Management of Gout in a Hospital Setting: A Lost Opportunity

Sarah Wright, Peter T. Chapman, Christopher Frampton, John L. O'Donnell, Rafi Raja, and Lisa K. Stamp

ABSTRACT. ***Objective.*** Management of gout is frequently suboptimal. The aim of this study was to determine the proportion of patients presenting to Christchurch Hospital for a gout flare and to determine whether management for both acute flares and urate lowering was in accordance with international recommendations.

Methods. A retrospective audit was undertaken of all admissions to Christchurch Hospital from June 1, 2013, to May 31, 2014, in which gout was coded as a primary or secondary discharge diagnosis. Information including demographics, comorbidities, concomitant medications, treatment of acute gout, and urate lowering was collected.

Results. A total of 235 acute admissions for gout in 216 individuals were identified. Eleven individuals had 2 admissions and 4 individuals had 3 admissions. In 95/235 admissions (40.4%), gout was the primary diagnosis. Gout accounted for 95/77,321 (0.12%) of acute admissions. The treatment of acute gout was prednisone monotherapy in 170/235 (72.3%) of admissions. Serum urate was measured at some point during 123/235 (52.3%) of admissions, with only 19/123 (15.4%) at target urate level (< 0.36 mmol/l). At 60 of the 235 admissions, urate-lowering therapy was already being prescribed. Nine out of 175 patients (5.1%) not treated with urate-lowering therapy at admission commenced allopurinol and 32/174 (18.4%) had commencement of urate-lowering therapy recommended in the discharge plan.

Conclusion. Rates of admission for gout are similar to that observed in other studies. Failure to initiate, change, or recommend alterations in urate-lowering therapy to achieve target urate in people with gout admitted to hospital represents a significant lost opportunity to improve longterm gout management. (First Release August 1 2017; J Rheumatol 2017;44:1493–8; doi:10.3899/jrheum.170387)

Key Indexing Terms:
GOUT URATE-LOWERING THERAPY MANAGEMENT HOSPITALIZATION

Gout is a common form of arthritis in New Zealand, affecting 3.75% of the general population with higher rates of 6.06% in New Zealand Māori (NZ Māori) and 7.63% in Polynesian people[1]. Early in the disease course, gout typically presents as an intermittent acute inflammatory arthritis. These gout flares cause significant pain and short-term disability. Over time, inadequately treated gout can lead to recurrent flares, presence of tophi, joint damage, and chronic gouty arthritis. Inadequately treated gout has been associated with significant healthcare costs[2,3], and while admission to hospital is reducing for rheumatoid arthritis, admissions for gout remain static or are increasing[4,5]. Longterm urate lowering is critical to the successful management of gout and while a definition of remission has not yet been formally agreed, serum urate < 0.36 mmol/l and the absence of gout flares and tophi have been identified as key features of remission[6]. International recommendations for the management of gout focus on effective management of gout flares and sustained urate lowering as well as identification and management of comorbidities[7,8,9]. Previous studies of in-hospital gout management in both Australia and New Zealand have revealed considerable variability in the acute management and suboptimal urate-lowering therapy[10,11], although improvements in management of gout flares have been noted after introduction of a protocol[12].

The aim of our study was to determine the proportion of patients presenting to Christchurch Hospital for a gout flare and to determine whether management for both acute flares and urate lowering was in accordance with international recommendations. In addition, we wished to determine whether there were differences in those individuals seen by

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the rheumatology service compared with those not seen, and in those with gout as the primary discharge diagnosis compared to those with gout as a secondary discharge diagnosis.

MATERIALS AND METHODS

A retrospective audit was undertaken of all acute admissions to Christchurch Hospital from June 1, 2013, to May 31, 2014, in which gout was coded as a primary or secondary discharge diagnosis. Ethical approval was obtained from the University of Otago, Human Ethics Committee (HD14/33).

