Original Article

Frequency and risk factors of impaired left ventricular global longitudinal strain in patients with end-stage renal disease: a two-dimensional speckle-tracking echocardiographic study

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Background: It has been identified that two-dimensional speckle-tracking imaging (2D-STI) enables the early detection of left ventricular (LV) systolic dysfunction. This study's objective was to evaluate the frequency of impaired LV global longitudinal strain (GLS) and investigate the factors in end-stage renal disease (ESRD) patients with preserved LV ejection fraction (LVEF) associated with the impaired GLS.

Methods: A total of 100 ESRD patients with preserved LVEF who underwent transthoracic echocardiography (TTE) were studied. The GLS was calculated as the average of peak longitudinal strain from 18 myocardial segments obtained utilizing the three-standard apical imagings. According to a predefined cutoff, a GLS absolute value of less than 18% was considered subclinical LV systolic dysfunction.

Results: Impaired LV GLS <18% was detected in 58 participants (58/100, 58%). Multivariate analysis exhibited that increased LV mass index was independently associated with impaired GLS <18% [odds ratio (OR): 1.028, 95% confidence interval (CI): 1.004–1.052, P=0.020]. For sequential logistic regression models, model 1, based on parameters included in multivariate logistic regression (χ^2=30.0), was improved by the addition of the LV mass index (χ^2=37.4, P<0.01).

Conclusions: The frequency of impaired LV GLS in ESRD patients with preserved LVEF was relatively high. An increased LVEF was independently associated with impaired LV GLS.

Keywords: End-stage renal disease (ESRD); speckle-tracking imaging (STI); strain

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Introduction

Patients with end-stage renal disease (ESRD) are highly prone to cardiovascular diseases (CVDs) in comparison with the general population (1,2), and more than 60% of ESRD deaths are caused by CVD such as malignant arrhythmia, chronic heart failure, sudden cardiac death, and so on. Left ventricular (LV) dysfunction is one of the leading causes of cardiac death, which is common in patients with ESRD. Therefore, early detection of LV dysfunction in these patients is vitally necessary. However, conventional echocardiography is not sensitive in detecting early deterioration of cardiac function as global LV parameters of the cardiac cavity size and ejection fraction (EF) are generally normal, despite the presence of subtle LV systolic dysfunction (3).

Myocardial strain acquired by two-dimensional speckle-tracking imaging (2D-STI) has emerged in the last decade as a reliable technique for detecting subclinical LV systolic dysfunction, which has allowed advancements in LV quantification beyond LVEF (4). Moreover, global longitudinal systolic strain (GLS), derived from 2D-STI, has been demonstrated as a superior predictor of adverse cardiovascular outcomes and all-cause mortality

with advanced kidney disease compared to LVEF (5-7). Nonetheless, the question has arisen regarding what ESRD patients' factors are related to impaired LV systolic longitudinal strain. Accordingly, this study's objective was to evaluate the factors in ESRD patients with preserved LVEF associated with impaired LV longitudinal systolic function.

Methods

Study population

A total of 112 inpatients and outpatients of our hospital with ESRD were recruited from September 2019 to May 2020. The inclusion criteria were as follows: >18 years old; estimated glomerular filtration rate (eGFR) of ≤15 mL/min/1.73 m^2 by the Modification of Diet in Renal Disease formula (8) or on maintenance hemodialysis, LVEF >50% normal sinus rhythm. There were 12 patients excluded due to coronary artery disease (n=7), severe valve diseases (n=2), and suboptimal image quality (n=3). Therefore, 100 participants were finally included in this study. The Ethics Committee approved the study of the Second Affiliated Hospital of Nanchang University, and individual consent for this retrospective analysis was waived. This study was performed following the World Medical Association Declaration of Helsinki.

