

More allopurinol is needed to get gout patients <0.36 mmol/L: a gout audit in the form of a before–after trial

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ABSTRACT

AIM: To establish a benchmark for gout control using the proportion of patients with serum uric acid (SUA) <0.36 mmol/L, assess patients' understanding of their preventive medication and trial a mail and phone intervention to improve gout control.

METHODS: Patients clinically diagnosed with gout and baseline SUAs were identified in two South Auckland practices. A mail and phone intervention was introduced aimed at improving the control of gout. Intervention #1 took place in one practice over three months. Intervention #2 occurred in the other practice four to 16 months following baseline.

RESULTS: No significant change in SUA from intervention #1 after three months. The second intervention by mail and phone resulted in improvement in SUA levels with a greater proportion of those with SUA <0.36 mmol/L and the difference in means statistically significant ($p=0.039$ two-tailed paired t-test). Benchmarking for usual care was established at 38–43% SUA <0.36 level. It was possible to increase from 38% to 50%. Issues relating to gout identified included lack of understanding of the need for long-term allopurinol and diagnosis and management for patients for whom English is not their first language.

STRATEGIES FOR IMPROVEMENT:

1. Community workers who speak Pacific languages may assist GPs in communicating to non-English speaking patients.
2. Alternative diagnoses should be considered in symptomatic patients with prolonged normouricaemia.
3. GPs should gradually introduce allopurinol after acute gout attacks, emphasising importance of prophylaxis.
4. A campaign to inform patients about benefits of allopurinol should be considered.
5. A simple one keystroke audit is needed for gout audit and benchmarking.
6. GP guidelines for gout diagnosis and management should be available.

KEYWORDS: Gout; uric acid; clinical audit; benchmarking; family practice

Introduction

It is now widely accepted that the target level of serum uric acid (SUA) for patients with recurrent gout is <0.36 mmol/L.¹ Below this level it is unlikely that patients get gout and, if they do, a reconsideration of their diagnosis is warranted. The impetus for this audit came from a presentation by Dr Karen Lindsay, a Rheumatology Research Fellow at Middlemore Hospital, South

Auckland, New Zealand (NZ) in May 2007. She reported that Counties Manukau (South Auckland District Health Board) had an admission rate for gout of 46/100 000 compared with a NZ mean of 19/100 000. Ninety-four percent of these admissions were for Maori and Pacific patients in approximately equal numbers. The rate for Maori and Pacific patients was about 150/100 000 citizens while for non-Maori/non-

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Pacific was about 10/100 000.² She presented the results of in-depth qualitative interviews with 11 patients with severe gout. Some had been started on allopurinol, but only one had been commenced by their GP. Her conclusions were that in South Auckland there was a:

- large burden of untreated gout;
- burden on family—24-hour care for patients with gout;
- widespread tolerance (of gout);
- struggle to work (for those affected); and
- minimal knowledge or understanding of gout.

While the principal author was somewhat critical of the GP management of these particular patients, the anecdotal GP view of gout management was that allopurinol was often prescribed but many patients failed to take it indefinitely. The question arose of how well-informed patients are about their gout. A small summer student project was conducted from November 2007 to February 2008 (extended to March 2009) to:

1. establish a benchmark for gout management in terms of patients with SUA <0.36 mmol/L in South Auckland (there is no existing benchmark for Auckland nor NZ);
2. explore patients' understanding of their gout and their gout medication; and
3. conduct an intervention to up-titrate gout patients' allopurinol to achieve SUA <0.36 mmol/L.

