PATIENT EDUCATION · GOUT & CKD

Gout & Uric Acid in Kidney Disease

Acute flares, diet, medications, and the gout–CKD cycle. Practical guidance for Filipino patients with gout, hyperuricemia, or chronic kidney disease. Covers **allopurinol use in CKD, low-purine Filipino foods, flare management,** and long-term uric acid targets.

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<6.0 mg/dL	↑ Risk	Allopurinol	Diet
Uric Acid Target (on treatment)	CKD Progression with high UA	Primary ULT Treatment	Modifies Uric Acid Risk

Uric acid target: <6 mg/dL for all gout patients on treatment; <5 mg/dL for tophaceous gout. Many Filipinos with gout remain undiagnosed or undertreated — allopurinol is inexpensive and highly effective when used correctly.

1 What Is Uric Acid? — The Purine Breakdown Product

Uric acid is the final breakdown product of **purines** — nitrogen-containing compounds found in many foods and naturally produced by the body during cell turnover. Purines are converted to uric acid by the enzyme **xanthine oxidase**. In healthy kidneys, about 70% of uric acid is excreted in urine. In CKD, impaired renal excretion causes serum uric acid to accumulate — a condition called **hyperuricemia** (serum UA >7 mg/dL in men, >6 mg/dL in women).

When uric acid concentration exceeds its solubility threshold (~6.8 mg/dL), it crystallizes as **monosodium urate (MSU) crystals** that deposit in joints, soft tissues, and renal tubules — causing acute gout attacks, tophi, and urate nephropathy. Uric acid targets: men <6 mg/dL on treatment; women <5 mg/dL; tophaceous gout <5 mg/dL.

⚠ The Gout–CKD Vicious Cycle

High Uric Acid → Crystal deposits in renal tubules → tubular inflammation & fibrosis → **CKD Progression**

Worsening CKD → Impaired uric acid excretion → serum UA rises further → **More crystal deposition**

Breaking this cycle with urate-lowering therapy (allopurinol) may slow CKD progression — start low (50 mg/day), titrate slowly, monitor creatinine every 4–6 weeks during titration.

2 Two Presentations of Gout

Acute Gout Flare

Sudden-onset, excruciating joint pain — often nocturnal

Classic site: First metatarsophalangeal joint (big toe) — podagra. Also ankle, knee, wrist, elbow.

Features: severe pain (often 10/10), warmth, erythema, swelling, exquisite tenderness — cannot tolerate a bedsheet touching the joint.

Duration: Untreated: 7–14 days. Treated early: 3–5 days.

Triggers: dehydration, alcohol (especially beer), organ meats, diuretics, contrast dye, illness, surgery.

UA target on treatment: <6 mg/dL (<5 mg/dL if tophi)

Chronic Tophaceous Gout

Years of uncontrolled hyperuricemia → crystal accumulation in tissues

Tophi: visible chalky MSU crystal deposits — ear helix, olecranon bursa, finger joints, Achilles tendon.

Joint destruction: chronic synovitis → erosive arthropathy → deformity and disability. X-ray: "punched-out" erosions with overhanging edges.

Kidney damage: urate nephropathy, uric acid kidney stones, interstitial nephritis → CKD progression.

Prevention: persistent ULT to keep UA <6 mg/dL — tophi dissolve slowly over 1–3 years on adequate therapy.

Note: Tophi are NOT an emergency unless they rupture (infection risk) or compress a nerve

 References: ACR Gout Guidelines 2020 · EULAR Gout 2016 · KDIGO CKD 2024 · Neogi et al., ACR 2015 classification criteria.

