

The association of gout with an increased risk of hypertension and diabetes mellitus among stroke survivors in New Zealand: A cross-sectional study using routinely collected electronic health data

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Abstract

Background: There is a paucity of data relating to the association of gout with the occurrence of hypertension and diabetes mellitus in patients with stroke. This study aimed to determine the association of gout with the risk of hypertension and diabetes mellitus in a cohort of stroke patients from Auckland, Aotearoa New Zealand.

Methods: A cross-sectional study was conducted among stroke survivors in South and East Auckland, New Zealand from the years 2010 to 2014. Electronic health record data were collected and analysed using Statistical Package for Social Science version 23. Multivariate logistic regression modelling adjusted for age, gender, and ethnicity was conducted to determine the association of gout with the risk of hypertension and diabetes mellitus in patients discharged with a diagnosis of stroke.

Results: The age-, gender-, and ethnicity-adjusted odds ratio for having hypertension and diabetes mellitus among stroke survivors with gout history were 3.25 (95% confidence interval 1.32–8.03) and 1.94 (95% confidence interval 1.12–3.36), respectively. Māori stroke survivors with gout history had the highest risk of having diabetes mellitus with age- and gender-adjusted odds ratio of 5.10 (95% confidence interval 1.90–18.93).

Conclusion: The findings from this study suggest gout may be independently associated with an increased risk of hypertension and diabetes mellitus in patients with stroke. Māori who are the indigenous population of New Zealand show a greater risk of diabetes mellitus associated with a gout diagnosis compared to other populations. This finding highlights the importance of the need for further research with Māori stroke survivors and other indigenous populations.

Keywords

Cardiology, cardiology, cerebrovascular disease/stroke, diagnostic testing, gout, stroke, uric acid, hypertension, link between gout and hypertension and diabetes mellitus, other diagnostic testing

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Introduction

Stroke continues to be a major health concern globally and this is related to its mortality, morbidity, disability, and socio-economic impacts.^{1–3} Hence, stroke management remains strongly focused on cardiovascular (CV) risk factor-targeted preventive strategies. Hypertension and diabetes mellitus are well-known traditional CV risk factors for stroke and are the two major targets in primary and secondary stroke prevention.⁴ Apart from the conventional CV risk factors, gout and its precursor, serum urate,^{5,6} are also currently known to contribute to the occurrence and progression of

cardiovascular diseases (CVDs) like stroke and some of the known CV risk factors.^{5–21}

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Gout precursor, serum urate or uric acid, although known to be a major antioxidant, primarily in the human plasma (extracellularly),⁷ is also being held to have paradoxical pro-oxidative effects intracellularly which could explain its role in the pathogenesis of CVDs⁶⁻⁷ most frequently at hyperuricaemic levels⁵⁻¹¹ (more than 6–7 mg/dl or 0.36–0.42 mmol/l).⁵ Fenech et al.⁵ reported individual studies providing evidence on the possible pathophysiologic mechanisms behind the development of CVDs as influenced by serum urate. Some of which included the production of pro-oxidative, pro-inflammatory, and vasoconstrictive substances favouring microvascular changes, and the presence of considerable amount of urate in human atherosclerotic plaque may apparently enhance thrombus formation and platelet adhesiveness.⁵ Meanwhile, the association of hyperuricaemia and gout with insulin resistance syndrome was also reported by George et al.,²² and this serves as another supporting theory on the possible pathophysiologic mechanism behind the occurrence of diabetes mellitus in the presence of gout.

Just like stroke, gout has become a global concern related to its increasing prevalence worldwide with New Zealand having one of the highest prevalences.²³ The under-treatment of gout with allopurinol in New Zealand had been reported,²⁴ and this could also be contributing to increasing prevalence of gout in the country apart from the combination of genetic and environmental factors such as lifestyle.^{23,24}

Although there have been international reports on the association of gout with diabetes mellitus and hypertension,¹⁶⁻¹⁹ the data providing supporting evidence to this area of research are limited in New Zealand. One study was conducted demonstrating the co-existence of gout with diabetes mellitus, and it specifically demonstrated only the high crude co-prevalence and age-standardised prevalence of diabetes and CVDs in those with gout.²⁰ At the time of completion of this study, there was no study yet done in New Zealand directly associating gout with the risk of diabetes mellitus and hypertension. It was observed, however, that there were a number of stroke patients, with co-existing gout in a public hospital in Auckland – a lot of whom with hypertension and diabetes mellitus at the same time. It was therefore the aim of this study to determine the association of gout with the risk of hypertension and diabetes mellitus among stroke survivors in Auckland, New Zealand.

