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Incident chronic kidney disease and the rate of kidney function decline in individuals with hypertension

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Abstract

Background. Little is known about the decline of kidney function in patients with normal kidney function at baseline. Our objectives were to (i) identify predictors of incident chronic kidney disease (CKD) and (ii) to estimate rate of decline in kidney function.

Methods. The study used a retrospective cohort of adult patients in a hypertension registry in an inner-city health care delivery system in Denver, Colorado. The primary outcome was development of incident CKD, and the secondary outcome was rate of change of estimated glomerular filtration rate (eGFR) over time.

Results. After a mean follow-up of 45 months, 429 (4.1%) of 10 420 patients with hypertension developed CKD. In multivariate models, factors that independently predicted incident CKD were baseline age [odds ratio (OR) 1.13 per 10 years, 95% confidence interval (CI), 1.03–1.24], baseline eGFR (OR 0.69 per 10 units, 95% CI 0.65–0.73), diabetes (OR 3.66, 95% CI 2.97–4.51) and vascular disease (OR 1.67, 95% CI 1.32–2.10). We found no independent association between age, gender or race/ethnicity and eGFR slope. In patients who did not have diabetes or vascular disease, eGFR declined at 1.5 mL/min/1.73 m^2 per year. Diabetes at baseline was associated with an additional decline of 1.38 mL/min/1.73 m^2.

Conclusions. Diabetes was the strongest predictor of both incident CKD as well as eGFR slope. Rates of incident CKD or in decline of kidney function did not differ by race or ethnicity in this cohort.

Keywords: chronic kidney disease; hypertension; progression

Introduction

Chronic kidney disease (CKD) is common, affecting 13.1% of the United States (US) population; 26.3 million Americans are estimated to have stages 1–4 CKD [1]. Hypertension is closely associated with CKD; 70% of patients with an elevated serum creatinine (defined as ≥1.6 mg/dL for men and ≥1.4 mg/dL for women) have hypertension [2], and 26.8% of patients with end-stage renal disease (ESRD) were deemed to have kidney failure as a result of hypertension by treating nephrologists [3]. Although high blood pressure is a major promoter of the decline in glomerular filtration rate (GFR) in diabetic and non-diabetic kidney disease [4], only a small percentage of patients with hypertension will develop CKD [5]. Therefore being able to identify those hypertensive individuals at

high risk for decline in kidney function early in the course of their disease process may help to guide their treatment.

Existing studies on the rate of decline in kidney function have been based on subjects in whom CKD (defined as GFR <60 mL/min/1.73 m^2) has already developed; little information is available on rates of decline in kidney function prior to the onset of CKD [6], specifically in known hypertensive cohorts. In addition, many inferences have been drawn from post-hoc analyses of subjects enrolled in clinical trials, who may not be representative of patients encountered in routine clinical care.

Therefore, the objectives of the current study were to (i) identify predictors of incident CKD and (ii) to estimate rate of decline in kidney function in a racially and ethnically diverse hypertensive patient population with normal or near normal kidney function at baseline.

Methods

Subjects

The data source for the study was an electronic registry of patients with hypertension at Denver Health, a large safety-net community health system in Denver, Colorado, USA which provides integrated primary and specialty care and emergency medicine and acute hospital care for the socially disadvantaged, vulnerable population of Denver [7,8]. The registry assembled information from patient registration files (age, sex, race/ethnicity, marital status and language), claims files (site, date, payer and diagnostic codes for all visits), laboratory files (dates and results of blood and urine tests) and pharmacy files. Patients were included in the registry if they received care between 1 January 2000 and 31 December 2006 and had at least one ICD-9CM (International Classification of Diseases, 9th Revision, Clinical Modification) code for hypertension (401, 401.0, 401.1, 401.9, 405, 405.0, 405.01, 405.09, 405.1, 405.11, 405.19, 405.9, 405.91, 405.99, 642.0x, 642.1x, 642.2x, 642.3x, 642.4x, 642.5x, 642.6x, 642.7x, 642.9x) coded in either an inpatient or outpatient administrative claim. We have previously found that this method for identifying patients with hypertension (defined as blood pressure >130/80 mm Hg in diabetic patients or >140/90 mm Hg in patients without diabetes) has a sensitivity of 88% and a specificity of 78% [9]. For the current analysis, we excluded patients with missing information on gender, individuals with no contact with the Denver Health system after their hypertension diagnosis, individuals who were <21 years of age at the time of first visit for hypertension and individuals who received pregnancy-related care during the study time period (because of known physiologic changes in the serum creatinine during pregnancy). We also excluded patients with a baseline estimated glomerular filtration rate (eGFR) <60 or >200 mL/min/1.73 m^2 or who did not have two baseline eGFR values ≥ 60 mL/min/1.73 m^2 at least 1 month apart using the Modification of Diet in Renal Diseases (MDRD) prediction equation [10]. Thus, patients included in this analysis were required to have a minimum of three serum creatinine measurements for inclusion, the first two to establish the baseline eGFR and the third, at least 1 year after the index date in order to calculate the rate of change of eGFR over time.