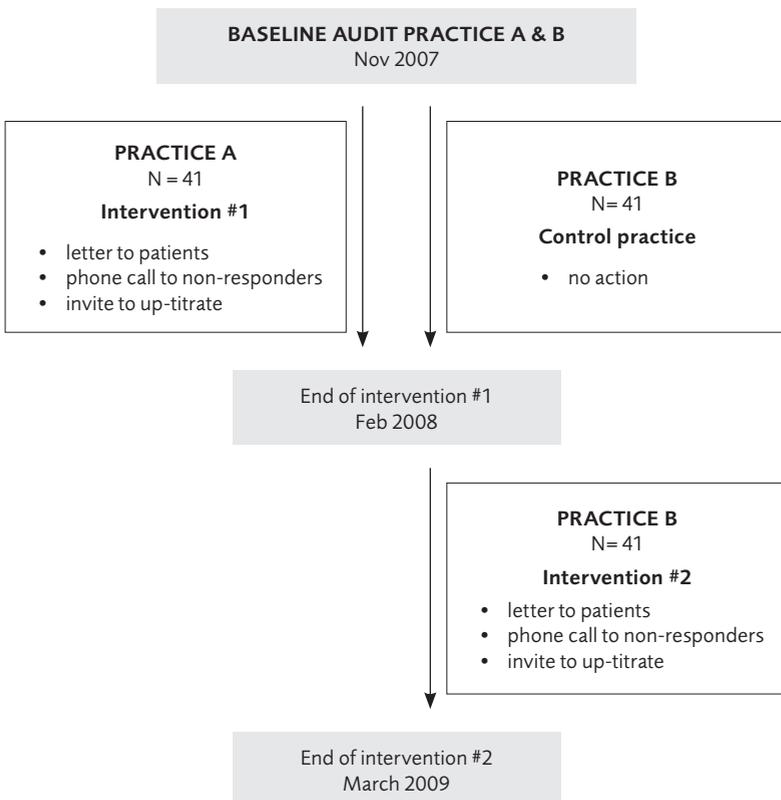
Method

A pilot before–after controlled trial of intervening to up-titrate allopurinol to achieve the desired target for SUA <0.36 mmol/L was conducted.¹ This aspect of the study is known as Intervention #1. Two practices in the same suburb of South Auckland were chosen to be part of the study, Practice A as intervention and Practice B as control. Both practices had a baseline audit conducted using the practice management system (PMS) Medtech-32. The control practice was not informed of the audit until the end of the project in February 2008. the PMS query builder was used to find patients with a classification of gout, or prescription of allopurinol, colchicine or probenecid. A patient having any one of these four items was considered to have a diagnosis of gout. It was decided not to search on non-steroidal anti-inflammatory drugs and oral steroids because these would have identified too many false positives. Practice A had 41 patients retrieved by the audit who were selected for the intervention. The study design is summarised in Figure 1.

Intervention #1: The first intervention consisted of sending out a questionnaire to all 41 patients asking about the number of attacks of gout they had had in the past year and a variety of questions about their concordance with taking medication. Those whose SUA had not been done in the previous year were sent a lab form for a blood test. Those who had a normal SUA (i.e <0.36 mmol/L within the past year) and on allopurinol were considered to be well controlled. Telephone calls were made to patients who had not responded to the initial letter. The control practice had no action until after the end of study audit in February 2008.

Intervention #2: The first intervention did not achieve any sizable change in SUA levels. It was decided to intervene in the control Practice B starting in May 2008 with a follow-up in March 2009.

Figure 1



One of the doctors in this practice (DH) sent a letter to those patients who either did not have a uric acid recorded or had SUA >0.36 mmol/L. When DH left her role was undertaken by GS. Patients were phoned and asked to make an appointment.

The study was approved by the Northern Regional Ethics Committee.

Results

Intervention #1
(November 2007 to February 2008)

Of the 41 letters sent to patients in Practice A, 17 were returned. Of those 17, 11 appeared controlled (SUA <0.36 mmol/L) and six did not. Of those six, four agreed to up-titrate and two had lower SUA and two had higher SUA at the end of the study. Of the 13 patients who answered the question about ‘how long are you meant to take allopurinol?’ Eleven said short-term and two said forever. Table 1 summarises the baseline comparison of the intervention (A) and control practice (B).

Table 2 summarises the state of the patients at the end of the intervention #1 study. Each category was chosen for individual patients to best characterise the most important (as judged by the authors) issue regarding their gout management. For example, if no one at home could speak English this was the reason given rather than saying the patient declined or did not engage. The control practice B had four changes in SUA over the three months of the study—two patients were 0.01 mmol/L higher, one was 0.06 mmol/L higher and one was 0.1 mmol/L lower. The intervention practice A had three patients whose SUA decreased (0.11, 0.11 and 0.14 mmol/L (only the latter was below 0.36 mmol/L) and three whose SUA increased (0.03, 0.05 and 0.12). Two patients in this group did not have a diagnosis of gout on re-examination of their charts.

Intervention #2
(February 2008 to March 2009)

Of the 41 patients in practice B in intervention #2, 36 had a SUA prior to November 2007 and a final SUA by March 2009 (see Table 3). The mean SUA fell from 0.4 to 0.36 ($p=0.039$ paired t-test) while the proportion of those controlled (<0.36) rose from 38% to 50% ($p=0.48$ McNemars test).

