Kidney Volume in Kidney Function Assessment: Determinants and Clinical Correlates in Systemic Hypertension and Chronic Kidney Disease in Southwestern, Nigeria

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Abstract

Background: The kidney volume is a very reliable ultrasound measure, reflecting contributions from all kidney parts. It could be affected by gender, body size and disease conditions. Its use in renal function assessment is based on its correlation with the glomerular filtration rate (GFR). Objectives: To assess the determinants and clinical correlates of kidney volume in hypertension and in chronic kidney disease (CKD). Materials and Methods: The two-center study was carried out at the Federal Medical Centre, Abeokuta (June-December 2017) and Babcock University Teaching Hospital, Ilishan-Remo (August 2019-January 2020). The kidneys of sixty participants who had hypertension without kidney disease (HWKD) and 58 with CKD were scanned from the front and back and their blood samples were taken for electrolytes and hemoglobin concentration. Result: The participants with CKD were significantly older than those with hypertension, P < 0.001. The mean kidney volume of hypertensives, 132.4 ± 18.3, was significantly higher than those with CKD, 63.7 ± 5.9, P < 0.001. The glomerular filtration rate (GFR) and hemoglobin levels were significantly higher in hypertensives than in CKD, P < 0.001, P < 0.001 respectively. The systolic blood pressure (SBP), creatinine and the albumin creatinine ratio (ACR) were significantly higher in CKD than in hypertension, P < 0.001, P < 0.001 and P < 0.001 respectively. Conclusion: The mean kidney volume was higher in hypertension and in males. The GFR and hemoglobin levels were significantly
higher in hypertension than in CKD while blood pressure and ACR were significantly higher in CKD than in hypertension. Kidney volume was positively and negatively correlated with GFR and ACR respectively.

Keywords
Kidney Volume, Hemoglobin, Albumin Creatinine Ratio, Hypertension, Correlation

1. Introduction
The tradition of assessing kidney size and functional status using the kidney length is fast eroding. The kidney volume and the cortical thickness are increasingly been incorporated into renal ultrasonography (RUS) due to several factors that undermine the reliability of RUS determined kidney length [1]. The ease of determining the KL compared to the KV and the CT also contributed to its continued use in many centers particularly in resources and personnel scare settings like in most low-income nations like Nigeria [2]. Weish-Rasheid et al. found among healthy volunteers, a positive association between kidney length and the body weight [3]. Makusidi and his group found a positive correlation between the glomerular filtration rate and the kidney volume and kidney length in patients with CKD [4]. Korkmaz et al. found the renal cortical thickness as a more reliable means of assessing the renal function through RUS compared to the kidney length [5].

The Kidney volume has been reported to be the most reliable parameter for assessing the kidney functional state at ultrasonography [6]. Sanusi et al. found a significant positive correlation between the kidney volume and other markers of kidney function assessment among patients with CKD. He and his group found significant interclass agreement between the various parameters of renal function assessment [7]. Buchholz et al. found the kidney volume a very reliable RUS parameter for assessing functional status among a healthy population [8]. Jovanovic et al. [9] found no correlation between the volume and length of the kidneys in a study where the authors also found no significant kidney size reduction with age as widely reported. An argument in favor of kidney volume is the incorporation of all renal tissue unlike other parameters, such as, cortical thickness (CT) where segmental physiologic or pathologic changes affecting the assessed part may undermine the reliability of the results. Polonia et al. reported a progressive yearly decline in renal function by 3.3 ± 8.2 ml/min among Type 2 diabetics [10]. Mclachlan et al. in a longitudinal study reported that from middle age, there is a reduction in kidney size of 0.5 cm per decade [11]. The decline in kidney function from middle age can therefore be partly or wholly attributable to the reduction in kidney size. This reduction has been reported not to be uniform as the renal cortex is reported to be more affected compared with the me-
This further brings to light, the choice and reliability of each RUS measure in conditions with relative cortical sparring such as (diabetes, HIV associated nephropathy) or relative medulla disease (sickle cell nephropathy, obstructive uropathy and toxic nephropathies) or relative pelvicalyceal disease, for instance, hydronephrosis [13].

