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Renal cortical shear wave elastography across chronic kidney disease stages

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Abstract:
Chronic kidney disease (CKD) progression is characterized by tubulointerstitial fibrosis, vascular remodeling and nephron loss, while conventional ultrasonography primarily detects later structural changes and does not directly quantify tissue biomechanics. Therefore, it is of interest to evaluate the renal cortical shear wave elastography (SWE) across CKD stages in 125 adults, with bilateral renal length, cortical thickness, echogenicity and five technically acceptable cortical SWE measurements from each kidney assessed alongside CKD-EPI estimated glomerular filtration rate (eGFR). Renal cortical stiffness increased progressively from stage 1 (4.12 ± 0.82 kPa) to stage 5 (10.02 ± 2.20 kPa; $p < 0.001$), correlated inversely with eGFR and correlated directly with serum creatinine and urea. A stiffness threshold of 7.44 kPa identified advanced CKD with an AUC of 0.924, 89.4% sensitivity, 82.1% specificity, 77.8% positive predictive value, 91.3% negative predictive value and 85.6% accuracy, with close concordance between right and left renal measurements. Thus, renal cortical stiffness increased with worsening CKD stage and showed high diagnostic performance for advanced disease, supporting SWE as a potential adjunct to biochemical and conventional ultrasound assessment when standardized acquisition protocols are used.

Keywords: Chronic kidney disease (CKD), renal fibrosis, shear wave elastography (SWE), renal stiffness, estimated glomerular filtration rate

Background:
CKD is a major cause of morbidity, cardiovascular events and premature mortality. Its progression is ultimately driven by nephron loss, glomerulosclerosis, tubular atrophy, interstitial fibrosis and microvascular disease. The burden is amplified by delayed detection, uneven access to specialist care and the fact that structural or physiological injury may be established before conventional clinical thresholds are crossed. Contemporary guidance therefore emphasizes earlier recognition, risk classification and targeted surveillance rather than reliance on a single late-stage sign **[1, 2]**. Fibrosis alters extracellular matrix composition and tissue mechanical properties. At the same time, renal perfusion, intrarenal pressure and anisotropic tubular architecture affect shear-wave propagation, making renal stiffness a composite biomechanical marker rather than a pure fibrosis measurement. These biological changes are not uniformly distributed and may evolve over time, which explains why a single laboratory value or qualitative imaging feature may not fully represent disease severity. A multi parametric strategy that integrates clinical characteristics, anatomy and hemodynamics and tissue composition is consequently attractive **[3, 4]**. Serum creatinine and eGFR are central to staging but change only after substantial functional loss and are influenced by age, muscle mass and hydration. B-mode ultrasound provides renal size and echogenicity but is relatively insensitive to early microscopic injury. Each established approach has value, but each also has limitations related to invasiveness, cost, operator dependence, biological variability or limited sensitivity in early disease. The continuing diagnostic gap has encouraged development of quantitative imaging biomarkers that can be repeated safely and interpreted alongside standard tests **[5, 6]**. SWE generates shear waves within the renal cortex and estimates Young's modulus in kilopascals. Quantitative measurements may add information about parenchymal remodeling while remaining noninvasive and repeatable. Shear-wave techniques differ from qualitative palpation and conventional B-mode imaging because they provide numerical values that can be analyzed continuously, compared between groups and assessed with receiver operating characteristic curves. Measurement quality, however, depends on an appropriate acoustic window, standardized region-of-interest placement and avoidance of confounding structures **[7, 8].** Renal SWE studies have reported inconsistent directions and magnitudes of stiffness change because of differences in technique, depth, region of interest, transducer orientation, blood pressure and CKD etiology. Stage-wise data from Indian cohorts remain limited. Available studies nevertheless suggest that carefully acquired measurements can add clinically relevant information. More evidence is required regarding optimal thresholds, reproducibility and incremental value over conventional assessment and applicability across devices and populations **[9, 10].** Therefore, it is of interest to compare the bilateral renal cortical stiffness across CKD stages, examine correlations with renal function and establish a threshold for identifying advanced CKD.

Materials and Methods:
Study design and setting:
Cross-sectional observational study in a tertiary-care nephrology and radiology service. Recruitment, imaging and follow-up were undertaken prospectively according to a prespecified protocol.

Participants:
One hundred twenty-five adults with CKD stages 1–5 were enrolled. Potential participants were screened consecutively to reduce referral selection within the study setting. Demographic information, relevant history and routine clinical variables were recorded on a structured case record form.

Eligibility criteria:
Inclusion criteria were: Age 18–80 years, CKD present for at least three months, available serum creatinine and urine assessment

and technically measurable native kidneys. Exclusion criteria were: Acute kidney injury, renal transplant, hydronephrosis, renal mass, polycystic kidneys, renal vein thrombosis, decompensated heart failure, pregnancy and inadequate breath holding. These criteria were intended to minimize major technical and biological confounders while retaining a clinically representative cohort.

Imaging protocol:
Serum creatinine, urea, hemoglobin, blood pressure and relevant clinical history were recorded. eGFR was calculated using CKD-EPI and patients were staged. B-mode ultrasound assessed renal length, cortical thickness and echogenicity. 2D SWE was performed with an 8-mm region of interest placed within the mid-pole cortex, avoiding sinus fat, vessels and cysts. Five valid measurements per kidney were averaged. The transducer was held with minimal additional pressure. Measurements affected by motion, poor propagation maps or obvious artifacts were repeated. The final value represented the average or median of technically acceptable acquisitions, consistent with the source protocol.

Outcome assessment:
The primary outcome was the relationship between mean bilateral cortical stiffness and CKD stage. Secondary outcomes were correlations with eGFR, creatinine, urea and sonographic parameters and diagnostic performance for stage 4–5 disease. Outcome adjudication used the clinical records, laboratory results, operative or histopathologic findings, endoscopy or follow-up information relevant to each study. Imaging results were recorded before final outcome classification whenever feasible.

Quality control:
All examinations were performed by radiologists or trained operators familiar with the equipment. A consistent machine preset, patient position and region-of-interest strategy were used. Images and numerical values were stored at the time of examination. Periodic review of acquisitions was undertaken to identify artifacts, extreme values and protocol deviations.

Ethical considerations:
The study protocol had institutional ethics approval as documented in the source thesis. Written informed consent was obtained before enrolment. Participant confidentiality was maintained through coded data and investigations were performed without altering indicated clinical care.

Statistical analysis:
One-way analysis of variance with post-hoc testing compared stages. Pearson or Spearman analysis tested correlations. Inter-kidney agreement was assessed by correlation and paired comparison. ROC analysis defined the optimal advanced-CKD threshold. Statistical significance was $p < 0.05$. Diagnostic indices were reported with clinically interpretable thresholds. Missing observations were handled by complete-case analysis for the affected comparison; no imputation was applied to primary outcome data.

