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Shear Wave Elastography in Evaluation of Renal Parenchymal Stiffness in Patients with Chronic Kidney Disease

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ABSTRACT
Background: *Chronic kidney disease (CKD) represents a global health challenge with progressive renal parenchymal fibrosis as its hallmark pathological feature. Traditional assessment methods, particularly renal biopsy, are invasive and carry significant complications. Shear wave elastography (SWE) offers a non-invasive alternative for quantifying tissue stiffness through measurement of Young's modulus.*
Objectives: *To compare renal parenchymal stiffness in patients with chronic kidney disease versus healthy controls using conventional B-mode ultrasonography and shear wave elastography, and to establish diagnostic cutoff values for distinguishing diseased from normal renal parenchyma.*
Methods: *This cross-sectional analytical study included 60 participants (30 CKD patients and 30 healthy controls) aged above 18 years at Ramaiah Medical College Hospital, Bengaluru from May 2023 to January 2025. Participants underwent conventional grayscale ultrasound and SWE examination using Philips Affiniti 70 ultrasound system with curved-array transducer (1-5 MHz). Young's modulus values were measured in kilopascals from the renal cortex. Clinical parameters including serum creatinine and estimated glomerular filtration rate (eGFR) were documented. Statistical analysis employed independent t-test, Pearson and Spearman correlation, and receiver operating characteristic curve analysis.*
Results: *The mean Young's modulus in CKD patients was significantly higher than controls (7.12 ± 3.21 kPa vs 2.89 ± 1.02 kPa). Strong negative correlation was observed between Young's modulus and eGFR in CKD patients ($r = -0.455$, $p = 0.012$). An optimal cutoff value of 4.00 kPa demonstrated excellent diagnostic performance with sensitivity of 93.3%, specificity of 93.3%, and area under curve of 0.967. No significant difference was found between left and right kidney measurements ($p = 0.821$).*
Conclusion: *Shear wave elastography provides a reliable, non-invasive method for assessing renal parenchymal stiffness in chronic kidney disease with excellent diagnostic accuracy. A cutoff value of 4.00 kPa effectively distinguishes diseased from normal renal parenchyma, potentially facilitating early detection and monitoring of CKD progression.*
Keywords: *Shear wave elastography, chronic kidney disease, Young's modulus, renal fibrosis, ultrasound elastography, non-invasive imaging*

I. INTRODUCTION

Chronic kidney disease (CKD) represents one of the most significant global health challenges of the 21st century, affecting more than 500 million individuals worldwide and contributing to approximately 1.2 million deaths annually (1). The burden of CKD continues to escalate, with projections indicating that by 2030, over 2 million patients will require renal replacement therapy through dialysis or transplantation (2). This progressive condition is characterized by sustained deterioration of renal function over a period of three months or longer, manifesting through structural or functional abnormalities with or without reduction in glomerular filtration rate (3).

According to the Kidney Disease Improving Global Outcomes (KDIGO) guidelines, chronic kidney disease is defined as kidney damage persisting for at least three months, identified by structural or functional abnormalities of the kidney with or without a reduction in glomerular filtration rate, or as a GFR less than 60 mL/min/1.73 m² for at least three months, regardless of the presence of kidney damage (4). The principal etiological factors driving the CKD epidemic include diabetes mellitus, hypertension, primary glomerular diseases, and various hereditary kidney disorders. The prevalence of CKD ranges between 8% and 16% globally,

with substantial regional variations influenced by demographic characteristics, socioeconomic factors, and access to healthcare services (5).

The pathophysiological hallmark of chronic kidney disease progression is tubulointerstitial fibrosis, characterized by excessive accumulation of extracellular matrix proteins in the renal parenchyma. This fibrotic process represents the final common pathway for virtually all forms of chronic kidney injury, irrespective of the initial insult (6). Progressive interstitial fibrosis leads to nephron loss, glomerulosclerosis, tubular atrophy, and ultimately irreversible decline in renal function. The degree of renal fibrosis has been demonstrated to correlate strongly with disease severity and serves as a critical predictor of progression to end-stage renal disease (7).

Traditionally, renal biopsy has remained the gold standard for assessing the extent and nature of renal parenchymal damage. Histopathological examination provides definitive information regarding the degree of glomerulosclerosis, tubular atrophy, interstitial fibrosis, and vascular changes. However, renal biopsy is fraught with significant limitations that restrict its widespread clinical application. The procedure is invasive, requires specialized expertise, carries substantial risk of complications including hemorrhage and infection, and is associated with costs exceeding one thousand US dollars per procedure (8). Furthermore, renal biopsy is subject to sampling error, as the obtained tissue core represents only a minuscule fraction of the total renal parenchyma. In advanced fibrotic kidneys, biopsy samples frequently contain insufficient glomerular tissue to permit accurate histopathological diagnosis (9).

These inherent limitations of renal biopsy have catalyzed intense research efforts aimed at developing non-invasive methods for quantitative assessment of renal fibrosis. Conventional grayscale ultrasonography has long been utilized as the primary imaging modality for evaluating kidney morphology in clinical practice. Standard ultrasonographic parameters including renal length, cortical thickness, parenchymal thickness, and assessment of cortical echogenicity provide valuable information regarding kidney structure. Increased renal cortical echogenicity has been correlated with interstitial fibrosis, tubular atrophy, and glomerulosclerosis in histological studies (10). However, these conventional parameters are subjective, operator-dependent, and cannot be objectively quantified. Moreover, they demonstrate limited sensitivity and specificity in detecting early renal parenchymal changes before significant morphological alterations become apparent (11).

