Vascular Stiffness in Children With Chronic Kidney Disease

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See Editorial Commentary, pp 791–794

Abstract—Carotid-femoral pulse wave velocity (cfPWV) is a measure of arterial stiffness associated with cardiovascular events in the general population and in adults with chronic kidney disease. However, few data exist regarding cfPWV in children with chronic kidney disease. We compared observed cfPWV assessed via applanation tonometry in children enrolled in the CKiD cohort study (Chronic Kidney Disease in Children) to normative data in healthy children and examined risk factors associated with elevated cfPWV. cfPWV Z score for height/gender and age/gender was calculated from and compared with published pediatric norms. Multivariable linear regression was used to assess the relationship between cfPWV and age, gender, race, body mass index, diagnosis, urine protein–creatinine ratio, mean arterial pressure, heart rate, number of antihypertensive medications, uric acid, and serum low-density lipoprotein. Of the 95 participants with measured cfPWV, 60% were male, 19% were black, 46% had glomerular cause of chronic kidney disease, 22% had urine protein–creatinine ratio 0.5 to 2.0 mg/mg and 9% had >2.0 mg/mg, mean age was 15.1 years, average mean arterial pressure was 80 mm Hg, and median glomerular filtration rate was 63 mL/min per 1.73 m^2. Mean cfPWV was 5.0 m/s (SD, 0.8 m/s); mean cfPWV Z score by height/gender norms was −0.1 (SD, 1.1). cfPWV increased significantly with age, mean arterial pressure, and black race in multivariable analysis; no other variables, including glomerular filtration rate, were independently associated with cfPWV. In this pediatric cohort with mild kidney dysfunction, arterial stiffness was comparable to that of normal children. Future research is needed to examine the impact of chronic kidney disease progression on arterial stiffness and associated cardiovascular parameters in children. **(*Hypertension*. 2017;69:863-869. DOI: 10.1161/HYPERTENSIONAHA.116.07653.)**

Key Words: arteriosclerosis ■ chronic kidney disease ■ pediatrics ■ pulse wave velocity ■ vascular stiffness

Arterial stiffness is associated with cardiovascular events and mortality in otherwise healthy and hypertensive adult populations.[1,2] Models incorporating pulse wave velocity (PWV), a measure of central arterial stiffness, suggest that 1 SD increment in PWV is equal to ≈10 years of aging.[3] In adults with chronic kidney disease (CKD), arterial stiffness is increased compared with healthy adults,[4] is associated with cardiovascular events and mortality,[5] and in many reports, is inversely related with level of kidney function.[6–8] There is emerging literature describing arterial stiffness in pediatric populations[9] and the relation of arterial stiffness to pediatric CKD, but this research has primarily focused on end-stage renal disease.[10–14] Recently, in a single-center study of children with predialysis CKD, Sinha et al[15] demonstrated that PWV did not differ by level of glomerular filtration rate (GFR), nor did it vary from PWV in healthy control children.

Assessment of arterial stiffness in children with mild to moderate CKD is important to understand the interactions between early CKD and the cardiovascular system, potentially identifying high-risk populations that could benefit from targeted intervention. Hence, the aims of this study were to measure carotid-femoral pulse wave velocity (cfPWV) via applanation tonometry in a North American cohort of children with mild to moderate CKD; to compare these values to published reference ranges in healthy children; and to examine risk factors associated with elevated cfPWV in children with CKD, particularly measured GFR.

Methods

Study Population

Participants were children with mild to moderate CKD enrolled in the CKiD study (Chronic Kidney Disease in Children), a multicenter, prospective, observational cohort study conducted in North America. The design and methods of the CKiD study have been described elsewhere.[16] Briefly, eligible participants were 1 to 16 years old at baseline, with an estimated GFR between 30 and 90 mL/min per 1.73 m^2 via the Schwartz formula (cohort 1) or between 45 and 90 mL/min per 1.73 m^2

Received October 15, 2016; first decision November 12, 2016; revision accepted February 08, 2017.

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Presented in part at the American Society of Nephrology Kidney Week, San Diego, CA, November 3–8, 2015.

