Correlation of Left Atrial Volume Index with Estimated Glomerular Filtration Rate in Chronic Kidney Disease: A Cross-Sectional Echocardiographic Study

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Keywords. Chronic kidney disease (CKD); Left atrial volume index (LAVi); Estimated glomerular filtration rate (eGFR)

Brief Communication

Introduction. Chronic kidney disease (CKD) drives progressive left heart remodeling through volume overload, inflammation, and hemodynamic stress, manifesting as left atrial enlargement and diastolic dysfunction. This cross-sectional study evaluated correlations between estimated glomerular filtration rate (eGFR) and left heart echocardiographic parameters—including left atrial volume index (LAVi), E/e' ratio, and atrial filling-related parameters—in CKD stages 2-4 to assess their utility as rapid, non-invasive predictors of renal function decline, particularly in emergency settings without laboratory access.

Methods. 100 consecutive CKD patients (eGFR < 60 mL/min/1.73 m², excluding LVEF < 35%, atrial fibrillation, poor acoustic windows) underwent transthoracic echocardiography (Philips EPIQ CVx) at Modarres Hospital, Tehran (2020-2021). Confirmed left heart parameters included: LAVi (biplane area-length method, indexed to BSA); E/e' ratio (pulsed-wave mitral E-velocity ÷ septal tissue Doppler e'-velocity); atrial filling parameters per ASE guidelines. eGFR calculated via CKD-EPI. Statistical analysis comprised Pearson/Spearman correlations, t-tests/Mann-Whitney U, and ROC curves (SPSS v22, $P < .05$).

Results. Mean age 61.1 ± 12.7 years (65 males); eGFR 60.76 ± 14.15 mL/min/1.73 m². Strong inverse correlation: eGFR vs LAVi (r = -0.92, $P < .001$). Moderate inverse correlations: eGFR vs E/e' (r = -0.54, $P < .0001$). Atrial filling parameters progressively impaired with CKD advancement. LAVi demonstrated excellent discriminatory performance: AUC 0.98 (95% CI 0.97-1.00) for stage 2 vs 3-4; AUC 0.92 (95% CI 0.86-0.97) for stage 3. Optimal cutoffs: ≥ 30 mL/m² (sens 100%, spec 91.4%); ≥ 41 mL/m² (sens 100%, spec 97.9%). Performance robust across LVEF subgroups.

Conclusion. LAVi, E/e', and atrial filling parameters exhibit strong, progressive correlations with eGFR decline in CKD, offering excellent bedside discrimination of disease stages independent of laboratory measures. These left heart indices support rapid cardio-renal risk stratification; multicenter longitudinal validation warranted.

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INTRODUCTION

Chronic kidney disease (CKD) represents a global public health issue, with an estimated prevalence approaching 13.1% of the population in certain regions[1,2] and disproportionately higher rates in low- and middle-income countries.[3] Beyond its

renal implications, CKD is intricately associated with cardiovascular diseases (CVD), which remains the leading cause of mortality in this population.[4] Patients with CKD are significantly more likely to experience cardiovascular events, such as coronary artery disease, heart failure, arrhythmias, and sudden cardiac death, than to progress to end-stage kidney disease (ESKD).[4] The elevated cardiovascular risk is present even in the early stages of CKD and intensifies as renal function declines.[4] Shared risk factors, such as hypertension, diabetes mellitus, and dyslipidemia, and CKD-specific mechanisms such as chronic inflammation, oxidative stress, vascular calcification, and abnormal calcium-phosphate metabolism, drive the pathophysiological interplay between CKD and CVD.[5] Furthermore, CKD contributes to adverse cardiac remodeling, including left ventricular hypertrophy and myocardial fibrosis, underscoring the bidirectional relationship between cardiac dysfunction and declining renal function.[6] The complex cardio-renal interaction highlights the need for integrated approaches for managing CKD and mitigating its cardiovascular outcomes.