The gold standard measure for assessing kidney function, the GFR, has an advantage of relevance in acute, subacute and chronic kidney dysfunction as functional alterations are produced in responses to physiologic or pathologic changes and these commonly involve the glomerular filtration [14]. Still, a delayed response is expected when the tubulointerstitial bed is affected causing altered absorptive and secretory function which manifests with urinary changes [15]. Further delays are expected when the responses involve chronic inflammatory changes that lead to fibrosis, thickening, sclerosis, scarring and shortenings, features commonly associated with changes in sizes, texture and structure, that are assessed with RUS. Despite this lag behind in urinary (ACR) and radiologic measures (kidney volume), some reliability is still found when correlation studies are carried out to determine their associations with the GFR [16].

Anemia has been known to be associated with kidney disease and its severity can also be of prognostic value in CKD. Even within this general statement, it is reported that anemia from diseases affecting the tubulointerstitial bed tend to be more severe due to the affection of the fibroblast cells of the renal peritubularrinterstitium, where erythropoietin is produced from [17]. The findings of hyponatremia, hyperkalemia and metabolic acidosis in CKD are also related to the affection of the Na’K’ ATPase, sodium hydrogen exchanger (NaHE3), and the acid secreting Type A intercalated cells of the distal tubules [18]. The defective ion exchange at these transport and exchange levels is seen as the kidney function worsen hence hyponatremia, hypernatremia and metabolic acidosis are common from the third to fourth stage of CKD.

Many studies that assessed kidney volume were either in comparison with other RUS measures or correlations with GFR. Moreover, most of these studies were carried out in the developed world. In our study therefore, we assessed the determinants of kidney volume in hypertension and in chronic kidney disease and determined the correlation between kidney volume and serum (GFR and hemoglobin concentration), and urinary (ACR) measures of kidney function as a way of reducing the knowledge gap.

2. Materials and Method

This was a two-center, hospital based prospective study that lasted for 18 months at the Radiology suites of the Federal Medical Center Abeokuta (January-December 2017) and Babcock University Teaching Hospital (August 2019-January 2020), 118 participants, ≥16 years, who gave written informed consent were consecutively recruited. The participants were made up of 60 hypertensives and 58 with stage 3 or 4 CKD recruited from the Nephrology clinic). Diabetics with or with-
out nephropathy, and those with renal graft, solitary kidney, pelvic tumors, infections, hydronephrosis and obstructive uropathy, renal artery stenosis and missing data were excluded.

Data was taken from a structured interviewer-administered questionnaire and participants’ case notes. Sociodemographic data was retrieved from hospital’s case files. Participants’ height and weight were measured using SECA stadiometer (Amazon, United Kingdom) and SECA weighing scale (Amazon, United Kingdom), without shoes, and on light covering. The blood pressure (BP) was taken after 5 minutes rest with a mercury sphygmomanometer (ACCOSON, England). Kidneys were scanned from both front and back after a 3 hour fast. All RUS were done by the same Radiologist.

Kidney volume in cm³ was calculated by multiplying the length by breath by width in cm [19].

After RUS, blood was taken for creatinine based eGFR and hemoglobin concentration.

Definitions:
Kidney dysfunction using serum-eGFR < 60 ml/min [20].
Kidney dysfunction using urine-ACR > 3.4 mg/mmol [21].
Anemia: Hemoglobin concentration < 13 g/dl [22].
Reduced kidney volume <50 cm³ [19].

Sample size was calculated from the formula on comparative study using a previous study’s prevalence [23]. Categorical variables are presented as proportions and frequencies and compared using Chi square. Continuous variables are presented as mean with standard deviation and compared with student t-test. ANOVA was used to compare three or more variables. Correlation analyses were carried out between kidney volume and the following measures: eGFR, hemoglobin concentration and the urine albumin creatinine ratio.

### 3. Results

One hundred and eighteen adults participated and had their samples analyzed. The mean age of the participants was 53.3 ± 14.6 years, those with hypertension was 48.4 ± 7.3 and those with CKD was 58.5 ± 6.8, P < 0.001. Table 1 showed that the sociodemographic, clinical and laboratory characteristics of the participants. The mean BMI of participants with hypertension and those with CKD were 23.5 ± 6.2 and 24.4 ± 3.4 respectively, P = 0.06.

