

Gout update

Dave Cox. Long-term Conditions Nurse

Gout





He mana tō te whānau
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Tōkeke
Equitable

Manawa whakaute
Respectful

Pono
Transparent



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Managing Gout - It's not rocket science

Contents.

- What is gout?
- Gout in Aotearoa/ New Zealand
- Risk factors
- Diagnosis
- Treating an acute attack
- Prevention of further attacks
- Self-management
- CPPD (Pseudogout)
- Further resources and help
- Summary

Gout



What is gout?

- Gout is an arthritis caused by the inflammatory response to intra-articular monosodium urate crystals.
- In early disease, gout presents as recurrent episodes of self-limiting acute inflammatory attacks ('flares') of arthritis.
- These attacks most often affect the 1st metatarsophalangeal joint (Podagra), midfoot and ankle.
- In the presence of prolonged hyperuricaemia, some patients develop recurrent polyarticular attacks, chronic tophaceous disease, erosive arthritis and renal disease (urate nephropathy and uric acid stones) (1)

Typical acute attack (Podagra)



Atlas of Healthcare Variation | Gout



Key finding
200,000 people were identified as having gout in 2018.

[Method](#)

[Help](#)

[Click for commentary](#)



Summary

New Zealand: 1. Prevalence of identified gout, percent

Select indicator and filters

- 1. Prevalence of identified gout, percent
- 2. Urate-lowering therapy use in people with gout, percent
- 3. Regularly receiving urate-lowering therapy, percent
- 4. NSAID use in people with gout, percent
- 5. NSAID but no urate-lowering therapy, percent
- 6. People with gout dispensed any of colchicine, prednisone or NSAID, percent
- 7. People with gout dispensed any of colchicine, prednisone or NSAID but no ...
- 8. Serum urate test in 6 months of urate-lowering therapy, percent
- 9. Hospital admissions with primary diagnosis of gout, rate per 100,000

Age group:

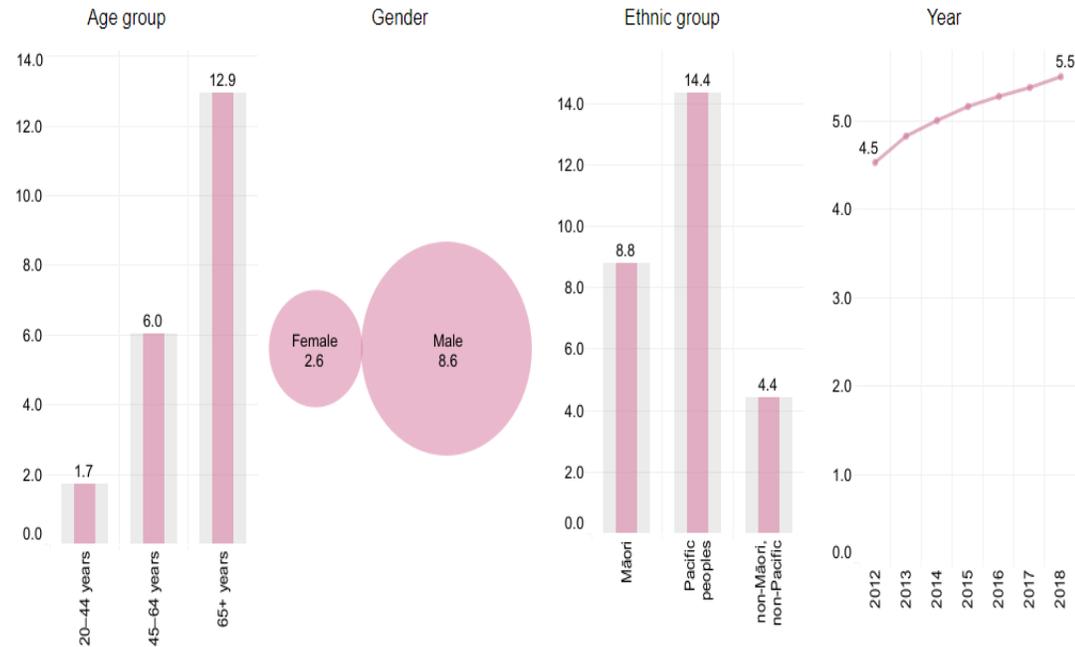
Gender:

Ethnic group:

Year:

District health board (DHB):

■ Selected DHB ■ New Zealand



PHARMAC insights

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Gout insights Impact on Māori

Establishing the baseline: November 2021

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Pacific peoples health - Gout data insights

Establishing the baseline: April 2022

Risk factors for gout

- Hyperuricaemia
- Male sex
- Māori and Pacific ethnicity
- Chronic renal impairment
- Hypertension
- Obesity
- Diuretic use
- Coronary heart disease

Gout and genetics

- Recent research has found that variants in specific genes are associated with a reduced ability for renal excretion of uric acid, and therefore increased risk of hyperuricaemia.
A variant within the SLC2A9 gene increases the risk of gout by more than five times in Maori and Pacific peoples, and a variant within the ABCG2 gene increases the risk in European and Pacific Peoples but not in Maori ^(4, 5)

Diagnosis of gout

Acute pain and swelling, often in 1st MCP joint (70% of first episodes present this way.)