Definitions

The index date for the study cohort was defined as the time of the first creatinine measurement after entry into the hypertension registry. We defined baseline comorbidities (diabetes, vascular disease, congestive heart failure, dyslipidemia, mental health conditions and substance abuse) using combinations of billing codes and laboratory values as previously described [9]. We defined vascular disease as a composite including coronary heart disease, peripheral vascular disease and cerebrovascular disease. The mental health diagnosis was comprised of individuals with depression, bipolar disorder and psychotic conditions. The substance abuse diagnosis combined alcohol dependence with illicit drug use or dependence. These comorbidities were counted as present at baseline if they were first recorded on visits prior to or up to 180 days after the index date. Estimated GFR was defined using the four-variable abbreviated MDRD study equation: eGFR = 186 × [serum creatinine (mg/dL)]$^{-1.154}$ × [age]$^{-0.203}$ × [0.742 if female] × [1.210 if black] [10]. As only 37% of our study population had a payer source which would require them to fill their medications within the Denver Health system, we did not include concurrent prescribed medications in this analysis.

All creatinine measurements were performed on a Roche Hitachi Modular Analytics P model analyzer (Roche Diagnostics, Indianapolis, IN, USA) by the modified Jaffe reaction and were calibrated to the isotope dilution mass spectrometry (IDMS) traceable creatinine standard. All available creatinine measurements (both inpatient and outpatient) were included in the analysis. A second analysis including only creatinine measurements from outpatient visits was performed which demonstrated similar associations and is not reported here.

The primary outcome of interest, incident CKD, was defined using the National Kidney Foundation Disease Outcome Quality Initiative (NKF/DOQI) definition of eGFR <60 mL/min/1.73 m^2 persisting for at least 3 months [10]. We operationalized this definition as a final eGFR <60 mL/min/1.73 m^2 with at least one other eGFR 3 months prior <60 mL/min/1.73 m^2 and no intervening eGFR measurements ≥ 60 mL/min/1.73 m^2. The secondary outcome was the rate of change in eGFR per 6-month time interval, defined as slope of the linear relationship between eGFR and time.

Statistical analyses

We compared baseline characteristics of those who developed CKD to those who did not using *t* tests or Wilcoxon rank sum tests for continuous variables and chi-square or Fisher exact tests for dichotomous or categorical predictors. We used stepwise multivariable logistic regression analysis to identify predictors of the primary outcome (development of incident CKD). Logistic regression was chosen over a Cox model due to (i) high variability of the timing and number of creatinine measurements for each patient and (ii) exact timing that participants reached the renal endpoint of CKD could not be established. Covariates were considered candidates for inclusion in the model if the *P* value on bivariate testing was <0.25. To derive the final model, variables were excluded using backwards elimination until all *P* values were <0.25. Model discrimination was assessed with the *c*-statistic, and model calibration was assessed with the Hosmer–Lemeshow statistic.

We used general linear mixed-effects models (random slope and random intercept) to estimate the rate of decline in eGFR and the degree to which the baseline covariates predicted eGFR. Prior to calculating eGFR, all creatinine measurements in each 6-month block were averaged to smooth high within-patient variability [11]. Non-physiologic eGFR values of >200 mL/min/1.73 m^2 were set to a maximum value of 200 mL/min/1.73 m^2 for eGFR values after the baseline measurements for analyses examining rate of decline in eGFR [12]. Repeated measures within each patient were modeled as a linear time trend (growth curve) model. Time was coded into 6-month periods after baseline (e.g. if eGFR based on mean of creatinine measurements within the first 6 months, time = 0; if eGFR based on mean of creatinine measurements from 6 to 12 months, time = 1, etc.). All analyses were conducted with SAS Version 9.1 (SAS Institute, Cary, NC, USA).

