K. Ting, T. K. Gill, H. Keen, G. R. Tucker, and C. L. Hill

Prevalence and associations of gout and hyperuricaemia: results from an Australian population-based study

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TITLE:

Prevalence and Associations of Gout and Hyperuricaemia: Results from an Australian Population-based Study

ABSTRACT:

Background:

Despite gout and hyperuricaemia being major co-morbid health issues worldwide, there is a large epidemiological gap regarding their impact in the Australian community.

Aims:

To determine the prevalence and associations of self-reported medically diagnosed gout, and hyperuricaemia, in an Australian population-based cohort.

Methods:

The North West Adelaide Health Study (NWAHS) is a longitudinal cohort study consisting of three stages of data collection. Each stage comprised a self-complete questionnaire, clinic assessment and Computer Assisted Telephone Interview (CATI). In Stage 3 (2008-2010) participants were asked if a doctor had ever diagnosed them with gout. Additional data included demographics, co-morbidities, laboratory data and SF-36. Participants were defined as having gout if they had self-reported medically diagnosed gout or were taking any gout specific medication (allopurinol, colchicine, probenecid). Hyperuricaemia was defined as a serum uric acid level >0.42mmol/L in men and >0.34mmol/L in women.

Results:

The overall prevalence of gout was 5.2%. Males were significantly more likely to have gout than females (8.5% vs 2.1%, $p<0.001$). The overall prevalence of hyperuricaemia was 16.6%, with being male again identified as a significant risk factor (17.8% vs 15.4%, $p<0.01$). Both gout and hyperuricaemia were associated with male sex, body mass index, and renal disease, after

multivariable adjustment. There was no significant difference reported in quality of life (mean SF-36) scores in gout participants, compared to unaffected individuals.

Conclusion:

In the South Australian population the prevalence of gout and hyperuricaemia is high. This study emphasises the need for optimal diagnosis and management of gout in Australia.

KEYWORDS:

Gout, Hyperuricaemia, South Australia, Prevalence, Epidemiology

AUTHORS & INSTITUTIONS:

Authors' names:

Kimberley Ting[1], Tiffany K Gill[2], Helen Keen[3], Graeme R Tucker[2], Catherine L Hill[1,4]

Authors' affiliation(s):

[1]Department of Rheumatology, The Queen Elizabeth Hospital, Woodville, South Australia [2]School of Medicine, University of Adelaide, Adelaide, South Australia, [3]School of Medicine and Pharmacology, University of Western Australia, [4] The Health Observatory, Discipline of Medicine, The University of Adelaide, South Australia

Corresponding author full contact details:

Name: Catherine L Hill

Address: 28 Woodville Road, Woodville South

Post code: 5011

City: Adelaide

Country: Australia

Email: Catherine.hill@health.sa.gov.au

BACKGROUND:

Gout is a major health issue worldwide. It is significantly associated with cardiovascular disease, and is an independent risk factor for all cause mortality.[1,2] Estimates from WHO Global Burden of Disease studies show a 49% increase in gout disability-adjusted life years from 1990 to 2010.[3] Hyperuricaemia, a pre-requisite for gout, is associated with the metabolic syndrome and is an independent risk factor for stroke and chronic kidney disease.[4,5] In keeping with such disease burden, gout and hyperuricaemia pose a significant socio-economic loss; in the United states minimal estimates for annual costs exceed 6 million US dollars.[6]

The prevalence of gout and hyperuricaemia varies depending on population and disease definition. One of the most extensive epidemiological studies was undertaken by Winnard et al., who used national health data sets to sample more than 4 million of the Aoteoroa New Zealand (NZ) population;[7] this group identified a prevalence of gout of 3.75%. Both Winnard and Stamp et al also found a higher prevalence or hyperuricaemia in the NZ Maori population compared with the Non-Maori population.[7,8] In the general US population, the prevalence of medical record diagnosed gout was 3.9% and the prevalence of hyperuricaemia was 21.4%.[9] In the UK and Europe the prevalence of gout is considerably lower; a retrospective analysis of general practitioner records from 2000-2005 revealed a gout prevalence of only 1.4%.[10] Similarly an Italian 2005-2009 primary care database revealed a gout and hyperuricaemia prevalence of only 0.9% and 11.9% respectively.[11]