Sometimes with fever and malaise.

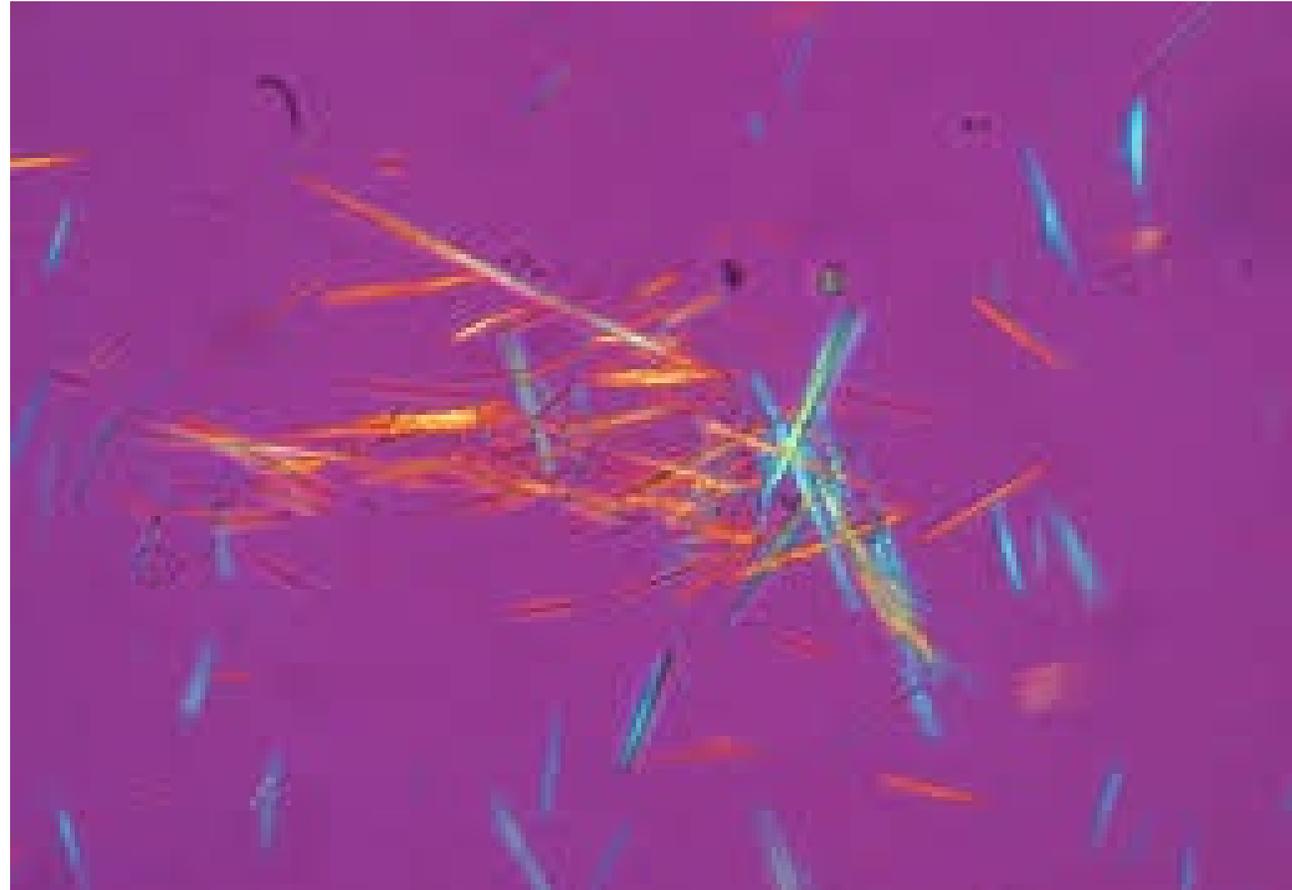
If in any doubt- joint aspiration is the 'gold standard'

