Socio-economic status influences chronic kidney disease prevalence in primary care: a community-based cross-sectional analysis

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ABSTRACT

Background. Primary care chronic kidney disease (CKD) registers report widely varying prevalence within the UK. We examined the effects of laboratory ascertainment and adjusting for practice-level variables on the variation in CKD prevalence. We carried out an Ayrshire-wide laboratory database analysis of primary care practices (PCPs).

Methods. We analysed 54 PCPs with 313 639 registered patients aged ≥18. All patients with a low estimated glomerular filtration rate (<60 mL/min/1.73 m2) had their serum creatinine values extracted from 1st January 2009 to 31st March 2012. Individuals with CKD stage 3–5 were identified with an algorithm that confirmed chronicity. These data were linked to PCP attributes from Information Services Division, Scotland. Using laboratory-ascertained CKD prevalence, we examined whether adjusting for practice-level factors [socio-economic status (SES), rurality and patients to general practitioner ratio (PGR)] and patient-level factors (age, gender) explained some of the observed variation among PCPs. Individual and combined hierarchical multilinear regression models were used.

Results. Eighteen thousand two hundred and eighty-five (5.8%) had CKD stage 3–5 on 31 March 2011. SES, rurality and PGR predicted 39% (F(3,50) = 12.37, P < 0.001) of the variation in prevalence with SES exerting the most influence (25%). With the stepwise addition of explanatory variables, variation between practices fell from 3.9-fold using PCP register prevalence to laboratory ascertained (3.1-fold variation), with age and gender adjustment (further fall to 2.1-fold), and lastly to 1.8-fold variation with adjustment for SES. Funnel plots using these adjustments reduced the number of outliers outside of 3 SD from 15 to 7 to 6, and outliers between 2 and 3 SD by 16 to 13 to 5.

Conclusions. Laboratory ascertainment is practicable, reduces variation and facilitates benchmarking. PCP attributes other than age and gender impact on prevalence. Over a third of variation in CKD prevalence among PCPs can be explained by rurality, PGR and especially SES even after age and gender stratification.

Keywords: chronic kidney disease, laboratory ascertainment, rurality, Scottish index of multiple deprivation, variation

INTRODUCTION

Chronic kidney disease (CKD) is common [1–5] and a major public health concern as it is associated with increased cardiovascular risk and, in subgroups, an increased risk of progressive renal decline [6–9]. CKD stages 3–5 (CKD 3–5) is a laboratory diagnosis based on estimated glomerular filtration rates (eGFRs) of <60 mL/min/1.73 m2. UK laboratories have reported eGFR routinely along with serum creatinine measurement in adults since 2006 [10]. However, to fulfil the chronicity criterion, the reduced eGFR must be present for ≥90 days, an important aspect which is often neglected in epidemiological studies.

CKD has been a domain in the UK General Practice Quality and Outcomes Framework (QOF) since 2006 [11]. Primary care practices (PCPs) receive performance-related payment to maintain a register of adult patients with CKD 3–5. The prevalence of CKD 3–5 registered by PCPs rose from 1.8% of the total Scottish population in 2006–07 (the first year CKD was included in the QOF) to 3.3% in 2010–11, presumed due to increasing ascertainment. Prevalence has now reached a plateau, most recently at 3.2% in 2012–13 [12].

This national prevalence rate masks substantial variation. In 2012–13, prevalence ranged from 2.5% in the NHS Lothian health board to 4.9% in NHS Ayrshire & Arran (A&A) [12]. Within health boards, there is even larger variation between PCPs. For example, in A&A, prevalence in individual PCPs
varied from 1.7 to 10.5%. We hypothesized that these large variations were due to differences in population characteristics. Therefore, the aim of this study was to establish the prevalence of CKD 3–5 using laboratory data (including the chronicity criterion) and to identify population-level factors that explain variation between PCPs.

