Management of gout in a South Auckland general practice

Esther Reaves MBChB, BSc (Hons); Bruce Arroll MBChB, PhD, FRNZCGP

ABSTRACT

BACKGROUND AND CONTEXT: In New Zealand, the highest prevalence of gout is in Maori and Pacific people. Counties Manukau District Health Board (CMDHB) has the highest Maori and Pacific population of any New Zealand District Health Board. A CMDHB study found that a high proportion of patients with gout were also at increased risk of cardiovascular disease.

ASSESSMENT OF PROBLEMS: The primary objective was to examine whether the control of gout had changed over time at one clinic. The secondary objective was to assess the management of cardiovascular risk factors in patients with gout at that clinic.

RESULTS: The mean serum uric acid level of patients with gout in the practice had risen in comparison with a similar audit carried out in March 2009. This indicates that the control of gout for patients at the practice has worsened over time. Many patients had not had an annual serum uric acid test.

STRATEGIES FOR IMPROVEMENT: A repeat uric acid level was scheduled for all patients with gout in the practice, with follow-up appointments to be arranged if the result was abnormal.

LESSONS: Gout is often suboptimally managed. Serum uric acid levels may only be tested when a patient presents with an acute attack of gout. Consideration should be given to a minimum of annual serum uric acid levels. Appropriate management of modifiable cardiovascular risk factors in this particular cohort is important and should be a particular focus of care.

KEYWORDS: Allopurinol; cardiovascular diseases; gout; primary health care; uric acid

Background

Gout is one of the commonest forms of inflammatory arthritis in men and older women. It is caused by deposition of uric acid crystals in joints. Definitive diagnosis of gout is by aspiration of a joint, but more often the diagnosis is a clinical diagnosis made in primary care. However, studies focusing on primary care have found that management of gout was suboptimal and did not comply with recent European League Against Rheumatism (EULAR) recommendations. In New Zealand, the Maori and Pacific population has a higher prevalence of gout than the population with European ancestry. It is also known that this population group has a genetic predisposition to the development of hyperuricaemia and gout. Counties Manukau District Health Board (CMDHB) has the highest Maori and Pacific population compared with other district health boards (DHBs) in New Zealand. Therefore, optimal management of these conditions in this population group and DHB is of particular importance. Allopurinol has become established as the first-line treatment in New Zealand in long-term management of gout. Despite the ready availability of allopurinol, gout is a significant cause of admissions to hospital and referral to Rheumatology Clinics in this area. Maori and Pacific men develop gout earlier in life than men in other ethnic groups, with the result that it impacts to a greater degree on their ability to work. Thus, it is important that primary care management of gout in CMDHB is optimal.
The primary purpose of this study was to assess the management of gout in one primary care clinic and to establish whether or not the management of gout in this South Auckland practice has improved since a similar audit was conducted in 2009. An analysis of gout management between 2000 and 2005 concluded that a diagnosis of gout warrants a cardiovascular risk assessment, due to the comorbidities associated with gout. Other studies have suggested that the presence of hyperuricaemia warrants investigation of associated diseases, such as diabetes and cardiovascular disease. Based on another recent study, 59% of patients with gout in CMDHB have an increased risk of a cardiovascular event within the next five years. These findings suggest an association between gout and cardiovascular risk factors. In view of this, the present study also assessed cardiovascular risk factor management in this group of patients.

**Assessment of problem**

A clinical audit was performed at a primary care clinic in South Auckland on 6 November 2012. The audit sought to identify registered patients who had been either classified as having gout at any time in their lives, or who had been prescribed allopurinol or colchicine in the period from 6 November 2010 to 6 November 2012, inclusive. The lists of patients resulting from each search were then combined and sorted using their National Health Index (NHI) number. Duplicates were removed but a note of how the patient had been identified was included. Recorded data included age, gender, ethnicity, New Zealand Index of Socioeconomic Deprivation 2006 (NZDep2006) quintile (a non-occupational index of socioeconomic deprivation where patients in quintile 1 are the least deprived and those in quintile 5 the most deprived), most recently prescribed dose of allopurinol and colchicine, serum uric acid (SUA) level, and date of measurement. In connection with the cardiovascular disease risk score calculation, the following data were also tabulated: haemoglobin A1c (HbA1c), systolic blood pressure (SBP), high density lipoprotein (HDL) cholesterol, total cholesterol, smoking status, presence or absence of a family history of cardiovascular disease, and presence or absence of left ventricular hypertrophy on an electrocardiogram (ECG). The presence or absence of a family history of cardiovascular disease was determined as per the New Zealand Cardiovascular Guidelines. Where there was uncertainty or a lack of specificity, due to under-recording, a family history of cardiovascular disease was taken as being absent. A further search was made of the notes of patients who had been included on the grounds of having been prescribed allopurinol or colchicine but who had not been given a formal classification of gout. Patients were excluded from the dataset if they had no mention of gout in their notes, or if a reason other than gout was given for being prescribed either allopurinol or colchicine. The level of control of gout was determined by the percentage of patients whose last measured SUA level was ≤0.36 mmol/L.

