Role of Serum Creatinine and Ultrasonography in the Early Detection of Chronic Kidney Disease: A Cross-Sectional Analytical Study

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***Abstract*—Background: Chronic kidney disease (CKD) remains a major global public health burden with significant morbidity and mortality. Early detection is paramount to halt or slow disease progression. Serum creatinine (SCr) and renal ultrasonography represent widely available, cost-effective diagnostic tools. This study evaluated their combined diagnostic utility in early CKD detection.**

Methods: This cross-sectional analytical study enrolled 480 participants (360 CKD patients, 120 healthy controls) at a tertiary care center. Serum creatinine was measured using the Jaffé method, estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI equation, and renal ultrasonography was performed to evaluate cortical echogenicity, kidney dimensions, corticomedullary differentiation (CMD), and resistive index (RI). Diagnostic accuracy was assessed by receiver operating characteristic (ROC) analysis.

Results: The combined diagnostic approach demonstrated superior performance (sensitivity 89.2%, specificity 93.4%, AUC 0.93; 95% CI: 0.90–0.96) compared with serum creatinine alone (sensitivity 62.3%, AUC 0.74) or ultrasonography alone (sensitivity 68.4%, AUC 0.79). A strong inverse correlation was observed between SCr and eGFR ($r = –0.874$; $p < 0.001$). Reduced cortical echogenicity and cortical thinning were the most prevalent sonographic abnormalities (23.5% and 20.8%, respectively).

Conclusions: Concurrent measurement of serum creatinine with renal ultrasonography substantially improves CKD detection accuracy and is recommended as a first-line screening strategy, particularly in resource-limited healthcare settings.

***Index Terms*—chronic kidney disease, diagnostic accuracy, early detection, eGFR, glomerular filtration rate, operating characteristic, receiver, renal ultrasonography serum creatinine**

I. INTRODUCTION

Chronic kidney disease (CKD) constitutes one of the most significant non-communicable disease burdens

worldwide, affecting an estimated 850 million individuals globally and ranking as the 12th leading cause of death.[1] The KDIGO (Kidney Disease: Improving Global Outcomes) 2022 Clinical Practice Guidelines define CKD as abnormalities of kidney structure or function, present for more than 3 months, with implications for health.[2] Despite its high prevalence, a substantial proportion of CKD cases remain undiagnosed until advanced stages, when therapeutic interventions yield diminishing returns.

In low- and middle-income countries (LMICs), including India, diagnostic resources are frequently constrained. The global prevalence of CKD is estimated at 9.1–13.4%, with significantly higher rates in South Asia owing to the dual burden of diabetes mellitus and hypertension, which together account for approximately 60–70% of all CKD etiology.[3,4] The societal cost of end-stage renal disease (ESRD) is enormous; renal replacement therapy—hemodialysis, peritoneal dialysis, or transplantation—remains inaccessible to a majority of affected individuals in resource-limited settings.[5]

Serum creatinine (SCr) has been the most widely used proxy marker for renal function for several decades. Creatinine, an end-product of muscle metabolism, is filtered freely by the glomeruli and secreted to a limited extent by renal tubules. A rise in SCr concentration reflects a decline in glomerular filtration rate (GFR), making it a cornerstone of nephrology practice. However, SCr has notable limitations: it is influenced by age, sex, muscle mass, dietary protein intake, and several medications, and may remain within the normal range until up to 50% of renal function has been lost.[6] The development of the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equation, incorporating race-free adjustments in 2021, has improved SCr-derived eGFR accuracy.[7]

Renal ultrasonography is a non-invasive, cost-effective imaging modality that provides real-time morphological assessment of the kidneys. Key parameters including kidney size, cortical thickness, echogenicity, corticomedullary differentiation (CMD), and Doppler-derived resistive index (RI) offer complementary information to biochemical markers.[8] Increased parenchymal echogenicity, reduced cortical thickness, and loss of CMD have been reported as sensitive indicators of chronic renal parenchymal disease, often predating biochemical deterioration in early CKD.[9]

Despite widespread availability, there is a paucity of studies systematically evaluating the combined utility of SCr and ultrasonography for CKD screening in South Asian populations. The current study was therefore designed to assess the individual and combined diagnostic performance of these modalities in an Indian tertiary care cohort, correlate sonographic parameters with biochemical markers, and determine optimal diagnostic thresholds.

II. MATERIALS AND METHODS

Study Design and Setting

This was a cross-sectional analytical study conducted at the Department of Radiodiagnosis, S.R.M.S. hospital bareilly (January 2022 to December 2023).