WHAT IS URIC ACID? — PURINE METABOLISM & CKD

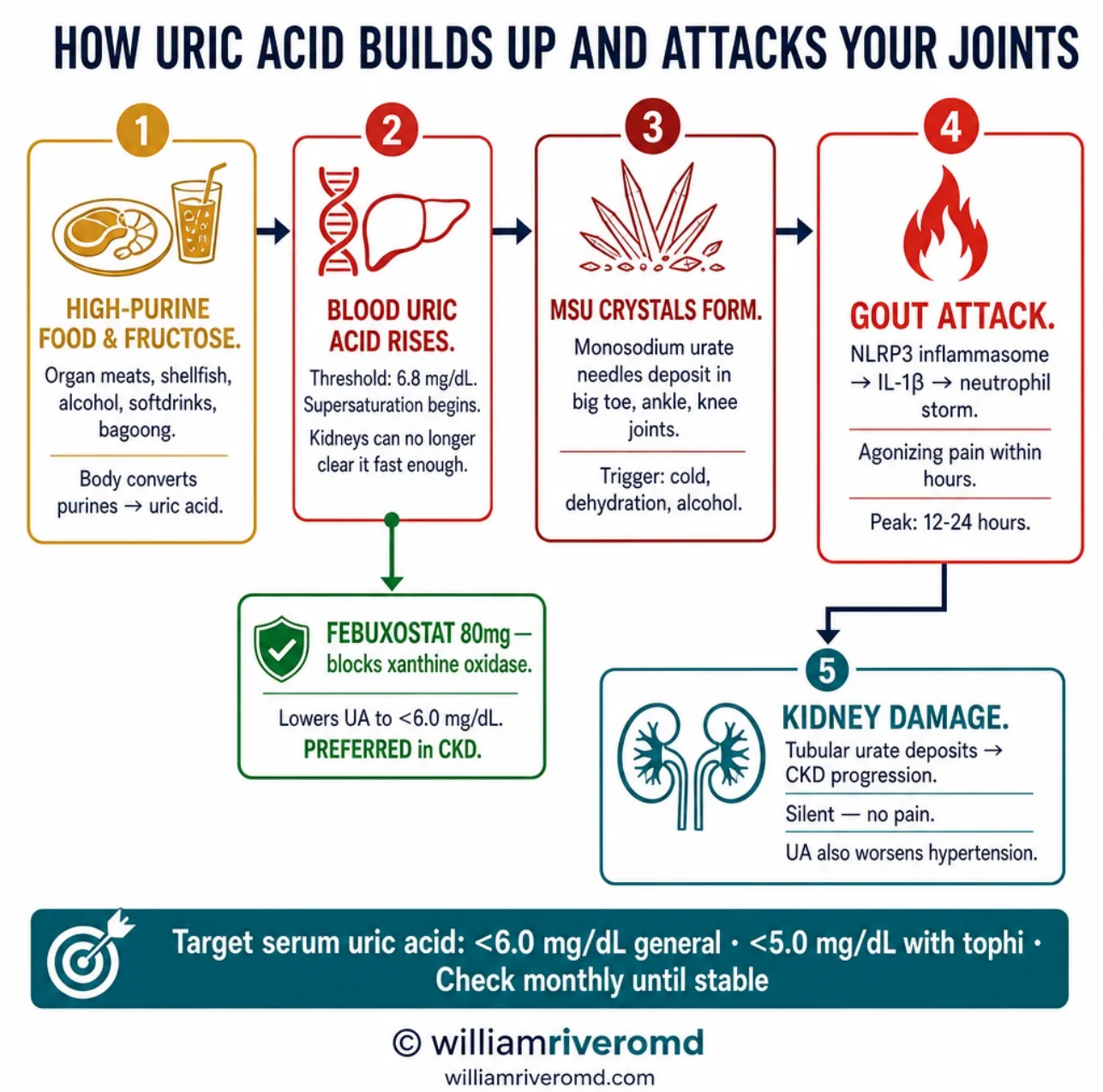

Fig. 1 — Uric acid is the end product of purine metabolism. Purines from food (organ meats, seafood, beer) and endogenous cell turnover are converted to uric acid by xanthine oxidase. In healthy kidneys, ~70% is excreted in urine. In CKD, impaired kidney excretion causes serum uric acid to accumulate, leading to crystal deposition in joints, soft tissues, and renal tubules. This is why gout is both more common and more severe in patients with chronic kidney disease.

