

Arterial stiffness in black African ancestry patients with chronic kidney disease living in Cameroon

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Background: Increased aortic pulse wave velocity (PWV), a direct measure of arterial stiffness (AS) is an independent predictor of cardiovascular events (CVEs) in chronic kidney disease (CKD) patients. This study assessed the patterns of PWV among Cameroonian patients with CKD in whom that marker of early vascular aging has not been explored so far.

Methods: We enrolled 150 Black African patients (mean age: 52±15 years, 56.7% males) with CKD in a cross-sectional study conducted at Douala General Hospital, Douala, Cameroon. Sociodemographic, anthropometric and biologic variables, blood pressure (BP) and PWV were recorded in all participants. Estimated aortic PWV was measured using a Mobil-O-Graph automatic brachial oscillometric device.

Results: PWV increased with aging ($P<0.0001$), and PWV adjusted for age, sex, body mass index and mean arterial BP (MAP) was higher in non-dialysed ($n=90$) than in hemodialysed ($n=60$) patients, even in pre-dialysis: 8.5 ± 2.0 vs. 7.9 ± 1.4 m/s ($P=0.026$); and in post-dialysis: 8.5 ± 2.0 vs. 7.8 ± 1.5 m/s ($P=0.008$). The mean PWV of all study participants was 8.2 ± 1.8 m/s, with 61.3% of patients having a PWV ≥ 8.2 m/s, indicative of subclinical damage to the aorta, which was more pronounced in non-dialysis (67.8%) than in hemodialysis (53.3%) patients ($P=0.033$). Multivariable analysis performed in all participants revealed that advanced age, MAP and tobacco use were independently associated with PWV (all $P<0.05$).

Conclusions: Our findings suggest increased AS in Cameroonian CKD non-dialyzed as compared to dialyzed patients. Slower PWV in patients on maintenance hemodialysis suggests improvement of aortic distensibility following dialysis. However, further large-scale studies are needed to confirm our findings and to improve understanding of the underlying mechanisms of arterial stiffening in black African ancestry patients with CKD.

Keywords: Arterial stiffness (AS); chronic kidney disease (CKD); hemodialysis; Mobil-O-graph; pulse wave velocity (PWV); Cameroon

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Introduction

Chronic kidney disease (CKD) is a growing worldwide public health challenge and represents a major cause of death, with the fastest increase over the last two decades (1)]. Among the determinants of high mortality rate in CKD patients, cardiovascular disease (CVD) accounts for up to 50% of all deaths (1,2). The high burden of CVD in CKD patients was ascribed to both conventional cardiovascular (CV) risk factors such as age, hypertension, diabetes, obesity, smoking, and dyslipidemia; and non-traditional or emerging risk factors which include arterial stiffness (AS) (3). Structural and functional changes in arteries are important features of CVD (4). The process of CV damage starts very early during CKD progression, long before end-stage renal disease (ESRD) is reached (1). Increased AS corresponds to damage of large arteries and was shown to be a significant predictive factor of all-cause and CV mortality in different populations (5), including patients with ESRD (3,6-8). Moreover, increased pulse wave velocity (PWV), which is the gold standard measure of AS (9), was shown to contribute to all-cause (5,7) and CV mortality in ESRD patients (6-8). PWV is linked to arterial wall structure and function, and is essentially influenced by age-related changes, blood pressure (BP) and other pathologic states such as CKD (4,6-8). Several mechanisms including those related to CKD have been suggested to be involved in accelerated vessel stiffening (3,8,10-13). Identified factors associated with increasing PWV in non-dialysed CKD patients are age, black ethnicity, male gender, hypertension and diabetes mellitus (13-16). Evidence indicates that stiffening of the aorta may decrease in patients on maintenance dialysis (17).

Despite the growing burden of CKD faced by many sub-Saharan African (SSA) countries, with an overall estimated CKD prevalence reaching 13.6% among adults (18), little is known about the prevalence and incidence of CVD in Black African patients with CKD. While hypertension and diabetes have been identified as common CV risk factors among CKD patients in Africa (18-20), data related to AS and CV outcomes in these patients have been poorly explored to date, especially in Cameroon. The present study aimed to assess the patterns of AS measured by PWV among black African ancestry CKD patients living in Cameroon.