Christchurch Hospital is a tertiary-level hospital in New Zealand servicing a catchment population of 529,905[13]. All cases of gout were identified through the International Classification of Diseases, 10th ed. code search of discharge summaries using codes "M10-". Discharges were classified as primary, the main reason for admission, or secondary, where an alternative diagnosis was the main reason for hospital admission. A retrospective review of written and electronic medical records and laboratory results was undertaken. Information on the following areas was collected: (1) 4 key comorbidities — cardiovascular disease (CVD; congestive cardiac failure, ischemic heart disease, or hypertension), cerebrovascular disease (stroke, transient ischemic attack), type 2 diabetes mellitus, and chronic kidney disease [CKD; defined as estimated glomerular filtration rate (eGFR) < 60 ml/min/1.73 m^2]; (2) 8 specific non-gout–related concomitant medications — diuretics, antihypertensive medication (angiotensin-converting enzyme inhibitors, angiotensin II receptor antagonists, calcium channel blockers, β blockers, and α blockers), cholesterol-lowering therapy, and antiplatelet agents; (3) laboratory assessments including eGFR, serum urate, and synovial fluid crystal analysis; (4) management of the gout flare including use of prednisone, nonsteroidal antiinflammatory drugs (NSAID), intraarticular steroid injection or colchicine, and documentation of the dose and duration of therapy; (5) the use of urate-lowering therapy at the time of admission, commenced during admission, or given instruction to commence by a general practitioner post-discharge, as well as dose changes; and (6) rheumatology service input defined as inpatient advice or planned followup.

Statistics. Summary statistics including means, SD, and ranges were used to summarize numeric measures and frequencies, and percentages were used to summarize categorical measures. Statistical comparisons between those with and without a primary diagnosis of gout and between those having a rheumatology consult and those not were undertaken using independent Student t tests for numeric measures, and chi-square and Fisher's exact tests as appropriate for categorical measures. A 2-tailed p value < 0.05 was taken to indicate statistical significance.

RESULTS

Over the 12-month audit period, there were 91,123 admissions to Christchurch Hospital consisting of 77,321 acute admissions and 13,802 elective admissions. There was a total of 235 admissions with gout as a coded discharge diagnosis in 216 individuals. Eleven individuals had 2 admissions and 4 individuals had 3 admissions. In 95/235 admissions (40.4%), gout was the primary diagnosis. Gout accounted for 95/77,321 (0.12%) of acute admissions and gout was a secondary diagnosis in 140/91,123 (0.15%) of total acute and elective admissions.

Of the 216 people, the mean age was 71.2 years (27–99 yrs), 78.2% were men, 172 (79.6%) identified as New Zealand European or European, and 32 (14.8%) NZ Māori or Pacific Island. There was a high prevalence of comorbid conditions, with 66/216 (30.6%) having 1, 56/216 (25.9%) having 2, and 40/216 (18.5%) with 3 or 4. The most common medical comorbidity was CKD (eGFR < 60 ml/min/1.73 m^2), which occurred in 128/205 (59.3%). CVD was present in 141/216 (65.3%) of patients; 61/216 (28.2%) had type 2 diabetes and 46/216 (21.3%) had cerebrovascular disease. Of the 8 non-gout medications recorded, the mean number per patient was 2.45 (0–7; Table 1).

The majority of admissions were to the Department of General Medicine 132/235 (56.2%), followed by Orthopedics 30/235 (12.8%) and Cardiology 24/235 (10.2%). The median (interquartile range) length of stay was 4.0 days (2–8 days).

During 72 of the 235 admissions (30.6%), a joint aspiration was undertaken, of which 61/72 (84.7%) were positive for monosodium urate crystals. Patients admitted under the orthopedic service were significantly more likely to have a joint aspiration compared with those admitted under the general medical service [21/30 (70%) vs 34/132 (25.8%); p < 0.001].

The treatment of acute gout was prednisone monotherapy in 170/235 (72.3%) of admissions, followed by NSAID 27/235 (11.5%), colchicine 11/235 (4.7%), and intraarticular steroid 3/235 (1.3%). The most common starting doses of prednisone were 40 mg daily (58/170; 34.1%) or 20 mg daily (78/170; 45.9%), and 55.3% of patients followed a tapered course. Colchicine was prescribed at 0.5 mg twice daily for 10/17 (58.8%) of the patients, with marked variability in the duration of therapy (3–30 days). Only 2 patients received the recommended dosing of 1.0 mg stat followed by 0.5 mg 1 h later. Dual therapy was prescribed consisting of either prednisone and colchicine (6 admissions) or prednisone and NSAID (5 admissions). Three patients did not receive any specific treatment for acute gout.

Table 1. Demographics and clinical characteristics of the 216 individuals admitted with gout. Values are n (%) unless otherwise specified.