Methods

Study design and setting

This was a cross-sectional study conducted among patients from South and East Auckland, New Zealand who had acute stroke between the years 2010

and 2014 and were admitted in Middlemore Hospital, Counties Manukau Health (CMH), Auckland, New Zealand.

Participants

The sample population were adult stroke survivors in New Zealand aged ≥ 18 years old, discharged from acute stroke and rehabilitation wards between the years 2010 and 2014 and were subsequently admitted for home-based rehabilitation to Community Stroke Rehabilitation (CSR) under the Assessment, Treatment, and Rehabilitation unit of same hospital in CMH.

Calculation for sample size was done using the OpenEpi²⁵ taking into consideration the power of the study (at least 90%), level of significance (alpha of 0.05), estimated population size (n = approximately 1000), and hypothesised % frequency of outcome of factor (20–30%). The calculated sample size for a study power of at least 90% was around 500.

Data collection

Demographic and clinical data, and diagnostic test results were collected and reviewed retrospectively by a single researcher from routinely collected health data from the patients' electronic hospital discharge summaries and official results of brain computed tomography (CT) scan and/or magnetic resonance imaging (MRI) (stored through health data software, Concerto, utilised in CMH).

Using the New Zealand alphanumeric code, National Health Index identifiers, all patients admitted to CSR in the years 2010–2014 were pulled out from the service's database and initial random sampling was conducted for selection of the first 500 patients for screening for eligibility using inclusion and exclusion criteria (Figure 1). Systematic random sampling was subsequently performed for continuous selection of patients for screening for eligibility until the final 500 eligible stroke survivors were completed for inclusion in the study. Inclusion criteria were as follows: patients with evidence of acute stroke (ischaemic or haemorrhagic) by brain CT scan and/or MRI during admission to hospital due to stroke. The primary sources of data for acute stroke, hypertension, diabetes mellitus, and gout diagnoses were from electronic hospital discharge summaries specifically from the discharge diagnoses and medical history sections, with the documentation of the administration of maintenance medications such as anti-hypertensives, hypoglycaemic agents, and allopurinol (with or without colchicine), serving as supporting evidence for the existence of the aforementioned conditions.

