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ORIGINAL RESEARCH ARTICLE

Left ventricular global longitudinal strain in patients with heart failure with preserved ejection fraction: outcomes following an acute heart failure hospitalization

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Abstract

Aims While abnormal resting LV GLS has been described in patients with chronic heart failure with preserved ejection fraction (HFpEF), its prognostic significance when measured during an acute heart failure hospitalization remains unclear. We assessed the association between left ventricular global longitudinal strain (LV GLS) and outcomes in patients hospitalized with acute HFpEF.

Methods and results We studied patients discharged alive for acute HFpEF from Duke University Medical Center between 2007 and 2010. Among patients with measurable LV GLS, we performed 2D, speckle-tracking analysis and Cox proportional hazards models assessed the association between continuous LV GLS and outcomes. Baseline characteristics were stratified by normal ($\leq -16\%$) or abnormal ($> -16\%$) LV GLS for comparison. Among 463 patients, the median LV GLS was -12.8% (Interquartile range, -15.8 to -10.8%) and was abnormal in 352 (76%). Overall patients in the cohort were generally elderly, female and had hypertension. After multivariable adjustment, worse outcomes were noted between LV GLS and mortality (HR 1.19 per 1% increase; 95% CI 1.00–1.42; $P = 0.046$) and a composite endpoint of mortality or rehospitalization at 30 days (HR 1.08 per 1% increase; 95% CI 0.99–1.18; $P = 0.08$). There was no association between LV GLS and mortality or a composite of mortality or rehospitalization at 1 year.

Conclusions A high prevalence of patients hospitalized with acute HFpEF have abnormal LV GLS suggesting unrecognized myocardial systolic dysfunction. Furthermore, worse LV GLS is associated with worse clinical outcomes at 30 days but not by1 year.

Keywords Global longitudinal strain; Heart failure with persevered ejection fraction; Echocardiography

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Introduction

Heart failure with preserved ejection fraction (HFpEF) affects approximately 50% of patients with clinical heart failure.[1] Mortality rates after the first hospitalization are as high as 43% and are similar to patients with heart failure with reduced ejection fraction (HFrEF).[1,2] However, unlike HFrEF, proven therapies to reduce mortality and hospitalization rates in HFpEF are lacking.[3] This is in a large part because of the complex and poorly understood pathophysiology of HFpEF.[4] Despite these abnormalities, left ventricular ejection fraction (LVEF) is normal, or preserved, on standard 2D echocardiography as per current taxonomy. Yet, HFpEF patients represent a heterogeneous population that may not be adequately characterized by LVEF[5]; many HFpEF patients may have unrecognized systolic dysfunction[6] and may be better risk stratified by an alternative tool for assessing myocardial contractile

function such as left ventricular (LV) global longitudinal stain (GLS).

Assessment of myocardial deformation using 2D speckle-tracking echocardiography for measurement of GLS has emerged as a more sensitive and objective modality than LVEF to quantify LV contractile performance[7] and may represent a useful tool for the HFpEF population.[4] Studies investigating surrogate markers of HF severity in the chronic, ambulatory HFpEF population found that impaired regional LV strain is associated with worse scores on the Duke Activity Status Index,[8] while abnormal LV GLS correlated with decreased peak oxygen consumption (VO2)[9] and higher levels of natriuretic peptides.[9–11] A recent meta-analysis of studies investigating mortality and hospitalizations in patients with diverse cardiac conditions found that GLS independently predicted mortality better than LVEF in almost 6000 patients with HFrEF, acute myocardial infarction, valvular heart disease and cardiac amyloidosis.[12] In patients with chronic HFpEF, GLS has been shown to be a potential predictor of HF related hospitalizations and cardiovascular (CV) death.[6,13–15] However, these studies have generally been small, restricted to clinical trial enrollees and excluded patients who were acutely hospitalized with HFpEF.

The complex pathophysiology of acute HFpEF coupled with poor stratification tools and lack of available therapies provides the rationale for assessing the utility of LV GLS in HFpEF. In this study, we retrospectively identified patients hospitalized with acute HFpEF who clinically required diuretic treatment. We investigated the association of LV GLS on 30-day and 1-year mortality and rehospitalizations and describe the prevalence and distribution of abnormal LV GLS among this patient population.

