Gout, diabetes and cardiovascular disease in the Aotearoa New Zealand adult population: co-prevalence and implications for clinical practice

Doone Winnard, Craig Wright, Gary Jackson, Peter Gow, Andrew Kerr, Andrew McLachlan, Brandon Orr-Walker, Nicola Dalbeth

Abstract

Aims To determine the co-prevalence of gout, diabetes and cardiovascular disease (CVD) in the entire Aotearoa New Zealand adult population to inform clinical practice.

Methods Algorithms based on hospital admissions, outpatient visits, drug dispensing, laboratory test data and mortality for the Aotearoa New Zealand Health Tracker (ANZHT) population aged ≥20 years (n = 3,036,093) were used to estimate the prevalence of those identified with gout, diabetes and CVD in 2009.

Results The crude prevalence in the adult ANZHT population of gout was 3.9%, of diabetes was 6.6%, and of CVD was 5.4%. For those identified with gout, 25.6% had diabetes and 22.7% had CVD. Both diabetes and CVD were more prevalent in those identified with gout, compared with those without gout (age-standardised rate (ASR) ratio 3.5 for diabetes and 2.7 for CVD, p for both <0.001).

Conclusions By applying algorithms based on hospital coding, community drug dispensing and laboratory test data sets, we have demonstrated a high co-prevalence of gout, diabetes and CVD in the adult population of Aotearoa New Zealand. Health service presentation with gout can be an important opportunity to assess risk and manage co-morbid disease. Prevention and management strategies are reinforcing for these metabolic conditions.

Gout is the most common inflammatory arthritis affecting men. This disease typically presents as recurrent self-limiting episodes of severe joint inflammation affecting the feet, described by patients as ‘like a fire in the joints’.1

Gout may result in significant work and social disability.1–3 Gout and hyperuricaemia have also been associated with insulin resistance and increased risk of hypertension, diabetes and cardiovascular disease (CVD). The exact role of serum urate in these conditions remains subject to investigation,4 but it has been suggested that the onset of gouty arthritis can identify a clinical population likely to have coincident metabolic risk5,6 and can therefore be an important opportunity for intervention to modify subsequent disease trajectories.7

Aotearoa New Zealand has one of the highest documented prevalences of gout worldwide, with very high rates in Māori and Pacific men.8–11 We have recently confirmed these findings in a study of the entire Aotearoa New Zealand population using the Aotearoa New Zealand Health Tracker (ANZHT) health dataset.12
The aim of this study was to investigate the co-prevalence of gout, diabetes and CVD in the Aotearoa New Zealand adult population to inform clinical practice.

Methods

Study population—The Aotearoa New Zealand Health Tracker (ANZHT) population was used to determine the prevalence of gout, diabetes and CVD in those aged ≥20 years, with stratification by age, gender, and ethnicity. The ANZHT population is based on ‘health service contact’, and attempts to align with Statistics New Zealand’s census-derived ‘usually resident’ definition (resident for more than 3 months).

All New Zealanders are assigned a unique alphanumeric code, their National Health Index (NHI) identifier, which is linked to most routinely collected national health databases. The NHI identifier can be encrypted and used to anonymously link the various databases. The denominator population for this study refers to people who were registered with a National Health Index identifier (NHI), and had any form of health services contact in Aotearoa New Zealand from 1 July 2008 to 30 June 2009. People without resident status were excluded if they did not receive services for a period greater than 3 months — to align with Statistics New Zealand’s definition of usually resident.

The health services contact includes:

1. Primary Health Organisation (PHO) Enrolment Register (General Practice consultation date or current PHO enrolment),
2. National Minimum Data Set (NMDS) Public Hospital Event (Admission or Discharge date),
3. Laboratory Testing Claims,
4. Community Pharmaceutical Dispensing,
5. General Medical Subsidy payments (GMS),
6. Client Claims Processing System payments (CCPS, Community aged and disability support events).

We identified a denominator ‘health services contact’ population of 3,036,093 people aged ≥20 years as at 30 June 2009. This was 98.6% of the Statistics New Zealand estimated usually resident population of people aged ≥20 (3,079,250) for the same period. Individuals included in the analysis also had to have been living in Aotearoa New Zealand, evidenced by some form of recorded health contact during July 2008 – June 2009 (some of whom died during the year).