Elastography represents a revolutionary advancement in medical imaging technology that enables non-invasive quantification of tissue stiffness. The fundamental principle underlying elastography is that pathological processes such as fibrosis, inflammation, and malignancy alter the mechanical properties of tissues. Shear wave elastography is a sophisticated dynamic elastographic technique that has gained widespread acceptance in hepatology for assessment of liver fibrosis (12). The technique employs focused acoustic radiation force impulses to generate shear waves within tissue. These shear waves propagate perpendicular to the direction of the ultrasound beam, and their velocity is directly proportional to tissue stiffness. Stiffer tissues permit faster shear wave propagation, while softer tissues result in slower propagation (13).

In SWE, tissue stiffness is quantified by measuring Young's modulus, expressed in kilopascals (kPa). Young's modulus represents the ratio of stress to strain and provides a quantitative measure of tissue elasticity. The shear wave velocity is tracked in real-time by ultrafast ultrasound imaging, and mathematical algorithms convert the velocity measurements into Young's modulus values. This approach offers several distinct advantages including real-time imaging capability, operator independence, reproducibility, and the ability to obtain measurements from large volumes of tissue (14).

The application of shear wave elastography to renal imaging has emerged as a promising frontier in nephrology. Several pioneering studies have demonstrated that SWE-derived estimates of renal Young's modulus are significantly elevated in patients with chronic kidney disease compared to healthy individuals (15). These findings suggest that SWE may serve as a valuable non-invasive biomarker for detecting and staging renal fibrosis. However, the technology faces certain challenges specific to renal imaging, including greater kidney depth from the skin surface compared to the liver, the rounded renal morphology which may cause refraction of acoustic pulses, and physiological factors such as respiratory motion and intrarenal blood flow that may influence measurements (16).

Despite these challenges, accumulating evidence supports the clinical utility of shear wave elastography in chronic kidney disease. The technique has demonstrated excellent feasibility, acceptable reproducibility, and strong correlations with established markers of renal function including estimated glomerular filtration rate and serum creatinine levels. Furthermore, SWE measurements have shown promise in differentiating various stages of chronic kidney disease and potentially predicting disease progression (17).

The present study was designed to systematically evaluate the application of shear wave elastography in assessing renal parenchymal stiffness in patients with chronic kidney disease. By comparing SWE-derived Young's modulus values between CKD patients and healthy controls, and correlating these measurements with conventional ultrasonographic parameters and biochemical markers of renal function, this investigation aims to establish the diagnostic performance of SWE and determine optimal cutoff values for clinical application. Such information would be invaluable in facilitating early non-invasive detection of renal parenchymal disease,

monitoring disease progression, and potentially guiding therapeutic interventions in patients with chronic kidney disease.

II. AIMS AND OBJECTIVES

The primary objective of this study was to compare renal parenchymal stiffness in patients with chronic kidney disease with a control population using conventional B-mode ultrasonography and shear wave elastography. Specific aims included: (1) to measure and compare Young's modulus values derived from shear wave elastography between CKD patients and healthy controls; (2) to correlate SWE-derived Young's modulus with estimated glomerular filtration rate and serum creatinine levels; (3) to assess conventional ultrasonographic parameters including renal cortical echogenicity and corticomedullary differentiation in both groups; (4) to determine optimal cutoff values of Young's modulus for distinguishing normal from diseased renal parenchyma; and (5) to evaluate the diagnostic performance of shear wave elastography in detecting chronic kidney disease using receiver operating characteristic curve analysis.

III. MATERIALS AND METHODS

Study Design and Setting

This cross-sectional analytical study was conducted in the Department of Radiodiagnosis at Ramaiah Medical College and Hospital, Bengaluru, Karnataka, India over a period extending from May 2023 to January 2025. The study protocol received approval from the Institutional Ethics Committee of Ramaiah Medical College, and written informed consent was obtained from all participants prior to enrollment. The study adhered to the principles outlined in the Declaration of Helsinki for medical research involving human subjects.

Study Population

The study population comprised 60 participants divided into two groups of equal size. The case group consisted of 30 patients diagnosed with chronic kidney disease based on KDIGO criteria, defined as eGFR less than 90 mL/min/1.73 m². The control group included 30 healthy individuals with eGFR greater than 90 mL/min/1.73 m² who presented to the Department of Radiodiagnosis for other reasons and had no evidence of renal disease.

Sample Size Calculation

Sample size determination was based on previous literature demonstrating mean and standard deviation of shear wave elastography Young's modulus in case and control groups as 7.61 ± 6.09 kPa and 3.55 ± 1.59 kPa respectively. With 90% statistical power, 95% confidence level, and an effect size of 1.05, the calculated minimum sample size was 25 participants in each group. To account for potential dropouts and enhance statistical robustness, 30 participants were enrolled in each group.

Inclusion Criteria

For the case group, inclusion criteria comprised patients aged 18 years or above diagnosed with chronic kidney disease who visited the Department of Radiodiagnosis at Ramaiah Medical College and Hospital. Chronic kidney disease was defined according to KDIGO guidelines as kidney damage lasting for three months or more identified by structural or functional abnormalities of the kidney with or without reduction in GFR, or as GFR less than 60 mL/min/1.73 m² for at least three months regardless of presence of kidney damage. For the control group, inclusion criteria included individuals aged 18 years or above with eGFR greater than 90 mL/min/1.73 m² and no clinical or biochemical evidence of renal disease.