Characteristics	Total
Male	169 (78.2)
Age, yrs, mean (range)	71.2 (27–99)
Ethnicity	
NZ European/European	172 (79.6)
Maori/Pacific Island	32 (14.8)
Comorbidities	
Type 2 diabetes mellitus	61 (28.2)
Cardiovascular disease	141 (65.3)
Cerebrovascular disease	46 (21.3)
CKD, eGFR < 60 ml/min/1.73 m^2, n = 205	128 (59.3)
≥ 2/4 comorbidities	96 (44.4)
Medications	
ACE inhibitor	72 (33.3)
Diuretic	86 (39.8)
α blocker	27 (12.5)
β blocker	112 (51.9)
Calcium channel blocker	36 (16.7)
Aspirin	97 (44.9)
Lipid-lowering agent	77 (35.6)
≥ 3 other medications	110 (50.9)

NZ: New Zealand; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate; ACE: angiotensin-converting enzyme.

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Serum urate was measured at some point in 123 of the 235 admissions (52.3%); mean serum urate was 0.52 mmol/l (range 0.17–1.0 mmol/l) and only 19/123 (15.4%) were at target urate < 0.36 mmol/l.

At 60 of the 235 admissions (25.5%), patients were receiving urate-lowering therapy, all allopurinol. The mean allopurinol dose was 171.3 mg/d (50–300 mg). Of these 61, 32 (52.5%) had serum urate measured, and 91/174 (52.3%) not receiving urate-lowering therapy at admission had a serum urate measured. Mean ± SD serum urate in those treated with allopurinol was significantly lower compared with those not receiving allopurinol (0.42 ± 0.16 mmol/l vs 0.55 ± 0.15 mmol/l; p < 0.001). Those receiving allopurinol were significantly more likely to be at target urate compared to those not treated with urate-lowering therapy (13/32 vs 6/91; p < 0.001).

Nine out of 175 patients (5.1%) not receiving urate-lowering therapy at admission commenced allopurinol during admission and 32/174 (18.4%) had commencement of urate-lowering therapy recommended in the discharge plan to the patients' general practitioner.

A rheumatology consult was undertaken in 39/235 (16.6%) of admissions. Those seen by the rheumatology service were significantly younger. They were also significantly more likely to have joint aspiration done, serum urate measured, and urate-lowering therapy altered (Table 2 and Table 3). There was no significant difference in length of stay (5.3 ± 5.3 days vs 6.7 ± 11.5 days; p = 0.44).

As expected, those with gout as the primary diagnosis were younger and had fewer medications as compared with those for whom gout was recorded as a secondary diagnosis (Table 2). In addition, those with gout as the primary diagnosis were more likely to be seen by the rheumatology service, to have a joint aspiration undertaken, and to have a change in urate-lowering therapy recommended (Table 3). Those with gout as the primary diagnosis had a significantly shorter length of stay compared to those with gout as a secondary diagnosis (3.4 ± 3.6 days vs 8.6 ± 13.2 days; p < 0.001).

DISCUSSION

Annually, a significant number of patients are either admitted with acute gout or with gout that complicates an admission to Christchurch Hospital. The rates observed in our study for admissions for acute gout were similar to those observed in a previous nationwide study between 1999 and 2009 in which gout accounted for 0.09%–0.11% of acute admissions in New Zealand, but higher than the 0.049%–0.070% of acute admissions observed in the United Kingdom[5]. Interestingly, the rates observed for gout as a secondary diagnosis were lower than those observed in the previous study, in which rates in New Zealand of 0.96% in 1999–2000 and 0.26% in 2008–2009 were observed[5]. There are several possibilities for this lower rate. First, the rate of gout-complicating admissions may have continued to decline over time. Second, the coding for gout-complicating admissions for alternative reasons may have been inaccurate. Third, there is a much lower proportion of Maori and Pacific Island people, who have high rates of gout, living in the Canterbury area, where the Christchurch hospital is located. The length of stay of 6.5 days is likely to result in significant healthcare costs that could be avoided by improved management of gout[3].

Prednisone was the most commonly used agent for acute gout. Although NSAID and colchicine are the recommended

Table 2. Demographics and clinical characteristics of those seen by rheumatology service compared to those not seen by rheumatology service and those with gout as the primary versus secondary diagnosis. Values are n (%) unless otherwise specified.

