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Research: Complications

Progression of chronic kidney disease in a multi-ethnic community cohort of patients with diabetes mellitus

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Abstract

Aims Ethnicity is a risk factor for the prevalence of severe chronic kidney disease among patients with diabetes. We studied the effect of ethnicity on progression of chronic kidney disease in people with diabetes managed in community settings.

Methods A 5-year retrospective, community-based cohort study of 3855 people with diabetes mellitus of white, black or South Asian ethnicity with an estimated glomerular filtration rate of < 60 ml min^{-1} 1.73 m^{-2} was undertaken. From 135 general practices in east London, all cases with at least 3 years clinical data were included. Using repeated-measures analysis, the annual decline in estimated glomerular filtration rate was calculated. Comparisons between the rate of decline in the three main ethnic groups, with and without proteinuria at baseline, were made.

Results The annual adjusted decline in estimated glomerular filtration rate for this cohort was 0.85 ml min^{-1} 1.73 m^{-2}. The rate of chronic kidney disease progression was significantly greater in South Asian groups (–1.01 ml min^{-1} 1.73 m^{-2}) compared with white groups (–0.70 ml min^{-1} 1.73 m^{-2}) ($P = 0.001$). For those with proteinuria at baseline, the annual decline was greater at 2.05 ml min^{-1} 1.73 m^{-2}, with both South Asian and black groups having a significantly faster rate of decline than white groups.

Conclusions For patients with diabetes and chronic kidney disease managed in primary care, the annual decline of renal function is less than previously thought and approximates the age-related annual decline of 1 ml min^{-1} 1.73 m^{-2}. Patients with proteinuria and those of South Asian and Black ethnicity need additional monitoring as they are at greater risk of rapid chronic kidney disease progression.

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Introduction

Chronic kidney disease and diabetes mellitus represent a growing public health problem in the UK [1,2]. Ethnicity has been identified as a risk factor for the prevalence and severity of concomitant chronic kidney disease in patients with diabetes mellitus. A previous study of chronic kidney disease and diabetes conducted in 151 general practices in east London identified both a higher prevalence of diabetes mellitus and a greater severity of associated chronic kidney disease in South Asian patients [3]. Severe chronic kidney disease (stage 4–5) was more prevalent in South Asians despite lower blood pressure compared with black or white patients. In line with these observations, ethnic minorities are over-represented on renal replacement therapy programmes in the UK [4] and the USA [5].

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Ethnicity has been associated with more rapid progression of chronic kidney disease [6–9]. Existing studies in the UK on the progression of chronic kidney disease in different ethnic groups are small and provide conflicting data. A hospital-based study of 45 patients [7] identified more rapid progression of kidney disease in South Asian patients compared with white and black patients. However, a similar hospital-based cohort of 39 patients showed no differences in the rate of chronic kidney disease progression by ethnic group [10].

Currently, there is insufficient evidence to determine why ethnic minorities are over-represented on UK renal replacement therapy programmes, and why South Asian patients with diabetes have a higher prevalence of more severe chronic kidney disease. No studies have examined ethnic differences in the progression of diabetes-related chronic kidney disease in primary care in the UK, where most cases of chronic kidney disease and diabetes mellitus are both identified and managed. Identifying patient groups at risk

of progressive chronic kidney disease offers the potential both to understand ethnic variations on renal replacement therapy programmes, and also to provide targeted health care to groups at higher risk of complications.

This study examines the rate of progression of chronic kidney disease in patients with diabetes (Type 1 and Type 2) managed in UK primary care and aims to determine whether there are differences in the progression of chronic kidney disease between the major ethnic groups.

Patients and methods

The study was set in the three geographically contiguous east London primary care trusts of Newham, Tower Hamlets, and City and Hackney, with a combined general practitioner-registered population of 829 710 in 2008 (the midpoint of this study). In the 2001 UK census, 51.3% of the population in these three primary care trusts was recorded as of non-white ethnic origin [11]. The populations of these primary care trusts are among the eight most socially deprived localities in Britain [11]. In April 2010, anonymized demographic and clinical data were obtained for all adults over the age of 18 years registered with 134 general practices in east London. This practice data covered more than 98% of the general practitioner-registered population in the three primary care trusts. All data were anonymous and managed according to UK National Health Service (NHS) information governance requirements. Ethics approval was obtained for this observational study (REC reference 09/H1102/71).