THE INFLAMMATORY CASCADE OF AN ACUTE GOUT ATTACK

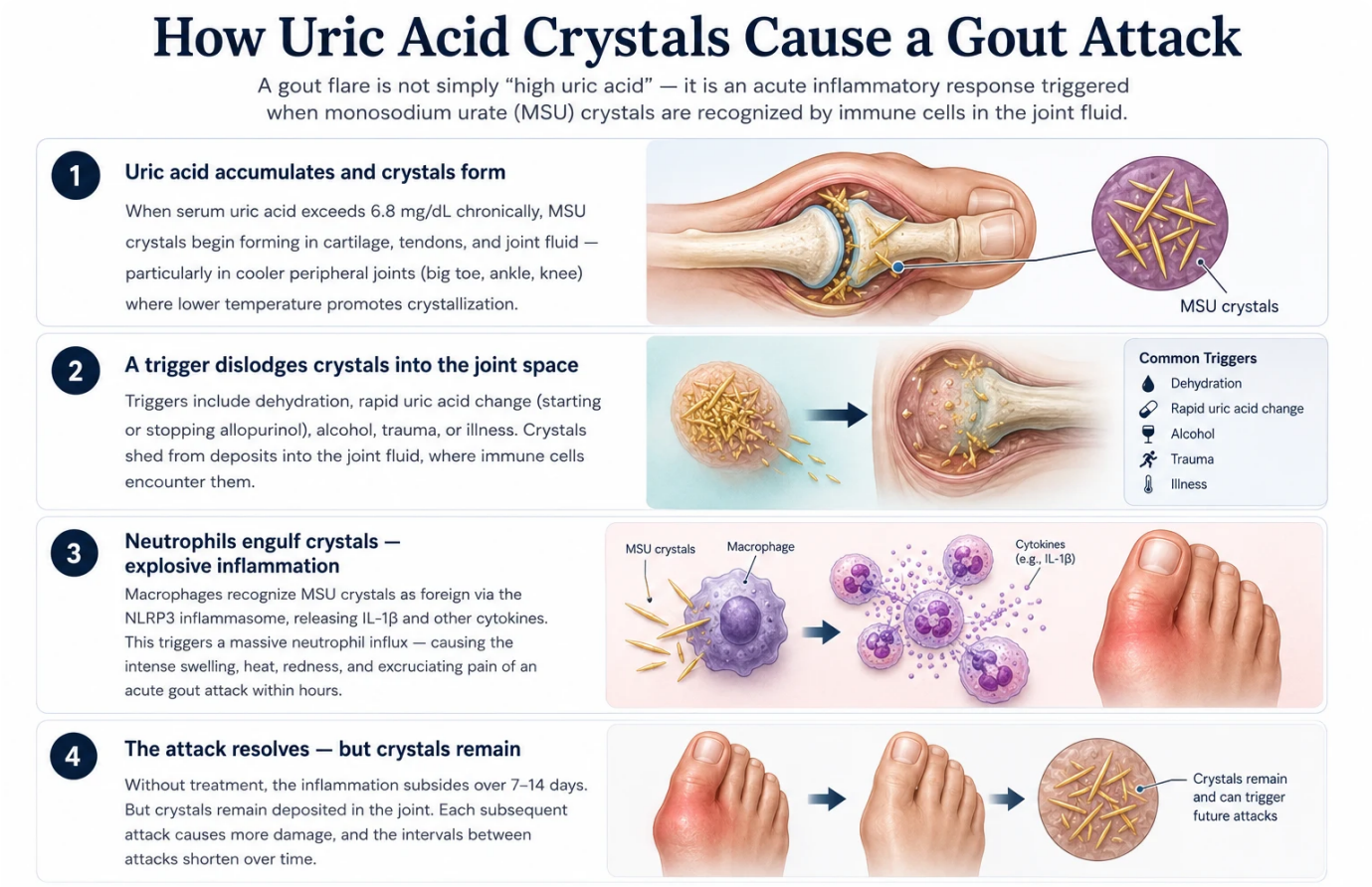

Fig. 2 — The inflammatory cascade of an acute gout attack: monosodium urate (MSU) crystals deposit in synovial fluid when serum uric acid exceeds the solubility threshold (~6.8 mg/dL). Neutrophils engulf the crystals and activate the NLRP3 inflammasome, releasing IL-1β, IL-6, and TNF-α — causing the hallmark intense joint inflammation, warmth, swelling, and severe pain. Colchicine inhibits neutrophil migration; NSAIDs and corticosteroids suppress the downstream inflammatory response.