Exclusion Criteria

Exclusion criteria for both groups included individuals with markedly reduced renal parenchymal thickness, subjects with renal cortex-to-skin distance exceeding 8 cm, participants who declined to provide informed consent, individuals unable to comply with breath-holding instructions during SWE examination, and patients with complete loss of corticomedullary differentiation. Additionally, pregnant women and patients with solitary kidneys were excluded from the study.

Clinical Data Collection

Comprehensive demographic and clinical information was systematically recorded for all participants. This included age, sex, detailed medical history with emphasis on presence of hypertension and diabetes mellitus, duration of known kidney disease for cases, and current medications. Laboratory investigations performed within one week of imaging included serum creatinine measured using standardized enzymatic methods, blood urea nitrogen, and complete blood count. Estimated glomerular filtration rate was calculated using the Chronic Kidney

Disease Epidemiology Collaboration (CKD-EPI) equation which incorporates serum creatinine, age, sex, and race.

Imaging Protocol

All ultrasonographic examinations were performed using the Philips Affiniti 70 ultrasound system equipped with dedicated shear wave elastography software and a curved-array transducer with frequency range of 1-5 MHz. A single experienced sonographer with over five years of expertise in elastography performed all examinations to ensure consistency and minimize inter-operator variability. Participants were positioned in the lateral decubitus posture with the examined kidney positioned uppermost.

Conventional Grayscale Ultrasound

Initial evaluation comprised comprehensive grayscale ultrasonographic examination of both kidneys. The kidney's long axis was identified in the coronal plane, and maximum longitudinal diameter was measured from pole to pole. Cortical thickness was measured perpendicular to the renal capsule from the outer margin of the renal cortex to the outer edge of the medullary pyramid at the mid-kidney level, avoiding the renal columns of Bertin. Parenchymal thickness was measured from the renal sinus to the outer renal margin. Renal cortical echogenicity was assessed subjectively and graded relative to adjacent liver parenchyma on the right side and splenic parenchyma on the left side. Corticomedullary differentiation was documented as maintained or poorly maintained based on visualization of distinct cortical and medullary zones.

Shear Wave Elastography Technique

For SWE imaging, the transducer was positioned gently parallel to the kidney's long axis without applying excessive pressure that could artificially alter tissue stiffness measurements. The SWE mode was activated and the color-coded elastogram was overlaid on the grayscale image. Participants were instructed to suspend respiration in mid-inspiration to minimize motion artifacts from respiratory excursion. Once a stable image was achieved with uniform color mapping across the renal cortex, measurements were obtained.

A standardized region of interest measuring approximately 1.5 cm in diameter was carefully positioned within the renal cortex at the mid-kidney level, specifically excluding the renal medulla, renal sinus, and any visible blood vessels. The region of interest was placed to ensure the shear waves traveled perpendicular to the radially arranged tubular system for optimal measurement accuracy. Five consecutive measurements were obtained from the mid-region of each kidney, and the mean Young's modulus value expressed in kilopascals was calculated for each kidney. Both kidneys were examined in all participants, and the average of bilateral measurements was used for final analysis.

Statistical Analysis

Data were entered into Microsoft Excel spreadsheets and analyzed using SPSS version 22 software (IBM SPSS Statistics, Somers NY, USA) and Epi-Info version 7.2.1 (CDC Atlanta). Categorical variables were presented as frequencies and proportions, with Chi-square test employed to assess statistical significance for qualitative variables. The assumption of normality for continuous variables was tested using the Shapiro-Wilk test. Continuous data demonstrating normal distribution were expressed as mean ± standard deviation, while non-normally distributed data were presented as median with interquartile range.

Independent samples t-test was used to compare mean Young's modulus values between case and control groups. Pearson correlation coefficient was calculated to assess linear relationships between Young's modulus and continuous variables including age, eGFR, and serum creatinine. Spearman rank correlation was employed for non-parametric correlations. Receiver operating characteristic curve analysis was performed to evaluate the diagnostic performance of Young's modulus in distinguishing CKD patients from controls, and to determine optimal cutoff values. The optimal cutoff was identified using Youden's index, which maximizes the sum of sensitivity and specificity. Area under the curve was calculated as a measure of overall diagnostic accuracy. Statistical significance was set at p-value less than 0.05 for all analyses.

IV. RESULTS

Demographic Characteristics

The study comprised 60 participants with mean age of 51.5 ± 13.8 years. In the overall population, 34 participants (56.7%) were male and 26 participants (43.3%) were female. The majority of subjects were in the age group of 60-69 years (30%), followed by 50-59 years (25%). The mean age of female subjects was 55.2 ± 12.3 years while male subjects had mean age of 48.7 ± 14.5 years.

In the control population, 16 participants (53.3%) were male and 14 participants (46.7%) were female. The case population comprised 18 males (60%) and 12 females (40%). Chi-square test of independence revealed no significant association between sex and case status ($\chi^2 = 0.068$, df = 1, p = 0.795). Fisher's exact test yielded

an odds ratio of 0.762 (95% CI: 0.274 - 2.121) with p-value of 0.795, confirming no significant sex-based difference in disease distribution. The risk of being a case among males was 47.1% compared to 53.8% among females, with risk difference of -6.8 percentage points.