**The presence of characteristic urate crystals
in the joint fluid.**

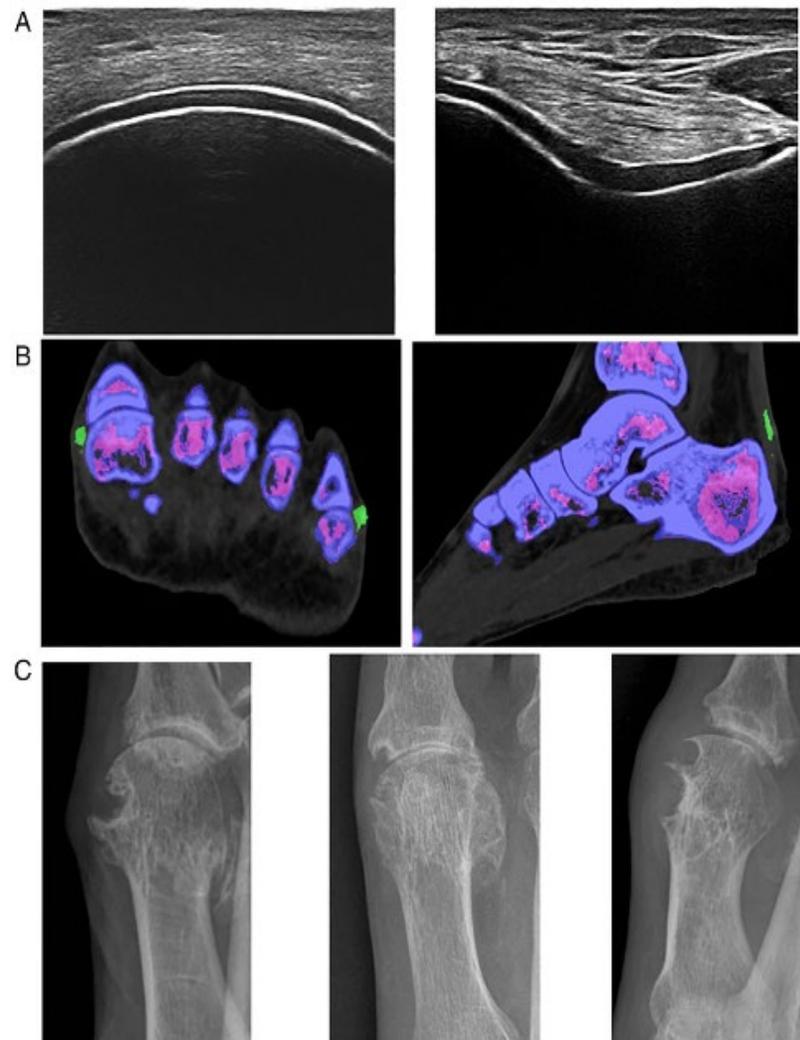
**A tophus proved to contain urate crystals
by chemical means or polarized light microscopy**

***RED FLAG - Sepsis**

Monosodium urate crystals



Imaging



Treatment summary for acute gout

- Exclude sepsis
- Prescribe NSAID for pain and inflammation and advise rest
- If NSAIDs are contraindicated and infection has been excluded, consider corticosteroids (oral or intra-articular) or colchicine. Steroids can be used for acute gout in patients with diabetes, but may need careful blood glucose management.
- If further pain relief is required, consider combination treatment and recheck diagnosis

Allopurinol

- Allopurinol remains the first choice of urate-lowering therapy (ULT), unless contra-indicated.
- Xanthine oxidase inhibitor
- Dosing can be difficult and should be calculated on estimated glomerular filtration rate (eGFR) ⁽¹¹⁾
- Allopurinol can be commenced during an acute attack
- Allopurinol should be continued if acute attack occurs

Suggested starting dose of Allopurinol – current guidelines on Southern Community HealthPathways

- Start low and go slowly.
- If eGFR is below 20, request a [non-acute rheumatology assessment](#).
- Decide on an appropriate starting dose, based on eGFR:
 - If eGFR 20 to 30, start 50 mg alternate days.
 - If eGFR 31 to 60, start 50 mg daily.
 - If eGFR above 60, start 100 mg daily.
- Increase dose by increments [every 4 weeks](#) based on eGFR, aiming for a target serum urate of less than 0.36 mmol/L.
 - If eGFR is 60 or less, increase by 50 mg.
 - If eGFR is above 60, increase by 100 mg.
 - The maximum dose of allopurinol is 900 mg per day.
 - The dose need not be limited by kidney function – seek [rheumatology advice](#) if there are concerns about dose.

Adverse effects with Allopurinol

The most common adverse effect is a rash (1–2%), which may be more common in patients with renal impairment.

Allopurinol Hypersensitivity Syndrome (AHS) is extremely rare but potentially fatal. It is characterised by fever, rash, eosinophilia, hepatitis and renal failure.

