# A close up of a logo Description automatically generatedQuestions and answers from the webinar ‘Equitable approaches to gout management’

The Health Quality & Safety Commission held a webinar on 1 July 2020 focused on equitable approaches to gout management.

The following questions were asked during the event or were unable to be answered due to time constraints. All questions have been answered and endorsed by the webinar panel.

## Medication dosing

**How often do you repeat serum urate levels when up-titrating allopurinol?**

Normally, I test the urate level each month or so while titrating the dose up. Once the urate is <0.36mmol/L, the recommendation is to check every 6–12 months.

**If a person has been on long term allopurinol and their urate levels are extremely low ie, below 0.2mmol/l, should the dose be reduced?**

**Is there any risk associated with low serum urate levels?**

I generally try to keep the urate above 0.20mmol/L. It’s probably not a big problem to have a level <0.20mmol/L, but some observational studies (Yu et al 2017) have suggested a risk of Parkinson’s disease with very low urate levels.

**Is it good practice to delay starting allopurinol for 14 days after initial gout flare if patient allopurinol naïve, and start with solely NSAIDS/prednisone or colchicine?**

Quite controversial! Several small RCTs (Taylor et al 2012; Hill et al 2015) have shown that starting allopurinol during a flare does not lead to worsening of the flare. I’m usually comfortable starting allopurinol at low dose, provided there is plenty of anti-inflammatory medication on board.

**How long after a gout flare would it be appropriate to check the urate level, as during a flare it would be lower?**

Generally, about two weeks after the flare has resolved.

**You say, ideally, urate is checked monthly to help up-titrate allopurinol. Due to the cost of blood tests this may be an issue for patients. Are there any suggestions around this issue?**

Point-of-care testing, as used in the Owning My Gout programme, are an option if obtaining bloods is difficult. Alternatively, you can dose titrate to a likely dose, then test urate, and increase the dose further if serum urate remains elevated.

**If the person has had low urate levels for a few months/years after starting allopurinol, is it still necessary to continue with the allopurinol?**

Long-term treatment is important. Within a few days of stopping allopurinol, the urate will be elevated again, with a risk of forming more crystals. The key benefits from allopurinol occur when the medication is used long-term to maintain the urate at a normal level, which will lead to crystals dissolving and preventing flares.

**I have noticed that both colchicine and allopurinol are sometimes prescribed together. Is this appropriate?**

Yes completely, unless the patient experiences side effects from daily colchicine. Colchicine can be prescribed to prevent gout flares for the first 3–6 months of establishing allopurinol. We found that patients who had urate >0.5 sometimes required ongoing prednisone 5mg or ongoing PRN naproxen to manage flares while titrating.

**Our pathways say colchicine should only be started within 72hrs of attack, do you follow that?**

Colchicine generally works best for a gout flare when started early. However, it is also used to prevent gout flares when starting allopurinol (prophylaxis), so this can be started any time for prophylaxis. In the Stop Gout programme, colchicine is started after two weeks of prednisone for the acute attack.

## Stop Gout/Owning My Gout programmes

**What does the script fund cover?**

The Stop Gout script fund covers the prescription co-payments of $15 (3x$5) for the three medicines given over the three-month programme.

**Is this programme in Northland District Health Board (DHB) only? Is there any chance it can be rolled out across the country?**

Gout Stop is only in Northland, Owning My Gout is only in Counties Manukau. It would be great to be national. With all DHBs required to address equity issues, gout should be a key condition to work on and, if not nationally, in those with the highest population of Māori/Pacific peoples as a priority.

**Did the preset scripts need to be loaded onto each practice’s patient management system individually?**

For the Stop Gout programme the primary health organisation, Mahitahi, loaded preset prescriptions remotely, so we just went into practices to show them how to find and use them.

**What was the cost to the patient?**

For Gout Stop, the cost to the patient was the GP consultation fee and prescription co-payment of $15. The prescription co-payment could be waived under the script fund if there was financial hardship.

**Does this initiative cover other long-term gout medications eg, probenecid for those who are intolerant or allergic to allopurinol?**

If people had severe side effects to allopurinol they were removed from the Owning My Gout programme and other therapies were trialed.

**Which DHBs was the Owning My Gout programme in?**

The Owning My Gout programme ran at Counties Manukau Health.

**In the programme what medicines are used in addition to allopurinol?**

In the Stop Gout programme, prednisone in the acute phase and colchicine daily ongoing while titration occurs.

**Are doses of allopurinol adjusted or modified as per renal function?**

Yes, the allopurinol doses were adjusted as recommended in the guidelines on HealthPathways.