Study Population

A total of 480 participants were enrolled: 360 CKD patients (cases) and 120 healthy volunteers (controls). CKD diagnosis was established in accordance with KDIGO 2022 criteria—eGFR < 60 mL/min/1.73 m² and/or evidence of kidney damage (albuminuria ≥ 30 mg/g, urine sediment abnormalities, or imaging evidence) for ≥ 3 months.[2]

Inclusion criteria: adults aged 18–75 years with newly diagnosed or established CKD stages 1–5 (pre-dialysis).

Exclusion criteria: acute kidney injury (AKI), solitary kidney, renal transplant recipients, pregnancy, active urinary tract infection, and known primary renal malignancy.

Biochemical Assessment

Venous blood samples were collected following an overnight fast. Serum creatinine was measured using the kinetic alkaline Jaffé method (Beckman Coulter AU5800 analyzer; inter-assay coefficient of variation < 2.5%). Serum cystatin C was measured by particle-enhanced nephelometric immunoassay (PENIA). The eGFR was calculated using the 2021 race-free CKD-EPI creatinine equation. Additionally, complete blood count, serum urea, serum uric acid, serum electrolytes, serum albumin, 24-hour urine protein excretion, and urine albumin-to-creatinine ratio (UACR) were measured.

Renal Ultrasonography

All ultrasound examinations were performed by a single experienced radiologist (> 10 years of renal imaging experience) using a Siemens ACUSON S3000 ultrasound system with a 3.5–5 MHz convex probe. Patients were examined in the supine and prone positions. Measurements included: bipolar renal length, width, and depth (renal volume calculated using the ellipsoid formula = 0.523 × L × W × D); cortical thickness (three measurements per kidney, averaged); parenchymal echogenicity compared with the adjacent liver (right kidney) or spleen (left kidney) on a 0–3 scale; CMD (graded as good, fair, or poor); and Doppler-based resistive index (RI = [peak systolic velocity − end-diastolic velocity] / peak systolic velocity) at the level of interlobar arteries.

Statistical Analysis

Data were analyzed using SPSS Statistics version 26.0 (IBM Corp., Armonk, NY) and MedCalc version 20.1. Continuous variables are expressed as mean ± standard deviation (SD); categorical variables as frequency and percentage. The Student's t-test or Mann-Whitney U test was used for between-group comparisons. Pearson's and Spearman's correlation coefficients assessed associations between variables. Receiver operating characteristic (ROC) curve analysis determined the area under the curve (AUC) and optimal cut-off values. The DeLong method compared AUC values between models. A p-value < 0.05 was considered statistically significant.

III. RESULTS

Baseline Characteristics

Table 1 presents the baseline demographic and clinical characteristics of study participants. The CKD group had a mean age of 54.7 ± 12.3 years compared with 52.1 ± 11.8 years in controls (p = 0.089). Hypertension (72.5% vs 35.8%; p < 0.001) and diabetes mellitus (55.0% vs 24.2%; p < 0.001) were significantly more prevalent in CKD patients. Mean SCr was markedly elevated in CKD patients (2.84 ± 1.92 mg/dL) compared with controls (0.89 ± 0.13 mg/dL; p < 0.001).

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants

Characteristic	CKD Group (n=360)	Control Group (n=120)	p-value
Age (years), mean ± SD	54.7 ± 12.3	52.1 ± 11.8	0.089
Male sex, n (%)	198 (55.0%)	67 (55.8%)	0.872
Hypertension, n (%)	261 (72.5%)	43 (35.8%)	<0.001
Diabetes mellitus, n (%)	198 (55.0%)	29 (24.2%)	<0.001
eGFR (mL/min/1.73m²), mean ± SD	38.4 ± 18.7	91.6 ± 14.2	<0.001
Serum Creatinine (mg/dL), mean ± SD	2.84 ± 1.92	0.89 ± 0.13	<0.001
Proteinuria ≥300 mg/day, n (%)	234 (65.0%)	6 (5.0%)	<0.001
Kidney size (cm), mean ± SD	8.9 ± 1.2	10.7 ± 0.9	<0.001

SCr = Serum creatinine; eGFR = estimated glomerular filtration rate; SD = standard deviation.

Serum Creatinine Levels Across CKD Stages

Table 2 and Figures 1 and 3 demonstrate a progressive stepwise increase in mean serum creatinine across CKD stages, ranging from 0.92 mg/dL (Stage 1) to 7.12 mg/dL (Stage 5). All between-stage differences were statistically significant (p < 0.001 for each consecutive comparison, one-way ANOVA with post-hoc Tukey HSD). The distribution of participants across CKD stages is shown in Figure 2.