Obesity, hypertension, dyslipidaemia and chronic renal failure have consistently been identified to be increased in the gout population.[8,10,11,12,13] The association of diabetes mellitus and a current smoking

history are less well defined with some studies finding them to be less prevalent in people with gout.[2,10,12]

To date health related quality of life (HRQoL) has not been extensively assessed in gout trials. One study found that after adjusting for potential medical and sociodemographic confounders gout was an independent predictor for lower quality of life and functional outcomes.[14] This study was, however, performed using gout patients from rheumatology clinics rather than the general population. Gout patients with frequent flares and the presence of tophi have lower HRQoL.[15]

Despite this international knowledge there is a gap in epidemiological evidence to both qualify and quantify the burden of gout and hyperuricaemia in the Australian community. Most recently an Australian General Practice observational study identified a crude gout prevalence of 1.54%, and recognised its poor management in Australian primary care[16]. Prior to this, a 2012 systematic review of Australian studies identified only 3 publications that investigated the prevalence of both gout and hyperuricaemia.[17] Of these only one used data in the 21st century and this study was limited to males aged 70 years and over. Apart from a 2002 epidemiological study dedicated to a select Aboriginal community in North Queensland, there have been no population studies investigating the prevalence and co-morbid associations of gout in Australia.[18]

Population-based data is required to quantify the burden of hyperuricaemia and gout in the Australian community, to enable clinicians and health policy advisers to deliver health care for gout and hyperuricaemia in the most effective manner.

The aim of this study is to assess the prevalence and associations of self-reported medically-diagnosed gout and hyperuricaemia in an Australian population-based cohort.

AIMS:

The aim of this study is to assess the prevalence and associations of self-reported medically-diagnosed gout and hyperuricaemia in an Australian population-based cohort.

METHODS:

Setting and Study Population:

The North West Adelaide Health Study (NWAHS) is a representative longitudinal study of 4056 adults aged 18 years and over at the time of recruitment from the north-west region of Adelaide, South Australia. Participants were randomly selected from the electronic white pages. The sample region represents approximately half of the metropolitan area (total population of approximately 1.2 million) and almost one-third of the population in South Australia (population of approximately 1.6 million), which has the second highest elderly population of all the Australian states and territories.[19] The region also reflects the demographic profile of the state, covering a broad range of socioeconomic areas.

Three phases of data collection have been conducted; in 1999-2003 (Stage 1), 2004-2006 (Stage 2) and 2008-2010 (Stage 3). Specifically, in stage 3 of the study participants were contacted and invited to participate in a computer-assisted telephone interview (CATI), self-completed questionnaire and a clinical assessment. Of the original 4056 participants, 346 participants were either deceased or unable to participate due to severe illness. Of the remaining 3710, 2710 (73.0%) completed the telephone

questionnaire2638 (71.1%) the self-reported questionnaire and 2487 (67.0%) attended the clinic assessment.

Data Collection

As part of the CATI, participants were asked 'Have you ever been told by a doctor that you have gout?" In addition age, sex and education level of the participants was determined. Self-reported doctor-diagnosed prevalence of co-morbidities were also determined from the CATI. Quality of life information was obtained using the Short Form 36 Version 2 (SF-36® V2).[20] Medication data was obtained using records matched to Medicare numbers using the Pharmaceutical Benefits Scheme (PBS) database. Participants were defined as having gout if they had self-reported medically diagnosed gout or were taking any gout specific medication (allopurinol, colchicine, probenecid). This definition has been used previously in population prevalence studies and has been shown to be both a reliable and sensitive definition for epidemiological purposes.[9,21]

The Socio-Economic Index for Areas (SEIFA) Index of Relative Social Disadvantage (IRSD) was determined from postcodes and used to provide a measure of socioeconomic status. These values are produced by the Australian Bureau of Statistics and are a composite measure of a range of socioeconomic characteristics based on Census data which is obtained every five years.[22] The IRSD scores were grouped into quintiles (highest, high, middle, low and lowest) for analysis, where the highest quintile represents postcodes with the highest IRSD scores (least disadvantaged areas) and the lowest quintile represents postcodes with the lowest IRSD scores (most disadvantaged areas).