Methods

Data source

We assessed adult patients with a HF-related hospital admission between 2007 through 2010 at Duke University Medical Center (DUMC) who had a 2D transthoracic echocardiogram anytime during the hospitalization with a visually estimated and measured biplane EF $\geq$50% and were discharged on a loop diuretic, either torsemide or furosemide. As diuretics are the only specific Class I recommendation for the medical management of HFpEF,[3] this requirement was chosen to better ensure that patients had clinically significant HF. All echocardiograms performed at DUMC since 1995 are prospectively archived in the Duke Echocardiography Lab Database.[16] To exclude disease that can mimic HFpEF, patients were excluded if they had an ICD-9 diagnosis code for primary pulmonary hypertension, severe aortic or mitral stenosis, or a prior aortic or mitral valve repair or replacement. The baseline characteristics of excluded patients did not have any considerable differences compared with the final cohort. Only the first hospitalization for HF during the period was used for each patient. Baseline clinical variables, including laboratory data, medications and billing codes, for each patient were obtained from the searchable, online Duke Enterprise Data Unified Content Explorer research portal.[17] Follow-up data were obtained from patients' medical records and through the Duke Databank of Cardiovascular Disease, an ongoing databank of all patients who undergo a cardiac catheterization at DUMC.[18] Patients with missing outcomes data had mortality determined through a search of the National Death Index.[19] This study was approved by the Duke University Institutional Review Board.

Echocardiographic assessment

For LV GLS analysis, all echocardiograms were transferred in Digital Imaging and Communications in Medicine format from Philips Xcelera (Philips Medical Systems, Eindhoven, The Netherlands) to a vendor independent software package (2D Cardiac Performance Analysis version 4.5, TomTec Imaging Systems, Unterschleissheim, Germany) at a frame rate of 30–50/s. Retrospective speckle-tracking longitudinal strain assessments on 2D images have been validated using TOMTEC software, even when the original study was not intended for this purpose.[20] All analyses were performed by a single experienced operator blinded to other patient characteristics and outcomes. Longitudinal strain assessments for the LV were performed in the apical 4-chamber, 3-chamber and 2-chamber views. For speckle tracking, the endocardial border was manually traced in end systole. The integrity of speckle tracking was visually ascertained. In the case of insufficient tracking, manual correction of the endocardial tracing was attempted and if still unsatisfactory, then the entire study was excluded from the analysis. In the small amount of studies in which patients were actively in atrial fibrillation, the previously validated index beat method was used to obtain longitudinal strain.[21] Longitudinal strain was calculated as the change in length divided by the original length of the speckle pattern over the cardiac cycle and expressed as a percentage; myocardial longitudinal lengthening was represented as positive strain and shortening as negative strain. Global longitudinal strain for the entire LV was averaged from the results of 18 segmental peak systolic strains. Normal LV GLS was defined as $\leq -16\%$, based on previous literature where normal LV GLS ranged from -15.9 to -22.1%.[6,22]

Diastolic dysfunction was analysed per American Society of Echocardiography guidelines[23] and included measurement of early (E) and late (A) diastolic mitral inflow velocities, mitral inflow deceleration times and spectral Doppler tissue

velocities of the septal mitral annulus (e'). Ratios for E/A and E/e' were subsequently calculated; E/A $>$ 0.96 and E/e' $>$ 15 were considered abnormal, per American Society of Echocardiography guidelines. Patients with active atrial fibrillation, poor image quality, E/A fusion or missing Doppler images were excluded from the analysis of diastolic dysfunction.

Outcomes of interest

The primary outcomes for the analysis were all-cause mortality through 30 days and 1 year post-discharge. The secondary outcomes were a composite endpoint of all-cause mortality or all-cause rehospitalization through 30 days and 1 year. Rehospitalization evaluation was limited to the Duke University Health system.