Filipino Foods — Purine & Uric Acid Content

Heat-map reference · Values approximate per 100g raw or per standard serving · FNRI Philippine tables + international purine databases

Food (Filipino / common name)	Purines (mg/100g)	Notes for Gout & CKD Patients
VERY HIGH PURINES (>200 MG/100G) — AVOID COMPLETELY DURING FLARE; SEVERELY LIMIT AT ALL TIMES		
Dilis — dried anchovies	600 mg	Extremely concentrated purines in dried form. A small handful delivers a massive uric acid load. Avoid entirely in gout.
Sardinas sa mantika — canned sardines in oil	480 mg	Very high purines from fish flesh and canning liquid. Avoid during flares; limit to once per week at most otherwise.
Atay ng baboy / manok — pork / chicken liver	444 mg	Highest purine load of any common Filipino food. Avoid completely — even 50g significantly raises serum UA.
Bato — kidney (pork / beef)	325 mg	Organ meat — extremely high purines. Avoid completely. Common in goto, pares, and some kare-kare preparations.
Utak — brain (pork)	250 mg	Very high purines. Avoid completely. Used in some sisig and kare-kare recipes.
Pusit — squid	200 mg	High purines. Avoid during flares. Limit to small portions (≤50g) on non-flare days, infrequently.
Chicken / beef broth cubes (Knorr, Maggi)	Very high	Highly concentrated meat extracts — among the highest purine sources per gram. Use pandan or tanglad (lemongrass) for flavor instead.
MODERATE PURINES (50–200 MG/100G) — LIMIT TO 1 SERVING PER DAY; AVOID ENTIRELY DURING ACTIVE FLARE		
Manok — chicken breast/thigh (100g)	175 mg	Moderate purines. One palm-sized piece (100g) per day is acceptable. Avoid skin. Boiled or grilled is preferred over fried.
Alimango / alimasag — crab (100g)	152 mg	Moderate-high. Limit to ≤50g during non-flare periods. Avoid entirely during flares.
Hipon — shrimp (100g)	150 mg	Limit to small portions (50g) occasionally. Higher purine than freshwater fish.
Baboy — pork (100g lean)	150 mg	Limit to 1 serving/day. Avoid fatty cuts (liempo). Isaw (intestine) = organ = very high purines — avoid.
Baka — beef (100g lean)	120 mg	1 serving/day acceptable. Avoid beef broth (concentrated purines). Avoid bulalo (bone marrow).
Bangus — milkfish (100g)	90 mg	Lower than many fish. One of the best fish choices for gout patients in the Philippines. 1 serving/day acceptable.
Tilapia (100g)	80 mg	Reasonable purine level. 1 piece/day acceptable. Grilled or steamed preferred.
Kabute — mushroom (1 cup)	60 mg	Plant purines — less readily absorbed than animal purines. Limit to 1 cup/day; avoid during active flare.
Monggo — mung beans (½ cup cooked)	50 mg	**Plant purines have minimal UA effect.** Do NOT eliminate monggo for gout — large studies show legumes do not significantly raise serum UA. Excellent fiber and protein source.
Kangkong (1 cup cooked)	40 mg	Very low actual risk despite being a moderate purine food. Eating kangkong does not significantly raise serum UA — safe daily vegetable.
Beer — 1 bottle (330 mL)	★ Multiple	**Single most potent gout trigger:** raises UA via (1) guanosine content, (2) ethanol → accelerated purine breakdown, (3) lactic acid → impaired renal UA excretion. AVOID completely in gout.
LOW PURINES — SAFE CHOICES; EAT FREELY (IN APPROPRIATE PORTIONS FOR OTHER DIETARY RESTRICTIONS)		
Itlog — eggs (chicken or duck)	<1 mg	Essentially zero purines. Excellent protein source for gout patients. No restriction needed.
Gatas — low-fat milk, yogurt	<5 mg	Low-fat dairy may actively **lower** serum uric acid by promoting renal UA excretion. 1–2 glasses/day beneficial (check phosphorus in CKD).
Kanin / bigas — white rice	<5 mg	Lowest purine of any staple. Safe for gout. Preferred over brown rice in CKD 4–5 (lower phosphorus).
Kamote, cassava, saging saba (as starch)	<10 mg	Low purine starchy foods — safe for gout. Saba banana: check potassium if fluid-restricted.
Most fruits (pakwan, papaya, mangga, pinya)	<15 mg	Safe. Exception: avoid sweetened fruit juices and sodas — fructose raises serum UA independently of purines.
Tofu — white / silken	Moderate plant	Despite moderate plant purines, tofu does NOT raise serum UA in clinical studies — may be mildly protective. Safe to eat daily.
Kape — coffee (1–2 cups/day)	—	Coffee (regular or decaf) **lowers serum uric acid** and reduces gout risk in epidemiological studies. 1–2 cups/day may be beneficial.
Seresa — cherries (½ cup daily)	<5 mg	Cherry consumption associated with 35% lower gout flare risk. Anthocyanins inhibit xanthine oxidase and have anti-inflammatory effects.