Clinical data collection

Practice computer databases were interrogated using Egton Medical Information Systems Web software [12]. Clinical and prescribing data were collected for the period 2 April 2005 to 2 April 2010.

Patients with Type 1 or Type 2 diabetes were included in the study if they had an estimated glomerular filtration rate (eGFR) of between 15 and 60 ml min^{-1} 1.73 m^{-2} on two or more readings at least 3 months apart, could provide $\geq$ 3 years of annual follow-up data, and were between 30 and 75 years at entry to the study. Patients with a record of being on either haemo- or peritoneal dialysis at entry to the study were excluded, and follow-up data were censored once dialysis was commenced. Age and date of diabetes diagnosis were included.

Variables for analysis included renal function as the eGFR expressed by the four-variable modification of diet in renal disease (MDRD) equation, systolic and diastolic blood pressure, glycated haemoglobin, proteinuria, total cholesterol, smoking status, BMI, vascular co-morbidity (hypertension, ischaemic heart disease, heart failure, cerebrovascular disease) and prescription data for angiotensin-converting enzyme inhibitor, angiotensin receptor blocker and all non-steroidal anti-inflammatory drugs. Presence of proteinuria was considered to be 'positive' if the patient had a protein: creatinine ratio value of $\geq$ 15 mg/mmol, an albumin:creatinine ratio value of $\geq$ 2.5 mg/mmol for men, or 3.5 mg/mmol for women, or a urine dipstick result $\geq$ 1 +.

Renal function is expressed as stage of chronic kidney disease defined by current UK guidelines [13] based on the eGFR value: stage 3a chronic kidney disease (eGFR 45–59 ml min^{-1} 1.73 m^{-2}), stage 3b chronic kidney disease (eGFR 30–44 ml min^{-1} 1.73 m^{-2}), stage 4 chronic kidney disease (eGFR 15–29 ml min^{-1} 1.73 m^{-2}) and stage 5 chronic kidney disease (eGFR < 15 ml min^{-1} 1.73 m^{-2}). All eGFR measures have been corrected for Black ethnicity (eGFR multiplied by 1.21). Different methods for measuring the serum creatinine (from which the eGFR is calculated) are used in the study area; however, as we report elsewhere [3], differences between techniques result in very small changes in eGFR that are likely to be equally distributed across the different ethnic groups and we do not consider that these differences will change the overall classification of chronic kidney disease.

For eGFR, all values in each of the study years were collected. Values which were $\geq$ 20 ml min^{-1} 1.73 m^{-2} higher than the preceding value were excluded as these were considered to be outliers. In the entire data set 310/70 672 (0.004%), values were excluded for this reason. This approach has been taken in other, similar studies [14]. An annual average eGFR was calculated for each patient year of the study. As serum creatinine recording was more complete than eGFR recording, the eGFR values used for analysis were derived from the creatinine value using the modification of diet in renal disease equation. For all other variables, the most recent value in each year of follow-up was used.

Ethnicity and demographic data

Self-reported ethnicity was recorded at the practice during registration or routine consultation. Ethnic categories are based on the UK 2001 census and for this study were condensed into three categories: white (British, Irish, other white), black (Black African, Black Caribbean, Black British, other black and mixed black), South Asian (Bangladeshi, Pakistani, Indian, Sri Lankan, British Asian, other South Asian or mixed Asian). Patients with mixed ethnicity were grouped with their parent ethnic minority. For example, an individual who had classified themselves as mixed South Asian and British is classed as South Asian for the purposes of this study. Patients with other, unknown, or missing ethnicity were not included in the analysis. The Townsend score (derived from census variables and linked to patient place of residence) was used as a measure of social deprivation [15].

Statistical analysis

All statistical analyses were performed using Stata version 10 [16]. Numerical variables with values outside of the valid range were removed from the data set.

To account for the hierarchical nature of the data—where multiple observations are nested in patients, who are nested in general practices—a multi-level linear regression model was used to calculate the annual change in mean eGFR.

To investigate whether the annual decline in eGFR was modified by ethnicity, an interaction term between years of follow-up and ethnic group was utilized. Adjusted analyses controlled for age at the start of the study, sex, deprivation, clinical factors, duration of diabetes at baseline, prescribed drugs, ischaemic heart disease, co-morbidity presence of proteinuria at baseline and eGFR value at baseline.

For systolic blood pressure, HbA_{1c} and BMI, the mean value for each patient across the whole study period were used. Prescription of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers and non-steroidal anti-inflammatory drugs (NSAIDs), were represented by a categorical variable for ever/never prescribed over the course of the study.