Methods

Study design and data collection

Between October 2015 and May 2016, 150 CKD patients

(56.7% males, mean age 52±15 years) comprising 90 non-dialysed and 60 hemodialysed patients were consecutively recruited in a cross-sectional study conducted in the hemodialysis unit of Douala General Hospital, Douala, Cameroon. To be included patients had to meet the Kidney Disease Outcomes Quality Initiative (KDOQI) criteria for CKD stages 1 to 5, with estimated glomerular filtration rate (eGFR) calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation and/or be on chronic hemodialysis (21). Exclusion criteria included: acute disease requiring hospitalization at the time of patient enrolment; evidence of stenosis of the subclavian artery of the arm without shunt or of the non-shunt arm chosen to be used for the Mobil-O-Graph measurements; atrial fibrillation, mental illness; inability to understand the information presented and to sign the informed consent. A questionnaire was used to assess socio-demographic variables, education level, patients' medical history (comorbidities, etiology of CKD, duration on dialysis, ongoing medications) and lifestyle data. Previously documented cardiovascular events (CVE) such as myocardial infarction, stroke, transient ischemic attack, peripheral vascular disease or heart failure were also recorded. Physical examination included measurements of BP, heart rate (HR), waist circumference (WC), body weight, and height. Body weight was measured in kilogram (kg), and height in meter (m) was measured to the nearest 0.5 cm with a fixed stadiometer. Body mass index (BMI; kg/m²) was calculated as weight (kg) divided by the square of height (m). Overweight was defined as a BMI ≥25 kg/m², and obesity as a BMI ≥30 kg/m². Abdominal obesity was defined as WC ≥102 cm for men and ≥88 cm for women (22).

All hemodynamic measurements were performed, after 15 minutes rest, in the sitting position and in standardized conditions (23). BP and PWV were recorded on the dominant arm and/or on the arm contralateral to the arteriovenous shunt in non dialysed and hemodialysed patients, respectively. Peripheral and central aortic systolic, diastolic and mean arterial BP (MAP), HR, and PWV were measured using a validated brachial cuff-based oscillometric method based on mathematical transformation of brachial pressure waveforms, using a common cuff (Mobil-O-Graph, I.E.M., Stolberg, North Rhine-Westphalia, Germany); which is a non-operator dependent automatic device. Two consecutive measurements were taken at time intervals of ≥5 minutes, using a cuff's width adjusted to the arm's circumference. The mean of the nearest two accurate hemodynamic readings was used. The reliability

of the Mobil-O-Graph in estimating the PWV by PWA (MobPWV) was demonstrated in previous studies (24,25). The device was approved by the Food and Drug Administration, with its BP detection unit validated according to the British Hypertension Society (26) and the European Society of Hypertension (27) recommendations. Pulse pressure (PP) was calculated as systolic minus diastolic BP. All procedures were performed by a single medical investigator. In dialysis patients, measurements were taken on dialysis day (prior to dialysis session; pre-dialysis measurement), and thereafter 24 hours after last dialysis (post-dialysis measurement).

Hypertension was defined as systolic BP ≥ 140 mmHg and/or diastolic BP ≥ 90 mmHg or ongoing antihypertensive medication. A standard carotid-femoral PWV (cfPWV) ≥ 10 m/s obtained by direct measurement method was proposed as indicative of subclinical aortic damage (22). The Mobil-O-Graph device used in this study provides an indirect measure of aortic PWV (mobPWV). Few studies have compared mobPWV and cfPWV. In hemodialysis patients, Sarafidis *et al.* (25) found that MobPWV gave lower values (-0.8 m/s) than Sphygmocor cfPWV (10.3 vs. 9.5 m/s, respectively), with Sphygmocor values obtained with subtracted distance using the suprasternal notch. Since the subtracted distance method underestimates standard cfPWV by 10% (28), and considering these findings, the cutoff value for MobPWV should be 8.2 m/s instead of 10 m/s (10% underestimation by the subtracted distance method amounting to 9 m/s minus 0.8 m/s lower values with MobPWV, yielding a figure of 8.2 m/s).

