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Ultrasonographic Renal Biometry in Diabetic Chronic Kidney Disease at Saint Joseph Hospital in Kinshasa, Democratic Republic of the Congo

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ABSTRACT

Introduction
Chronic kidney disease (CKD) is a major global public health challenge, particularly among patients with diabetes mellitus. In sub-Saharan Africa, ultrasonographic data on renal biometry in diabetic CKD remain limited, and local data from the Democratic Republic of the Congo (DRC) are scarce.

Purpose
To evaluate renal dimensions using transabdominal ultrasonography in patients followed for diabetic CKD at Saint Joseph Hospital in Kinshasa, to compare these measurements with those of patients with non-diabetic CKD, and to analyze morphological variations according to clinicobiological and ultrasonographic stages.

Methods
A single-center cross-sectional study was conducted from October 1, 2020, to September 30, 2021. A total of 160 adult patients with nephrologist-confirmed CKD were consecutively enrolled, including 79 diabetic and 81 non-diabetic patients. Clinical, biological, and ultrasonographic data were collected using a standardized case report form. Ultrasonographic measurements included renal long axis (bipolar length), short axis, width, cortical thickness, parenchymal echogenicity, corticomedullary (parenchymo-sinusal) differentiation, and interlobar renal resistive indices (RIs). Statistical comparisons were performed using the Chi-square test, Fisher's exact test, Student's independent t-test, analysis of variance (ANOVA), and paired t-tests, with statistical significance set at $p < .05$.

Results
The mean age of the cohort was 57.3 ± 11.7 years. The mean right renal long axis was significantly larger in diabetic patients than in non-diabetic patients (101.4 ± 10.1 mm vs. 86.7 ± 10.6 mm; $p < .001$), as was the mean left renal long axis (107.1 ± 36.7 mm vs. 88.4 ± 11.4 mm; $p < .001$). In both cohorts, the left kidney was significantly longer than the right kidney. Renal dimensions decreased progressively with advancing CKD stages, although this reduction was substantially more pronounced in the non-diabetic group. Inter-method agreement between the Kidney Disease: Improving Global Outcomes (KDIGO) functional stages and ultrasonographic structural stages was weak (kappa = 0.15; 95% CI [0.08, 0.24]).

Conclusion
Renal ultrasonography demonstrates relative preservation of renal dimensions in diabetic patients with CKD compared to non-diabetic patients, who exhibit more marked parenchymal atrophy. In resource-limited settings, ultrasound is an invaluable tool for structural characterization, but it must complement—and not replace—laboratory-based glomerular filtration estimates for clinical staging.

INTRODUCTION

Chronic kidney disease (CKD) is a progressive, irreversible syndrome characterized by structural or functional renal abnormalities associated with elevated cardiovascular morbidity and all-cause mortality. The Kidney Disease: Improving Global Outcomes (KDIGO) clinical practice guidelines define CKD as abnormalities of kidney structure or function, present for more than 3 months, with health implications (KDIGO CKD Work Group, 2024). Globally, CKD affects over 10% of the general population, exceeding 800 million individuals, with its prevalence continuously driven upward by aging populations, hypertension, obesity, and diabetes mellitus (Francis et al., 2024; Kovesdy, 2022).

The epidemiological burden of CKD is particularly pronounced in sub-Saharan Africa. In this region, healthcare systems face severe resource constraints that restrict early screening, systematic biological monitoring, and access to specialized nephrological care (Bello et al., 2024). Recent reports from the International Society of Nephrology (ISN) underscore the stark regional inequalities regarding access to basic laboratory diagnostics, imaging modalities, and renal replacement therapies (ISN, 2023). Under these conditions, non-invasive, cost-effective, and highly reproducible diagnostic tools like renal ultrasonography remain critical for clinical assessment and decision-making.

Diabetic kidney disease (DKD) is characterized by distinct, stage-dependent morphological patterns. In the early stages of diabetes, renal hyperfiltration and hypertrophy lead to preserved or significantly increased renal volume and cortical thickness (Mogensen & Andersen, 1973). As the disease advances to chronic stages, progressive glomerulosclerosis and interstitial fibrosis eventually trigger renal volume reduction, yet these dimensions often remain relatively preserved compared to non-diabetic etiologies of CKD, where cortical atrophy is typically earlier and more pronounced (Desta et al., 2020; Emilomo et al., 2018). Ultrasonographic biometry allows for the precise evaluation of renal bipolar length, cortical thickness, parenchymal echogenicity, and corticomedullary differentiation, serving as a vital surrogate marker for histopathological changes.

