Prevalence and Determinants of Chronic Kidney Disease Among Hypertensive Cameroonians According to Three Common Estimators of the Glomerular Filtration Rate

Francois Folefack Kaze, MD;[1,2] Andre-Pascal Kengne, MD, PhD;[3] Carine Tchendjou Magatsing, MD;[4] Marie-Patrice Halle, MD;[5] Euloge Yiagnigni, MD;[6] Kathleen Blackett Ngu, MD[2]

From the Department of Medicine and Specialties, Faculty of Medicine and Biomedical Sciences, The University of Yaoundé 1, Yaoundé, Cameroon;[1] Department of Internal Medicine, The Yaoundé University Teaching Hospital, Yaoundé, Cameroon;[2] South African Medical Research Council, University of Cape Town, Cape Town, South Africa;[3] Higher Institute of Health Sciences, Bangangté, Cameroon;[4] Department of Clinical Sciences, Faculty of Medicine and Pharmaceutical Sciences, The University of Douala, Douala, Cameroon;[5] and Department of Internal Medicine, Yaoundé Central Hospital, Yaoundé, Cameroon[6]

Hypertension is a risk factor for renal diseases, which, in turn, are precursors of hypertension. The authors assessed the prevalence and determinants of chronic kidney disease (CKD) among 336 hypertensive adult Cameroonians (mean age, 60.9±11.3 years; 63.4% women) at Yaoundé. Any participant with an estimated glomerular filtration rate <60 mL/min/1.73 m^2 regardless of the equation used (Cockcroft-Gault [CG], Modification of Diet in Renal Disease [MDRD], and Chronic Kidney Disease Epidemiology Collaboration [CKD-EPI]) and/or dipstick proteinuria was reviewed 3 months later. Participants presented a high prevalence of diabetes (18.5%), dyslipidemia (17.6%), gout/hyperuricemia (10.7%), overweight/obesity (68.8%), self-medication (37.5%), and alcohol consumption (33.3%). Hypertension was uncontrolled in 265 patients (78.9%). The prevalence of CKD was 49.7%, 50.0%, and 52.1% according to MDRD, CKD-EPI, and CG equations, respectively. Advanced age, adiposity, and severity of hypertension were determinants of CKD. Nearly half of the hypertensive patients had CKD regardless of the estimators used, predicted by well-known risk factors. *J Clin Hypertens (Greenwich).* 2016;18:408–414.

The prevalence of hypertension is growing worldwide, particularly in sub-Saharan Africa (SSA) where most people with the disease remain undiagnosed, untreated, or inadequately treated.[1,2] Hypertension is also a major risk factor for cardiovascular and renal diseases and, furthermore, kidney diseases are precursors of hypertension.[3]

Chronic kidney disease (CKD) affects 10% of adults worldwide and poses a major public health and socioeconomic challenge.[4,5] Studies have revealed that Africans are at higher risk for CKD, which is three to four times more frequent in this ethnic group than in Caucasians, occurs prematurely, and progresses rapidly to end-stage renal disease (ESRD).[6,7] Although a high prevalence of CKD have been reported in SSA settings, even higher figures ranging from 38.2% to 46.9% have been reported in high-risk populations including hypertensive patients.[8–12] Compared with Caucasians, Africans are at high risk for hypertensive nephrosclerosis (the second leading cause of ESRD), are five to eight times more likely to develop ESRD from hypertension, have renal involvement at a younger age, have blood pressure (BP) that responds less well to acute treatment, and have a faster decline in renal function at similar BP levels.[3,10,13–15]

In Cameroon, hypertension and other noncommunicable diseases (NCDs) including CKD are increasingly common, with unacceptably low awareness, treatment, and control rates.[16,17] However, CKD among people with hypertension, particularly those receiving routine care, has seldom been characterized. This has relevance in the context of highly advocated integrated approaches to the most common NCDs. Accordingly, we undertook this study to establish the prevalence and risk factors for CKD among hypertensive Cameroonians.

METHODS

Study Setting and Design

Participants were recruited at the hypertension clinics of Yaoundé Central and University Teaching Hospitals—the two major hypertension clinics in the capital city of Cameroon. We included all Cameroonian adults (aged ≥18 years) with a medical diagnosis of hypertension. Participants provided written informed consent and were advised to take their medications as usual on the day of recruitment. This study was approved by the administrative authorities of the involved hospitals and the Cameroon national ethics committee.