Blood samples were collected after an 8-hour overnight fast and sent to the biochemistry laboratory of Douala General Hospital for analysis. Serum creatinine, uricemia, calcium, phosphorus, hemoglobin, fasting plasma glucose (FPG), total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol values were measured using enzymatic colorimetric methods. Blood samples were collected before dialysis sessions in hemodialysis patients.

Diabetes mellitus was defined as FPG ≥ 1.26 g/L and/or being on glucose-lowering medication(s) (29). Smoking status was defined as never, former or current smoking, the latter also including occasional smokers. Alcohol use was defined as having at least one alcoholic drink per week, and classified along three categories: never, former and current users.

Kidney disease was defined using the KDOQI definition for CKD (21), i.e., lowering of eGFR and/or the presence of

(micro) albuminuria. Each patient was classified into one of 5 CKD stages: stage 1: kidney damage with normal or increased eGFR (>90 mL/min/1.73 m²); stage 2: mild decreased in eGFR ($60-89$ mL/min/1.73 m²); stage 3: moderate reduction in eGFR ($30-59$ mL/min/1.73 m²); stage 4: severe reduction in eGFR ($15-29$ mL/min/1.73 m²); stage 5: kidney failure (eGFR <15 mL/min/1.73 m² and/or chronic dialysis). The chronic hemodialysis group consisted of all patients with stage 5 CKD (eGFR ≤ 15 mL/min/1.73 m²) and on hemodialysis as renal replacement therapy.

Participation to the survey was voluntary and written signed informed consent was taken from all the patients before inclusion. The study was approved by the Ethical Institutional Research Board of the University of Douala, Cameroon (No. CEI-UD/362/12/2015/T).

Statistical analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences version 20.0 software (SPSS Inc, Chicago, Illinois, USA). Quantitative data are presented as mean \pm standard deviation (SD), and qualitative data are presented as frequencies or percentages. Comparison of quantitative data between non-dialyzed and hemodialysed patients was performed using Student *t*-test and qualitative data were compared using Chi square test. For comparison of hemodynamic parameters between pre- and post-dialysis, we used the non-parametric Wilcoxon rang test. Estimated standard cfPWV and mobPWV were adjusted for major confounders including age, sex, MAP and BMI in both non-dialysed CKD and hemodialysis participants using linear regression equation. A multiple regression analysis was performed to assess independent determinants of PWV. All relevant variables correlated with PWV (at least $P < 0.1$) were included in the model. Statistical significance was considered for $P < 0.05$.

Results

Baseline characteristics

As shown in *Table 1*, non-dialysed patients were older ($P=0.009$) and more had a BMI >25 kg/m² (0.012) than patients on maintenance dialysis. Most non-dialysed patients (33.3%) were in CKD stage 5 or CKD stage 4 (25.6%). Hypertension was the most frequent CV risk factor among study participants, followed by type 2 diabetes mellitus. Mean hemoglobin was lower in hemodialysis