Results

After excluding patients with <3 creatinine measurements, those with CKD at baseline or those who were in an indeterminate category (did not have both of their baseline eGFR measurements ≥60 mL/min/1.73 m^2) (Figure 1), a total of 10 420 patients with hypertension met our inclusion criteria. These individuals had a total of 134 983 creatinine measurements with a median of eight measurements [interquartile range (IQR) 5–15] and with a mean period of observation of 3.73 years (range 1–6.94 years). For the rate of change analysis, after averaging the creatinine values in 6-month time intervals, the median number of measurements per patient was 5 (interquartile range 3–7) (data not shown). The mean time in the hypertension registry was 62.4 months, and the mean duration of follow-up after the first creatinine measurement was 44.7 months. We had self-re-
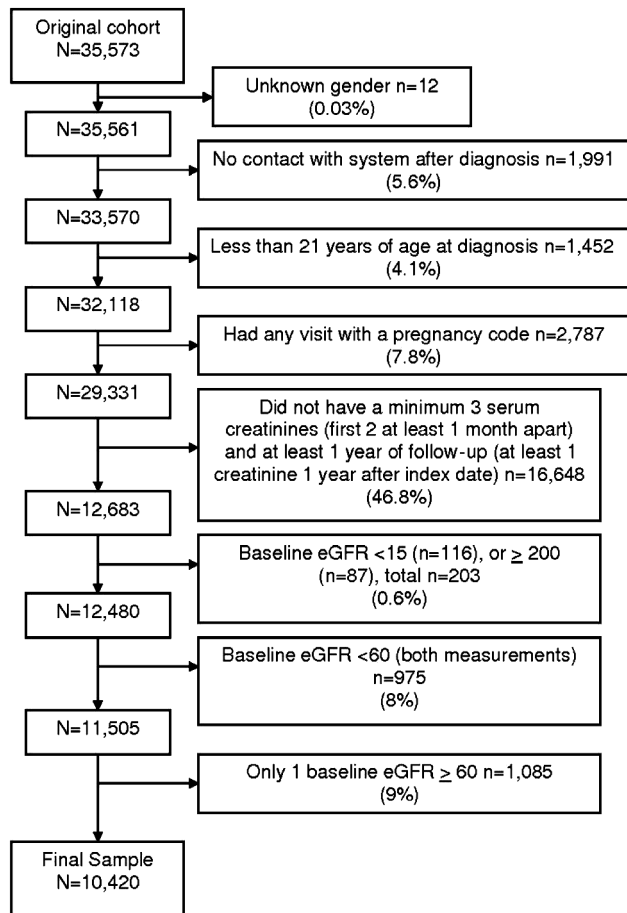
Fig. 1. Eligibility for incident CKD and GFR progression cohorts among patients included in the hypertension registry.

Table 1. Baseline characteristics

	Total n = 10420 n (%)
Age, years	55.0 (11.9)
Male sex	4672 (44.8%)
Race/ethnicity	2805 (26.9%)
African American	4556 (43.7%)
Hispanic	2722 (26.1%)
White	209 (2.0%)
Other	128 (1.2%)
Missing	
Marital status	3224 (30.9%)
Married	6467 (62.1%)
Other	729 (7.0%)
Unknown	
Language	7157 (68.7%)
English	1785 (17.1%)
Spanish	384 (3.7%)
Other	1094 (10.5%)
Missing	
Duration in hypertension registry, months	62.4 (19.2)
Period of observation, months	44.7 (20.1)
Comorbidities	
Diabetes	3822 (36.7%)
Vascular	1721 (16.5%)
Congestive heart failure	37 (0.4%)
Dyslipidemia	1766 (17.0%)
Mental health	2614 (25.1%)
Substance abuse	1666 (16.0%)
Baseline eGFR (mL/min/1.73 m^2)	98.7 (23.6)

Data are presented as mean (SD) or n (%). eGFR = estimated glomerular filtration rate.

ported income information for 68% of patients, who reported a median income of $7680 (IQR $4164–$12 992). Baseline characteristics of the whole cohort are shown in Table 1.