In advanced CKD, progressive atrial remodeling manifests through structural and functional changes, with the left atrial volume index (LAVi) emerging as both a prognostic and diagnostic tool.[7] Studies demonstrate that LAVi is a strong predictor of dialysis initiation, particularly in advanced CKD stages, where elevated values independently correlate with a shorter time before renal replacement therapy is required.[7-9] Simultaneously, LAVi is a diagnostic marker of chronic diastolic dysfunction, reflecting the cumulative hemodynamic burden through its strong inverse relationship with estimated glomerular filtration rate (eGFR) estimated glomerular filtration rate (eGFR), calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation and association with elevated cardiac filling pressures.[10] The role of left atrial volume index (LAVi) as a rapid, non-invasive predictor of kidney function across all stages of CKD remains unclear. Additionally, the usefulness of echocardiographic parameters such as LAVi, pulmonary artery pressure, and E/e' ratio which is a useful echocardiographic measure applied to evaluate left ventricular filling pressure and diastolic function, determined by dividing the early mitral inflow velocity (E wave) by the early mitral annular tissue velocity (e' wave), in urgent clinical settings without relying on laboratory eGFR measurements has not been well studied.

We aimed to assess the correlation between eGFR and leftheart echocardiographic parameters, including left atrial volume index (LAVi), pulmonary artery pressure (PAP), and E/e' ration to explore their potential prognostic role in predicting renal function.

MATERIALS AND METHODS

Participants and Study Design

This cross-sectional study was conducted on patients with chronic kidney disease (CKD) attending the Cardiology and Nephrology departments of Modarres Hospital, Tehran, Iran, between 2020 and 2021. A total of 100 participants were selected via consecutive, non-random sampling. The inclusion criteria comprised adult patients with an eGFR below 60 mL/min/1.73 m² for over three months and/or an albumin-to-creatinine ratio exceeding 30 mg/g in at least two urine samples. Patients were excluded based on the following criteria: atrial fibrillation or flutter, left bundle branch block, congestive heart failure, mitral valve stenosis or insufficiency, intracardiac shunts, poor acoustic windows, and left ventricular ejection fraction (LVEF) below 35%.

All participants provided written informed consent after receiving detailed explanations about the study protocol. The study was approved by the institutional ethics committee (IR.SBMU.RETECH.REC.1400.555) and conducted in accordance with the Declaration of Helsinki. Participants were not charged for any procedures, and clinical management decisions were independent of study participation. Personal identifying information was coded and securely stored to ensure confidentiality.

Echocardiographic Data Collection

Transthoracic echocardiography (TTE) was performed with patients positioned in the left lateral decubitus orientation using the Philips EPIQ CVx system (Philips Healthcare). Echocardiographic images were acquired and analyzed using the integrated software on the EPIQ CVx platform. Left atrial (LA) size was measured at end-systole, corresponding to maximal LA chamber volume, using apical 2-chamber and 4-chamber views. Left atrial volume (LAV) was calculated using

the biplane area-length method, and the left atrial volume index (LAVi) was derived by indexing LAV to the patient's body surface area. Additional parameters, including pulmonary artery pressure (PAP) and the E/e' ratio, were also measured according to standard echocardiographic protocols.

To assess LV filling pressure and diastolic function, E/e' ratio was utilized; which early transmitral flow velocity (E) was measured using pulsed-wave Doppler at the mitral leaflet tips in the apical four-chamber view, while early diastolic mitral annular velocity (e') was obtained by tissue Doppler imaging (TDI) at the septal annulus. The E/e' ratio was then calculated by dividing the transmitral E-wave velocity by the corresponding e' velocity. Averaged values from three consecutive cardiac cycles were used to minimize beat-to-beat variability, in accordance with the American Society of Echocardiography guidelines.[11] Pulmonary artery systolic pressure (PAP) was estimated from the peak tricuspid regurgitation velocity using the simplified Bernoulli equation with the addition of an estimated right atrial pressure derived from inferior vena cava respiratory variation.

All echocardiographic assessments were performed by a cardiologist with subspecialty training in echocardiography, ensuring consistency and accuracy.

Statistical Analysis

Data analysis was performed using SPSS software version 22 (IBM Corp., Armonk, NY, USA). Quantitative variables were presented as mean ± standard deviation (SD), and qualitative variables were expressed as counts and percentages. The normality of continuous variables was assessed using the Kolmogorov-Smirnov test.

Correlations between continuous variables, including eGFR and echocardiographic parameters (LAVi, PAP, and E/e'), were examined using Pearson's correlation coefficient for normally distributed data or Spearman's rank correlation for non-normally distributed data.

Inter-group comparisons of echocardiographic parameters (LAVi, PAP, and E/e') among CKD stages were performed using the independent samples t-test for normally distributed variables or the Mann-Whitney U test for non-parametric data.