Table 1 Baseline characteristics of the study population

Variables	Total, n=150	Non-dialysis, n=90	Hemodialysis, n=60	P
Age (years)	52±15	55±15	48±15	0.009
Male gender	85 (56.7)	53 (58.9)	32 (53.3)	0.6
BMI (kg/m ²)	26.2±4.7	27.0±4.8	25.0±4.3	0.009
Overweight/obesity	80 (53.3)	56 (62.2)	24 (40.0)	0.012
Abdominal obesity	51 (34.0)	35 (38.9)	16 (26.7)	0.17
Current smoking	2 (1.3)	1 (1.1)	1 (1.7)	0.8
Current alcohol use	24 (16.0)	19 (21.1)	5 (8.3)	0.07
Hypertension	131 (87.3)	77 (85.6)	54 (90.0)	0.3
Hypertension medication	118 (78.7)	70 (77.8)	48 (80.0)	0.9
Diabetes	49 (32.7)	32 (35.6)	17 (28.3)	0.5
Current dyslipidemia	93 (62.0)	55 (61.1)	38 (63.3)	0.9
Previous CVE	27 (18.0)	17 (18.9)	10 (16.7)	0.8
Blood glucose (g/L)	1.18±0.58	1.16±0.44	1.27±0.70	0.289
Hemoglobin (g/dL)	9.1±2.4	10.3±2.2	8.4±1.9	<0.0001
Serum calcium (mg/L)	8.8±1.6	8.79±0.88	8.84±1.96	0.858
Serum phosphate (mg/L)	4.2±1.7	4.27±1.58	4.09±1.66	0.529
Ca-Ph product (mg ² /dL ²)	37.3±15.8	37.16±12.88	37.59±16.11	0.8
Total cholesterol (g/L)	1.78±0.95	1.94±1.07	1.56±0.35	0.013
HDL cholesterol (g/L)	0.45±0.21	0.52±0.22	0.38±0.20	0.0002
Triglycerides (g/L)	1.05±0.49	1.19±1.00	0.88±0.33	0.032
LDL cholesterol (g/L)	1.08±0.89	1.13±0.65	1.09±0.55	0.6
Median GFR (mL/min/1.73 m ²)	26.5	27.0	0.29	0.048
Peripheral				
SBP (mmHg)	143±25	144±25	148±24	0.771
DBP (mmHg)	91±16	89±16	93±17	0.216
PP (mmHg)	53±16	55±16	56±14	0.386
HR (bpm)	80±13	78±12	78±12	0.881
Central				
SBP (mmHg)	130±22	131±22	134±21	0.469
DBP (mmHg)	92±17	91±17	94±18	0.15
PP (mmHg)	38±14	40±14	39±11	0.434
MAP (mmHg)	104±18	104±18	108±18	0.218
MobPWV (m/s)	8.2±1.8	8.5±2.0	7.9±1.4	0.025
Estimated cfPWV (m/s)	10.0±2.0	10.3±2.2	9.6±1.6	0.025
MobPWV* (m/s)	8.2±1.6	8.4±1.7	7.9±1.4	0.042
Estimated cfPWV* (m/s)	10.0±1.8	10.3±1.9	9.7±1.6	0.042

Data are mean ± standard deviation or number (percentages); PWV*, pulse wave velocity adjusted for age, MAP, sex, body mass index. HDL, high-density lipoprotein; LDL, non-high-density lipoprotein; Ca-Ph, calcium-phosphate; CVE, cardiovascular event.

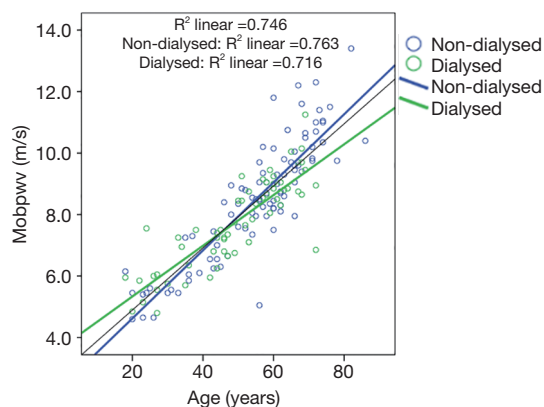


Figure 1 Correlation between age and pulse wave velocity in both non-dialysed and hemodialysed patients.

patients ($P < 0.0001$ vs. non-hemodialysis patients), whereas total cholesterol, triglycerides and HDL-cholesterol were significantly higher in non-dialysis than in hemodialysis patients (all $P < 0.05$). Alcohol consumption rate was slightly higher in non-dialysis than in hemodialysis patients ($P = 0.07$). Peripheral as well as central SBP, DBP, PP and MAP were similar between non-dialyzed and hemodialyzed patients. MobPWV and estimated cfPWV were significantly higher in non-dialyzed than in hemodialyzed patients, even after adjustment for age and BMI (all $P < 0.05$).