From the clinic assessment height and weight were measured using standardised protocols, and used to determine body mass index (BMI, weight (kg)/height (m^2)). The categories of BMI were determined according to the World Health Organization criteria.[23] Blood pressure was measured with a standard sphygmomanometer to determine the presence of high blood pressure (Grade 1 ≥ 140/90, Grade 2 ≥160/100, Grade 3 ≥ 180/110). Fasting blood tests were also undertaken to determine fasting plasma glucose (FPG), low density lipoprotein (LDL), high density lipoprotein (HDL), HbA1c, serum uric acid (SUA) and creatinine levels. Creatinine levels were used to calculate the estimated glomerular filtration rate for quantifying kidney disease. Hyperuricaemia was defined as a serum uric acid level >0.42mmol/L in men and >0.34mmol/L in women. This equates to the same definition of hyperuricaemia used in the US NHANES-III study.[9] Presence of diabetes was defined as self-reported diabetes or FPG>=7.0mm/L.

This study was approved by Human Research Ethics committee of The Queen Elizabeth Hospital, Adelaide, South Australia and all participants provided informed consent. Consent was obtained separately from participants to access PBS data, with 93.8% agreeing to provide their Medicare number.

Data Weighting and Statistical Analyses

In Stage 1, data were weighted by western and northern health regions, age group, sex and the probability of selection in the household to the Australian Bureau of Statistics 1999 Estimated Resident Population and the 2001 Australian Census data. Stage 3 was reweighted using the 2009 Estimated Resident Population, incorporating participation in each of the three components, to adjust the original weight from Stage 1 in the calculation. All analyses undertaken in this paper use this weight.

Statistical analyses were conducted using SPSS, version 22. Univariable logistic regression analysis compared the gout group to those without gout to determine the odds ratios for demographic factors. All variables, including co-morbidities, were then included in a multivariable logistic regression analysis, and non-significant variables were removed in a backward elimination process to determine factors ($p<0.05$) associated with gout. Statistically significant differences ($p<0.05$), adjusted for sex, age, and BMI, between those with and without gout, for HbA1c, fasting plasma glucose, creatinine and serum uric acid were also determined using multiple analyses of variance. The same process of analysis was used to determine odd ratios and associations for participants with hyperuricaemia compared to participants with normal serum uric acid.

RESULTS:

Overall 2389 participants provided a response to self-reported medically diagnosed gout, and 2462 provided a blood sample to be tested for serum urate. The overall prevalence of gout was 5.2% (Table 1). Males were significantly more likely to have gout than females (8.5% vs 2.1%, $p<0.01$). Gout was also associated with increasing age. Gout was not associated with socio-economic status or educational attainment (data not shown). Gout was associated with body mass index, hypertension, diabetes mellitus, chronic kidney disease and cardiovascular disease (Table 2). However when adjusted for co-morbid variables, only higher body mass index and chronic kidney disease were strongly associated with gout. Interestingly a current smoking history was inversely associated with gout (OR 0.40, 95%CI 0.19-0.84), although this association becomes non-significant after adjustment for age and BMI (Table 2).

The overall prevalence of hyperuricaemia was 16.6% (Table 3), with being male again identified as a significant risk factor (17.8% vs 15.4%, p<0.01). Hyperuricaemia was associated with higher body mass index, hypertension, chronic kidney disease and cardiovascular disease (Table 4). Akin to the findings with gout participants, following adjustment for co-morbid variables, higher body mass index and chronic kidney disease remained strongly association with hyperuricaemia. In contrast to gout participants, even after adjustment for age and BMI, a current smoking history remained significantly associated with hyperuricaemia (OR 0.69, 95%CI 0.48-1.00).