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via the CKiD formula (cohort 2). The collection of cfPWV was added to the study protocol in 2013 at a subset of sites with access to the necessary equipment and software. All subjects provided informed consent/assent according to local requirements, and the study received institutional review board approval at participating sites.

Measurements and Data Collection

Arterial stiffness was assessed by cfPWV via applanation tonometry using a SphygmoCor device (AtCor Medical, Inc, Australia), software version 9. All operators were trained in the collection of cfPWV and were certified by a qualification process prior to collecting study data. The qualification process involved the submission of 3 studies each on 3 test patients satisfying quality control parameters built into the software and within-between subject reproducibility statistics (coefficient of variation <10%). Qualification was provided after physician review (K.E.C. Meyers). Similarly, all incoming study data were evaluated for satisfaction of quality control parameters, and operators submitting deficient data on 2 consecutive study patients were required to repeat the qualification process on test patients.

cfPWV was collected 3× per participant. Prior to cfPWV, patients were placed in the supine position and rested for 5 minutes. Three electrocardiographic leads were attached. The right carotid artery was palpated and marked, and the distance between the carotid pulse site and the suprasternal notch was measured twice to the nearest millimeter and recorded in the software (proximal site). This procedure was repeated for the right femoral artery (distal site). For the distal site, distance between the right femoral artery and suprasternal notch was measured directly with the measuring tape against the skin as suggested by Weber et al.[17] The pulse was then captured for 10 seconds at the proximal and distal sites using a Millar tonometer. If the pulse acquisition site differed from the palpated site previously marked, the distance measurements were repeated and re-entered into the software. To assist with pulse acquisition, real-time feedback via color-coded visual guidance bars built-in to the software was displayed on the laptop screen, and autocapture of results occurred after 10 seconds of quality data were obtained.

A cfPWV Z score normalized separately by height and gender and by age and gender was calculated using published pediatric reference data.[18] Reusz et al[18] assessed cfPWV via applanation tonometry in over 1000 children and adolescents and provided formulas using the least mean squares method to calculate Z score. Although the published least mean squares formulas were provided for age ranges 7 to 19 years, inclusive, and for height ranges 120 to 195 cm (males) and 115 to 180 cm (females), several of our participants were outside of these age (N=11) or height (N=4) ranges. To calculate Z scores for all of our participants, we extended the least mean squares formula for 7-year-olds to participants <7 years and the formula for 19-year-olds for participants >19 years. Similarly, we extended the least mean squares formula for males with a height of 120 cm to male participants shorter than 120 cm and the formula for females with a height of 180 cm to female participants taller than 180 cm. To see if these assumptions impacted our results, we also calculated cfPWV Z score, excluding those outside the age or height ranges provided by Reusz et al.[18]

Casual blood pressure (BP) was reported as the average of 3 measurements collected in the sitting position at 30-second intervals by trained and annually recertified operators via auscultation using an aneroid sphygmomanometer, as previously published.[19] Given that both BP and cfPWV were collected by trained and certified operators using a standard protocol, these measurements may not always have been collected by the same person or in immediate succession. Systolic BP (SBP) and diastolic (DBP) percentiles were standardized by age, sex, and height following the Fourth report on the diagnosis, evaluation, and treatment of high BP in children and adolescents.[20]

Mean arterial pressure (MAP) was determined using the SphygmoCor device during an assessment of pulse wave analysis, performed immediately before or after cfPWV, using applanation tonometry of the radial pulse. MAP is determined by measuring the area under the radial pressure waveform curve taking into account the length of the cardiac cycle calibrated by the SBP and DBP measurements. For participants lacking an available radial waveform (N=5), MAP was estimated from the casual BP using the formula (DBP+[SBP–DBP]/3).