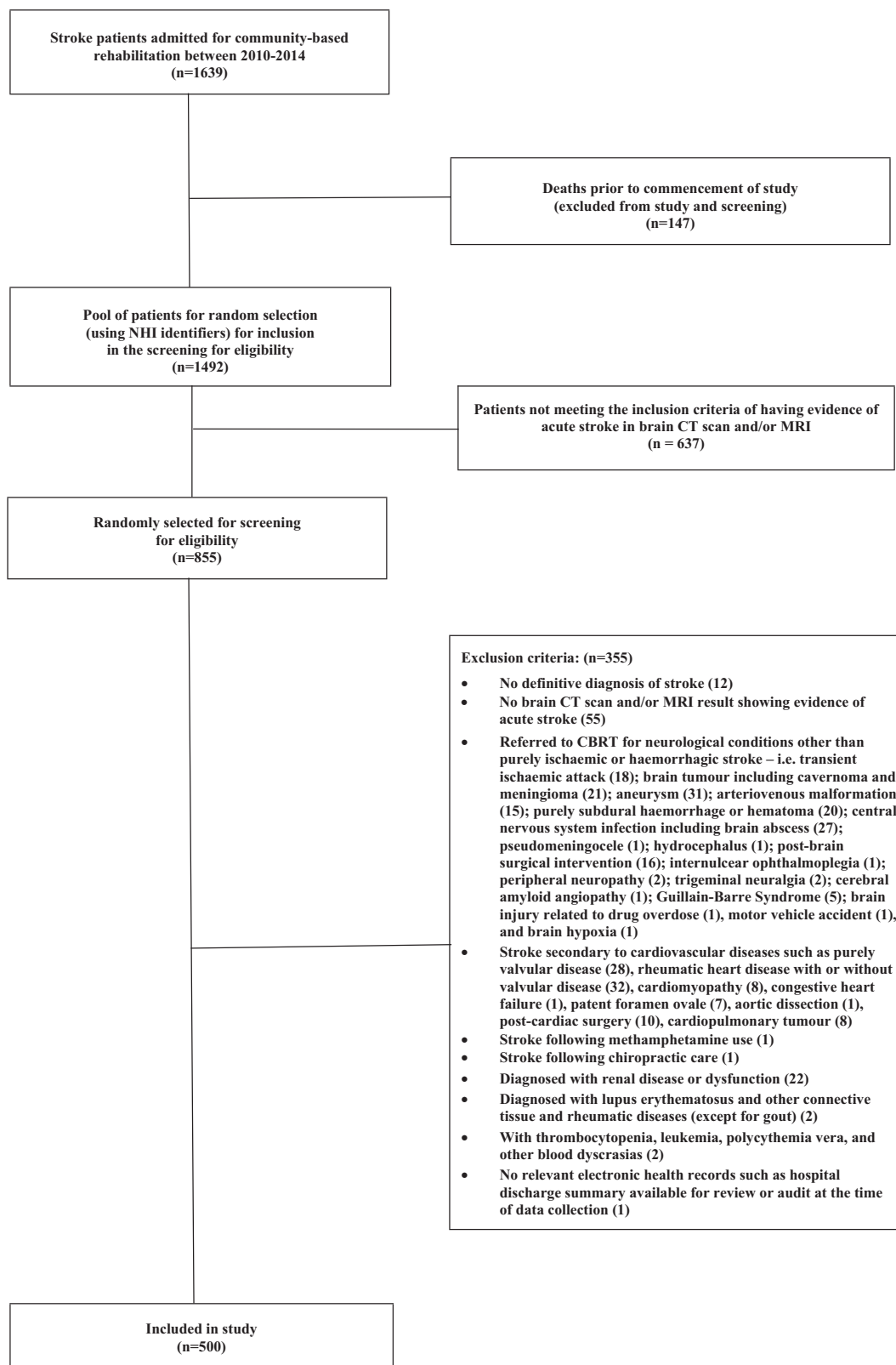


Figure 1. Flow chart of random selection of stroke survivors and screening for eligibility. CT: computed tomography; MRI: magnetic resonance imaging; NHI: national health index.

Stroke survivors were de-identified and their identifiers were replaced by Arabic numerals (1–500) during data collection and tabulation for analyses. Approval from CMH Research Office (Research Registration Number 2033) and University of Auckland Human Participants Ethics Committee (HPEC) (Reference 014887) was obtained for access and collection of patients' information from CMH electronic health records. No actual patient contact was made to collect any personal and clinical information.

Case definition

Stroke survivors were identified having a diagnosis of stroke, gout, hypertension, and diabetes mellitus based on the following (as applicable):

- Pre-existing condition – patients were known to have gout, hypertension, and diabetes mellitus based on their medical history with maintenance medications such as allopurinol (with or without colchicine), anti-hypertensives, and hypoglycaemic agents prior to admission to hospital due to acute stroke
- New diagnoses of the diseases during admission due to stroke between the years 2010 and 2014:

For acute stroke diagnoses – had evidence of acute ischaemic or haemorrhagic stroke on brain CT scan and/or MRI.

For diabetes mellitus diagnoses – established in discharge summaries, diagnoses based on the results of fasting glucose of >7 mmol/l, abnormal 2 h oral glucose tolerance test, and symptoms of hyperglycaemia with a random blood glucose of >11.1 mmol/l.²⁶

For hypertension diagnoses – defined as having blood pressure of $\geq 130/80$ mmHg based on the recommended target blood pressure of $<130/80$ mmHg by the New Zealand Guidelines Group for those with CVD (such as stroke) and following the guidelines in measuring the blood pressure which involves at least two measurements on two separate occasions.²⁷

- Documented prescription and administration of allopurinol (with or without colchicine and other analgesics or anti-inflammatory drugs), anti-hypertensive drugs, and hypoglycaemic agents provided supporting evidence on the diagnoses of gout, hypertension, and diabetes mellitus, respectively.