## Long-term conditions or comorbidities

**Can we comment on treating urate in context of lowering cardiovascular disease (CVD) risk?**

Data are still unclear whether urate contributes directly to CVD, and I wouldn’t start urate lowering therapy to prevent CVD for asymptomatic hyperuricemia. However, reducing exposure to NSAIDs by preventing gout flares may have some benefits in reducing CVD risk (plus chronic kidney disease (CKD) risk!). The CARES trial of people with gout indicated that allopurinol may be safer than febuxostat for patients with established CVD.

**Treatment of asymptomatic hyperuricemia is not currently recommended. Are we not concerned about the long-term association of increased cardiovascular risk, renal effects, diabetes etc?**

There is not enough evidence to treat asymptomatic hyperuricemia currently, but it will be interesting to watch this space, if this prevention was to occur nationally, what the impact might be on CVD rates.

## Gout management

**Role of HLA B\*5801 in different ethnic group to allopurinol?**

I test this for Chinese and other Asian patients. I would avoid allopurinol if B\*5801 positive.

**Just wondering about high rates of gout and genetics of Māori/Pacific. Was gout an historic issue? I’m thinking kaimoana would’ve been a vital source of food and purines? Has the modern processed diet increased gout rates?**

There are genetic differences which influence excretion of uric acid from the kidneys. Dietary habits only contribute 10–20 percent whereas genes and other biological factors (eg, CKD, medications) 80–90 percent. In addition to changes of diet with ‘modern living’, there is also more CKD, CVD, diuretics, and this may also be contributing to the increasing prevalence of gout in NZ and worldwide.

**I’ve heard tonight that dietary factors only contribute 20 percent to uric acid level. What advice would you give the patient with gout who enjoys 1 beer per day?**

Take urate lowering therapy and continue to enjoy your beer or kaimoana!

**I had a home visit today with an 18-year-old patient who lives with his koro, who also suffers long-term gout, nana was out in the kitchen listening. The kōrero about including whānau to make this work is so important to instigate understanding and compliance.**

Working with the whānau is so important. Changing understanding about gout within one whānau has such a flow on effect in the community.

**Have you thought about introducing nutritionists or dieticians into this initiative since diet is important and affects gout?**

The programmes did not include dieticians. The evidence for effectiveness of dietary management in changing serum urate is weak (Major et al 2018). Dietary changes only lead to small reductions in serum urate and spending a lot of time focusing on diet can deflect from the key messages about effective treatments such as allopurinol. However, you are right that the most common view in the community is that food is the cause of gout.

Bearing in mind the overwhelming evidence (Tin et al 2019; Major et al 2018; Hollis-Moffatt et al 2009; Phipps-Green et al 2016; Tanner et al 2017) about genetic factors, particularly for Māori and Pacific peoples, we need to provide information about how it is not the person’s fault and instead focus on the role of the kidneys.

**These kinds of interventions need to include lifestyle modification advice which will have wider benefits for patients than gout reduction.**

When I am talking to a person with gout, especially a younger person, my primary focus is to get them onto urate lowering therapy. Once urate lowering therapy is established in a sustainable way then I will have a conversation about lifestyle issues in a way that doesn’t blame the person.

**I work as a long-term condition (LTC) nurse and I believe the main problem with gout and other LTC s, comes down to the patient’s lack of understanding of their medications and conditions. Health literacy. Time constraints within general practice. Cost. All of what has been addressed in both programmes. I am in a role that allows this time and I have obtained great engagement from patient and whānau and improved health outcomes.**

Health professionals have a big role to play in building people’s health literacy however building health literacy is the responsibility of the whole system as outlined in the Framework for Health Literacy: <https://www.health.govt.nz/publication/framework-health-literacy>.

An article published in *NZMJ Digest* (Health Literacy NZ 2020) explains what the term ‘low health literacy’ means in relation to individuals and whānau.

**I wonder whether and where the weakest link of gout management is. We must identify the weakest link whether it’s adherence to medication, misunderstanding about medication, misinformation about side effect of NSAID vs allopurinol long term. So, I wonder whether involvement of pharmacists can be another target of improvement of equity.**

As we said on the night you have identified a whole lot of issues relating to medication which supports the involvement of pharmacists. Once people understand the need to take urate-lowering medicine then the involvement of pharmacists to titrate and support persistence is critical.

**Can we have access to the link for the study re nurse led clinics?**

A UK study highlights the effectiveness of nurse-led clinics for gout management (Doherty et al 2018).

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