Heart rate was recorded by the SphygmoCor device during tonometric acquisition of the carotid pulse during cfPWV. Height and weight were collected using a calibrated stadiometer and scale, respectively, and reported as the average of 2 measurements. Age–sex-specific height Z score was calculated using the 2000 Centers for Disease Control standard growth charts for US children.[21]

GFR was measured by plasma disappearance of iohexol.[22] Where a measured GFR was not available (N=4), GFR was estimated using equations developed by the CKiD study.[23] Urine protein and creatinine were analyzed by standard laboratory methods from a first morning urine sample and reported as a urine protein-to-creatinine ratio. Other laboratory measures included in the analysis were triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, calcium, phosphate, uric acid, and serum glucose. Where a fasting serum glucose was not available (N=9), a nonfasting serum glucose was used.

Demographic information was collected at study entry. Participants of Hispanic ethnicity were categorized as Hispanic; otherwise, participants were categorized as white, black, or other. Length of time with CKD was collected by self-report. Specific CKD diagnosis was collected at baseline to confirm study eligibility. As done previously, participants were categorized as having a CKD diagnosis of a glomerular origin (eg, chronic glomerulonephritis or focal segmental glomerulosclerosis) or nonglomerular origin (eg, obstructive uropathy or dysplastic kidney).

Statistical Analyses

Clinical and demographic characteristics of the study participants were summarized using mean±SD or median and interquartile range for skewed continuous data among included and excluded participants; differences were tested using Student's *t* test or Wilcoxon rank-sum test. Categorical variables were expressed as frequencies and percentages and compared using Chi-square or Fisher exact tests, as appropriate. The main outcome, mean cfPWV, was used as a continuous variable. Univariable regression models for cfPWV were used to assess the relation between cfPWV and various demographic and clinical variables previously described in the literature as related to PWV (age and MAP) or to CKD. Along with age and gender, all covariates with a P value <0.20 were then included in the adjusted model, except where determined to be highly collinear. The final multivariable linear regression model included age, gender, race, body mass index, diagnosis, urine protein–creatinine ratio, MAP, heart rate, number of antihypertensive medications, uric acid, and serum low-density lipoprotein. All analyses were performed using SAS software version 9.4 (SAS Institute, Inc., Cary, NC). A P value <0.05 was considered statistically significant.

Results

The number of eligible participants and description of excluded participants are summarized in Figure. Demographic and clinical characteristics of the 95 included participants and the 30 excluded participants are provided in Table 1. For included participants, the mean age was 15.1 years; 60% were male and 19% were black. Glomerular diseases accounted for almost half of the underlying diagnoses; median GFR was 63.1 mL/min per 1.73 m^2 (interquartile range, 42.9–78.5). There were 23 participants with CKD stage 1, 29 with stage 2, 15 with stage 3A, 25 with stage 3B, and 3 with stage 4. Mean cfPWV was 5.0 m/s (SD, 0.8 m/s). Mean cfPWV Z scores compared with published reference ranges with healthy children based on height/gender and age/gender were both −0.1 (SD, 1.1). Mean cfPWV Z scores were unchanged if we included only those 84 participants within the age range (Z score, −0.10;

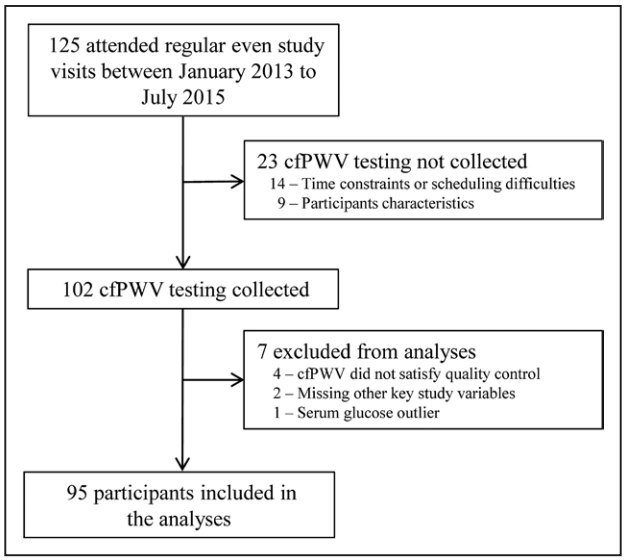

Figure. Flowchart for inclusion of participants. cfPWV indicates carotid-femoral pulse wave velocity.