The mean serum uric acid level in participants with gout was significantly higher compared to those without gout (p <0.01, Table 5). The mean serum creatinine in participants with gout and hyperuricaemia was significantly higher compared to those participants without gout and hyperuricaemia respectively (p<0.01). There was no significant difference reported in mean SF-36 scores in the gout participants compared to those without gout (Table 6).

Medication data revealed that 40.3% of gout participants were taking allopurinol, 13.7% taking colchicine and none taking probenicid. This may be an underestimation these medications are all very low cost medicine in Australia, and therefore not always recorded on the PBS database

DISCUSSION:

This study was undertaken to augment the limited epidemiological data available on gout and hyperuricaemia in Australia. Our study population, sampling from a region representing almost one-third of the South Australian population, dealt exclusively with participants with self-reported medically diagnosed gout. The overall prevalence of gout of 5.2% was high, comparable to large New

Zealand population studies and surprisingly close to the 6.05% prevalence in the Maori population.[7] It is higher than longitudinal databases of selected populations in the UK (1.4%), US (3.9%) and Italy (0.91%).[10,11,13] The only comparable Australian study is a 2002 Indigenous Australian study[18], for which self-reported gout was confirmed by clinical examination and investigation, which found an overall prevalence of 3.8% (males 9%, females 0.7%). From a participant self-reported population survey from the Australian Bureau of Statistics (1997-1999), the prevalence of gout was 1.7%.[17]

An recently published study of Australian general practice dataset demonstrated a prevalence of 1.54% using a definition of gout by use of allopurinol or colchicine, or diagnosis of gout (including tophus, tophi and podagra) within the general practice record. [16] In addition, allopurinol was only prescribed to 57% of patients with gout.[16]

Our study population also revealed a high prevalence of hyperuricaemia of 16.6%. This is similar to New Zealand (13.7%),[8] US (21.4%) and Italian (11.9%) studies.[11,13] The significantly increased prevalence of both gout and hyperuricaemia in the male population was also consistent with international data.[7,12] With respect to other traditional cardiovascular risk factors, obesity, hypertension and diabetes mellitus were all increased in the gout and hyperuricaemia participants however did not reach statistical significance following adjustment for age, sex and BMI. In particular the risk of diabetes in gout, independent of the known diabetic risk factor of obesity, has been further investigated in a UK study[24]. In this cohort analysis adjusted for BMI, the incident rate for diabetes in the total gout population was significantly higher than that in the total non-gout population. Of importance, the outcome assessed was defined as incident diabetes requiring at least one prescription for medication for the treatment of diabetes, suggesting the issue of diabetes and gout probably relates to control of diabetes.

After adjusting for co-morbid variables including obesity, our study agreed with previous studies correlating chronic kidney disease with gout and hyperuricaemia.[12] Similarly, after adjusting for age, sex and body mass index, we did not find a significant association between hyperuricaemia and cardiovascular disease. Our study supports the evidence that rather than being an independent risk factor for cardiovascular disease, hyperuricaemia is likely intricately related to cardiovascular risk factors, such as obesity, and is merely a marker of risk for future cardiovascular disease. This is consistent with the Framingham study[25], which did not demonstrate an increase in coronary artery disease or death from cardiovascular disease, unless a high cardiovascular risk cohort was selectively studied. Body mass index has been suggested as a major confounder in observational studies[26]; indeed in the very well clinically defined Fremantle Diabetes Study (FDS) no independent relationship between hyperuricaemia and mortality was seen after adjustment for significant variables such as BMI.[27]

Our study also found that the absolute percentage of current smokers was lower in patients with gout however this was not significant following adjustment. The role of smoking is interesting and there have been several recent studies, including an Australian GP prevalence study, suggesting an inverse relationship between smoking and gout.[2, 16,28] Most recently, data from the Framingham Heart Study cohort found that current cigarette smoking was associated with a lower incidence of gout independent of the prevalence of other traditional risk factors (HR 0.76 (95% CI 0.59, 0.98).[29] One theory postulated is that the oxidative stress from chronic exposure to cigarette smoke results in decreased uric acid production[30]. This would support out findings of lower serum uric in current smokers. Other studies of smoking suggest that smoking inhibits the innate immune responses[31], and one might hypothesise that this suppression might attenuate the neutrophil driven inflammatory response against monosodium urate crystals in gout. Further mechanistic and epidemiological studies

specifically investigating cigarette smoking on hyperuricaemia and gout would strengthen the current limited knowledge.

