



Managing gout in primary care

Part 1 – Talking about gout: time for a re-think

Gout is a treatable form of arthritis that is associated with poor health and reduced life expectancy. Too often, gout management is focused on controlling the patient's symptoms while their risk of irreversible joint damage and negative health outcomes continues to grow. Māori and Pacific peoples are disproportionately affected by gout and often receive sub-optimal care; it is time for a re-think to address this disparity.


KEY PRACTICE POINTS:

- Gout is a serious condition that is often associated with a range of long-term negative health outcomes, e.g. co-morbid cardiovascular disease, renal failure and reduced life expectancy
- The management of gout is sub-optimal in New Zealand, and changes need to be made both in community awareness and in the delivery of healthcare; after making a diagnosis, the emphasis should be placed on providing information to patients about their condition, addressing any misconceptions or concerns and supporting appropriate medicine use
- Gout flares can be treated with a NSAID, prednisone or low dose colchicine, depending on individual clinical circumstances; all are considered to be equally effective
- Discuss urate-lowering treatment with all patients with gout at their first presentation, recommend early initiation and encourage regular and consistent use; lifestyle changes alone are insufficient to prevent future gout flares from occurring
- Allopurinol can be initiated during an acute flare of gout if it is thought that this may improve the likelihood of the patient committing to long term treatment; however, this may not be appropriate for all patients

This is a revision of a previously published article.

What's new for this update:

- A general article revision
 - Update of statistics
 - Addition of genetics to the list of risk factors

 For further information about initiating and titrating allopurinol, see: "Part 2: Controlling gout with long-term medicines" available from: [bpac.org.nz/2021/gout-part2.aspx](https://www.bpac.org.nz/2021/gout-part2.aspx)

Gout is controllable with long-term treatment

Gout is the most common form of inflammatory arthritis.¹ It is caused by monosodium urate crystals accumulating in joint fluid, cartilage, bones, tendons and other tissues.² Urate is produced via the metabolism of dietary and endogenous purines.² When urate levels in the blood reach saturation point, monosodium urate crystals can form.² The inflammatory response to these crystals results in gout flares which are characterised by painful, red, hot, swollen joints. Over time, the duration and frequency of these flares may increase, resulting in chronic gouty arthritis and subcutaneous deposits of crystals referred to as tophi, both of which can lead to the destruction of joints.²

The risk factors for gout

Long-term hyperuricaemia is the most important risk factor for the development of gout, and in most patients this will be caused by declining renal function.² Detecting chronic kidney disease (CKD) early and preserving renal function is therefore an important gout-prevention strategy. Additional factors that contribute to hyperuricaemia and increase the risk of developing gout include:²⁻⁵

- Older age
- Genetics, e.g. variants of the *SLC2A9* fructose/urate co-transporter genes have been implicated in the greater number of Māori and Pacific peoples living with gout; it is thought that these genetic variants reduce the ability to excrete urate, contributing to hyperuricaemia and therefore the risk of gout
- Male sex
- Hypertension
- Obesity
- Use of certain medicines, e.g. diuretics or low dose aspirin (for more information on the appropriate selection and use of cardiovascular medicines, see: **Part 2: controlling gout with long-term urate-lowering medicines**)
- Excessive consumption of red meat, seafood, beer, spirits, sucrose or fructose-sweetened drinks

The burden of poorly-controlled gout is often over-looked

Gout is much more than an intensely painful condition that prevents people from working, performing daily activities and participating in their communities.¹ People with gout are also more likely than those without gout to die at a younger age due to co-morbid cardiovascular disease and renal complications.⁶ In New Zealand, 40% of people with gout have diabetes and/

or cardiovascular disease.⁷ Despite this, many people consider gout to be a condition that merely requires analgesics to control and are not aware of the potential long-term consequences.⁸ Raising community awareness about gout is an important role for health professionals in primary care.

Urate-lowering treatment improves long-term health outcomes

Reducing serum urate levels in patients with gout not only means that flares are less likely, it may also reduce the risk of adverse renal and cardiovascular outcomes. For example, a meta-analysis found that compared to patients who were not taking a urate-lowering medicine (or were taking a placebo), patients with hyperuricaemia and CKD who were taking a urate-lowering medicine:⁹

- Reduced their risk of cardiovascular events or renal failure by more than half
- Had slower rates of decline in renal function
- Reduced their proteinuria

More recently, it was found that people with gout and diabetes who were taking urate-lowering treatment had significantly lower risk of coronary artery disease or stroke.¹⁰

Māori and Pacific peoples with gout are not receiving adequate care

Gout management in New Zealand needs to change because Māori and Pacific peoples in particular are not receiving the medicines according to their level of need. Furthermore, research suggests that inequities between how gout is managed in Māori and non-Māori is ingrained in the current model of care, with no significant reduction in disparity in recent years.¹¹

Gout is more frequent and more severe in Māori and Pacific peoples

The prevalence and burden of gout in New Zealand is higher in Māori and Pacific peoples than in other groups. In 2019, gout was estimated to affect approximately 6% of people in New Zealand aged over 20 years; Māori and Pacific peoples aged 20 to 44 years have a three and seven times greater prevalence of gout than non-Māori and non-Pacific peoples, respectively.¹ The prevalence of gout increases with age; among males aged over 65 years the prevalence is 35% for Māori, 50% for Pacific peoples and 18% for non-Māori and non-Pacific groups.¹ Māori and Pacific peoples with gout are dispensed more prescriptions for NSAIDs each year (41% and 46%, respectively) than other ethnic groups (35%) and are therefore at greater risk of NSAID-related adverse effects, e.g. acute kidney injury and cardiovascular events.¹ Māori and Pacific patients with gout are also five and nine times more likely to be admitted to hospital due to gout than non-Māori, non-Pacific people.¹

Prescribers often delay initiation of urate-lowering treatment

Numerous studies from New Zealand and overseas show that urate-lowering treatment is often delayed well beyond the point when it is indicated. For example, a small qualitative study of Māori patients with gout found that on average urate-lowering treatment was not prescribed until 18 years after the appearance of symptoms.¹³ In 2019, dispensing data showed that Māori and Pacific peoples with gout were more likely to have received urate-lowering treatment (60%), i.e. dispensed medicine at least once in one year, than non-Māori and non-Pacific people with gout (56%). However, Māori and Pacific peoples were less likely to receive regular treatment (39% and 36%, respectively), i.e. dispensed medicine in three or four quarters in one year compared with non-Māori and non-Pacific people (43%).¹

Once urate-lowering medicines are started, monitoring is also often sub-optimal, meaning that many patients will still have serum urate concentrations above recommended levels for treating gout. A systematic review predominantly of studies from the United States and United Kingdom found only 28 – 38% of patients had their serum urate levels monitored regularly and 23% of patients taking allopurinol had serum urate levels above 0.36 mmol/L.¹⁴

Identifying the barriers to optimal management


The barriers to the early and optimal use of urate-lowering medicines are multi-factorial. Firstly, there is a lack of clarity in guidelines as to the best time to initiate treatment, and at times there are discrepancies between guidelines. Secondly, there is sometimes a perception among health professionals that gout management is acute, rather than preventative.⁸ The limited time that is available in consultations in primary care and the intermittent nature of gout flares also make it difficult for health professionals to focus on the long-term management of gout and promote patient education.¹⁵

Nurses and pharmacists have an important role in gout management

Most patients with gout are able to achieve serum urate targets if they are provided with effective support. This role is ideal for nurses in primary care; an essential component of gout education is overcoming misconceptions that are barriers to care (see: “Overcoming misconceptions that are barriers to managing gout”). A nurse-led programme in primary care in the United Kingdom found that with education and lifestyle advice, 92% of patients were able to achieve serum urate treatment targets.⁸

Community pharmacists can reduce delays in the diagnosis of gout and the initiation of urate-lowering treatments by

asking patients who are purchasing NSAIDs about their symptoms.⁸ Patients who may have gout, e.g. those with a history of gout-like flares, can be encouraged to present to general practice for an assessment, and those who know they have gout can be encouraged to discuss the possibility of starting urate-lowering treatment with a general practitioner.


 Owing My Gout and Gout Stop are initiatives led by community pharmacists, practice nurses and general practitioners to improve access to medicines for gout and to build on health literacy by educating participants. Further information is available from: https://www.arthritis.org.nz/wp-content/uploads/2020/07/Gout-programmes-evaluation-report-.FINAL_-200228.pdf. Another gout management improvement project was launched by the National Hauora Coalition in collaboration with Papakura Marae Health Clinic, further information is available from: <https://www.nhc.maori.nz/wp-content/uploads/2019/09/HCAAnnualReport-Final.pdf>

Overcoming misconceptions that are barriers to managing gout

Perceptions and beliefs about gout can contribute to delays in initiating urate-lowering treatment.¹⁶ Good communication helps to overcome misconceptions that are barriers to care. A structured approach to discussions is therefore recommended:

- Assess the patient’s understanding about gout
- Build on their knowledge by validating information that is correct, filling in knowledge gaps and correcting misconceptions
- Check that the patient has understood the information that has been delivered

The goal is to form a loop of communication, with gaps in understanding forming the basis for further discussion.

 Further information on effective discussion and communication about gout management with patients is available from: <https://bpac.org.nz/bpj/2014/april/gout.aspx>

Delivering the messages that patients and whānau need to hear


Do not blame yourself because you have gout. Lifestyle factors can trigger gout flares but are not the sole cause of the condition. Biological factors (e.g. chronic kidney disease, certain uric acid renal transporter alleles) and some medicines (e.g. diuretics) contribute significantly to the higher prevalence of gout in Māori and Pacific peoples compared with other ethnic groups.¹⁷ Explaining to patients that they may have a genetic predisposition to gout helps to dispel the perception that the condition is self-inflicted.

Gout is serious, it's not just "a pain in the toe". Patients should understand that gout is associated with an increased risk of co-morbid cardiovascular disease and renal complications. However, by educating patients to actively manage their condition, e.g. regularly taking preventative medicines and making lifestyle changes when appropriate, they can reduce this risk.

Gout is a long-term disease caused by deposits of urate crystals. These crystals are still present in the joint after a flare has settled. The crystals will only dissolve if the urate level in the blood is kept low (< 0.36 mmol/L) by regular use of medicines such as allopurinol.⁸

In the long-term, allopurinol can stop flares from happening. If patients regularly use urate-lowering treatment and serum urate levels are treated to target, flares of gout will be virtually eliminated for many patients within two years.¹⁸


Allopurinol is a safe and highly effective medicine. Urate-lowering medicines such as allopurinol are associated with an increased risk of flares in the first months of treatment and this may discourage some patients to take them, even if they have collected the prescription from the pharmacist.² Patients can be reassured that with prophylactic medicines and appropriate dose titration, the risk of allopurinol causing a flare will be substantially reduced and ongoing use will prevent future flares.

 Patient resources for gout, including Samoan and Tongan language versions, are available from: www.goodfellowunit.org/gout-study-project/gout-study-project

Diagnose gout, manage the flare and talk about long-term treatment

In primary care, gout is usually diagnosed clinically with supporting evidence provided by elevated serum urate levels; see "Diagnosing gout" for an example of a validated diagnosis tool and alternative diagnoses to consider.²

Caution is required when interpreting serum urate levels during a gout flare as up to 40% of patients are reported to have serum urate levels within the normal range;¹⁹ repeat testing for diagnostic purposes may be required once the flare has subsided.² Although the gold standard for diagnosing gout is the presence of monosodium urate crystals under polarised microscopy,² joint aspiration is usually not necessary unless there is a high suspicion of another cause, e.g. septic arthritis.

 **Best practice tip:** Request a renal function test at the same time as the serum urate to allow for the prompt initiation of urate-lowering treatment, should a diagnosis of gout be confirmed.

Medicines for gout flares are determined by the patient's characteristics

Patients with gout often initially present due to a flare, which will be the treatment priority. Rest and elevation of the affected joint should be encouraged during the gout flare, and some patients may find the use of an ice pack beneficial.⁴ Lifestyle changes to avoid obvious triggers, limit purine and fructose/sucrose intake, and reduce weight are important, but alone are insufficient for the management of gout.²

Rongoā rākau does not interfere with conventional gout treatments

Rongoā rākau (traditional plant remedies with healing properties) may be used by some Māori patients to treat flares of gout.¹³ This may be in the form of a poultice or plant material added to bathwater. Urate-lowering medicines can be used safely in combination with Rongoā rākau and should not be discouraged. Positive discussions about traditional medicines are helpful as they break down barriers with patients and allow prescribers to assess if any interactions with conventional medicines are likely.



Diagnosing gout

Table 1 provides an example of a scoring system to assess the likelihood of gout, which can support a clinical diagnosis. A score of eight or more is associated with a greater than 80% likelihood of gout.² A score of four or less rules out gout in almost 100% of patients and an alternative diagnosis should be considered.²

Table 1: Clinical score for the diagnosis of gout, adapted from Janssens *et al.* (2010)


Feature	Clinical score
Serum urate > 0.35 mmol/L	3.5
Metatarsophalangeal joint involvement	2.5
Male sex	2
Previous reported flare	2
Hypertension or ≥ 1 cardiovascular disease*	1.5
Joint erythema	1
Onset within one day	0.5
Score	Maximum 13

* Angina, myocardial infarction, heart failure, cerebrovascular event, transient ischaemic attack or peripheral vascular disease

Septic arthritis should be considered in patients with monoarticular joint pain, with erythema, warmth and joint immobility; systemic symptoms may also be present.²⁰ Often the patient will have an underlying condition affecting the joint, e.g. osteoarthritis, and concurrent

treatment with an immunosuppressive medicine increases the likelihood of infection.²⁰ The knee is most often affected by septic arthritis, followed by the hip, shoulder, ankle and wrist.²⁰ Patients with septic arthritis will often have an elevated serum white blood cell count and C-reactive protein levels may also be raised.²⁰

Acute calcium pyrophosphate crystal arthritis, also known as calcium pyrophosphate deposition (CPPD) disease, and previously known as pseudogout, is an arthritis caused by the accumulation of calcium pyrophosphate crystals.²¹ Acute calcium pyrophosphate crystal arthritis has a prevalence of 4 – 7% in European populations;²¹ the prevalence among Māori and Pacific peoples is unknown. Previous joint damage is a strong risk factor for calcium pyrophosphate crystal arthritis and it becomes more likely if the first onset of symptoms occurs later in life as it is rare in patients aged under 60 years.²¹ Patients with calcium pyrophosphate crystal arthritis often have systemic symptoms, including fever and chills, and elevated inflammatory markers, which can make it difficult to distinguish from infection.²¹ Where there is clinical uncertainty, calcium pyrophosphate crystal arthritis can be differentiated from gout and septic arthritis by requesting laboratory analysis of aspirated joint fluid.²¹ Radiography can also be used to support a diagnosis of acute calcium pyrophosphate crystal arthritis in joints that are unable to be aspirated.²¹ Unlike gout, calcium pyrophosphate-lowering medicines do not exist and treatment is focused on symptom relief.