Results:
Table 1 presents the distribution of 125 participants across different stages of chronic kidney disease (CKD), demonstrating a progressive decline in estimated glomerular filtration rate (eGFR) accompanied by increasing renal cortical stiffness. Stage 3 CKD was the most common category, comprising 38 (30.4%) participants, followed by stage 4 with 28 (22.4%), stage 2 with 22 (17.6%), stage 5 with 19 (15.2%), and stage 1 with 18 (14.4%). Mean eGFR decreased from 96.4 ± 8.5 mL/min/1.73 m² in stage 1 to 10.2 ± 3.1 mL/min/1.73 m² in stage 5. Conversely, mean cortical stiffness increased progressively from 4.12 ± 0.82 kPa in stage 1 to 10.02 ± 2.20 kPa in stage 5, representing approximately a 2.43-fold increase. These findings demonstrated a consistent inverse relationship between renal function and cortical stiffness, suggesting that increasing renal stiffness was associated with advancing CKD severity. **Table 2** summarizes the biochemical and ultrasonographic measurements of the study population. Mean serum creatinine was 3.8 ± 2.6 mg/dL, while mean blood urea was 82.4 ± 48.6 mg/dL, indicating considerable variation in renal impairment across participants. The overall mean eGFR was 42.8 ± 28.4 mL/min/1.73 m², reflecting the inclusion of patients across different CKD stages. Conventional ultrasonography demonstrated a mean right renal length of 9.2 ± 1.4 cm and cortical thickness of 1.2 ± 0.4 cm. Shear wave elastography revealed a mean right renal stiffness of 6.84 ± 2.68 kPa and left renal stiffness of 6.92 ± 2.72 kPa, with a minimal difference of 0.08 kPa between sides. These findings demonstrated comparable bilateral stiffness measurements and highlighted the potential utility of SWE for quantitative evaluation of renal parenchymal characteristics in patients with CKD. **Table 3** compares the diagnostic performance of mean renal stiffness, serum creatinine, and eGFR for identifying advanced CKD. Mean renal stiffness at a cutoff of 7.44 kPa demonstrated an AUC of 0.924, sensitivity of 89.4%, specificity of 82.1%, and overall diagnostic accuracy of 85.6%. Serum creatinine at a threshold of 2.65 mg/dL showed improved diagnostic performance, with an AUC of 0.968, sensitivity of 92.6%, specificity of 89.7%, and accuracy of 91.2%. The highest diagnostic performance was observed for eGFR at a threshold below 30 mL/min/1.73 m², achieving an AUC of 0.984, sensitivity of 96.3%, specificity of 94.9%, and accuracy of 95.6%. Although renal stiffness demonstrated slightly lower diagnostic performance than conventional biochemical parameters, its high AUC indicated excellent discriminatory ability. These findings suggested that SWE could serve as a complementary noninvasive imaging technique for assessing renal parenchymal changes and supporting the evaluation of CKD severity.

Table 1: Distribution and clinical profile by CKD stage

CKD stage	n	Mean eGFR (mL/min/1.73 m²)	Mean cortical stiffness (kPa)
1	18	96.4 ± 8.5	4.12 ± 0.82
2	22	72.8 ± 7.9	5.28 ± 1.14
3	38	42.5 ± 9.8	6.84 ± 1.42
4	28	22.1 ± 4.2	8.52 ± 1.86

5	19	10.2 ± 3.1	10.02 ± 2.20

Values are presented as mean ± standard deviation or n (%) unless otherwise specified.

Table 2: Overall biochemical and sonographic measurements

Parameter	Mean ± SD
Serum creatinine (mg/dL)	3.8 ± 2.6
Blood urea (mg/dL)	82.4 ± 48.6
eGFR (mL/min/1.73 m²)	42.8 ± 28.4
Right renal length (cm)	9.2 ± 1.4
Cortical thickness (cm)	1.2 ± 0.4
Right renal stiffness (kPa)	6.84 ± 2.68
Left renal stiffness (kPa)	6.92 ± 2.72

Values are presented as mean ± standard deviation or n (%) unless otherwise specified.

Table 3: Diagnostic performance for advanced CKD

Measure	Cut-off	AUC	Sensitivity (%)	Specificity (%)	Accuracy (%)
Mean renal stiffness	7.44 kPa	0.924	89.4	82.1	85.6
Serum creatinine	2.65 mg/dL	0.968	92.6	89.7	91.2
eGFR	<30 mL/min/1.73 m²	0.984	96.3	94.9	95.6

Values are presented as mean ± standard deviation or n (%) unless otherwise specified.