★ Beer triggers gout via multiple independent mechanisms beyond purine content. Plant purines (monggo, tofu, vegetables) have significantly less impact on serum UA than equivalent animal purines. Values are approximations — individual response varies.

Choi et al., NEJM 2004 · Zhang et al., Ann Rheum Dis 2012 · FNRI Philippine Food Composition Tables 2023 · ACR Gout Guidelines 2020 · **Educational use only.**

GOUT-FRIENDLY FILIPINO DIET — WHAT TO EAT & AVOID

Fig. 3 — Gout-friendly Filipino diet: emphasize white rice, eggs, low-fat protein (bangus, tilapia, tofu), fruits, and water. Avoid organ meats (atay, bato, utak), dried fish (dilis), beer and all alcohol, and high-fructose drinks (soft drinks, sweetened fruit juice). Tofu and low-fat dairy may actually lower serum uric acid levels. Monggo beans are safe — do not eliminate them based on old advice. Coffee (1–2 cups/day) is associated with reduced gout risk in large epidemiological studies.

HIDDEN URIC ACID TRIGGERS IN THE FILIPINO DIET

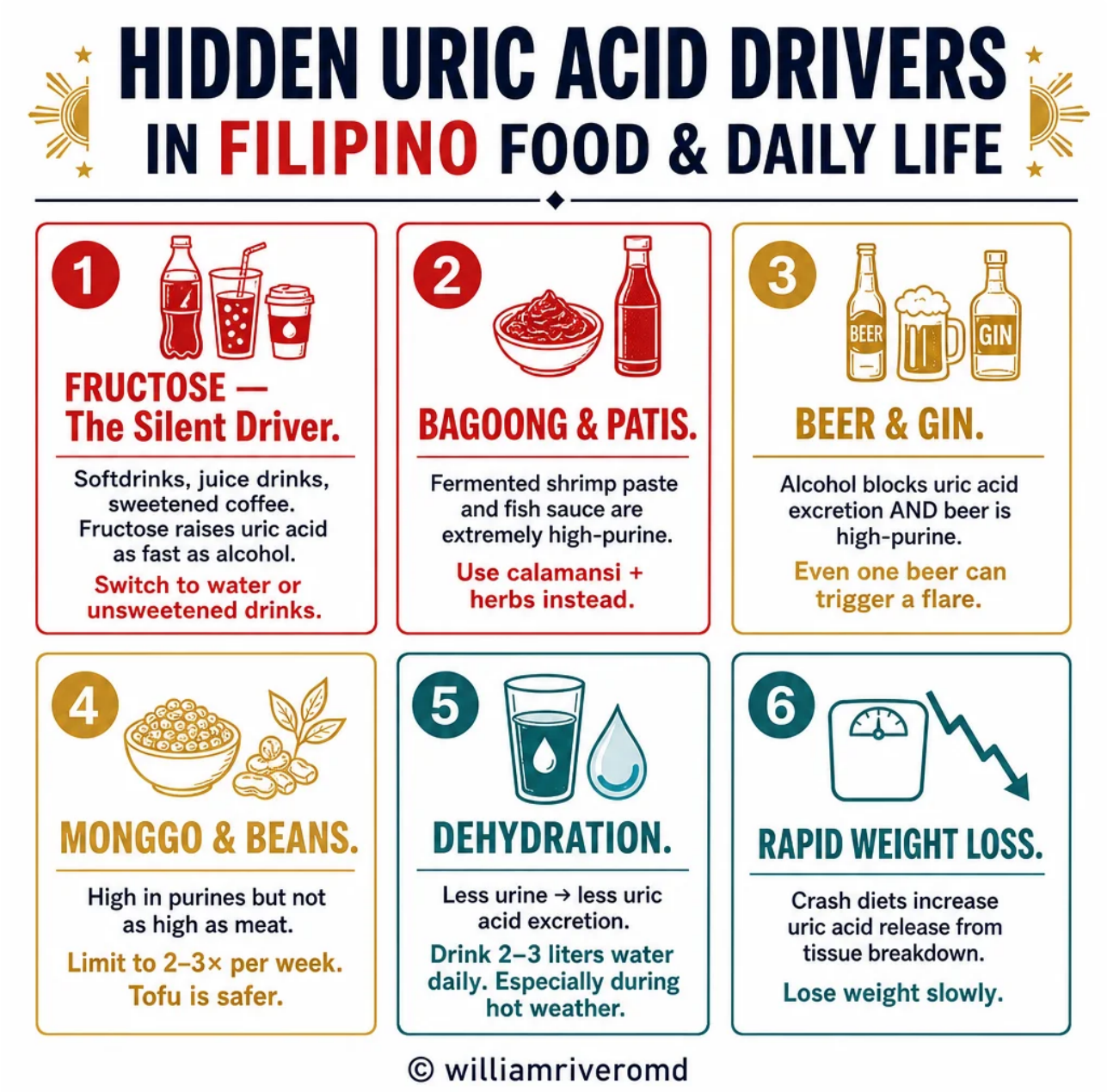

Fig. 4 — Hidden uric acid triggers in the Filipino diet: (1) **Fruit juice and softdrinks** — fructose raises uric acid by activating AMP deaminase, accelerating purine breakdown independently of dietary purines; (2) **Processed meat seasonings** (Knorr, Maggi liquid seasoning) — highly concentrated meat extracts with very high purine density; (3) **Instant soups and mami** with meat extract broth — concentrated animal purines hidden in the flavor base; (4) **Beer** — the single most potent gout trigger in the Filipino diet, acting through purine load, ethanol-driven purine catabolism, and lactic acidosis impairing renal uric acid excretion.

What To Do During an Acute Gout Flare — Start Within 12–24 Hours

1. **Rest the joint immediately** — Do NOT walk on a swollen foot or use the affected joint. Elevate the extremity to reduce swelling. Bed rest is appropriate for severe podagra.
2. **Ice pack — NOT heat** — Apply ice wrapped in cloth for 20 minutes on, 20 minutes off. Ice reduces inflammation and numbs pain. Never apply heat (a common Filipino home remedy) — heat worsens crystal precipitation and inflammation.