SD, 1.1) and 91 participants within the height range (Z score, −0.06; SD, 1.1) of the Reusz et al[18] formulas.

The univariable analysis with cfPWV as the dependent variable is shown in Table 2. cfPWV was significantly associated with age, race, height, body mass index, waist circumference, glomerular diagnosis, urine protein–creatinine ratio, SBP percentile, MAP, and uric acid. Results from the multivariable analysis (Table 3) revealed cfPWV had significant, independent associations with age, MAP, and race.

One participant had a measured cfPWV value above the third quartile plus 1.5 the interquartile range. In a subsequent analysis, this point of high influence was excluded from the regression models. In this analysis, the independent associations with age and MAP persisted. The point estimate for the association with black race was still large, but the significance of the association with race was lessened. The estimate of the association between black race and cfPWV was modified from 0.45±0.21 (P=0.03) to 0.23±0.19 (P=0.24) when this 1 point was removed from the analysis. Given that the cfPWV data collected for this 1 participant satisfied all quality control parameters and was a physiologically plausible value (8.2 m/s), we did not exclude it from our analyses.

Discussion

In this study, we assessed central arterial stiffness via cfPWV using applanation tonometry in a well-characterized cohort of children with mild to moderate CKD. The assessment of arterial stiffness in children with early CKD is important because arterial stiffness may lead to left ventricular hypertrophy and left ventricular dysfunction, both of which are widely reported in pediatric CKD.[24,25] Our analysis demonstrated that cfPWV was independently and significantly associated with increasing age and MAP, both of which are well-known primary determinants of PWV,[7,26] as well as black race, a novel finding in children with early CKD. Our study serves to confirm the findings of Sinha et al,[15] namely that that the cfPWV of

Table 1. Baseline Characteristics of Study Participants

Patient Characteristics	Included (N=95)	Excluded (N=30)	*P* Value
Age, y	15.1±3.7	13.1±4.4	0.01
Male	60.0 (57)	70.0 (21)	0.32
Race/ethnicity			0.34
White	66.3 (63)	50.0 (15)	
Black	19.0 (18)	26.7 (8)	
Hispanic	8.4 (8)	10.0 (3)	
Other	6.3 (6)	13.3 (4)	
Height, cm	162.6 (147.8, 170.4)	153.4 (135.6, 163.3)	0.01
Height Z score	−0.2±1.2	−0.5±1.2	0.20
BMI, kg/m^2	20.9 (18.8, 25.1)	18.5 (16.5, 25.5)	0.21
Waist circumference, cm	74.0±16.0	75.0±23.0	0.97
Diagnosis (% glomerular)	46.3 (44)	26.7 (8)	0.06
GFR, mL/min per 1.73 m^2	63.1 (42.9, 89.0)	64.7 (46.9, 78.5)	0.75
GFR stage			0.02
Stage 1 (≥90)	24.2 (23)	3.3 (1)	
Stage 2 (60–89)	30.5 (29)	60.0 (18)	
Stage 3A (45–59)	15.8 (15)	13.3 (4)	
Stage 3B (30–44)	26.3 (25)	20.0 (6)	
Stage 4 (15–29)	3.2 (3)	3.3 (1)	
Urine protein–creatinine, mg/mg*			0.52
<0.5	69.2 (63)	72.4 (21)	
0.5–2.0	22.0 (20)	13.8 (4)	
>2.0	8.8 (8)	13.8 (4)	
Systolic blood pressure percentile	44.1 (18.8, 68.6)	53.6 (26.5, 85.8)	0.21
Diastolic blood pressure percentile	57.1 (32.0, 77.7)	53.3 (33.0, 79.8)	0.51
Mean arterial pressure, mm Hg	80.2±9.7	83.9±9.5	0.07
Heart rate, bpm	70.0 (64.0, 78.0)	…	
Number of antihypertensive medications*			0.04
0	22.0 (20)	5.0 (1)	
1	60.4 (55)	60.0 (12)	
2	15.4 (14)	20.0 (4)	
3+	2.2 (2)	15.0 (3)	
Triglycerides, mg/dL	90.0 (68.0, 131.0)	97.0 (65.0, 145.0)	0.82
LDL, mg/dL	86.5 (72.0, 113.0)	85.5 (70.0, 106.0)	0.79
HDL, mg/dL	54.5 (45.0, 66.0)	54.0 (44.0, 62.0)	0.40