Conventional transthoracic echocardiography (TTE)

Comprehensive conventional TTE was performed by well-trained cardiologists in the left lateral decubitus position for optimal image quality, using a commercially available GE Healthcare Vivid E95 equipped with M5S 3.5 mHz transducer (GE Healthcare, Chicago, IL, USA). The dimension of LV at end-diastole (LVEDD) and end-systole (LVESD), interventricular septum thickness in diastole (IVSd), and posterior wall thickness (PWTd) were measured by M-mode echocardiography from the parasternal long-axis view. The LV mass was determined using the Devereux formula and was indexed by body surface area (BSA) for LV mass index (LVMI). Relative wall thickness (RWT) was calculated as the ratio of 2× PWTd/LVEDD. Hypertrophy of the LV (LVH) was defined as LVMI (male) >115 g/m^2 and LVMI (female) >95 g/m^2 (9). The LVEF was determined by utilizing biplane Simpson's method. From the apical 4-chamber view, mitral peak early diastolic velocity (E) and peak late diastolic velocity (A) were assessed by pulsed-wave Doppler. Tissue Doppler assessed the early diastolic peak velocity (e') at the septal mitral annulus site. The ratio (E/e') was calculated as a reliable index of LV filling pressures (10-12). All measurements were completed according to the guidelines published by the American Society of Echocardiography (13).

2D STI

To perform the speckle tracking strain analysis, three standard apical views (4-chamber, 2-chamber, and 3-chamber) were acquired by grayscale images at frame rates of 50–80 frames/sec and were stored in a cine-loop format. The analysis was executed online using GE propriety software (GE, Vingmed Ultrasound, Horten, Norway). Detailed information on the methodological procedure of 2D-STI has been previously described (14). In short, the software automatically determined the endocardial and epicardial boundaries at end-systole with automated function imaging (AFI) for each view, with adjustments performed manually until optimal tracking was achieved. The software automatically divided the LV into six segments in each view for 18 segments. Then, GLS was determined as the average of peak longitudinal strain for all the segments within the three standard apical views. For this study, GLS was represented as an absolute value. Participants were excluded if there was any suboptimal segment. As previously reported, a GLS absolute value of <18% was considered subclinical LV systolic dysfunction in participants with preserved LVEF, according to previous researches (9,12,15).

Statistical analysis

Data were expressed as mean ± standard deviation (SD) and frequencies (%) for continuous variables and categorical variables, respectively. Comparisons of continuous variables in two groups were performed using the Student's *t*-test or Mann-Whitney U test. Categorical variables were compared by χ^2 or Fisher's exact test. Pearson correlation coefficients were used to assess the correlation between two variables. Univariate logistic regression was used to determine possible correlations of reduced LV systolic function (GLS <18%), and parameters with P value <0.1 or clinically relevant ones on univariate analysis were incorporated into the multivariate analysis to determine the independent associations (P<0.05). Multicollinearity between variables was not be allowed in the multivariate analysis. A sequential logistic model for GLS <18% was introduced and tested by

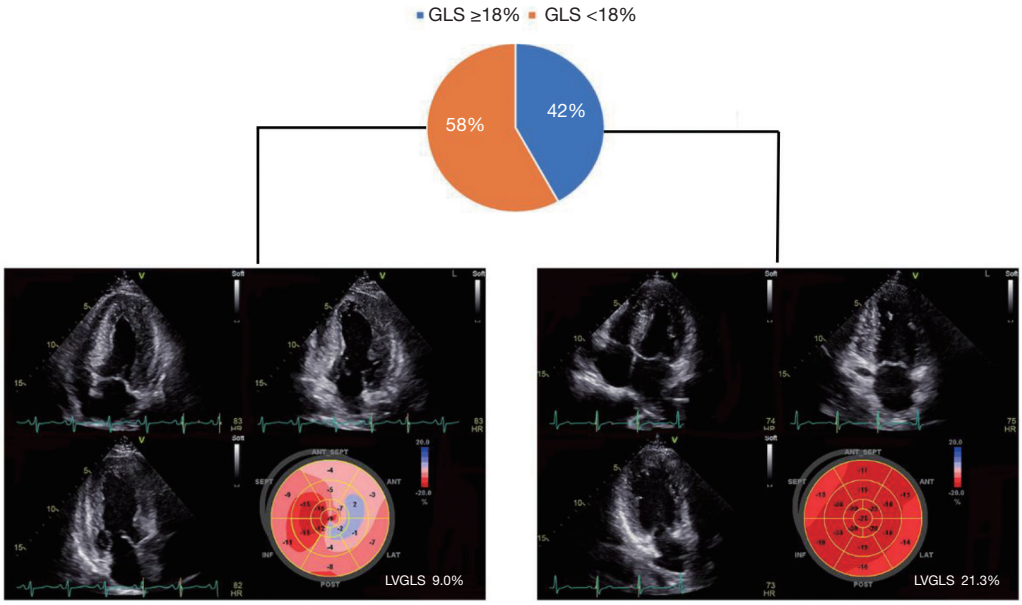

Figure 1 Prevalence of LV systolic dysfunction in terms of LV GLS in ESRD patients with preserved LVEF. Fifty-eight percent of the patients showed LV systolic dysfunction. The patient on the left-side exhibited severely impaired LV GLS, the patient on the right-side showed normal LV GLS. GLS is expressed as absolute values. LV, left ventricular; GLS, global longitudinal strain; ESRD, end-stage renal disease; LVEF, LV ejection fraction.

