys healthy enough to clear urate and handle urate-lowering drugs?
- 24-hour urine uric acid (optional, if considering probenecid or recurrent stones) → Is the patient an overproducer of urate or an underexcreter?
- X-ray or ultrasound of the affected joint (if diagnosis is uncertain or to look for chronic damage) → Are there signs of chronic gout or other joint disease?

- Complete blood count (CBC) and inflammatory markers (ESR, CRP) if infection is a concern
→ Is there infection or another cause of inflammation?

Treatment logic

- Colchicine (1.2 mg, then 0.6 mg one hour later, then 0.6 mg once or twice daily), NSAIDs (indomethacin 50 mg three times daily or naproxen 500 mg twice daily), or corticosteroids (prednisone 30–40 mg daily for 5 days, or intra-articular injection) → Stops the immune system's attack on the uric acid crystals inside the joint. Colchicine blocks neutrophils (the white blood cells that swarm the crystals). NSAIDs block prostaglandins (chemicals that cause pain and swelling). Steroids shut down the whole inflammatory cascade. All three shrink the swelling, redness, and pain during an acute flare. This is the fire extinguisher — it puts out the flare fast.
- Allopurinol (start 100 mg daily, increase slowly to 300–800 mg daily, target urate < 6 mg/dL) or febuxostat (start 40 mg daily, increase to 80 mg if needed) → Blocks xanthine oxidase, the enzyme that converts purines (from food and dying cells) into uric acid. This cuts down how much uric acid the body makes, so blood urate drops below the threshold where crystals form (6.8 mg/dL). Over months, existing crystals dissolve. This fixes the root problem. Allopurinol is first choice (cheaper, long track record). Febuxostat is second choice (more potent, safe if patient had mild allopurinol rash, but slight cardiovascular risk in some studies).
- Probenecid (start 250 mg twice daily, increase to 500–1000 mg twice daily) → Blocks the kidney tubes from reabsorbing uric acid, so more urate gets flushed out in the pee and blood levels drop. This works best if the kidneys are healthy (eGFR > 50) and the patient is an underexcreter (most gout patients are). Must drink plenty of water to dilute the urine and prevent kidney stones.
- Preventive (Prophylactic) colchicine (0.6 mg once or twice daily) or low-dose NSAID (naproxen 250 mg twice daily) for 3 to 6 months when starting or adjusting urate-lowering therapy → Prevents flares during the risky months when you are lowering urate and dissolving crystals. As blood urate drops, crystals start to break up and shed into the joint, which can trigger new flares (paradoxical flares). Prevention (Prophylaxis) keeps the immune system calm during this remodeling period. ACR and EULAR guidelines both recommend this.

- Lifestyle changes: cut alcohol (especially beer and liquor), avoid purine-rich foods (red meat, organ meat, shellfish, high-fructose corn syrup drinks), lose weight if overweight, drink 2+ liters of water daily, avoid dehydration → Reduces the purine load entering the body (less raw material to make uric acid) and improves kidney urate excretion (better hydration helps kidneys flush urate, weight loss improves insulin resistance which helps kidneys work better). Tackles both sides of the imbalance: less urate made, more urate cleared. Can lower urate by 1 to 2 mg/dL in some patients.
- Pegloticase (IV infusion 8 mg every 2 weeks) → An enzyme (PEGylated uricase) that breaks down uric acid directly in the bloodstream into allantoin, a harmless waste product that the kidneys easily pee out. Urate drops very fast (often to near zero). Used only for severe refractory gout when allopurinol, febuxostat, and probenecid have all failed and the patient has disabling tophi or frequent flares.

Medications

Colchicine · Anti-inflammatory (microtubule inhibitor)

Colchicine stops white blood cells from swarming into the joint and releasing chemicals that cause pain and swelling. It works by jamming the cell's internal scaffolding (microtubules) so the cells cannot move or dump their inflammatory cargo.