WHAT GAP THIS FILLS

What we already know: If patients with recurrent gout have their serum uric acid (SUA) levels controlled at <0.36 mmol/L they are unlikely to get further attacks of gout. The drug allopurinol can be up-titrated until an SUA <0.36 is obtained. However many patients do not take allopurinol long-term. Maori and Pacific patients have a 10- to 15-fold increase in hospital admissions for gout compared with non-Maori/non-Pacific.

What this study adds: Practices can audit for gout patients and establish benchmarking for adequate SUA control. Mail and telephone contacting of patients with gout can significantly improve their ongoing use of allopurinol and reach the target SUA level of <0.36 mmol/L.

Table 1. Baseline results

	Practice A N=41	Practice B N=41
Average age	60 years	72 years
Ethnicity	European 42% Maori 23% Pacific 23% Other 12%	European 58% Maori 28% Pacific 8% Other 5%
Average SUA	0.40	0.40
Median SUA	0.38	0.395
Range of SUA	0.25–0.7	0.17–0.62
% <0.36 SUA	43%	38%

SUA = Serum uric acid

Table 2. Status of patients at practice A at the end of intervention #1

Status	Number	%
Controlled (SUA <0.36 mmol/L)	17	41
Contacted but did not engage	5	12
Wrong diagnosis	2	5
Declined	1	2
No phone	3	7
English difficulties	3	7
No response	5	12
Agreed to up-titrate	4	10
Did not up-titrate	1	2
Total	41	98

Table 3. Change in practice B for intervention #2 (November 2007 to March 2009)

N = 36*	November 2007	March 2009
Mean SUA	0.40	0.36 [†]
Median SUA	0.40	0.345
Range of SUA	0.17–0.62	0.18–0.57
% <0.36 SUA	39% (14/36)	50% (18/36) [‡]

* 36 patients had a SUA on both occasions

† $p=0.039$ paired two-tailed t-test of the means

‡ $p=0.48$ for difference in proportions using McNemars paired test

Strategies for quality improvement

The end of study results show very little change from the intervention #1. This may be due to the short time duration for that part of the study. The intervention #2 showed greater improvement and this did reach statistical significance for the difference in mean SUA, but not in the changes in proportions of those controlled with SUA <0.36 mmol/L.

A weakness of the study is the small numbers, short duration of intervention #1 (three months) and limited resources which did not allow for a bigger study group. By missing those who were only ever treated with non-steroidal anti-inflammatory agents we are probably excluding younger patients and those who are less concordant at taking long-term medication.

Strengths of the study were that the two general practices were reasonably similar apart from the higher number of Pacific patients in practice A, and the percentage of those well controlled on allopurinol was similar in both clinics which is in some ways a form of validity.

It is not clear how many GPs are aware that 0.36 is now the target for gout control. The Auckland community laboratory was reporting normal uric acid up to 0.42 and this may have led to confusion in the community. The patients had almost all been prescribed allopurinol hence they are likely a more compliant group and the results need to be seen in that light.

Lessons and messages

1. Language issues

This was an issue for Pacific patients and it may mean that community workers who speak Pacific languages are engaged to assist GPs with the management of gout. One of their roles would be to emphasise the need for long-term consumption of allopurinol when indicated.

2. Wrong diagnosis

Several patients in this study were coded for gout, but did not meet diagnostic criteria, and had other diagnoses to account for their symptoms. Alternative diagnoses should be considered if the patient has ongoing symptoms in the face of prolonged

normouricaemia (SUA <0.36 mmol/L). In this situation, referral to rheumatology should be considered.

3. Non-engagement

This is an issue that may be the most difficult for GPs to manage. It may require an emphasis on the need for long-term use of allopurinol at the time of the acute diagnosis. Management of gout in primary care is particularly difficult due to concerns about allopurinol worsening disease if started during an acute gout attack. Strategies for ensuring that allopurinol is started after an acute gout attack are needed, emphasising the importance of prophylaxis and gradual introduction of allopurinol.

4. Long-term treatment

GPs may need to emphasise the long-term need for allopurinol once it is started. A campaign to inform patients about the benefits of allopurinol should be considered.

5. Audit tools and benchmarks for gout audits

Audit tools should be made available for GPs to do their own audits. It would be useful to get audits of more NZ practices to establish the benchmark. Is the 43% in this study better or worse than the NZ average? Both practices had a similar level so it may be that most NZ practices are in this ballpark. This current audit took three query builders to get three lists from which the duplicates were removed. A more automated system of audit would make it a 'painless' and simple procedure. A simple one keystroke audit is needed.

6. Guidelines for the diagnosis and management of gout

These should be made available to GPs.

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COMPETING INTERESTS

None declared.