3. **Take colchicine or NSAIDs as prescribed — START EARLY** — The earlier treatment starts, the faster resolution. If colchicine: 1 mg at first sign of flare, then 0.5 mg after 1 hour (low-dose protocol). Dose-reduce in CKD (see table below). If NSAIDs: only if eGFR >30 and no contraindications.
4. **Increase water intake to 2–3 L/day** — Unless your nephrologist has restricted fluids. Increased urine output helps clear uric acid and reduces crystal supersaturation. Avoid all alcohol.
5. **Stop all alcohol immediately** — Even one beer during a flare prolongs the attack by 24–48 hours. Alcohol is the most common Filipino gout flare trigger.
6. **Continue allopurinol — do NOT stop during a flare** — Stopping and restarting allopurinol causes uric acid fluctuations that prolong the attack. If already on allopurinol, keep taking it at the same dose throughout the flare.

IMPORTANT — NSAID Safety Warning for CKD Patients

Do NOT take NSAIDs (mefenamic acid, ibuprofen, diclofenac, naproxen) if your eGFR is <30 mL/min/1.73m² — they acutely constrict the afferent arteriole and can cause a sudden, potentially irreversible drop in kidney function (NSAID-induced AKI). Use **colchicine with dose reduction** instead (≤0.5 mg/day if eGFR <30). Corticosteroids (prednisolone) are the preferred alternative when both NSAIDs and colchicine are contraindicated. **Always ask your nephrologist before taking any pain medication during a flare.**

When to Go to the Emergency Room

Emergency Red Flag	Reason
Fever >38.5°C WITH joint swelling	May be **septic arthritis** (joint infection) — a surgical emergency. Requires joint aspiration to distinguish from gout. Delay risks joint destruction.
Tophi breaking through the skin	Open wound with urate crystal exposure → high infection risk → sepsis. Requires urgent wound care and antibiotics.
Multiple joints simultaneously swollen	Polyarticular gout or alternative diagnosis (RA, reactive arthritis, bacterial). Needs urgent evaluation and joint aspiration.
Severe pain uncontrolled by prescribed medications	IV colchicine or IV corticosteroids may be required. Do not exceed prescribed colchicine dose — toxicity is dangerous in CKD.