Receiver operating characteristic (ROC) curve analyses were conducted to evaluate the ability of LAVi to discriminate among different CKD stages. Optimal cutoff values were determined via exploratory data analysis and validated statistically through ROC curves. Comparisons of ROC curves between LVEF subgroups (< 50% and ≥ 50%) were performed to assess potential confounding effects of ejection fraction on the association between LAVi and CKD stage.

A two-tailed *P*-value less than .05 was considered statistically significant for all analyses.

RESULTS

In this study, 100 CKD patients, including 65 males and 35 females, were examined, with a mean age of 61.1 ± 12.7 years (ranging from 32 to 80). Table 1 provides additional details on patients' clinical characteristics.

Our findings demonstrated a strong and statistically significant inverse correlation between LAVi and eGFR (r = -0.92, $P < .001$) (Figure 1). Furthermore, there was a significant but weak positive correlation between eGFR and LVEF (r = 0.25, $P = .005$). Conversely, significant, moderate, inverse correlations were identified between eGFR and both PAP (r = -0.54, $P < .0001$)

Table 1. Descriptive statistics on patients' clinical characteristics

Variable	Mean ± SD	Range
Creatinine (mg/dL)	1.25 ± 0.38	0.8-3
eGFR (mL/min/1.73 m^2)	60.76 ± 14.15	22-88
LVEF (%)	52.17 ± 6.2	35-60
LAVi (mL/m^2)	30.22 ± 6.71	17-46
PAP (mmHg)	28.12 ± 5.94	20-43
E/e'	11.18 ± 3.99	5.5-23

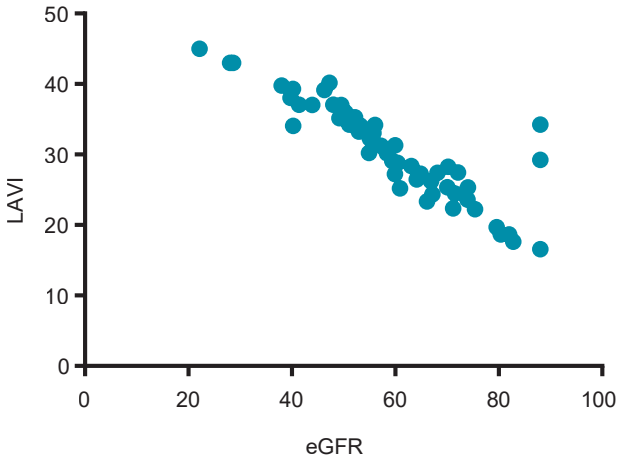

Figure 1. Correlation between eGFR and LAVi (r = -0.92, $P < .0001$).

and the E/e′ ratio (r = -0.54, $P < .0001$).

Discriminatory Performance of LVAi for CKD Staging

ROC curve analyses demonstrated that LAVi effectively distinguished progressive CKD stages (Figure 2). When stage 2 was defined as the classification threshold, the area under the curve (AUC) for LAVi was 0.98 (95% CI: 0.97–1.00), indicating outstanding discriminatory ability. Using stage 3 as the state variable yielded an AUC of 0.92 (95% CI: 0.86–0.97), suggesting moderate accuracy. These findings confirm that left atrial enlargement, quantified by LAVi, correlates with structural cardiac remodeling associated with renal functional decline.

Subgroup Analysis by Ejection Fraction

To address the impact of ejection fraction on the relationship between left atrial volume index (LAVi) and advanced CKD, a subgroup analysis was performed based on left ventricular ejection fraction (LVEF). Patients were stratified into two categories: those with LVEF < 50% (including HFmrEF) and those with LVEF ≥ 50% (HFpEF). ROC curves for LAVi in identifying advanced CKD were constructed separately for each subgroup.

As demonstrated by the ROC curves in Figure 3, LAVi retained its strong discriminatory power for advanced CKD in both EF subgroups, with nearly identical performance across the two categories (AUC difference = 0.015,95% CI: -0.008 to 0.038; $P = .20$). The sensitivity and specificity metrics remained consistently high regardless of LVEF status. These findings suggest that while HFmrEF and HFpEF may act as potential confounders in studies of left atrial remodeling, the association between elevated LAVi and advanced CKD in our cohort is considerable and not significantly attenuated by differences in ejection fraction.