Hemodynamic variables

PWV increased significantly ($P = 0.002$) with aging in both non-dialysis and hemodialysis patients (Figure 1). After adjustment for age, MAP, sex and BMI, estimated cfPWV was markedly higher in non-dialysis than in hemodialyzed patients, even in pre-dialysis ($P = 0.026$) and in post-dialysis ($P = 0.008$) (Table 1), suggesting that chronic dialysis may favourably impact AS. MAP and both peripheral and central systolic and diastolic BP were similar between the two groups. As illustrated in Table 2, hemodialysis acutely and significantly decreased peripheral PP (by 6 mmHg; $P = 0.008$), and increased HR (by approximately 5 bpm; $P = 0.001$).

As shown in Figure 2, 61.3% of the total study population exhibited age, MAP, sex and BMI-adjusted PWV: MobPWV ≥ 8.2 m/s; estimated standard cfPWV ≥ 10.0 m/s, suggesting subclinical aortic damage. Most participants with high PWV were non-dialysed patients ($P = 0.033$).

PWV according to CV risk factors and other CKD-related

parameters

In the overall study population (Table 3), PWV was higher in patients aged ≥ 50 years, in hypertensive patients and/or in diabetic patients with CKD (all $P < 0.01$) and with a trend for higher PWV in patients with previous CVE ($P = 0.064$). There was no significant difference between those in CKD stage 5 and those in early CKD stages.

Determinants of aortic PWV

As illustrated in Table 4, multivariable regression analysis performed in the total study population, and in non-dialyzed group taken separately revealed that advanced age, MAP and current smoking were positively and independently associated with PWV (all $P < 0.05$). In the hemodialysis group in predialysis as well as in post dialysis, older age, MAP and diabetes emerged as independent correlates of PWV ($P < 0.05$).

Discussion

We are not aware of another study exploring aortic stiffness as assessed by PWV estimated by Mobil-O-Graph in CKD patients born and living in Cameroon, and this study provides direct evidence of higher age, MAP, sex and BMI adjusted PWV in non-dialysis patients vs. those on chronic maintenance dialysis. AS was mostly driven by age, BP, tobacco use and diabetes. It should be noted that high-risk patients exhibiting increased PWV, a major vascular aging biomarker suggestive of subclinical aortic damage, were mostly found among non-dialysed patients.

Increased AS was reported in both normotensive and hypertensives black populations (30), as well as in the present study of black Cameroonian indigenous CKD patients, especially in the non-dialysed patients. Our findings are in line with previous observations of Caucasian patients with CKD, in whom early vascular ageing, characterized by aortic stiffening combined with outward arterial remodeling, was observed according to progression of CKD and in ESRD (11-13). Increased arterial stiffening in CKD patients is the result of aging and of non-specific and ESRD related risk factors, such as medial calcification, volume overload, uraemia-related endothelial dysfunction, increased extracellular matrix and intimal fibroelastic thickening (13,31). Our findings also in the line of Shinohara *et al.* (32), who previously reported

Table 2 Comparison of hemodynamic parameters between hemodialysed patients in pre-dialysis and in post dialysis

Variables	Total	Pre-dialysis	24 h post-dialysis	P
Peripheral				
SBP (mmHg)	143±25	148±24	143±25	0.008
DBP (mmHg)	91±16	93±17	92±17	0.667
PP (mmHg)	53±16	56±14	50±15	0.008
HR (bpm)	80±13	78±12	83±14	0.001
Central				
SBP (mmHg)	130±22	134±21	129±21	0.06
DBP (mmHg)	92±17	94±18	93±18	0.687
PP (mmHg)	38±14	39±11	36±13	0.28
MAP (mmHg)	104±18	108±18	105±18	0.129
MobPWV (m/s)	8.2±1.8	7.9±1.4	7.8±1.5	0.15
Estimated cfPWV (m/s)	10.0±2.0	9.6±1.6	9.5±1.6	0.15
MobPWV* (m/s)	8.2±1.6	7.9±1.4	7.8±1.5	0.25
Estimated cfPWV* (m/s)	10.0±1.8	9.7±1.6	9.6±1.6	0.25

PWV*, pulse wave velocity adjusted for age, MAP, sex, body mass index. HR, heart rate; HD, hemodialysis; SBP, systolic blood pressure, DBP, diastolic blood pressure, PP, pulse pressure, MAP, mean arterial blood pressure.