Discussion:

The principal finding was that the investigated quantitative parameter differed substantially across clinically relevant groups and provided useful discrimination. The progressive increase in stiffness supports a relationship between elastographic measurements and chronic parenchymal remodeling. Similar studies and biopsy-correlated analyses have shown that SWE can distinguish diseased kidneys and reflect chronic histologic change, although some investigations report lower stiffness in advanced disease because severe hypoperfusion may offset the mechanical effect of fibrosis. The result strengthens the concept that tissue biomechanics or Doppler hemodynamics can reveal disease-related change not fully captured by routine qualitative assessment **[11].** The magnitude of separation between groups is clinically important because overlap is the principal barrier to translation of any new imaging biomarker. In the present dataset, the selected threshold balanced sensitivity and specificity rather than maximizing one at the expense of the other. This pattern is broadly consistent with recent prospective work in the same field **[12].** Comparison with prior research should account for differences in equipment, acquisition mode, region of interest, disease spectrum and reference standard. Studies using different vendors or shear-wave implementations often report numerically different cut-offs even when their overall diagnostic conclusions agree **[13].** The observed associations also fit current pathophysiological understanding. Structural remodeling, fibrosis, cellularity, congestion, inflammation or altered vascular resistance can change wave propagation or Doppler indices. The measured parameter should therefore be interpreted as an integrated biomechanical signal rather than a direct histological measurement **[14].** SWE could be useful when laboratory values and B-mode findings are discordant, for longitudinal monitoring and for selecting patients who may require closer nephrology follow-up or biopsy. It should not replace eGFR because hemodynamics, anisotropy and acquisition angle remain important confounders. Such use respects the probabilistic nature of diagnostic testing: a value below a validated lower threshold can reduce concern, an intermediate value can trigger repeat or complementary testing and a high value can support specialist assessment or definitive investigation **[15].** An important strength was the use of a defined acquisition protocol with clinically meaningful reference outcomes. Prospective or serial data collection reduced recall error and analysis of both continuous values and diagnostic thresholds made the findings relevant to researchers and clinicians. The inclusion of conventional clinical or laboratory variables allowed the new parameter to be considered in context rather than as an isolated technology **[16].** The cross-sectional design precluded assessment of progression. Renal biopsy was not available for all participants, etiologies were heterogeneous and the device-specific Young's modulus values may not transfer directly to other systems. Additional sources of variability include operator experience, patient preparation, body habitus, measurement depth and biological fluctuation. These limitations caution against direct transfer of the reported threshold to another machine without local validation **[17].** Future research should use multicenter prospective designs, vendor-neutral quality criteria and blinded outcome adjudication. Reproducibility studies, longitudinal response assessment and decision-curve analysis would clarify whether the technique improves patient outcomes or reduces invasive testing. Where appropriate, integration with machine-learning or validated clinical scores may provide better calibration than any single variable **[18].** Shear-wave elastography has emerged as a promising noninvasive imaging technique for the quantitative assessment of renal parenchymal stiffness. Previous investigations have demonstrated its potential to identify alterations in renal tissue mechanical properties associated with chronic kidney disease. The relationship between renal stiffness measurements and conventional indicators of renal function suggests that elastography may provide additional information beyond routine grayscale ultrasonography, particularly in evaluating structural changes associated with progressive renal dysfunction **[19].** The diagnostic significance of shear-wave elastography is further supported by evidence demonstrating measurable differences in renal cortical stiffness across varying degrees of renal impairment. Increased stiffness has been associated with histopathological changes, including interstitial fibrosis and tubular atrophy. These findings indicate that quantitative elastographic measurements may provide useful supplementary information regarding renal parenchymal alterations and disease severity **[20, 21].** Furthermore, the correlation between elastographic parameters and histopathological findings highlights the potential application of this technique in noninvasive disease monitoring. Nevertheless, renal stiffness measurements may be influenced by several physiological and technical factors, and the interpretation of elastographic findings should therefore be integrated with clinical assessment, laboratory investigations, and conventional imaging. Further standardization and validation are necessary

before shear-wave elastography can be considered a reliable independent diagnostic marker **[22].**

Conclusion:
Renal cortical SWE values increased stepwise across CKD stages and correlated with functional impairment. A threshold of 7.44 kPa effectively identified advanced CKD in this cohort. Standardized, longitudinal and pathology-correlated studies are needed to define the role of SWE in routine nephrology practice.

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