1. Urate crystals sit in the joint and trigger an alarm.
2. Neutrophils (white blood cells) try to swallow the crystals and release chemicals (like interleukin-1) that cause pain and swelling.
3. Colchicine binds to tubulin (the building block of the cell's skeleton) and stops the neutrophil from moving and releasing those chemicals.
4. Fewer neutrophils reach the joint, so the flare calms down.

SAFETY WATCH

Diarrhea, nausea, and belly cramps are common—start with a low dose (0.6 mg once or twice daily for a flare, NOT the old high-dose regimen). · Do NOT use with strong CYP3A4 or P-glycoprotein inhibitors (like clarithromycin, ritonavir) in people with kidney or liver disease—can cause life-threatening toxicity. · Reduce dose if kidneys are weak (CrCl under 30 mL/min) or avoid if on dialysis. · Watch for muscle pain or weakness—rare but serious sign of muscle breakdown (rhabdomyolysis), especially with statins.

NSAIDs (e.g., indomethacin, naproxen, ibuprofen) · Nonsteroidal anti-inflammatory drugs

NSAIDs block an enzyme called COX (cyclooxygenase) that makes prostaglandins—chemicals that turn up pain, heat, and swelling in the joint. By shutting down prostaglandin production, NSAIDs cool the flare.

1. Urate crystals trigger white blood cells to release prostaglandins and other inflammatory signals.
2. Prostaglandins widen blood vessels, increase fluid leakage into the joint, and sensitize pain nerves.
3. NSAIDs block COX-1 and COX-2 enzymes so prostaglandins cannot be made.
4. Less prostaglandin means less swelling, heat, and pain in the joint.

SAFETY WATCH

Can cause stomach ulcers and bleeding—use the lowest dose for the shortest time; consider a proton-pump inhibitor (PPI) if you have ulcer history. · Can raise blood pressure and worsen heart failure—avoid in people with heart disease or uncontrolled high blood pressure (hypertension). · Can harm kidneys, especially in older adults, people with chronic kidney disease, or those taking diuretics—check kidney function. · Avoid in people with aspirin allergy or asthma triggered by NSAIDs. · Indomethacin can cause more CNS side effects (dizziness, confusion) in older adults.

Corticosteroids (e.g., prednisone, methylprednisolone; or intra-articular triamcinolone) · Corticosteroid (broad anti-inflammatory)

Steroids turn down the entire inflammatory alarm system. They enter immune cells and flip genetic switches that stop production of inflammatory chemicals (cytokines, prostaglandins) and keep white blood cells from swarming the joint.

1. Urate crystals activate immune cells to release waves of inflammatory signals.
2. Steroids cross into the cell and bind to receptors in the cytoplasm.
3. The steroid-receptor complex moves into the nucleus and changes gene activity—blocking genes that make inflammatory chemicals and turning on genes that calm inflammation.
4. White blood cells make fewer attack chemicals, blood vessels leak less fluid, and the joint cools down.

SAFETY WATCH

Short courses (3–7 days) for a gout flare are generally safe, but watch blood sugar in people with diabetes. · Can worsen infections—avoid if active infection is suspected. · Long-term use (not typical for gout flares) can cause bone thinning, weight gain, high blood pressure, and mood changes. · Injecting a joint is safe if you are sure it is gout and NOT joint infection (septic arthritis)—never inject an infected joint.

Allopurinol · Xanthine oxidase inhibitor

Allopurinol blocks xanthine oxidase, the enzyme in the liver that turns purines (from food and dying cells) into uric acid. Less uric acid is made, so blood levels drop and crystals slowly dissolve.

1. Your body breaks down purines (from meat, seafood, beer, and your own cells) into hypoxanthine, then xanthine.
2. Xanthine oxidase is the enzyme that converts xanthine into uric acid.
3. Allopurinol (and its active form, oxypurinol) jams into the xanthine oxidase enzyme and blocks it.
4. Xanthine and hypoxanthine build up a little (they are more soluble and harmless), and uric acid production drops.
5. Blood uric acid falls below 6 mg/dL (below 5 mg/dL if you have tophi), and crystals in joints slowly dissolve.