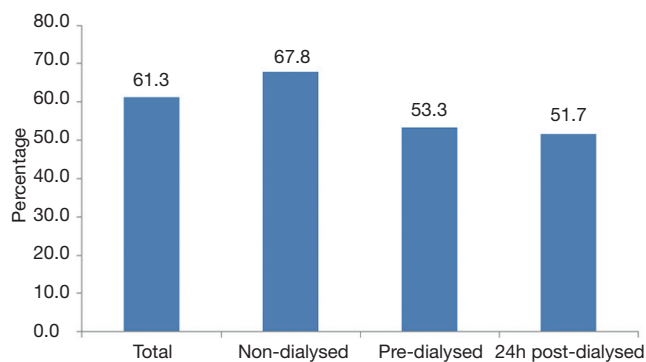


Figure 2 Proportion of patients with increased PWV (MobPWV ≥ 8.2 m/s; estimated standard cfPWV ≥ 10.0 m/s).

that predialysis CKD patients are more prone to accelerated arterial wall stiffening than chronic hemodialysis patients. It was suggested that hemodialysis could directly control or reduce some of the metabolic alterations underlying AS in patients with advanced CKD. Thus, hemodialysis patients show cyclic changes in PWV related to hydration status and BP values (33), with a recent study suggesting that daily

Table 3 Comparison of mean pulse wave velocity with cardiovascular risk factors and other parameters in the total study population

Variables	No	Yes	P
Age ≥ 50 years	6.9±3.4	9.3±1.3	<0.0001
Male sex	8.6±3.0	8.0±1.9	0.7
Smoking	8.3±2.7	9.1±1.8	0.4
Alcohol use	8.3±2.8	8.5±1.6	0.7
Overweight/obesity	8.2±3.5	8.6±1.6	0.3
Abdominal obesity	8.2±3.0	8.6±1.6	0.3
Hypertension	6.8±1.7	8.6±2.7	0.005
Diabetes	7.8±2.9	9.5±1.2	<0.0001
Previous CV event	8.3±2.8	8.9±1.2	0.064
Anemia	8.3±1.8	8.6±2.8	0.2
High Ca-Ph	8.6±2.8	8.2±1.9	0.5
Dyslipidemia	8.0±2.1	8.6±2.9	0.1
Stage 5 CKD	8.5±1.9	8.3±3.0	0.5

Data are mean \pm standard deviation of pulse wave velocity. Ca-Ph, calcium phosphorus product.

Table 4 Multivariable linear regression analysis of some factors correlating independently with pulse wave velocity

Variables	All participants (n=150)	Non-dialyzed (n=90)	Pre-dialyzed (n=60)	Post-dialyzed (n=60)
Age (years)	0.83±0.05 ^c	0.84±0.05 ^c	0.82±0.09 ^c	0.78±0.08 ^c
BMI (kg/m ²)	0.03±0.04	-0.01±0.05	0.06±0.07	0.06±0.07
MAP (mmHg)	0.23±0.04 ^c	0.23±0.05 ^c	0.36±0.08 ^c	0.27±0.07 ^b
Diabetes	0.05±0.05	0.00±0.05	0.22±0.09 ^b	0.22±0.09 ^a
Previous CVE	0.03±0.04	0.04±0.05	0.02±0.07	0.00±0.07
Current smoking	0.08±0.04 ^a	0.09±0.05 ^a	0.06±0.07	0.07±0.07
Adjusted R ²	0.782	0.820	0.734	0.757
P value for the model	<0.0001	<0.0001	<0.0001	<0.0001

^a, P<0.05; ^b, P<0.01; ^c, P<0.001; Data are $\beta \pm$ SE. β , standard regression coefficient. SE, standard error; BMI, body mass index; CVE, cardiovascular event; MAP, mean arterial pressure.

dialysis may be used in patients with high PWV levels to reduce mortality risk (33).