Statistical methods

Patient demographics, medical history, laboratory findings, echocardiography variables and in-hospital therapies were summarized as frequencies and percentages for categorical variables and by medians (25th and 75th percentiles) for continuous variables, and stratified by either normal or abnormal LV GLS. Baseline characteristics were compared using the Wilcoxon rank-sum tests for continuous variables, and Pearson chi-square or exact tests for categorical variables as appropriate.

Unadjusted Kaplan–Meier (KM) curves were generated for the primary and composite endpoints. We generated Cox proportional hazards regression models to assess the association between LV GLS and all-cause mortality and the composite of all-cause mortality or rehospitalization. Unadjusted and adjusted models were used, applying the adjustment covariates of age, sex, blood-urea-nitrogen (BUN) levels, N-terminal pro-brain natriuretic peptide (NT-proBNP) levels, right ventricular (RV) systolic pressure, moderate mitral stenosis, moderate aortic stenosis, E/e' ratios and history of chronic kidney disease (CKD), coronary artery disease (CAD), chronic obstructive pulmonary disease (COPD), hypertension (HTN) and diabetes. Candidate variables were selected for use in the multivariable model based on clinical judgement. Multiple imputation with fully conditional specification methods were used for imputation of missing adjustment covariates. Twenty-five imputations were carried out, and results reflect the combined analyses accounting for uncertainty because of missingness. Hazard ratios (HRs) for 30-day and 1-year outcomes were calculated with corresponding 95% confidence intervals (CIs). The analysis was performed considering a continuous LV GLS measure, and HRs reported per 1% increase in LV GLS. Linearity assumptions were assessed for continuous LV GLS and continuous adjustment covariates; transformations were applied as necessary with no violation associated with LV GLS. Proportional hazards assumptions were assessed for all variables and transformations applied when necessary; no violations were detected for LV GLS. Statistical significance was assessed using 2-sided *P* values. A *P* value $<$0.05 was considered statistically significant. All statistical computations were generated using SAS version 9.2 or higher (SAS Institute Inc., Cary, NC, USA).

Results

We identified 739 unique patients at DUMC from 2007 to 2010 who were hospitalized for acute HFpEF with an LVEF $\geq$50% and discharged on loop diuretics. We could perform LV GLS analysis on 600 patients with 139 excluded because of poor image quality secondary to reduced echogenicity. We excluded another 55 patients who had primary pulmonary hypertension, 64 patients with severe aortic or mitral stenosis or prior aortic or mitral valve repair/replacement, and 18 patients with measured biplane EF $<$50%. Our final cohort included 463 patients. Of these patients, 24% (n = 111) had normal LV GLS, and 76% (n = 352) had impaired LV GLS. *Figure 1* shows the distribution of LV GLS among acute HFpEF patients. *Table 1* presents the baseline characteristics of the patients stratified by normal or impaired LV GLS. Compared with those with normal LV GLS, patients with impaired LV GLS were more likely to be men (41% vs. 29%, *P* = 0.019) and had higher levels of proBNP levels (2497 vs. 1713, *P* = 0.046). There was no difference between the groups in BUN or creatinine levels, frequency

Figure 1 Distribution of left ventricular global longitudinal strain (LV GLS) in patients with acute heart failure with preserved ejection fraction. Mean LV GLS −13.4% (SD 3.8).