As shown in Table 2, 429 patients (4.1% of the study cohort) developed CKD. The results of the logistic regression for risk of incident CKD are shown in Table 3. Age (OR 1.13 per 10 years, 95% CI 1.03–1.24), baseline eGFR (0.69 per 10 units, 0.65–0.73), presence of diabetes (3.66, 2.97–4.51) and vascular disease (1.67, 1.32–2.10) independently predicted incident CKD (Table 3). The *c*-statistic for this model was 0.81, indicating good discrimination. Race and ethnicity were not independent predictors of incident CKD in this cohort. In order to test whether there was a systematic difference in patients who had insufficient creatinine measurements to meet the incident CKD definition, we performed a sensitivity analysis, requiring just a single final eGFR <60 mL/min/1.73 m^2 to define incident CKD. In this analysis, the incidence of CKD was higher (10.2%), but there was no significant difference in any of the associations in bivariate or multivariate testing.

Predictors of eGFR at baseline and of the change of eGFR over time are shown in Table 4. The intercept values represent the within sub-group difference in baseline eGFR. The slope values represent the rate of change of eGFR over a 6 month time interval, where a negative number represents a decline in kidney function. African American race, Hispanic ethnicity and presence of diabetes were associated with higher eGFR at baseline. Older age and female sex were associated with lower eGFR at baseline. However, none of these factors was independently associated with the eGFR slope.

Figures 2 and 3 represent model-based estimates of the eGFR slopes, created by using the intercepts, slopes and duration of observation from the models to construct the graphs. For patients who did not have diabetes or vascular disease at baseline, an eGFR decline of 1.52 mL/min/1.73 m^2 per year was observed (Figure 2). Presence of diabetes at baseline was associated with an additional decline of 1.38 mL/min/1.73 m^2 per year, resulting in an overall decline of 2.9 mL/min/1.73 m^2 per year. Although the presence of vascular disease alone at baseline was not significantly associated with a worsening decline in eGFR, the combination of both diabetes and vascular disease resulted in an additional decline of 0.62 mL/min/1.73 m^2 per year with an overall decline of 3.52 mL/min/1.73 m^2 per year (Figure 3).

In an attempt to assess the impact of blood pressure control on our findings, we conducted a secondary analysis in the 528 individuals (5.1% of the original cohort) who had blood pressure readings at the time of their entry into the cohort. In this small sub-group, baseline systolic blood

Table 2. Bivariate associations of baseline characteristics with incident CKD

	No CKD n = 9991 (95.9%) n (%)	Incident CKD n = 429 (4.1%) n (%)	P value
Age at time zero, years	54.8 (11.8)/54.3	59.8 (11.0)	<0.0001
Male sex	4510 (45.1%)	162 (37.8%)	0.003
Race/ethnicity	2696 (27.0%)	109 (25.4%)	
African American	4335 (43.4%)	221 (51.5%)	
Hispanic	2633 (26.4%)	89 (20.8%)	
White	201 (2.0%)	8 (1.9%)	
Other	126 (1.3%)	2 (0.5%)	0.008
Missing			
Marital status	3081 (30.8%)	143 (33.3%)	
Married	6208 (62.1%)	259 (60.4%)	
Other	702 (7.0%)	27 (6.3%)	0.51
Unknown			
Language	6866 (68.7%)	291 (67.8%)	
English	1707 (17.1%)	78 (18.2%)	
Spanish	367 (3.7%)	17 (4.0%)	
Other	1051 (10.5%)	43 (10.0%)	0.91
Missing			
Duration in hypertension registry, months	61.2 (19.2)	69.6 (16.8)	<0.0001
Period of observation, months	44.1 (20.0)	57.2 (18.6)	<0.0001
Comorbidities			
Diabetes	3539 (35.4%)	283 (66.0%)	<0.0001
Vascular	1606 (16.1%)	115 (26.8%)	<0.0001
Congestive heart failure	34 (0.3%)	3 (0.7%)	0.19
Dyslipidemia	1685 (16.9%)	81 (18.9%)	0.28
Mental health	2537 (25.4%)	77 (18.0%)	0.0005
Substance abuse	1623 (16.2%)	43 (10.0%)	0.0006
Baseline eGFR (mL/min/1.73 m^2)	99.3 (23.5)	84.2 (20.2)	<0.0001

Data are presented as mean (SD) or *n* (%). eGFR = estimated glomerular filtration rate; CKD = chronic kidney disease.