Of note, advanced age emerged as independent determinant of increased PWV in the overall study population, as well as in non-dialysed and hemodialysed patients taken separately. Our findings are in line with previous surveys of Caucasian CKD patients which identified age as a strong and independent marker of increased AS (3,6-8,34). Arterial stiffening is an unavoidable participant to ageing processes. Thus, the aorta and large arteries become progressively less distensible with advancing age, with resultant reduced capacity to buffer pulsations from the contracting ventricle (35,36).

We found that MAP, which reflects the steady component of BP, emerged as an independent marker of increased PWV. This observation fits with previous studies demonstrating that BP is a major determinant of PWV (7,12,28,30,34). Indeed, aortic stiffening can at first be functional, resulting from higher BP without structural changes to the artery. Hypertension was also recognized as a major determinant of increased AS due to the associated medial hypertrophy which arises from chronic elevation of BP, which is usually the case in CKD patients (7,31,34), as well as in this study population, in which more than three quarter of patients were hypertensive.

Evidence also indicates that increased AS contributes to elevated PP, the dynamic component of BP, and inversely a decrease in BP can attenuate vascular stiffness (7). Thus, the lower PP observed following an hemodialysis session was in keeping with the lower PWV observed in ESRD patients on maintenance dialysis. However, the beneficial role of hemodialysis on AS is still debated (36). Some studies

(37,38), but not all (17,33) found a progressive increase of AS in hemodialysis patients. Part of the discrepancy among observations could be attributed to differences in study design. Our survey was strictly cross-sectional and had a relatively limited sample size. Differences in type of device used to assess aortic PWV, and possibly differences in age and comorbidity status of CKD patients could also represent confounders. However, our results establish unequivocally that a high proportion of Cameroonian patients with CKD exhibit major impairment in functional and structural large artery wall properties before initiation of hemodialysis.

Increased AS has also been reported in diabetic patients (12), and this was the case for Cameroonian CKD diabetic participants in this study. Yet diabetes, which was independently related to PWV in Caucasian CKD patients (34), emerged as independent determinant of PWV only in patients on maintenance dialysis, but not in non-dialysed patients in our study, probably because PWV was adjusted for obesity, which is a major determinant of type 2 diabetes mellitus and which was more pronounced in non-dialysed participants.

In Caucasian patients with CKD, some studies (14,31,38-40), but not all (11,13,17,33), reported increased PWV with declining kidney function assessed by eGFR, a validated marker of CKD progression. In the present survey, no association was observed between PWV and eGFR. This could be attributable, at least partly, to the limited sample size of the study population.

There are some limitations to this report. First, only cross-sectional data were included in the analysis, making

impossible to draw any firm conclusions regarding the causality of the relationships between AS and the determinants identified. Long-term prospective study is needed to better characterize the impact of CKD on the natural history of AS and its determinants in CKD patients living in Cameroon. Secondly, being a single-centre study with limited sample size, the power to detect differences within our study population was constrained. Thirdly, we used the Mobil-O-graph which represents an oscillometric method for estimating aortic PWV. Few studies have used this device in CKD patients, and it may underestimate aortic PWV (25). Pending to its validation in Black African ancestry population, including patients with CKD, our data should be interpreted with caution. A fourth limitation of this study was the absence of normal control subjects, which may allow to determine the impact of CKD on AS in black African indigenous patients living in Cameroon with this condition. A fifth limitation is the lack of correlations between PWV findings and other stiffness index such like left ventricular diastolic function as assessed by echocardiography. In mitigation, these limitations cannot alter the overall scope of our observations, since data were rigorously collected, and a single trained medical operator performed all measurements, eliminating inter-observer variability.

In conclusion, this study reveals higher aortic PWV in black Cameroonian non-dialysed CKD patients in comparison to those on maintenance dialysis. Slower PWV observed in patients on maintenance hemodialysis suggests that dialysis may contribute to improvement of their aortic distensibility. PWV was mainly modulated by advancing age, blood pressure, smoking and diabetes. However, further large-scale studies are needed to confirm our findings and to elicit underlying mechanisms of arterial stiffening in black African ancestry patients with CKD.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Ethical Statement: The study was approved by institutional ethics committee Board of the Douala University (No. 326) and informed consent was taken from all the patients.

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