Data Collection

Final year undergraduate medical students collected data on the following: demographics (age, sex, and level of education), duration of hypertension, ongoing

Address for correspondence: Francois Folefack Kaze, MD, Department of Medicine and Specialties, Faculty of Medicine and Biomedical Sciences, The University of Yaoundé, Yaoundé, PO Box 33127, Cameroon
E-mail: f_kaze@yahoo.fr

Manuscript received: August 22, 2015; **revised:** October 21, 2015; **accepted:** October 22, 2015
DOI: 10.1111/jch.12781

treatment, family history of CKD, comorbidities (diabetes, gout, human immunodeficiency virus [HIV], and viral hepatitis B and C), lifestyle characteristics, and practice of self-medication. Anthropometric measurements and BP were taken three times and the average was used in all analyses. BP was measured in the office according to World Health Organization (WHO) guidelines[18] using an automated sphygmomanometer (OMRON HEM705CP; Omron Matsusaka Co, Matsusaka, Japan)[19] and appropriate cuff size on the right arm with participants in a sitting position after 30 minutes of rest. For each participant, we drew 3 mL of whole blood from an antecubital vein for serum creatinine (SCr) and fasting capillary glucose (after an overnight fast of at least 8 hours), and collected mid-stream second morning urine for dipstick tests. The urine dipstick tests used were CombiScreen 7SL PLUS 7 test strips (Analyticon Biotechnologies AG, D-35104 Lichentenfeis, Germany). Serum creatinine was measured with a kinetic modification of the Jaffé reaction using Human visual spectrophotometer (Human Gesellschaft, Biochemica und Diagnostica mbH, Wiesbaden, Germany) and the Beckman creatinine analyzer (Beckman CX systems instruments, Anaheim, CA, USA), and converted to standardized as $SCr_{Standardized}=0.95*SCr_{Jaffe} - 0.10$.[20,21] In participants with an estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m^2 and/or dipstick proteinuria, the chronicity was confirmed on another sample 3 months later.

Definitions and Calculations

eGFR (mL/min) was measured using the Cockcroft-Gault (CG), the four-variable Modification of Diet in Renal Disease (MDRD), and the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equations.[22–24] CKD was defined by a confirmed positive dipstick proteinuria or albuminuria (at least traces) and/or eGFR <60 mL/min/1.73 m^2. The Kidney Disease Improving Global Outcomes (KDIGO) guidelines were used to stage participants for GFR and albuminuria categories of CKD.[25] The GFR categories included: G1 (eGFR ≥90), G2 (eGFR 60–89), G3 (eGFR 30–59), G4 (eGFR 15–29), and G5 (eGFR <15). The albuminuria categories of CKD were as follows: A1 (negative), A2 (traces to 1+ [30]), and A3 (at least 2+ [100]). The diagnosis of diabetes mellitus, dyslipidemia, and gout was made by the attending physician. Controlled BP was based on the average value of the three previous consultations' BP levels of <140 mm Hg for systolic BP and <90 mm Hg for diastolic BP. Hyperglycemia refers to a fasting capillary glucose level of at least 1.26 g/L. Self-medication referred to recurrent use of medicines at least once every 3 months; these medicines included herbal medicines from African pharmacopeia and street medications, which are Western drugs usually of uncertain origins that are sold in shops and along market streets without any control.

Statistical Analysis

Data were analyzed using SAS/STAT version 9.1 software for Windows (SAS Institute Inc, Cary, NC). We have reported the results as means and standard deviations (SDs) and counts and percentages. Group comparisons used chi-square tests and variants for qualitative variables, and Student *t* test and nonparametric equivalents for quantitative variables. Age- and sex-adjusted logistic regression models were used to investigate the predictors of CKD, CKD stages G3 and G4, and albuminuria. A *P* value <.05 was used to characterize statistically significant results.

Sample Size Estimation

The study was planned with the intention of being able to investigate about 12 predictors of CKD. For such a purpose and assuming a ratio of eight participants with CKD per candidate predictor,[26] a minimum of 96 participants with prevalent CKD were required. We further assumed the prevalence of CKD to be about 38% in our population, which is the bottom figure of reported prevalence of CKD in people with hypertension in SSA.[8–12] Based on the above parameters, and considering a 95% probability of observing at least 96 participants with prevalent CKD, we needed to examine at least 288 participants with hypertension.

RESULTS

Baseline Characteristics of the Study Population

A total of 336 hypertensive patients, 213 (63.4%) women, participated in the study. The mean age of participants was 60.9±11.3 years, and up to 10.4% did not have any formal education. As shown in Table I, only 0.6% of the participants reported an existing family history of kidney disease although the group presented a high prevalence of CKD risk factors including diabetes (18.5%), dyslipidemia (17.6%), gout (10.7%), overweight/obesity (68.8%), self-medication (37.5%), alcohol consumption (33.3%), and smoking (3.4%). There was a mild decrease in mean eGFR, which was almost similar across the various estimators, while study participants presented a high frequency of dipstick abnormalities including albuminuria (39.3%), hematuria (14.8%), and leukocyturia (8%) (Table II).