METHODS

Participants and setting

A&A is a health board in the West of Scotland and is responsible for commissioning and providing healthcare for its geographically defined population of 373,712 residents (2011 census data) [13]. The area is a mix of rural, urban and island populations. In 2010–11, there were 57 PCPs led by two or more general practitioners (GPs), falling to 55 PCPs in 2011–12 due to amalgamation. The PCPs are independent contractors to the board and are responsible for the primary care needs of their registered patients. Almost the entire population is registered with a PCP. Each practice serves a median catchment population of 6533 (IQR 4079–9097.5; range: 1280–14,804) [14]. One PCP situated in the north of A&A uses laboratories in another health board and was therefore excluded from statistical analysis. For the remaining 54 PCPs, all samples for serum creatinine estimation are sent to our main or satellite laboratory.

Laboratory data

Our laboratories used the Jaffe assay method, performed using Roche Modular P Units and calibrated to the UK National External Quality Assessment Service adjustment for isotope dilution mass spectrometry (IDMS) traceability [15]. eGFR was calculated using the IDMS-traceable 4-variable Modification of Diet in Renal Disease equation [16], which is the method stipulated for QOF reporting. The laboratory only reports eGFR in adults, aged ≥18 years, if <60 mL/min/1.73 m². Ethnicity is not routinely recorded; however, the population is predominantly white (98.84% white, 0.37% Indo-Asian, 0.24% Chinese, 0.23% mixed ethnicity, 0.12% African or Caribbean origin) [11, 13].

We extracted all serum creatinine results recorded within the A&A laboratory database from 1 January 2009 to 31 March 2012 (39 months). All patients with one or more eGFR results <60 mL/min/1.73 m² during the period were included. We excluded those who were not residents of A&A, did not have a Community Health Index number (CHI: the NHS Scotland unique identifier) or did not have two or more serum creatinine samples >90 days apart at any point during the index period. The CHI was used to identify individuals’ PCP. CHI is in use for 97% of the population [17] and in our laboratory over 98% of all biochemistry requests utilize CHI. Data linkage was carried out to attribute all deaths within the survey period to the appropriate PCP using data provided by the Information Services Division of NHS Scotland. Patients who died before the census date for each year (April 1st) were excluded from the annual prevalence figures.

Assessment of chronicity of kidney disease was then performed. If a patient’s eGFR was <60 mL/min/1.73 m² on ≥2 occasions, they were considered to have CKD 3–5. If eGFR was ≥60 mL/min/1.73 m² on all measurements during the study period, they were not considered to have CKD 3–5. However, if a patient had eGFR above and below 60 mL/min/1.73 m² during the study period, further consideration was given as follows: the number of days between serum creatinine results was calculated for each individual, and any changes in eGFR across the 60 mL/min/1.73 m² threshold were noted. It was assumed that changes in eGFR across the threshold occurred at the midpoint between the sample dates (Figure 1). CKD was diagnosed when eGFR was <60 mL/min/1.73 m² for >50% of the time. We believe this method accounts for chronicity and approximates real-world decision making. We identified a cohort from 1 April 2010 to 31 March 2011, using this approach. For annual assessment of prevalence, the QOF allows the inclusion of data from the preceding 15-month period. Therefore, to avoid excluding CKD 3–5 patients in the year of interest, we also used laboratory data from 2009 to 2010. We replicated this approach for the following year (2011–12) to ensure reproducibility.

Primary care practice data

QOF data are published annually by the Information Services Division (ISD) of NHS Scotland and include CKD 3–5 prevalence by PCP [18]. ISD reports CKD prevalence as a percentage of the total population. We obtained the number of registered patients in each PCP by age and gender (as of October 2011) from ISD, and adjusted the denominator to the adult population, which is used throughout the analysis. Practice-level data including socio-economic status (SES), rurality, mean patient age, patient gender split, practice list size and number of GPs in post (headcount) were obtained from ISD [18].
SES was assessed using the 2009 Scottish Index of Multiple Deprivation (SIMD) at the practice level [14]. SIMD identifies areas of relative deprivation across Scotland by ranking small areas (1–6505 data zones) using a composite of 38 indicators of SES which are then subdivided into quintiles [19].

Rurality was assessed using the Scottish Government’s 8-fold Urban/Rural Classification where Class 1 denotes large urban areas with settlements of over 125 000 people and Class 8 denotes very remote areas with a population of <3000 people and with a drive time of over 60 min to a settlement of 10 000 or more [20].