Table 1: Demographic Profile of Study Participants

Parameter	Category	Count	Percentage (%)
Age Group	20-29 years	5	8.3
	30-39 years	9	15.0
	40-49 years	10	16.7
	50-59 years	15	25.0
	60-69 years	18	30.0
	70-79 years	2	3.3
	80-89 years	1	1.7
Sex	Male	34	56.7
	Female	26	43.3

Biochemical Parameters

Serum creatinine levels demonstrated significant differences between case and control populations. The mean serum creatinine in the case population was 3.04 ± 1.66 mg/dL compared to 0.95 ± 0.36 mg/dL in the control population. Correlation analysis revealed a statistically significant positive relationship between age and serum creatinine values. Pearson correlation coefficient was $r = 0.266$ with $p = 0.040$, while Spearman correlation demonstrated stronger association with $\rho = 0.458$ and $p < 0.001$. This moderate positive correlation indicated that serum creatinine levels tended to increase with advancing age, with the Spearman correlation suggesting a non-linear relationship.

Interestingly, no significant association was observed between sex and serum creatinine levels. Pearson correlation yielded $r = -0.0001$ with $p = 0.9996$, while Spearman correlation showed $\rho = -0.122$ with $p = 0.351$. Mean serum creatinine values were virtually identical between males (1.99 ± 1.89 mg/dL) and females (1.99 ± 1.13 mg/dL), indicating that sex did not significantly influence creatinine levels in this study population.

Table 2: Comparison of Serum Creatinine Between Case and Control Groups

Group	Mean Serum Creatinine (mg/dL)	Standard Deviation (mg/dL)
Case	3.04	1.66
Control	0.95	0.36

Conventional Ultrasonographic Findings

Renal cortical echogenicity assessment revealed distinct patterns between case and control populations. In the control population, 27 subjects (90%) demonstrated normal cortical echogenicity (hypoechoic or isoechoic relative to liver parenchyma), while 3 subjects (10%) showed mildly increased echogenicity. The presence of increased echogenicity in a small subset of controls may be attributed to physiological variations, transient states of dehydration, or subclinical parenchymal changes not yet manifesting as functional impairment. In contrast, all 30 subjects (100%) in the case population exhibited increased renal cortical echogenicity, consistent with the expected pathological changes of chronic kidney disease including interstitial fibrosis and tubular atrophy.

Corticomedullary differentiation was maintained in all 30 subjects (100%) of the control population, indicating preserved renal architecture. In the case population, corticomedullary differentiation was maintained in 22 subjects (73.3%) while it was poorly maintained in 8 subjects (26.7%). The loss or diminution of corticomedullary differentiation in these 8 patients suggested more advanced disease with extensive parenchymal damage and architectural disruption. Complete loss of corticomedullary differentiation was an exclusion criterion for this study.

Shear Wave Elastography Measurements

Age-Related Variations in Young's Modulus

Pearson correlation analysis was performed to evaluate the relationship between age and renal parenchymal stiffness in both populations. In the control population, a weak to moderate positive correlation was observed between age and Young's modulus for both kidneys. For the right kidney, Pearson coefficient was $r = 0.395$ with $p = 0.031$ (statistically significant), while for the left kidney, $r = 0.342$ with $p = 0.064$ (approaching

significance). This finding suggested that even in healthy individuals, renal parenchymal stiffness tends to increase modestly with advancing age, potentially reflecting normal physiological changes including age-related nephrosclerosis and mild interstitial fibrosis.

In contrast, the case population demonstrated an unexpected negative correlation between age and Young's modulus, though this relationship did not achieve statistical significance. For the right kidney, $r = -0.225$ with $p = 0.231$, and for the left kidney, $r = -0.251$ with $p = 0.181$. This paradoxical finding may reflect the complex interplay of disease severity, duration, and underlying pathophysiology in CKD patients, where the pathological process may override normal age-related changes.

Table 3: Pearson Correlation Between Age and Renal Young's Modulus

Parameter	Case Population (r)	Case P-value	Control Population (r)	Control P-value
Age vs Right Kidney YM	-0.225	0.231	0.395	0.031
Age vs Left Kidney YM	-0.251	0.181	0.342	0.064
Age vs Mean YM	-0.241	0.200	0.379	0.039

Comparison of Young's Modulus Between Kidneys

The mean Young's modulus values for left and right kidneys in the case population were 6.44 ± 2.50 kPa and 6.45 ± 2.70 kPa respectively, demonstrating remarkable bilateral symmetry. Similarly, in the control population, mean values were 2.75 ± 0.90 kPa for the left kidney and 2.79 ± 0.98 kPa for the right kidney. Paired differences analysis revealed a mean difference between right and left kidney measurements of only 0.024 ± 0.680 kPa with median difference of 0.000 kPa.

Wilcoxon signed-rank test yielded $W = 690.0000$ with $p = 0.8213$, confirming no statistically significant difference between bilateral kidney measurements. The effect size calculated using Cohen's d was extremely small at 0.0088, further supporting the absence of clinically meaningful differences. Strong bilateral correlation was demonstrated with Pearson correlation coefficient $r = 0.9685$ ($p < 0.0001$) and Spearman correlation $\rho = 0.9734$ ($p < 0.0001$). Levene's test for equality of variances yielded $F = 0.0001$ with $p = 0.9920$, indicating equal variance between kidneys. These findings support the validity of using either kidney for assessment and the bilateral nature of CKD progression in this cohort.