(*Continued*)

Table 1. Continued

Patient Characteristics	Included (N=95)	Excluded (N=30)	*P* Value
Calcium×phosphate, mg^2/dL^2	38.3 (33.3, 42.7)	40.6 (32.2–45.9)	0.23
Uric acid, mg/dL	6.5±1.6	6.3±1.5	0.64
Serum glucose, mg/dL	87.0 (81.0, 92.0)	90.5 (87.0, 95.0)	0.01
cfPWV, m/s	5.0±0.8	…	
cfPWV Z score by age/gender	–0.1±1.1	…	
cfPWV Z score by height/gender	–0.1±1.1	…	
Length of time with CKD, y	10.6 (3.8, 15.2)	12.2 (7.8, 15.5)	0.22

Data are means±SD, medians (25th, 75th percentiles) or n (%). BMI indicates body mass index; cfPWV, carotid-femoral pulse wave velocity; CKD, chronic kidney disease; GFR, glomerular filtration rate; HDL, high-density lipoproteins; and LDL, low-density lipoproteins.

*Included N=91.

Table 2. Outcome cfPWV: Univariable Analysis

Patient Characteristics	Estimate±SE	*P* Value
Age, y	0.10±0.02	<0.0001
Male	–0.16±0.17	0.35
Black	0.47±0.21	0.03
Height, cm	0.02±0.00	<0.001
BMI, kg/m^2	0.05±0.01	0.001
Waist circumference, cm	0.01±0.01	0.03
Glomerular diagnosis	0.38±0.17	0.03
GFR, per 10 mL/min per 1.73 m^2	–0.00±0.03	0.96
Urine protein–creatinine ratio, mg/mg		
<0.5	Reference	Reference
≥0.5	0.38±0.19	0.05
Systolic blood pressure percentile	0.01±0.00	0.02
Diastolic blood pressure percentile	0.00±0.00	0.14
Mean arterial pressure, mm Hg	0.03±0.01	0.001
Heart rate, bpm	–0.01±0.01	0.12
Number of antihypertensive medications		
0	Reference	Reference
1	0.34±0.21	0.11
≥2	0.46±0.28	0.10
Triglycerides, per 10 mg/dL	0.01±0.02	0.40
LDL cholesterol, per 10 mg/dL	0.03±0.02	0.14
HDL cholesterol, per 10 mg/dL	0.05±0.05	0.26
Calcium×phosphate, mg^2/dL^2	–0.01±0.01	0.60
Uric acid, mg/dL	0.12±0.05	0.03
Serum glucose, per 10 mg/dL	0.05±0.09	0.57
Length of time with CKD	0.01±0.01	0.71

BMI indicates body mass index; cfPWV, carotid-femoral pulse wave velocity; CKD, chronic kidney disease; GFR, glomerular filtration rate; LDL, low-density lipoproteins; HDL, high-density lipoproteins; and SE, standard error.

children with early CKD was not significantly different from healthy children nor was related to GFR, and also extends these findings to a North American cohort using applanation tonometry and measured GFR.

There are several different methods to measure arterial stiffness, the most commonly recommended is cfPWV via applanation tonometry.[8] cfPWV is a simple, noninvasive assessment of functional stiffness of the central arterial system and is the gold standard because of its ability to predict future cardiovascular events and agreement with invasive measures in adults (eg, cardiac catheterization), although comparison to direct invasive measures in children has not been performed.[8,27] cfPWV can also be measured using an oscillometric device (cuff-based) instead of using applanation tonometry (transducer-based). Proponents of the oscillometric method cite that it is quicker and easier to perform and may be better tolerated by children. One study comparing different methods of collecting cfPWV in children found that tonometric assessment failed in 22% of the 156 participants, although the timing of the procedure (conducted after oscillometric assessment) may have contributed to this high failure rate.[28] Although our study did have missing cfPWV in 18% of eligible participants, only 7% were because of failure of tonometric acquisition (Figure). However, multiple reports have concluded that PWV values obtained from oscillometric and tonometric devices are not interchangeable,[28,29] and our group decided to use tonometric cfPWV because of its extensive prior use in clinical research and its endorsement as method of choice by consensus bodies.[7,27]

