SUMMARY Gout: what's in and what's out

The bottom line

Genetic factors and renal function are the key drivers of gout risk; **dietary and lifestyle changes alone are insufficient for preventing flares** (but are still important)

Gout is not "curable", but it can be controlled; long-term adherence to urate-lowering treatment can prevent flares from occurring and the risk of adverse effects can be managed

Avoid	Encourage
• Excess purines e.g. beer, shellfish, liver, marmite • Fructose, particularly fruit juice • Tomatoes • A sedentary lifestyle	• Dairy products • Vegetables • Staying hydrated (≥2 L water/day) • Maintaining a healthy BMI • Exercise

Vitamin C supplementation does not significantly lower serum urate levels as previously thought

Adherence to urate-lowering treatment is the key to long-term management

Initial steps

Establish diagnosis and severity
e.g. symptoms, number of joints involved, level of pain, measure serum urate

Patient education on cause, lifestyle changes and urate lowering treatment

Assess renal function – review/manage co-morbidities and medicine use to preserve this

Acute flare management

NSAIDs (naproxen preferred)
Corticosteroids (prednisone)
Low-dose colchicine*

- All acute treatment options are considered equally effective for managing flares
- Choice depends on preference, co-morbidities and other medicine use
- **Combination treatment may be required for severe gout flares**

Initiate urate-lowering medicine if indicated (even during acute treatment of flares)
Allopurinol is preferred unless contraindicated, ineffective or not tolerated

flare prophylaxis is usually provided for ≥ 6 months (see medicine table at end for options). However, new guidance suggests that 3–6 months might be suitable for some patients.

Up-titrate dose† until serum urate target of < 0.36 mmol/L is reached**
See treatment options table for medicine specific guidance

Once the target is reached, review patient every 6–12 months or after a flare

Symptoms controlled and target achieved → **Continue long-term treatment**

Gout flare or target not achieved → **Acute management for flares; re-review ULT dosing, lifestyle, co-morbidities and medicines**

Long-term management

* Although colchicine is sometimes avoided by New Zealand clinicians, use in low doses has comparable efficacy to NSAIDs and prednisone if given within 36 hours of the flare onset, with an improved safety profile compared to traditional high-dose regimens; † For allopurinol, up-titration should be performed every four weeks;
** A serum urate target of < 0.30 mmol/L is recommended if tophi are present

Diagnosing gout

Gout is diagnosed clinically with supporting evidence from elevated serum urate levels **(see the "practice tool" for more information)**

Repeat serum urate testing may be required if levels are assessed during a flare as they may be "abnormally normal"

Request a renal function test at the same time as testing serum urate (this will inform subsequent treatment decisions)

For more information on manging gout in primary care, see:

- **https://bpac.org.nz/2018/gout-part1.aspx** and
- **https://bpac.org.nz/2018/gout-part2.aspx**

BMI, body mass index; NSAIDs, non-steroidal anti-inflammatory drugs.1. Khanna D, Fitzgerald JD, Khanna PP, *et al.* Arthritis Care Res. 2012;64:1431–46; 2. Khanna D, Khanna PP, Fitzgerald JD, *et al.* Arthritis Care Res. 2012;64:1447–61; 3. Graf SW, Whittle SL, Wechalekar MD, et al. Int J Rheum Dis. 2015;18:341–51; 4. Richette P, Doherty M, Pascual E, *et al.* Ann Rheum Dis. 2017;76:29–42; 5. Edwards NL, Sundy JS, Forsythe A, *et al.* J Med Econ. 2011;14:10–5; 6. Janssens HJEM, Fransen J, van de Lisdonk EH, *et al.* Arch Intern Med. 2010;170:1120–6; 7. NZ Formulary. NZF v95. 2020. Available from: www.nzf.org.nz (Accessed May, 2020); 8. Wood R, Fermer S, Ramachandran S, *et al.* J Rheumatol. 2016;43:1897–903. 9. FitzGerald JD, Dalbeth N, Mikuls T *et al.* Arthritis Care & Research. 2020;72:744–60.

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Indications for urate-lowering treatment:

Symptomatic hyperuricaemia, with at least one of:

- ≥ 2 gout flares/year
- Tophi/tophus
- Renal impairment (eGFR <60 mL/min/1.73 m^2)
- Past urolithiasis
- Serum urate level ≥0.54 mmol/L

Stress the importance of adherence

Treat to a serum urate target of < 0.36 mmol/L (or < 0.30 mmol/L if tophi are present)

Consider checking anyone of Han Chinese, Korean or Thai ancestry for the *HLA B5801* allele before administering allopurinol as this can predispose them to hypersensitivity reactions

Urate lowering medicines are not indicated for the treatment of asymptomatic hyperuricaemia

