

Gout

A plain-English guide for you and your family

WHAT IS HAPPENING

Your body constantly breaks down purines (building blocks from food like red meat, seafood, alcohol, and from your own cells) into uric acid. Normally, two-thirds of that uric acid leaves through your kidneys in urine, and one-third leaves through your gut in stool, keeping blood levels safely below 6–7 mg/dL. But when too much uric acid piles up — either because you're making too much (from a diet heavy in purines, alcohol, or sudden cancer cell breakdown) or getting rid of too little (from kidney disease, water pills, or dehydration) — the uric acid crosses a tipping point and crystallizes into needle-shaped solids inside joints, especially the big toe. Your immune system sees these crystals as dangerous invaders and floods the joint with white blood cells and inflammatory chemicals, causing the joint to swell, turn red and hot, and hurt intensely. Over months to years, repeated attacks scar the joint, destroy cartilage, and form lumpy uric acid deposits called tophi under the skin and in the kidneys, where they can also form kidney stones and damage kidney tissue. Every symptom traces back to this one upstream problem: uric acid concentration climbing past the point where it stays dissolved in blood.

What you might feel

- Sudden, severe pain in one joint — often the big toe, ankle, or knee — that comes on at night or early morning
- The joint becomes hot, red, and swollen; the skin may look shiny and tight
- The joint is so tender that even a bedsheet touching it can hurt
- Pain and swelling usually peak within 12 to 24 hours
- Attacks are triggered by alcohol (especially beer), red meat, shellfish, sugary drinks, water pills (diuretics), hospital stay, surgery, or starting uric-acid-lowering medicine
- Firm, chalky lumps (tophi) under the skin near joints or on the ears
- Fever and feeling generally unwell during a severe attack
- Multiple joints flaring at once, or an attack that won't go away

Call the doctor right away if...

- 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)

What the care team will do

- Check temperature, heart rate, and blood pressure, and look at the affected joint for extreme redness, warmth, and swelling
- Give the ordered anti-inflammatory medicine (colchicine, NSAID, or steroid) as soon as possible, ideally within 24 hours of symptom start
- Elevate the affected joint on pillows and keep bed sheets or blankets off the area using a bed cradle or box
- Hold any scheduled allopurinol or febuxostat if this is a new gout attack and the patient is not already taking it
- Encourage drinking 8 to 12 cups of water daily unless the patient has heart failure or kidney failure with fluid restrictions
- Check the medication list and hold diuretics (water pills) if the provider approves, especially thiazides and furosemide
- Teach the patient to avoid alcohol, red meat, organ meats (liver, kidney), shellfish, and high-fructose drinks during the attack and long-term
- Monitor for side effects: diarrhea with colchicine, stomach pain or black stools with NSAIDs, high blood sugar or mood changes with steroids

Medicines you may be given — and why

Colchicine

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.

NSAIDs (e.g., indomethacin, naproxen, ibuprofen)

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.

Corticosteroids (e.g., prednisone, methylprednisolone; or intra-articular triamcinolone)

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.

Allopurinol

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.

Febuxostat

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.

Probenecid

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.

Pegloticase

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.

Where this information comes from

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

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