Statistical analysis was carried out using the statistics software, SPSS version 16. Data are presented as mean ± standard deviation or median (inter-quartile range) as appropriate. Univariate and stepwise multivariate regression analyses were carried out as described in the text.

RESULTS

Study population

The study population is described in a flowchart in Figure 2. From the laboratory database, we identified all patients with CKD 3–5 and confirmed chronicity. This report focuses on the cohort from 2010 to 2011, with population demographics for the year presented in Table 1 and Figure 3.

Based on the laboratory data, and using the chronicity criterion, the prevalence of CKD 3–5 was 5.8% of adults (range: 3.0–9.1%). The prevalence for females was 7.3% and males 4.2%. In Scotland, the QOF reports CKD 3–5 prevalence using the total population as the denominator, which was 4.3%. Once expressed using the adult population as the denominator, prevalence was 5.4% (range between PCPs 2.8–11.0%).

Variation in practice-level CKD prevalence

Laboratory data confirmed variation in CKD prevalence at the PCP level, but to a lesser degree than the QOF data. There was a strong correlation between CKD laboratory prevalence (LabP) and age (Pearson’s r = 0.69, P < 0.001). We therefore generated CKD prevalences for each PCP, stratified to the age and gender structure of NHS Ayshire & Arran’s population (AGP), prior to further analyses.

After standardization for age and gender, SES was found to have a strong positive association with CKD prevalence (Figure 4). There were complex interactions between SES and other PCP demographic factors; level of rurality, mean age and patients to GP ratio (PGR) (Table 2).

In a stepwise multivariate regression model, SES, rurality, mean age and PGR were included as independent variables against the dependent variable, AGP. The strongest and most parsimonious model to emerge (Table 3) features only SES, rurality and PGR. The coefficients are positive, indicating that higher standardized CKD prevalence is associated with poorer SES and rurality and with more PGR. These three factors combined explained 39% (adjusted $R^2 = 0.392$) of the variability in prevalence ($F(3,50) = 12.37, P < 0.001$). SES was the single most influential predictor, accounting for 25% of the variability.

By applying the coefficient from the univariate regression of AGP and SES, we created funnel plots of CKD prevalence (LabP) before and after adjusting for age and sex then SES (Figure 5). Adjusting for age and gender, and then SES reduces the number of PCPs outside of 3 SDs from 15 to 7 to 6. For PCPs between 2 and 3 SDs, the number of practices fell from 16 to 13 to 5. Variation in prevalence between practices was 3.9-fold for QOF prevalences, 3.1 for laboratory prevalences, 2.1 for age- and gender-adjusted laboratory prevalences and 1.8 for age-, gender- and SES-adjusted laboratory prevalences.

DISCUSSION

We identified all patients with CKD 3–5 in a large, geographically defined Scottish population, using laboratory data, and taking into account chronicity (i.e. persistence of reduced eGFR for >90 days). CKD prevalence remained stable at 5.6–5.8% of adults from 2009–10 to 2011–12. Our study had three main findings. First, a laboratory case-finding approach was practical and identified more patients than PCP registers, equating to an additional 1255 patients (7% of the CKD population). There was a 3.9-fold variation in CKD prevalence between PCPs using QOF registers, ranging from 2.8 to 11.0%. Our second main finding was to demonstrate a reduction in variation to 3-fold by using laboratory-defined prevalence (3.0–9.1%). However, substantial variation persisted, suggesting it cannot be explained by clinical practice variation in registering disease alone. Age and gender are well-established predictors of CKD prevalence [3, 5], and the demographics of PCPs differ which results in age and gender acting as both confounders and effect modifiers in the relationship between PCPs and CKD prevalence. Adjusting for age and gender reduced this variation between PCPs substantially. Our third finding was that after age and gender, PCP population-weighted SES was the single most important factor for explaining the residual variation in CKD prevalence among PCPs.

