



Original Article



Association of Serum Creatinine and Cortical Thickness with Renal Echogenicity on Ultrasound in Chronic Kidney Disease

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ABSTRACT

The Incidence of chronic kidney disease (CKD) is increasing day by day all over the globe. In assessing the progression of CKD, both the function and structure of the kidney need to be evaluated. **Objectives:** To determine the association between serum creatinine and cortical thickness with renal echogenicity on ultrasound in patients with CKD. **Methods:** This cross-sectional analytical research was carried out at the Department of Radiology and Biochemistry, Shahida Islam Medical and Dental College, Lodhran, for six months (June 2024 to November 2024). All patients who were referred for ultrasonography of the kidneys, with serum creatinine and serum urea checked on the same day on which U/S was carried out, were included. Patients who were k/c of acute renal injury, on hemodialysis, peritoneal dialysis, renal transplant, chronic liver disease, fatty liver, or those having a solitary kidney were all excluded. SPSS version 23.0 was used for data analysis. A one-way ANOVA test was applied to test for association, keeping $p < 0.05$ statistically significant. **Results:** Mean age of participants was 52.26 ± 12.50 years (95% CI: 39.76–64.76). The study found that higher serum creatinine and urea levels were significantly associated with increased cortical echogenicity and reduced cortical thickness on ultrasound. The mean serum creatinine was 2.11 ± 1.2 mg/dL, and cortical thickness progressively declined from 1.11 cm to 0.71 ± 0.10 cm across echogenicity grades. **Conclusions:** The study found a significant association between elevated serum creatinine and cortical thickness levels with renal echogenicity on ultrasound in CKD patients.

INTRODUCTION

Chronic kidney disease (CKD) is affecting people increasingly all over the world, and its incidence is increasing. A characteristic feature of CKD is a steady decline in the function of the kidney [1]. Chronic kidney disease (CKD) is characterized by a decrease in the glomerular filtration rate (GFR) and/or an elevation in urine albumin excretion [2]. Among CKD patients, impaired structure and function of the kidney are interrelated.

Therefore, in assessing the progression of CKD, both the function and structure of the kidney need to be evaluated. Previously, information regarding kidney function was provided by laboratory testing, while structural information was provided through imaging techniques [3]. At present, various imaging techniques such as ultrasound (U/S), CT-scan (Computed Tomography), and MRI (Magnetic Resonance Imaging) tend to provide information about the



kidney's structural alterations; however, little input is provided with regard to functional impairments in CKD [4]. Ideally, diagnosing CKD must provide both functional and structural information in detail [5]. A deranged level of serum creatinine for months to years is indicative of CKD. Based on kidney damage, the extent of CKD is termed by the decrease in GFR (i.e., <60 ml/min per 1.7 m² for over three months [6]. With regard to the structural function of the kidney, the most commonly used modality is ultrasonography. It is a non-invasive and inexpensive technique that shows sufficient anatomy of the kidneys that is required for diagnosing kidney diseases without any exposure of the patient to contrast or radiation [7]. Therefore, U/S has become the standard radiological procedure of choice worldwide. Such factors tend to promote detecting CKD early and predict derangement of kidney function tests necessary for making a clinical diagnosis [8]. U/S helps to identify the length of the kidney, its thickness, and the echogenicity of the renal parenchyma in addition to details of the dilated collecting system [9]. The details help in assisting in identifying the extent of damage to renal parenchyma and reversibility. The decision to perform a renal biopsy depends on the extent of damage. Literature states U/S reports abnormal renal findings in as high as 67 % of cases with CKD [10]. The morphology of the kidney can be assessed by various means, such as by measurement of renal length, volume, and cortical thickness [11]. The function of the kidneys can be estimated via renal length and cortical thickness. Through assessment of these functions, clinical decisions can easily be made [12]. Serial ultrasonography findings can help in assessing the progression of renal diseases or their normality [13]. Even though the volume of renal parenchyma is relatively accurate, especially in end-stage kidney diseases, measuring the kidney's longitudinal length is enough for patients with normal or abnormal kidney function [14]. Some studies have reported that the calculation of renal volume via ultrasound provides a more exact measurement of functioning kidney compared to renal length. Recently, a study reported length of the kidney and volume substantially correlated with the estimated GFR [15]. In CKD patients, cortical echogenicity tends to increase at U/S. Additionally, the renal cortex seldom becomes thinned [16]. To date, the relationship in-between the function of the kidneys and cortical thickness has not been well established in terms of U/S for structural integrity of kidneys and serum creatinine for functional evaluation of the kidneys [17].