We did not find any association between gout and socio-economic status. One could reasonably hypothesise that gout, like other cardiovascular risk factors, is increased in lower socio-economic regions. In New Zealand, Winnard et al. found that participants in socio-economically deprived areas were 1.4 times more likely to have gout compared with those in socio-economically advantaged areas. The impact of socio-economic status on the burden of disease in Australia warrants further study.

As already alluded to, there is also sparse information locally and internationally regarding gout and health related quality of life. An Italian study specifically investigating the impact of gout, also utilising SF-36 scores, found significant debility in those with chronic gout.[14] However this study recruited patients from rheumatology centres, who are more likely to be more severe cases and potentially during flares of gout, resulting in worse SF-36 scores.[14] Unlike these clinic based studies, we did not find an association between gout and quality of life in this population-based study. This may be related to the fact that we didn't have any measures of gout severity such as flare frequency or tophi.

In our study, gout was defined by self-reported medically diagnosed gout. Whilst the gold-standard definition of gout is the presence of monosodium urate crystals in synovial fluid analysis or tophi[30], this can not be practically applied to large population-based studies. The definition of gout in epidemiological studies varies and includes, either in isolation or combination, physician diagnosed gout based on history and examination, prescription practices, and analysis of healthcare datasets or coding systems. We believe self-reporting of medically diagnosed gout is a reliable definition for epidemiological purposes where establishing demographic and clinical associations in a population

study is the aim. This is supported by a 2011 US study specifically addressing the question of reliability and sensitivity of self-reported physician diagnosed gout.[21] Using a large cohort consisting of more than 32,000 participants this group found that self-reported physician diagnosed gout was consistent both over multiple questionnaires, and with the duration of time. They also found a sensitivity of 84% compared against the gold standard, defined as a hospital discharge diagnosis of gout or use of gout specific medications (colchicine, probenicid, allopurinol).

Our study was limited in that we did not address the relationship between gout and medications. Therefore, we were unable to assess the prevalence or adherence to gout specific medications. We also did not have data on potentially hyperuricaemic agents such as diuretics and aspirin. The relationship between features of gout reported to impact quality of life - tophi, polyarticular involvement and recurrent attacks[14,15] – was also not investigated. In addition, we had no information on the severity of gout in terms of frequency of acute attacks, presence of tophi or joint damage.

CONCLUSION:

This Australian population-based study demonstrates the high prevalence of gout and hyperuricaemia in Australia. This highlights the need for improved diagnosis and management of gout in Australia.

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Table 1: Prevalence and demographic of Gout

	Gout %	No Gout %	Adjusted OR* (95%CI)	p value
Total (n=2389)	5.2%	94.8%		
Female (n=1230)	2.1%	97.9%	1.00 (ref)	
Male (n=1159)	8.5%	91.5%	5.29 (3.24-8.64)	<0.01
Age				
22-44 years (n=1012)	1.6%	98.4%	1.00 (ref)	
45-54 years (n=484)	3.5%	96.5%	1.93 (0.93-4.00)	0.08
55-64 years (n=385)	7.0%	93.0%	3.31 (1.66-6.62)	<0.01
65-74 years (n=262)	11.8%	88.2%	5.78 (2.84-11.75)	<0.01
≥75 years (n=244)	12.7%	87.3%	4.09 (1.79-9.35)	<0.01
Socioeconomic Status				