Characteristics	Seen by Rheumatology, n = 39	Not Seen by Rheumatology, n = 196	p	Primary Diagnosis, n = 95	Secondary Diagnosis, n = 140	p
Age, yrs, mean (SD)	64.0 (16.9)	73.6 (15.1)	< 0.001	68.2 (17.5)	74.6 (13.9)	0.002
Comorbidities						
Type 2 diabetes mellitus	14 (35.9)	54 (27.6)	0.31	29 (30.5)	40 (28.6)	0.71
Cardiovascular disease	23 (59)	146 (74.5)	0.04	62 (65.3)	108 (77.1)	0.046
Cerebrovascular disease	4 (10.3)	47 (23.9)	0.6	14 (14.7)	37 (26.4)	0.03
CKD, GFR < 60 ml/min/1.73 m^2	22 (57.9)	120 (61.2)	0.42	52/88 (59.1)	90/136 (66.2)	0.28
eGFR ml/min/1.73m^2, mean (SD)	54.6 (23.5)	51.8 (23.5)	0.52	54.4 (25.9)	50.9 (22.0)	0.29
≥ 2/4 comorbidities	17 (43.6)	101 (51.5)	0.35	46 (48.4)	73 (52.1)	0.58
Concomitant medications						
ACE inhibitor	13 (33.3)	68 (34.7)	0.85	24 (25.3)	58 (41.4)	0.01
Diuretic	16 (41)	88 (44.9)	0.64	39 (41.1)	66 (47.1)	0.36
α blocker	3 (7.7)	27 (13.8)	0.29	13 (13.7)	18 (12.9)	0.85
β blocker	18 (46.2)	107 (54.6)	0.32	44 (46.3)	81 (57.9)	0.08
Calcium channel blocker	4 (10.3)	36 (18.5)	0.21	14 (14.7)	27 (19.3)	0.37
Aspirin	19 (48.7)	91 (46.4)	0.82	40 (42.1)	71 (50.7)	0.19
Lipid-lowering agent	15 (38.5)	70 (35.7)	0.76	28 (29.5)	58 (41.4)	0.06
≥ 3 other medications	21 (53.8)	103 (52.6)	0.91	42 (44.2)	83 (59.3)	0.02

CKD: chronic kidney disease; GFR: glomerular filtration rate; eGFR: estimated GFR; ACE: angiotensin-converting enzyme.

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Table 3. In-hospital management of those seen by rheumatology service compared to those not seen by rheumatology service and those with gout as the primary versus secondary diagnosis. Values are n (%) unless otherwise specified.

Variables	Total Admissions, n = 235	Seen by Rheumatology, n = 39	Not Seen by Rheumatology, n = 196	p	Primary Diagnosis, n = 95	Secondary Diagnosis, n = 140	p
Aspiration undertaken	72 (30.6)	22 (56.4)	50 (25.5)	< 0.001	52 (54.7)	20 (14.3)	< 0.001
Serum urate checked	123 (52.3)	32 (82.1)	91 (46.4)	< 0.001	58 (61.1)	65 (46.4)	0.03
Urate-lowering therapy altered, change considered or recommended to GP on discharge	94 (40)	32 (82.1)	62 (31.6)	< 0.001	56 (58.9)	38 (27.1)	< 0.001
In-hospital, days, mean (SD)	6.5 (10.7)	5.3 (5.3)	6.7 (11.5)	0.44	3.4 (3.6)	8.6 (13.2)	< 0.001
Seen by rheumatology service	39 (16.6)	N/A	N/A		24 (25.3)	15 (10.7)	0.003

GP: general practitioner; N/A: not assessed.

first-line therapies for gout, these agents were used in only 11.5% and 4.7% of cases, respectively. This most likely reflects the population, which had multiple comorbidities including CKD and concomitant medications, perhaps precluding the use of NSAID and colchicine. This high use of prednisone is similar to that observed in another New Zealand hospital, but much higher than that reported in an Australian study in which only 28% of inpatients received oral prednisone for acute gout[11] (Table 4)[5,10,11,12,14].

There are several key aspects of the longterm management of gout that were suboptimal. In only 26% of admissions were patients receiving urate-lowering at the time of admission and in only 52.3% of admissions was serum urate measured. Further, the majority of those receiving urate-lowering therapy who had a serum urate measured were not at target urate levels. A small minority of patients commenced or had the dose of allopurinol changed during their inpatient stay and a minority had a recommendation about urate-lowering therapy made to their general practitioner in the discharge documents. This failure to initiate, change, or recommend alterations in urate-lowering therapy to achieve target urate represents a significant lost opportunity to improve longterm gout management and is similar to that observed in other studies (Table 4). Although the new American College of Physicians gout guidelines[15] do not advocate testing serum urate, the treat-to-target urate strategy suggested by both the American College of Rheumatology (ACR)[8] and the European League Against Rheumatism (EULAR)[7] for individuals with "symptomatic" gout severe enough to warrant admission to hospital would support consideration of a change in urate-lowering therapy. In those individuals for whom gout is a secondary diagnosis and the acute gout attack may have been precipitated by alterations in therapy, such as diuretics for heart failure or sepsis, checking serum urate just prior to discharge or recommending it is checked after discharge may be more appropriate.