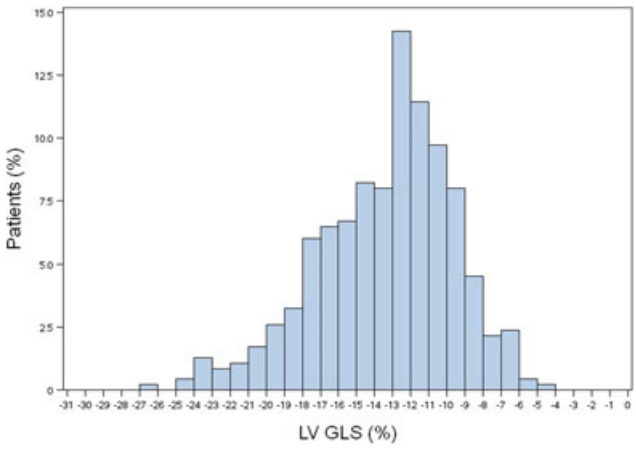

Table 1. Baseline acute HFpEF patient characteristics

	Normal LV GLS *n*=111	Abnormal LV GLS *n*=352	Total *n*=463	*P* value
LV GLS, %	−17.9 (−19.9–−16.9)	−11.9 (−13.5–−10.2)	−12.8 (−15.8–−10.8)	<0.0001
Demographics				
Age, years	68 (61–80)	70 (58–80)	69 (59–80)	0.757
Sex				0.019
Male	32 (28.8%)	145 (41.2%)	177 (38.2%)	
Female	79 (71.2%)	207 (58.8%)	286 (61.8%)	
Race				0.073
White or Caucasian	74 (66.7%)	194 (55.1%)	268 (57.9%)	
Black or African American	29 (26.1%)	133 (37.8%)	162 (35.0%)	
Other	8 (7.2%)	25 (7.1%)	33 (7.1%)	
Medical History				
Hypertension	105 (94.6%)	336 (95.5%)	441 (95.2%)	0.710
Chronic Kidney Disease	61 (55.0%)	222 (63.1%)	283 (61.1%)	0.126
Diabetes	67 (60.4%)	210 (59.7%)	277 (59.8%)	0.895
Coronary Artery Disease	68 (61.3%)	238 (67.6%)	306 (66.1%)	0.218
CABG or PCI	23 (20.7%)	77 (21.9%)	100 (21.6%)	0.797
COPD	25 (22.5%)	58 (16.5%)	83 (17.9%)	0.148
Atrial fibrillation/flutter	53 (47.7%)	192 (54.5%)	245 (52.9%)	0.211
Active Atrial fibrillation/flutter	4/104 (3.9%)	27/348 (7.7%)	31/452 (6.9%)	0.166
Medications during Hospitalization				
Spironolactone or eplerenone	19 (17.1%)	61 (17.3%)	80 (17.3%)	0.959
ACE-inhibitor or ARB	65 (58.6%)	238 (67.6%)	303 (65.4%)	0.080
Beta-blocker	85 (76.6%)	294 (83.5%)	379 (81.9%)	0.098
Clinical Variables				
BUN, mg/dL	26 (18–44)	24 (15–37)	24 (15–39)	0.095
Creatinine, mg/dL	1.30 (0.90–2.00)	1.20 (0.90–1.80)	1.30 (0.90–1.90)	0.694
NT-proBNP, pg/mL [*n*]	1713 (622–5084) [86]	2497 (1125–6480) [256]	2242 (912–6369) [342]	0.046
Systolic blood pressure, mmHg [n]	128 (118–145) [96]	131 (117–151) [285]	130 (117–150) [381]	0.524
Echocardiography Variables				
Mitral Regurgitation				0.388
None/ Trivial/Mild	95/103 (92.2%)	301/337 (89.3%)	396/440 (90.0%)	
Moderate/Severe	8/103 (7.8%)	36/337 (10.7%)	44/440 (10.0%)	
Mitral Stenosis				0.675
None/Trivial/Mild	105/107 (98.1%)	335/340 (98.5%)	440/447 (98.4%)	
Moderate	2/107 (1.9%)	5/340 (1.5%)	7/447 (1.6%)	
Aortic Regurgitation				0.741
None/Trivial/Mild	100/102 (98.0%)	325/336 (96.7%)	425/438 (97.0%)	
Moderate/Severe	2/102 (2.0%)	11/336 (3.3%)	13/438 (3.0%)	
Aortic Stenosis				1.000
None/Trivial/Mild	101/103 (98.1%)	331/337 (98.2%)	432/440 (98.2%)	
Moderate	2/103 (1.9%)	6/337 (1.8%)	8/440 (1.8%)	
E/e' [*n*]	17.8 (12.9–25.0) [93]	18.5 (14.8–26.2) [300]	18.4 (14.2–25.6) [393]	0.270
E/A [*n*]	1.2 (0.9–1.6) [92]	1.3 (0.9–1.8) [254]	1.2 (0.9–1.8) [346]	0.500
RV Size				0.678
Normal	92/111 (82.9%)	287/349 (82.2%)	379/460 (82.4%)	
Small	1/111 (0.9%)	1/349 (0.3%)	2/460 (0.4%)	
Mildly Enlarged	10/111 (9.0%)	39/349 (11.2%)	49/460 (10.7%)	
Moderately Enlarged	7/111 (6.3%)	16/349 (4.6%)	23/460 (5.0%)	
Severely Enlarged	1/111 (0.9%)	6/349 (1.7%)	7/460 (1.5%)	
RV Systolic Pressure [*n*]	40.0 (32.0–51.0) [67]	42.0 (35.0–50.0) [186]	42.0 (34.0–50.0) [253]	0.226