Others have used a variety of case-finding approaches to investigate CKD prevalence: population surveys [5, 21], laboratory-based case findings [22, 23], primary care record searches [3, 24] or PCP registers [25]. However, there has been relatively limited attention to describing and understanding the variation in prevalence between PCPs. In England, the NHS Atlas of Variation has examined variation in CKD prevalence as reported on QOF registers and has benchmarked this against age- and gender-adjusted expected prevalences. In 2008–09 [26], the observed/expected ratio was 0.2–0.9 in primary care trusts (a 4.5-fold variation), when benchmarked against the NEOERICA study [3], which used primary care records and a single eGFR estimate. Variation at individual practice level was 10-fold. In 2010–11 [27], observed/expected ratios varied from 0.3 to 1.4 (4.7-fold), when benchmarked against the Health Survey for England [5], which uses a population survey approach based on a single eGFR and excluding institutionalized adults. Underlying explanatory factors for the variation were not sought in these studies. We also found that rurality was associated with higher CKD prevalence and this association persisted after stratifying the age and gender profile of the
population. We can find no other studies replicating this. There are complex interactions between age, SES and rurality, but each added to the model and remained significant in our multivariate model.

The association between CKD prevalence and SES has been described elsewhere but with differences in methods and indicators used to indicate level of SES. In a Swedish case–control study, individual-level SES as defined by occupation class or educational attainment was negatively associated with an increased risk of chronic renal failure (approximating to CKD stage 4) after adjusting for age, sex, BMI, smoking, alcohol and analgesic use [28]. In an English laboratory-based study [29], incidence of CKD 3–5 was associated with area-level SES with the highest SES group having a relative risk of 0.80 (0.69–0.93) and the lowest quintile 1.17 (1.02–1.33). The lower SES quintiles also had worse outcomes. Studies from a UK secondary care population demonstrated an association between a lower SES with increased referrals for CKD and also lower eGFR at presentation [30, 31]. An English study using a population survey found an increased risk of age- and sex-adjusted CKD with several individual-level socio-economic markers. After additional adjustment for ethnicity, smoking, BMI,
hypertension and diabetes mellitus, only renting household tenure versus ownership remained significant [32]. A large UK study based on primary care records showed an odds ratio of 0.92 for SES and CKD prevalence after multivariate analysis. However, the authors considered this insignificant having pre-specified significance at an odds ratio of 0.67. We found a linear relationship between CKD prevalence and the mean SES of the population served by PCPs. Whether the slope we identified applies to other populations in the UK and beyond requires confirmation.

Our study has limitations. We assumed that those with no serum creatinine results do not have CKD. We have also assumed that those with a reduced eGFR, but no confirmatory sample >90 days later, do not have CKD. Both assumptions could lead to an underestimate of CKD prevalence, and a population survey with repeated eGFR estimates would be necessary to address those flaws. However, the logistics and cost of such a study would be prohibitive. Most CKD occurs in older people, and we have previously shown that 71% of over 65 year olds had a serum creatinine checked in a single year (Figure 3); and that despite a 20% rise in the population having been sampled between 2004 and 2010, there was no change in CKD prevalence [2]. We therefore believe that relatively few CKD patients have been missed.

We chose a particular method of defining chronicity, which we believe mimics clinical decision making. Other methods are possible. De Lusignan et al. [33] examined a variety of approaches to defining CKD prevalence. When they required two eGFRs >90 days apart, with no intervening results >60 mL/min/1.73 m², they found a prevalence of CKD stages 3–5 of 5.41%. If intervening results were ignored, this rose to 5.55%. Our approach is different again, but likely to give an intermediate value. More importantly, many CKD prevalence studies are based on a single eGFR, with no attempt to confirm the chronicity [2, 3, 5, 21]. This leads to an overestimate of prevalence (6.41% in the study above). In our study, we demonstrate the practical application of the chronicity criteria in a large cohort with multiple serial results at varied intervals. This approach avoids misclassifying acute kidney injury as CKD. It also provides one possible approach to classify ‘borderline’ cases with eGFR measurements close to the 60 mL/min/1.73 m² threshold, in a fashion that mimics clinical decision making. Identifying the optimal way of classifying these borderline patients would require