The analysis was conducted firstly for the whole study population, and then separately for those with and without positive proteinuria at baseline

Results

From a total population of 48 406 patients with diabetes in April 2010, 3855 individuals with chronic kidney disease were identified for inclusion in the retrospective cohort (Fig. 1).

The study cohort comprised 1725 South Asian patients (44.7%), 1509 white patients (39.1%) and 621 black patients (16.2%). Over the course of the study, 2089 patients had data in each of the 5 years (54.2%), 986 patients had data in 4 out of 5 years (25.6%) and 780 had data in 3 out of 5 years (20.2%). In 2006, 3065 patients joined the study. An additional 439 joined in 2007 and 351 in 2008. Patients could not enter the study after this point as a minimum of 3 years of data was required. Two hundred and eleven patients left the study in 2008, 372 left in 2009 and 3272 remained until 2010. Reasons for leaving the study included moving out of the study area, commencement of dialysis or death.

The South Asian group is younger than other groups, with a greater proportion of women. Black groups have the highest rate of hypertension and the lowest rate of ischaemic

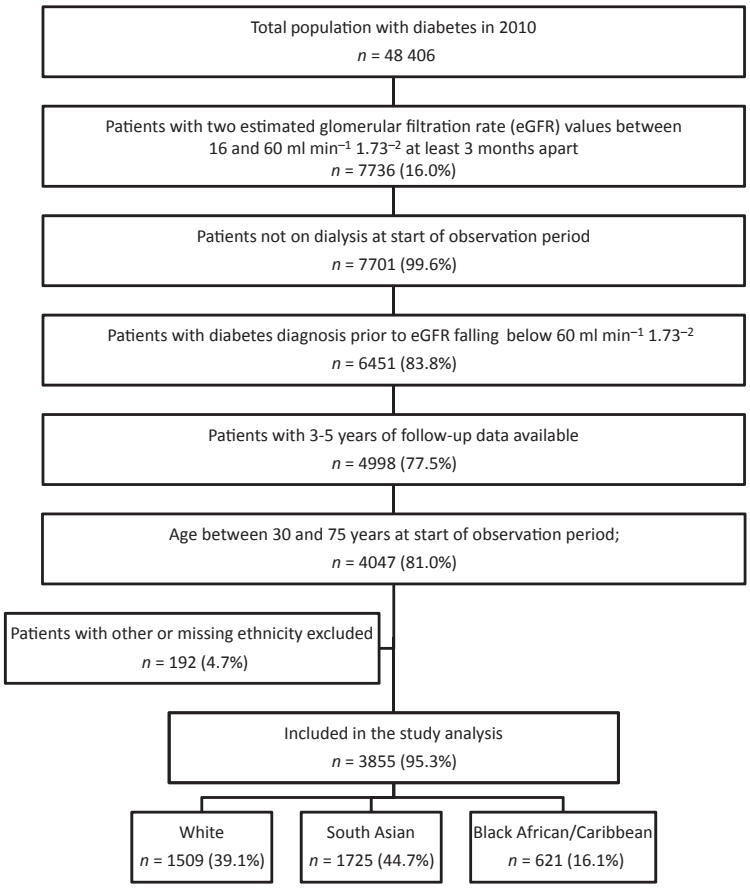

FIGURE 1 Study sample derivation

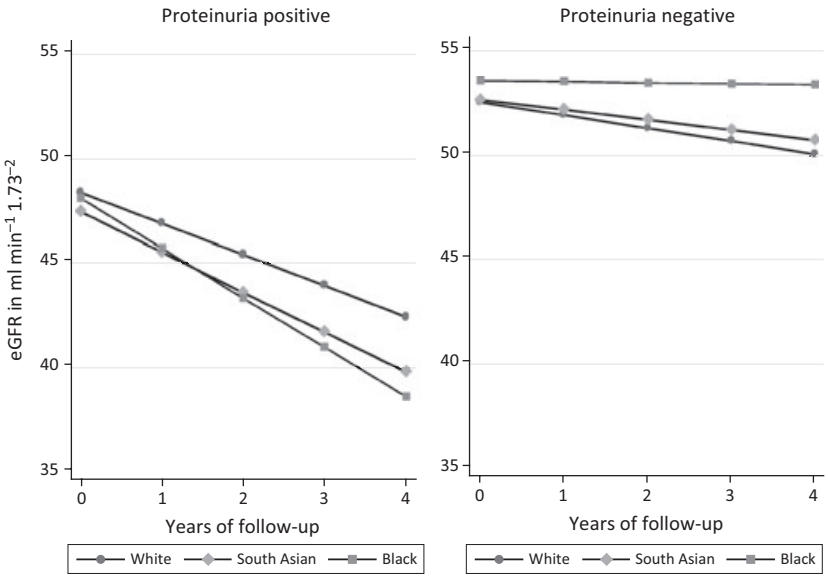

FIGURE 2 Predicted values from linear regression for annualized change in estimated glomerular filtration rate (eGFR) by ethnic group for study patients with positive and negative proteinuria at baseline.