ACE, angiotensin-converting enzyme; ARB, angiotensin II receptor blocker; BUN, blood-urea-nitrogen; CABG, coronary artery bypass graft; COPD, chronic obstructive pulmonary disease; LV GLS, left ventricular global longitudinal strain; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PCI, percutaneous coronary intervention.
Values are expressed as total N (%) or median (25^{th} – 75^{th} percentiles).
All statistics were calculated from total N noted at the top of the column unless otherwise indicated.

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of underlying co-morbid diseases including CKD, HTN, diabetes, CAD or prior revascularization by coronary artery bypass grafting or percutaneous coronary intervention. The measurable E/e' and E/A ratios were elevated with a median of 18.4 (25th –75th percentile, 14.2–25.6) and 1.2 (25th – 75th percentile, 0.9–1.8), respectively. The majority of patients had normal RV size (82%) and contractility (87%) with no differences between groups.

To examine intraobserver reliability, we reassessed LV GLS, biplane ejection fraction (EF), and peak mitral annulus velocity for medial/septal annulus (Em) on a random 10% sampling of the final population. We found correlation values of 0.74 and 0.90 for LV GLS and Em, respectively. For biplane EF, an agreement statistic capturing differences of 5% yielded coverage probability of 0.94.[24]

Table 2 presents the number of events in patients with normal and abnormal LV GLS, and *Table 3* presents the outcomes data from the unadjusted and adjusted Cox regression models, reporting HRs per 1% increase in LV GLS. On adjusted analysis, LV GLS was associated with increased mortality (HR 1.19 per 1% increase; 95% CI 1.00–1.42; $P = 0.046$) and a nominal increase in composite endpoint of mortality or rehospitalization at 30 days (HR 1.08 per 1% increase; 95% CI 0.99–1.18; $P = 0.08$). There was no statistically significant association between LV GLS and mortality (HR 1.02 per 1% increase; 95% CI: 0.96–1.08; $P = 0.56$) or a composite of mortality or rehospitalization (HR 1.03 per 1% increase; 95% CI: 0.98–1.08; $P = 0.20$) at 1 year. *Figure 2* presents the unadjusted KM curves for mortality between patients with normal and abnormal LV GLS. *Figure 3* presents the unadjusted KM curves for the composite outcome of mortality or rehospitalization. *Figures S1* and *S2* present the unadjusted KM curves for mortality and the composite outcome of mortality or rehospitalization, respectively, with patients separated into tertiles based on LV GLS.

Table 2. Primary outcomes total events

Endpoint	Normal LV GLS	Abnormal LV GLS
30-day death	2	14
30-day rehospitalization	6	26
30-day death or rehospitalization	8	40
1-year death	20	77
1-year rehospitalization	10	57
1year death or rehospitalization	30	134

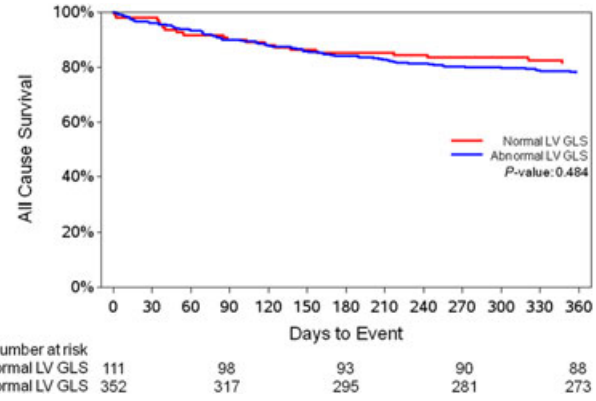
Table 3. Outcomes associated with LV GLS after acute HFpEF hospitalization (per 1% increase in LV GLS)