Mean kidney volume of participants with hypertension and with CKD was 132.4 ± 18.3 and 63.7 ± 5.9 respectively, P < 0.001. The radiological findings of the participants are shown in Table 2. The mean GFR of participants with hypertension and with CKD were 100.3 ± 22.5 and 46.1 ± 8.4 respectively, P < 0.001. The mean ACR of participants with hypertension and with CKD was 15.0 ± 5.7 and 35.6 ± 8.9 respectively, P < 0.001. The mean hemoglobin concentration of participants with hypertension and with CKD was 14.9 ± 1.2 and 12.2 ± 1.1 respectively, P = 0.001.

Table 3 shows participants’ characteristics based on the status of their kidney
Table 1. Sociodemographic, clinical and laboratory characteristics of participants.

| Variables        | Hypertension Mean ± SD | CKD Mean ± SD |
|------------------|------------------------|---------------|
|                  | N = 60 (%)             | N = 58 (%)    | \(X^2\) t-test | P-value |
| Gender           |                        |               |
| Males            | 38 (63.3)              | 35 (60.3)     | 0.8           | 0.05    |
| Females          | 22 (36.7)              | 23 (39.7)     |               |         |
| Age, years       |                        |               |
| 16.0 - 39.9      | 9 (15.0)               | 5 (8.6)       | 1.6           | 0.02    |
| 40.0 - 59.9      | 22 (36.7)              | 22 (38.0)     |               |         |
| ≥60.0            | 29 (48.3)              | 31 (53.4)     |               |         |
| BMI, kg/m\(^2\)  |                        |               |
| <19.5            | 8 (13.3)               | 6 (10.3)      | 1.2           | 0.04    |
| 19.5 - 24.9      | 23 (38.3)              | 24 (41.4)     |               |         |
| >25.0            | 29 (48.4)              | 28 (48.3)     |               |         |
| Mean SBP, mmHg   | 144.2 ± 7.9            | 149.3 ± 5.5   | 3.3           | 0.002   |
| Mean DBP, mmHg   | 93.4 ± 2.7             | 97.6 ± 7.1    | 1.9           | 0.04    |
| Mean ACR, mg/mmol| 21.1 ± 3.5             | 38.5 ± 9.5    | 6.8           | <0.001  |
| Mean sodium, mmol/l| 142.6 ± 7.4           | 136.1 ± 3.2   | 2.4           | 0.01    |
| Mean potassium, mmol/l| 3.7 ± 2.2          | 4.3 ± 1.8     | 2.9           | 0.003   |
| Mean Bicarbonate, mmol/l| 24.7 ± 5.8           | 19.6 ± 2.5    | 3.3           | 0.001   |
| Mean chloride, mmol/l| 104.4 ± 12.2          | 99.7 ± 9.7    | 3.1           | 0.001   |
| Mean urea, mmol/l | 8.7 ± 1.4              | 11.6 ± 3.5    | 4.9           | <0.001  |
| Uric acid, mmol/l | 0.6 ± 0.2              | 0.9 ± 0.5     | 3.8           | 0.001   |
| Mean creatinine, umol/l| 102.5 ± 8.8         | 159.2 ± 11.3  | 5.7           | <0.001  |
| Mean eGFR, ml/min | 91.6 ± 3.4             | 47.1 ± 10.6   | 7.5           | <0.001  |
| Mean Hb conc, g/dl| 14.7 ± 3.4             | 12.9 ± 2.6    | 5.2           | <0.001  |

CKD—chronic kidney disease, BMI—body mass index, SBP—systolic blood pressure, DBP—diastolic blood pressure, SPO\(_2\)—percentage oxygen saturation.

Table 2. Radiological findings of participants.

| Variables        | Hypertension Mean ± SD | CKD Mean ± SD | t-test | P-value |
|------------------|------------------------|---------------|--------|---------|
|                  | N = 60 (%)             | N = 58 (%)    |        |         |
| Mean kidney length, cm | 10.9 ± 3.5            | 9.1 ± 2.6     | 5.6    | <0.001  |
| Mean kidney breath, cm  | 4.5 ± 1.4             | 3.4 ± 1.0     | 5.9    | <0.001  |
| Mean kidney width, cm  | 2.9 ± 1.2             | 2.2 ± 1.1     | 4.2    | <0.001  |
| Mean kidney volume, cm\(^3\) | 132.4 ± 18.3       | 63.7 ± 5.9    | 8.4    | <0.001  |

CKD—chronic kidney disease.

volume. The mean age and BMI of hypertensives with reduced kidney volume was less than those with CKD that had reduced kidney volume, \(P < 0.001\) and \(P = 0.04\) respectively.