Cardiovascular risk scoring was calculated using a formula based on the Framingham equation. Cardiovascular risk scores (risk of cardiovascular disease in five years) were grouped as follows: mild (0–10%), moderate and high (10–20%), and very high (>20%), as per the New Zealand Cardiovascular Guidelines grouping. A patient’s cardiovascular risk score was incremented by five percentage points if he/she fulfilled either or both of the following criteria: Maori/Pacific ethnicity, or family history of premature cardiovascular disease.

A previous audit was carried out in the South Auckland practice in March 2009. This audit studied the impact of an active intervention over the course of a year, to up-titrate allopurinol in order to achieve a target SUA level <0.36 mmol/L. Patients identified for inclusion were those with a classification of gout, or prescription of allopurinol, colchicine or probenecid. This current audit did not include a prescription of probenecid in its identification of patients, as only one patient was prescribed this in the South Auckland practice at the time of the 2009 audit. This study then compared the results of the audit in March 2009 with the results of this study’s audit, to determine if control of gout had improved with time.

**Results of assessment/measurement**

A total of 196 patients qualified for inclusion in the audit. One patient was excluded due to a diagnosis of myeloproliferative disease and no
mention of gout in their notes. The percentage of patients with gout in the total practice population was 4.2% (196/4641). This audit found that 9.5% of men in the practice population have gout. Furthermore, 94% (60/64) of the women in the audit were aged over 50 years, emphasising that gout predominantly appears in post-menopausal women. This audit found that Maori women were on average 11 years younger than their European counterparts. Table 1 provides a comparison of patient characteristics.

Despite the majority having a formal classification of gout, 5.1% (10/196) had no record in their notes of an SUA ever being measured. This study found that, in comparison to the previous audit performed in March 2009, there has been a significant increase ($p=0.009$) in the mean SUA level (Table 2). This study also found that there was a non-significant decrease ($p=0.08$) in the proportion of patients with an SUA level $\leq 0.36$ mmol/L.

Table 3 provides a summary of the cardiovascular risk scores and individual risk factor target achievement for this group of patients. On further analysis, 47% of patients with hyperuricaemia (SUA level $>0.36$ mmol/L) had an SBP $>130$ mm Hg. In total, 36% (68/189) of the sample population had a high or very high risk of cardiovascular disease. As a consequence of this finding, this study assessed whether the New Zealand targets for cardiovascular risk factors were being achieved. Of note, only 26% of patients with a very high cardiovascular risk score had achieved a target SBP of $<130$ mm Hg. For all cardiovascular risk groups, target SBP was achieved by the lowest proportion of patients compared with the proportion of patients achieving other recommended targets. Other recommended targets included achieving a target HDL $>1$ mmol/L, a target HbA1c $\leq 53$ mmol/mol and smoking cessation. The very high cardiovascular risk group was less successful than the lower cardiovascular risk groups in achieving recommended cardiovascular risk targets. The highest proportion of patients achieving target SBP were those with a less than 15% risk of cardiovascular disease in five years. Of note, only 28% (19/68) of patients in the very high cardiovascular risk group were achieving the target SUA level.
urate-lowering therapy.\textsuperscript{21,22} The South Auckland practice that was audited had stopped carrying out three-monthly SUA testing and had not instigated any other form of regular uric acid testing. The practice of only measuring uric acid level in acute gout attacks is counterintuitive, given that in the majority of cases SUA level is low or normal.\textsuperscript{23} Regular SUA testing would afford an opportunity to assess patient medication compliance, which, given the use of alternative remedies and over-the-counter medication amongst the Maori and Pacific communities, may be worthwhile.\textsuperscript{14} Suboptimal management of gout may also be influenced by the health literacy of patients. \textit{Korero Marama} found that Maori have poorer health literacy skills compared with non-Maori.\textsuperscript{24} This could have a significant impact, in terms of patients not appreciating the need for long-term medication and how to manage gout as a chronic condition.

Overall the practice performed poorly in terms of gout management, with only 19.9\% of the patients in the sample achieving the target SUA level in the past year. This suggests that, despite more than half of patients having an SUA measurement in the past year, gout continues to be poorly controlled. Nevertheless, in comparison to the audit in 2009, there has been an increase in the proportion of patients prescribed allopurinol. Despite this improvement, allopurinol dosing is still suboptimal, as only half of the patients recently prescribed allopurinol achieved the target SUA level. A common concern of practitioners is worsening of renal impairment in those with gout by increasing the dose of allopurinol. Table 4 provides a summary comparison between those patients prescribed allopurinol and those patients who were not, with their SUA level. The suboptimal management of gout despite allopurinol therapy has also been reported in the UK and Germany.\textsuperscript{2} The ultimate therapeutic aim of prescribing allopurinol is to cause dissolution and prevent formation of urate crystals; this optimally happens at an SUA level ≤0.36 mmol/L. To ensure this level is reached, consideration should be given to the instigation of dedicated primary care nurses to manage gout and its associated comorbidities, as has been implemented in the management of diabetes, with some success.\textsuperscript{25}