Treatment options for managing gout in primary care

Treatment	Medicine/option		Dose range	Notes
Lifestyle modifications	Healthy overall diet (reduce purines and fructose), exercise, maintenance of normal BMI, adequate hydration			Lifestyle changes are important but are insufficient to control gout alone
Acute treatment (usually monotherapy, but combination treatment may be required for severe flares)	**NSAIDs**	**Naproxen** (preferred)	750 mg initially, 500 mg eight hours later, then 250 mg every eight hours until the flare settles	All options are equally effective for manging flares Naproxen is the preferred NSAID but celecoxib* can be used in patients intolerant to this Stop colchicine prophylaxis before initiating acute colchicine treatment. In elderly patients or in those with renal impairment, initial colchicine dose should not exceed 1 mg in first 24 hours; total max 3 mg over four days; do not repeat course within three days
		Celecoxib*	Up to 800 mg once, followed by 400 mg on day 1, then 400 mg BD for one week	
	Corticosteroids	**Prednisone**	Oral: 20–40 mg once daily for five days; tapering the dose over ten days can reduce the likelihood of a rebound flare, but is not always necessary	
	Low-dose colchicine (most effective if within 36 hours of flare onset)		1 mg immediately, followed by 500 µg after one hour on day 1, and then BD dosing of 500 µg until the flare settles (this is an alternative dose now recommended by many experts; see NZF for traditional regimen)	
Flare prophylaxis (prescribed alongside urate- lowering treatment)	**NSAIDs with PPI**	**Naproxen**	250 mg BD (with PPI if indicated)	Usually provide prophylactic medicines for six months alongside urate-lowering treatment; continue if symptoms do not resolve. However, new guidance suggests that 3–6 months might be suitable for some patients Only use prednisone for prophylaxis if NSAIDs and colchicine are contraindicated
	Very low-dose colchicine*		500 µg BD; reduce to 500 µg once daily or every other day if needed	
	Corticosteroids	**Prednisone**	5 mg once daily; tapered slowly on withdrawal	
Long-term treatment	**Urate-lowering medicine‡**	**Allopurinol** (First-line)	100 mg once daily (adapt to renal function);† increase dose every 4 weeks if required (up to 900 mg max usually; lower if renal function is impaired)	Probenecid can be used as either an add-on to allopurinol (or febuxostat), or as a second-line monotherapy if serum urate target is not met Febuxostat (and benzbromarone)‡ are typically third-line for patients where allopurinol and/or probenecid are ineffective, contraindicated or not tolerated; for more information on the risks associated with these medicines and guidance about use, see: **https://bpac.org.nz/2018/gout-part2.aspx** New ACR 2020 guidelines prioritise feboxustat as the second-line ULT (over probenecid) in patients without a history of CVD that have previously taken allopurinol. However, this is not yet reflected in New Zealand guidance and the Special Authority criteria for funding requires that probenecid is trialled first unless contraindicated
		Probenecid (Second-line)	250 mg BD for one week, then 500 mg BD; increase dose up to 1 g BD (2 g total) if required; avoid if eGFR is < 30 mL/min/1.73m^2	
		Febuxostat** (Third-line)	80 mg once daily; increase dose up to 120 mg once daily after 2–4 weeks if required; use with caution if eGFR is < 30 mL/min/1.73m^2	

* Unapproved indication; † If the patient's eGFR is 30–60 mL/min/1.73m^2, then use an initial dose of 50 mg once daily; if their eGFR is < 30 mL/min/1.73m^2, use 50 mg every other day; ** Special Authority required. There is an increased risk of cardiovascular and all-cause mortality with febuxostat compared with allopurinol; ‡ Benzbromarone is an additional urate-lowering treatment that is an unapproved medicine in New Zealand but can be obtained fully subsidised with Special Authority. However, as of May 2020, benzbromarone is out of stock at a wholesaler level in New Zealand, and PHARMAC has advised that it will likely delist this ULT from the Pharmaceutical schedule (no date is currently set). No new patients should be started on benzbromarone and those currently taking it should be switched to another ULT. Benzbromarone is associated with an increased risk of hepatotoxicity, so liver function monitoring is recommended.

BD, twice daily; eGFR, estimated glomerular filtration rate; NSAIDs, non-steroidal anti-inflammatory drugs; PPI, proton pump inhibitor.1. Khanna D, Fitzgerald JD, Khanna PP, *et al.* Arthritis Care Res. 2012;64:1431–46; 2. Khanna D, Khanna PP, Fitzgerald JD, *et al.* Arthritis Care Res. 2012;64:1447–61; 3. Graf SW, Whittle SL, Wechalekar MD, et al. Int J Rheum Dis. 2015;18:341–51; 4. Richette P, Doherty M, Pascual E, *et al.* Ann Rheum Dis. 2017;76:29–42; 5. Edwards NL, Sundy JS, Forsythe A, *et al.* J Med Econ. 2011;14:10–5; 6. Janssens HJEM, Fransen J, van de Lisdonk EH, *et al.* Arch Intern Med. 2010;170:1120–6; 7. NZ Formulary. NZF v95. Available from: www.nzf.org.nz (Accessed May, 2020); 8. Wood R, Fermer S, Ramachandran S, *et al.* J Rheumatol. 2016;43:1897–903. ; 9. FitzGerald JD, Dalbeth N, Mikuls T *et al.* Arthritis Care & Research. 2020;72:744–60.