χ^2 tests to determine the incremental value of independent risk factors. All tests were two-sided, and P value <0.05 was considered to indicate statistical significance. All statistical analyses were processed with the software SPSS version 26.0 (IBM, SPSS Inc., Chicago, IL, USA).

Results

Clinical and laboratory characteristics

Of 100 ESRD participants (aged 52.3±15.1 years, 68% male) with LVEF ≥50%, 42 (42/100, 42%) had LV GLS ≥18% and 58 (58/100, 58%) showed LV GLS <18% (*Figure 1*). The baseline clinical characteristics of the two groups are depicted in *Table 1*. There were no inter-group differences for age, gender, body mass index, or BSA (P>0.05). Participants with LV GLS <18% had higher systolic blood pressure (SBP), diastolic blood pressure (DBP), and lower eGFR compared to those with LV GLS ≥18%. Compared to participants with LV GLS ≥18%, heart rate (HR), serum creatinine, phosphorus, and product of calcium-phosphorus (Ca × P) in those with GLS <18% tended to be higher, but the differences were not statistically significant.

Echocardiographic parameters for patients with GLS <18% and GLS ≥18%

Compared to LV GLS ≥18% participants, conventional parameters in IVSd, PWTd, LVEDD, LVESD, LVEF, RWT, LVMI, and E/e' were significantly aggravated in patients with a GLS <18%. Additionally, the prevalence of LVH in participants with GLS <18% was relatively high. Among the 68 ESRD participants with LVH, 50% (21/42) of those with GLS ≥18% had LVH, and 81% (47/58) of those with GLS <18% showed LVH. Compared to ESRD participants with non-LVH, GLS was significantly decreased in those with LVH (19.0%±2.4% *vs.* 16.4%±3.2%, P<0.001) (*Table 2*).

Association of LV geometry and clinical parameters with GLS

Linear correlation analyses showed that GLS correlated significantly with LVMI (r=–0.576, P<0.001), eGFR (r=0.284, P<0.01), LVEF (r=0.468, P<0.01), LVEDD (r=–0.328, P<0.01), LVESD (r=–0.467, P<0.01), and RWT (r=–0.373, P<0.01) in all participants. With the increase of

Table 1 Baseline characteristics and laboratory variables

Variables	ESRD	GLS ≥18% (n=42)	GLS <18% (n=58)	P value
Clinical characteristics				
Age, year	52±15	55±14	50±16	0.160
Gender	32 [32]	12 [29]	20 [35]	0.532
Body mass index, kg/m^2	21.98±3.46	21.87±3.22	22.06±3.65	0.778
BSA, m^2	1.64±0.16	1.63±0.17	1.65±0.16	0.523
HR, beats/min	78±13	75±12	80±14	0.054
SBP, mmHg	147±26	136±24	155±25	<0.001
DBP, mmHg	87±16	82±15	90±17	0.022
Hemodialysis	35 [35]	15 [36]	20 [35]	0.899
Hypertension	80 [80]	34 [81]	46 [79]	0.839
Diabetes mellitus	24 [24]	8 [19]	16 [28]	0.324
Medications				
ACE inhibitor/ARB	10 [10]	3 [7]	7 [12]	0.418
β-blocker	20 [20]	9 [21]	11 [19]	0.761
α-blocker	8 [8]	5 [12]	3 [5]	0.221
Calcium antagonist	53 [53]	22 [52]	31 [53]	0.916
Diuretic	27 [27]	12 [29]	10 [17]	0.177
Laboratory results				
Serum creatinine, μmol/L	869.06±361.21	800.13±340.07	918.98±370.66	0.105
eGFR, mL/min/1.73 m^2	7.16±4.57	8.51±6.05	6.18±2.77	0.011
Hemoglobin, g/L	94.75±22.49	97.52±23.31	92.74±21.87	0.296
Albumin, g/L	36.79±5.47	36.74±5.83	36.82±5.25	0.937
Calcium, mmol/L	2.14±0.27	2.16±0.25	2.12±0.28	0.518
Phosphate, mmol/L	1.79±0.54	1.66±0.47	1.87±0.57	0.052
Calcium-phosphor, mmol^2/L^2	3.80±1.24	3.55±1.00	3.98±1.37	0.093

Continuous data are presented as mean ± SD. Categorical data are presented as numbers [%]. GLS is expressed as absolute values. ESRD, end-stage renal disease; GLS, global longitudinal strain; BSA, body surface area; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; eGFR, estimated glomerular filtration rate; SD, standard deviation.