### Table 1. Population characteristics in 2010–11

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Female (%)</td>
<td>52.2</td>
<td>51.4</td>
</tr>
<tr>
<td>Mean Age ± SD*</td>
<td>48 ± 18</td>
<td>50 ± 18</td>
</tr>
<tr>
<td>Mean SIMD ± SD*</td>
<td>3.0 ± 1.4</td>
<td>3.4 ± 1.4</td>
</tr>
<tr>
<td>Proportion of population** (%)</td>
<td>65 and over</td>
<td>20.9</td>
</tr>
<tr>
<td>75 and over</td>
<td>9.6</td>
<td>10.1</td>
</tr>
<tr>
<td>85 and over</td>
<td>2.5</td>
<td>2.6</td>
</tr>
</tbody>
</table>

*Excludes population age <18 years.
**Scottish Index of Multiple Deprivation scale, least socio-economically disadvantaged = 1 and most disadvantaged = 5.

### Table 2. Univariate relationships between primary care practice, demographic factors and laboratory chronic kidney disease prevalence after adjustment for age and gender (AGP)

<table>
<thead>
<tr>
<th>n</th>
<th>Mean age</th>
<th>PGR</th>
<th>SES</th>
<th>Rurality</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGP</td>
<td>−0.262</td>
<td>0.060</td>
<td>0.581**</td>
<td>0.270*</td>
</tr>
<tr>
<td>Mean age</td>
<td>−0.461**</td>
<td>0.389**</td>
<td>0.363**</td>
<td></td>
</tr>
<tr>
<td>PGR</td>
<td>−0.037</td>
<td>−0.430**</td>
<td>0.233</td>
<td></td>
</tr>
<tr>
<td>SES</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

PGR, patients to GP ratio; SES, socio-economic status.
*Pearson’s r.
**Spearman’s ρ.
*P < 0.05.
**P < 0.01.
longitudinal outcome data, which was beyond the scope of this study.

The difference between the QOF-reported prevalence and LabP range from −4.2 to 2.5%. However, for information governance reasons, we were unable to access PCP registers, to directly assess their accuracy compared with laboratory data. This means that the discrepancy between PCP registers and the gold standard laboratory data may actually be larger still. It is worthwhile noting that within the limitation of our data, there appears to be both under- and over-reporting within PCP registers which we believe is unintentional and generally reflects the inefficiencies of the systems in use. Using a particularly stringent definition of CKD stages 3–5, which required food-fasted samples, we previously found that 27% of patients on CKD registers did not have CKD stage 3–5 [34]. If this finding is generalizable, it could mean that up to a further 4457 patients with laboratory-identified CKD are not included on PCP registers. We were also unable to access individual patient comorbidities, which would have allowed more detailed investigation of the association between socio-economic status and CKD prevalence.

Identification of patients with CKD 3–5 is the first step in optimizing the renal health of the population. Patients with CKD but not on the register may not receive appropriately targeted monitoring, referral and treatment. Patients incorrectly on the register may receive inappropriate interventions. Currently, the UK relies on populating CKD registers with patients identified and coded by PCPs. Some PCPs perform this task manually, while others use software packages that interrogate PCP electronic records, but do not correct the serum creatinine for IDMS standardization. The large proportion of serum creatinine results carried out by other community health services or in secondary care is mostly not included.

Laboratory information is both necessary and sufficient to identify patients with CKD 3–5. We have shown that laboratory case findings are technically feasible and desirable. The methods described in this paper are easily replicable throughout the

Table 3. Predictors of age and gender standardized CKD prevalence at primary care practice level: regression coefficients

<table>
<thead>
<tr>
<th></th>
<th>B (s.e.)</th>
<th>Standardized</th>
<th>t</th>
<th>95% CI</th>
<th>Part correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Constant)</td>
<td>2.053 (0.728)</td>
<td>2.822**</td>
<td>0.592, 3.515</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SES</td>
<td>0.714 (0.155)</td>
<td>0.512</td>
<td>4.635**</td>
<td>0.406, 0.497</td>
<td></td>
</tr>
<tr>
<td>Rurality</td>
<td>0.163 (0.061)</td>
<td>0.356</td>
<td>2.662**</td>
<td>0.040, 0.285</td>
<td></td>
</tr>
<tr>
<td>PGR</td>
<td>0.088 (0.041)</td>
<td>0.279</td>
<td>2.139*</td>
<td>0.005, 0.229</td>
<td></td>
</tr>
</tbody>
</table>

SES, socio-economic status; PGR, patients (hundreds) to GP ratio; CI, confidence intervals.