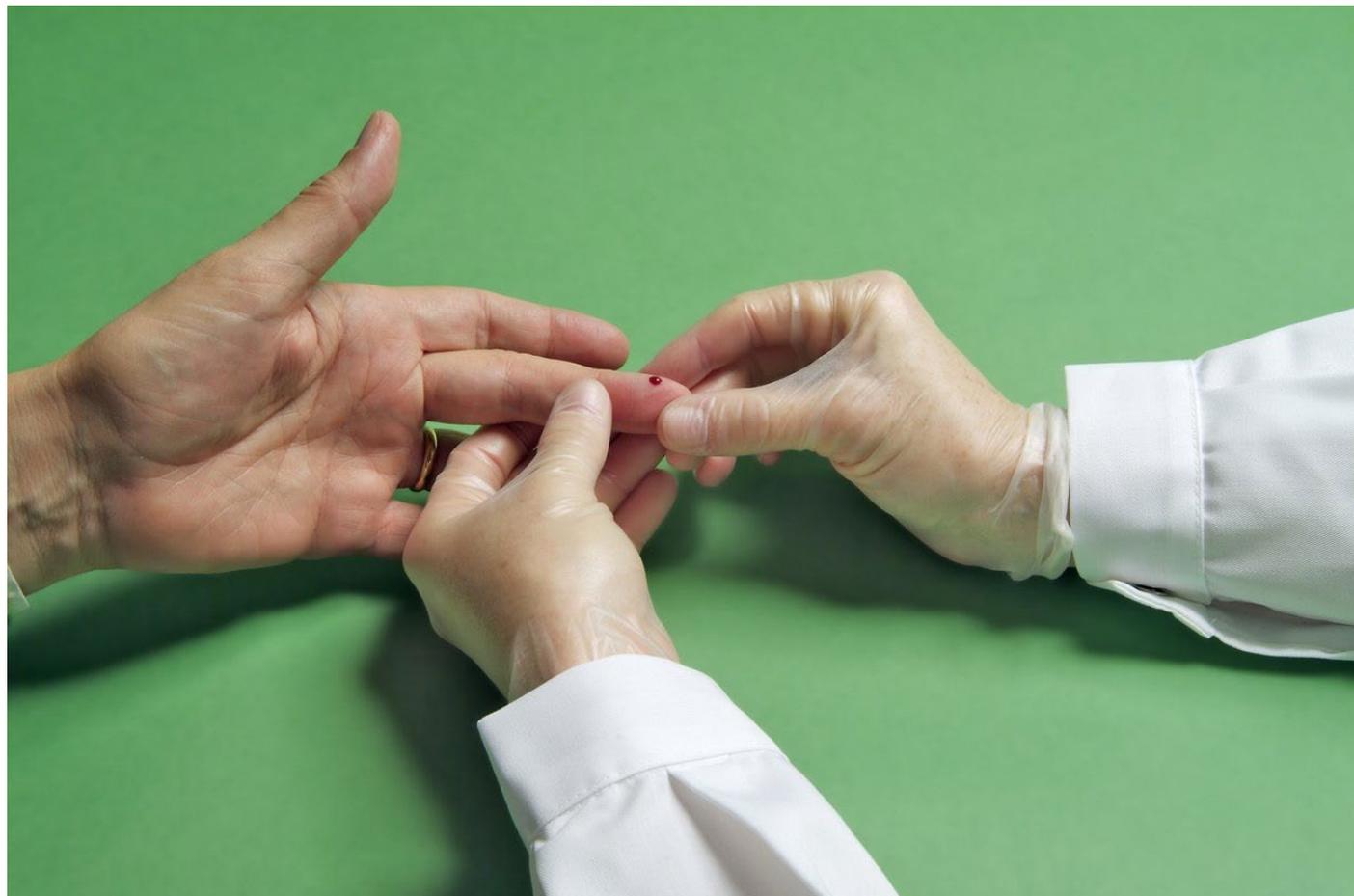
Consider HLA –B*5801 screening for people of Korean, Han Chinese and Thai descent. (HLA – B*5801 +ve = Significant increase in AHS risk)

Adverse effects can occur at any dose.

Treat to target!

- Aim for target serum urate level of $<0.36\text{mmol/l}$
- For tophaceous gout aim for lower target (0.30mmol/l)
(*European guidelines)
- Non-adherence to ULT is a challenge.
- Flares can persist up to 12 months once target achieved.

Point of care testing



Colchicine

Colchicine has a narrow therapeutic margin and considerable variation in absorption between individuals. Toxic effects include diarrhoea, nausea and vomiting, electrolyte imbalance, alopecia, haematological effects, pancreatitis, and failure of kidneys, liver or respiratory system. High doses can be fatal.

Using Colchicine in an acute attack - Current advice on Southern Community HealthPathways

- **Colchicine**
- Use 1 mg immediately, then 0.5 mg one hour later, and then 0.5 mg twice a day. Never exceed this dose.
- Warn patients to discontinue if they get diarrhoea.
- Avoid if severe renal or hepatic impairment.
- Check for contraindications (some medications)

Probenecid

- The uricosuric agent probenecid is an effective urate lowering drug with normal renal function and urate under-excretion
- It is useful in combination with allopurinol if there is persistent hyperuricaemia despite therapeutic doses of allopurinol, or in allopurinol intolerance. ⁽¹²⁾
- The typical dose is 250mg BD for 2 weeks, then 500mg BD thereafter
- Probenecid is contraindicated in patients with a history of renal stones
- Patients need to be advised to ensure high fluid intake while taking probenecid (about 2 x litres of water a day)

Febuxostat

- Second-line ULT
- Dose need not be adjusted in mild-moderate renal impairment (ie creatinine clearance > 30 mL/min) (13)
- May be more effective than fixed dose allopurinol
- Appears to be well tolerated in older patients.

Febuxostat risks ⁽¹³⁾

- Main risk is hepatotoxicity
- May increase cardiovascular risk
- Hypersensitivity reactions
- Prophylaxis for gout flares needs to be considered on initiation of treatment

Prescribing Febuxostat ⁽¹³⁾

- Ensure an adequate trail of allopurinol and probenecid.
- Start febuxostat at 80mg daily.
- Co-prescribe prophylaxis with low dose colchicine or a low dose NSAID when starting febuxostat.
- Monitor LFTs
- Should be continued in acute attack
- Encourage adherence to all urate lowering therapies.

Recommendations for optimal use of urate lowering therapies (ULT)(19)

- ULT should be explained to patients, when the diagnosis is confirmed, as a treatment to continually prevent the return of gout.
- ULT should be discussed and offered to patients who have a diagnosis of gout, especially in those who have > 2 attacks in 12 months, tophi, chronic gouty arthritis, joint damage, renal impairment, urolithiasis.

Recommendations for optimal use of urate lowering therapies (ULT)

- Aim of ULT is to reduce and maintain the sUA to or below a target of 0.36mmol/l (0.30mmol/l in tophaceous gout)
- Allopurinol is the first-line ULT to consider. It should be started at a low dose (50-100mg daily) and then the dose increased, if necessary, in 50-100mg increments approximately every 4 weeks until the target sUA has been achieved, (maximum dose 900mg daily)

Recommendations for optimal use of urate lowering therapies (ULT)

- Febuxostat can be used as a second-line xanthine oxidase inhibitor for patients in whom allopurinol is not tolerated, or whose renal impairment prevents allopurinol dose escalation sufficient to meet therapeutic target
- Start at a dose of 80mg daily and, if necessary, increase to 120mg daily after 4 weeks to achieve therapeutic target.

Recommendations for optimal use of urate lowering therapies (ULT)⁽¹⁹⁾

- Uricosuric agents can be used for patients who are resistant to, or intolerant of xanthine oxidase inhibitors in patients who have normal or mildly impaired renal function
- Colchicine 500µg OD or BD should be considered as prophylaxis against acute attacks resulting from initiation or up-titration of any ULT.
- For patients who cannot tolerate colchicine, a low –dose NSAID or coxib, with gastroprotection, can be used as an alternative, provided there are no contra-indications.
- Monthly serum urate and renal function checks until target serum urate is achieved.
- *Note: SGLT2 inhibitors used in Type 2 diabetes management can also lower serum urate*

Lifestyle modification(21)

Encourage:

- Maintain ideal weight
- Drink at least 2L of water per day
- Exercise moderately, but rest, elevate and cool affected joints during an acute attack
- Include low fat dairy, soy, vegetable proteins and foods high in Vit C in their diet.

Lifestyle modification(21)

Discourage

- Dehydration
- Alcohol, particularly beer (if not avoided, then limit intake)
- Excess intake of purine- rich foods, eg red meat and offal (liver and kidneys), shellfish, oily fish, yeast extracts
- Soft drinks containing sucrose and fructose which interfere with tubular excretion of urate.

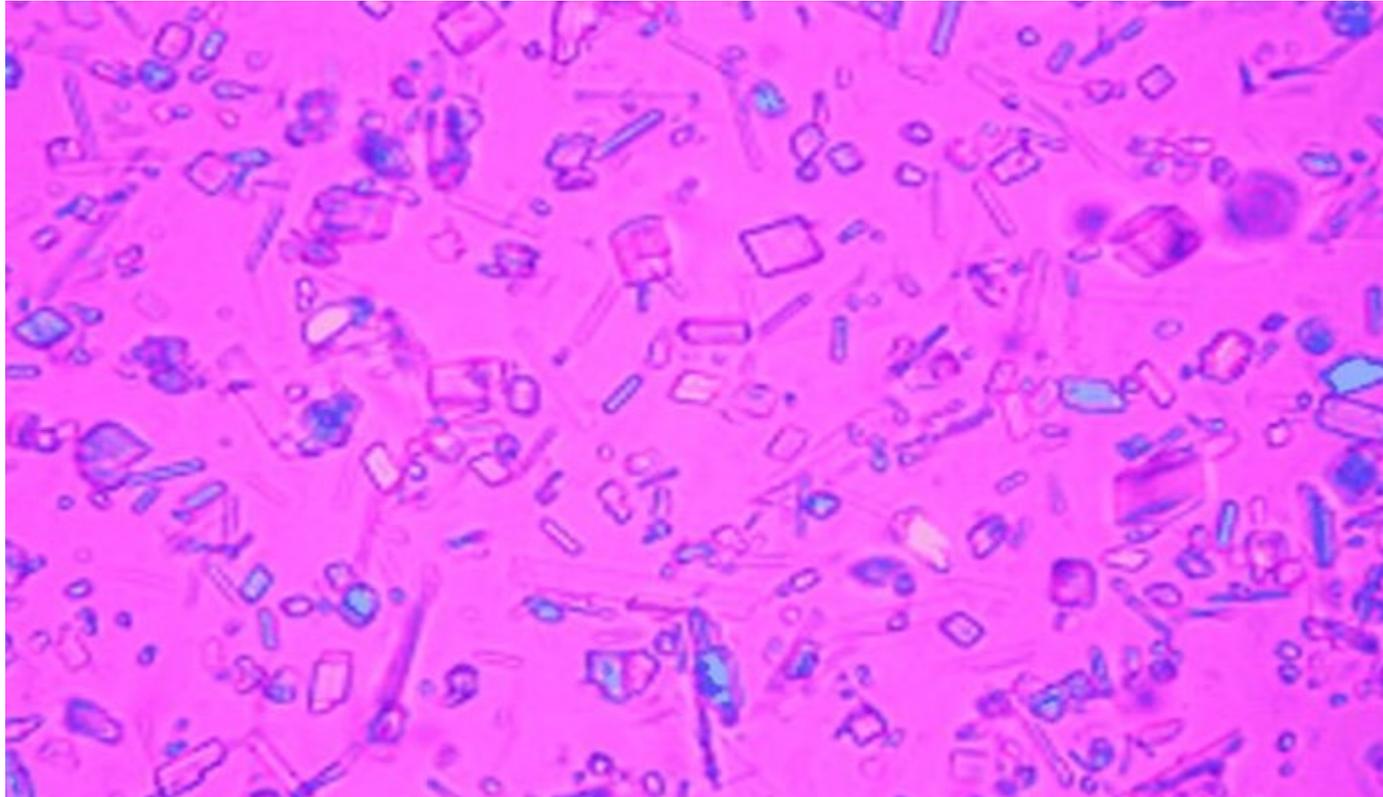
When to refer to a Rheumatologist ⁽²⁰⁾

- Persistent hyperuricaemia or gout attacks despite maximum tolerated ULT treatment.
- Doubt about diagnosis
- Failure to achieve prompt resolution of acute attacks
- Development of progressive bone and joint damage on X-ray

CPPD (Pseudogout)

- Calcium pyrophosphate dihydrate crystal deposition disease (**CPPD**) is a form of arthritis that causes pain, stiffness, tenderness, redness, warmth, and swelling (inflammation) in some joints. It usually affects one joint at a time, but sometimes it may affect several joints at once.

CPPD crystals



Southern Community HealthPathways

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- Infectious Diseases ▾
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- Nephrology ▾
- Neurology ▾
- Oncology ▾
- Pain Management ▾
- Palliative Care ▾
- Respiratory ▾
- Rheumatology ▴
- DMARD and Immunosuppressive Initiation ▾
- Fibromyalgia ▾
- Giant Cell Arteritis (GCA) or Temporal Arteritis
- Gout
- Inflammatory Arthritis
- Polymyalgia Rheumatica (PMR)
- Spondyloarthritis

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Gout

Red flags ?

▶ Exclude septic arthritis in a single swollen painful joint. The history and examination in gout and septic arthritis may be similar. Arrange joint aspiration if any doubt.

Background

About gout ▾

Assessment

In an acute attack:

1. Typical history presents with an acute swollen tender joint over a 6 to 12 hour period. Often associated with fever and malaise.
2. Ask about previous episodes and self-treatment.
3. 1st MTP joint is involved in 50% of all attacks, and 70% of first attacks. Other common joints are knee, foot, wrist, ankle, hand, and elbow.
4. Check for systemic symptoms, fever, whether other joints are involved, or any **tophi** ▾ are present.
5. If serum urate is measured, note that levels may be normal in up to 50% of people with acute gout. Check renal function if not done within 3 months.
6. Consider the possibility of **septic arthritis** or **pseudogout** (calcium pyrophosphate deposition)

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Further resource for health professionals

Gout Guide

Tools and resources to
improve gout outcomes



Welcome to the Gout Guide for health providers

"Gout is so treatable compared to other long-term conditions. Treatment is so important for our patients so they can work and participate in family life and their communities." – Dr Harley Aish, Otago Family and Christian Medical Centre.

The Gout Guide is a dynamic, must-have resource for GPs, nurses, pharmacists, health coaches and all healthcare teams in Aotearoa New Zealand!

As primary care leaders, we're on a shared mission to improve gout outcomes equitably. Let's rethink our strategies and stimulate conversation about gout within our teams and with whānau Māori and Pacific communities.

The Gout Guide builds on findings from several gout projects including the Whanganui GOUT STOP programme and ProCare Gout Collaborative. It provides practical tools and insights for a fresh take on gout treatment. Completed in June 2023, the guide developed following a project funded by Te Whatu Ora, Long-Term Conditions Directorate. Ongoing support is provided by Health Literacy NZ and Health Navigator Charitable Trust. [Read more.](#)

Join us on this journey to equity and excellence in gout care. Together, we'll make a big difference!

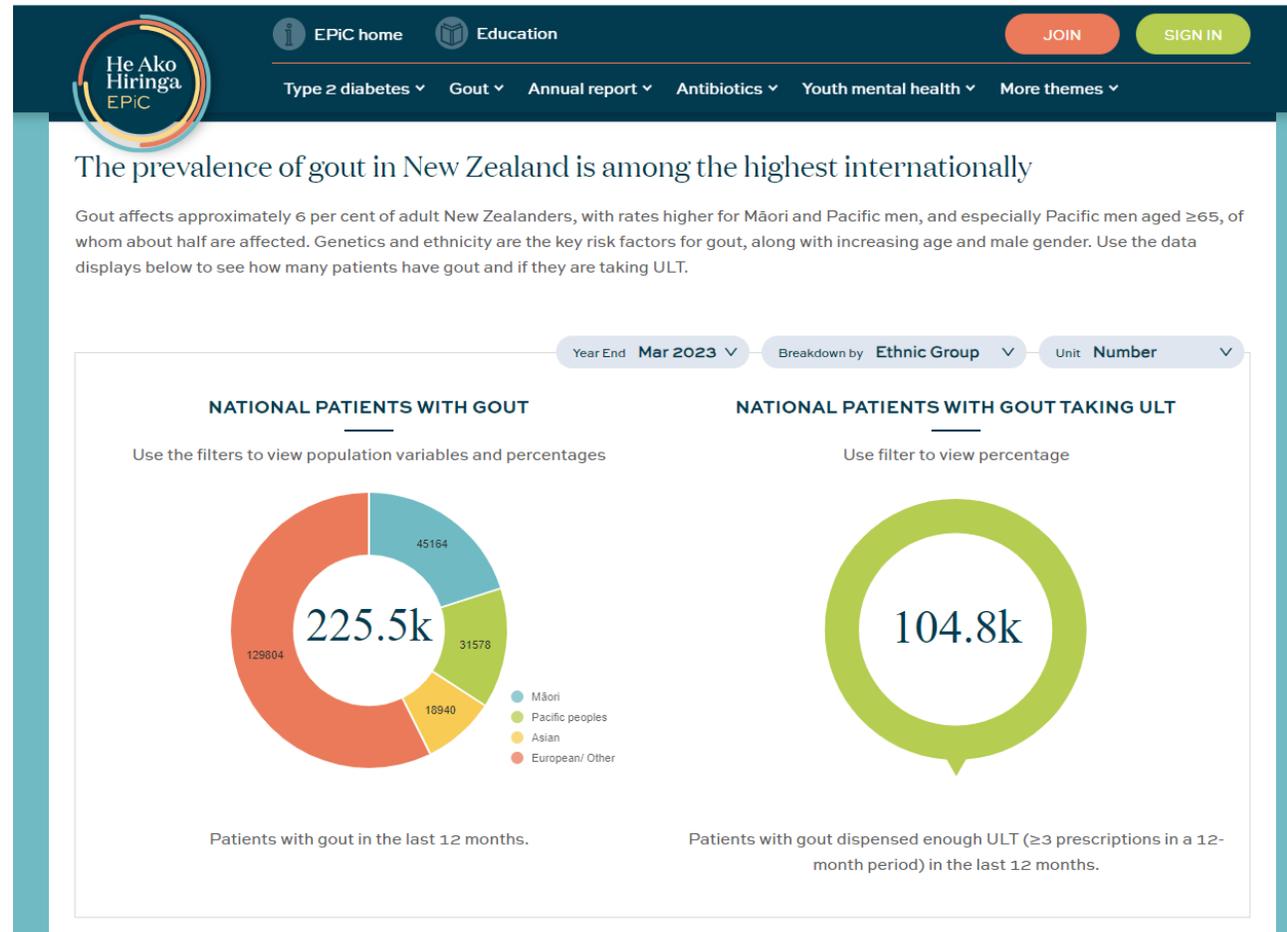
See steps to get started below the Gout Guide Pages display.

 Gallery view  List

Gout Guide Pages

<https://goutguide.nz/>

He Ako Hiringa – EPIc dashboard



Healthify (formerly Health Navigator)

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Low on data? Visit zero.govt.nz, scroll down the page then click on our logo to return to our site and browse for free.



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Gout | Mate waikawa kai kōiwi

Key points about gout (mate waikawa kai kōiwi)

- Gout is a type of long-term inflammatory arthritis.
- It's due to a build-up of uric acid in joints and causes sudden, severe joint pain and swelling, especially in your toes and fingers.
- If you don't get treatment it can cause permanent damage to your joints and can harm your kidneys.
- See your healthcare team for treatment to help during an attack and to stop further attacks.



Booklet – Downloadable from Health Navigator. Hard copies can be ordered from Health Literacy NZ and Physiotherapy NZ websites.



Patient support organization.

Arthritis New Zealand

0800 663 463

www.arthritis.org.nz

info@arthritis.org.nz

Food for thought.....