Q1 (n= 607)	5.8%	94.2%	1.00 (ref)	
Q2 (n=648)	4.9%	95.1%	1.06 (0.62-1.83)	0.83
Q3 (n=535)	4.7%	95.3%	0.97 (0.54-1.76)	0.93
Q4 (n=455)	6.2%	93.8%	1.03 (0.58-1.86)	0.91
Q5(n=120)	3.3%	96.7%	0.99 (0.33-2.92)	0.98

*The odds ratios (ORs) were adjusted for the following variables: age, sex, body mass index. Q1 = Quintile 1 (least disadvantaged), Q2 = Quintile 2, Q3 = Quintile 3, Q4 = Quintile 4, Q5 = Quintile 5 (most disadvantaged)

Ref = reference value.

Table 2: Associations of Gout and other comorbidities

	Gout % (n = 124)	**No Gout % (n =2265)**	**Adjusted OR* (95% CI)**	**p value**
Body Mass Index				
BMI <25	11.4%	28.5%	1.00	
BMI 25-<30	39.8%	38.3%	1.49 (0.78-2.83)	0.22
BMI 30-<35	30.9%	20.6%	2.39 (1.22-4.68)	0.01
BMI 35+	17.9%	12.6%	3.29 (1.55-6.99)	<0.01
Hypertension				
No Hypertension	55.3%	81.3%	1.00	
Grade 1	31.7%	14.8%	1.54 (0.96-2.47)	0.07
Grade 2 / Grade 3	13.0%	3.9%	1.77 (0.88-3.56)	0.11
Diabetes Mellitus	23.4%	7.6%	0.71(0.41-1.23)	0.22
Smoking				
Non-smoker	49.2%	50.5%	1.00	
Current	6.7%	17.8%	0.93 (0.61-1.44)	0.76
Past history	44.2%	31.6%	0.49 (0.22-1.09)	0.08
Cardiovascular disease	21.8%	6.3%	0.62 (0.35-1.10)	0.10
Chronic kidney disease				
Stage 2	81.3%	96.0%	1.00	

Stage 3	15.4%	3.9%	2.53 (1.29-4.95)	0.01
Stage 4/Stage 5	3.3%	0.1%	8.06 (1.18-55.17)	0.03

*The odds ratios (ORs) were adjusted for the following variables: age, sex, body mass index. Hypertension as classified by National Heart Foundation - (Grade 1 ≥ 140/90, Grade 2 ≥160/100, Grade 3 ≥ 180/110). Chronic kidney disease categorised as per National Kidney Foundation. LDL = low-density lipoprotein, diabetes, smoking status, cardio vascular disease. Ref = reference value.

Table 3: Prevalence and demographic of Hyperuricaemia

	Hyperuricaemia (%)	**No Hyperuricaemia (%)**	**Adjusted OR* (95%CI)**	**p value**
Total (n=2462)	16.6%	83.4%		
Female (1271)	15.4%	82.6%	1.00 (ref)	
Male (n=1191)	17.8%	82.2%	1.44 (1.12-1.85)	0.01
Age				
22-44 years (n=1012)	13.8%	86.2%	1.00 (ref)	
45-54 years (n= 488)	14.2%	85.8%	0.74 (0.52-1.04)	0.08
55-64 years (n=387)	16.2%	83.8%	0.86 (0.60-0.1.25)	0.44
65-74 years (n=263)	18.4%	81.6%	0.93 (0.60-1.45)	0.76
≥75 years (n=246)	29.3%	70.7%	1.08 (0.66-1.77)	0.76
Socioeconomic status				

Q1 (n=613)	21.5%	78.5%	1.00 (ref)	
Q2 (n=646)	17.5%	82.5%	0.83 (0.61-1.14)	0.25
Q3 (n=543)	10.7%	89.3%	0.54 (0.37-0.77)	<0.01
Q4 (n=449)	14.5%	85.5%	0.67 (0.47-0.97)	0.03
Q5 (n=120)	18.3%	81.7%	1.17 (0.68-2.03)	0.57