Flare Duration Guide

Untreated flare: 7–14 days to resolution
Treated early (within 12–24 hrs): 3–5 days
Treated late (>48 hrs): 5–10 days
Recurrent flares without ULT: progressively longer and more severe over years.

⚠ Colchicine Dose Reduction in CKD

eGFR >60: Standard: 1 mg then 0.5 mg after 1 hr (acute)
eGFR 30–60: 0.5 mg once for acute; 0.5 mg/day prophylaxis
eGFR <30: Single 0.5 mg dose only; avoid repeat dosing; avoid for prophylaxis
Dialysis: 0.5 mg every 48–72 hrs with close monitoring

Khanna et al., ACR Gout Guidelines 2020 · Richette et al., EULAR 2016 · Neogi T., NEJM 2011 · Stamp et al., Drug Saf 2011 · **Educational use only.**

ACUTE GOUT FLARE — CLASSIC PRESENTATION & JOINT SITES

Fig. 5 — Acute gout flare: classic presentation with erythema, warmth, swelling, and exquisite tenderness of the first metatarsophalangeal joint (podagra) — the most common initial site. The ankle, knee, and wrist are also frequent targets. Patients often describe the pain as the worst of their life — unable to tolerate a bedsheet touching the joint. The differential diagnosis includes septic arthritis (requires fever + joint aspiration to exclude), pseudogout (calcium pyrophosphate crystals), and cellulitis. Early treatment within 12–24 hours of symptom onset dramatically shortens flare duration from 7–14 days to 3–5 days.

Gout Medications — Dosing & CKD Considerations

Medication	Dose	Use	Key Notes for CKD Patients
Allopurinol	50–300 mg/day	Long-term ULT (first-line)	**Start at 50 mg/day in CKD** — titrate by 50 mg every 2–4 weeks to target UA <6 mg/dL. Metabolized to oxypurinol, which accumulates in CKD. **STOP immediately if any rash develops** — risk of severe DRESS syndrome (drug reaction with eosinophilia and systemic symptoms), which can be fatal. HLA-B*58:01 screening recommended in Filipino and Han Chinese patients before starting (higher DRESS risk).
Febuxostat	40–80 mg/day	Long-term ULT (second-line)	Alternative for allopurinol-intolerant patients. No dose adjustment needed for mild–moderate CKD. **Caution in cardiovascular disease** — CARES trial showed possible increased CV mortality vs allopurinol. Avoid in unstable angina or recent MI. More expensive than allopurinol in the Philippines.
Colchicine	0.5–1 mg/day (prophylaxis); 1 mg acute	Flare prevention & acute treatment	**Reduce dose in CKD** (see Page 7). Risk of neuromuscular toxicity with accumulation in CKD. Never combine with clarithromycin or strong CYP3A4 inhibitors. Continue prophylactic colchicine for 3–6 months after starting ULT to prevent mobilization flares.
NSAIDs (mefenamic acid, ibuprofen, diclofenac)	Short course (3–5 days)	Acute flare (if eGFR >30)	**Nephrotoxic — AVOID in CKD stages 4–5 (eGFR <30).** Even 3 days can precipitate AKI requiring dialysis in advanced CKD. Avoid in patients on ACEi/ARB + diuretic combination (triple whammy = high AKI risk).
Prednisolone	20–40 mg/day × 3–5 days	Acute flare (when NSAIDs & colchicine contraindicated)	Preferred when NSAIDs are contraindicated (eGFR <30) and colchicine is not tolerated. Short course generally safe. **Monitor blood sugar** — can precipitate hyperglycemia in diabetics. Exclude active infection before use.