Table 3. Multivariate analysis of predictors of incident CKD

	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	Adjusted P value
Age at time zero (per 10 years)	1.42 (1.31–1.53)	1.13 (1.03–1.24)	0.008
Gender	Ref.		
Male	1.36 (1.11–1.66)		
Female			
Race/ethnicity	1.20 (0.90–1.59)		
African American	1.51 (1.17–1.94)		
Hispanic	Ref.		
White	0.91 (0.47–1.76)		
Other/missing			
Duration of hypertension, per year	1.37 (1.27–1.47)		
Period of observation, per 6 months	1.23 (1.19–1.27)	1.21 (1.17–1.25)	<0.0001
Baseline eGFR**, per 10 units	0.67 (0.64–0.72)	0.69 (0.65–0.73)	<0.0001
Comorbidities			
Diabetes	3.53 (2.88–4.33)	3.66 (2.97–4.51)	<0.0001
Vascular	1.91 (1.53–2.38)	1.67 (1.32–2.10)	<0.0001
Congestive heart failure	2.06 (0.63–6.74)		
Mental health	0.64 (0.50–0.83)		
Substance abuse	0.57 (0.42–0.79)		

OR = odds ratio; CI = confidence interval; eGFR = estimated glomerular filtration rate. *c*-statistic: 0.81. Hosmer–Lemeshow Goodness-of-Fit Test: $P = 0.01$.
**Baseline eGFR is the first measured eGFR after the diagnosis of hypertension.

pressure was not a statistically significant predictor of incident CKD. However, each increase of 10 mm Hg in systolic blood pressure resulted in a decline of 0.29 mL/min/ 1.73 m^2 in eGFR per 6-month period ($P = 0.09$). Inclusion of blood pressure in the multivariate models did not alter the findings of the main analysis.

Table 4. Coefficients of the intercepts and slopes of eGFR progression analysis

Variables	Intercept coefficient (SE)	P value
Variables that affect the intercept		
Intercept[a]	96.17 (.47)	<0.0001
Race/ethnicity		<0.0001
African American	4.05 (.54)	
Latino	2.96 (.49)	
Other race	−1.65 (1.15)	
Female gender	−2.73 (.40)	<0.0001
Age at baseline	−.58 (.02)	<0.0001
Diabetes	1.68 (.47)	0.0004
Vascular disease	.10 (.72)	0.89
Diabetes × vascular disease	−1.65 (1.11)	0.14
Variables that affect slope	**Slope coefficient[b] (SE)**	**P value**
Time[c]	−.76 (.04)	<.0001
Time × diabetes	−.69 (.06)	<0.0001
Time × vascular disease	−.09 (.10)	0.3556
Time × diabetes × vascular disease	−.31 (.16)	0.0464

[a]eGFR at baseline when covariates = 0 (e.g. white, male, age 55, no diabetes or vascular disease).
[b]Change in eGFR per 6 months.
[c]Slope at baseline when covariates = 0 (e.g. white, male, age 55, no diabetes or vascular disease).
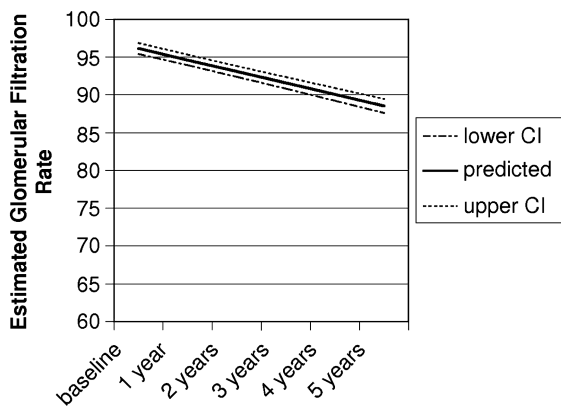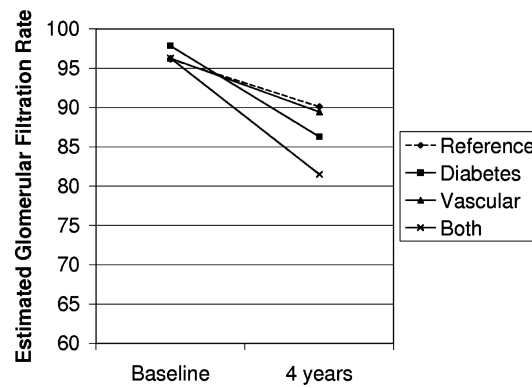

Fig. 2. eGFR change over time in patients without diabetes or vascular disease.