SAFETY WATCH

Start low (50–100 mg daily) and titrate up every 2–5 weeks to reach target uric acid—rushing the dose can trigger flares. · Use preventive (prophylactic) colchicine (0.6 mg once or twice daily) or low-dose NSAID for the first 3–6 months to prevent flares when you start or increase the dose. · Rare but serious: allopurinol hypersensitivity syndrome (fever, rash, liver and kidney damage, often fatal)—more common in people with the HLA-B*5801 gene variant (test Asian patients, especially Han Chinese, Thai, and Korean, before starting). · Stop immediately if rash appears. · Reduce dose in chronic kidney disease (CKD), but do NOT avoid allopurinol in CKD—dose can be adjusted safely. · Can interact with azathioprine and 6-mercaptopurine (increases toxicity)—reduce dose of those drugs.

Febuxostat · Xanthine oxidase inhibitor (non-purine analog)

Febuxostat also blocks xanthine oxidase, stopping the liver from making uric acid. It is chemically different from allopurinol and works well even in mild-to-moderate kidney disease.

1. Purines are broken down into xanthine.

2. Xanthine oxidase would normally turn xanthine into uric acid.
3. Febuxostat binds tightly to xanthine oxidase and blocks it.
4. Uric acid production drops, blood levels fall, and crystals dissolve over time.

SAFETY WATCH

Start at 40 mg daily, increase to 80 mg if uric acid target is not reached after 2–4 weeks. · Use flare prevention (prophylaxis) (colchicine or low-dose NSAID) for 3–6 months when starting. · FDA boxed warning: may increase risk of cardiovascular death compared to allopurinol in people with heart disease—use with caution in patients with coronary artery disease or heart failure; consider allopurinol first. · Liver enzyme elevation can occur—check liver tests at baseline and periodically. · Does NOT require HLA-B*5801 testing. · Safe in mild-to-moderate kidney disease without dose adjustment.

Probenecid · Uricosuric agent

Probenecid blocks the kidney's reabsorption pumps (urate transporters) so more uric acid leaves the body in the urine instead of being pulled back into the blood.

1. Your kidneys filter uric acid out of the blood into the urine.
2. Normally, transporter proteins (URAT1 and others) in the kidney tubule pull about 90% of that uric acid back into the blood.
3. Probenecid blocks those transporters.
4. More uric acid stays in the urine and leaves the body.
5. Blood uric acid levels drop, and crystals dissolve.

SAFETY WATCH

Start 250 mg twice daily, increase to 500 mg twice daily after one week, max 2–3 g/day in divided doses. · Use flare prevention (prophylaxis) (colchicine or low-dose NSAID) for 3–6 months. · Drink plenty of water (at least 2 liters/day) to prevent kidney stones. · Do NOT use if you have a history of kidney stones or CrCl under 50 mL/min. · Do NOT use if you are an overproducer of uric acid (24-hour urine uric acid over 800 mg—increases stone risk). · Can reduce excretion of other drugs (e.g., penicillin, methotrexate)—watch for interactions. · Rarely causes rash or stomach upset.

Pegloticase · Recombinant uricase enzyme (PEGylated)

Pegloticase is a lab-made version of uricase (an enzyme humans lost through evolution) attached to polyethylene glycol (PEG) to make it last longer. It directly breaks down uric acid in the blood into allantoin, which dissolves easily and leaves in the urine.

1. Most mammals have uricase, which breaks uric acid into allantoin (very soluble, harmless).

2. Humans lost the gene for uricase millions of years ago, so our uric acid can build up.
3. Pegloticase is infused into the bloodstream and acts like the missing enzyme.
4. It converts uric acid into allantoin.
5. Allantoin dissolves in the blood and kidneys flush it out.
6. Blood uric acid drops dramatically (often to near zero), and tophi shrink rapidly.