LVMI, the absolute values of GLS decreased accordingly (*Figure 2*).

Related factors of reduced GLS for ESRD patients

Univariable and multivariable analyses were performed to determine independent factors associated with impaired GLS (*Table 3*). In univariable analysis, SBP, DBP, eGFR, LVEF, LVEDD, LVESD, IVSd, PWTd, LVMI, and E/e' ratio were associated with GLS <18%. Parameters with P value <0.1 or clinical relevance on univariate analysis were incorporated into the multivariate analysis. To avoid multicollinearity, IVSd, PWTd, and LVEDD were not included in the multivariable analysis. By further multivariate analysis, an increased LVMI was independently associated with GLS <18% [odds ratio (OR): 1.028;

Table 2 Echocardiographic variables in patients with ESRD

Variables	ESRD	GLS ≥18% (n=42)	GLS <18% (n=58)	P value
IVSd, mm	11.50±1.95	10.64±1.45	12.12±2.04	<0.001
PWTd, mm	10.94±1.89	10.05±1.29	11.59±2.00	<0.001
LVEDD, mm	49.19±5.01	47.86±4.91	50.16±4.91	0.023
LVESD, mm	32.21±4.86	30.43±4.38	33.50±4.82	0.002
RWT	0.45±0.08	0.42±0.07	0.46±0.08	0.001
LVMI, g/m^2	129.57±42.59	110.00±24.26	143.75±47.33	<0.001
LVH, n [%]	68 [68]	21 [50]	47 [81]	0.001
LVEF, %	62.29±6.17	64.60±6.15	60.62±5.68	0.001
E/e'	13.21±3.75	11.77±3.26	14.26±3.76	0.001
GLS, %	17.25±3.20	19.97±1.85	15.28±2.42	<0.001

Continuous data are presented as mean ± SD. Categorical data are presented as numbers [%]. GLS is expressed as absolute values. ESRD, end-stage renal disease; GLS, global longitudinal strain; IVSd, interventricular septum thickness in diastole; PWTd, posterior wall thickness in diastole; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; RWT, relative wall thickness; LVMI, left ventricular mass index; LVH, left ventricular hypertrophy; LVEF, left ventricular ejection fraction; E/e', peak early diastolic velocity (by pulsed-wave Doppler)/early diastolic peak velocity (by tissue Doppler); SD, standard deviation.

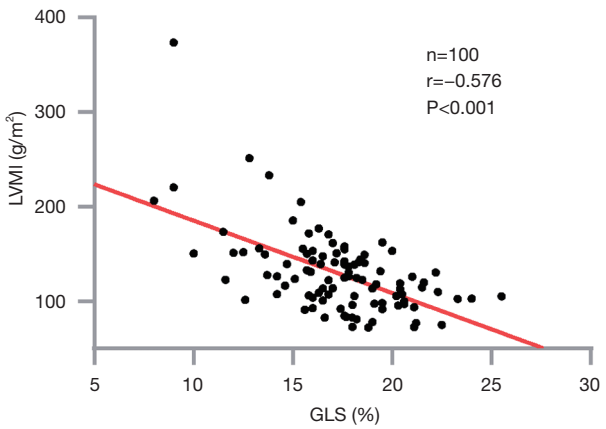

Figure 2 Linear correlation analyses. LV GLS decreased significantly with LVMI in patients. GLS is expressed as absolute values. LV GLS, left ventricular global longitudinal strain; LVMI, LV mass index.

95% confidence interval (CI): 1.004–1.052, P=0.020]. A sequential logistic analysis for predicting GLS <18% was done, starting with multivariate logistic regression (model 1: SBP, DBP, eGFR, LVEF, LVESD, E/e'), and LVMI was added to model 1 via stepwise block analysis to assess its incremental value. Model 1 (χ^2=30.0) was improved by the addition of LVMI (χ^2=37.4, P<0.01), as shown in *Figure 3*.