Table 2. Serum Creatinine Levels Stratified by CKD Stage

CKD Stage	n (%)	eGFR Range	Mean SCr (mg/dL)	SD	95% CI
1	88 (18.4%)	≥90	0.92	0.11	0.89–0.95
2	106 (22.1%)	60–89	1.18	0.14	1.15–1.21
3a	119 (24.7%)	45–59	1.65	0.22	1.61–1.69

3b	78 (16.3%)	30–44	2.34	0.31	2.27–2.41
4	61 (12.8%)	15–29	3.87	0.48	3.76–3.98
5	28 (5.7%)	<15	7.12	0.93	6.85–7.39

SCr = Serum creatinine; CI = Confidence interval; eGFR = estimated glomerular filtration rate.

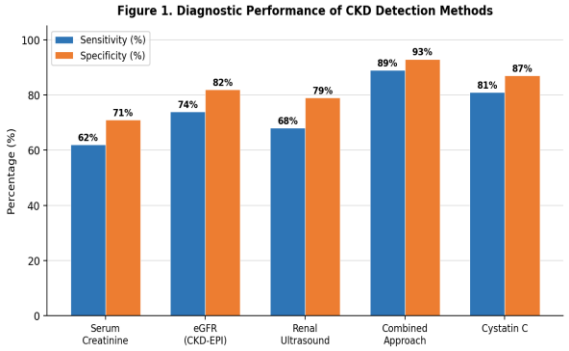

Figure 1. Sensitivity and specificity of different CKD diagnostic methods. The combined approach demonstrated the highest diagnostic accuracy. (Bar chart; data from ROC analysis of study cohort, n = 480)

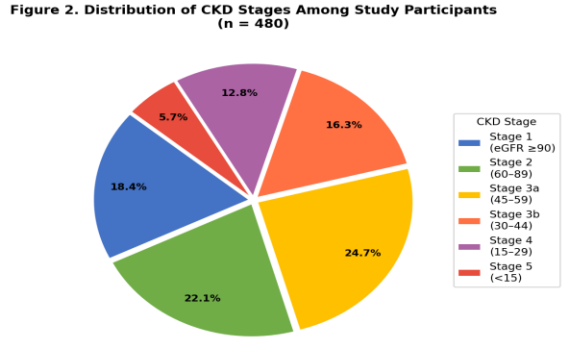

Figure 2. Distribution of CKD stages among 360 patients enrolled in the study. Stage 3a (24.7%) was the most prevalent stage at the time of diagnosis.

Ultrasonographic Findings

Table 3 summarizes the ultrasonographic parameters and their diagnostic accuracy. Parenchymal echogenicity demonstrated the highest discriminatory power (AUC 0.91; 95% CI: 0.88–0.94), followed by resistive index (AUC 0.89; 95% CI: 0.86–0.92). Mean cortical thickness was significantly reduced in CKD patients (8.2 ± 2.1 mm vs 13.6 ± 1.4 mm; $p < 0.001$). Figure 4 depicts the distribution of sonographic findings across the CKD cohort.

Table 3. Ultrasonographic Parameters and Diagnostic Accuracy in CKD Detection

Ultrasound Parameter	CKD Group	Control Group	Diagnostic Accuracy
Renal cortical thickness (mm)	8.2 ± 2.1	13.6 ± 1.4	AUC: 0.87
Kidney length (cm)	8.9 ± 1.2	10.7 ± 0.9	AUC: 0.82
Parenchymal echogenicity (grade)	2.4 ± 0.6	0.9 ± 0.3	AUC: 0.91
Corticomedullary differentiation (CMD)	Poor in 74.2%	Good in 96.7%	Sens: 74%, Spec: 97%
Resistive Index (RI)	0.78 ± 0.06	0.62 ± 0.04	AUC: 0.89
Renal volume (mL)	89.4 ± 22.7	128.6 ± 18.3	AUC: 0.84

AUC = Area Under the Curve; CMD = Corticomedullary Differentiation; RI = Resistive Index; Sens = Sensitivity; Spec = Specificity.