*The odds ratios (ORs) were adjusted for the following variables: age, sex, body mass index. Ref = reference value. Q1 = Quintile 1 (least disadvantaged), Q2 = Quintile 2, Q3 = Quintile 3, Q4 = Quintile 4, Q5 = Quintile 5 (most disadvantaged)

Table 4: Associations of Hyperuricaemia and other co-morbidities

	Hyperuricaemia % (n=413)	**No Hyperuricaemia % (n=2034)**	**Adjusted OR (95% CI)**	**p value**
Body Mass Index				
BMI <25	10.4%	28.6%	1.00 (ref)	
BMI 25-<30	35.8%	41.1%	1.78 (1.22-2.60)	<0.01
BMI 30-<35	29.3%	20.2%	3.24 (2.18-4.83)	<0.01
BMI 35+	24.5%	10.2%	7.56 (5.02-11.41)	<0.01
Hypertension				
No Hypertension	63.0%	74.1%	1.00 (ref)	
Grade 1	26.9%	20.5%	1.21 (0.87-1.67)	0.27
Grade 2 / Grade 3	10.2%	5.5%	1.44 (0.83-2.51)	0.20
Diabetes Mellitus	15.5%	10.4%	0.91 (0.59-1.41)	0.68
Smoking				
Non-smoker	45.7%	45.2%	1.00 (ref)	
Current	8.1%	15.4%	0.68 (0.47-0.99)	0.04
Past history	46.2%	39.4%	1.00 (0.76-1.31)	1.00

CVD	12.6%	9.2%	1.25 (0.77-2.03)	0.38
Chronic kidney disease				
Stage 2	75.7%	96.1%	1.00 (ref)	
Stage 3	22.6%	3.6%	10.31 (6.26-16.98)	<0.01
Stage 4/Stage 5	1.7%	0.3%	6.61 (1.22-35.71)	0.03

*The odds ratios (ORs) were adjusted for the following variables: age, sex, body mass index (BMI). BMI as defined by WHO criteria. Hypertension as classified by National Heart Foundation - (Grade 1 ≥ 140/90, Grade 2 ≥160/100, Grade 3 ≥ 180/110). Chronic kidney disease categorised as per National Kidney Foundation. LDL = low-density lipoprotein, diabetes, smoking status, cardio vascular disease. Ref = reference value

Table 5 Mean laboratory values for participants with gout and hyperuricaemia

	Gout	No gout	p value
LDL	2.87	3.06	0.03
HDL	1.51	1.46	0.16
Serum urate	0.35	0.32	<0.01
HbA1c	5.74	5.69	0.43
Creatinine	82.13	73.24	<0.01
Fasting plasma glucose	5.17	5.14	0.80
	Hyperuricaemia	No Hyperuricaemia	p value

LDL	3.13	3.03	0.05
HDL	1.46	1.47	0.71
HbA1c	5.71	5.70	0.75
Creatinine	83.39	72.05	<0.01
Fasting plasma glucose	5.13	5.15	0.76

*The means were adjusted for the following variables: age, sex, body mass index (BMI). BMI as defined by WHO criteria. LDL = low-density lipoprotein, HDL = high-density lipoprotein. HbA1c = Haemoglobin A1c.

Table 6: Mean SF-36 scores for those with gout and without gout by gender

	Males		Females		Gout v No gout (persons)	Males with gout v Females with gout
	Gout	No Gout	Gout	No Gout	p-value	p-value
Physical functioning	81.04	83.42	68.43	78.37	0.26	0.12
Role physical	80.04	85.67	81.89	82.42	0.09	0.38
Bodily pain	72.00	72.78	70.85	69.56	0.71	0.76
General health	63.67	68.19	67.58	67.84	0.05	0.24
Vitality	58.61	59.95	57.46	54.71	0.47	0.74
Social functioning	81.67	84.37	85.05	79.79	0.82	0.37
Role emotional	89.51	90.61	90.74	87.35	0.59	0.53
Mental health	77.23	78.11	81.35	75.02	0.31	0.19