Table 1 Baseline characteristics of study population (cohort size = 3855)

Demographic characteristics	No. with data	White (n = 1509)	South Asian (n = 1725)	Black (n = 621)	*P*-value* South Asians vs. white	*P*-value* black vs. white
Mean years follow-up (SD)	3855	4.4 (0.8)	4.3 (0.8)	4.3 (0.8)	< 0.001	0.119
Median number of eGFR values (range)	3855	10 (3–57)	10 (3–76)	10 (3–45)	0.004	0.702
Mean number of years with diabetes at baseline (SD)	3855	7.8 (8.6)	8.8 (7.7)	9.9 (8.1)	0.002	< 0.001
Baseline measures						
% men	3855	61.1	50.7	59.6	< 0.001	0.221
Mean age (SD)	3855	65.3 (8.1)	63.1 (8.5)	64.3 (8.2)	< 0.001	0.013
Mean systolic blood pressure (SD)	3778	136.4 (17.8)	134.9 (18.9)	140.4 (18.9)	0.023	< 0.001
Mean diastolic blood pressure (SD)	3778	74.6 (10.2)	74.3 (10.2)	77.7 (10.4)	0.510	< 0.001
Mean HbA_{1c} (IFCC aligned) (SD)	3454	60.3 (16.7)	65.4 (17.6)	64.4 (20.4)	< 0.001	< 0.001
Mean HbA_{1c} (%) (SD)	3454	7.7 (1.5)	8.1 (1.6)	8.1 (1.9)	< 0.001	< 0.001
% current smokers	3057	13.8	6.7	4.5	< 0.001	< 0.001
% with proteinuria test recorded	3855	70.0	69.3	73.3	0.690	0.129
% with positive proteinuria	2720	26.5	36.4	30.3	< 0.001	0.131
% on angiotensin-converting enzyme inhibitors/angiotensin receptor blockers	3855	79.5	79.7	83.6	0.927	0.032
% on NSAIDs	3855	68.8	71.4	72.1	0.103	0.126
Baseline co-morbidities						
% with ischaemic heart disease	3855	28.9	31.7	13.9	0.089	< 0.001
% with hypertension	3855	78.1	73.7	88.7	0.004	< 0.001
% with stroke	3855	11.1	10.8	10.6	0.791	0.735
% with heart failure	3855	8.4	8.5	6.6	0.961	0.159

**P*-values for differences by ethnic group are derived using linear regression for continuous variables, and logistic regression for categorical variables.
eGFR, estimated glomerular filtration rate; IFCC, International Federation of Clinical Chemistry; NSAID, non-steroidal anti-inflammatory drug.

heart disease. The white group is the oldest, with the highest rates of smoking and the best control of diabetes (HbA_{1c})

A record of proteinuria testing was available for 70% of the sample at baseline; there was no variation in recording frequency by ethnicity. A positive proteinuria result was most common in the South Asian group (Table 1).

Annual decline in eGFR

A crude overall decline in eGFR of 3.44 ml min^{-1} 1.73^{-2} was observed for the whole study cohort over the 4-year follow-up period. Adjusted linear regression indicated an annualized change of –0.85 ml min^{-1} 1.73^{-2} in the whole cohort. There was a statistically significantly greater decline in South Asian compared with white groups ($P < 0.001$, Table 2).

Annual decline in mean eGFR for those with proteinuria positive at baseline

In the first year of follow-up, 2720 (70%) had a proteinuria test recorded. Of this group, 857 (31.5%) were classed as having proteinuria at baseline (see Patients and methods). Compared with the whole study cohort, the crude overall decline in the subpopulation who are proteinuria positive at baseline is over double that of the whole cohort (Table 2). Adjusted linear regression indicates that the annual change in mean eGFR increased from –0.85 ml min^{-1} 1.73^{-2} to –2.05 ml min^{-1} 1.73^{-2} in this subgroup. Furthermore, the annual change in mean eGFR is significantly greater for non-white groups in comparison with the reference population ($P = 0.034$ for South Asian, $P = 0.001$ for black groups; see Fig. 1 and Table 2).