In the Democratic Republic of the Congo (DRC), peer-reviewed data addressing ultrasonographic renal biometry in patients with diabetic CKD remain extremely scarce. This diagnostic knowledge gap limits the ability of local clinicians to interpret renal dimensions in diabetic patients presenting with impaired kidney function.

Study Objectives and Hypotheses

The primary objective of this study was to compare ultrasonographic renal dimensions between diabetic and non-diabetic CKD patients followed at Saint Joseph Hospital in Kinshasa.

Our primary hypothesis was that diabetic CKD patients exhibit significantly larger (more preserved) renal dimensions compared to non-diabetic CKD patients at comparable levels of renal dysfunction. Secondary objectives included:

a. Describing variations in renal dimensions across KDIGO functional stages.
b. Comparing measurements between the right and left kidneys.
c. Assessing the level of agreement between KDIGO clinicobiological staging and structural ultrasonographic staging.

METHODS

Study Design, Setting, and Period

This single-center, cross-sectional, descriptive, and analytical study was conducted in the Department of Radiology and Medical Imaging at Saint Joseph Hospital—a tertiary reference facility in Kinshasa, DRC—from October 1, 2020, to September 30, 2021. The study protocol was approved by the Institutional Review Board/Ethics Committee of the School of Public Health at the University of Kinshasa.

Participant Recruitment and Eligibility Criteria

A consecutive, non-probabilistic sampling strategy was used to recruit adult patients referred for renal ultrasonography who were being actively managed for CKD by the hospital's nephrology service. No a priori sample size calculation was performed; instead, all eligible patients presenting during the 12-month study window were consecutively enrolled to maximize

statistical power, yielding a final sample size of 160 patients (79 diabetic and 81 non-diabetic).

Inclusion Criteria:

i. Age ≥ 18 years.
ii. Confirmed diagnosis of CKD established by a staff nephrologist (based on KDIGO criteria).
iii. Availability of comprehensive clinical records and recent serum biochemistry (urea, creatinine, eGFR).
iv. Successful completion of a high-quality, interpretable transabdominal renal ultrasound.
v. Provision of written informed consent.

Exclusion Criteria:

i. Polycystic kidney disease or major congenital renal malformations.
ii. Known renal space-occupying lesions (benign or malignant tumors).
iii. Unilateral or bilateral renal artery stenosis.
iv. Superimposed acute kidney injury (AKI) or acute-on-chronic renal failure.
v. History of renal surgery (e.g., partial/total nephrectomy) likely to distort native biometry.

Ultrasonographic Protocol

All ultrasound examinations were performed using a Sonoscape S40 ultrasound system (SonoScape Medical Corp., Shenzhen, China; commissioned in 2018) equipped with a 2.0–5.0 MHz multi-frequency convex transducer. To standardize pelvic and abdominal acoustic windows, patients were scanned with a moderately distended bladder (achieved by instructing patients to avoid voiding for 3 to 4 hours prior to the exam while maintaining normal fluid intake).

Scanning was performed in multiple patient positions:

Right Kidney: Evaluated via a lateral or anterolateral approach at the right flank with the patient in the left lateral decubitus position.

Left Kidney: Evaluated via a lateral or posterolateral approach at the left flank with the patient in the right lateral decubitus position.

Deep inspiration and brief breath-holding maneuvers were utilized to displace the liver and spleen and optimize visualization of the superior and inferior renal poles.