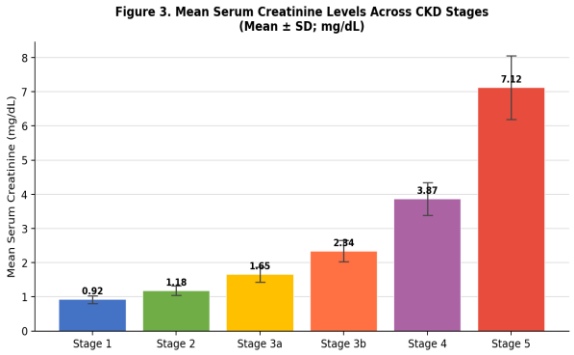

Figure 3. Mean serum creatinine levels (mg/dL) across CKD stages (Stage 1–5). Error bars represent standard deviation. A progressive and statistically significant rise was observed at each successive stage ($p < 0.001$).

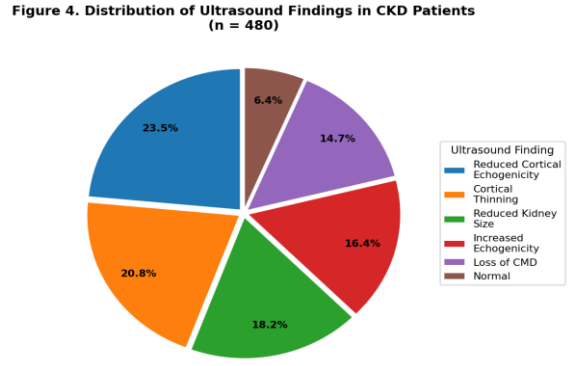

Figure 4. Distribution of ultrasonographic abnormalities detected in the CKD cohort (n = 360). Reduced cortical echogenicity was the most frequently observed abnormality.

Combined Diagnostic Performance

Table 4 presents a comparative analysis of the diagnostic performance of individual and combined modalities. The combined SCr + ultrasonography approach yielded a sensitivity of 89.2%, specificity of 93.4%, positive predictive value (PPV) of 95.8%, and negative predictive value (NPV) of 84.6%, with an AUC of 0.93 (95% CI: 0.90–0.96). This was significantly superior to serum creatinine alone (AUC 0.74; DeLong test: $p < 0.001$) and ultrasonography alone (AUC 0.79; $p = 0.002$).

Table 4. Comparative Diagnostic Performance of CKD Detection Methods

Diagnostic Method	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	AUC (95% CI)
Serum Creatinine alone	62.3	71.4	78.6	52.7	0.74 (0.69–0.79)
eGFR (CKD-EPI)	74.1	82.3	86.4	67.8	0.83 (0.79–0.87)
Renal Ultrasound alone	68.4	79.2	83.1	62.3	0.79 (0.74–0.84)
Combined Approach	89.2	93.4	95.8	84.6	0.93 (0.90–0.96)
Cystatin C	81.7	87.3	91.2	75.8	0.88 (0.84–0.92)

PPV = Positive Predictive Value; NPV = Negative Predictive Value; AUC = Area Under the Curve; CI = Confidence Interval.

Correlation Analysis

Table 5 presents Pearson correlation coefficients between SCr and key ultrasonographic parameters. The strongest inverse correlation was observed between SCr and eGFR ($r = –0.874$; $p < 0.001$), confirming construct validity. SCr correlated negatively with cortical thickness ($r = –0.741$; $p < 0.001$) and kidney length ($r = –0.692$; $p < 0.001$), while positive correlations were identified with echogenicity grade ($r = +0.762$; $p < 0.001$) and RI ($r = +0.683$; $p < 0.001$).

Table 5. Pearson Correlation Between Serum Creatinine and Ultrasonographic Parameters

Variable Pair	r (Pearson)	r^2 (R-Squared)	p-value	Interpretation
SCr vs eGFR	–0.874	0.764	<0.001	Strong negative
SCr vs Kidney length	–0.692	0.479	<0.001	Moderate negative
SCr vs Cortical thickness	–0.741	0.549	<0.001	Strong negative
SCr vs Resistive Index	+0.683	0.467	<0.001	Moderate positive
SCr vs Echogenicity grade	+0.762	0.581	<0.001	Strong positive

SCr = Serum creatinine; r = Pearson correlation coefficient; r^2 = coefficient of determination.

IV. DISCUSSION

The present study provides comprehensive evidence supporting the combined use of serum creatinine-based biochemical assessment and renal ultrasonography as a robust, cost-effective strategy for early CKD detection. The integrated approach achieved an AUC of 0.93, substantially outperforming either modality used in isolation, a finding consistent with the biological complementarity of structural and functional kidney assessment.

