




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 associated with a range of long-term negative health outcomes, e.g. cardiovascular disease, renal failure and reduced life expectancy
- The management of gout is frequently hindered by negative stereotypes and a reluctance to initiate urate-lowering treatment early
- Gout flares can be treated with a NSAID, prednisone or colchicine, depending on individual clinical circumstances
- Discuss urate-lowering treatment with all patients with gout at their first presentation and recommend early initiation
- Allopurinol can be initiated during an acute flare of gout; there is little evidence supporting delaying treatment until the flare has settled

 For further information about initiating and titrating allopurinol, see: **Part 2–Controlling gout with long-term urate-lowering treatment.**

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.³ When urate levels in the blood reach saturation point, monosodium urate crystals can form. 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 many patients this will be

caused by declining renal function.¹ Detecting chronic kidney disease (CKD) early and preserving renal function is therefore an important gout-prevention strategy. Additional factors that contribute to hyperuricaemia, and are associated with an increased risk of developing gout, include increasing age, male sex, hypertension, obesity, use of diuretics, antihypertensive medicines and low dose aspirin and excessive consumption of red meat, seafood, beer, spirits, sucrose or fructose-sweetened drinks.¹

The burden of poorly-controlled gout is often overlooked

Gout is much more than an intensely painful condition that prevents people from working, performing daily activities and participating in their communities.⁴ People with gout are also more likely than those without gout to die at a younger age due to cardiovascular and renal complications.¹ In New Zealand, 40% of people with gout have diabetes and/or cardiovascular disease.⁵ Despite this, many patients consider gout to be a condition that merely requires analgesics to control and are not aware of the potential long-term consequences.⁶ 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 gout flares are less likely, it may also help 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:⁷

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

A recent study found that patients with gout and diabetes who were taking urate-lowering treatment had significantly lower risk of myocardial infarction or stroke.⁸

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 they need to manage their health effectively. Furthermore, research suggests that disparities between how gout is managed in Māori and non-Māori is ingrained in the current model of care, with no reduction in the disparity between 2006/7 and 2012/13.⁹

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

The prevalence and burden of gout in New Zealand is higher in Māori and Pacific patients than in other groups. In 2014, 7.6% of Māori and 12.7% of Pacific peoples aged over 20 years were identified as having gout, compared to 4% of people of New Zealand European or Asian descent.¹⁰ The prevalence of gout increases with age; among males aged over 65 years the prevalence is 36.7% for Māori, 46.0% for Pacific peoples and 16.5% for people of New Zealand European or Asian descent.¹⁰ Māori patients with gout are dispensed more prescriptions for NSAIDs each year (60%) than New Zealand European-Other patients with gout (54%) and are therefore at greater risk of NSAID-related adverse effects, e.g. acute kidney injury and cardiovascular events.¹⁰ Māori and Pacific patients with gout are also five times more likely to be admitted to hospital due to gout than people of New Zealand European or Asian descent.¹⁰

Māori and Pacific patients with gout are often under-treated

Figure 1 shows that in 2017, 3.4% of Māori and 4.8% of Pacific patients were dispensed a urate-lowering medicine, compared to 2.2% of New Zealand Europeans.¹¹ However, the prevalence of gout is approximately twice as high in Māori and more than three times higher in Pacific peoples, compared to New Zealand European and Asian peoples.⁵ These results strongly suggest that gout in both Māori and Pacific peoples is under-treated.

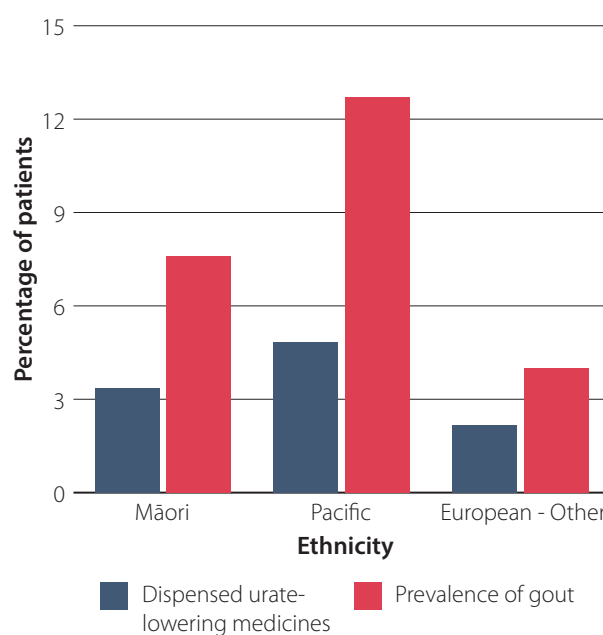


Figure 1: Percentage of enrolled patients dispensed allopurinol, benzbromarone, febuxostat or probenecid from a community pharmacy in New Zealand by ethnicity in 2017, compared to prevalence of gout in patients aged over 20 years in 2014^{10,11}

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.⁴ In the United Kingdom, a study of more than 52,000 patients with gout found that one year after diagnosis only 17% had a prescription for a urate-lowering medicine, at five years this had risen to 30%, and after ten years only 41% of patients had a prescription for a urate-lowering medicine.¹² A systematic review assessing the management of gout in primary care found that the proportion of patients with gout who were taking urate-lowering medicines was variable, depending on the study. However, six out of eight of the most robust studies found that less than 50% of patients with gout were taking urate-lowering treatment.¹³

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 found that in one study, one-quarter of patients received a serum urate test in the first six months of urate-lowering treatment, and in another study, only one-third of patients had been tested after the first year of urate-lowering treatment.¹³


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.¹⁴ 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.¹⁴

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 below). 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.¹⁵

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. 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 is a project involving community pharmacists and practice nurses in South Auckland. An electronic shared-care plan is initiated by a practice nurse, and community pharmacists use Standing Orders for titrating allopurinol in collaboration with the General Practice team. Further information is available from: <http://koawatea.co.nz/wp-content/uploads/2015/06/LS4-Manaaki-Hauora-Project-Review-Owning-My-Gout-March-2016.pdf>

Overcoming misconceptions that are barriers to managing gout

Perceptions and beliefs about gout can contribute to delays in initiating urate-lowering treatment.⁶ 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 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. Gout is a multi-factorial condition that is not solely caused by lifestyle factors. Genetic variations in uric acid renal transporters contribute significantly to the ethnic differences in gout prevalence.¹⁶ Explaining to Māori and Pacific 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". By actively managing their condition, e.g. making lifestyle changes and

maintaining treatment adherence, patients with gout can reduce their risk of cardiovascular and renal complications.

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 medicines such as allopurinol.

In the long-term, allopurinol can stop flares from happening. If patients adhere to urate-lowering treatment and serum urate levels are treated to target, flares of gout will be virtually eliminated for many patients within two years.¹⁷

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.⁴ 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 adherence to treatment 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

Diagnosing gout

Table 1 provides an example of a scoring system for the clinical diagnosis of gout. A score of eight or more is associated with a greater than 80% likelihood of gout.¹⁸ A score of four or less rules out gout in almost 100% of patients and an alternative diagnosis should be considered.¹⁸

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 the condition becomes more likely if the first onset of symptoms occur 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>

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.¹⁸

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. Although the gold standard for diagnosing gout is the presence of monosodium urate crystals under polarised microscopy,¹ 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. Patients should rest and elevate the affected joint during a gout flare and some may find the use of a ice pack beneficial.¹

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

Options for the treatment of gout flares are:^{22, 23}

- Naproxen 750 mg initially, 500 mg eight hours later, then 250 mg every eight hours until the flare has settled
- Prednisone 20 – 40 mg, once daily, for five days; tapering the dose over ten days can reduce the likelihood of a rebound flare, but tapering is not always necessary
- Colchicine 1 mg immediately, followed by 500 micrograms after one hour on day one, and then twice daily dosing of 500 micrograms*²²
- Triamcinolone acetonide intra-articular injection, 2.5 – 40 mg, determined by the size of the affected joint

* This is an alternative dose to the traditional regimen, which is now recommended by many experts. In patients with an estimated glomerular filtration rate (eGFR) < 50 mL/minute/1.73m² the initial dose should not exceed 1 mg in the first 24 hours, with a total maximum of 3 mg over four days

There is insufficient evidence to directly compare the efficacy of medicines for the treatment of gout flares.²² 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

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.²⁴ The adverse effects of colchicine may also be exacerbated by medicine interactions.²⁴ Caution is advised when prescribing colchicine to patients who are taking medicines that inhibit the CYP 3A4 enzyme and/or p-glycoprotein, e.g. erythromycin, clarithromycin and verapamil.¹ There have also been reports of myopathy and rhabdomyolysis in patients taking colchicine with statins.¹ 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. Patients should be advised to stop taking colchicine and seek medical attention if they experience nausea, vomiting, diarrhoea or abdominal pain.²⁴

Further information is available from: 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



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.²²

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. 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.²²

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.²² 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.

Aim to initiate urate-lowering treatment early

Patients with hyperuricaemia and the following characteristics should start urate-lowering treatment:^{1,22}

- Two or more flares per year
- Tophi or erosions on X-ray
- Renal impairment
- Kidney stones

If the patient is presenting for the first time with a gout flare, ask about any prior episodes that they may have treated themselves that are likely to have been gout; frequency and severity of gout can often be underestimated and therefore under-treated. A recently published 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.²⁵ 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.

Practice changing point: urate-lowering treatment can be initiated during a flare

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. However, there is little evidence to support this delay and two randomised controlled trials have found no increase in pain, flares or markers of inflammation when allopurinol was initiated during a flare, compared to waiting ten days.^{26,27}

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.⁴ 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.



Best practice tip: If allopurinol is initiated 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. In some cases medicines used for the treatment of gout flares will be continued at lower doses for flare prophylaxis.

Adherence is the key to long-term management

Explain to patients that adherence to urate-lowering medicines needs to be life-long to prevent flares of gout 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 continue urate-lowering treatment during any

future flares. If urate-lowering treatment is stopped, even after years of being symptom-free, most patients will experience a return of flares within four years.²⁸

Keep reading: Part 2 – Controlling gout with long-term medicines.

Acknowledgement: Thank you to **Professor Nicola Dalbeth**, Rheumatologist Auckland DHB and School of Medicine, University of Auckland and **Professor Lisa Stamp**, Rheumatologist, Department of Medicine, University of Otago, Christchurch for expert review of this article.

References

- Hui M, Carr A, Cameron S, et al. The British Society for Rheumatology Guideline for the Management of Gout. *Rheumatology* 2017;56:1246. doi:10.1093/rheumatology/kex250
- Qaseem A, Harris RP, Forciea MA, et al. Management of Acute and Recurrent Gout: A Clinical Practice Guideline From the American College of Physicians. *Ann Intern Med* 2017;166:58–68. doi:10.7326/M16-0570
- Maiuolo J, Oppedisano F, Gratteri S, et al. Regulation of uric acid metabolism and excretion. *Int J Cardiol* 2016;213:8–14. doi:10.1016/j.ijcard.2015.08.109
- Te Karu L, Bryant L, Elley CR. Maori experiences and perceptions of gout and its treatment: a kaupapa Maori qualitative study. *J Prim Health Care* 2013;5:214–22.
- Winnard D, Wright C, Jackson G, et al. Gout, diabetes and cardiovascular disease in the Aotearoa New Zealand adult population: co-prevalence and implications for clinical practice. *N Z Med J* 2012;126:53–64.
- Spencer K, Carr A, Doherty M. Patient and provider barriers to effective management of gout in general practice: a qualitative study. *Ann Rheum Dis* 2012;71:1490–5. doi:10.1136/annrheumdis-2011-200801
- Su X, Xu B, Yan B, et al. Effects of uric acid-lowering therapy in patients with chronic kidney disease: A meta-analysis. *PLoS ONE* 2017;12:e0187550. doi:10.1371/journal.pone.0187550
- Singh JA, Ramachandran R, Yu S, et al. Allopurinol use and the risk of acute cardiovascular events in patients with gout and diabetes. *BMC Cardiovasc Disord* 2017;17:76. doi:10.1186/s12872-017-0513-6
- Auckland UniServices Limited. Supply of quantitative research and analysis services: Variation in medicines use by ethnicity: a comparison between 2006/7 and 2012/13. 2018. Available from: www.pharmac.govt.nz/assets/2018-01-19-Variation-in-medicines-use-by-ethnicity-Final-Report.pdf (Accessed Apr, 2018)
- Health Quality & Safety Commission New Zealand. Gout. 2016. Available from: www.hqsc.govt.nz/our-programmes/health-quality-evaluation/projects/atlas-of-healthcare-variation/gout (Accessed Apr, 2018)
- Ministry of Health. Pharmaceutical Claims Collection. 2016.
- Kuo C-F, Grainge MJ, Mallen C, et al. Eligibility for and prescription of urate-lowering treatment in patients with incident gout in England. *JAMA* 2014;312:2684–6. doi:10.1001/jama.2014.14484
- Jeyaruban A, Larkins S, Soden M. Management of gout in general practice—a systematic review. *Clin Rheumatol* 2015;34:9–16. doi:10.1007/s10067-014-2783-z
- Humphrey C, Hulme R, Dalbeth N, et al. A qualitative study to explore health professionals' experience of treating gout: understanding perceived barriers to effective gout management. *J Prim Health Care* 2016;8:149–56. doi:10.1071/HC15017
- Rees F, Jenkins W, Doherty M. Patients with gout adhere to curative treatment if informed appropriately: proof-of-concept observational study. *Ann Rheum Dis* 2013;72:826–30. doi:10.1136/annrheumdis-2012-201676
- Flynn T. Experiencing Gout in New Zealand: The Genetic and Dietary Influences of a Complex Disease. 2016. Available from: <https://ourarchive.otago.ac.nz/handle/10523/6661?show=full> (Accessed Jan, 2018)
- Perez-Ruiz F. Treating to target: a strategy to cure gout. *Rheumatology* 2009;48 Suppl 2:ii9–ii14. doi:10.1093/rheumatology/kep087
- Janssens HJEM, Franssen J, van de Lisdonk EH, et al. A diagnostic rule for acute gouty arthritis in primary care without joint fluid analysis. *Arch Intern Med* 2010;170:1120–6. doi:10.1001/archinternmed.2010.196
- Dalbeth N, Winnard D, Gow PJ, et al. Urate testing in gout: why, when and how. *N Z Med J* 2015;128:65–8.
- Horowitz DL, Katzap E, Horowitz S, et al. Approach to septic arthritis. *Am Fam Physician* 2011;84:653–60.
- Rosenthal AK, Ryan LM. Calcium Pyrophosphate Deposition Disease. *N Engl J Med* 2016;374:2575–84. doi:10.1056/NEJMra1511117
- Richette P, Doherty M, Pascual E, et al. 2016 updated EULAR evidence-based recommendations for the management of gout. *Ann Rheum Dis* 2017;76:29–42. doi:10.1136/annrheumdis-2016-209707
- New Zealand Formulary (NZF). NZF v70. 2018. Available from: www.nzf.org.nz (Accessed Apr, 2018)
- MEDSAFE. Spotlight on colchicine. Prescriber update 2018;39:8–9.
- Dalbeth N, Saag KG, Palmer WE, et al. Effects of Febuxostat in Early Gout: A Randomized, Double-Blind, Placebo-Controlled Study. *Arthritis Rheumatol* 2017;69:2386–95. doi:10.1002/art.40233
- Taylor TH, Mecchella JN, Larson RJ, et al. Initiation of allopurinol at first medical contact for acute attacks of gout: a randomized clinical trial. *Am J Med* 2012;125:1126–1134.e7. doi:10.1016/j.amjmed.2012.05.025
- Hill EM, Sky K, Sit M, et al. Does starting allopurinol prolong acute treated gout? A randomized clinical trial. *J Clin Rheumatol* 2015;21:120–5. doi:10.1097/RHU.0000000000000235
- Perez-Ruiz F, Herrero-Beites AM, Carmona L. A two-stage approach to the treatment of hyperuricemia in gout: the 'dirty dish' hypothesis. *Arthritis Rheum* 2011;63:4002–6. doi:10.1002/art.30649



This article is available online at:
www.bpac.org.nz/2018/gout-part1.aspx