For each kidney, primary biometric dimensions were recorded:

a) *Long Axis (Bipolar Length):* Measured as the maximum distance between the upper and lower poles on a longitudinal plane showing the central sinus echoes.
b) *Short Axis (Anteroposterior Diameter):* Measured on a true transverse plane at the level of the renal hilum.
c) *Width (Transverse Diameter):* Measured from the lateral border to the medial renal lip at the level of the hilum on the transverse view.
d) *Cortical Thickness:* Measured from the outer margin of the renal cortex to the outer margin of the renal pyramids (medullary pyramids), strictly avoiding the columns of Bertin and areas with acoustic shadowing.
e) *Renal Resistive Index (RI):* Obtained by performing pulsed-wave Doppler at the level of the interlobar arteries (at the margins of the renal pyramids). The RI was calculated using the standard formula:

$$RI = \frac{\text{Peak Systolic Velocity - End DiastolicVelocity}}{\text{Peak Systolic Velocity}}$$

All scans were performed by an experienced radiologist or a senior radiology resident under direct faculty supervision. To ensure technical accuracy, measurements were repeated thrice, and the average value was recorded. Interobserver and intraobserver variabilities were not formally quantified, which represents a methodological limitation of this study.

Variables and Operational Definitions

1. *Chronic Kidney Disease (CKD):* Defined according to the KDIGO guidelines as an estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m² or the presence of structural/markers of kidney damage persisting for ≥ 3 months (KDIGO CKD Work Group, 2024).

2. *Glomerular Filtration Rate Estimation:* Calculated from serum creatinine values using the available laboratory records. Since different formulas (e.g., MDRD or CKD-EPI) were used across partner laboratories without consistent documentation, this variability was noted as a limitation.

3. *Diabetes Mellitus:* Confirmed based on documented clinical history, active use of oral hypoglycemic agents or insulin, or persistent chronic fasting hyperglycemia (≥ 126 mg/dL) documented by the managing clinician. Differentiation between type 1 and type 2 diabetes relied on clinical presentation, age of onset, c-peptide levels (where available), and treatment history.
4. *Ultrasonographic Staging of CKD:* Based on a validated 4-tier morphological classification (Zongo et al., 2001):
   - *Stage 0:* Normal renal size and cortical echogenicity (hypoechoic compared to liver/spleen parenchyma).
   - *Stage I:* Cortical echogenicity equal to that of the liver or spleen (isoechoic) with preserved corticomedullary differentiation.
   - *Stage II:* Cortical echogenicity greater than that of the liver or spleen (hyperechoic) with partially preserved corticomedullary differentiation.
   - *Stage III:* Marked cortical hyperechogenicity with complete loss of corticomedullary differentiation (parenchymo-sinusal blurring) and/or marked renal atrophy.

Table 1:
Clinical and Functional Staging of CKD Based on KDIGO Guidelines

KDIGO Stage	Functional Description	eGFR Range (mL/min/1.73 m²)
G1	Normal or high kidney function with structural markers	≥ 90
G2	Mildly decreased GFR with structural markers	60–89
G3a	Mildly to moderately decreased GFR	45–59
G3b	Moderately to severely decreased GFR	30–44
G4	Severely decreased GFR	15–29
G5	Kidney failure (End-stage kidney disease)	< 15

Statistical Analysis

Data were cleaned, coded, and entered into Microsoft Excel 2010, and analyzed using SPSS Version 24.0 (IBM Corp., Armonk, NY, USA).

Descriptive Statistics: Continuous variables were expressed as mean ± standard deviation (SD) after confirming normality using the Kolmogorov-Smirnov test. Categorical variables were expressed as frequencies and percentages.

Bivariate Analyses:

- ✧ Group comparisons of continuous variables between diabetic and non-diabetic patients were performed using Student's independent t-test.
- ✧ Comparisons across multiple groups (e.g., across KDIGO stages) were evaluated using one-way Analysis of Variance (ANOVA).
- ✧ Paired continuous data (e.g., comparing right versus left kidney measurements within the same patient) were analyzed using Student's paired t-test.
- ✧ Categorical distributions were compared using Pearson's Chi-square (χ^2) test or Fisher's exact test where cell counts were < 5.

Agreement Analysis: Inter-method agreement between the clinical/functional KDIGO stages (ordinal scale) and the structural ultrasonographic stages (ordinal scale) was quantified using Cohen's weighted kappa (k) coefficient with its 95% confidence interval (CI). Kappa values were interpreted as: <0.20 (poor/weak agreement), 0.21–0.40 (fair), 0.41–0.60 (moderate), 0.61–0.80 (substantial), and 0.81–1.00 (almost perfect).

Missing Data: Analyses were performed on a complete-case basis; missing variables were excluded without statistical imputation. A two-tailed p-value < .05 was considered statistically significant.