Table 4: Comparison of Young's Modulus Between Right and Left Kidneys

Group	Right Kidney YM (kPa)	Left Kidney YM (kPa)
Control	2.79 ± 0.98	2.75 ± 0.90
Case	6.45 ± 2.70	6.44 ± 2.50

Sex-Related Variations

Analysis of sex-related differences in renal stiffness revealed no significant correlation between sex and Young's modulus values. The correlation coefficient was -0.07, indicating a weak negative relationship that lacked clinical significance. Mean Young's modulus for male subjects was 5.42 kPa compared to 6.18 kPa for female subjects. This difference was not statistically significant, suggesting that sex does not substantially influence renal parenchymal stiffness measurements in this population.

Comparison Between Case and Control Groups

The primary finding of this study was the highly significant difference in renal parenchymal stiffness between CKD patients and healthy controls. The overall population (n=60) demonstrated mean Young's modulus of 4.618 ± 2.730 kPa with median of 4.050 kPa (IQR: 2.475 - 5.675 kPa) and range of 1.000 - 17.000 kPa. When stratified by disease status, the mean Young's modulus in the case population was 7.12 ± 3.21 kPa compared to 2.89 ± 1.02 kPa in controls, representing an approximately 2.5-fold increase in renal parenchymal stiffness among CKD patients. This difference was highly statistically significant and clinically meaningful, reflecting the underlying pathological changes of interstitial fibrosis and parenchymal scarring characteristic of chronic kidney disease.

Table 5: Comparison of Mean Young's Modulus Between Groups

Group	Mean YM (kPa)	Standard Deviation (kPa)
Control	2.89	1.02
Case	7.12	3.21

Correlation with Renal Function Parameters

Correlation with Estimated Glomerular Filtration Rate

Pearson correlation analysis revealed significant negative correlations between Young's modulus measurements and eGFR in the case population. For the right kidney, r = -0.453 with p = 0.012; for the left kidney, r = -0.443 with p = 0.014; and for mean Young's modulus, r = -0.455 with p = 0.012. These correlations were statistically significant and indicated that as renal function declined (lower eGFR), parenchymal stiffness increased (higher Young's modulus). This inverse relationship supports the hypothesis that SWE-derived stiffness measurements reflect the degree of renal fibrosis and functional impairment.

In the control population, negative correlations were also observed but with weaker magnitude and lacking statistical significance. For the right kidney, r = -0.339 with p = 0.067; for the left kidney, r = -0.285 with p = 0.127; and for mean Young's modulus, r = -0.320 with p = 0.084. The weaker correlations in controls likely reflect the normal physiological range of both eGFR and tissue stiffness in healthy kidneys.

Spearman correlation analysis corroborated these findings. In the case population, significant negative correlations were observed for right kidney Young's modulus versus eGFR (ρ = -0.385, p = 0.035) and mean Young's modulus versus eGFR (ρ = -0.393, p = 0.031). In the control population, right kidney measurements also showed significant correlation (ρ = -0.414, p = 0.021) along with mean Young's modulus (ρ = -0.372, p = 0.042).

Linear regression analysis demonstrated that for each unit increase in mean Young's modulus, the estimated glomerular filtration rate decreased by approximately 10.6 mL/min/1.73 m². This quantitative relationship provides clinically useful information for predicting renal function based on elastography measurements.

Table 6: Pearson Correlation Between Young's Modulus and eGFR

Parameter	Case Population (r)	Case P-value	Significance	Control Population (r)	Control P-value	Significance
Right Kidney YM vs eGFR	-0.453	0.0118	Significant	-0.339	0.066	Not Significant
Left Kidney YM vs eGFR	-0.443	0.0142	Significant	-0.285	0.127	Not Significant
Mean YM vs eGFR	-0.455	0.0161	Significant	-0.320	0.084	Not Significant

Table 7: Spearman Correlation Between Young's Modulus and eGFR

Parameter	Case Population (ρ)	Case P-value	Significance	Control Population (ρ)	Control P-value	Significance
Right Kidney YM vs eGFR	-0.385	0.035	Significant	-0.414	0.021	Significant
Left Kidney YM vs eGFR	-0.352	0.056	Not Significant	-0.294	0.115	Not Significant
Mean YM vs eGFR	-0.393	0.031	Significant	-0.372	0.042	Significant

Diagnostic Performance Analysis

Receiver operating characteristic curve analysis was performed to evaluate the diagnostic accuracy of Young's modulus measurements in distinguishing CKD patients from healthy controls. The analysis yielded excellent results demonstrating high discriminatory power. For serum creatinine as a reference standard, the area under the ROC curve was 0.91, indicating excellent discrimination between case and control groups. For SWE measurements, left kidney Young's modulus achieved AUC of 0.92 while right kidney demonstrated AUC of 0.91. The nearly identical curves for both kidneys confirmed consistent diagnostic performance regardless of which kidney was examined.

Optimal cutoff values were determined using Youden's index to maximize diagnostic accuracy. For mean Young's modulus, the optimal cutoff was identified as 4.00 kPa. At this threshold, the diagnostic performance was exceptional with sensitivity of 93.3%, specificity of 93.3%, positive predictive value of 93.3%, negative predictive value of 93.3%, overall accuracy of 93.3%, and area under the curve of 0.967. These values indicate that Young's modulus of 4.00 kPa or greater correctly identifies 93.3% of CKD patients while correctly excluding 93.3% of healthy individuals.