The mean systolic BP was 154±26 mm Hg and diastolic BP was 90±15 mm Hg. Equivalent figures were 126±8 mm Hg and 76±7 mm Hg for the controlled group and 162±23 mm Hg and 94±14 mm Hg for the uncontrolled group (all *P*<.001). The duration of hypertension was 6.7±7.9 years in the global population, 8.3±8.6 years in the controlled group, and 6.2 years in the uncontrolled group (*P*=.047). The median (25–75th percentiles) number of antihypertensive drugs was two (1–2), with no statistical difference between the two groups (*P*=.120) (Table I).

Hypertension was uncontrolled in 265 patients (78.9%) and was significantly associated with younger age (*P*=.01), shorter duration of the disease (*P*=.047),

TABLE I. Clinical Characteristics by BP Control

Characteristics	Overall	Controlled BP Group	Uncontrolled BP Group	*P* Value
No. (%)	336 (100)	71 (21.1)	265 (78.9)	
Mean age, y (SD)	60.9 (11.3)	64.0 (10.6)	60.1 (11.4)	.01
Men:women, No.	123:213	20:51	103:162	.097
Level of education, No. (%)				
None	35 (10.4)	9 (12.7)	26 (9.8)	.614
Primary	121 (36.0)	24 (33.8)	97 (36.6)	
Secondary	124 (36.9)	29 (40.8)	95 (35.8)	
Higher	56 (16.7)	9 (12.7)	47 (17.7)	
Familial history of kidney disease, No. (%)				
No	328 (97.6)	70 (98.6)	258 (97.4)	.596
Yes	2 (0.6)	0 (0)	2 (0.8)	
Unknown	6 (1.8)	1 (1.4)	5 (1.9)	
Diabetes, No. (%)	62 (18.5)	4 (5.6)	58 (21.9)	.001
Gout, No. (%)	36 (10.7)	8 (11.3)	28 (10.6)	.505
Dyslipidemia, No. (%)	59 (17.6)	44 (16.6)	15 (21.1)	.374
HIV infection, No. (%)				
No	306 (91.1)	69 (97.2)	237 (89.4)	.026
Yes	12 (3.6)	0 (0)	12 (4.5)	
Unknown	18 (5.4)	2 (2.8)	16 (6.0)	
Current or former tobacco use, No. (%)	13 (3.9)	3 (4.2)	10 (3.8)	.542
Current or former alcohol use, No. (%)	112 (33.3)	26 (36.6)	86 (32.5)	.508
Self-medication, No. (%)	126 (37.5)	25 (35.2)	101 (38.1)	.654
Mean duration of hypertension, y (SD)	6.7 (7.9)	8.3 (8.6)	6.2 (7.6)	.047
Mean SBP, mm Hg (SD)	154 (26)	126 (8)	162 (23)	<.001
Mean DBP, mm Hg (SD)	90 (15)	76 (7)	94 (14)	<.001
Hypertension treatment, No. (%)				
ACE inhibitor	192 (57.1)	49 (69.0)	143 (54.0)	.023
ARB	9 (2.7)	2 (2.8)	7 (2.6)	>.999
CCB	132 (39.3)	24 (33.8)	108 (40.7)	.287
Diuretics	9 (2.7)	2 (2.8)	7 (2.6)	.086
Blocker	42 (12.5)	9 (12.7)	33 (12.4)	.960
Central-acting drugs	2 (0.6)	0 (0)	2 (0.7)	>.999
Median (25–75th percentiles) number of antihypertensive drugs	2 (1–2)	2 (2–2)	2 (1–2)	.120
Mean BMI, kg/m^2 (SD)	29.4 (14.8)	27.7 (5.1)	29.9 (16.4)	.282
BMI ≥25 kg/m^2, No. (%)	231 (68.8)	48 (67.6)	183 (69.1)	.815

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BMI, body mass index; BP, blood pressure; CCB, calcium channel blocker; DBP, diastolic blood pressure; HIV, human immunodeficiency virus; SBP, systolic blood pressure; SD, standard deviation.