Age was categorised into three groups (spaced equally): 18–41, 42–65, and more than 65 years old. However, age as a continuous variable was the covariate used in the logistic regression modelling in this study as it contributed more significantly in improving the models beyond just statistical significance than age as a

categorical variable. Although evidence from studies has shown variations in gout prevalence rates by gender,^{23,28} gender-stratification for predicting the occurrence of hypertension and diabetes mellitus in the presence of gout history was not possible to be performed in this study, for doing so would violate the assumptions of logistic regression modelling.

Ethnicity was categorised into four groups – Māori, Pacific Islander, European, and 'other ethnic groups' – based on Statistics New Zealand's ethnic groups categorisation.²⁹ Ethnicity stratification was conducted in this study for predicting the occurrence of hypertension and diabetes mellitus in the presence of history of gout.

Statistical analysis

Statistical Package for Social Sciences software, version 23³⁰ was utilised for data analyses. Data were presented as frequencies, reported as n (%) for categorical variables, and as median and inter-quartile range for continuous variable, such as age. Comparisons between groups with and without gout history or diagnosis and among ethnicities were conducted using multivariate logistic regression modelling. Multivariate logistic regression models were also utilised for determining the odds ratios (ORs), 95% confidence intervals (CIs), and p-values for comparisons of the risks of gout, hypertension, and diabetes mellitus; and for adjustment for confounding variables – age, gender, and ethnicity (adjustment was for first-order effects only, and not for interactions). Chi-square test was also utilised to predetermine the presence of statistical relationship between exposures (ethnicity and gout history) and disease outcomes (hypertension and diabetes mellitus).

Results

There were 1639 identified patients admitted to CSR for home-based rehabilitation between the years 2010 and 2014, and 1492 (91%) of this comprised the pool of patients for inclusion in the random selection for screening for eligibility (less than the 147 deaths prior to start of data collection) (Figure 1). In total, 855 stroke survivors underwent systematic random selection for screening for eligibility after excluding 637 who did not meet the inclusion criteria of having evidence of acute stroke (ischaemic or haemorrhagic) by brain CT scan and/or MRI. Final 500 were included in the study after the exclusion of 355 utilising the study's exclusion criteria (Figure 1).

Details of the demographic and clinical characteristics of the stroke survivors included in this study are presented in Tables 1 and 2. Of the eligible 500 stroke survivors included in the study, 291 (58%) were male stroke survivors (Table 1), and 220 (44%) belonged to

Table 1. Characteristics of the sample population of stroke survivors from South and East Auckland, New Zealand between the years 2010 and 2014.

	n (%)
Gender	
Male	291 (58.2)
Female	209 (41.8)
Age (years)	
Median	67
Interquartile range (IQR)	57–76
Age range	32–96
Age group	
18–41	12 (2.4)
42–65	220 (44.0)
>65	268 (53.6)
Ethnicity	
Māori	82 (16.4)
Pacific Islander	139 (27.8)
European	220 (44.0)
Other ethnic groups	59 (11.8)
Stroke type	
Ischaemic	432 (86.4)
Haemorrhagic	68 (13.6)
Gout	
With gout history	84 (16.8)
Without gout history	416 (83.2)
Selected stroke risk factors	
Hypertension	423 (84.6)
Diabetes mellitus	159 (31.8)
Impaired glucose tolerance	86 (17.2)
Total	500 (100)

the European ethnic group, 139 (28%) Pacific Islander, 82 (16%) to Māori, and 59 (12%) to ‘other ethnic groups’ comprising of Indians, Chinese, and other Asians (Tables 1 and 2). Ages of the sample population ranged from 32 to 96 years (Table 1). Majority of stroke survivors had ischaemic stroke – 432 (86%) – observed in 94% of Māori, 90% of European, 81% of Pacific Islander, and 75% of ‘other ethnic groups’ (Tables 1 and 2). Sixty-eight (14%) who had haemorrhagic stroke was observed in nearly 5% of Māori stroke survivors, 11% of Europeans, 19% of Pacific Islanders, and 22% of ‘other ethnic groups’ (Table 2).

By stroke risk factors, 423 (85%) had hypertension (Tables 1 and 2). High proportion of hypertension was noted in each of the ethnic groups – 90% of Pacific Islander stroke survivors, 85% of ‘other ethnic groups’, 83% of Europeans, and 80% of Māori. Highest proportion of diabetes mellitus was noted among Pacific Islander stroke survivors at 54%, followed by Māori and ‘other ethnic groups’ at 29%, and then by European at 20%. By gout history, 84 (17%) had gout (Tables 1 and 2).

The association between gout and stroke risk factors, hypertension and diabetes mellitus

Among stroke survivors with gout history, 93% (78) had hypertension and 46% (39) had diabetes mellitus (Table 3). Higher gout crude prevalence rate (24%) was noted among stroke survivors with diabetes mellitus, while the crude rate among those with hypertension was 18% (Table 4).

A multivariate logistic regression was conducted for predicting the occurrence of hypertension and diabetes mellitus in those with history of gout (Table 5). There was a statistically significant association between gout and hypertension at $p < 0.05$. Stroke survivors with gout history had 3.3 times higher odds (95% CI 1.32–8.03; $p = 0.011$; adjusted for age, gender, and ethnicity) and 2.7 times higher odds (95% CI 1.12–6.38; $p = 0.026$; unadjusted for age, gender, and ethnicity) of having hypertension, compared to stroke survivors without gout history. Māori stroke survivors with gout history had 10 times higher odds (95% CI 1.26–83.87; $p = 0.030$; adjusted for age and gender) of having hypertension, compared to Māori stroke survivors without gout history. There was no statistically significant association between gout and hypertension among Pacific Islander stroke survivors at $p < 0.05$ (95% CI 0.65–43.49 and 0.54–34.11; adjusted and unadjusted for age and gender, respectively) and among European stroke survivors at $p < 0.05$ level (95% CI 0.40–4.05 and 0.35–3.38; adjusted and unadjusted for age and gender, respectively).

There was also a statistically significant association between gout and diabetes mellitus at $p < 0.05$. Stroke survivors with gout history had 1.9 times higher odds (95% CI 1.12–3.36; $p = 0.018$; adjusted for age, gender, and ethnicity) and 2.1 higher odds (95% CI 1.27–3.52; $p = 0.004$; unadjusted for age, gender, and ethnicity) of having diabetes mellitus, compared to stroke survivors without gout history. Māori stroke survivors with gout history had five times higher odds (95% CI 1.90–18.93; $p = 0.002$; adjusted for age and gender) of having diabetes mellitus, compared to Māori stroke survivors without gout history. There was no statistically significant relationship between gout and diabetes mellitus among Pacific Islander stroke survivors at $p < 0.05$ (95% CI 0.35–1.87 and 0.35–1.84; adjusted and unadjusted for age and gender, respectively) and among European stroke survivors at $p < 0.05$ (95% CI 0.82–6.65 and 0.73–5.39; adjusted and unadjusted for age and gender, respectively).

Discussion

Gout and hypertension

This study demonstrated an increased risk of hypertension occurrence in the presence of a history of gout

Table 2. Characteristics of the sample population of stroke survivors, per ethnicity distribution.

	Māori n = 82	Pacific Islander n = 139	European n = 220	Other ethnic groups n = 59	Total n
Gender					
Male	38 (13.1)	82 (28.1)	134 (46.0)	37 (12.7)	291
Female	44 (21.1)	57 (27.3)	86 (41.1)	22 (10.5)	209
Age (years)					
Median	60.50	61	74	63	
Interquartile range (IQR)	50–65.25	52–71	66–82	56–73	
Age range	32–80	33–93	40–96	40–86	
Age group					
18–41	4 (33.3)	6 (50.0)	1 (8.3)	1 (8.3)	12
42–65	58 (26.4)	80 (36.4)	47 (21.4)	35 (15.9)	220
>65	20 (7.5)	53 (19.8)	172 (64.2)	23 (8.6)	268
Stroke type					
Ischaemic	77 (17.8)	113 (26.2)	198 (45.8)	44 (10.2)	432
Haemorrhagic	4 (5.8)	26 (38.2)	25 (36.8)	13 (19.1)	68
Selected stroke risk factors					
Hypertension	66 (15.6)	125 (29.6)	182 (43.0)	50 (11.8)	423
Diabetes mellitus	24 (15.1)	75 (47.2)	43 (27.0)	17 (10.7)	159
Impaired glucose tolerance	15 (17.4)	18 (20.9)	43 (50.0)	10 (11.6)	86
Gout					
With gout history	28 (33.3)	32 (38.1)	24 (28.6)	0 (0.0)	84
Without gout history	54 (13.0)	107 (25.7)	196 (47.1)	59 (13.7)	416

Table 3. Characteristics of sample population of stroke survivors by stroke risk factors, hypertension and diabetes mellitus according to gout history.

	Hypertension n (%)	Diabetes mellitus n (%)
With gout history (n=84)	78 (93)	39 (46)
Without gout history (n=416)	345 (83)	120 (29)

Table 4. Crude prevalence of gout in stroke survivors with hypertension and diabetes mellitus.

	With gout history n (%)	Without gout history n (%)
Hypertension (n=423)	39 (24.5)	345 (81.6)
Diabetes mellitus (n=159)	78 (18.4)	120 (75.5)

among stroke survivors, and that those with gout history were three times more likely to have hypertension compared to those without history of gout, adjusted for age, gender, and ethnicity. Although still limited, there have been previous reports on the association of gout with the risk of hypertension. In a population-based

study, it was shown that those with gout had an 18% higher risk of developing hypertension with hazard ratio (HR) 1.18 (95% CI 1.02–1.37), compared to those without gout.¹⁶ Differences in the study results could be attributed to the differences in the measure of effects used, type of study population and its characteristics (stroke patients in our study), sample size, and study design; and the effects of potential confounders such as comorbidities and lifestyle factors, which were not adjusted for in our study.

As an additional contribution of our study in this area of the association of gout with increased risk of hypertension, difference in the odds of developing hypertension by ethnicity was also demonstrated. It was shown that Māori stroke survivors with gout history were 10 times more likely to have hypertension compared to Māori stroke survivors without gout history, adjusted for age and gender. The odds were notably high, but the precision was relatively low reflected in the wide CI (1.26–83.87), and this could be attributed to the small sample size and possible effects of potential confounders. This significant association between gout and the risk of hypertension, however, was not demonstrated among Pacific Islander and European stroke survivors in our study, indicating that Māori ethnicity was the major contributor in the magnitude of the association between gout and the increased risk of hypertension.

Table 5. Multivariate logistic regression predicting the occurrence of hypertension and diabetes mellitus in the presence of history of gout.

	Adjusted for covariates ^a				Unadjusted for covariates ^b			
	OR	95 % CI for OR		p-value	OR	95 % CI for OR		p-value
		Lower	Upper			Lower	Upper	
All stroke survivors								
Hypertension	3.25	1.32	8.03	0.011	2.68	1.12	6.38	0.026
Diabetes mellitus	1.94	1.12	3.36	0.018	2.11	1.27	3.52	0.004
Māori								
Hypertension	10.27	1.26	83.87	0.030	9.45	1.17	76.15	0.035
Diabetes mellitus	5.10	1.90	18.93	0.002	5.29	1.77	15.81	0.003
Pacific Islander								
Hypertension	5.31	0.65	43.49	0.120	4.29	0.54	34.11	0.169
Diabetes mellitus	0.80	0.35	1.87	0.611	0.80	0.35	1.84	0.603
European								
Hypertension	1.26	0.40	4.05	0.693	1.09	0.35	3.38	0.885
Diabetes mellitus	2.33	0.82	6.65	0.113	1.98	0.73	5.39	0.183

^{a,b}Age, gender, and ethnicity for all stroke survivors; age and gender for individual ethnic group.