The rheumatology service was only involved in 16% of admissions. This is lower than that reported in a similar hospital audit in Australia, where 40/118 (33.9%) were seen by rheumatology services[11]. Those individuals seen by the rheumatology service were more likely to have changes to urate-lowering therapy. The data in our audit also suggest that those individuals with gout as a secondary discharge diagnosis rather than primary discharge diagnosis are less likely to be seen by the rheumatology service, less likely to have a joint aspirated, and less likely to have changes in urate-lowering therapy.

Given the high prevalence of gout, it is not practical for all patients to be seen by a rheumatology service. Appropriate and effective management by other healthcare professionals, in particular general physicians and general practitioners, is vital to improving outcomes for people with gout. Our audit would suggest that the focus of efforts should be on improving the implementation and uptake of the treat-to-target serum urate strategy, with more intensive use of urate-lowering therapy rather than on acute gout management. Traditional teaching has been that urate-lowering therapy should not be commenced during an acute attack, but started about 2 weeks after the attack has settled. Two small clinical trials have suggested that starting urate-lowering therapy during a gout attack that is being treated appropriately does not prolong the attack[16,17]. To our knowledge, there is currently no consensus on this aspect of management, with the ACR guidelines recommending that urate-lowering therapy be started during an attack as long as the attack is treated appropriately, and EULAR making no recommendation in this regard, citing lack of evidence[7,8]. Thus at the very least, recommendations should be made on discharge about urate-lowering therapy.

Patient education and understanding of the disease are also vital to ensuring adherence with urate-lowering therapy. The greater patients' understanding of their illness, the more likely they are to adhere to urate-lowering therapy[18]. Evidence suggests that few patients receive clear explanations of gout or appropriate management advice[19], resulting in patients both holding misconceptions about gout and having unanswered questions about its etiology and management[20,21]. Hospital admission provides an ideal opportunity to provide patient education. An inpatient and hospital health professional may have more time than is available in primary

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Table 4. Studies examining hospital admissions and treatment for gout.

Study	Yr(s)	Study Design	% Admissions for Gout of Total Hospital Admissions	Treatment of Acute Gout (%)				Urate-lowering Therapy (%)					Discharge Recommendations
				Prednisone	NSAID	Colchicine	ACTH	ULT at Admission	ULT Stopped	ULT Started	ULT Dose Increased	ULT Dose Decreased	
Our current study (NZ)	2013–2014	Retrospective note review	Primary diagnosis: 0.12% of acute admissions; secondary diagnosis: 0.15% admissions	72.3	11.5	4.7	0	25.5	N/A	5.1	3.3	N/A	18.4% starting ULT advised to GP
Kennedy, *et al*[10] (NZ)	2012	Retrospective note review	N/A	61	40	40	15	50		31	11	N/A	Advised to see GP *et al*[10] 36%, "treat-to-target" serum urate was initiated/recommended 9%
Gnanenthiran, *et al*[11] (Australia)	2005–2006	Retrospective note review	N/A	28	32	75	1	26.9	56	N/A	19	8	N/A
Petersel and Schlesinger[14] (USA)	2007	Retrospective note review	N/A		21	53		27	3	N/A	N/A	N/A	N/A
Kamalaraj, *et al*[12] (Australia)	2008–2009	Retrospective note review	N/A	32.6	22.5	76.4	0	18	N/A	N/A	N/A	N/A	N/A
Robinson, *et al*[5] (NZ and England)	1999–2009	Administrative data	NZ 1999: 0.09% primary, 0.96 secondary; 2008–09: 0.11% primary, 0.26% secondary; UK 1999: 0.021%; 2008–09: 0.031%	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

NSAID: nonsteroidal antiinflammatory drug; ACTH: adrenocorticotropic hormone; ULT: urate-lowering therapy; NZ: New Zealand; GP: general practitioner; N/A: not assessed.

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care to reinforce education. This may also be a time when people with gout are more receptive to receiving information given the disease has resulted in hospitalization.

Our audit confirmed the need for improved longterm management of those admitted with gout. While our study is not dissimilar to previous studies of the hospitalizations for gout, we believe the focus needs to shift to include urate lowering as well as antiinflammatory treatment during the inpatient period. Strategies to improve treat-to-target urate in hospital as well as clear discharge advice to the general practitioner are important and should be emphasized in hospital protocols.

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