The mean systolic and diastolic blood pressure of participants with HWKD that had reduced kidney volume were lower than participants with CKD who had reduced kidney volume, \(P < 0.001\) and \(P = 0.001\) respectively. The mean serum creatinine and urine ACR of participants with HWKD who had reduced
Table 3. Participants' demographic, clinical and laboratory findings based on kidney volume status.

| Variables                  | Hypertensives KV < 50 cm³ | CKD KV < 50 cm³ | X² t-test | P-value |
|----------------------------|---------------------------|-----------------|-----------|---------|
|                            | N = 12 (%)                | N = 24 (%)      |           |         |
|                            | Mean ± SD                 | Mean ± SD       |           |         |
| Males                      | 5 (41.7)                  | 13 (54.2)       | 3.4       | 0.002   |
| Females                    | 7 (58.3)                  | 11 (45.8)       |           |         |
| Mean age, yrs              | 53.1 ± 12.3               | 60.3 ± 8.3      | 4.7       | <0.001  |
| Mean BMI, kg/m²            | 24.6 ± 5.1                | 25.9 ± 7.4      | 1.9       | 0.04    |
| Mean SBP, mmHg             | 152.6 ± 2.8               | 163.6 ± 12.4    | 5.5       | <0.001  |
| Mean DBP, mmHg             | 99.1 ± 2.3                | 105.7 ± 7.7     | 3.2       | 0.001   |
| Mean creatinine, umol/l    | 137.2 ± 5.5               | 176.4 ± 8.4     | 6.4       | <0.001  |
| Mean eGFR, ml/min          | 63.5 ± 12.8               | 45.7 ± 8.9      | 7.1       | <0.001  |
| Mean ACR, mg/mmol          | 28.4 ± 6.2                | 33.8 ± 14.7     | 4.0       | <0.001  |
| Mean Hb conc, g/dl         | 12.8 ± 4.2                | 10.9 ± 5.4      | 3.7       | <0.001  |

CKD—chronic kidney disease, SBP—systolic blood pressure, DBP—diastolic blood pressure, Hb—hemoglobin concentration, eGFR—estimated glomerular filtration rate, ACR—albumin creatinine ratio, BMI—body mass index.

kidney volume were less compared with participants with CKD who had reduced kidney volume, P < 0.001 and P < 0.001. The mean eGFR and hemoglobin concentration of participants with HWKD who had reduced kidney volume were higher than in participants with CKD who had reduced kidney volume, P < 0.001 and P < 0.001 respectively.

Pearson’s correlation analysis carried out to ascertain the strength of association between kidney volume and different assessment measures of kidney function in Table 4, showed that the positive correlation between kidney volume and GFR was stronger in CKD sufferers (OR—0.11, CI—0.114 - 0.120) than in HWKD (OR—0.098, CI—0.082 - 0.101). The positive correlation between kidney volume and hemoglobin concentration was stronger in hypertensives without kidney disease (OR—0.206, CI—0.198 - 0.245) than in CKD sufferers (OR—0.097, CI—0.095 - 0.098). The negative correlation between kidney volume and urine albumin creatinine ratio was stronger in the CKD (OR—0.128, CI—0.119 - 0.129) population than in HWKD (OR—0.056, CI—0.055 - 0.058).

4. Discussion

The kidney volume of hypertensives in our study was higher than those with CKD and the kidney volume was positively correlated with the GFR and the hemoglobin concentration as there was a negative correlation between kidney volume and the urine albumin creatinine ratio. The higher kidney volumes in hypertensives than in CKD mirrors findings by Paquette et al. who reported that the mean kidney volume of CKD sufferers was lower than those in health, and those with hypertension [24]. The higher kidney volume could be a pointer...
Table 4. Pearson’s linear correlation coefficient between kidney volume and eGFR, and ACR, and Hemoglobin concentration between kidney length and cortical thickness.

| Variables                             | r     | CI            | P    | Correlation          |
|---------------------------------------|-------|---------------|------|----------------------|
| KV and glomerular filtration rate     |       |               |      |                      |
| Participants with hypertension        | 0.098 | 0.082 - 0.101 | 0.55 | very weakly positive |
| Participants with CKD                 | 0.118 | 0.114 - 0.120 | 0.39 | very weakly positive |
| KV and hemoglobin concentration       |       |               |      |                      |
| Participants with hypertension        | 0.206 | 0.198 - 0.245 | 0.07 | insignificantly positive |
| Participants with CKD                 | 0.097 | 0.095 - 0.098 | 0.62 | very weakly positive |
| KV and albumin creatinine ratio       |       |               |      |                      |
| Participants with hypertension        | 0.056 | 0.055 - 0.058 | 0.62 | very weakly negative |
| Participants with CKD                 | 0.128 | 0.119 - 0.129 | 0.57 | very weakly negative |

r—correlation coefficient, CI—confidence interval 95%, KV—kidney volume, GFR—glomerular filtration rate, CKD—chronic kidney disease, ACR—albumin creatinine ratio.

a preserved nephron tissue mass and function. Moreover, hypertension without kidney disease is commonly compensated for by renal tissue hypertrophy with or without hyperplasia. Though these compensatory structural changes could be deleterious on the long run, they present with a “step up” in size and function in the initial stages and this could be detected in RUS. The renal hypertrophy is commonly associated with hyperfiltration and salt wasting [25].

More men than women participated in the study both as hypertensives and with CKD and this agrees with previous findings that found both hypertension and CKD to be commoner in males [26]. The dominance of men in the hypertension and CKD population has partly be attributed to the higher responsiveness to the renin angiotensin aldosterone system (RAAS) pathway. In addition to the greater male responsiveness, Miller et al. reported that when RAAS inhibition is commenced, males, after eight weeks showed a depressed response to these drugs [27]. Males that are compliant with their treatment regimen tend to have more poor blood pressure control than treatment compliant women. An implication of this is a faster progression from hypertension to CKD in males than in women. Estrogens as vasodilators in activity also contribute to the lower female prevalence of HTN and CKD [28]. Another reason for the higher prevalence of CKD and faster progression to end stage in males could also be due to the pro-apoptotic and pro-fibrotic activity of androgens in the renal tubules leading to chronic tubulointerstitial nephritis and fibrosis [29].

Hypertensives were younger than those with CKD in our study similar to findings from a previous study [30]. Depending on several factors ranging from genetic to hormonal, environmental and exposures to injurious substances, the length of the lag phase between hypertension and CKD is also dependent on the level of compliance with treatment regimen by hypertensives. Hypertension cause endothelial damage in the kidneys and vascular tissues leading to platelet aggregation, stasis, microthrombi, release of vasoconstricting and pro-inflammatory

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cytokines, sluggish flow and atherosclerosis. The resulting chronic hypoperfusion leads to chronic ischemic injury, necrosis with nephron loss, sclerosis and fibrosis. The structural end point is a smaller, more echogenic kidney which can further be indented and calcific (SICK syndrome) found in chronic inflammatory conditions like pyelonephritis, diabetes, reflux disease, analgesic and sickle cell nephropathies [31]. The terminal structural entity therefore, is reduced kidney size and volume in CKD as seen in this study [32].

The higher BMI among participants with CKD than hypertensives in this study reflect the renal function decline typically from the third and fourth stages of CKD that is associated with reduced glomerular filtration, distortion of the glomerular tuft leading to retention of nitrogenous waste and water, and proteinuria. There is interstitial spaces fluid retention (edema) due to reduced oncotic pressure (from hypoalbuminemia) commonly found in CKD sufferers, hence the higher BMI in them, despite the reduced kidney volume [33]. The higher ACR found in the CKD population in this study compared with those with hypertension without kidney disease (HWKD) was also reported by Poudel et al. who found a higher ACR in CKD sufferers than in hypertensives without kidney disease [34]. As injury from hypertension continues overtime, the podocytes undergoing surface capping shedding immune complexes into the subepithelial spaces. This leads to foot processes replacement by continuous cytoplasmic bands along the glomerular basement membrane (GBM) with loss of the charge and size selectivity. Podocyte fusion (effacement) causes the loss of albumin, large and/or negatively charged substances into the urine [35].

