Original Article

Value of two-dimensional speckle-tracking echocardiography in the assessment of left atrial function in patients with chronic kidney disease

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Background: The rising prevalence of chronic kidney disease (CKD) has emerged as a global public health concern, posing a significant threat to human health. This study aimed to assess changes in left atrial (LA) function in patients with CKD with left ventricular hypertrophy (LVH) using two-dimensional speckle-tracking echocardiography (2D-STE) and to investigate the independent correlations between baseline parameters and LA strain and strain rate.

Methods: We conducted a prospective study that included patients diagnosed with CKD at Shenzhen People's Hospital between November 2020 and September 2021. Healthy participants were enrolled as a healthy control group. Conventional transthoracic echocardiography was performed to obtain conventional ultrasound parameters, with cines analyzed offline to determine strain and strain rate parameters. Single-factor analysis of variance was used to compare the groups. The relationship between different variables and LA strain and strain rate was analyzed by general linear regression. The relationship between left ventricular mass index (LVMI) and LA strain and strain rate was analyzed by multifactor linear regression.

Results: The study included 236 participants: 166 patients with CKD (85 in the $CKD_{non\text{-}LVH\ (N\text{-}LVH)}$ group and 81 in the CKD_{LVH} group) and 70 healthy controls (CON group). The results showed that LA volume in the $CKD_{N\text{-}LVH}$ group was not significantly different compared with that in the CON group (P>0.05), but the remaining LA strain and strain rate parameters were decreased (P<0.05), except for the LA global longitudinal strain during early diastole (LA Se) and LA global longitudinal strain rate during late diastole (LA SRa) (P>0.05). In the CKD_{LVH} group, LA strain and strain rate were further reduced as compared to those in the $CKD_{N\text{-}LVH}$ group (P<0.05). Additionally, LA strain and strain rate were negatively correlated with age [*vs.* LA global longitudinal strain during systole (LA Ss): R=–0.36, P<0.001; *vs.* LA global longitudinal strain rate during systole (LA SRs): R=–0.24, P<0.001], systolic blood pressure (*vs.* LA Ss: R=–0.38, P<0.001; *vs.* LA SRs: R=–0.43, P<0.001), A peak (*vs.* LA Ss: R=–0.36, P<0.001; *vs.* LA SRs: R=–0.34, P<0.001), E/*e'* (*vs.* LA Ss: R=–0.44, P<0.001; *vs.* LA SRs: R=–0.54, P<0.001), LA volume index (LAVI) (*vs.* LA Ss: R=–0.35,

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P<0.001; *vs.* LA SRs: R=–0.52, P<0.001), and LVMI (*vs.* LA Ss: R=–0.46, P<0.001; *vs.* LA SRs: R=–0.55, P<0.001); meanwhile, LA strain and strain rate were positively correlated with glomerular filtration rate (GFR) (*vs.* LA Ss: R=0.50, P<0.001; *vs.* LA SRs: R=0.50, P<0.001) and *e'* (*vs.* LA Ss: R=0.58, P<0.001; *vs.* LA SRs: R=0.54, P<0.001). LVMI had an independent negative effect on all LA strain and strain rates (*vs.* LA Ss: β=–0.29, P<0.001; *vs.* LA SRs: β=–0.42, P<0.001).

Conclusions: LA strain and strain rate are valuable indicators for detecting early LA functional changes in patients with CKD. LVMI independently negatively impacts all LA strain and strain rates and may be a predictor of cardiovascular events.

Keywords: Chronic kidney disease (CKD); two-dimensional speckle-tracking echocardiography (2D-STE); strain; strain rate; left ventricular hypertrophy (LVH)

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Introduction

The rising prevalence of chronic kidney disease (CKD) has emerged as a global public health concern, posing a significant threat to human health (1). In 2017, a systematic analysis of the global all-age Global Burden of Disease (GBD) study project found that there were 697.5 million cases of CKD at all stages worldwide, representing a global prevalence of 9.1%, with CKD and its impact on cardiovascular disease (CVD) causing 2.6 million deaths (2). As renal dysfunction progresses, the prevalence of CVD in patients with CKD is directly related to the severity of CKD (3). Many patients with CKD succumb to severe complications related to renal dysfunction before reaching the end stage of the disease, with CVD being the most prevalent complication and leading cause of death among this population (1).

During the cardiac cycle, the left atrium (LA) is closely linked to the left ventricle, and LA function is essential for maintaining efficient cardiac function, especially during exercise (4,5). It helps increase stroke volume and cardiac output by enhancing left ventricular (LV) filling. Structural and functional changes in the LA, such as dilation and reduced compliance, are associated with adverse cardiovascular outcomes, including atrial fibrillation, heart failure, and stroke (4,6,7).

Changes in LA volume and diameters are independent risk factors for CVD, death, or atrial fibrillation in patients with CKD (8-10). Moreover, research has indicated (11-13) that alterations in LA function often occur before structural changes, and that two-dimensional speckle-tracking echocardiography (2D-STE) is more effective in detecting these functional changes earlier than is conventional echocardiography.

2D-STE tracks the spatial movement of echo specks within the myocardium to reflect the real-time movement and deformation of the myocardial tissue. It is a critical tool for evaluating LA function, as it can evaluate LA mechanics by measuring strain and strain rate, which reflect the deformation and deformation rate of the atrial myocardium, respectively. 2D-STE is superior to conventional echocardiography in detecting subclinical myocardial dysfunction due to its higher sensitivity, angle independence, comprehensive assessment of myocardial mechanics, and superior prognostic ability (14,15).

LA strain and LA volume index (LAVI) are more sensitive parameters than are traditional echocardiographic parameters and LV strain in patients with early CKD (16). Saito *et al.* (17). demonstrated that myocardial fibrosis can be detected by 2D strain. Given that the atrium has thinner walls than the ventricle has, it is possible that alterations in myocardial deformation may be detected earlier in the atrium through use of strain analysis. With the deterioration of renal function, the risk of CVDs in patients with CKD significantly increases, which emphasizes the importance of early identification and intervention.

Although numerous studies have employed 2D-STE to evaluate changes in left ventricle geometry (such as increased wall thickness and/or dilation) and function in patients with CKD (18-20), few studies have focused on changes in LA function. Therefore, we conducted this study to assess the changes in LA function in patients with CKD with LV hypertrophy (LVH) using 2D-STE, and to explore

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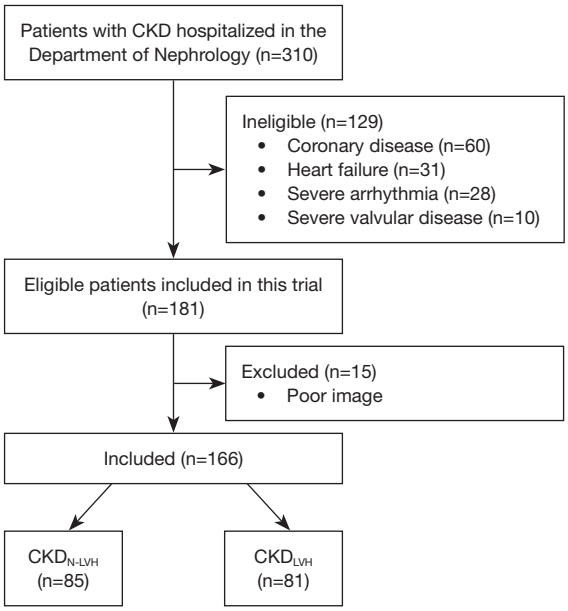

Figure 1 The study flowchart of the inclusion and exclusion of patients with CKD. CKD, chronic kidney disease; LVH, left ventricular hypertrophy; N-LVH, non-LVH.

the independent correlations between baseline parameters and LA strain and strain rate. We present this article in accordance with the STROBE reporting checklist (available at https://qims.amegroups.com/article/view/10.21037/qims-24-1537/rc).

Methods

Study population

The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Provincial Ethics Committee of Shenzhen People's Hospital (No. LL-KY-2021282). All individuals in this prospective study provided written informed consent prior to their participation.

The study included 166 patients with CKD who visited Shenzhen People's Hospital between November 2020 and September 2021. The diagnostic criteria for CKD (21) were a glomerular filtration rate (GFR) <60 mL/min/1.73 m^2 for ≥3 months with or without renal injury, including structural and functional renal abnormalities for ≥3 months with or without a GFR decline. Patients were excluded if they had a low ejection fraction (EF <50%), coronary artery disease, severe valvular disease, congenital heart disease, or severe arrhythmia (including atrial fibrillation) or had undergone renal transplantation. All patients were clinically and hemodynamically stable. The study flowchart of the inclusion and exclusion of patients with CKD is shown in *Figure 1*. Additionally, 70 healthy participants were collected as a healthy control group, and people with underlying diseases such as heart disease, diabetes, liver and kidney diseases, etc. were excluded.

Clinical characteristics

Clinical data collected included gender, age, height, weight, blood pressure, heart rate, GFR, serum creatinine (SCr), blood urea nitrogen (BUN), echocardiographic parameters, the body mass index (BMI) [BMI = weight (kg)/height^2 (m^2)], and body surface area (BSA) [BSA = 0.010061 × height (cm) + 0.010124 × weight (kg) – 0.010099].

Echocardiographic analysis

A comprehensive transthoracic echocardiography (TTE) scan was conducted with the participants at rest with a Vivid E95 diagnostic ultrasound machine (GE HealthCare, Chicago, IL, USA) equipped with an M5S probe at 3.5 MHz. The participants were instructed to lie in the left decubitus position, and the electrocardiogram was connected and recorded simultaneously to ensure optimal image quality. LV ejection fraction (LVEF) was determined using the biplane Simpson method. M-mode echocardiographic measurements included LV end-diastolic dimension (LVEDD) and LV end-systolic dimension (LVESD), interventricular septal thickness at end-diastole (IVSd), posterior wall thickness at end-diastole (PWTd), and systolic anterior-posterior LA diameters in the end-systolic period (LAd) in long-axis views from the parasternal sternum. LV mass (LVM) and LV mass index (LVMI) were calculated using the Devereux correction formula as follows: LVM (g) = 0.8 × {1.04[(IVSd + PWTd + LVEDD)^3 – LVEDD^3] + 0.6}; LVMI (g/m^2) = LVM/BSA. The criterion for LVH being LVMI was ≥115 g/m^2 for men and was ≥95 g/m^2 for women (22). LV end-diastolic volume (LVEDV) was calculated using the Teichholtz correction formula (23) as follows: LVEDV =7.0 × LVEDD^3/(2.4 + LVEDD). The LV end-diastolic volume index (LVEDVI) was normalized to BSA (LVEDV/BSA). All recordings and measurements adhered to the guidelines of the American Society of Echocardiography (24). Pulsed Doppler was employed to measure early diastolic peak flow velocity (E peak), late

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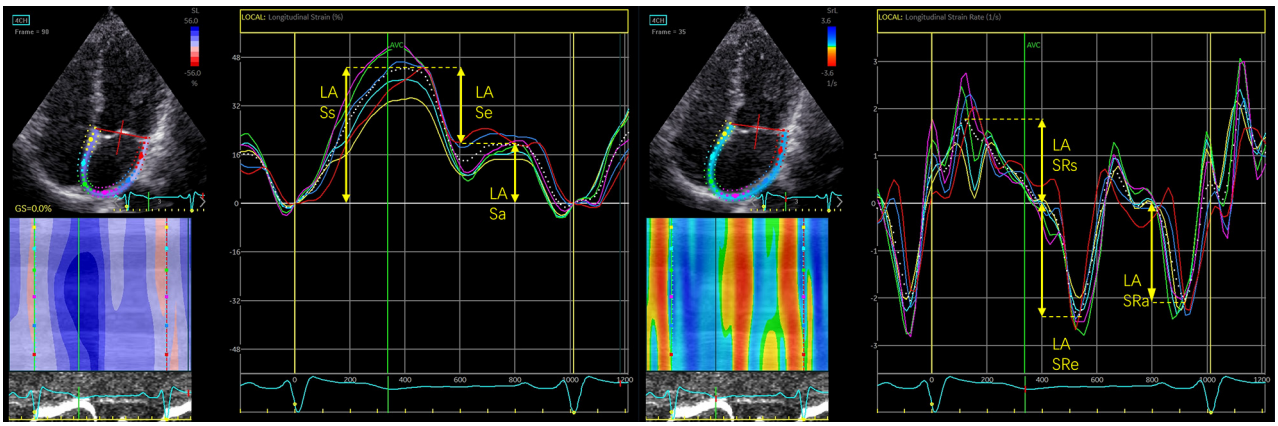

Figure 2 Schematic of strain measurements (left) and strain rate measurements (right) in normal participants. LA Ss, left atrial global longitudinal strain during systole; LA Se, left atrial global longitudinal strain during early diastole; LA Sa, left atrial global longitudinal strain during late diastole; LA SRs, left atrial global longitudinal strain rate during systole; LA SRe, left atrial global longitudinal strain rate during early diastole; LA SRa, left atrial global longitudinal strain rate during late diastole; GS, global strain; SL, strain longitudinal; SrL, strain rate longitudinal; AVC, aortic valve closure.

diastolic peak flow velocity (A peak), the E/A ratio, and E peak deceleration time (DT) at the mitral orifice. Tissue Doppler imaging (TDI) velocity mode was used to measure the peak early diastolic velocity (*e'*) through placement of the sampling volume in the septal and lateral wall myocardium at the level of the mitral annulus. The average *e'* from these two values was used to calculate the E/*e'* ratio.

LA volume analysis

Apical four-chamber and two-chamber view cines (70–90 fps) of the LA apex were acquired. These cines were imported into EchoPAC version 203 software (GE HealthCare) in stored digital raw data format for analysis. The LA endocardium was manually outlined in each of the two apical dynamic images. The LA maximum volume (LAVmax) was measured at end-systole just before the mitral valve opened, the LA minimum volume (LAVmin) was measured at end-diastole at the apex of the R-wave, and the presystolic LA volume (LAVpre) was measured at the onset of the P-wave (25). The LA total emptying volume (LAVt; LAVt = LAVmax – LAVmin), LA total emptying fraction (LAVtEF; LAVtEF = LAVt/LAVmax), LA passive emptying volume (LAVp; LAVp = LAVmax – LAVpre), LA passive emptying fraction (LAVpEF; LAVpEF = LAVp/LAVmax), conduit volume (CV; CV=SV – LAVt), LA active emptying volume (LAVa; LAVa = LAVpre – LAVmin), and LA active emptying fraction (LAVaEF = LAVa/LAVpre) were calculated based on these three LA volume indicators (25). All calculated LA volume indicators were adjusted for BSA to obtain the LAVI. LAVt and LAVtEF reflect LA reservoir function; LAVp, LAVpEF, and CV reflect LA conduit function; and LAVa and LAVaEF LA booster pump function.

LA strain and strain rate analysis

Apical four-chamber and two-chamber views cines (70–90 fps) of the LA apex were obtained. These images were imported in stored digital raw data format into EchoPAC version 203 software (GE HealthCare) for analysis. The 2D-STE analysis software was used to outline the LA endocardial surface in the dynamic 2D image when it was most distinct. The region of interest was automatically generated by the software, with the width of the region adjusted to include the entire layer of the myocardium. After running the program, the software automatically tracked the myocardial motion in the region of interest frame by frame to generate the strain and strain rate curves of the atrial wall. The global longitudinal strains (GLSs) (LA Ss, LA Se, LA Sa) and strain rates (LA SRs, LA SRe, LA SRa) of the LA wall were recorded during systole, early diastole, and late diastole, respectively, and the specific measurement methods were shown in *Figure 2*.

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Table 1 Clinical and laboratory characteristics of the groups

Variables	CON (n=70)	$CKD_{N\text{-}LVH}$ (n=85)	CKD_{LVH} (n=81)	P
Age (years)	51.11±9.53	53.27±13.21	54.52±14.22	0.252
Gender (male)	33 (47.1)	59 (69.4)	44 (54.3)	0.060
SBP (mmHg)	117.56±9.58	134.08±19.08*	149.46±17.14*#	<0.001
DBP (mmHg)	75.57±8.37	84.14±11.66*	87.28±12.92*	<0.001
BMI (kg/m^2)	23.26±3.41	23.26±3.41	24.19±4.77	0.145
BSA (m^2)	1.70±0.19	1.71±0.19	1.67±0.18	0.369
HR (bpm)	67.94±9.43	74.72±11.19*	71.98±11.26*	0.001
GFR (mL/min/1.73 m^2)	102.20±16.48	29.37±23.14*	15.14±18.04*#	<0.001
SCr (μmol/L)	71.53±15.70	428.19±413.71*	636.09±386.14*#	<0.001
BUN (mmol/L)	4.94±3.12	16.01±10.84*	20.99±11.31*#	<0.001

Continuous data are expressed as the mean ± standard deviation. Categorical data are expressed as number (%). *, P<0.05 indicates significant difference from the control group; #, P<0.05 indicates significant difference from the $CKD_{N\text{-}LVH}$ group. CON, control; CKD, chronic kidney disease; LVH, left ventricular hypertrophy; N-LVH, non-LVH; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; BSA, body surface area; HR, heart rate; GFR, glomerular filtration rate; SCr, serum creatinine; BUN, blood urea nitrogen.

Statistical analysis

The statistical software SPSS 26.0 (IBM. Corp., Armonk, NY, USA) was used for data analysis. Continuous variables are presented as the mean ± standard deviation or as the median with interquartile range. One-way analysis of variance (ANOVA) was employed for comparisons between multiple groups when the data followed a normal distribution and variances were homogeneous. For homogeneous variances, the least significant difference (LSD) *t*-test method was used, while the Kruskal-Wallis test was applied for inhomogeneous variances. The Chi-squared test was used for comparing count data. General linear regression was conducted to examine the relationship between different variables and LA strain and strain rate. Multifactorial linear regression analyzed the relationship between LVMI and LA strain and strain rate. The first multivariate model was adjusted for age, systolic blood pressure (SBP), diastolic blood pressure (DBP), BSA, GFR, and BUN; the second model was adjusted for E peak, A peak, *e*', E/*e*', DT, LV GLS, LVEF, LVEDVI, and LAVI; the third model included all parameters from models I and II. Interobserver agreement was assessed by two independent researchers who randomly selected 20 participants. Intraobserver agreement was evaluated by the same researcher, who analyzed 20 participants twice. The intragroup correlation coefficient (ICC) and the Bland-Altman method were used to test intra- and interobserver agreement for LA strain and strain rate. All statistical tests were two-tailed, with P<0.05 considered statistically significant.

Results

Clinical and laboratory characteristics

The study included 236 participants: 166 patients with CKD (85 in the $CKD_{N\text{-}LVH}$ group and 81 in the CKD_{LVH} group) and 70 healthy controls (CON group). The mean age of the patients with CKD was 53.88±13.68 years, with 103 males and 63 females. The clinical and laboratory characteristics of the groups are detailed in *Table 1*. There were no statistically significant differences in age, gender, BMI, or BSA between the groups (P>0.05). Compared to the CON group, the CKD group exhibited higher SBP and DBP, faster heart rates, lower GFR, and higher SCr and BUN levels (P<0.05).

TTE parameters and LV longitudinal strain

Table 2 summarizes the TTE parameters and LV longitudinal strain. In the CKD_{LVH} group, LVEF, *e*' average TDI, and GLS were lower compared to the other two groups, while LVMI, LVEDVI, E peak, A peak, and E/*e*' were higher. The LA volume in the CKD_{LVH} group was higher than that in the other groups, and each EF value of

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Table 2 Echocardiographic parameters in each group

Variables	CON (n=70)	$CKD_{N\text{-}LVH}$ (n=85)	CKD_{LVH} (n=81)	P
LVEF (%)	68.72±6.05	68.86±5.83	66.43±6.42*#	0.020
LVMI (g/m^2)	76.43±14.58	85.27±15.91*	135.65±30.12*#	<0.001
LVEDVI (mL)	57.80±10.66	58.65±11.81	74.45±16*#	<0.001
E peak (m/s)	0.74±0.18	0.71±0.18	0.81±0.27*#	0.013
A peak (m/s)	0.62±0.16	0.83±0.19*	0.93±0.23*#	<0.001
e' average TDI (cm/s)	10.51±2.19	7.89±2.09*	6.3±2.17*#	<0.001
E/*e*', mean	7.13±1.62	9.45±2.92*	13.56±4.77*#	<0.001
DT (seconds)	192.30±28.23	205.35±40.3	206.28±43.31	0.078
GLS (%)	19.58±2.25	17.42±1.91*	16.7±2.5*#	<0.001
LAVImax (mL)	21.41±5.54	23.53±8.45	36.27±14.3*#	<0.001
LAVIpre (mL)	13.63±4.34	15.37±6.08	25.58±11*#	<0.001
LAVImin (mL)	8.58±3.05	10.03±4.04	17.12±8.42*#	<0.001
LAVIt (mL)	12.83±3.40	13.5±5.35	19.15±7.35*#	<0.001
LAVtEF (%)	60.27±7.80	57.16±7.93	53.68±8.78*#	<0.001
LAVIp (mL)	7.78±2.30	8.15±3.83	10.69±4.94*#	<0.001
LAVpEF (%)	36.58±8.22	34.47±9.35	30.05±8.84*	<0.001
CVI (mL)	26.86±6.99	26.63±7.28	30.02±9.68*#	0.013
LAVIa (mL)	5.05±1.93	5.35±2.71	8.46±4.03*#	<0.001
LAVaEF (%)	37.05±9.63	34.51±8.72	33.76±9.48*	0.079

Continuous data are expressed as the mean ± standard deviation. *, P<0.05 indicates significant difference from CON group; #, P<0.05 indicates significant difference from $CKD_{N\text{-}LVH}$ group. CON, control; CKD, chronic kidney disease; LVH, left ventricular hypertrophy; N-LVH, non-LVH; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index; LVEDVI, left ventricular end-diastolic volume index; E, early diastolic peak flow velocity; A, late diastolic peak flow velocity; *e*', tissue Doppler early diastolic peak velocity; TDI, tissue Doppler imaging; DT, mitral early diastolic E peak deceleration time; GLS, left ventricular global longitudinal strain; LAVI, left atrial volume index; LAVImax, left atrial maximum volume index; LAVIpre, left atrial presystolic volume index; LAVImin, left atrial minimum volume index; LAVIt, left atrial total emptying volume index; LAVtEF, left atrial total emptying fraction; LAVIp, left atrial passive emptying volume index; LAVpEF, left atrial passive emptying fraction; CVI, conduit volume index; LAVIa, left atrial active emptying volume index; LAVaEF, left atrial active emptying fraction.

the LA of the CKD_{LVH} group was lower compared to that of the other groups. LVMI, A peak, and E/*e*' were higher in the CKD_{LVH} group compared to the CON group, while the *e*' average TDI, and GLS were lower compared to the CON group (P<0.05).

LA strain and strain rate analysis

Strain and strain rate were lower in the CKD group compared to the CON group. Specifically, LA Ss, LA Se, LA SRs, LA SRe, and LA SRa were even further reduced in the CKD_{LVH} group as compared to the $CKD_{N\text{-}LVH}$ group (P<0.05). *Figure 3* illustrates the pattern of LA strain and strain rate in three patients from different study groups, with detailed data provided in *Figure 4* and Table S1.

Regression analysis

The univariate correlations of LA strain and strain rate in the study population are presented in *Tables 3*,*4*. LVMI showed a negative correlation with LA Ss, LA Se, LA Sa, LA SRs, LA SRe, and LA SRa. LA strain and strain rate were negatively correlated with age, SBP, A peak, E/*e*', and LAVI and were positively correlated with GFR and *e*'. Multivariate

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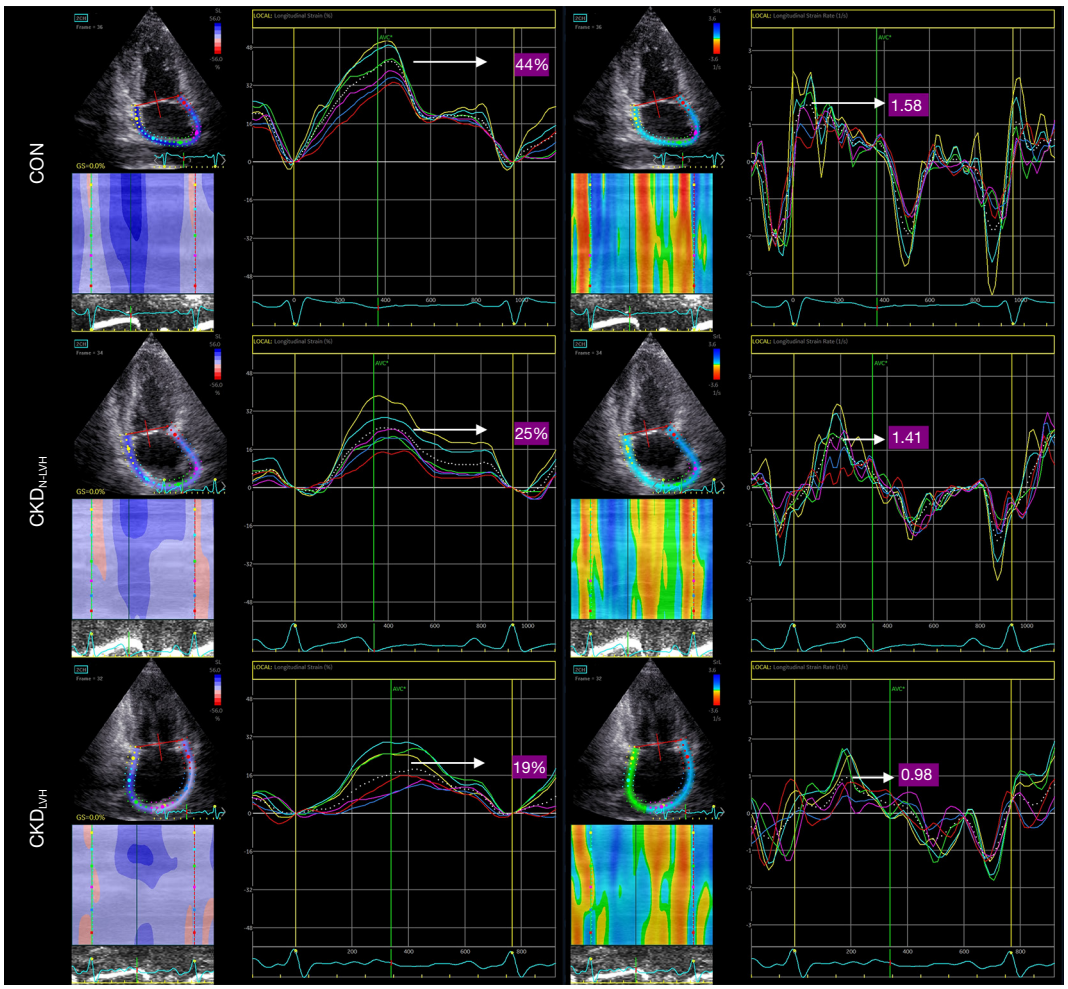

Figure 3 LA strain and strain rate curves obtained from apical two-chamber views from a representative sample of three patients from each study group. AVC, aortic valve closure; LA, left atrial; CKD, chronic kidney disease; LVH, left ventricular hypertrophy; CON, control; N-LVH, non-LVH; SL, strain longitudinal; SrL, strain rate longitudinal.

analysis confirmed that LVMI had an independent negative effect on all LA strain and strain rates (*Table 5*).

Intraobserver and interobserver variability analysis

Intraobserver and interobserver agreement were evaluated for each LA strain and strain rate indicator. The intraobserver ICC values for LA Ss, LA Se, LA Sa, LA SRs, LA SRe, and LA SRa were 0.948 [95% confidence interval (CI): 0.873–0.979], 0.961 (95% CI: 0.905–0.985), 0.902 (95% CI: 0.769–0.960), 0.814 (95% CI: 0.588–0.922), 0.920 (95% CI: 0.809–0.967), and 0.874 (95% CI: 0.709–0.948), respectively (*Figure 5A-5F*). The interobserver ICC values were 0.918 (95% CI: 0.806–0.967), 0.972 (95% CI: 0.932–0.989), 0.833 (95% CI: 0.626–0.931), 0.858 (95% CI: 0.676–0.941), 0.947 (95% CI: 0.871–0.979), and 0.869 (95% CI: 0.699–0.946), respectively (*Figure 5G-5L*). All parameters had intra- and interobserver ICC values above 0.75, indicating good repeatability.

Discussion

Principal findings

This study primarily focused on using LA strain and strain rate techniques to evaluate LA function in patients with CKD. Several key findings emerged. First, conventional ultrasound did not show abnormal LA volume in the CKD$_{N-LVH}$ group compared to the CON group, but strain

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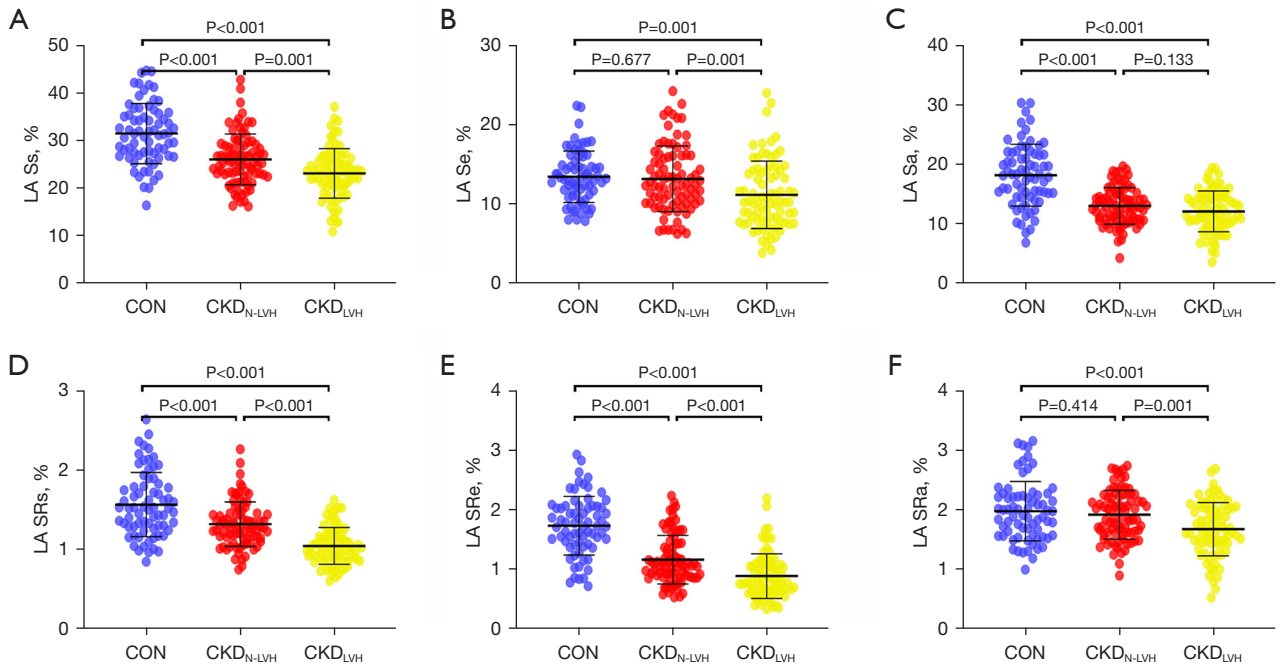

Figure 4 Effect of left ventricular hypertrophy on strain and strain rate. (A) Comparison with LA Ss between the CKD_{LVH}, CKD_{N-LVH}, and CON group. (B) Comparison with LA Se between the CKD_{LVH}, CKD_{N-LVH}, and CON group. (C) Comparison with LA Sa between the CKD_{LVH}, CKD_{N-LVH}, and CON group. (D) Comparison with LA SRs between the CKD_{LVH}, CKD_{N-LVH}, and CON group. (E) Comparison with LA SRe between the CKD_{LVH}, CKD_{N-LVH}, and CON group. (F) Comparison with LA SRa between the CKD_{LVH}, CKD_{N-LVH}, and CON group. CON, control; CKD, chronic kidney disease; LVH, left ventricular hypertrophy; N-LVH, non-LVH; LA Ss, left atrial global longitudinal strain during systole; LA Se, left atrial global longitudinal strain during early diastole; LA Sa, left atrial global longitudinal strain during late diastole; LA SRs, left atrial global longitudinal strain rate during systole; LA SRe, left atrial global longitudinal strain rate during early diastole; LA SRa, left atrial global longitudinal strain rate during late diastole.

and strain rate indices were changed, with reductions in all except LA Se and LA SRa. The LA strain and strain rate were further reduced in the CKD_{LVH} group compared to the CKD_{N-LVH} group. These metrics were negatively correlated with age, SBP, A peak, E/*e*', LAVI, and LVMI but positively correlated with GFR and *e*'. LVMI had an independent negative impact on all LA strain and strain rates (26).

The role of LA volume in CKD

The study found that LA volume was increased in the CKD_{LVH} group compared to the CKD_{N-LVH} group, with no significant difference between the CKD_{N-LVH} group and the CON group. This suggests that LAVI is closely associated with LVH in patients with CKD, as LVH is a common change in uremic cardiomyopathy and leads to elevated LAVI (27). LAVI is also an independent risk factor for cardiovascular events in patients with CKD (28,29). In a study involving 49 patients with CKD (stages 3–5), LAVI was significantly higher in the CKD group relative to LVMI and was a predictor of adverse cardiac events. Moreover, patients with LAVI >32 mL/m^2 have significantly lower event-free survival than those with normal (<28 mL/m^2) or mildly dilated (28–32 mL/m^2) LAVI (30). Another study involving 210 nondialysis patients with CKD (stages 4–5) found that LAVI assessed by TTE was independently associated with new-onset atrial fibrillation (31). Atrial fibrillation is linked to an increased risk of stroke, congestive heart failure, myocardial infarction, CKD progression, and death in patients with CKD (32,33). LAVI, similar to LA and LV strain analyses, is a reliable indicator of myocardial involvement in patients with CKD and is superior to E/*e*'

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Table 3 Univariate correlation analysis of left atrial strain

Variables	LA Ss		LA Se		LA Sa	
	R	P	R	P	R	P
Age	−0.36	<0.001	−0.44	<0.001	−0.12	0.076
SBP	−0.38	<0.001	−0.19	0.004	−0.37	<0.001
DBP	−0.17	0.008	−0.07	0.257	−0.30	<0.001
BMI	−0.12	0.133	−0.11	0.142	−0.05	0.569
BSA	−0.14	0.034	−0.09	0.170	−0.11	0.080
HR	−0.01	0.906	−0.06	0.399	−0.04	0.573
GFR	0.50	<0.001	0.20	0.002	0.52	<0.001
SCr	−0.25	<0.001	−0.09	0.185	−0.27	<0.001
BUN	−0.28	<0.001	−0.12	0.064	−0.28	<0.001
LVEF	0.15	0.019	0.04	0.546	0.18	0.006
LVMI	−0.46	<0.001	−0.24	<0.001	−0.43	<0.001
LVEDVI	−0.25	<0.001	−0.09	0.162	−0.27	<0.001
E peak	0.06	0.399	0.16	0.017	−0.06	0.378
A peak	−0.36	<0.001	−0.28	<0.001	−0.20	0.002
e’	0.58	<0.001	0.44	<0.001	0.43	<0.001
E/*e*’	−0.44	<0.001	−0.28	<0.001	−0.36	<0.001
DT	−0.10	0.136	−0.17	0.012	0.02	0.797
LAVImax	−0.35	<0.001	−0.17	0.011	−0.35	<0.001
LAVIpre	−0.44	<0.001	−0.27	<0.001	−0.38	<0.001
LAVImin	−0.47	<0.001	−0.26	<0.001	−0.43	<0.001

SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; BSA, body surface area; HR, heart rate; GFR, glomerular filtration rate; SCr, serum creatinine; BUN, blood urea nitrogen; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index; LVEDVI, left ventricular end-diastolic volume index; E, early diastolic peak flow velocity; A, late diastolic peak flow velocity; *e*’, tissue Doppler early diastolic peak velocity; DT, mitral valve early diastolic E peak deceleration time; LAVImax, left atrial maximum volume index; LAVIpre, left atrial presystolic volume index; LAVImin, left atrial minimum volume index; LA Ss, left atrial global longitudinal strain during systole; LA Se, left atrial global longitudinal strain during early diastole; LA Sa, left atrial global longitudinal strain during late diastole.

for evaluating LV diastolic function (34).

The role of LA strain and strain rate in CKD

The study found that LA volume in the $CKD_{N\text{-}LVH}$ group did not show significant changes, while patients in the CKD_{LVH} group exhibited a notable increase in LA volume. However, LA strain and strain rate indicators were reduced in the CKD group, with the CKD_{LVH} group showing even greater reductions compared to the $CKD_{N\text{-}LVH}$ group. This suggests that LA strain and strain rates are already abnormal in patients with CKD with normal LA volume, indicating that functional changes in the LA occur before structural changes. In a study of 33 patients with CKD (stage 3), both global systolic strain (GS) and strain rate of the left atrium were reduced in the CKD group, despite the LAVI being similar in the CKD group combined with hypertension (HT) and the HT group. This implies that changes in LA function may precede LA enlargement even in disease states (35). LA GS and strain rate may serve as more sensitive noninvasive tools for detecting cardiovascular involvement in CKD. Thus, using strain and strain rate parameters to evaluate

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Table 4 Univariate correlation analysis of left atrial strain rate

Variables	LA SRs		LA SRe		LA SRa	
	R	P	R	P	R	P
Age	−0.24	<0.001	−0.52	<0.001	−0.10	0.142
SBP	−0.43	<0.001	−0.51	<0.001	−0.14	0.032
DBP	−0.18	0.006	−0.21	0.001	0.00	0.981
BMI	−0.21	0.007	−0.13	0.085	−0.08	0.333
BSA	−0.03	0.686	−0.13	0.043	0.05	0.934
HR	0.20	0.003	−0.07	0.265	0.33	<0.001
GFR	0.50	<0.001	0.64	<0.001	0.20	0.002
SCr	−0.31	<0.001	−0.40	<0.001	−0.11	0.107
BUN	−0.32	<0.001	−0.44	<0.001	−0.13	0.045
LVEF	0.15	<0.018	0.02	0.711	0.23	<0.001
LVMI	−0.55	<0.001	−0.48	<0.001	−0.42	<0.001
LVEDVI	−0.42	<0.001	−0.30	<0.001	−0.34	<0.001
E peak	−0.12	00.065	0.17	0.008	−0.34	<0.001
A peak	−0.34	<0.001	−0.61	<0.001	0.00	0.998
e’	0.54	<0.001	0.82	<0.001	0.13	0.045
E/*e*’	−0.54	<0.001	−0.53	<0.001	−0.39	<0.001
DT	−0.10	0.144	−0.27	<0.001	0.04	0.600
LAVImax	−0.52	<0.001	−0.40	<0.001	−0.43	<0.001
LAVIpre	−0.56	<0.001	−0.49	<0.001	−0.45	<0.001
LAVImin	−0.57	<0.001	−0.45	<0.001	−0.51	<0.001

SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; BSA, body surface area; HR, heart rate; GFR, glomerular filtration rate; SCr, serum creatinine; BUN, blood urea nitrogen; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index; LVEDVI, left ventricular end-diastolic volume index; E, early diastolic peak flow velocity; A, late diastolic peak flow velocity; *e*’, tissue Doppler early diastolic peak velocity; DT, mitral valve early diastolic E peak deceleration time; LAVImax, left atrial maximum volume index; LAVIpre, left atrial presystolic volume index; LAVImin, left atrial minimum volume index; LA SRs, left atrial global longitudinal strain rate during systole; LA SRe, left atrial global longitudinal strain rate during early diastole; LA SRa, left atrial global longitudinal strain rate during late diastole.

Table 5 Multivariate regression analysis of left atrial strain and strain rate

Variables	Model 1		Model 2		Model 3	
	β	P	β	P	β	P
LA Ss						
LVMI	−0.29	<0.001	−0.21	<0.001	−0.20	0.001
Age	−0.34	<0.001	−	−	−0.35	<0.001
SBP	0.02	0.797	−	−	0.08	0.923
A peak	−	−	0.06	0.441	0.20	0.010
E/*e*’	−	−	0.13	0.431	0.24	0.880
LAVI	−	−	−0.14	0.080	−0.13	0.088

Table 5 *(continued)*

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Table 5 *(continued)*

Variables	Model 1		Model 2		Model 3	
	β	P	β	P	β	P
LA Se						
LVMI	−0.20	0.001	−0.08	0.416	−0.16	0.113
Age	−0.40	<0.001	−	−	−0.32	<0.001
SBP	−0.12	0.211	−	−	−0.06	0.548
A peak	−	−	−0.08	0.322	−0.01	0.940
E/*e*’	−	−	−0.26	0.184	−0.14	0.453
LAVI	−	−	−0.08	0.399	−0.08	0.358
LA Sa						
LVMI	−0.25	<0.001	−0.26	<0.001	−0.13	0.040
Age	−0.11	0.079	−	−	−0.21	0.003
SBP	0.13	0.134	−	−	0.06	0.493
A peak	−	−	0.15	0.025	0.29	0.001
E/*e*’	−	−	0.40	0.108	0.16	0.370
LAVI	−	−	−0.13	0.141	−0.11	0.184
LA SRs	−	−	−	−	−	−
LVMI	−0.42	<0.001	−0.20	0.009	−0.20	0.009
Age	−0.15	0.010	−	−	−0.15	0.027
SBP	−0.12	0.167	−	−	−0.05	0.590
A peak	−	−	0.09	0.222	0.16	0.051
E/*e*’	−	−	0.01	0.929	−0.05	0.777
LAVI	−	−	−0.23	0.004	−0.23	0.004
LA SRe						
LVMI	−0.16	<0.001	−0.10	0.108	−0.06	0.311
Age	−0.46	<0.001	−	−	−0.24	<0.001
SBP	−0.08	0.235	−	−	0.05	0.390
A peak	−	−	−0.24	<0.001	−0.14	0.012
E/*e*’	−	−	0.27	0.018	0.18	0.097
LAVI	−	−	−0.15	0.007	−0.14	0.011
LA SRa						
LVMI	−0.46	<0.001	−0.18	0.028	−0.20	0.011
Age	−0.04	0.529	−	−	−0.29	<0.001
SBP	0.05	0.619	−	−	0.01	0.890
A peak	−	−	0.35	<0.001	0.44	<0.001
E/*e*’	−	−	−0.19	0.296	−0.24	0.184
LAVI	−	−	−0.21	0.018	−0.22	0.013

Model 1: adjustment for age, systolic blood pressure (SBP), diastolic blood pressure (DBP), body surface area (BSA), glomerular filtration rate (GFR), and blood urea nitrogen (BUN); Model 2: adjustment for early diastolic peak flow velocity (E peak), late diastolic peak flow velocity (A peak), tissue Doppler early diastolic peak velocity (e’), E/e’, mitral valve early diastolic E peak deceleration time (DT), left ventricular global longitudinal strain (LV GLS), left ventricular ejection fraction (LVEF), left ventricular end-diastolic volume index (LVEDVI), and left atrial volume index(LAVI); Model 3: including all parameters of model 1 and model 2. LA Ss, left atrial global longitudinal strain during systole; LA Se, left atrial global longitudinal strain during early diastole; LA Sa, left atrial global longitudinal strain during late diastole; LA SRs, left atrial global longitudinal strain rate during systole; LA SRe, left atrial global longitudinal strain rate during early diastole; LA SRa, left atrial global longitudinal strain rate during late diastole; LVMI, left ventricular mass index; SBP, systolic blood pressure; E, early diastolic peak flow velocity; A, late diastolic peak flow velocity; *e*’, tissue Doppler early diastolic peak velocity; LAVI, left atrial volume index.