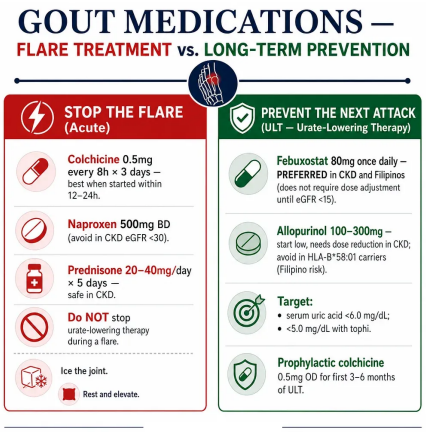

Fig. 6 — Urate-lowering therapies (ULT: allopurinol, febuxostat) and anti-inflammatory agents (colchicine, NSAIDs, corticosteroids) used in gout management. In CKD, allopurinol must be started at 50 mg/day and titrated slowly; NSAIDs are avoided if eGFR <30.

How Gout Damages the Kidneys

Urate Nephropathy

MSU crystal deposits in renal interstitium and tubules → tubular obstruction → chronic interstitial nephritis → progressive CKD. Occurs with persistently elevated serum UA even between gout attacks. Effective ULT (allopurinol) slows or stabilizes CKD progression in several studies.

Uric Acid Kidney Stones

Supersaturation in acidic urine (common in gout + metabolic syndrome) → uric acid nephrolithiasis. Radiolucent on plain X-ray. Treatment: urinary alkalinization with potassium citrate (target urine pH 6.0–6.5) + hydration 2–2.5 L/day + allopurinol if recurrent.

Cardiovascular Risk

Hyperuricemia associated with hypertension (inhibits NO production → vasoconstriction), endothelial dysfunction, and CKD progression. Treating gout reduces overall inflammatory burden and cardiovascular risk.

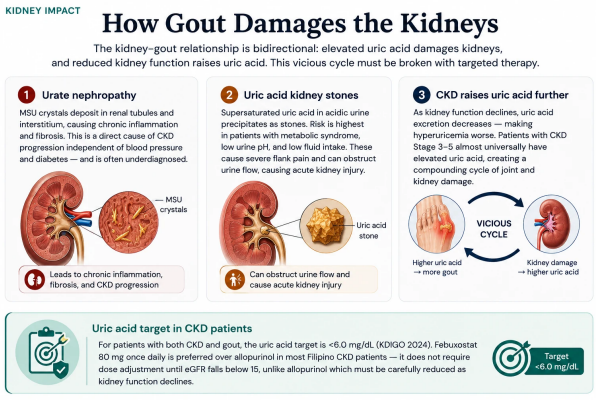

Fig. 7 — Mechanisms by which chronic hyperuricemia and gout damage kidney tissue, accelerate CKD progression, and increase cardiovascular risk. MSU crystal deposition in tubules causes obstructive nephropathy; uric acid stones cause obstructive AKI; endothelial inflammation drives hypertension and glomerular injury.

Long-Term Management Goals

Your Long-Term Gout & Kidney Protection Plan

- **Serum uric acid target:** <6 mg/dL on urate-lowering therapy (<5 mg/dL if tophi present). Check UA every 3–6 months until target reached, then annually.
- **Hydration:** 2 L water/day (unless fluid-restricted by nephrologist) — the single most important non-pharmacologic intervention.
- **Diet:** Eliminate organ meats, dried fish (dilis), and all alcohol. Reduce sweetened drinks and fruit juices (fructose raises UA). Low-fat dairy, tofu, eggs, and coffee are safe or beneficial.
- **Weight loss if overweight:** Reduces UA production and improves insulin sensitivity (hyperinsulinemia impairs renal UA excretion). Even 5 kg loss can lower serum UA by 0.5–1 mg/dL.
- **Coffee 1–2 cups/day** may help lower serum UA based on epidemiological evidence.
- **Never stop allopurinol without your doctor's advice** — stopping causes rebound hyperuricemia and increased flare risk.
- **Monitor kidney function** (creatinine, eGFR, urinalysis) every 3–6 months while adjusting allopurinol doses.

References: ACR Gout Guidelines (Khanna et al.) 2020 · EULAR Gout Recommendations (Richette et al.) 2016 · KDIGO CKD Guidelines 2024 · Choi HK NEJM 2004 · Stamp LK Drug Saf 2011 ·