Determination of LVAi Cutoff values

The optimal cut-off values for LAVi were established through an iterative process of exploratory data analysis to effectively differentiate between different stages of CKD. We initially depicted the distribution of LAVi values across CKD stages using scatter plots (Figure 4), enabling us to observe natural clustering and distinction of LAVi in relation to to CKD severity. Based on these visual patterns, two cut-points were selected at 30 and 41 mL/m² that define three LAVi categories reflecting increasing left atrial enlargement severity. These cutoffs align with clinical echocardiographic

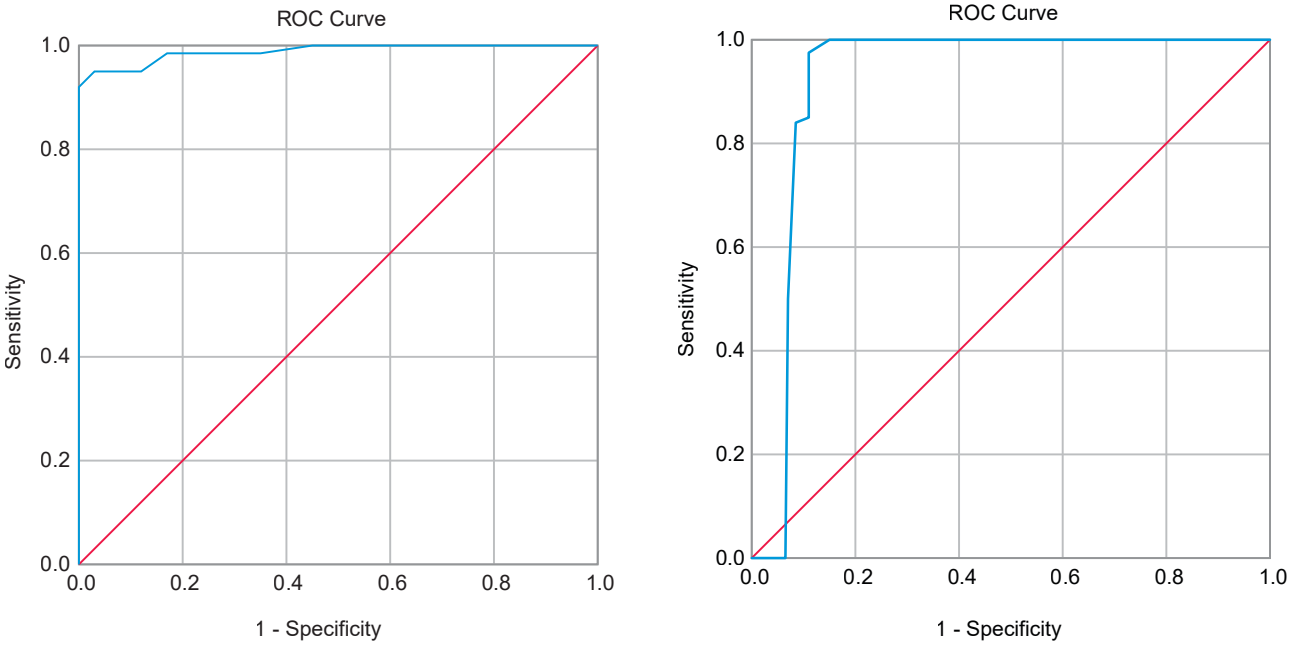

Figure 2. ROC curves: panel 1 illustrates the ROC curve for distinguishing stage 2 from stage 3 and 4 CKD based on LAVi. Panel 2 illustrates the ROC curve for distinguishing stage 3 from 2 and 4 based on LAVi.