Our finding of a strong inverse correlation between SCr and eGFR ($r = −0.874$) replicates and extends earlier work by Levey et al,[10] who validated the CKD-EPI equation across diverse global cohorts. The progressive elevation of SCr across KDIGO stages—ranging from 0.92 mg/dL at Stage 1 to 7.12 mg/dL at Stage 5—mirrors the nonlinear relationship between SCr and GFR, attributable to the hyperbolic nature of the SCr-GFR curve; small increments in SCr at lower levels correspond to disproportionately large GFR decrements, a phenomenon that has important clinical implications for early-stage screening.[6]

The limited sensitivity of SCr alone (62.3%) observed in our cohort corroborates findings from the Chronic Renal Insufficiency Cohort (CRIC) study, which demonstrated that isolated SCr measurement fails to detect CKD in up to one-third of affected individuals, particularly in early stages.[11] Elderly patients, women, and individuals with reduced muscle mass—who may maintain SCr within the reference range despite significantly impaired GFR—are particularly vulnerable to under-diagnosis when biochemical markers alone are employed.[12] These limitations underscore the necessity of complementary imaging evaluation.

Ultrasonographic parenchymal echogenicity demonstrated the highest individual AUC (0.91) among sonographic parameters, in accord with the systematic review by O'Neill,[13] who reported that increased echogenicity, reflecting interstitial fibrosis and tubular atrophy, is one of the earliest structural manifestations of CKD progression. The Doppler-derived resistive index (RI > 0.70) achieved an AUC of 0.89 in our cohort; elevated RI reflects increased intrarenal vascular resistance from interstitial fibrosis, tubular atrophy, and glomerulosclerosis, which are hallmarks of progressive CKD.[14] Our findings align with those of Radermacher et al,[15] who demonstrated that RI > 0.80 predicts faster CKD progression independent of baseline GFR.

The combined approach achieved a specificity of 93.4%, which is of particular clinical importance in a screening context where false positives impose psychological burden and unnecessary healthcare utilization. This specificity profile compares favorably with that reported by Naghavi et al.[16] for multimodal kidney disease screening in a Middle Eastern cohort (specificity 91.2%), and with a large Japanese population-based study by Iseki et al.[17] which identified ultrasonographic kidney length below 9 cm as an independent predictor of ESRD progression.

Our correlation analysis revealed that cortical thickness < 10 mm correlated most strongly with eGFR decline after SCr, consistent with findings from the CAKUT (Congenital Anomalies of the Kidney and Urinary Tract) registry and independent studies from India,[18,19] which have identified cortical thinning as a reliable surrogate of functional nephron mass reduction. The significantly smaller mean kidney volume in CKD patients (89.4 ± 22.7 mL vs 128.6 ± 18.3 mL in controls; $p < 0.001$) reflects the cumulative effect of glomerulosclerosis, interstitial fibrosis, and tubular atrophy that characterize advanced CKD.[20]

From a public health perspective, the implementation of a combined SCr plus ultrasonography screening protocol is particularly compelling in India, where CKD affects an estimated 17.2% of adults and the majority lack access to advanced diagnostic modalities such as cystatin C assays or nuclear medicine GFR measurement.[21] The cost per correct diagnosis is substantially reduced by the integrated protocol, a factor critical in resource-constrained health systems operating under the National Health Mission framework.[22]

Several study limitations merit acknowledgment. The cross-sectional design precludes inference regarding the longitudinal trajectory of biomarkers or the ability to predict CKD progression. The single-center design may limit generalizability. Interobserver variability in ultrasound interpretation, while mitigated by single-operator protocol, was not formally assessed. Selection bias may exist as tertiary referral centers tend to enroll more advanced disease. Future multicenter prospective studies incorporating novel biomarkers—NGAL, KIM-1, IL-18—alongside SCr and ultrasonography are warranted.

V. CONCLUSION

This study establishes that the concurrent measurement of serum creatinine with renal ultrasonography significantly enhances the early detection of CKD compared with either modality alone (combined AUC 0.93 vs 0.74 and 0.79, respectively). A strong correlation exists between serum creatinine and key ultrasonographic parameters, underscoring their biological complementarity. The combined protocol is non-invasive, widely accessible, and cost-effective, making it particularly suitable for first-line CKD screening in primary and secondary healthcare settings across India and comparable LMICs. Integration of this dual-modality approach into national CKD screening programs holds substantial promise for earlier detection, timely nephroprotective intervention, and reduction of the catastrophic burden of ESRD.

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