Endpoint	Hazard Ratio (95% CI)	*P* value	C-statistic
30-day death or rehospitalization			
Unadjusted	1.06 (0.98–1.15)	0.138	0.61
Adjusted[a]	1.08 (0.99–1.18)	0.082	0.72
30 day death			
Unadjusted	1.13 (0.98–1.31)	0.084	0.68
Adjusted[a]	1.19 (1.00–1.42)	0.046	0.88
1-year death or rehospitalization			
Unadjusted	1.03 (0.99–1.08)	0.151	0.55
Adjusted[a]	1.03 (0.98–1.08)	0.204	0.76
1-year death			
Unadjusted	1.02 (0.97–1.08)	0.467	0.56
Adjusted[a]	1.02 (0.96–1.08)	0.562	0.91

CI, confidence interval

[a]Adjustment variables: Year of echo, age, sex, BUN, systolic blood pressure, NT-proBNP, RV systolic pressure, moderate mitral stenosis, moderate aortic stenosis, E/e', and history of CKD, HTN, CAD, COPD, and diabetes

Figure 2 Unadjusted Kaplan–Meier Curves: All-cause survival at 1 year. LV GLS, left ventricular global longitudinal strain.

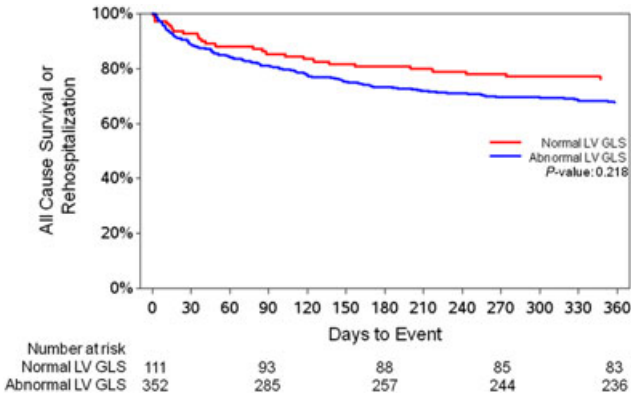


Discussion

In this single-centre cohort study, we found that the majority of patients admitted with acute HFpEF had abnormal LV GLS. In our unadjusted analyses, we observed no statistically significant association between LV GLS and mortality or a composite endpoint of mortality or rehospitalization at 30 days or 1 year. Abnormal LV GLS was associated with significantly worse 30-day mortality rates and nominally higher composite outcomes after adjustment for co-morbid variables; however, there remained no association with clinical outcomes at 1 year post-discharge. To our knowledge, this is the first study to evaluate patients hospitalized with acute HFpEF and describe the association of LV GLS with both 30-day and 1-year clinical outcomes.

After adjustment, we found an association between worse LV GLS and rates of mortality at 30 days post-discharge. It is known that mortality rates following a hospitalization for acute HFpEF are similar to rates for HFrEF,[25] even up to

Figure 3 Unadjusted Kaplan–Meier Curves: All-cause survival or rehospitalization at 1 year. LV GLS, left ventricular global longitudinal strain.



5 years post-discharge.[1,2] Yet, limited data exist on biomarkers, clinical or echocardiographic variables that may predict worse short-term post-discharge outcomes, and the association of HFpEF echocardiographic variables with clinical outcomes have mostly been focused on measurements of diastolic dysfunction among chronic HFpEF patients.[26–29] Thus, the use of LV GLS to identify a subset of acute HFpEF patients with worse short-term outcomes, independent of diastolic dysfunction, may represent a novel tool to identify high-risk patients with unique cardiac pathophysiology for potential interventions prior to discharge.