Gout and diabetes mellitus

The crude prevalence rate of diabetes mellitus among stroke survivors with gout history in our study was much higher (46%), compared to the rates in a study done by Winnard et al.²⁰ in the adult Aotearoa New Zealand Health Tracker (ANZHT) population – 26% crude co-prevalence and 17% age-standardised prevalence of diabetes in those with gout. In a study done by Liu et al.,²¹ high prevalence of gout (>20%) among men with type 2 diabetes was revealed. Meanwhile, in a population-based cohort study done by Rho et al.,¹⁷ the incidence rates of diabetes in men and women with gout were 9.5 and 10.1 cases per 1000 person years, respectively. Our study further demonstrated that stroke survivors with gout history were nearly twice as likely (1.94) to have diabetes mellitus compared to those without gout history, adjusted for age, gender, and ethnicity. In contrast to hypertension as an outcome, when no adjustment for age, gender, and ethnicity was made, the OR for having diabetes mellitus slightly increased to 2.11, indicating the effect of these covariates in the models and resultant estimate. While in the study done by Winnard et al.²⁰ in the adult ANZHT population, a rate ratio of 3.5 for diabetes in those with gout, compared to those without gout, was revealed. In a prospective study by Choi et al.¹⁸ among men, the multivariate relative risk for incident type 2 diabetes was 1.34 (95% CI 1.09–1.64) in those with gout, compared to those without gout. In the study done by Rho et al.,¹⁷ it was shown that the univariate HR for diabetes mellitus was 1.71 (95% CI 1.51–1.93) in women with gout and 1.22 (95% CI 1.13–1.31) in men with gout, while the multivariate HR for diabetes

mellitus was 1.48 (95% CI 1.29–1.68) in women with gout and 1.15 (95% CI 1.06–1.24) in men with gout. Stratification by gender was not possible in our study having gout history as the exposure and the stroke risk factors, hypertension and diabetes mellitus, as outcomes, for doing so would violate the assumptions of logistic regression modelling.

By ethnicity, Māori stroke survivors with gout history were five times more likely to develop diabetes mellitus compared to Māori stroke survivors without gout history, adjusted for age and gender. Unlike in hypertension, the OR CI for the risk of diabetes mellitus was narrower (1.90–18.93) indicating a relatively more precise estimate. In the study done by Winnard et al.²⁰ in the adult ANZHT population, the rate ratio for diabetes in those with gout, compared to those without gout, among Māori was 2.5. The significant association between gout and diabetes mellitus was not demonstrated among Pacific Islander and European stroke survivors with gout history in our study. However, in the study done by Winnard et al.²⁰ in the adult ANZHT population, the rate ratios for diabetes among European and Pacific Islander populations with gout, compared to those without gout, were 3.7 and 1.6, respectively.

Limitations

The generalisability of our study results may be limited primarily because our study was done restrictively among stroke patients. Our study results are also merely indicative for reasons related to the effect of the potential confounders (including comorbidities and lifestyle factors) apart from the cross-sectional

nature of our study (exclusively done among stroke survivors in the years 2010–2014 and with cross-sectional studies known for cause-and-effect issues). Some strategies undertaken to minimise bias in our study included adjustment for known potential confounders such as age, gender, and ethnicity using multivariate logistic regression modelling, randomisation, and restriction of patients included in our study.

Conclusion

The findings from our study suggest that gout may be associated independently with an increased risk of hypertension and diabetes mellitus in those who had a stroke. Māori who are the indigenous population of New Zealand could have markedly contributed to the association, particularly showing a greater risk of diabetes mellitus associated with a gout diagnosis compared to other populations. This finding highlights the importance of the need for further research with Māori stroke survivors and other indigenous populations. Future prospective large studies may also potentially provide an invaluable new insight into CV risk assessment with consideration of routine screening for serum urate levels both in primary and secondary care. Consideration should also be shown in regard to rehabilitation of stroke survivors with gout, management of gout, and CVDs such as stroke and its risk factors, including aggressive treatment of gout and its precursor, elevated serum urate, with allopurinol.

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Ethical approval

The study was approved by CMH Research Office (Research Registration Number 2033) and University of Auckland HPEC (Reference 014887). There was no actual patient contact made to collect any personal and clinical information.

Guarantor

JS is the guarantor for this study.

Contributorship

Conception and design: DEDSG; Data collection: DEDSG; Data analysis: DEDSG and JS; Manuscript drafting and revision: DEDSG and JS; Approved the final version of the manuscript: all authors.

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