Fig. 3. eGFR progression for reference group (no diabetes or vascular disease), presence of diabetes, vascular disease, and both diabetes and vascular disease.

Discussion

Within a large community-based, racially and ethnically diverse sample of hypertensive patients with normal or near normal kidney function at baseline, diabetes was the strongest predictor of incident CKD and resulted in a more rapid decline in kidney function than in non-diabetics. In addition, the combination of diabetes and vascular disease at baseline was associated with even more rapid decline in eGFR than diabetes alone. Contrary to previous literature, we did not find differences in rate of decline in kidney function by race and ethnicity in a group of diverse patients with similar access to care and socioeconomic status.

In the current study, the rate of decline of kidney function in patients without diabetes or vascular disease at baseline was greater than the expected 1 mL/min/1.73 m^2 that is widely considered to be the "normal" decline in kidney function with age [10]. This decline is also greater than what was found in the Baltimore Longitudinal Study of Aging [6] (0.75 mL/min/year) in a European study of patients with existing CKD (1.03 mL/min/1.73 m^2 per year) [13] and an elderly Canadian community-based cohort with CKD at baseline (0.8 mL/min/1.73 m^2 for females and 1.4 mL/min/1.73 m^2 per year decline for males) [14]. However, slopes for diabetic patients were similar in our study to the slopes reported by Hemmelgarn and colleagues (2.9 mL/min/1.73 m^2 vs. 2.7 mL/min/1.73 m^2 per year, respectively) [14]. It is conceivable that the apparent discrepant results among the published studies [6,13,14] and our analysis are due to the fact that the patients included in this cohort were all hypertensive, had a higher burden of comorbidities and were of a lower socioeconomic status.

Two prior studies have assessed predictors of incident CKD. In an analysis of the Atherosclerosis Risk in Communities (ARIC) and the Cardiovascular Health Study (CHS) datasets, Kshirsagar et al. developed a prediction rule to predict incident CKD [15]. Contrary to our results, these investigators found white race to be associated with incident CKD. Other independent predictors of CKD in this study included age, female sex, diabetes, hypertension, cardiovascular disease, peripheral vascular disease, anemia, congestive heart failure and low high-density lipoprotein. This analysis was limited by (i) a shorter follow-up of the African American sub-group, (ii) using only a single eGFR <60 mL/min/1.73 m^2 to define incident CKD and (iii) failure to perform analysis on eGFR decline.

Fox et al. assessed a subset of the Framingham cohort with normal kidney function at baseline that attended two examinations 10 years apart [16]. Their primary outcome was development of CKD defined as a single eGFR <59.25 mL/min/1.72 m^2 for women and 64.25 mL/min/1.72 m^2 for men. In their multivariate model, they found age, presence of diabetes, BMI (body mass index), smoking, baseline eGFR and male sex to be associated with incident CKD. The study population was predominantly white, so they were unable to assess the effect of race/ethnicity on the development of CKD.

African Americans have an incidence of ESRD 3.6 times greater than non-Hispanic whites [3] although their prevalence of CKD in the USA is similar [1,17]. This discrepancy has been attributed to a faster decline in kidney function in African Americans with existing kidney disease [17]. Furthermore, Latino individuals represent the largest minority group in the USA and have 1.5 times higher rates of incident ESRD than non-Hispanic whites. However, data on rates of progression in this group are scarce [18]. To our knowledge, this is the first study to look at rate of decline of eGFR over time in a hypertensive, racially and ethnically diverse community-based sample with normal or near normal kidney function at baseline. Our findings that race or ethnicity is not associated with incident CKD or decline of eGFR from normal kidney function suggest that the increased incidence of ESRD observed in these populations is due to factors that occur after the onset of CKD. In this regard, Hsu *et al.* demonstrated that the incidence of CKD was not higher in African Americans when compared to whites but that African Americans with CKD presumably progressed to ESRD at five times the rate of whites with CKD [17]. Peralta *et al.* demonstrated that Latinos with CKD had nearly a 2-fold risk of developing ESRD when compared to whites with CKD [18]. Finally, differences observed between studies may not account for important differences in access to health care, the quality of care for individuals with hypertension or for important comorbid health conditions such as diabetes or vascular disease, which may accelerate the decline in kidney function.