Ethnicity data for this population were taken from the 2009 second quarter PHO enrolment database and the NHI extract for the 2009 second Quarter. Ethnicity was prioritised from multiple ethnic codes to Māori (ethnic code 21), Pacific people (30–37), Asian (41–44), European (10–12) and ‘Other’ New Zealanders.

Case definition—People were identified as having gout, diabetes and/or CVD in the ANZHT population based on, where appropriate:

- Discharge diagnoses (primary or secondary) for public hospital admissions from 1988-2009,
- Related procedures codes for public hospital procedures from 1988–2009,
- Attendance at hospital outpatients from July 2006 to December 2009 identified by purchase units within the National Non-admitted Patient Collection (NNPAC),
- Dispensing of medication deemed relatively specific for the disease group in question from a community pharmacy between July 2001 and December 2009,
- Request of laboratory tests deemed relatively specific for the disease group in question, during a two year period between 1 July 1996 and 31 December 2009.12-15

Description of the variables for each condition is outlined in Table 1, with detail of the International Statistical Classification of Diseases and Related Health Problems (ICD) codes used in Appendix 1. A stacked bar graph shows the relative contribution of each category of variables to the diagnosis of each condition in Appendix 2. As outlined in our previous publication validating the gout diagnosis algorithm, for individuals who had been diagnosed with leukaemia or lymphoma (ICD-10-AM C80-C96) in the previous 24 months, dispensing of allopurinol was excluded as an indicator of gout.12
Table 1. Algorithm variables used to identify people with gout, diabetes and CVD\textsuperscript{12-15}

<table>
<thead>
<tr>
<th>Variables</th>
<th>Gout</th>
<th>Diabetes</th>
<th>CVD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital diagnosis codes</td>
<td>ICD codes for gout.</td>
<td>ICD codes for diabetes including pre-existing diabetes in pregnancy but excluding diabetes arising from pregnancy.</td>
<td>ICD codes for coronary heart disease, ischaemic stroke, atherosclerotic cerebrovascular disease, and peripheral vascular disease.</td>
</tr>
<tr>
<td>Hospital procedure codes</td>
<td>–</td>
<td>–</td>
<td>ICD procedures codes for coronary artery bypass graft, coronary angioplasty or stenting, and peripheral vascular procedures.</td>
</tr>
<tr>
<td>Hospital outpatients</td>
<td>–</td>
<td>Two or more clinic visits for diabetes education and management; and/or retinal screening.</td>
<td>–</td>
</tr>
<tr>
<td>Medication dispensed</td>
<td>Allopurinol, Colchicine</td>
<td>Two or more scripts for all subsidised forms of insulin, oral hypoglycaemics and glucagon. Glucose test strips and insulin needles were not included. Metformin to women of 12 to 45 years of age was not included.</td>
<td>Two or more scripts for specific anti-anginals: glyceryl trinitrate, isosorbide dinitrate, isosorbide mononitrate, nicorandil, and perhexiline.</td>
</tr>
<tr>
<td>Laboratory request</td>
<td>–</td>
<td>Four or more requests for HbA1c and one or more requests for Albumin Creatinine Ratio.</td>
<td>–</td>
</tr>
</tbody>
</table>

Probenecid was not included in the gout algorithm as it is used infrequently for the treatment of gout, and is also used to increase antibiotic blood levels for the treatment of bacterial infections in primary care and thus could confound results.

An indicative mortality rate was calculated by counting records in the study population where the date of death (from the Births Deaths and Marriages death registration) for an individual on the NHI was between 1 July 2008 and 30 June 2009. This is termed indicative as the current study is a cross-sectional rather than cohort study, and the possibility of bias exists.

**Statistical methods** — The data handling, calculation of crude and age-standardised prevalence and calculation of rate ratios were all undertaken in SAS 9.1 using PROC SQL. The age variable was converted to five-year age categories (20-24, 25-29, to 85+) for all calculations. The prevalence of diabetes and CVD in those with gout was examined and compared with those not identified as having gout. Confidence intervals were not used as the study is enumerating the entire Aotearoa New Zealand population. The age-standardised mortality of those with and without gout was also compared. Prevalence estimates were calculated as age-standardised to the WHO World Standard Population (2001) to facilitate ethnic and international comparisons.