We found higher levels of serum sodium and bicarbonate with lower potassium, urea and creatinine levels in hypertensives without kidney disease than in CKD as was reported in previous studies [36] [37]. With nephron loss and reduction in renal mass and kidney volume, the secretory and absorptive functions of the renal tubules are depressed coupled with reduced activities of the ion exchangers, transporters and diffusive forces of the tubular apical and basolateral membranes [38]. The higher blood pressure, with salt and water retention in the CKD population, lead to compensatory pressure natriuresis under stimulation by the natriuretic peptides and this partly accounts for the lower sodium found in them as seen in this study [39]. The loss of nephrons could also explain the higher serum potassium in the CKD population from reduced distal tubular (principal cells) exchange between the sodium and potassium and (Type A intercalated cells) exchange between potassium and hydrogen ions [40]. The loss of renal mass and volume with decreasing GFR could explain the decline in nitrogenous waste removal leading to higher blood level as was seen in our study and as previously reported [41] [42].

The higher hemoglobin concentration among HWKD compared to the CKD population found in our study is similar to findings from studies that established links between CKD and anemia [43]. Apart from reduced intake, digestion and assimilation (malnutrition), and increased losses (hemolysis), the reduced nephron mass and volume could cause reduced erythropoietin production from
the peritubular renal interstitium hence higher rates of anemia in the CKD population than in hypertensives without CKD [17]. Due to the pan-renal injury induced by hypertension unlike some diseases with segmental renal tissue affection, the mass and volume of all anatomical and physiological units of the kidney are commonly reduced in hypertensive kidney disease and this is commonly seen in renal measures using RUS as seen in our study and as reported previously [44].

Females, increasing age, higher BMI, higher blood pressures, lower sodium, bicarbonate and hemoglobin concentration, with elevated nitrogenous waste and urine ACR were more associated with reduced kidney volume in participants. We infer that despite the higher prevalence and severity of hypertension and CKD in males (thereby causing higher losses in kidney volume), the higher prevalence of reduced kidney volume in females in this study could be explained by their lower kidney sizes in health and therefore in disease, as reported by Piras et al. [45].

Our study showed in both hypertensives without kidney disease and in CKD, a positive correlation between the kidney volume and 1) the GFR and 2) the hemoglobin concentration. Previous studies have shown a direct relationship between nephron mass (kidney volume) and kidney function in health and in disease [24]. The negative correlation between the kidney volume and urine ACR we found is in agreement with a previous study that found an inverse relationship between kidney function and the degree of proteinuria [34]. The loss of nephron mass and kidney volume is commonly associated with chronic inflammatory changes leading to sclerosis and loss of function of the glomerular tuft with loss of negatively charged heparansulphate which allows a greater albumin loss in the urine [35].

We encountered some limitations in this study, one, creatinine based eGFR was done once hence it was difficult to ascertain conditions that could cause transient reductions or increases in kidney function in participants. RUS, being operator dependent, there could have been mis-representation of structures. It was also difficult to ascertain participants’ compliance with the prescribed 3 hour fast. More studies involving a healthy population, and other racial groups are needed to formulate policies that would be widely applicable.

Conclusion: Kidney volume, being the summation of all kidney parts gives a better representation of the amount of renal tissue compared to other RUS measures. The kidney volumes of participants with CKD were less than those with hypertension without kidney disease. The mean kidney volume of females was less in both hypertensives without kidney disease and in CKD despite higher prevalence of both conditions in males. There was a direct relationship between kidney volume and serum sodium, bicarbonate, hemoglobin concentration and the GFR but an inverse relationship with the BMI, blood pressure, serum creatinine, potassium, and urine ACR. Correlation analysis between kidney volume and GFR were stronger in CKD than in hypertension. With hemoglobin concentration, correlation was stronger in hypertension than CKD but with the
ACR, correlation with kidney volume was stronger in CKD than in hypertension without kidney disease.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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