Discussion

Main findings

The present study's major finding was that the frequency of LV systolic dysfunction in ESRD participants with preserved LVEF was relatively high. Over half (58%) of ESRD participants showed LV systolic dysfunction, defined by LV GLS <18% in normal LVEF. Compared to participants with LV GLS ≥18%, LV geometry was larger or thicker in those with LV GLS <18%. An increased LVMI was independently associated with abnormal GLS, which was consistent with previous studies (6,16,17).

Impaired LV GLS in ESRD patients without overt heart failure

Our study exhibited a high proportion of impaired GLS, with 58% (58/100) incidence among ESRD patients, which was similar to previous reports describing impaired LV GLS in ESRD patients with preserved LVEF. In a study by Huang *et al.* study (7), 42% (80/190) of hemodialysis participants had LV longitudinal systolic dysfunction defined as GLS <16%. Similarly, Rhea *et al.* reported 47% (66/139) of advanced chronic kidney disease (CKD) participants as impaired GLS (determined as GLS <18%) (15).

Table 3 Univariate and multivariate logistic regression analysis for detecting GLS <18% ESRD patients with preserved LVEF

Variables	Univariate		Multivariate	
	OR (95% CI)	P value	OR (95% CI)	P value
eGFR, mL/min/1.73 m^2	0.877 (0.785–0.987)	0.022	0.985 (0.864–1.123)	0.819
SBP, mmHg	1.032 (1.013–1.051)	0.001	1.005 (0.971–1.041)	0.775
DBP, mmHg	1.031 (1.004–1.059)	0.026	1.035 (0.980–1.093)	0.213
HR, beats/min	1.032 (0.999–1.067)	0.059	1.039 (0.993–1.087)	0.096
IVSd, mm	1.739 (1.289–2.346)	<0.001	–	–
PWTd, mm	1.924 (1.392–2.660)	<0.001	–	–
LVEDD, mm	1.103 (1.011–1.204)	0.027	–	–
LVESD, mm	1.158 (1.052–1.276)	0.003	0.909 (0.744–1.110)	0.350
LVMI, g/m^2	1.035 (1.017–1.054)	<0.001	1.028 (1.004–1.052)	0.020
LVEF, %	0.890 (0.825–0.959)	0.002	0.886 (0.767–1.022)	0.096
E/e'	1.239 (1.083–1.417)	0.002	1.160 (0.972–1.384)	0.099

GLS is expressed as absolute values. GLS, global longitudinal strain; ESRD, end-stage renal disease; LVEF, left ventricular ejection fraction; OR, odds ratio; CI, confidence interval; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; IVSd, interventricular septum thickness in diastole; PWTd, posterior wall thickness in diastole; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LVMI, left ventricular mass index; E/e', peak early diastolic velocity (by pulsed-wave Doppler)/early diastolic peak velocity (by tissue Doppler).

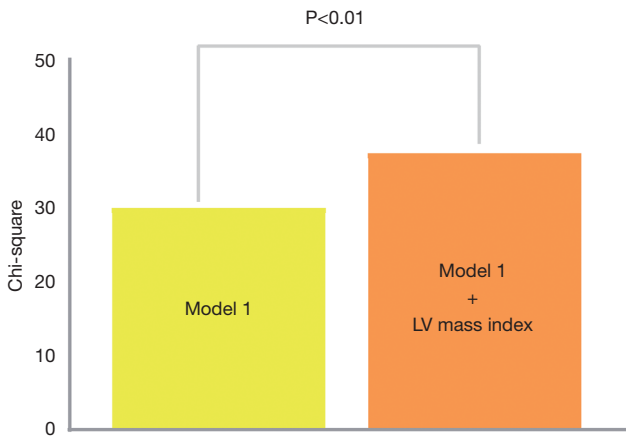

Figure 3 Incremental value by sequential logistic regression models for identifying LV GLS <18%, presented as global χ^2 values. Model 1 (SBP, DBP, eGFR, LVEF, LVESD, E/e') based on significant univariate logistic regression parameters (χ^2=30.0) was improved by the addition of LVMI (χ^2=37.4, P<0.01). GLS is expressed as absolute values. LV GLS, left ventricular global longitudinal strain; SBP, systolic blood pressure; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; E/e', peak early diastolic velocity (by pulsed-wave Doppler)/early diastolic peak velocity (by tissue Doppler); LVEF, LV ejection fraction; LVESD, LV end-systolic diameter; LVMI, LV mass index.