**Ethical considerations** — Institutional ethical approval was not required for this study, consistent with the Aotearoa New Zealand Ministry of Health guidelines for use of non-identifiable Ministry of Health data. The study involved no personal contact with the populations involved and only aggregated results are reported.
Results

Disease prevalence—In the adult ANZHT population the crude prevalence of gout was 3.9% (119,234/3,036,093), of diabetes 6.6% (202,003/3,036,093), and of CVD 5.4% (165,042/3,036,093), for 2008/09 (Table 2). The age-standardised prevalence of gout was significantly higher in Māori and Pacific peoples (7.7% and 8.6% respectively), than in those of European and Asian ethnicities (2.3% and 2.2% respectively).

Table 2. Ethnic and gender specific disease prevalence (crude & age-standardised rate ANZHT using the WHO standard population) – ≥20 years

<table>
<thead>
<tr>
<th>Variables</th>
<th>Number (count)</th>
<th>Crude ANZHT rate</th>
<th>Age-standardised rates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Gout</td>
<td>Diabetes</td>
<td>CVD</td>
</tr>
<tr>
<td>Māori</td>
<td>23,460</td>
<td>31,032</td>
<td>15,246</td>
</tr>
<tr>
<td>Pacific</td>
<td>14,055</td>
<td>24,743</td>
<td>5,857</td>
</tr>
<tr>
<td>European</td>
<td>77,013</td>
<td>125,384</td>
<td>137,972</td>
</tr>
<tr>
<td>Asian</td>
<td>4,706</td>
<td>20,844</td>
<td>5,967</td>
</tr>
<tr>
<td>All</td>
<td>119,234</td>
<td>202,003</td>
<td>165,042</td>
</tr>
<tr>
<td>Male</td>
<td>89,112</td>
<td>102,177</td>
<td>89,458</td>
</tr>
<tr>
<td>Female</td>
<td>30,122</td>
<td>99,826</td>
<td>75,584</td>
</tr>
</tbody>
</table>

Co-prevalent disease and mortality—For those identified with gout, 25.6% were also identified as having diabetes (30,579/119,234) and 22.7% also identified as having CVD (27,131/119,234); of those, 8.6% (10,276/119,234) had both diabetes and CVD (Table 3). Diabetes and/or CVD affected 40% of those with gout. For Māori, Pacific peoples and Asian populations one-third of those with gout also had diabetes.

Both diabetes and CVD were more prevalent in those identified with gout, compared with those without gout (age-standardised rate (ASR) ratio 3.5 for diabetes and 2.7 for CVD, p for both <0.001) (Table 3). The increased risk of co-prevalent disease was most marked for European populations and for females – that is, for the groups with the lowest gout prevalence.

Apparent mortality rates were also increased in those with gout (ASR ratio 2.4, p<0.005) compared to those without gout. The increased risk was greatest for those in Asian populations (ASR ratio 2.8, p<0.005) and for females (ASR ratio 3.5). A person with diabetes who also had gout had an age-standardised mortality rate ratio of 2.0 compared with a person with diabetes who did not have gout (p<0.001). Likewise a person with CVD and gout had an apparent age-standardised mortality rate ratio of 1.4 compared with a person with CVD who did not have gout (p<0.001).
Table 3. People with gout: ethnic and gender specific co-prevalence of diabetes, CVD, or both

<table>
<thead>
<tr>
<th>Variables</th>
<th>Prevalence in those with gout (crude) - coprevalence</th>
<th>Prevalence in those with gout (age-standardised)</th>
<th>Rate ratio compared to all without gout</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Diabetes</td>
<td>CVD</td>
<td>Both</td>
</tr>
<tr>
<td>Māori</td>
<td>32%</td>
<td>20%</td>
<td>10%</td>
</tr>
<tr>
<td>Pacific</td>
<td>35%</td>
<td>12%</td>
<td>7%</td>
</tr>
<tr>
<td>European</td>
<td>22%</td>
<td>26%</td>
<td>8%</td>
</tr>
<tr>
<td>Asian</td>
<td>31%</td>
<td>15%</td>
<td>8%</td>
</tr>
<tr>
<td>All</td>
<td>26%</td>
<td>23%</td>
<td>9%</td>
</tr>
<tr>
<td>Male</td>
<td>23%</td>
<td>21%</td>
<td>7%</td>
</tr>
<tr>
<td>Female</td>
<td>33%</td>
<td>29%</td>
<td>12%</td>
</tr>
</tbody>
</table>

Age-standardised rate (ASR) ANZHT using WHO standard population. p<0.001 for all rate ratios.