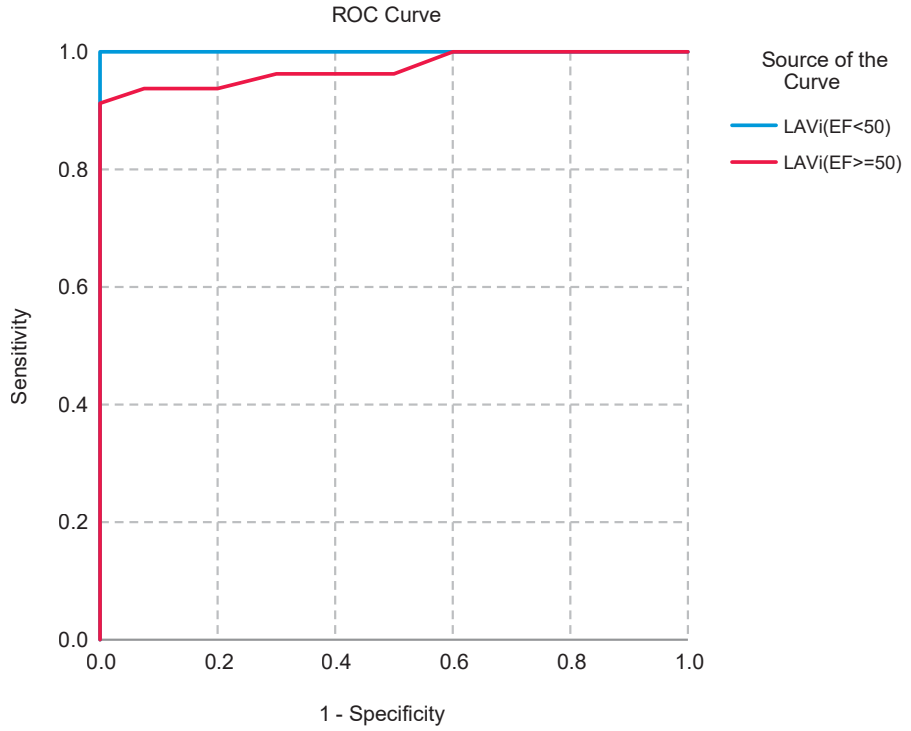

Figure 3. ROC curve discriminatory ability of LAVi in CKD patients with different LVEF status.

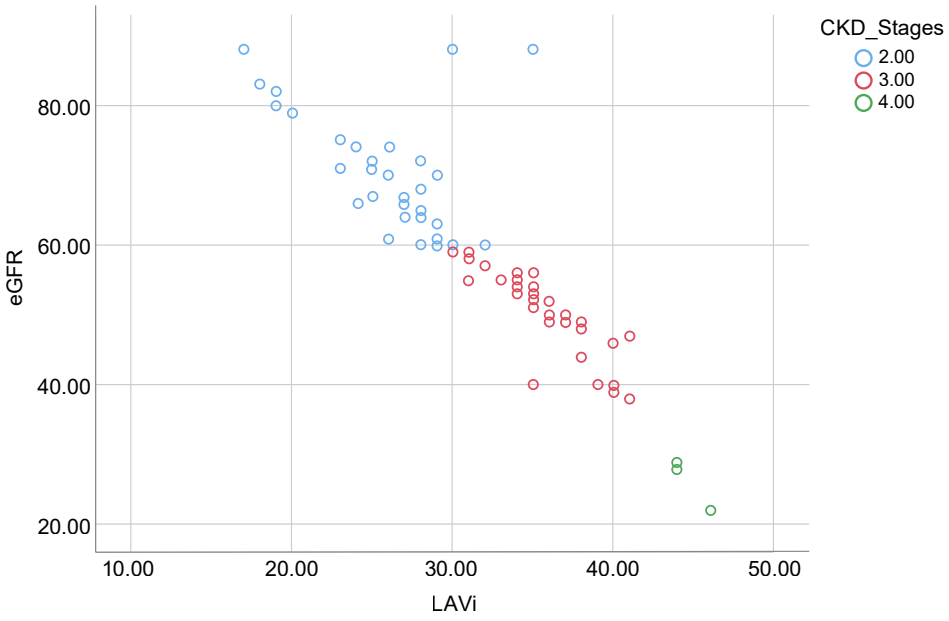

Figure 4. Scatter plot of eGFR based on LVAi.

interpretations and were subsequently validated statistically using receiver operating characteristic (ROC) curve analysis, demonstrating their efficacy in differentiating CKD stages. This combined graphical and statistical approach ensured that the cut-offs were both data-driven and clinically relevant, enhancing the precision of LAVi as a marker of cardiac remodeling in CKD.