 Further information on diagnosing and managing calcium pyrophosphate crystal arthritis is available from: <https://bpac.org.nz/bpj/2013/october/cppd.aspx>



A NSAID, corticosteroids or colchicine may be prescribed to treat gout flares

There are several options that can be used for the acute treatment of gout flares, depending on specific patient characteristics (Table 2). There is insufficient evidence to directly compare the efficacy of medicines for the treatment of gout flares.³ Medicine selection is therefore based on the patient's preference, renal function, the presence of co-morbidities, e.g. prednisone may be preferred over a NSAID or colchicine in a patient with reduced renal function, and the concurrent use of medicines that may interact with colchicine (see: "Particular care is required with colchicine"). If a patient is experiencing severe flares of gout, e.g. involving multiple joints, it may be appropriate to prescribe combination treatment, e.g. a NSAID with colchicine or corticosteroids with colchicine.²³

Once treatment for the acute flare has been completed, flare prophylaxis can be started or resumed if appropriate (for further information on flare prophylaxis, see: "Part 2: Controlling gout with long-term medicines" available from: bpac.org.nz/2021gout-part2.aspx).

N.B. The initiation of urate-lowering treatment can also be considered during an acute flare in some patients, see: "Talk about urate-lowering treatment before the patient leaves".

Provide a "pill in the pocket" for managing future flares

Patients with gout require ready access to medicines for managing flares until they achieve long-term symptom control with urate-lowering treatment.² It is often necessary to prescribe an extra quantity of medicine for this purpose;

emphasise to patients that they should stop taking the medicine when the flare has settled, unlike urate-lowering treatment which should be taken every day. Medicines should be stored in a secure and safe location at work and at home. Special care should be taken with colchicine as relatively small overdoses can be fatal. Patients should take medicines promptly for acute flares and those taking colchicine should do so within 12 hours of flare onset.³

Talk about urate-lowering treatment before the patient leaves

Urate-lowering treatment should be discussed with all patients with gout once a diagnosis has been established.³ This includes patients who are currently experiencing a gout flare, as they should be provided with the opportunity to manage their gout immediately, and some may not return for a follow-up consultation once the pain of the flare has resolved. The discussion should also cover the importance of titrating the dose of urate-lowering treatment over time for it to be effective.



Urate lowering medicines are not indicated for the treatment of asymptomatic hyperuricaemia.⁴

Aim to initiate urate-lowering treatment early

Patients with symptomatic hyperuricaemia and any of the following should start urate-lowering treatment:^{3,4,6}

- Two or more flares per year (this includes any flares the patient did not seek medical evaluation for)
- Tophi or erosions on X-ray
- Renal impairment (eGFR < 60 mL/min/1.73 m²)

Table 2: Treatment options for an acute gout flare.²²

Medicine	Dose	Additional notes
Naproxen	750 mg initially, 500 mg eight hours later, then 250 mg every eight hours until the flare has settled	Avoid if eGFR < 30 mL/min/1.73m ²
Prednisone	20 – 40 mg, once daily, for five days	Tapering the dose over 10 – 14 days can reduce the likelihood of a rebound flare, but tapering is not always necessary
Colchicine	Low dose regimen: 1 mg immediately, followed by 500 micrograms after one hour; maximum dose of 1.5 mg over a one-hour period. If eGFR 10 – 50 mL/min/1.73m ² , reduce the initial dose by half (i.e. 500 micrograms); do not exceed 1.5 mg over three days.	Do not repeat acute course within three days. Do not commence prophylaxis (very low dose colchicine) until 12 hours or more after the acute dose is taken. Colchicine should ideally be avoided, or used with caution, in frail patients, those who weigh < 50 kg, or patients with hepatic or renal impairment (eGFR 10 – 50 mL/min/1.73m ²). Colchicine is contraindicated in patients with an eGFR < 10 mL/min/1.73m ² .
Corticosteroid (triamcinolone acetonide)	Intra-articular injection, 2.5 – 40 mg	Dose determined by the size of the affected joint

- Past urolithiasis
- Serum urate level \geq 0.54 mmol/L

A randomised controlled trial has demonstrated that urate-lowering treatment in patients with early gout (with one or two prior flares) resulted in reduced incidence of gout flares and improved MRI-determined synovitis.²⁴ Patients who are initiated on urate-lowering treatment are less likely to require treatment for gout flares and are therefore less likely to experience adverse effects from repeated exposure to NSAIDs.