The present study aimed to determine the association between serum creatinine and cortical thickness with renal echogenicity on ultrasound in patients with CKD.

METHODS

This cross-sectional analytical study was conducted in the Departments of Radiology and Biochemistry, Shahida Islam Medical & Dental College, Lodhran, over six months (June–November 2024), after obtaining ethical approval from the Institutional Review Board of Shahida Islam Medical Complex (IRB No. SIMC/ET.C./00030/24). All patients referred for renal ultrasonography with serum creatinine and serum urea tested on the same day were eligible. Non-probability consecutive sampling was used. Informed consent was obtained from all participants. Inclusion criteria were male and female patients aged >18 years presenting for chronic kidney disease (CKD) workup or known CKD with GFR <60 ml/min/1.73m², calculated using the Modification of Diet in Renal Disease (MDRD) equation. Exclusion criteria were patients with acute kidney injury, on hemodialysis or peritoneal dialysis, renal transplant recipients, those with chronic liver disease, fatty liver, or solitary kidney. Sample size was calculated using OpenEpi, based on a local prevalence of CKD (21.2%), 95% confidence interval, and 5% margin of error, resulting in 257 subjects [18]. Serum creatinine and urea were measured using an automated chemistry analyzer (Roche Cobas c311 or equivalent) via an enzymatic colorimetric method. Renal ultrasound was performed using a GE LOGIQ P5 system with a 3.5–5.5 MHz curved array transducer, utilizing low tissue harmonic and speckle reduction imaging. Manual gain and time-gain compensation were optimized. Kidney length, width, and cortical thickness were measured in the largest longitudinal and transverse sections. Renal echogenicity was compared with adjacent liver and spleen and graded as follows: Grade 0 (Normal): Cortex hypoechoic or isoechoic vs. liver/spleen, preserved corticomedullary differentiation (CMD). Grade 1: Cortex isoechoic to liver/spleen, CMD preserved. Grade 2: Cortex mildly hyperechoic, partial CMD loss. Grade 3: Cortex markedly hyperechoic, poor/absent CMD. Grade 4: Cortex markedly hyperechoic with complete CMD loss, indistinct renal architecture [9]. Data were analyzed using SPSS version 23.0. Quantitative variables (age, kidney size, cortical thickness) were presented as mean ± SD, while categorical variables (echogenicity grades) were expressed as frequency and percentage. One-way ANOVA was applied to assess associations between serum creatinine, cortical thickness, and renal echogenicity. Normality of data was confirmed using the Shapiro-Wilk test (p=0.51). A p-value <0.05 was considered statistically significant.

RESULTS

From the total of 257 patients, the baseline demographics and laboratory values of patients included in the study are presented in Table 1. The mean age of participants was

52.26 ± 12.50 years (95% CI: 39.76–64.76). The mean serum creatinine was 2.11 ± 1.2 mg/dL (95% CI: 0.58–3.31), while the mean serum urea was 64.25 ± 15.75 mg/dL (95% CI: 48.5–80), both elevated beyond normal ranges, consistent with chronic kidney disease. The mean longitudinal kidney length was 9.25 ± 1.1 cm (95% CI: 8.15–10.35), parenchymal thickness was 4.88 ± 0.92 cm (95% CI: 3.96–5.80), and cortical thickness was 0.82 ± 0.24 cm (95% CI: 0.61–1.11) (Table 1).

Table 1: Baseline Demographics of Patients Included in the Study (N=257)

Variables	Mean ± SD	95 % Confidence Interval	
		Lower Limit	Upper Limit
Age (years)	52.26 ± 12.50	39.76	64.76
Serum Creatinine (mg/dL)	2.11 ± 1.2	0.58	3.31
Serum Urea (mg/dL)	64.25 ± 15.75	48.5	80
Longitudinal Length (cm)	9.25 ± 1.1	8.15	10.35
Parenchymal Thickness (cm)	4.88 ± 0.92	3.96	5.80
Cortical Thickness (cm)	0.82 ± .24	0.61	1.11

A progressive increase in serum creatinine was noted with higher echogenicity grades. Patients with Grade 0 echogenicity had a mean serum creatinine of 0.91 ± 0.77 mg/dL (95% CI: 0.58–1.22), while those with Grade 4 had the highest mean of 2.81 ± 0.99 mg/dL (95% CI: 1.56–3.31). The association was statistically significant ($p < 0.001$), indicating a strong correlation between worsening echogenicity and elevated serum creatinine (Table 2).

Table 2: Association of Serum Creatinine with Cortical Echogenicity of Kidneys (N=257)

Grading of Echogenicity (Based on U/S)	Serum Creatinine (mg/dL)			p-Value
	Mean ± SD	95 % Confidence Interval		
		Lower Bound	Upper Bound	
Grade 0 (n=74)	0.91 ± 0.77	0.58	1.22	<0.001
Grade 1 (n=61)	1.88 ± 0.87	0.82	2.1	
Grade 2 (n=58)	2.26 ± 0.95	1.2	2.56	
Grade 3 (n=44)	2.54 ± 0.96	1.33	2.64	
Grade 4 (n=20)	2.81 ± 0.99	1.56	3.31	

Patients with Grade 0 echogenicity had the highest cortical thickness of 1.11 cm, while those with Grade 4 showed the lowest, at 0.71 ± 0.10 cm (95% CI: 0.66–0.72). A steady decrease in mean cortical thickness was observed from Grade 1 (1.02 ± 0.067 cm) to Grade 3 (0.81 ± 0.14 cm), with a significant trend ($p < 0.001$), suggesting cortical thinning correlates with more advanced renal parenchymal changes (Table 3).

Table 3: Association of Cortical Thickness with Cortical Echogenicity of Kidneys (n=257)

Grading of Echogenicity (Based on U/S)	Cortical Thickness (cm)			p-Value
	Mean ± SD	95 % Confidence Interval		
		Lower Bound	Upper Bound	
Grade 0 (n=74)	1.11	1.11	1.11	<0.001
Grade 1 (n=61)	1.02 ± 0.067	0.98	1.06	

Grade 2 (n=58)	0.92 ± 0.13	0.83	0.99
Grade 3 (n=44)	0.81 ± 0.14	0.77	0.95
Grade 4 (n=20)	0.71 ± 0.1	0.66	0.72

DISCUSSION

The results of this study showed that of 257 patients with chronic kidney disease, the mean serum creatinine and urea levels were elevated at 2.11 ± 1.2 mg/dL and 64.25 ± 15.75 mg/dL, respectively. Kidney ultrasound findings showed a mean cortical thickness of 0.82 ± 0.24 cm and a mean longitudinal length of 9.25 ± 1.1 cm. A statistically significant increase in serum creatinine and a decrease in cortical thickness were observed with higher grades of cortical echogenicity ($p < 0.001$), indicating a strong association between biochemical deterioration and structural kidney damage. The ultrasound findings reported in this research, for instance, echogenicity, longitudinal length, cortical and parenchymal thickness, all parameters tend to be affected by CKD. In addition, GFR and grading of disease (stage) can be determined by assessing endogenous levels of serum creatinine [19]. According to Kovesdy, the normal upper limit of the length of the kidney was recorded at 12 cm, while in our research, the mean longitudinal length was 9.25 ± 1.1 cm [20]. In another research by Roy and Pal, the length of the kidney below 8 cm was defined as reduced length and was attributed to CKD [21]. As the length of the kidney decreased, renal function was also observed to decline along with renal length. This is traditionally attributed to CKD [22]. A similar finding was reported in our study as well, where both cortical thickness and serum creatinine were significantly associated with renal echogenicity in CKD patients. Therefore, in determining the progression of CKD, estimating renal length must be preferred to renal volume. In addition, assessing levels of serum creatinine can also aid in the assessment of disease progression [22]. The mean serum creatinine levels observed in our research, according to the grade of renal echogenicity on U/S, were found to be in line with other studies. Wang et al. study found a substantial association ($p < 0.05$) between the parameters [23]. Ammirati et al. observed values ($p < 0.05$) similar to those in our study [24]. Serum creatinine levels have long been used to assess kidney function, not only in CKD but also in other kidney-related disorders. In our study, the mean cortical thickness was 8.2 ± 2.4 mm and showed a significant association with renal echogenicity grades ($p < 0.001$). Similar to our findings, other researchers have also reported increased renal echogenicity with decreased cortical thickness [25]. Although our study included CKD patients with varying grades of renal echogenicity, it was not without limitations. Being single-centered with a limited sample size, the findings may not be generalizable, and observer bias could not be fully excluded. Nonetheless,

the study highlights important ultrasound parameters associated with both structural (cortical thickness) and functional (serum creatinine) aspects of kidney function.

CONCLUSIONS

This study demonstrated a significant association between elevated serum creatinine and cortical thickness with renal echogenicity on ultrasound in CKD patients. Ultrasonographic parameters can serve as reliable indicators of renal function and disease progression.

Authors Contribution

Conceptualization: MI

Methodology: MFA, NM

Formal analysis: NM, SA

Writing review and editing: MFA, SA, SSS, KA

All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

All the authors declare no conflict of interest.

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