Discussion

Using national level health datasets, we have demonstrated a high co-prevalence of gout, diabetes and CVD in the adult population of Aotearoa New Zealand. One in three of the Māori, Pacific and Asian populations with gout were identified as having diabetes. Forty percent of the population with gout were identified as having diabetes and/or CVD, with nearly one in ten having all three conditions. While women have a much lower prevalence of gout, those with gout have higher co-prevalence of diabetes and/or CVD than males, at 50%.

In addition, while mortality rates are indicative because of the cross sectional nature of this study, those with gout appeared to have significantly higher age-standardised mortality rates; twice the mortality rate for males and more than three-fold for females with gout.

These data suggest that gout is an important marker of health risk and this has important implications for clinical practice.

The tendency for gout, diabetes and CVD to co-exist was described in Aoteaora New Zealand 50 years ago but this is the first study to examine the co-prevalence of gout, diabetes and CVD in an entire national population. The use of a unique identifier within the Aoteaora New Zealand health system, the National Health Index (NHI), enables the linking of national datasets as described in this paper.

Use of the NHI for enumerating both the numerator and denominator avoids numerator/denominator bias. This is particularly important in relation to assigned ethnicity because of historical issues related to undercounting of the Māori population to varying degrees in different Aotearoa New Zealand datasets. The use of administrative data also reduces the need for complex and expensive surveys and may have more predictable biases than unknown biases related to survey response rates. Internationally, algorithms based on diagnostic coding and pharmaceutical prescribing have been used to identify cases of gout in population based studies, although these studies based on health care databases have described more limited populations than our study using national level databases.
Record linkage using similar algorithms has been used to examine the prevalence of diabetes and CVD within Aotearoa New Zealand and internationally and the validity of the decision rules continues to be explored in the Aotearoa New Zealand setting.

This is the first national level study of the association of gout, diabetes and CVD in the Aotearoa New Zealand adult population. In a previous study of 100 consecutive patients with gout treated in secondary care in South Auckland, Colvine et al reported that 51% of patients were at very high risk of a CVD event (≥20% risk of CVD events in 5 years) and 33% had diabetes.

Cross-sectional studies have demonstrated an association between the diagnosis of gout, coronary heart disease and diabetes in primary care in the UK and the Netherlands, and between gout and diabetes in population studies in the US (MRFIT trial) and the Netherlands. Longitudinal studies have shown associations between gout and/or hyperuricaemia and cardiovascular events and mortality and recent meta-analyses have confirmed the independent association between hyperuricaemia and stroke and, to a lesser extent, hyperuricaemia and coronary heart disease.

While it is not currently advocated that serum urate testing be used as a prognostic tool in cardiovascular disease, or that asymptomatic hyperuricaemia be treated in the hope of independently reducing cardiovascular risk, the EULAR guidelines for the management of gout do recognise the importance of risk factor assessment for patients presenting with gout.

An Aotearoa New Zealand study of secondary care referred gout patients found that despite the high risk of CVD events, only 50–65% were receiving indicated treatment with aspirin, statin, beta-blocker, and/or angiotensin converting enzyme (ACE) inhibitor according to the NZ guidelines for the management of cardiovascular risk and diabetes, and recommended targets for CVD risk factors such as blood pressure and lipids were frequently not attained.

Internationally it has been suggested that almost every first attack of gout urges patients to seek medical help. However while clinical experience suggests that acute gout does drive people to seek relief through medical attendance, qualitative studies in NZ suggest that gout is so prevalent in Māori and Pacific families and whānau (extended family), and is associated with whakamā (shame), that patients may in fact use the medication of other family members, or seek over-the-counter treatment from a pharmacy.

Qualitative research also suggests that young men with gout, especially when it is not typical podagra, may initially believe they have a sports injury; presentation in this instance may be to a physiotherapist or a sports clinician. This highlights the importance of all frontline health professionals being aware not only of the appropriate treatment of gout but that gout can be a marker of metabolic disease and an opportunity for early intervention to prevent future ill-health.

An Aotearoa New Zealand study of the prevalence of metabolic syndrome from a cross-sectional survey undertaken in 2002-03 found that approximately 70% of those who had the syndrome did not yet have type 2 diabetes, highlighting the opportunity for intervention to prevent diabetes. While weight loss and regular exercise are
challenging to achieve, they are the foundation of management of the metabolic syndrome and for the prevention of diabetes. Similar interventions have been shown to improve hyperuricaemia and frequency of gout attacks.