Ethical considerations

The study was approved by the Ethics Committee of the School of Public Health of the University of Kinshasa, acting as the National Ethics Committee (approval No. ESP/CE/27B/2020). Data confidentiality was respected. All included patients received information on the voluntary nature of participation, and informed consent was obtained before inclusion.

RESULTS

General, Sociodemographic, and Clinical Characteristics

The study population consisted of 160 patients: 79 (49.4%) with diabetic CKD and 81 (50.6%) with non-diabetic CKD.

The mean age of the overall population was 57.3 ± 11.7 years. Diabetic patients were significantly older than non-diabetic patients (59.6 ± 11.8 years vs. 55.1 ± 11.1 years; p = .041). A slight male predominance was observed (53.1% overall), with no significant differences in sex distribution between the two cohorts.

Table 2:
Sociodemographic Characteristics of the Study Population

Variables	Overall Cohort (N=160)	Diabetic Patients (n=79)	Non-Diabetic Patients (n=81)	Statistical Significance (p)
Age (years)	**57.3 ± 11.7**	**59.6 ± 11.8**	**55.1 ± 11.1**	**.041**
< 60years	89 (55.6%)	38 (48.1%)	51 (63.0%)	
≥ 60years	71 (44.4%)	41 (51.9%)	30 (37.0%)	
Sex				**.756**
Male	85 (53.1%)	41 (51.9%)	44 (54.3%)	
Female	75 (46.9%)	38 (48.1%)	37 (45.7%)	

Note: Statistically significant (p < .05).

Clinical parameters revealed that systemic hypertension (64.2% vs. 45.6%; p = .013) and lower limb edema (71.6% vs. 50.6%; p = .005) were significantly more prevalent among non-diabetic patients. Active vomiting (uremic symptom) was exclusively reported in the non-diabetic cohort (25.9% vs. 0.0%; p < .001). Conversely, mean Body Mass Index (BMI) was significantly higher in diabetic patients (26.7 ± 4.7 kg/m² vs. 25.0 ± 4.6 kg/m²; p = .027).

Table 3:
Clinical Presentation and Hemodynamic Parameters

Clinical Variables	Overall Cohort (N=160)	Diabetic Patients (n=79)	Non-Diabetic Patients (n=81)	Statistical Significance (p)
Hypertension	88 (55.0%)	36 (45.6%)	52 (64.2%)	.013
Lower limb edema	98 (61.3%)	40 (50.6%)	58 (71.6%)	.005
Vomiting	21 (13.1%)	0 (0.0%)	21 (25.9%)	< .001
SBP (mmHg)	133.5 ± 20.5	134.0 ± 23.0	133.0 ±17.8	.755
DBP (mmHg)	82.2 ± 10.8	80.7 ±11.2	83.7 ±10.4	.084
BMI (kg/m²)	25.8 ± 4.7	26.7 ± 4.7	25.0 ±4.6	.027

Note: SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; BMI: Body Mass Index. Statistically significant (p < .05).

Biological Profiles and Diabetic Subtypes

Among the 79 diabetic patients, type 2 diabetes mellitus was highly predominant (n = 67; 84.8%), while type 1 diabetes was present in 12 patients (15.2%). Over half of the diabetic cohort (54.4%) had a documented duration of diabetes exceeding 10 years.

While mean blood urea nitrogen and serum creatinine levels did not differ significantly between the two groups, the mean eGFR was significantly lower in the non-diabetic cohort (34.5 ± 13.4 mL/min/1.73 m² vs. 48.0 ± 14.1 mL/min/1.73 m²; p = .004), indicating more advanced functional impairment in the non-diabetic group at presentation.

Table 4:
Comparative Serum Biochemistry

Biological Parameters	Overall Cohort (N=160)	Diabetic Patients (n=79)	Non-Diabetic Patients (n=81)	Statistical Significance (p)
Urea (mg/dL)	96.2 ± 27.6	95.8 ± 30.7	96.5 ± 26.7	.932
Creatinine (mg/dL)	2.6 ± 0.4	2.6 ± 0.9	2.6 ± 0.3	.987
eGFR (mL/min/1.73 m²)	41.2 ± 29.9	48.0 ±14.1	34.5 ± 13.4	.004

Note: eGFR: estimated Glomerular Filtration Rate. Statistically significant (p < .05).