Annual decline in mean eGFR for those without proteinuria at baseline

For the 1863 individuals who were proteinuria negative at baseline, the adjusted linear regression indicates that the annual change in eGFR is –0.42 ml min^{-1} 1.73^{-2}, with the amount of annual change significantly less in black groups compared with white ($P = 0.002$ for black groups, see Table 2), with no difference between white and South Asian patients.

Discussion

Main findings

Data from this large, multi-ethnic, primary care cohort, followed over 4 years, demonstrates that progression of chronic kidney disease in patients with diabetes mellitus managed in primary care settings is slow and is equivalent to the consensus on age-related renal function decline of approximately 1 ml min^{-1} $year^{-1}$ [17,18].

Table 2 Crude and adjusted change in estimated glomerular filtration rate (eGFR) over total follow up period by ethnic group

	Whole study population ($n = 3855$)				Proteinuria positive at baseline ($n = 857$)				Proteinuria negative at baseline ($n = 1863$)			
Crude change in eGFR over total study period in ml min^{-1} 1.73^{-2}*	%	Mean baseline eGFR	Mean final eGFR	Overall difference	%	Mean baseline eGFR	Mean final eGFR	Overall difference	%	Mean baseline eGFR	Mean final eGFR	Overall difference
Whole population	100	51.39	47.94	–3.44	100	49.40	40.97	–8.42	100	52.75	51.32	–1.44
White	37.29	51.34	48.68	–2.66	32.79	49.76	42.51	–7.25	41.89	52.46	51.17	–1.29
South Asian	42.62	51.04	46.79	–4.25	51.11	48.69	40.52	–8.17	40.97	52.89	50.57	–2.02
Black	15.34	52.48	49.35	–3.13	16.10	50.92	39.31	–11.61	17.14	53.85	53.47	–0.38
Adjusted annual change in eGFR†	Loss of eGFR in ml min^{-1} 1.73^{-2}		*P*-value		Loss of eGFR in ml min^{-1} 1.73^{-2}		*P*-value		Loss of eGFR in ml min^{-1} 1.73^{-2}		*P*-value	
Whole population	–0.85		< 0.001		–2.05		< 0.001		–0.42		< 0.001	
White (reference)	–0.72		—		–1.72		—		–0.50		—	
South Asian	–1.01		0.001		–2.11		0.034		–0.49		0.965	
Black	–0.73		0.906		–2.51		0.001		–0.05		0.002	

*Adjusted analysis for the whole population controls for age at baseline, mean systolic blood pressure, mean HbA_{1c}, mean BMI, prescription of non-steroidal anti-inflammatory drugs and angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, presence of ischaemic heart disease ever, presence of proteinuria ever, sex, deprivation, duration of diabetes at baseline and baseline eGFR.

†Adjusted analysis for the proteinuria positive/negative subgroups control for age at baseline, mean systolic blood pressure, mean HbA_{1c}, mean BMI, prescription of NSAIDs and angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, presence of ischaemic heart disease ever, sex, deprivation, duration of diabetes at baseline and baseline eGFR.

In the entire community cohort, we find that differences in rate of progression are present, with South Asian groups progressing faster than the white reference group. These differences occur despite lower blood pressure in the South Asian group. However, the absolute rate of progression is slow and the differences between groups are small in patients without proteinuria at baseline.

We demonstrate that, among the cohort of patients with proteinuria at baseline, the rate of chronic kidney disease progression is doubled. Analysis by ethnicity shows that both South Asian and black groups with proteinuria progress faster than the white reference population, even after adjustment for key clinical and demographic data. This may be one reason why there is a higher prevalence of severe chronic kidney disease in these populations [3].

Difference in chronic kidney disease progression by ethnicity

There are several potential mechanisms by which differences in progression by ethnicity may be mediated. A lower nephron mass, often the result of adverse fetal programming in ethnic minority groups, predisposes to glomerular hyperfiltration and consequent glomerular fibrosis, resulting in the development of proteinuria and, in turn, more rapid progression of kidney disease [19].