Diagnostic Performance of LAVi Cutoffs for CKD Stage Classification

Table 2 summarizes the diagnostic measures for

Table 2. Diagnostic accuracy of LAVi for discrimination between stages of CKD

Comparison (CKD Stage)	LAVi Cutoff (mL/m²)	Sensitivity % (95% CI)	Specificity % (95% CI)	PPV % (95% CI)	NPV% (95% CI)	Interpretation Summary
Stage ≥ 3 vs. Stage 2	≥ 30	100 (91–100)	91.4 (82–97)	88.4 (76–95)	100 (93–100)	Excellent discrimination between early and moderate/ advanced CKD
Stage 3 vs. Stages 2 and 4	30–41 (Category 2)	94.7 (82–99)	91.4 (81–97)	87.8 (73–95)	96.4 (86–99)	LAVi 30–41 effectively identifies Stage 3 CKD
Stage 4 vs. Stages 2 and 3	> 41	100 (40–100)	97.9 (93–100)	66.7 (22–96)	100 (96–100)	Very high accuracy for advanced CKD, limited by the small Stage 4 sample size

LAVi categories based on the cutoff values at 30 and 41 mL/m², discriminating CKD stages 2, 3, and 4. Sensitivity, specificity, positive predictive values (PPV), and negative predictive values (NPV) are calculated for each discrimination, with 95% confidence intervals (CI) computed using the exact Clopper–Pearson method.

Notes

Sensitivity: Proportion of true positive CKD stage cases correctly identified by LAVi category.

Specificity: Proportion of true negative cases correctly excluded by LAVi category.

PPV: Probability that patients with positive LAVi test truly have the CKD stage.

NPV: Probability that patients with negative LAVi test truly do not have CKD stage.

This table demonstrates that LAVi is a strong marker for discriminating CKD progression, especially for distinguishing stage 2 from higher stages and stage 4 from earlier ones. The intermediate category (30–41 mL/m²) performs well for identifying stage 3. The wide confidence intervals in stage 4 sensitivity metrics reflect the limited number of stage 4 patients.

DISCUSSION

This study demonstrates a strong association between worsening renal function and progressive left atrial enlargement in patients with chronic kidney disease, as evidenced by the significant inverse correlation between eGFR and LAVi identified in our cohort. The differentiating ability of LAVi for staging CKD was outstanding, with ROC curve analyses revealing near-perfect efficacy in distinguishing both early (stage 2) from moderate/ advanced (stage 3/4) disease. These results provide compelling evidence that elevated left atrial volume, assessed via echocardiography, reflects structural cardiac remodeling closely associated with declining kidney function, suggesting the potential of LAVi as a mean to identify individuals with advanced kidney disease.

Our finding of a significant inverse correlation between eGFR and LAVi is consistent with prior research demonstrating that left atrial enlargement is more prevalent and prominent in CKD patients.[12] Consisting with existing literature, LAVi is significantly higher in CKD patients compared to those without kidney disease, and progressive increases in left atrial dimensions are associated with worsening CKD stages.[13,14] The prevalence of left atrial enlargement varies considerably in ESKD patients undergoing hemodialysis, ranging from 8.3% to as high as 81%.[15,16] These structural changes also create a substrate for atrial arrhythmias. CKD-induced left atrial enlargement significantly increases atrial fibrillation (AF) vulnerability in experimental models, exhibiting a substantially higher AF development rate in CKD subjects compared to controls.[17]

The association between deteriorating renal function and increased LAVi is probably multifactorial. Volume overload, a common consequence of impaired kidney function in CKD, increases left ventricular end-diastolic pressure, leading to left atrial enlargement.[18] The presence of shared risk factors, such as diabetes mellitus, hypertension, and coronary artery disease, also contribute. Furthermore, CKD-induced inflammation, driven by uremia, and heightened wall stress promote left atrial dilatation.[19,20] The resulting cardiac fibrosis is exacerbated by neurohormonal alterations, oxidative stress, and sympathetic nervous system activation.[19] Additionally, increased activity of renin-angiotensin-aldosterone system (RAAS) promotes myocardial fibrosis through mediators like transforming growth factor-beta 1

(TGF-β1), with these changes manifesting earlier and more prominently in the thin-walled atrium.[19] This structural remodeling is accompanied by molecular changes, including alterations in atrial connexins (gap junction proteins) and activation of inflammatory pathways, which further facilitate arrhythmogenesis.[17]

In addition to LAVi, our results demonstrated significant moderate correlations between pulmonary artery pressure (PAP) and E/e′ ratio with declining renal function. Elevated PAP, indicative of pulmonary hypertension, is a common comorbidity in patients with chronic kidney disease, with a prevalence estimate reaching up to 78% in CKD populations who are referred for right heart catheterization.[21] The presence of elevated PAP reflects increased pulmonary vascular resistance and left heart dysfunction secondary to volume overload, factors frequently observed in advanced CKD.[22] Importantly, elevated PAP has been demonstrated to independently predict adverse cardiovascular outcomes and increased mortality risk in patients with stage 2–4 CKD,[23] underscoring its clinical and prognostic significance.