Comparative Renal Biometry and Doppler Indices

Ultrasonographic renal measurements demonstrated that diabetic patients had significantly larger renal dimensions compared to non-diabetic patients across almost all parameters.

Right Kidney: Bipolar length (101.4 ± 10.1 mm vs. 86.7 ± 10.6 mm; p < .001), short axis (42.9 ± 5.8 mm vs. 32.4 ± 6.3 mm; p < .001), and width (56.9 ± 8.9 mm vs. 45.5 ± 7.3 mm; p < .001) were significantly higher in the diabetic cohort.

Left Kidney: Similarly, bipolar length (107.1 ± 36.7 mm vs. 88.4 ± 11.4 mm; p < .001), short axis (45.4 ± 6.5 mm vs. 34.7 ± 6.4 mm; p < .001), and width (58.2 ± 8.3 mm vs. 47.9 ± 6.9 mm; p < .001) were significantly larger in diabetic patients.

Intriguingly, cortical thickness was significantly lower in diabetic patients than in non-diabetic patients in both the right kidney (7.8 ± 1.1 mm vs. 8.8 ± 1.5 mm; p < .001) and left kidney (7.9 ± 1.0 mm vs. 8.9 ± 1.6 mm; p < .001). Interlobar renal resistive indices (RI) were slightly higher in diabetic patients, reaching statistical significance on the right side (0.72 ± 0.07 vs. 0.69 ± 0.07; p = .026).

Table 5:
Ultrasonographic Biometry and Doppler Indices of the Right Kidney

Morphological Parameters	Overall Cohort (N=160)	Diabetic Patients (n=79)	Non-Diabetic Patients (n=81)	Statistical Significance (p)
Long axis (mm)	93.9 ±12.7	101.4 ±10.1	86.7 ±10.6	< .001
Short axis (mm)	37.7 ±7.9	42.9 ±5.8	32.4 ±6.3	< .001
Width (mm)	51.1 ±9.9	56.9 ±8.9	45.5 ±7.3	< .001
Cortical thickness (mm)	8.3 ±1.4	7.8 ±1.1	8.8 ±1.5	< .001
Interlobar RI	0.71 ±0.07	0.72 ±0.07	0.69 ±0.07	

Note: RI: Resistive Index. Statistically significant (p < .05).

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Table 6:
Ultrasonographic Biometry and Doppler Indices of the Left Kidney

Morphological Parameters	Overall Cohort (N=160)	Diabetic Patients (n=79)	Non-Diabetic Patients (n=81)	Statistical Significance (p)
Long axis (mm)	97.7 ± 28.5	107.1 ± 36.7	88.4 ± 11.4	< .001
Short axis (mm)	39.9 ± 8.4	45.4 ± 6.5	34.7 ± 6.4	< .001
Width (mm)	53.0 ± 9.2	58.2 ± 8.3	47.9 ± 6.9	< .001
Cortical thickness (mm)	8.4 ± 1.5	7.9 ± 1.0	8.9 ± 1.6	< .001
Interlobar RI	0.71 ± 0.08	0.72 ± 0.09	0.70 ± 0.08	

Note: RI: Resistive Index. Statistically significant (p < .05).

Paired Comparisons of Right vs. Left Renal Bipolar Length

A paired t-test confirmed that the left kidney was significantly longer than the right kidney in both study cohorts:

Diabetic Cohort: Left kidney (107.1 ± 36.7 mm) vs. Right kidney (101.4 ± 10.1 mm); Mean difference = 5.7 mm (p = .014).

Non-Diabetic Cohort: Left kidney (88.4 ± 11.4 mm) vs. Right kidney (86.7 ± 10.6 mm); Mean difference = 1.7 mm (p = .001).

Agreement Between Clinicobiological Staging (KDIGO) and Ultrasonographic Staging

There was a highly noticeable discordance between the functional (KDIGO) stages and structural (ultrasound-derived) stages. For instance, of the 69 patients categorized under advanced functional stages (KDIGO G4 or G5), 8 patients (11.6%) still exhibited completely normal renal morphology on ultrasound (Stage 0), and 29 patients (42.0%) showed only mild structural alterations (Stage I).