Elevated blood pressure is a strong mediator for the progression of diabetic nephropathy [20.21]. It has been shown that mean blood pressure varies by ethnicity and, more specifically, black populations have a higher average blood pressure than white populations, and South Asians in turn have a lower mean blood pressure compared with white populations [22–24]. Reducing the blood pressure in black populations to those in current UK guidelines for patients with diabetes and nephropathy (< 130/80 mmHg) [25] is an appropriate therapeutic target. For the South Asian population, the UK guideline target blood pressure may reflect mild hypertension and thus contribute to the progression of chronic kidney disease.

Strengths and weaknesses

This study is the largest primary care cohort of people with diabetes and concomitant chronic kidney disease to examine the progression of chronic kidney disease by ethnicity. Length of follow-up was similar for all groups, and ethnicity recording for those on the diabetes register was 95% complete.

Patients were only included in the cohort if the diagnosis of chronic kidney disease was confirmed by two separate measures of eGFR, in line with current definitions of chronic kidney disease. Patient turnover was less than expected for inner London, with 80% of patients in this cohort providing follow-up data for either 4 or 5 years. Completeness of ethnicity data and the setting within a multi-ethnic area of London makes the results relevant to other areas in the UK with similar populations. In the UK health system, access to primary care is free, and hence reduces the confounding attributable to differential access to treatment by ethnicity noted in some studies based in the USA [26].

A potential weakness of the study is that proteinuria status was incomplete and the recording method was heterogeneous, including dipstick testing, hence we cannot examine the effect of the intensity of proteinuria on chronic kidney disease progression. Data on over-the-counter drug use, particularly that of NSAIDs, which may adversely affect renal function and could vary by ethnicity, are not available. Despite the long follow-up time, we did not have access to data on hospital admission, attendance at specialist diabetes or renal outpatient clinics or mortality. We have condensed the ethnic groups, based on the 2001 census categories, into three broad categories, but it is possible that progression differs between different subsets within the major ethnic groups. This question is being addressed in a concurrent study. We were not able to determine the exact cause of underlying kidney disease in this primary care cohort as these patients are rarely investigated with renal biopsy. Some patients may have an additional renal lesion along with diabetic nephropathy [27]; however, we are not aware of evidence that the prevalence of non-diabetic renal disease in patients with diabetes differs significantly by ethnic group

Implications for clinical practice

Early chronic kidney disease associated with diabetes mellitus is unlikely to progress rapidly in a primary care setting. The challenge for clinicians is to identify who may be at greater risk of progression, to optimize management and decide who might need referral for specialist secondary care. Patients of South Asian or Black ethnicity, as well as the presence of proteinuria, will identify patients at the highest risk of rapid progression and most in need of aggressive therapeutic intervention or specialist referral. For black groups, a therapeutic approach that brings blood pressure down to the targets suggested by UK guidelines is appropriate. In contrast, South Asian patients may require a target blood pressure lower than that suggested by current guidelines [25].

In this cohort, 70% had been prescribed NSAID medication. Data on over-the-counter NSAID medication use is not routinely collected in the UK, although high proportions self-treat for pain and musculoskeletal conditions [28]. Increased awareness among patients and clinicians of the potential nephrotoxic effects of these medications is a further target for intervention.

Conclusion

In community settings, the progression of chronic kidney disease in patients with diabetes mellitus is slow and the

majority of cases can be managed safely by primary care teams. The presence of proteinuria doubles the average rate of decline. The UK Quality and Outcomes Framework for general practice [29] reflects this additional risk by requiring annual proteinuria testing and initiation of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers for those who are proteinuria positive.

Our findings confirm that ethnicity is an independent risk factor for more rapid progression of chronic kidney disease in patients with diabetes mellitus, particularly among those patients with proteinuria. The mechanism underlying these findings is likely to be multifactorial, including the result of fetal programming combined with additional renal insults, cultural differences (such as medicines adherence, use of clinical services, lifestyle factors) and socio-economic factors which we are not able to capture in routine clinical databases.

The high prevalence of diabetes mellitus in South Asian populations identifies this group as high risk for progressive chronic kidney disease and cardiovascular consequences. Guidelines may need to reflect risk based on ethnicity, as well as factors such as proteinuria to optimize reno-protective strategies, and facilitate early consultation with secondary care.

Further studies are required to understand the mechanisms mediating faster progression in South Asians, and the increased rates of progression in both South Asian and black populations with proteinuria. Identifying preventative strategies for these groups may contribute to a reduction in the over-representation of ethnic minority groups on UK renal replacement programmes.

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Competing interests

None declared.

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