Randomized Controlled Trial > Rheumatology (Oxford). 2020 Mar 1;59(3):575-579.

doi: 10.1093/rheumatology/kez333.

Nurse-led care is preferred over GP-led care of gout and improves gout outcomes: results of Nottingham Gout Treatment Trial follow-up study

Amy Fuller^{1 2}, Wendy Jenkins¹, Michael Doherty^{1 2}, Abhishek Abhishek^{1 2}

Affiliations + expand

PMID: 31410473 DOI: 10.1093/rheumatology/kez333

Outcomes of nurse-led care

At Year 2	Nurse led care according to BSR guidelines, n=255	Usual GP care, n=262	P
Uric acid at target level	95%	30%	<0.001
On allopurinol	96%	56%	<0.001
Mean allopurinol dose	460 mg/day	230 mg/day	<0.001
Two or more attacks	8%	24%	<0.001
Tophi present	2.6%	9.6%	<0.002
Health related quality of life	41.31	37.87	<0.05
Patient concern	37	54	<0.001
Patient unmet need	21	34	<0.001

The nurse-led intervention was cost-effective in the short-term and potentially cost-saving in the long-term.

Doherty *Lancet* 2018





Managing Gout - It's not rocket science

References

1. http://www.bpac.org.nz/BPJ/2007/September/docs/bpj8_gout_pages_9-18.pdf
2. Gibson T, Waterworth R, Hatfield P, et al. Hyperuricaemia, gout and kidney function in New Zealand Maori men. *Br J Rheumatol* 1984; 23(4):276-82
3. <https://www.hqsc.govt.nz/our-programmes/health-quality-evaluation/projects/atlas-of-healthcare-variation/gout/>
4. Merriman T, Dalbeth N. The genetic basis of hyperuricaemia and gout. *Joint Bone Spine* 2011; 78 (1): 35-40
5. Phipps-Green A, Hollis Moffat J, Dalbeth N, et al. A strong case for the ABCG2 gene is the susceptibility to gout in New Zealand Pacific island and Caucasian, but not Maori, case and control sample sets. *Human Molecular Gene* 2010; 19 (24) : 4813-9
- 6 Wallace SL, Robinson H, Masi AT, et al. Preliminary criteria for the classification of the acute arthritis of primary gout. *Arthritis Rheum* 1977; 20(3): 895-900
7. Zhang W, Doherty M, Pascaul E, et al. EULAR evidence based recommendations for gout. Part 1: Diagnosis. Report of a task force of the Standing Committee for International Clinical Studies including Therapeutics (ESCSIT). *Ann Rheum Dis* 2006; 65 (10): 1301-11
8. Champion EW, Glynn RJ, DeLarby LO. Asymptomatic hyperuricaemia. Risks and consequences in the Normative Aging Study. *Am J Med* 1987; 82(3): 421-6

References

9. Stamp LK, Zhu X, Balbeth N, et al. Serum urate as a possible biomarker in chronic gout-evidence that serum urate fulfills the OMERACT validation for soluble biomarkers. *Semin Arthritis Rheum* 2011; 40:483-500
10. Logan JA, Morrison E, McGill PE. Serum uric acid in acute gout. *Ann Rheum Dis* 1997; 56: 696-7
11. Stamp L, Taylor W, Jones P, et al. Starting dose is a risk factor for allopurinol hypersensitivity syndrome. A proposed safe starting dose of allopurinol. *Arthritis Rheum* ; 64 (8) : 2529-36
12. Reinders MK, van Roon EN, Houtman PM, et al. Biochemical effectiveness of allopurinol-probenecid in previously benzbromarone treated gout patients. *Clin Rheumatol* 2007
13. <http://www.bpac.org.nz/BPJ/2014/July/febuxostat.aspx>
14. Roberts R, Wallace M, Wright D, et al. Frequency of CYP2C9 polymorphisms in Polynesian people and potential relevance to management of gout with benzbromarone. *Joint Bone Spine* 2014;81:160-3
15. Merriman T, Choi H, Dalbeth N. The genetic basis of gout. *Rheum Clin N Am* 2014;40: 279-90
16. Jones P. The modern management of gout. *New Ethicals Journal* 2001; 4: 29-31
17. Jansen TL, Richette P, Perez-Ruz F, et al. International position paper on febuxostat. *Clin Rheumatol* 2010; 29:835-40
18. Baker JF, Krishnan E, Chen L, Schumacher HR. Serum uric acid and cardiovascular disease: recent developments, and where do they leave us? *Am J Med* 2005;118:816-26
19. Hui M, Carr A, Cameron S, et al. The British Society for Rheumatology Guideline for the Management of Gout. *Rheumatology* 2017; 7:e1-e20
20. http://www.bpac.org.nz/BPJ/2007/September/docs/bpj8_gout_pages_9-18.pdf
21. <http://www.bpac.org.nz/BPJ/2013/March/managing-gout.aspx>

Thank you





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