For the right kidney specifically, optimal cutoff was 4.20 kPa with sensitivity of 90.0%, specificity of 93.3%, positive predictive value of 93.1%, negative predictive value of 90.3%, accuracy of 91.7%, and AUC of

0.960. For the left kidney, optimal cutoff was 4.10 kPa with sensitivity of 93.3%, specificity of 93.3%, positive predictive value of 93.3%, negative predictive value of 93.3%, accuracy of 93.3%, and AUC of 0.966. The consistently high diagnostic performance across all measurements validates SWE as a reliable tool for detecting chronic kidney disease.

Table 8: Diagnostic Performance of Young's Modulus Measurements

Parameter	Optimal Cutoff (kPa)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)	AUC
Right Kidney YM	4.20	90.0	93.3	93.1	90.3	91.7	0.960
Left Kidney YM	4.10	93.3	93.3	93.3	93.3	93.3	0.966
Mean YM	4.00	93.3	93.3	93.3	93.3	93.3	0.967

V. DISCUSSION

This cross-sectional analytical study systematically evaluated the utility of shear wave elastography in assessing renal parenchymal stiffness in patients with chronic kidney disease compared to healthy controls. The findings demonstrate that SWE provides a reliable, non-invasive method for quantifying renal tissue stiffness with excellent diagnostic performance in distinguishing diseased from normal kidneys. The results contribute valuable evidence supporting the clinical application of this emerging technology in nephrology.

The demographic characteristics of the study population revealed that the majority of participants were in the sixth and seventh decades of life, with peak incidence in the 60-69 year age group (30%) followed by the 50-59 year age group (25%). This age distribution is consistent with the known epidemiology of chronic kidney disease, which demonstrates increasing prevalence with advancing age due to cumulative exposure to risk factors, age-related decline in renal function, and higher burden of comorbidities such as hypertension and diabetes mellitus (1,2). The sex distribution showed no significant predilection, with 56.7% male and 43.3% female participants overall. While some epidemiological studies have reported higher CKD prevalence in females, our study demonstrated no statistically significant association between sex and disease status, likely reflecting the relatively small sample size and specific population characteristics.

The biochemical analysis revealed expected differences in serum creatinine levels between case and control populations, with CKD patients demonstrating significantly elevated values (3.04 ± 1.66 mg/dL versus 0.95 ± 0.36 mg/dL). The statistically significant positive correlation between age and serum creatinine (Spearman $\rho = 0.458$, $p < 0.001$) aligns with established physiological principles. Progressive age-related decline in renal function occurs through multiple mechanisms including reduction in renal blood flow, loss of functioning nephrons, glomerular sclerosis, interstitial fibrosis, and tubular atrophy. Studies have documented approximately 1 mL/min/year decline in GFR after age 40 in the general population, though substantial individual variability exists (3,4). The moderate strength of this correlation suggests that while age influences renal function, other factors including genetics, comorbidities, and environmental exposures play important roles.

Interestingly, no significant correlation was observed between sex and serum creatinine in this study, with virtually identical mean values in males and females (1.99 ± 1.89 mg/dL versus 1.99 ± 1.13 mg/dL). This finding contrasts with some previous reports indicating lower serum creatinine in women due to lower muscle mass (5). The discrepancy may be explained by several factors including the specific characteristics of the study population, presence of advanced CKD in the case group where disease effects may override normal sex-based differences, and relatively small sample size that may lack power to detect subtle variations. In advanced renal disease, impaired creatinine filtration elevates serum levels in both sexes, potentially masking baseline differences.

Conventional ultrasonographic evaluation provided important complementary information. The finding of increased renal cortical echogenicity in 100% of CKD patients compared to 90% normal echogenicity in controls is consistent with established literature correlating hyperechoic renal cortex with chronic parenchymal disease (6,7). Increased cortical echogenicity results from altered acoustic properties of diseased tissue, related to interstitial fibrosis, tubular atrophy, and glomerulosclerosis. The presence of mildly increased echogenicity in 10% of controls may represent normal anatomical variation, subclinical changes not yet manifesting functionally, or transient states such as dehydration. Studies have demonstrated that acute kidney injury from various causes including dehydration, sepsis, and acute glomerulonephritis can also produce increased echogenicity due to inflammatory infiltrates and proteinaceous casts (8).

The assessment of corticomedullary differentiation revealed maintenance in all controls but poor differentiation in 26.7% of CKD patients, indicating advanced disease with architectural disruption. The corticomedullary junction normally appears distinct due to different echogenic properties of cortex and medulla. Progressive loss of this differentiation correlates with severity of chronic parenchymal damage and represents an

unfavorable prognostic marker (9,10). Complete loss of corticomedullary differentiation was an exclusion criterion as it represents end-stage disease where elastography measurements may be unreliable.

The core findings of this study relate to shear wave elastography measurements. The mean Young's modulus in CKD patients was significantly elevated compared to controls (7.12 ± 3.21 kPa versus 2.89 ± 1.02 kPa, representing approximately 2.5-fold increase). This substantial difference reflects the fundamental pathophysiological process of chronic kidney disease, namely progressive interstitial fibrosis with accumulation of extracellular matrix proteins. As fibrosis progresses, affected tissue becomes stiffer, allowing shear waves to propagate more rapidly, resulting in higher Young's modulus values (11,12). These findings are remarkably consistent with published literature. A study conducted by Leong and colleagues reported mean Young's modulus of 4.40 kPa in controls compared to elevated values in CKD patients, with strong negative correlation between Young's modulus and eGFR ($r = -0.576$, $p < 0.0001$) (13). Similarly, Samir and colleagues found median Young's modulus of 4.40 kPa in controls versus 9.40 kPa in CKD patients (14).

The control group values in our study (mean 2.89 kPa) fall within the range reported in various previous investigations, though some variability exists in published normal values. This variability likely reflects differences in elastography equipment and techniques (point shear wave versus two-dimensional shear wave elastography), different ultrasound systems, varying measurement protocols, operator technique variations, and diverse population characteristics including age, body habitus, and ethnicity (15,16). Some studies have reported normal kidney Young's modulus ranging from 2.15 to 5.70 kPa, highlighting the need for institution-specific reference ranges.

An unexpected finding was the weak negative correlation between age and Young's modulus in the CKD group ($r = -0.225$ to -0.251), though not statistically significant. In contrast, the control group showed weak positive correlation ($r = 0.342$ to 0.395), statistically significant for the right kidney. The positive correlation in controls suggests that even in healthy individuals, normal aging processes produce modest increases in renal stiffness through mechanisms including age-related nephrosclerosis, mild interstitial fibrosis, vascular changes, and glomerular obsolescence. Similar findings were reported in some studies demonstrating age-related increases in tissue stiffness (17). However, other investigators found no significant age-related changes in renal elasticity, highlighting ongoing debate regarding this relationship.

The paradoxical negative correlation in CKD patients, while not significant, merits consideration. One hypothesis is that advanced CKD involves complex pathological processes beyond simple fibrosis, including parenchymal atrophy, cyst formation, vascular sclerosis, and altered tissue architecture that may not uniformly increase stiffness. Additionally, in severely damaged kidneys, loss of functioning parenchyma and replacement with fluid-filled spaces might reduce measured stiffness in some cases. The small sample size and heterogeneous disease etiologies may have contributed to this unexpected finding. Larger studies with subgroup analysis by CKD stage and etiology would help clarify these relationships.

The remarkable bilateral symmetry of Young's modulus measurements (mean difference 0.024 kPa, $p = 0.821$) with very strong correlation between kidneys ($r = 0.9685$) is clinically significant. This finding demonstrates that CKD typically progresses bilaterally in a symmetric fashion, validates the reliability and reproducibility of SWE measurements, and indicates that either kidney can be used for assessment when bilateral examination is not feasible. The high correlation also suggests that unilateral measurements may suffice in clinical practice, potentially reducing examination time and patient burden. However, in cases of unilateral renal disease, asymmetric progression, or discordant measurements, bilateral evaluation remains important.

The absence of significant sex-related differences in Young's modulus (correlation coefficient -0.07) aligns with findings from several previous studies (18). While males and females differ in muscle mass affecting creatinine levels, the mechanical properties of renal parenchyma appear similar between sexes when normalized for body size. This finding simplifies interpretation of elastography results as sex-specific reference ranges may not be necessary, though further validation in larger populations is warranted.

The strong negative correlations between Young's modulus and estimated glomerular filtration rate in CKD patients ($r = -0.453$ to -0.455, $p = 0.012$ to 0.014) represent a key validation of SWE as a marker of renal function. As eGFR declines indicating worsening renal function, Young's modulus increases reflecting progressive fibrosis. This inverse relationship has strong biological plausibility and is supported by extensive literature. The aforementioned study by Leong demonstrated similar negative correlation ($r = -0.576$, $p < 0.0001$) (13). Linear regression analysis in our study revealed that each 1 kPa increase in Young's modulus corresponded to approximately 10.6 mL/min decrease in eGFR, providing a quantitative framework for predicting functional impairment based on elastography measurements.

The weaker, non-significant correlations in the control group ($r = -0.285$ to -0.339) likely reflect the narrow physiological range of both variables in healthy individuals. Within the normal range, small variations in eGFR and tissue stiffness may not demonstrate strong linear relationships, and other factors such as measurement variability and biological variation may obscure subtle associations.

Some studies have reported contrasting findings, with positive relationships between shear wave velocity (which directly relates to Young's modulus) and eGFR. These discrepancies may stem from methodological

differences including different elastography techniques, varying stages of CKD studied, different definitions of renal regions of interest, and heterogeneous patient populations. Advanced CKD with parenchymal atrophy and cystic changes may produce different stiffness patterns than early disease dominated by active inflammation and fibrosis. Standardization of measurement protocols and inclusion of histopathological correlation would help reconcile these discordant findings.

The diagnostic performance analysis yielded exceptional results, with the optimal Young's modulus cutoff of 4.00 kPa demonstrating sensitivity of 93.3%, specificity of 93.3%, and area under the curve of 0.967. These performance metrics indicate that SWE is highly accurate in distinguishing CKD patients from healthy individuals. The AUC approaching 1.0 represents near-perfect discrimination. For comparison, a study by Bob and colleagues reported cutoff of 4.31 kPa with sensitivity of 80.3% and specificity of 79.5% (19), while others reported cutoff around 5.0 kPa with good diagnostic performance (20). The slight variations in optimal cutoffs across studies likely reflect differences in study populations, disease severity distribution, and technical factors.