Recently published guidelines have recommended screening for hyperlipidaemia, hypertension and diabetes when making decisions about the management of gout.\textsuperscript{10} In our study, 26\% of patients with a very high cardiovascular risk had not been prescribed allopurinol. This finding raises

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**Table 3. Summary of cardiovascular risk scores for the total group (n=189)\textsuperscript{*} and proportion of the group achieving the individual cardiovascular risk factor targets**

<table>
<thead>
<tr>
<th>CV risk score</th>
<th>CV risk 0–&lt;10%</th>
<th>39% (74/189)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV risk 10–&lt;20%</td>
<td>40% (76/189)</td>
<td></td>
</tr>
<tr>
<td>CV risk &gt;20%</td>
<td>21% (39/189)</td>
<td></td>
</tr>
<tr>
<td>Individual risk factor targets\textsuperscript{†}</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Target SBP &lt;130 mm Hg</td>
<td>44% (83/189)</td>
<td></td>
</tr>
<tr>
<td>Target HDL cholesterol &gt;1 mmol/L</td>
<td>71% (134/189)</td>
<td></td>
</tr>
<tr>
<td>Target HbA1c &lt;53 mmol/L</td>
<td>85% (160/189)</td>
<td></td>
</tr>
</tbody>
</table>

\textsuperscript{*} Seven patients could not be given a cardiovascular risk score as certain values were unrecorded

\textsuperscript{†} Data were not recorded for all individual risk factor targets for all patients included in the study (N=196)

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**Table 4. Comparison of mean SUA level and proportion at the target SUA ≤0.36 mmol/L for the total patient group and according to whether allopurinol prescribed for use**

<table>
<thead>
<tr>
<th></th>
<th>Patients (N=196)</th>
<th>Currently prescribed allopurinol\textsuperscript{*} (n=92)</th>
<th>Never prescribed allopurinol (n=70)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean SUA level</td>
<td>0.41 mmol/L</td>
<td>0.37 mmol/L</td>
<td>0.45 mmol/L</td>
</tr>
<tr>
<td>Number at target</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SUA ≤0.36 mmol/L</td>
<td>35% (68/196)\textsuperscript{†}</td>
<td>50% (46/92)</td>
<td>13% (9/70)</td>
</tr>
</tbody>
</table>

SUA: serum uric acid level

\textsuperscript{*} Taken as being prescribed in the past three months

\textsuperscript{†} Nine patients had not had an SUA measurement
concern given reports that indicate that gout may increase the risk of acute myocardial infarction.26 Optimal management of gout, particularly in patients with a very high cardiovascular risk, is thus necessary. Research carried out in secondary care suggests that patients with gout in this setting would benefit from cardiovascular risk management.17 However, given the high risk of comorbidities associated with gout, this study further emphasises the need for cardiovascular risk assessment and management as part of primary care management of gout.16,27 This is in agreement with recently published EULAR recommendations and research on the management of gout.2,10 An initial assessment of a patient’s cardiovascular risk should be carried out on diagnosis of gout and, from there, follow-up should be based on the level of risk, according to New Zealand Cardiovascular Guidelines.19 This allows not only for better control of comorbidities, with modifiable risk factors being identified early, but also a holistic approach to be adopted with regard to patient care. This could be achieved by primary care practitioners allowing an extended consultation time with the patient on first presentation of gout, to afford an opportunity for all components of the patient’s care to be addressed.28 Given the high prevalence of gout in Maori and Pacific people, with an additional increased cardiovascular risk, it is important to include an assessment of cardiovascular risk with a diagnosis of gout in this patient group.17,19

Following the audit, results were provided as feedback to relevant staff at the primary care practice at which the audit was based. After some discussion, it was proposed that patients with gout should have a repeat uric acid level taken, with follow-up appointments to be arranged if the result was abnormal.

Lessons and messages

Despite interventions, gout continues to be poorly controlled in this South Auckland practice. However, with the introduction of annual measurement of SUA level, lack of compliance with medication and forewarning of future gout attacks will be identified. This study highlights that practitioners need to be aware of the increasing evidence of high-risk comorbidities associated with gout. Our results also suggest that there is a lack of allopurinol up-titration, in order to achieve the target SUA level. Therefore, primary care physicians should review the dose of allopurinol in patients with gout that is persistently poorly controlled. Consideration should be given to the use of dedicated primary care nurses in the management of gout. Alternatively an extended consultation time with the practitioner would allow cardiovascular risk assessment and management to be carried out as part of the primary care management of gout on first presentation. This would allow the adoption of a holistic approach to patient care and modifiable cardiovascular risk factors to be addressed earlier.

There needs to be recognition of the impact of gout on the health of patients in South Auckland and of its implications for the future financial burden on the health care system. Improved management of gout in primary care will permit secondary care resources to be more appropriately distributed.

References


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COMPETING INTERESTS
None declared.