The E/e′ ratio, a non-invasive echocardiographic marker of left ventricular filling pressures and diastolic dysfunction, also demonstrated significant correlation with CKD severity in our study. Previous investigations have demonstrated that E/e′ serves as a strong predictor of mortality and cardiovascular events in individuals with CKD, and elevated E/e′ values correlating with an increased risk of CKD progression.[24] In dialysis-dependent CKD patients, E/e′ ratios exceeding 14 have been linked to higher risks of heart failure and mortality.[25] This parameter reflects not only structural left atrial remodeling but also functional impairment of diastolic relaxation and ventricular compliance that progressively worsens with declining renal function.[26]

Together, PAP and E/e′ provide complementary prognostic information as markers of hemodynamic stress and subclinical cardiac dysfunction beyond atrial enlargement. These echocardiographic parameters may serve as valuable adjuncts for earlier detection and risk stratification of cardiovascular complications in CKD patients. Future studies should explore their combined prognostic value for predicting clinical outcomes such as heart failure progression, hospitalization, and mortality in this high-risk population.

Limitations

Several limitations should be considered when interpreting the results of this study. First, the cross-sectional design precludes determining causality; we can only infer association rather than a direct cause-and-effect relationship between declining eGFR and increased LAVi. Second, the use of non-random sample from a single center with modest sample size (n = 100), may limit the generalizability of the findings. Third, our cohort included only patients with CKD at stages 2–4, with no patients in stage 5 (eGFR < 15 mL/min/1.73m²); therefore, the discriminatory ability of LAVi in end-stage kidney disease cannot be extrapolated from this study. Fourth, we lacked detailed information on all potential confounders, such as the duration and severity of hypertension, proteinuria levels, or the specific medications (particularly renin-angiotensin system inhibitors or diuretics) used by the patients. Fifth, although the AUC values for LAVi discrimination were excellent, the small sample size may have contributed to overfitting, and these results require validation in larger, multi-center cohorts. Finally, the exclusion of patients with LVEF < 35% means that our findings may not be applicable to patients with significantly reduced systolic function, though subgroup analysis by LVEF did not reveal substantial confounding in our sample.

Future studies are recommended to employ longitudinal design, larger and more ethnically diverse populations from multiple centers, and collect complete data on relevant confounders to validate these findings and establish the clinical utility of LAVi-guided risk stratification and intervention in CKD populations.

CONCLUSION

This study demonstrates a strong inverse correlation between eGFR and LAVi, PAP, and E/e′ in CKD patients. LAVi showed excellent discriminatory ability for identifying advanced CKD stages and remained strong even after stratification by ejection fraction. These findings suggest that LAVi, along with complementary echocardiographic parameters, could serve as a valuable, readily accessible marker for assessing

cardiac remodeling and risk stratification in CKD. The association between renal dysfunction and left atrial enlargement highlights the importance of comprehensive cardiac evaluation in CKD patients.

ABBREVIATIONS

AF: Atrial Fibrillation
CKD: Chronic Kidney Disease
CVD: Cardiovascular Disease
eGFR: Estimated Glomerular Filtration Rate
ESKD: End-Stage Kidney Disease
LA: Left Atrium
LAV: Left Atrial Volume
LAVi: Left Atrial Volume Index
LV: Left Ventricle
LVEF: Left Ventricular Ejection Fraction
PAP: Pulmonary Artery Pressure
P-WD: P-wave Dispersion
RAAS: Renin-Angiotensin-Aldosterone System
SD: Standard Deviation
TGF-β1: Transforming Growth Factor Beta 1
TTE: Transthoracic Echocardiography

DECLARATIONS

Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Shahid Beheshti University of Medical Sciences (IR.SBMU.RETECH.REC.1400.555). All participants provided written informed consent prior to enrollment in the study.

Consent for Publication

Not applicable. This manuscript does not contain any individual person's data in any form (including individual details, images, or videos).

Availability of Data and Materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

AB: Conceived the study, designed the protocol, supervised data collection, and critically revised the manuscript.

SJB: conducted the initial statistical analysis and drafted the initial manuscript.

AM: Assisted with data collection, performed a detailed literature review, and contributed to the writing and editing of the manuscript.

MH: Assisted with data collection, performed a detailed literature review, and contributed to the writing and editing of the manuscript.

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