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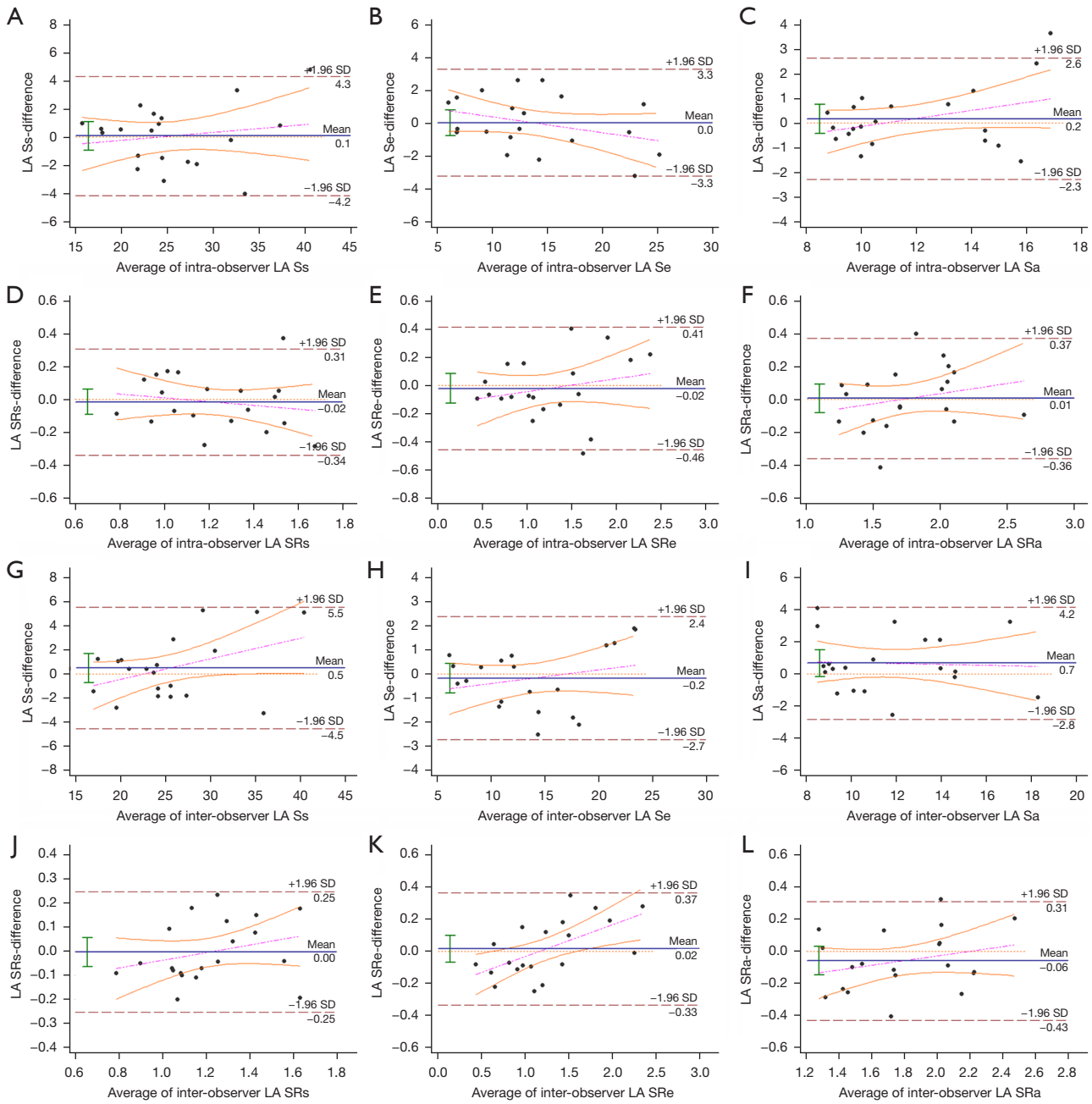

Figure 5 Bland-Altman plots of intraobserver and interobserver consistency. Intraobserver consistency for LA Ss (A), LA Se (B), LA Sa (C), LA SRs (D), LA SRe (E), and LA SRa (F). Interobserver consistency for LA Ss (G), LA Se (H), LA Sa (I), LA SRs (J), LA SRe (K), and LA SRa (L). LA Ss, left atrial global longitudinal strain during systole; LA Se, left atrial global longitudinal strain during early diastole; LA Sa, left atrial global longitudinal strain during late diastole; LA SRs, left atrial global longitudinal strain rate during systole; LA SRe, left atrial global longitudinal strain rate during early diastole; LA SRa, left atrial global longitudinal strain rate during late diastole; SD, standard deviation.

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LA function is feasible, and these parameters have become sensitive indicators for assessing LA function (36-38). LA strain is increasingly being adopted as a surrogate for LV diastolic dysfunction (39).

The LA strain index also predicts poor outcomes in patients with CKD. In a study of 76 patients with CKD (stage 3), LA GS was the most sensitive predictor of CKD presence, outperforming LAVI. Adding LA strain to conventional echocardiographic parameters significantly improved the ability to detect cardiovascular lesions (C statistic value =0.65 *vs.* C-statistic value =0.84; P<0.001). Another study reported that increased LAVI, decreased LV GLS, and presence of CKD were independent predictors of LA strain, while LVMI, E/*e'* ratio, and CKD were predictors of LAVI (16). In a study involving 243 patients with CKD (stages 3–4), reduced LA strain rate was found to be an independent predictor of cardiovascular death and major adverse cardiovascular events (MACEs). Multivariate regression analysis of univariate predictors identified LA strain rate as the only independent predictor of primary and secondary endpoints. Receiver operating characteristic curve analysis indicated that LA strain rate was a stronger predictor of adverse events [area under the curve (AUC) =0.84] compared to the Framingham (AUC =0.58) and atherosclerotic CVD (AUC =0.59) risk scores (40).

Relationship between LVMI and LA function

In our study, LVMI was negatively correlated with LA strain and strain rate. Additionally, LA strain and strain rate were negatively correlated with age, SBP, A peak, E/*e'*, and LAVI and was positively correlated with GFR and *e'*. Multivariate analysis confirmed that LVMI had an independent negative impact on all LA strain and strain rates.

LVH is a risk factor for cardiac disease and all-cause mortality (41,42). It is common in CKD, with its incidence increasing as renal function declines (43). Factors contributing to LVH in patients with CKD include HT, phosphate metabolism disorders, uremic toxins, and chronic inflammation (44-46). LVH involves specific remodeling changes at both the macro- and microlevels, including the activation of apoptotic signaling and metabolic pathways. These changes can increase extracellular matrix production and lead to intercellular fibrosis in cardiomyocytes, resulting in systolic and diastolic dysfunction (3) and the formation of arrhythmogenic substrates (47). LVH is strongly associated with LA volume and function (48,49). Increased LA stiffness (50) and fibrosis (51) in patients with CKD reduce LA contraction function, leading to an enlarged atrial volume.

Study limitations

There are several limitations to this study that should be addressed. First, we employed a prospective design, and the follow-up portion of this study is ongoing and will be completed in the future. However, due to incomplete follow-up data, statistical analysis of a segment of the data has not been conducted. Therefore, the prognostic significance of the findings is unclear and needs to be verified by further studies. Second, participants were only enrolled from a single center and constituted a relatively small sample size. Therefore, a study with a larger sample size to validate our findings is currently underway. Third, strain echocardiographic imaging involves a number of drawbacks, such as intervendor variability, the dependence on the operator's experience, differences in frame rate and loading conditions, and external mechanical factors (52-55). Fourth, the study did not stage CKD, which could have affected the statistical results for patients with different CKD stages. Fifth, EchoPAC does not yet have a speckle-tracking technique specifically designed for LA studies. Consequently, a speckle-tracking technique designed for the LV was used. However, earlier studies have confirmed that it is feasible to measure LA strain using this software (51,56,57).

Conclusions

LA strain and strain rate are valuable indicators for detecting early changes in LA function in patients with CKD. LVMI, which has an independent negative effect on both LA strain and strain rate, is significantly correlated with these measures and may also be a predictor of cardiovascular events.

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://qims.amegroups.com/article/view/10.21037/qims-24-1537/rc

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Provincial Ethics Committee of Shenzhen People's Hospital (No. LL-KY-2021282). All individuals provided written informed consent to participate in this prospective study.

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