A recent analysis of gout in patients with diabetes in Aotearoa New Zealand demonstrated a 5% relative increase in prevalence of gout with every kg/m² increase in BMI, highlighting the importance of weight management for the prevention and treatment of gout.

Care with pharmacotherapy is also important as gout is associated with the use of several medications – in particular diuretics and low dose aspirin, both of which are used in the treatment of cardiovascular disease.

The use of aspirin remains a priority for secondary prevention of CVD in those with gout, but monitoring of serum uric acid is appropriate to ensure gout control is not compromised. Both losartan and calcium channel blockers have urate-lowering effects which may be useful in the treatment of hypertension in those with gout and atorvastatin is also urate-lowering and may be useful in patients with gout who need a statin.

The potential limitations of this study are acknowledged. Sensitivity of the algorithms used in this study may be limited by the fact that only those who present to the health system and are diagnosed or fill their prescriptions in a particular ‘time window’ are counted. In the case of gout, people with gout may self-manage their gout flares by purchasing over-the-counter non-steroidal anti-inflammatory drugs directly from a pharmacy, borrowing medication or using alternative therapies which are not captured in health systems data.

In Aotearoa New Zealand, the use of traditional Māori remedies (rongoā) is important among the Māori community (personal communication, Leanne Te Karu, Māori Pharmacists Association). Thus, our data may be an underestimate of the true prevalence of gout in the community. We have recently published an analysis applying capture-recapture methods to national administrative data sets which suggests that potentially 20% of people in Aotearoa New Zealand with gout are not being identified and treated specifically for their gout. In addition, this is a cross sectional study and the diagnosis of all three conditions will be subject to survivor bias, again potentially underestimating prevalence.

In relation to specificity, allopurinol use for asymptomatic hyperuricaemia is very uncommon in Aotearoa New Zealand and the exclusion of patients with haematological malignancies largely mitigates other use. Colchicine use for other conditions is very infrequent.

In the case of diabetes and CVD, those treated by lifestyle modification without medications and who do not receive care in the hospital setting may be missed. In the case of diabetes, excluding dispensing of metformin for women 12–45 years on the grounds that such prescriptions could be related to treatment of polycystic ovarian syndrome is likely to decrease the sensitivity of the diabetes algorithm.

The HbA1c blood test is being used increasingly for screening and diagnosis of diabetes and the algorithm for diabetes has recently been adjusted to four HbA1c
tests within two years rather than three to improve specificity by reducing the chance that use for screening results in misdiagnosis of disease.

Mild CVD that is managed within primary care without the use of specific antianginal medications may not be captured by the algorithm. The limitation of medications to specific antianginals for the identification of CVD improves specificity while likely reducing sensitivity.

In summary, by applying algorithms based on diagnostic and procedural coding, drug dispensing claims and laboratory requests to national health data sets we have demonstrated a high co-prevalence of gout, diabetes and CVD in the adult population of Aotearoa New Zealand.

A diagnosis of gout warrants clinical attention to risk factor management and optimal disease management in the context of these co-prevalent conditions. Public health attention to the drivers of metabolic disease is also important to help improve health outcomes and reduce the related health system costs.

Competing interests: Nil.

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References:


## Appendix 1. Algorithm Variables, Hospitalisation and Procedure Codes

<table>
<thead>
<tr>
<th>Disease</th>
<th>Hospitalisation Codes</th>
<th>Procedure Codes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gout</td>
<td>ICD-9: 274, ICD-10 M10 (And excluding ICD-10-AM C80-C96, leukaemia and lymphoma)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ICD-10: E10-E14 (diabetes codes), O24.0 to O24.3 (referring to pre-existing diabetes in pregnancy), ICD-9: 250 (diabetes codes); but not ICD 10:O24.4 (diabetes arising from pregnancy)</td>
<td></td>
</tr>
<tr>
<td>CVD</td>
<td>ICD-10: 120x to 125x; ICD-9: 410x to 414x (Coronary artery disease)</td>
<td>ICD-10: 3850500 (Coronary endarterectomy)</td>
</tr>
<tr>
<td></td>
<td>ICD-10: E1053, E1153, E1453 (Diabetic ischaemic cardiomyopathy)</td>
<td>ICD-10: 3530400-3530501, 3531000-3531005; ICD-9: 360x (Coronary angioplasty or stent, Percutaneous coronary intervention)</td>
</tr>
<tr>
<td></td>
<td>ICD-10: I63x, I64x, I66x, I678, I693, I694, I698; ICD-9: 434x, 436x, 4371, 438x (Ischaemic stroke)</td>
<td>ICD-10: 3849700-3850304, 9020100-9020103; ICD-9: 361x (Coronary Artery Bypass Graft)</td>
</tr>
<tr>
<td></td>
<td>ICD-10: G45x (except G453), G46x; ICD-9:435x (Transient Ischaemic Attack)</td>
<td>ICD-10: 3863700, 3845619, 3865308 (Re-operation &amp; other procedures on coronary arteries)</td>
</tr>
<tr>
<td></td>
<td>ICD-10: I670, I671; ICD-9: 4373 (Atherosclerotic cerebrovascular disease: Dissection cerebral arteries, non-ruptured cerebral aneurysm)</td>
<td>ICD-9: 362x (Heart revascularisation by arterial implant)</td>
</tr>
<tr>
<td></td>
<td>ICD-10: I670x, I671x; ICD-9: 4373 (Atherosclerotic peripheral vascular disease: ICD-10: 165x; ICD-9: 433x (Oclusion and stenosis of precerebral arteries)</td>
<td>ICD-10: 3857200, [684][685] 3855000-3857101, [693] 3870600, 3870601, 3871200 (Operative management of acute rupture or dissection of thoracic aorta 3857200 but other codes (repair of ascending [684][685] and descending[686], or replacement of aneurysm with graft [715] will be coded first) other aortic repair procedures [693])</td>
</tr>
<tr>
<td></td>
<td>ICD-9: 4372 (Atherosclerotic peripheral vascular disease: Dissection cerebral arteries, non-ruptured cerebral aneurysm)</td>
<td>ICD-10: 3270000-3276318 (Arterial bypass graft [711][712][713])</td>
</tr>
<tr>
<td></td>
<td>ICD-10: 165x; ICD-9: 433x (Oclusion and stenosis of precerebral arteries)</td>
<td>ICD-10: 3270000-3354200, 3335400, 9021100-9021210, 9022900</td>
</tr>
<tr>
<td></td>
<td>ICD-9: 171x; ICD-9: 441x, 443.2 (Aortic aneurysm and dissection, other arterial dissection)</td>
<td>ICD-10: 3922 (Peripheral arterial shunts/bypasses: Peripheral arterial bypass, endarterectomy, repair aneurysm, peripheral arterial bypass graft, aorto-subclavian-carotid bypass)</td>
</tr>
<tr>
<td></td>
<td>ICD-10: 172x; ICD-9: 442x (other aneurysm)</td>
<td>ICD-9: 3924 (Aorto-renal bypass, angioplasty/stent peripheral)</td>
</tr>
<tr>
<td></td>
<td>ICD-10: 174x; ICD-9: 444x (Arterial embolism and thrombosis)</td>
<td>ICD-10: 3530000-3530305, 3530600-3530905</td>
</tr>
<tr>
<td></td>
<td>ICD-10: I739, I7021, E1051, E1052, E1151, E1152, E1451, E1452 (2nd and 3rd edition);</td>
<td>ICD-9: 3925 (Aorto-iliac femoral bypass)</td>
</tr>
<tr>
<td></td>
<td>ICD-10: 4439, 44021, 44022, 44023, 44024, 25072, 25073 (Intermittent claudication, gangrene, or diabetic peripheral angiopathy with or without gangrene)</td>
<td>ICD-9: 3926 (Other intra-abdominal vascular shunt or bypass)</td>
</tr>
<tr>
<td></td>
<td>ICD-10: E1050, E1059, E1150, E1159, E1450, E1459; ICD-9: 25070, 25071 (Diabetic circulatory complication)</td>
<td>ICD-9: 3928 (Other (peripheral) vascular shunt or bypass)</td>
</tr>
</tbody>
</table>

CVD=Cardiovascular disease; ICD9-CM used up to 2000 in New Zealand; ICD10-AM thereafter. ICD10-AM procedure codes given by [block] or individual code.
Appendix 2. Relative contribution of each category of variables to diagnosis of the three conditions, gout, diabetes, and CVD

Note: Diagnosis and procedure codes from hospitalisation for CVD are grouped into one category, and hospitalisation and outpatient visits for Diabetes are grouped into one category to improve readability of the graph.