The difference in the incidence of abnormal GLS in CKD patients with retained LVEF may depend on patient characteristics, such as the severity of renal function and dialysis availability. The main pathophysiological explanation may be that LV longitudinal dysfunction reflected early CKD-related myocardial changes such as myocardial ischemia, hypertrophy, and fibrosis because subendocardial longitudinal myocardial fibers were susceptible to reduced coronary perfusion and increased wall stress (18,19). Similarly, GLS was also shown to enable the detection of subclinical myocardial dysfunction of many CVDs, including hypertension and ischemic heart diseases (20).

Related risk factors of LV longitudinal systolic myocardial dysfunction

In our study, increased LVMI was independently associated with abnormal GLS in ESRD participants with preserved LVEF, which was concordant with previous studies (6,16,17). The increase of LVMI suggested varying degrees of LVH, which was a common sequela in ESRD. Wang *et al.* and Nardi *et al.* have reported the prevalence of LVH as 74.5% and 73.7% of ESRD patients, respectively (17,21).

The development of LVH was virtually attributed to the presence of all of these factors (arterial hypertension, volume expansion, calcium-phosphate metabolic abnormalities, arterial stiffness, anemia, and activation of the renin-angiotensin-aldosterone system) (22). As expected, in our analysis of 68 (68%) cases of ESRD with LVH, the prevalence of LVH in patients with GLS <18% was relatively higher than that of GLS ≥18% (50% *vs.* 81%). Compared to ESRD patients with non-LVH, GLS was significantly decreased in patients with LVH, suggesting that reduced GLS was closely associated with LVH. From the pathophysiologic standpoint, the close correlation between decreased GLS in ESRD and larger LVH/LVMI can be explained as follows: pathological myocardial hypertrophy induced the activation of cellular apoptotic and autophagic signals that increased collagen production within the extracellular matrix (ECM), leading to myocardial fibrosis (MF) (23). Increasing levels of MF led to ventricular stiffening, thereby impairing the systolic and diastolic function. In contrast, higher blood pressure (higher afterload) was not an independent factor for detecting GLS <18%, although it frequently existed in ESRD. The reason was most likely that hypertension was not the only driver in reducing GLS in patients with ESRD. In early-stage CKD, higher blood pressure remained the driving force behind the development of LV systolic dysfunction, but its influence diminished as the CKD stage progressed to ESRD (24).

Clinical implications

Our findings indicated that a significant proportion of ESRD patients without overt heart failure have asymptomatic systolic longitudinal myocardial dysfunction. This high proportion of cardiac abnormalities requires close follow-up to detect the early development of heart failure and strictly control cardiovascular risk factors, including rigorous control of blood pressure for more stable and tightly controlled volume load, and early application of drugs to reduce MF [angiotensin-converting enzyme (ACE) inhibitors and β-blockers] to improve ventricular remodeling. Guidelines recommend an echocardiogram for all dialysis patients 1–3 months after renal replacement therapy initiation and every 3 years subsequently (25). Our opinion is that all newly admitted ESRD patients should undergo a comprehensive echocardiographic examination including GLS, and follow-up with serial studies at shorter intervals of 12–18 months should be necessitated as it seems to add prognostic value (26,27). Also, we believe that repeat echocardiography after about 6 months is useful for risk stratification with symptomatic changes, new clinical events, or treatment likely to affect cardiac function (27). It remains to be studied whether shorter repeated echocardiographic monitoring, including GLS, conveys comparable prognostic information.

Limitations

This study had some limitations. First, there were a relatively small number of ESRD participants from a single study center. Second, the study was cross-sectional, making the findings' prognostic significance unclear and requiring further verification. Finally, the study did not include a healthy control group. A more comprehensive analysis may be needed.

Conclusions

Our study confirmed that reduced GLS frequently occurs in patients with ESRD. Moreover, increased LVMI was shown to be significantly associated with abnormal LV longitudinal systolic function.

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Footnote

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at http://dx.doi.org/10.21037/qims-20-1034). The authors have no conflicts of interest to declare.

Ethical Statement: This study was approved by the Ethics Committee of the Second Affiliated Hospital of Nanchang University and individual consent for this retrospective analysis was waived. This study was performed in accordance with the World Medical Association Declaration of Helsinki.

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