SAFETY WATCH

Given as IV infusion every 2 weeks (8 mg in 250 mL over at least 2 hours) in a monitored setting. · Reserved for people with severe tophaceous gout who have failed or cannot tolerate xanthine oxidase inhibitors. · High risk of infusion reactions (flushing, chest discomfort, hives, shortness of breath, anaphylaxis)—premedicate with antihistamines and corticosteroids. · Check uric acid level before EVERY infusion—if it rises above 6 mg/dL, the drug is likely not working (antibodies have formed), and continuing increases reaction risk; consider stopping. · Use flare prevention (prophylaxis) (colchicine ± low-dose NSAID or prednisone) starting at least 1 week before first infusion and continue for at least 6 months. · Avoid in people with G6PD deficiency (can cause severe breakdown of red blood cells)—test before starting. · Do NOT give with other uric-acid-lowering drugs during pegloticase therapy (can interfere with monitoring).

Red flags

- Fever above 100.4°F (38°C) with joint pain — could be a joint infection, not gout
- Joint pain in more than two joints at once, or pain that spreads quickly
- Severe pain that doesn't improve at all after three days of treatment
- Red, hot, swollen joint in someone with a weakened immune system (on steroids, chemotherapy, or HIV)

Nursing priorities

1. Check temperature, heart rate, and blood pressure, and look at the affected joint for extreme redness, warmth, and swelling — Septic arthritis (joint infection) looks like gout but causes fever and very angry-looking joints; it needs emergency joint fluid removal and IV antibiotics within hours or the infection destroys cartilage and can spread to the bloodstream causing sepsis
2. Give the ordered anti-inflammatory medicine (colchicine, NSAID, or steroid) as soon as possible, ideally within 24 hours of symptom start — Gout pain comes from white blood cells swarming the uric acid crystals and releasing inflammatory chemicals; early treatment stops this cascade faster, shortens the attack from weeks to days, and reduces joint damage

3. Elevate the affected joint on pillows and keep bed sheets or blankets off the area using a bed cradle or box — The inflamed joint is so tender that even light touch from sheets causes severe pain; elevation reduces blood pooling and swelling by using gravity to drain fluid back toward the heart
4. Hold any scheduled allopurinol or febuxostat if this is a new gout attack and the patient is not already taking it — Starting urate-lowering drugs during an acute flare makes the attack worse by causing sudden shifts in uric acid levels that shake more crystals loose into the joint; these drugs should only start after the flare is completely gone
5. Encourage drinking 8 to 12 cups of water daily unless the patient has heart failure or kidney failure with fluid restrictions — High fluid intake dilutes uric acid in the blood and urine so less crystallizes in joints; it also helps kidneys flush uric acid out in the pee, lowering total body uric acid load
6. Check the medication list and hold diuretics (water pills) if the provider approves, especially thiazides and furosemide — Diuretics make kidneys reabsorb uric acid back into the blood instead of peeing it out, which raises uric acid levels and can trigger or prolong gout attacks
7. Teach the patient to avoid alcohol, red meat, organ meats (liver, kidney), shellfish, and high-fructose drinks during the attack and long-term — These foods are high in purines (building blocks that break down into uric acid) and alcohol blocks the kidneys from removing uric acid; avoiding them lowers uric acid production and prevents future flares
8. Monitor for side effects: diarrhea with colchicine, stomach pain or black stools with NSAIDs, high blood sugar or mood changes with steroids — Colchicine is toxic to gut cells and causes watery diarrhea; NSAIDs damage the stomach lining causing ulcers and bleeding; steroids raise blood glucose by making cells resist insulin and can cause agitation or confusion, especially in elderly patients

Sources

- American College of Rheumatology 2020 Gout Management Guideline
- Cochrane Database of Systematic Reviews
- New England Journal of Medicine
- CDC Arthritis Program

Last reviewed: 2026-07-07