The consistently high diagnostic performance for both kidneys individually (AUC 0.960-0.966) and for mean values (AUC 0.967) validates the robustness of SWE measurements. The excellent positive and negative predictive values (93.3% for both) indicate that a Young's modulus above 4.00 kPa strongly suggests CKD, while values below this threshold effectively rule out significant renal disease. These performance characteristics compare favorably to other non-invasive tests and approach the accuracy of more invasive procedures.

It is important to acknowledge that while SWE demonstrates excellent accuracy for detecting CKD presence, its ability to differentiate individual CKD stages appears more limited. Some studies have shown that while early and advanced stages can be distinguished, discrimination between consecutive stages (e.g., stage 2 versus stage 3) may be challenging (21). The relatively wide overlap in stiffness values across adjacent stages, combined with measurement variability and biological factors, limits precise staging capability. Future studies should evaluate longitudinal changes in Young's modulus to assess its utility in monitoring disease progression over time.

Several technical considerations merit discussion. Renal elastography faces unique challenges compared to liver elastography, which has become well-established clinically. The kidneys are typically located deeper from the skin surface than the liver, potentially affecting acoustic radiation force and shear wave propagation. The rounded renal morphology may cause refraction of acoustic pulses leading to variability in shear wave generation. Respiratory motion and intrarenal blood flow create inherent physiological variability. Despite these challenges, our study demonstrates that with careful technique including breath-holding, appropriate transducer positioning, and multiple measurements, reproducible results can be obtained.

The study has several strengths including prospective design, adequate sample size based on power calculation, single experienced operator minimizing inter-operator variability, comprehensive assessment combining conventional ultrasonography with elastography, systematic correlation with biochemical markers of renal function, and rigorous statistical analysis including ROC curve evaluation. However, certain limitations must be acknowledged. The cross-sectional design precludes assessment of temporal changes in stiffness during disease progression. The relatively small sample size of 30 per group limits subgroup analyses and may not capture the full spectrum of CKD presentations. Absence of histopathological correlation prevents direct validation of the relationship between measured stiffness and actual fibrosis quantification. The study was conducted at a single tertiary care center which may limit generalizability to other populations and settings. Inclusion of patients with various CKD etiologies introduces heterogeneity, though this reflects real-world clinical practice. Finally, the study did not evaluate inter- and intra-observer reliability, though use of a single experienced operator minimizes this concern.

Future research directions should include longitudinal studies tracking Young's modulus changes over time to evaluate prognostic value for predicting CKD progression and response to therapeutic interventions. Histopathological correlation studies directly comparing elastography measurements with quantitative fibrosis assessment from renal biopsies would provide definitive validation. Multi-center studies with larger sample sizes would establish generalizable reference ranges and validate cutoff values. Investigation of elastography performance in specific CKD etiologies (diabetic nephropathy, hypertensive nephrosclerosis, glomerulonephritis) may identify disease-specific patterns. Technical refinements to optimize measurement protocols and improve reliability would enhance clinical applicability. Finally, cost-effectiveness analyses comparing SWE with other diagnostic modalities would inform clinical implementation strategies.

In conclusion, this study provides robust evidence supporting shear wave elastography as a valuable non-invasive tool for assessing renal parenchymal stiffness in chronic kidney disease. The technology demonstrates excellent diagnostic accuracy with the optimal cutoff of 4.00 kPa effectively distinguishing diseased from normal kidneys. The strong inverse correlation with eGFR validates Young's modulus as a marker of renal function. SWE offers several advantages over renal biopsy including non-invasive nature, absence of complications, lower cost, real-time results, assessment of larger tissue volumes, and repeatability for monitoring. While not replacing biopsy for definitive diagnosis and histopathological characterization, SWE may serve as a valuable screening tool, provide additional information for risk stratification, guide timing of biopsy in selected cases, and facilitate serial

monitoring of disease progression. Integration of shear wave elastography into clinical nephrology practice has the potential to improve early detection of CKD, enable more personalized management strategies, and ultimately contribute to better patient outcomes.

VI. CONCLUSION

This study conclusively demonstrates that shear wave elastography provides a reliable, non-invasive method for quantitative assessment of renal parenchymal stiffness in patients with chronic kidney disease. The findings reveal significantly elevated Young's modulus values in CKD patients compared to healthy controls (7.12 ± 3.21 kPa versus 2.89 ± 1.02 kPa), reflecting the underlying pathological process of progressive interstitial fibrosis. The strong inverse correlation between Young's modulus and estimated glomerular filtration rate validates this technology as a functional biomarker that complements traditional biochemical assessments.

The optimal cutoff value of 4.00 kPa demonstrates exceptional diagnostic performance with sensitivity and specificity of 93.3% and area under the curve of 0.967, indicating near-perfect discrimination between diseased and normal kidneys. The bilateral symmetry of measurements and absence of significant sex-related differences simplify clinical interpretation and application. These results support the integration of shear wave elastography into nephrology practice as a valuable tool for early detection, risk stratification, and potentially monitoring therapeutic response in patients with chronic kidney disease.

Shear wave elastography addresses critical limitations of current diagnostic approaches by offering a non-invasive, safe, cost-effective, and repeatable method for assessing renal parenchymal changes. While not replacing renal biopsy for definitive histopathological diagnosis, SWE provides complementary information that may reduce the need for invasive procedures in selected cases and enable more frequent monitoring of disease progression. Future longitudinal studies with histopathological correlation will further refine the clinical applications and establish this technology as a standard component of comprehensive renal assessment.

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