*P < 0.05.

**P < 0.01.

**Figure 5:** Funnel plots of CKD 3–5 prevalence by primary care practice population (A–D). (A) Funnel plot constructed using primary care practice QOF reported CKD stage 3–5, unadjusted prevalences. (B) Funnel plot constructed using primary care practice unadjusted laboratory-ascertained CKD stage 3–5 prevalences. (C) Funnel plot constructed using primary care practice laboratory prevalences, adjusted for population age and gender profile. (D) Funnel plot constructed using primary care practice laboratory prevalences, adjusted for age, gender and socio-economic status.
UK and any other health system with high-quality information systems at the population level. Implementation would improve the accuracy and completeness of CKD registers that may lead to improved care and outcomes. There are very few studies in CKD examining the impact of QOF on outcomes, with one study demonstrating a sustained improvement in the mean blood pressure of CKD patients after the introduction of QOF [35]. Another study found that the proportion attaining blood pressure targets, along with diabetes prevalence, age and ethnicity combined predicted 40% of the variation in the incidence of renal replacement therapy [36].

A similar approach could also be applied to proteinuria, as laboratory quantification is recommended [37, 38]. However, ascertainment will be less complete, as a smaller proportion of the population are tested. Even among patients with CKD 3–5, only 82% have had proteinuria quantified [12].

The mechanism by which low SES is associated with increased CKD prevalence is not clear. Other factors associated with CKD are known to be more common in lower SES populations, including obesity, smoking, diabetes mellitus and vascular disease and so low SES may simply be a composite marker for these [39]. The Whitehall II study demonstrated that in white London civil servants, 25% of the observed association between SES and decreased GFR could be explained by the combined effect of obesity and metabolic syndrome [40]. However, even after adjustment for known risk factors associated with low SES, residual increased risk can remain [41].

On a more practical level, others have assumed that an age- and gender-adjusted prevalence is a reasonable benchmark to use for PCPs or regions [27], with the assumption that those with lower than expected prevalence are failing to identify or register patients. Our study shows the importance of also including socio-economic status when benchmarking, as the number of statistical outliers fell from 20 to 11 practices. Using this approach will allow further investigation to be targeted at a smaller group of outliers.

In conclusion, we have demonstrated that using a laboratory case-finding approach identifies patients who would otherwise not be included in CKD registers. We have also shown that this approach reduces the variation in CKD prevalence between PCPs. Using such an approach is both practicable and likely to improve the accuracy and completeness of CKD registers. Accurate and complete CKD registers are a prerequisite for target monitoring, interventions and appropriate referrals to secondary care. We have also confirmed the association between SES and CKD prevalence, and shown the importance of adjusting for SES when aiming to detect unwarranted variations in CKD prevalence.

ACKNOWLEDGEMENTS

We thank Gordon Duncan, Area Laboratory Systems Manager at University Hospital Crosshouse for his help writing the database query and extracting the laboratory data required to conduct this study and the Research and Development Department at University Hospital Crosshouse for their help in navigating local information governance safeguards and liaising with the regional ethics department.

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CONFLICT OF INTEREST STATEMENT

Ethical permission was sought, but not required as the study was classified as an audit. All local information governance safeguards were adhered to during the study. The protocol for this study is available on request. Aggregated data may be made available but are entirely dependent upon approval by the information governance guardians on a case-by-case basis. All authors have contributed substantially to the work to be listed as such. This manuscript and the results have not been published elsewhere except in abstract. All authors have completed the ICMJE uniform disclosure form and declare no support or financial relationship to any organisation with a vested interest in the submitted work. The authors also affirm that the manuscript is an honest, accurate and transparent account of the study being reported and that no important aspects of the study have been omitted. No form of funding was sought or utilised for the duration of this study.


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