The relationship between LV GLS was no longer statistically significant at 1 year in contradistinction to previous studies that have found LV GLS to be an important predictor of clinical outcomes such as mortality or hospitalizations in patients with chronic HFpEF.[6,13–15] For instance, recently Shah *et al.*[6] found that abnormal LV longitudinal strain was a predictor of CV death as well as a composite of HF hospitalizations, CV death or aborted cardiac arrest in 447 chronic HFpEF patients enrolled in the TOPCAT trial with a median follow up of 2.6 years. Our study differs from that of Shah *et al.* in several key areas. The patients included in the Shah *et al.* analysis were subject to strict inclusion and exclusion entry criteria for the TOPCAT trial. Patients with severe COPD, uncontrolled HTN, severe renal dysfunction or recent myocardial infarction, coronary artery bypass grafting, stroke or percutaneous coronary intervention were excluded.[30] Comparatively, our patient population was focused on acute HF and more representative of HFpEF patients with substantial co-morbid disease(s) encountered in the general hospital setting. Shah *et al.* noted worse clinical outcomes beyond 2 years; yet, our study did not find a significant association at 1 year post-discharge. There are important differences between acute and chronic HF patients. For instance, chronic HF patients' therapies target neurohormonal regulation, preventing cardiac remodelling and management of co-morbid diseases. While acute HF therapies target decongestion, maintaining adequate cardiac output, preventing kidney insufficiency and reversing inciting causes of decompensation. These differences in haemodynamic and congestive states between acute and chronic patients may have important unrecognized implications regarding the association of abnormal LV GLS on longer-term outcomes. Another potential explanation is that HFpEF is primarily a disease of the elderly[1] and 40–50% of patients with HFpEF die from non-cardiovascular causes.[31] Therefore, with many competing co-morbidities, abnormalities in LV contractility during acute HF, as determined by LV GLS, may play a lesser role in longer-term outcomes.

Although HFpEF patients are more likely to be women, we found a higher proportion of men with abnormal LV GLS among those with acute HFpEF. This finding is in line with contemporary data from Shah *et al.*[6] who also noted a statistically significant increase in male prevalence among chronic HFpEF patients with worse strain. The increased prevalence of men with abnormal GLS and the association of abnormal GLS with worse clinical outcomes provides the rationale for future prospective studies to investigate this unique association, particularly among post-hospitalization outcomes were there remains a paucity of data. There was no difference between the two groups in echocardiographic measurements of diastolic function, such as E/e' and E/A ratios, and overall the ratios were elevated indicating the presence of diastolic dysfunction throughout our population. Our study found higher levels of NT-proBNP among patients with abnormal LV GLS, which are in line with prior data noting significantly higher levels of BNP or NT-proBNP among chronic HFpEF patients with abnormal compared with normal LV GLS.[9–11] Thus, in a population with preserved LVEF, similar echocardiographic measures of diastolic dysfunction and elevated levels of NT-proBNP, LV GLS represents a useful tool to identify myocardial dysfunction independent of diastolic dysfunction that contributes to the complex pathophysiology of acute HFpEF.

There are several limitations to the present study. First, this was a single-centre retrospective analysis, and our data on subsequent hospitalizations were limited to the Duke University Health system. While our statistical models adjusted for variables likely to affect mortality, measured and unmeasured variables may contribute in unknown ways. Only patients who had an echocardiogram during their hospitalization were included, and inherent bias may exist regarding which patients received an echocardiogram. We only included data from the index echocardiogram, thus we only captured each patient at a single snapshot in time, and there were no serial measurements of LV GLS. Furthermore, there remain limitations to the generalizability of any single-centre study utilizing speckle-tracking

echocardiography and strain analysis. While acknowledging the variability of different vendor acquisition and strain analysis platforms, we used a vendor independent approach to mitigate this potential limitation, which relies heavily on obtaining reproducible high-quality images. While echocardiograms were obtained during the acute hospitalization, there is likely heterogeneity among our cohort with respect to the amount of therapy (i.e. diuresis) received prior to evaluation, which may have affected our results, as LV GLS remains susceptible to loading conditions.

In conclusion, among patients hospitalized with acute HFpEF, there is a high prevalence of abnormal LV GLS. We found that LV GLS is associated with worse 30-day but not 1-year post-discharge outcomes. