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Gout in Indigenous people: inequity and culturally appropriate management

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The delayed right to appropriate management is a right denied

Gout is known to be associated with various comorbidities, including hypertension, diabetes, obesity, chronic kidney disease, ischaemic heart disease, and hyperlipidaemia. Untangling the relation between hyperuricaemia, gout, and cardiovascular disease has been complicated at least in part because the drug treatments used to treat gout flares have been reported to have differential effects on cardiovascular disease risk. For example, non-steroidal anti-inflammatory drugs (NSAID) have been reported to increase the risk of cardiovascular events[1] and colchicine to reduce the risk.[2] Additionally, allopurinol, the most commonly used urate-lowering treatment, might reduce cardiovascular disease risk.[3] While mendelian randomisation studies do not support a causative role for hyperuricaemia in most of the cardiovascular disease risk factors associated with gout, identification and management of these comorbidities remain important given their impact on cardiovascular disease risk.[4]

Like other Indigenous populations worldwide, Māori, the Indigenous people of Aotearoa, New Zealand, have high rates of gout and inequitable health outcomes, both with respect to gout and generally compared with the country's settler population.[5] At least part of the reason for gout mismanagement in Māori is the clear evidence of inequity in regular dispensing of urate-lowering treatment and culturally safe access to primary care services.[5]

In a linked paper, Cai et al (doi:10.1136/bmjmed-2021-000081) report the association between gout and cardiovascular disease by using linked administrative data highlighting disadvantaged outcomes for Māori.[6] For both men and women, compared with New Zealand Europeans, Māori had a much higher risk of a fatal or non-fatal cardiovascular event within five years (adjusted hazard ratio 1.79 (95% confidence interval 1.21 to 1.90) for women and 1.59 (1.51 to 1.68) for men). As these drugs are known to increase cardiovascular disease risk, it is unfortunate that the authors were unable to capture NSAID dispensing data. Additionally, NSAIDs are readily available for purchase as a general sales medicine (eg, in supermarkets and pharmacies) in Aotearoa New Zealand and sharing of drugs used to treat gout flares among whānau (family) members is not uncommon.[7] The use of NSAIDs might have contributed to the observed results. The study also highlights an increased risk of cardiovascular disease in men with gout and no previous cardiovascular disease, but not in women who had allopurinol dispensed for less than three of four quarters in the year (adjusted hazard ratio 1.15 (95% confidence interval 1.05 to 1.25)) and in those with serum urate concentrations higher than the recommended treatment target of 0.36 mmol/L (1.16 (95% 1.04 to 1.30)). Taken together, these data provide further evidence that people with gout should receive treatment to lower urate concentrations using the treat-to-target urate approach, benefits of which might extend beyond reducing flares and tophi.[8]

In comparison to other studies that suggest colchicine might reduce cardiovascular disease, Cai et al reported that colchicine dispensing in men with gout who have no history of previous cardiovascular disease was associated with an increased risk of cardiovascular events. Importantly, studies examining the role of colchicine in cardiovascular disease have used regular low dose colchicine (0.5 mg) daily for long periods.[2] One meta-analysis reported a 32% reduction in the composite of cardiovascular mortality, myocardial infarction, ischaemic stroke, and urgent coronary revascularisation with this approach.[9] In the current study, Cai and researchers[6] were unable to determine whether colchicine dispensing was for flare prophylaxis or flare treatment. Compared with studies of cardiovascular disease, when used to treat gout flares, colchicine is only used for a short period (ie, days), and the proinflammatory state induced by the gout flare might negate any protective effects on cardiovascular disease risk by colchicine. Although the results need to be interpreted with caution, the evidence gives further weight to the need to manage gout appropriately with urate-lowering treatment in a treat-to-target urate fashion to prevent gout flares and thereby avoid the need for colchicine.

Although studies have shown that gout can be effectively managed with urate-lowering treatment, ensuring that target concentrations of urate are met and maintained has proved difficult in routine clinical practice. For Māori and Pasifika people, who have the highest prevalence of gout in the world, the urgency to transform health engagement dominated by western processes and remove the barriers creating inequity has always been pressing.[10] Health systems have systemically not delivered a resolution for a health condition that should be manageable. Should further evidence be required, this study adds to the previous layers to demand gout prevention and reduce cardiovascular disease risk and deaths.

Action must now focus on strategies to implement and maintain urate-lowering treatments that eliminate, rather than increase, health inequities, particularly for Indigenous populations.

Prioritisation of equity to access and reducing the barriers to regular urate-lowering treatment will require healthcare providers and systems to transfer power to Indigenous populations to develop culturally appropriate and acceptable strategies. If strategies are to be systematically studied, Indigenous peoples' knowledge and worldview must be valued throughout all phases of study design, incorporating data and tissue sample sovereignty, equal explanatory power, and strengthened reporting involving Indigenous people.[11] The time to achieve optimal management of gout has passed. Accepting the status quo is accepting a decrease in life expectancy, disproportionately burdened on Māori and Pasifika people in Aotearoa New Zealand.

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