TABLE II. Biological Characteristics by BP Control

Characteristics	Overall	Controlled BP Group	Uncontrolled BP Group	*P* Value
No. (%)	336 (100)	71 (21.1)	265 (78.9)	
Mean fasting capillary glucose, g/L (SD)	1.01 (0.40)	0.89 (0.19)	1.05 (0.43	.004
Hyperglycemia, No. (%)	48 (14.3)	3 (4.2)	45 (17.0)	.006
Dipstick abnormalities, No. (%)				
Albuminuria	132 (39.3)	25 (35.2)	107 (40.4)	.319
Hematuria	50 (14.9)	13 (18.3)	37 (14.0)	.361
Leukocyturia	27 (8.0)	4 (5.6)	23 (8.7)	.402
Mean serum creatinine, mg/L (SD)	13.0 (10.4)	11.7 (5.1)	13.4 (11.4)	.229
Mean creatinine clearance, mL/min/1.73 m^2 (SD)				
MDRD	71.0 (29.1)	71.3 (31.1)	70.9 (28.7)	.928
CG	72.8 (35.0)	68.0 (29.9)	74.1 (36.2)	.193
CKD-EPI	70.1 (26.0)	68.9 (21.7)	70.5 (27.0)	.660

Abbreviations: BP, blood pressure; CG, Cockroft-Gault; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration; MDRD, Modification of Diet in Renal Disease; SD, standard deviation.

uncontrolled diabetes (*P*=.006), HIV infection (*P*=.026), and lower use of angiotensin-converting enzyme inhibitors (*P*=.023) (Tables I and II).

Prevalence and Correlates of CKL As Well As Albuminuria and G3 to G5 GFR Categories

Nearly half of patients had CKD regardless the estimators, with prevalence rates of 49.7%, 50.0%, and 52.1% according to MDRD, CKD-EPI, and CG equations, respectively (Table III and Table SI). Using albuminuria and GFR categories of the KDIGO classification of CKD, 116 patients (34.5%) had albuminuria A2 to A3, among whom 21 (6.2%) and 95 (28.3%) were in categories A2 and A3, respectively. Similarly, 109 patients (32.5%) were in G3 to G5 categories according to MDRD and CKD-EPI equations, and 113 patients (33.6%) according to the CG formula (Tables III and Table SI). With GFR estimated by the MDRD equation, the prevalence of G3, G4, and G5 stages was 28.3%, 2.1%, and 2.1%, respectively. Equivalent figures were 27.7%, 3.0%, and 1.8% with the CKD-EPI equation, and 25.3%, 6.8%, and 1.5% with the CG formula.

Advanced age was the only factor significantly associated with the presence of CKD and G3 to G5 categories of CKD regardless of the equation used (all *P*<.001), while albuminuria was associated with female sex (*P*=.009). Raised systolic BP was associated with albuminuria (*P*=.025) and G3 to G5 categories of CKD estimated by MDRD (*P*=.047) and CKD-EPI (*P*=.043) equations. Longer duration of hypertension was associated with G3 to G5 categories of CKD-EPI–diagnosed CKD (*P*=.03), while the presence of diabetes was associated with G3 to G5 categories of CKD estimated by CG (*P*=.008) and MDRD (*P*=.05) equations. As expected, low body mass index (BMI) was associated with G3 to G5 categories of CKD estimated by the CG formula (Table III and Table SI).

Predictors of CKD As Well As Albuminuria and G3 to G5 GFR Categories in Age- and Sex-Adjusted Logistic Regressions

Table IV and Table SII show the age- and sex-adjusted predictors of CKD as well as albuminuria and the G3 to G5 GFR categories of CKD based on each of the three estimators. With the exception of albuminuria, advanced age was consistently and positively associated with all these outcomes, with the magnitude of the effects per year increase in age being 4% (2%–6%) to 6% (3%–8%) for CKD and 5% (2%–7%) to 9% (6%–12%) for G3 to G5 categories of CKD. Raised systolic BP was significantly and positively associated with most of the outcomes, while female sex was significantly and negatively associated with albuminuria (odds ratio [OR], 0.53; 95% confidence interval [CI], 0.33–0.84 [*P*=.007]). Increasing BMI was significantly and negatively associated with G3 to G5 categories of CKD estimated by the CG equation and as a consequence, overweight/obesity was associated with lower odds of these categories of CKD (OR, 0.48; 95% CI, 0.28–0.82 [*P*=.007]). The presence of dyslipidemia was significantly and negatively associated with G3 to G5 categories of CKD estimated by CG (OR, 0.49; 95% CI, 0.24–0.99 [*P*=.048]), while the increasing number of

TABLE III. Characteristics According to Albuminuria, GFR Categories, and CKD Based on the Four-Variable MDRD Equation

	Albuminuria-Based CKD			GFR-Based CKD Stages			CKD		
Variable	A1	A2 to A3	*P* Value	G1 and G2	G3 to G5	*P* Value	No	Yes	*P* Value
Participants, No. (%)	220 (65.5)	116 (34.5)		227 (67.5)	109 (32.5)		169 (50.3)	167 (49.7)	
Women, No. (%)	151 (68.6)	62 (53.4)	.009	142 (62.5)	71 (65.1)	.717	111 (65.7)	102 (61.1)	.428
Mean age, y (SD)	60.5 (11.4)	61.7 (11.1)	.369	59.1 (11.4)	64.6 (10.1)	<.001	58.6 (11.4)	63.2 (10.7)	<.001
Diabetes, No. (%)	35 (15.9)	27 (23.3)	.105	35 (15.4)	27 (24.8)	.05	28 (16.6)	34 (20.3)	.401
Gout, No. (%)	20 (9.1)	16 (13.8)	.197	20 (8.8)	16 (14.7)	.131	14 (8.3)	22 (13.2)	.161
Dyslipidemia, No. (%)	40 (18.2)	19 (16.4)	.764	44 (19.4)	15 (13.8)	.224	34 (20.1)	25 (15.0)	.252
Smoking, No. (%)	9 (4.1)	4 (4.4)	>.999	10 (4.4)	3 (2.7)	.559	8 (4.7)	5 (3.0)	.573
HIV, No. (%)	9 (4.1)	3 (2.6)	.193	10 (4.4)	2 (1.8)	.297	8 (4.7)	4 (2.4)	.171
Self-medication, No. (%)	84 (38.2)	42 (36.2)	.813	86 (37.9)	40 (36.7)	.904	65 (38.4)	61 (36.5)	.736
Mean duration hypertension, y (SD)	7.1 (8.0)	5.8 (7.5)	.164	6.1 (7.9)	7.8 (7.6)	.053	6.4 (7.9)	6.9 (7.8)	.551
Mean SBP, mm Hg (SD)	152.0 (23.8)	158.6 (28.4)	.025	152.4 (23.4)	158.3 (29.5)	.047	151.9 (23.4)	156.8 (27.6)	.080
Mean DBP, mm Hg (SD)	89.7 (13.8)	91.8 (16.1)	.227	89.9 (14.4)	91.5 (15.1)	.345	89.7 (14.0)	91.7 (15.2)	.387
Controlled hypertension, No. (%)	48 (21.8)	23 (19.8)	.779	50 (22.0)	21 (19.3)	.669	36 (21.3)	35 (20.9)	>.999
Antihypertensive drugs ≥3, No. (%)	40 (18.2)	23 (19.8)	.749	40 (17.6)	23 (21.1)	.557	30 (17.7)	33 (19.8)	.965
Mean BMI, kg/m^2 (SD)	29.6 (17.7)	28.9 (5.9)	.696	30.2 (17.6)	27.6 (4.8)	.131	30.2 (20.0)	28.6 (5.6)	.310
BMI ≥25 kg/m^2, No. (%)	146 (66.4)	85 (73.3)	.217	157 (69.2)	74 (67.9)	.803	112 (66.3)	119 (71.2)	.348
Mean fasting capillary glucose, g/L (SD)	0.99 (0.4)	1.04 (0.3)	.371	0.99 (0.3)	1.04 (0.4)	.254	1.00 (0.4)	1.02 (0.4)	.578

Abbreviations: BMI, body mass index; CKD, chronic kidney disease; DBP, diastolic blood pressure; GFR, glomerular filtration rate; HIV, human immunodeficiency virus; MDRD, Modification of Diet in Renal Disease; SBP, systolic blood pressure; SD, standard deviation.

TABLE IV. Predictors of Albuminuria and G3 to G5 Categories and CKD Based on the Four-Variable MDRD Equation in Age- and Sex-Adjusted Logistic Regressions

	Albuminuria		CKD Stages G3 to G5		CKD	
Variable	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value
Women	0.53 (0.33–0.84)	.007	1.20 (0.73–1.95)	.475	0.86 (0.55–1.36)	.525
Age, y	1.01 (0.99–1.03	.462	1.05 (1.02–1.07)	<.001	1.04 (1.02–1.06)	<.001
Diabetes	1.43 (0.81–2.55)	.220	1.66 (0.92–2.98)	.092	1.11 (0.63–1.97)	.709
Gout	1.29 (0.62–2.66)	.495	1.75 (0.84–3.66)	.134	1.45 (0.70–3.03)	.315
Dyslipidemia	0.86 (0.47–1.57)	.617	0.63 (0.33–1.22)	.173	0.66 (0.37–1.19)	.168
Smoking	0.65 (0.19–2.21)	.489	0.73 (0.19–2.83)	.649	0.63 (0.19–2.04)	.444
Self-medication	0.88 (0.55–1.42)	.609	0.96 (0.59–1.56)	.870	0.89 (0.57–1.40)	.626
Duration of hypertension, y	0.98 (0.95–1.01)	.159	1.01 (0.98–1.04)	.371	1.00 (0.97–1.02)	.774
Number of antihypertensive drugs	1.02 (0.78–1.32)	.882	1.26 (0.96–1.66)	.101	1.08 (0.84–1.38)	.559
SBP, mm Hg	1.01 (1.00–1.02)	.018	1.01 (1.00–1.02)	.031	1.01 (1.00–1.02)	.049
DBP, mm Hg	1.01 (0.99–1.03)	.204	1.01 (1.00–1.03)	.080	1.01 (1.00–1.03)	.127
BMI, kg/m^2	1.00 (0.98–1.02)	.953	0.97 (0.93–1.01)	.200	0.99 (0.98–1.01)	.553
BMI ≥25 kg/m^2	1.64 (0.97–2.77)	.063	1.18 (0.70–1.98)	.543	1.63 (0.99–2.67)	.053
Fasting capillary glucose	1.20 (0.67–2.08)	.526	1.28 (0.72–2.28)	.390	1.06 (0.61–1.84)	.826
Hyperglycemia	1.51 (0.80–2.83)	.201	1.60 (0.84–3.05)	.156	1.10 (0.58–2.06)	.771
Controlled hypertension	0.92 (0.52–1.63)	.769	0.69 (0.38–1.24)	.215	0.85 (0.50–1.47)	.574

Abbreviations: BMI, body mass index; CI, confidence interval; CKD, chronic kidney disease; DBP, diastolic blood pressure; MDRD, Modification of Diet in Renal Disease; OR, odds ratio; SBP, systolic blood pressure.

antihypertensive drugs was significantly and positively associated with these stages using the same equation (OR, 1.44; 95% CI, 1.06–1.94 [*P*=.007]).

DISCUSSION

Our study among this group of hypertensive Cameroonians revealed that nearly half had CKD regardless of the estimators used. Based on the KDIGO classification of CKD,[25] about one third of participants had albuminuria A2 to A3 categories and were in stages CKD G3 to G5 regardless of the equation used to estimate GFR. Albuminuria and G3 to G5 categories of CKD were predicted by advanced age, raised systolic BP, adiposity, and increased number of antihypertensive drugs.

Across estimators of GFR, the CG equation diagnosed more participants with CKD while CKD-EPI and MDRD diagnosed the same proportion of participants with CKD, largely in line with existing and extensively discussed reports from the general population.[8,27,28] Although none of these equations has been validated in Africans populations, the similar yields of the MDRD and CKD-EPI equations suggest their possible applicability in this population, based on the results of previous studies and guidelines recommendations, while awaiting development of an appropriate CKD equation for Africans.[25,27,28]

We observed that nearly half of the study participants presented with CKD, a high prevalence of CKD similar to data reported in other SSA countries ranging from 38.2% to 46.9% depending on the definition of CKD, and the estimation method of GFR and proteinuria.[9–12] This could be related to the high proportion of those with uncontrolled hypertension and the high prevalence of endemic infections including HIV, hepatitis B and C virus, and bacterial and parasitic infections, which were not screened in this study. It can also be favored by the severity of the disease, as suggested by the association of prevalent CKD with raised systolic BP and increased number of antihypertensive drugs used, despite the fact that nearly 60% of patients were taking an angiotensin-converting enzyme inhibitor or an angiotensin receptor blocker, in line with guidelines.[29–31] It is well documented that hypertensive patients with longer duration of the disease and difficulties in achieving BP control and who are less respondent to acute BP treatment or require increasing numbers of antihypertensive drugs are more likely to have associated target organ damage and underlying kidney disease.[3,11,12,32] All the above features were predictors of CKD in our patients. Moreover, the high frequency of well-known CKD initiation and progression of risk factors observed in this population could further contribute to the observed high prevalence as reported in previous SSA studies.[9,10]

The African ethnicity of our participants is another explanation of the high prevalence of CKD. Indeed, Africans are at high risk for hypertension-associated renal disease, which is the second worldwide leading cause of ESRD.[3] Compared with Caucasians, they are five to eight times more likely to develop ESRD from hypertension-associated renal diseases and show a faster decline in renal function at similar BP levels.[3,13,14] However, with the late referral of CKD patients to the nephrologist in this setting, mostly at an ESRD stage when kidney biopsy is less contributive in determining the cause of CKD,[33] the high frequency of CKD attributed to hypertension-associated renal diseases in

Africans could be related to MYH9 gene polymorphisms. This may be associated with focal and segmental sclerosis and may explain the poor response to BP control in those with a clinical diagnosis of hypertension-associated renal diseases.[34,35]

The reported high prevalence of adiposity in this study could be an additional contributor to the high prevalence of CKD observed elsewhere.[9,10] Obesity is a well-known risk factor for CKD, which leads to albuminuria probably secondary to the hyperfiltration state with focal and segmental glomerulosclerosis lesions.[25] We also observed that advanced age was predictive of prevalent CKD as well described in the literature.[9,10,25] This could be related to arterial stiffness, abnormalities of vascular reactivity, and self-medication including anti-inflammatory drugs.[3]

Study Limitations and Strengths

The present study has some limitations including the semiquantitative assessment of urinary albumin excretion using dipsticks, the nonvalidation of any of the equations used in Africans populations, the nonsystematic screening of participants for endemic infections conferring high risk for CKD such as HIV infection, hepatitis B and C viral infection, and the nonexhaustive assessment of socioeconomic status, which has been shown to be associated with CKD.[9,36,37] However, this study to our knowledge is the first in SSA to provide data on the epidemiology of kidney disease among hypertensive patients using the three estimators of GFR with a 3-month confirmation of the chronicity according to the KDIGO guidelines for CKD screening.[25] It also provides a complete picture of CKD prevalence including albuminuria and GFR categories as well as their determinants in the African population with hypertension.

CONCLUSIONS

This study revealed that nearly half of hypertensive patients had CKD regardless of the estimators used. The high prevalence of CKD as well as albuminuria and advanced stages of CKD was predicted by advanced age, raised systolic BP, adiposity, and increased number of antihypertensive drugs. These results invite actions for adequate management of hypertension, systematic screening of hypertensive patients for CKD, and early referral to nephrologists for optimal management.

Acknowledgments: We thank the staff of the Yaoundé Central and University Teaching Hospitals hypertension clinics and the biochemistry laboratory technicians of the Yaoundé University Teaching Hospital.

Disclosures: The authors report no specific funding in relation to this research and no conflicts of interest to disclose.

Authors' Contributions: FFK: Conception and design of the study, supervision of data collection, interpretation of data, and drafting of the manuscript. APK: Data analysis and interpretation and drafting of the manuscript. CTM: Conception and design of the study, data collection, and critical revision of the manuscript. MPH: Conception and design of the study and critical revision of the manuscript. EY: Conception and design of the study and critical revision of the manuscript. KBN: Conception and design of the study and critical revision of the manuscript. All authors approved the final manuscript.

References

1. Kearney PM, Whelton M, Reynolds K, et al. Global burden of hypertension: analysis of worldwide data. *Lancet*. 2005;365:217–223.
2. Ataklte F, Erqou S, Kaptoge S, et al. Burden of undiagnosed hypertension in sub-Saharan Africa: a systematic review and meta-analysis. *Hypertension* 2014;65:291–298.
3. Barri YM. Hypertension and kidney disease: a deadly connection. *Curr Hypertens Rep*. 2008;10:39–45.
4. World Kidney Day. World kidney day: FAQS. Available from: http://www.worldkidneyday.org/faqs Accessed 13, August 2014. 2014.
5. Lozano R, Naghavi M, Foreman K, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*. 2012;380:2095–2128.
6. Naicker S. End-stage renal disease in sub-Saharan Africa. *Ethn Dis* 2009;1(suppl 1):S1–S13.
7. U S Renal Data System. *Atlas of Chronic Kidney Disease and End-Stage Renal Disease in the United States*. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2010. USRDS 2010 Annual Data Report.
8. Sumaili EK, Krzesinski JM, Zinga CV, et al. Prevalence of chronic kidney disease in Kinshasa: results of a pilot study from the Democratic Republic of Congo. *Nephrol Dial Transplant*. 2009;24:117–122.
9. Sumaili EK, Cohen EP, Zinga CV, et al. High prevalence of undiagnosed chronic kidney disease among at-risk population in Kinshasa, the Democratic Republic of Congo. *BMC Nephrol*. 2009;10:18.
10. Osafo C, Mate-Kole M, Affram K, Adu D. Prevalence of chronic kidney disease in hypertensive patients in Ghana. *Ren Fail*. 2011;33:388–392.
11. Lengani A, Laville M, Serme D, et al. Renal insufficiency in arterial hypertension in black Africa. *Presse Med*. 1994;23:788–792.
12. Laville M, Lengani A, Serme D, et al. Epidemiological profile of hypertensive disease and renal risk factors in black Africa. *J Hypertens*. 1994;12:839–843; discussion 845.
13. Hsu CY, Lin F, Vittinghoff E, Shlipak MG. Racial differences in the progression from chronic renal insufficiency to end-stage renal disease in the United States. *J Am Soc Nephrol*. 2003;14:2902–2907.
14. Wright JT Jr, Kusek JW, Toto RD, et al. Design and baseline characteristics of participants in the African American Study of Kidney Disease and Hypertension (AASK) Pilot Study. *Control Clin Trials*. 1996;17(4 Suppl):3S–16S.
15. Naicker S. End-stage renal disease in sub-Saharan and South Africa. *Kidney Int Suppl*. 2003;83:S119–S122.
16. Mbanya JC, Minkoulou EM, Salah JN, Balkau B. The prevalence of hypertension in rural and urban Cameroon. *Int J Epidemiol*. 1998;27:181–185.
17. Katte JC, Dzudie A, Sobngwi E, et al. Coincidence of diabetes mellitus and hypertension in a semi-urban Cameroonian population: a cross-sectional study. *BMC Public Health*. 2014;14:696.
18. Chalmers J, MacMahon S, Mancia G, et al. 1999 World Health Organization-International Society of Hypertension Guidelines for the management of hypertension. Guidelines sub-committee of the World Health Organization. *Clin Exp Hypertens*. 1999;21:1009–1060.
19. O'Brien E. A website for blood pressure measuring devices: dableducational.com. *Blood Press Monit*. 2003;8:177–180.
20. Rule AD, Bailey KR, Schwartz GL, et al. For estimating creatinine clearance measuring muscle mass gives better results than those based on demographics. *Kidney Int*. 2009;75:1071–1078.
21. Levey AS, Coresh J, Greene T, et al. Expressing the Modification of Diet in Renal Disease Study equation for estimating glomerular filtration rate with standardized serum creatinine values. *Clin Chem*. 2007;53:766–772.
22. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. *Nephron*. 1976;16:31–41.
23. Levey AS, Coresh J, Greene T, et al. Using standardized serum creatinine values in the modification of diet in renal disease study equation for estimating glomerular filtration rate. *Ann Intern Med*. 2006;145:247–254.
24. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. *Ann Intern Med*. 2009;150:604–612.
25. K/DIGO Guidelines. Definition and classification. *Kidney Int Suppl* 2013;3:19–62.

26. Peduzzi P, Concato J, Kemper E, et al. A simulation study of the number of events per variable in logistic regression analysis. *J Clin Epidemiol*. 1996;49:1373–1379.
27. Matsha TE, Yako YY, Rensburg MA, et al. Chronic kidney diseases in mixed ancestry south African populations: prevalence, determinants and concordance between kidney function estimators. *BMC Nephrol*. 2013;14:75.
28. Eastwood JB, Kerry SM, Plange-Rhule J, et al. Assessment of GFR by four methods in adults in Ashanti, Ghana: the need for an eGFR equation for lean African populations. *Nephrol Dial Transplant*. 2010;25:2178–2187.
29. KDIGO Blood Pressure Work Group. KDIGO clinical practice guideline for the management of blood pressure in chronic kidney disease. *Kidney Int*. 2012;2(Suppl):337–414.
30. Mancia G, Fagard R, Narkiewicz K, et al. 2013 ESH/ESC Guidelines for the management of arterial hypertension: the Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *J Hypertens*. 2013;31:1281–1357.
31. James PA, Oparil S, Carter BL, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA*. 2014;311:507–520.
32. Klahr S, Levey AS, Beck GJ, et al. The effects of dietary protein restriction and blood-pressure control on the progression of chronic renal disease. Modification of Diet in Renal Disease Study Group. *N Engl J Med*. 1994;330:877–884.
33. Halle MP, Kengne AP, Ashuntantang G. Referral of patients with kidney impairment for specialist care in a developing country of sub-Saharan Africa. *Ren Fail*. 2009;31:341–348.
34. Freedman BI, Hicks PJ, Bostrom MA, et al. Polymorphisms in the non-muscle myosin heavy chain 9 gene (MYH9) are strongly associated with end-stage renal disease historically attributed to hypertension in African Americans. *Kidney Int*. 2009;75:736–745.
35. Matsha TE, Masconi K, Yako YY, et al. Polymorphisms in the non-muscle myosin heavy chain gene (MYH9) are associated with lower glomerular filtration rate in mixed ancestry diabetic subjects from South Africa. *PLoS ONE*. 2012;7:e52529.
36. Stanifer JW, Jing B, Tolan S, et al. The epidemiology of chronic kidney disease in sub-Saharan Africa: a systematic review and meta-analysis. *Lancet Glob Health*. 2014;2:e174–e181.
37. Amato D, Alvarez-Aguilar C, Castaneda-Limones R, et al. Prevalence of chronic kidney disease in an urban Mexican population. *Kidney Int Suppl*. 2005;97:S11–S17.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Table SI. Characteristics according to glomerular filtration rate categories and chronic kidney disease based on Cockroft-Gault and CKD-EPI equations.

Table SII. Predictors of G3 to G5 categories and chronic kidney disease based on Cockroft-Gault and CKD-EPI equations in age- and sex-adjusted logistic regressions.