Initiation of urate-lowering treatment can be considered during a flare for some patients, but caution is required

The optimal timing of urate-lowering treatment initiation is still debated.²⁵ Traditionally, initiation of urate-lowering treatment has been delayed until the pain of a flare has resolved. The rationale being that dispersion of urate crystals during the initiation phase of treatment may make the patient's pain worse. There is now some limited evidence which suggests that initiating a urate-lowering treatment during a flare may have no significant impact on the duration of the flare or on its severity for some patients.^{4, 25, 26} As a result, the 2020 American College of Rheumatology (ACR) now conditionally recommend starting treatment during a flare rather than waiting for it to resolve. However, the two randomised controlled trials (RCT) used to justify this recommendation had very small cohorts, alongside strict exclusion criteria and medicine regimens that do not reflect how gout is usually managed in a New Zealand.²⁵ A third open-label RCT published in 2021 also found that early allopurinol treatment did not significantly worsen or prolong acute flares, however, the study population had considerably different baseline characteristics to people affected by gout in New Zealand.²⁶ While this strategy may be acceptable for some patients with early stage gout – particularly if they express a preference for starting long-term prevention as early as possible – further evidence is required before it can be conclusively recommended for all patient groups.

✔ Best practice tip: If a decision is made to initiate allopurinol during a flare, start at a low dose and ensure that the patient understands that they need to continue allopurinol after the flare has resolved, even when other medicines for treating the flare are ceased. Medicines used for the treatment of gout flares can be continued at lower doses for flare prophylaxis. In patients for whom urate-lowering treatment is indicated but allopurinol is not immediately initiated, ensure that either a prescription is written for them to pick up once the flare resolves, or that a follow-up appointment is scheduled.

Regular use is the key to long-term management


Explain to patients that the use of urate-lowering medicines needs to be regular and life-long to prevent flares of gout


Particular care is required with colchicine

Colchicine has a narrow therapeutic index meaning that the range between therapeutic and toxic effects is small and can overlap. Serious adverse effects associated with colchicine include paralytic ileus, myopathy, myocardial toxicity and blood dyscrasias. Colchicine is contraindicated in patients with significant gastrointestinal or cardiac conditions or pre-existing blood dyscrasias.²² The adverse effects of colchicine may also be exacerbated by medicine interactions.²² Caution is advised when prescribing colchicine to patients who are taking medicines that inhibit the CYP3A4 enzyme and/or P-glycoprotein, e.g. erythromycin, clarithromycin and verapamil.²² There have also been reports of myopathy and rhabdomyolysis in patients taking colchicine with statins.²² Colchicine is very toxic in overdose and there is no reversal agent; deaths have occurred with accidental overdose as low as 6 – 7 mg.

Prescribe the lowest effective dose of colchicine for the patient, and provide clear instructions on how and when to take it.⁴ Patients should be advised to stop taking colchicine and seek medical attention if they experience nausea, vomiting, diarrhoea or abdominal pain.²²

If the patient is taking very low dose colchicine for flare prophylaxis, this must be stopped during low dose colchicine treatment for an acute flare.

 Further information is available from: <https://bpac.org.nz/bpj/2014/september/safer-prescribing.aspx>

 The NZF interactions checker provides details on medicine interactions and their clinical significance, available from: www.nzf.org.nz



from returning. If initiation of urate-lowering treatment has been delayed until after a flare has been resolved, ensure that patients know that they should usually continue urate-lowering treatment during any future flares (assuming they have been adherent to allopurinol in the weeks and months leading up to the event). If urate-lowering treatment is stopped, even after years of being symptom-free, most patients will eventually experience a return of flares.³

 Keep reading: “Part 2: Controlling gout with long-term medicines” available from: [bpac.org.nz/2021gout-part2.aspx](https://www.bpac.org.nz/2021gout-part2.aspx)

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