Conversely, among the 13 patients with clinical Stage 1 CKD, 5 (38.5%) already displayed Stage I structural changes on ultrasound. The calculated Cohen's weighted kappa coefficient was k = 0.15 (95% CI [0.08, 0.24]), indicating weak/poor agreement between clinical staging and ultrasound findings.

Table 7:
Cross-Tabulation and Inter-Method Agreement Between KDIGO and Ultrasonographic Stages

KDIGO Stage	Ultrasound Stage 0	Ultrasound Stage I	Ultrasound Stage II	Ultrasound Stage III	Total Patients (N)
Stage 1	8	5	0	0	13
Stage 2	14	4	2	0	20
Stage 3	24	22	7	5	58
Stage 4–5	8	29	30	2	69
Total	**54**	**60**	**39**	**7**	**160**

Note: Cohen's weighted k = 0.15 (95% CI [0.08, 0.24]), indicating poor agreement.

Qualitative Structural Ultrasonographic Characteristics

Poor medullary visibility was significantly more frequent in non-diabetic patients (28.4% vs. 10.1%; p = .003). Similarly, cortical hyperechogenicity (38.3% vs. 19.0%; p = .018) and complete loss of corticomedullary differentiation (69.1% vs. 6.3%; p < .001) were significantly more prevalent in the non-diabetic cohort, validating the hypothesis of advanced parenchymal damage and fibrosis in the non-diabetic group.

Table 8:
Qualitative Morphological Findings of the Kidneys

Structural Parameters	Overall Cohort (N=160)	Diabetic Patients (n=79)	Non-Diabetic Patients (n=81)	Statistical Significance (p)
Medullary Visibility				**.003**
Poor	31 (19.4%)	8 (10.1%)	23 (28.4%)	
Good	129 (80.6%)	71 (89.9%)	58 (71.6%)	
Cortical Echogenicity				**.018**
Hypoechoic *(Normal)*	54 (33.8%)	28 (35.4%)	26 (32.1%)	
Isoechoic	60 (37.5%)	36 (45.6%)	24 (29.6%)	
Hyperechoic	46 (28.8%)	15 (19.0%)	31 (38.3%)	
Loss of Differentiation				**< .001**
Absent *(Preserved)*	99 (61.9%)	74 (93.7%)	25 (30.9%)	
Present *(Lost)*	61 (38.1%)	5 (6.3%)	56 (69.1%)	

Note: Statistically significant (p < .05).

DISCUSSION

This single-center study confirms that adult Congolese patients with diabetic CKD exhibit significantly larger and more preserved renal dimensions (bipolar length, short axis, and width) compared to patients with non-diabetic CKD, despite presenting with clinically comparable levels of nitrogenous waste retention (urea and creatinine). Conversely, non-diabetic patients displayed more advanced morphological deterioration, marked by smaller kidney sizes, higher rates of parenchymal hyperechogenicity, and more frequent loss of corticomedullary differentiation. These findings have immediate diagnostic and prognostic implications in resource-limited clinical environments like sub-Saharan Africa.

The sociodemographic characteristics of our cohort are highly consistent with epidemiological data from sub-Saharan Africa. The higher mean age and marked predominance of type 2 diabetes (84.8%) among our diabetic patients align with the rising global incidence of age-related metabolic syndromes (Kovesdy, 2022). Furthermore, the longer duration of diabetes (exceeding 10 years in over 50% of the sample) represents the classic

temporal profile required for microvascular complications and diabetic nephropathy to clinically manifest.

The relative preservation of renal volume in diabetic CKD observed in our study is well-supported by pathophysiological mechanisms. Early-stage diabetic nephropathy is characterized by renal hyperfiltration, glomerular capillary shear stress, and subsequent cellular hypertrophy driven by hyperglycemia-activated growth factors (Mogensen & Andersen, 1973). This initial hypertrophy delays the apparent "shrinkage" of the kidneys during progressive CKD. Our results corroborate findings by Desta et al. (2020) in Ethiopia and Emilomo et al. (2018) in Nigeria, both of whom demonstrated that diabetic patients maintain relatively larger kidney sizes despite advanced stages of chronic parenchymal disease.

An intriguing paradox in our findings was that cortical thickness was significantly lower in diabetic patients than in non-diabetic patients, despite their larger overall bipolar lengths and diameters. This suggests that the relative preservation of overall renal volume in diabetes might be driven by expansion of the renal sinus fat or medullary compartment rather than true cortical preservation, a phenomenon that warrants future histopathological and tomographic validation.

Our study demonstrated a statistically significant difference between right and left kidney lengths, with the left kidney being longer in both cohorts. This biometric discrepancy is a well-established anatomical finding globally, routinely attributed to the shorter length of the left renal artery, which leads to increased perfusion, as well as the anatomical space constraints imposed by the liver on the right side (Pedersen et al., 1993).

The very weak agreement between the KDIGO clinical classification and the structural ultrasonographic classification (k = 0.15; 95% CI [0.08, 0.24]) is one of the most clinically critical findings of this study. This morphological-functional discordance indicates that biological deterioration of the GFR often precedes macroscopic alterations visible on standard gray-scale ultrasound. In clinical practice, particularly in sub-Saharan Africa, where diagnostic imaging is sometimes used as a cheap surrogate for laboratory testing, this is a dangerous pitfall: a patient with completely normal-looking kidneys on ultrasound may already be in stage 4 or 5 CKD. Conversely, a finding of normal kidney size in a diabetic patient must never be misconstrued as "healthy" or "reversible" kidney function.

In resource-limited healthcare environments, renal ultrasound remains an invaluable, highly accessible, and non-invasive diagnostic pillar (Bello et al., 2024). It allows clinicians to rapidly rule out obstructive uropathy, detect chronic structural remodeling, and differentiate acute kidney injury (characterized by normal-to-large echogenic kidneys) from chronic, irreversible diseases (characterized by shrunken, hyperechoic kidneys with blurred corticomedullary boundaries) (Zongo et al., 2001). However, as our study clearly demonstrates, its results must always be integrated with serum chemistry and albuminuria levels.

Limitations and Strengths

Strengths: This study provides rare, systematically collected biophysical data from the Democratic Republic of the Congo, comparing similar-sized groups of diabetic and non-diabetic patients using clinical, biological, and detailed structural parameters.

Limitations: The cross-sectional design prevents us from establishing causal pathways or tracking intra-individual renal shrinkage over time. The study was single-center, limiting generalizability to the broader Congolese population. We did not formally measure interobserver or intraobserver variability, which is a known source of bias in ultrasonographic biometry. The specific equations used to calculate eGFR by different referring laboratories were not standardized, and histopathological confirmation of diabetic nephropathy (renal biopsy) was not performed due to local clinical constraints. Lastly, the lack of multivariate regression analysis limited our ability to control for confounding variables such as age, sex, BMI, and hypertension.

CONCLUSION

In conclusion, this study demonstrates that patients with diabetic CKD at Saint Joseph Hospital in Kinshasa exhibit significantly larger and more structurally preserved renal dimensions than non-diabetic CKD patients, despite similar levels of renal impairment. While renal size

decreases with advancing stages of CKD in both populations, this atrophy is significantly more pronounced in non-diabetic patients.

Due to the weak agreement observed between functional KDIGO stages and structural ultrasonographic stages, renal ultrasound must be used as a complementary tool to guide morphological characterization and rule out obstructive causes, and not as a replacement for biochemical assessment. Future prospective, multicenter, and longitudinal studies with standardized eGFR equations and multivariate models are highly recommended to establish definitive local reference values for the Congolese population.

Use of Artificial Intelligence (AI) Declaration: The authors declare that no generative artificial intelligence was used to produce, modify, or manipulate the primary research data. This manuscript underwent formal editorial and linguistic refinement under the sole responsibility and oversight of the authors.

Authors' Contributions: DLM and AMA conceived and designed the study. DLM and DMI were responsible for data acquisition and drafting the manuscript. SYT, DLM, NMK, and RNK performed the data analysis and interpretation. AMT, JMT, GLM, and MLT contributed to the critical revision of the manuscript and gave final approval for publication. .

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Informed Consent: All participants provided written informed consent prior to inclusion.

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

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Moyo, N. K.[1]: Nil identified.
Nyemabo, R. K.[1]: Nil identified.
Lelo, M. T.[1]: Nil identified.
Molua, A. A.[1,2]: Nil identified.

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