We observed that the cfPWV in our cohort was comparable to published reference ranges for healthy children standardized by height and gender and separately by age and gender. Our study population had relatively preserved kidney function, evidenced by a median GFR of 63 mL/min per 1.73 m^2. In addition, only 9% had a urine protein–creatinine ratio >2.0 mg/mg, and BP was seemingly well controlled, with 78% of participants being prescribed at least 1 antihypertensive medication and median SBP and DBP percentiles of 44% and 57%, respectively. Similarly, our study population had a relatively large proportion of children with glomerular cause of CKD with shorter disease duration and, hence, less exposure to reduced GFR, elevated BP, and other factors that may contribute to increased PWV. Given the evidence supporting elevated PWV in pediatric end-stage renal disease populations,[10–14] our results are consistent with the concept that increased arterial stiffness becomes more prominent as CKD progresses toward end-stage renal disease.

Several studies conducted in adults with CKD have shown an association between increased PWV and decreased GFR.[4,7] Fewer data exist on arterial stiffness in children with CKD. A recent study by Sinha et al[15] compared oscillometric cfPWV in 188 children aged 2 to 18 years with CKD stages 1, 2, 3, 4, and 5 (26%, 25%, 30%, 16%, and 3%, respectively) with 38 age- and BP-matched healthy controls. Similar to our results, the

Table 3. Adjusted Associations Between cfPWV and Selected Factors

Patient Characteristics	Estimate±SE	*P* Value
Age, y	0.08±0.03	0.004
Male	0.07±0.18	0.68
Black	0.45±0.21	0.03
Glomerular diagnosis	0.26±0.17	0.14
Urine protein–creatinine ratio (mg/mg)		
<0.5	Reference	Reference
≥0.5	−0.06±0.19	0.74
BMI, kg/m^2	−0.00±0.02	0.87
Mean arterial pressure, mm Hg	0.02±0.01	0.01
Heart rate, bpm	−0.00±0.01	0.91
Number of anti-HTN medications		
0	Reference	Reference
1	0.10±0.20	0.63
≥2	0.29±0.27	0.27
LDL cholesterol, per 10 mg/dL	0.00±0.03	0.88
Uric acid, mg/dL	0.05±0.05	0.38

BMI indicates body mass index; cfPWV, carotid-femoral pulse wave velocity; LDL, low-density lipoproteins; and SE, standard error.

authors found that cfPWV values did not differ between those with CKD and healthy controls (5.3±0.9 versus 5.3±0.8 m/s, respectively) and were not significantly related to estimated GFR.[15] While Sinha et al[15] did observe differences in other measures of arterial stiffness, such as carotid augmentation index and circumferential wall stress, between CKD patients with suboptimal BP (defined as ≥75th percentile) and normotensive controls, this relationship did not persist for cfPWV. A study by Patange et al[30] found that radial augmentation index was inversely proportional to estimated GFR and was worse in patients with any CKD as compared with healthy controls. Although Patange et al[30] used a surrogate measure of arterial stiffness and had relatively few patients with mild to moderate CKD, these results suggest that arterial stiffness worsens with kidney disease progression in children. Similarly, in a study by Dursun et al,[31] children on dialysis had higher Doppler-ultrasound aortic PWV as compared with nondialysis CKD patients, while nondialysis CKD patients were similar to healthy controls.

We also found that black race, which was significantly associated with elevated cfPWV in univariable analysis, retained significance after controlling for other factors in multivariable analysis. Several reports have suggested that arterial stiffness may be worse in blacks compared with whites in healthy adults and adults with CKD,[8,32] but there are few data regarding cfPWV in healthy black children, and to our knowledge, whether this relationship exists in children with CKD has not been described. Because the number of black participants in our study was small, these findings will need to be explored further in other studies. Given the increased burden of cardiovascular disease among black Americans as compared with whites,[33] the interaction between race and the progression of arterial stiffness and kidney disease will be important to further delineate in future research.

We did not find that the number of prescribed antihypertensive medications was significantly related to cfPWV. This may suggest that BP control was generally similar across our study population, regardless of the number of prescribed antihypertensive medications. Finally, to further explore the impact of GFR on cfPWV, we performed an additional limited regression analysis looking at cfPWV across different CKD stages, with adjustment for age, race, body mass index, and MAP. Similar to our primary model, the major covariates significantly associated with cfPWV were age, black race, and MAP, with no significant relation to CKD staging.

Our study has several potential limitations. First, our study is cross-sectional and, thus, represents a snapshot of arterial stiffness. The prospective design of the CKiD study will allow us to perform longitudinal assessments of cfPWV in the cohort, which could help elucidate changes in arterial stiffness as kidney function declines or as exposure to CKD increases. Second, participants enrolled at cfPWV participating centers of the CKiD study may differ from the general population of children with CKD. However, the demographic characteristics and distribution of underlying diagnoses is similar to the typical North American pediatric end-stage renal disease population as reported by the United States Renal Data System.[34] Third, despite our efforts to standardize cfPWV collection procedures and the intensive qualification process, the presence of multiple operators at different clinical sites may bias the results. Similarly, given the different certification process for the collection of BP and cfPWV possibly necessitating 2 different operators at a given clinical site, these separate measurements may not have been performed in immediate succession. Finally, we measured cfPWV using a SphygmoCor device and compared these results to normative data collected using a PulsePen device.[18] Although some authorities suggest that results are comparable between these 2 applanation tonometric devices,[29] we cannot exclude that there were additional problems related to comparability. Despite these limitations, our analysis is strengthened by our relatively large sample size, a diverse cohort with a mixture of glomerular and nonglomerular CKD diagnoses, a measurement of kidney function using plasma disappearance of iohexol (as opposed to GFR estimating equations), and systematically collected cardiovascular and laboratory parameters.

Perspectives

This study represents an examination of children enrolled in a national cohort with mild to moderate CKD using cfPWV, an understudied topic with implications for the treatment of childhood CKD. Our initial cross-sectional findings provide some assurance that early CKD is not characterized by substantial arterial stiffness in children. We also found in our analysis that black race was associated with increased arterial stiffness. This should be examined in future studies. The longitudinal nature of CKiD should help us determine the effects of aging, ongoing CKD, and worsening of kidney function on arterial stiffness in children with CKD.

Sources of Funding

Funding was provided by the National Institute of Diabetes and Digestive and Kidney Diseases, the Eunice Kennedy Shriver National Institute of Child Health and Human Development, and the National Heart, Lung, and Blood Institute (U01-DK-66143, U01-DK-66174, U01DK-082194, U01-DK-66116).

Disclosures

None.

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Novelty and Significance

What Is New?

- We examined arterial stiffness using carotid-femoral pulse wave velocity by applanation tonometry in a nationally representative North American sample of children with mild to moderate chronic kidney disease and a measured glomerular filtration rate.

What Is Relevant?

- We found that arterial stiffness was not significantly related to measures of kidney function in children with mild to moderate chronic kidney disease and was not increased when compared with data collected in healthy children.

Summary

Carotid-femoral pulse wave velocity was significantly associated with higher age and mean arterial pressure, as well as black race in multivariable analysis. The impaired arterial stiffness seen in children with end-stage renal disease was not evident in our sample of children with mild to moderate chronic kidney disease, suggesting that these vascular changes occur later in the disease process.

Hypertension
Journal of the American Heart Association

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Hypertension. 2017;69:863-869; originally published online April 3, 2017;
doi: 10.1161/HYPERTENSIONAHA.116.07653
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0194-911X. Online ISSN: 1524-4563