An important strength of our study was our inclusion of all three major racial/ethnic groups in the USA. Another advantage of our study was the requirement that the development of CKD be persistent for at least 3 months (NKF/DOQI definition of CKD). In addition, we included an assessment of the rate of decline of eGFR that has not been previously reported for individuals with normal kidney function at baseline. Thus, assessing progression by rate of change of eGFR over time allows for more complete assessment of the risk factors for progression [19], not just those who achieve the dichotomous primary outcome of incident CKD.

This study has a number of limitations. First, we relied on estimating GFR from the MDRD study equation rather than using a direct method of measuring the GFR. Our study relied on laboratory values measured in routine clinical care so that a direct measure of GFR (mGFR) was not possible. In addition, the MDRD study equation does not perform as well at higher levels of GFR [20] and may have resulted in inaccuracy in classifying patients. Despite these concerns, the MDRD estimating equation is recommended for use by the NKF/DOQI guidelines and is the most widely used prediction equation in clinical practice. In addition, because estimating equations are derived from cross sectional data, there is some concern about the ability of estimating equations to assess changes in GFR over time. Xie *et al.* evaluated the performance of the MDRD study equation to assess eGFR slope when compared to a direct mGFR. They found that the MDRD eGFR underestimated the slope decline by 28%, but none of the tested clinical variables predicted a systematic difference between mGFR

and eGFR slope [21]. Therefore, using the MDRD equation may have led us to underestimate the eGFR slope, but it was unlikely to affect the predictors associated with more rapid decline in GFR.

Second, the use of creatinine values measured in routine clinical care can result in systematic bias. Patients with more comorbidities are more likely to be engaged in the medical system and may have more frequent laboratory monitoring than their healthier counterparts. In addition, patients who were demonstrating a decline in kidney function may have received more measurements of serum creatinine. Third, we were unable to assess the severity of hypertension in our patients in our registry due to incomplete pharmacy data and limited blood pressure data. Other limitations include inability to control for use of antihypertensive medications, variability of duration of follow-up within the cohort and restriction of our analysis to patients with hypertension which did not allow assessment of the impact of hypertension on the development of incident CKD as we did not have a comparator group without hypertension. It also likely led to estimates of eGFR slopes that are steeper (more negative) than one would expect for patients without hypertension.

In conclusion, we found diabetes to be the strongest predictor of both incident CKD and eGFR decline in a diverse cohort of hypertensive patients from an inner-city, integrated delivery system. In addition, we found no difference by race/ethnicity in rates of incident CKD or in decline of kidney function. The presence of vascular disease at baseline in patients with diabetes was additive. Our findings suggest that efforts to prevent the development of CKD should focus on awareness of risk factors for diabetes and vascular disease. Further work on preventing the development of and slowing the progression of kidney disease in hypertensive patients is necessary.

Conflict of interest statement. The authors report no financial conflicts of interest.

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Results of surgical treatment for renovascular hypertension in children: 30-year single-centre experience

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Abstract

Background. We retrospectively reviewed the medical records of all patients who underwent surgery as part of the treatment of renovascular hypertension (RVH) at our centre between 1979 and 2008.

Patients. Thirty-seven children (65% male) with a median age of 7.6 (0.4–17.9) years were identified with a median systolic blood pressure (SBP) of 140 (105–300) mm Hg prior to surgery. Bilateral renal artery stenosis and intra-renal disease were present in 19 (51%) patients, mid-aortic syndrome in 15 (40%), involvement of visceral arteries in eight out of 35 (23%) and coexisting cerebral disease in eight out of 30 (26%) investigated patients.

Results. Surgical procedures (*n* = 53) included (i) nephrectomy (18, of which two unplanned and two secondary due to technical failure), (ii) renovascular surgery on the renal arteries (28, of which 18 had autologous surgery and 10 synthetic grafts inserted for revascularisation) and (iii) aortic reconstruction with (6) and without (1) a synthetic graft. Post-operative complications were haemorrhage (5), septicaemia (5) and chylous ascites (1). There were no perioperative deaths; two children died during follow-up. The SBP post-surgery improved to a median value of 116 (range 90–160) mm Hg. Twelve months after surgery, 16 (43%) children had normal blood pressure without treatment, 15 (41%) normal or improved on one to four antihypertensive drugs and four (11%) unchanged; no data were available for two (5%) children.

Conclusion. Surgery effectively treated the hypertension of 90% of our children, when performed in conjunction with medical therapy and interventional radiology. In spite of aggressive surgical treatment, RVH is sometimes a progressive disease.

Keywords: bypass; children; graft; nephrectomy; renovascular hypertension

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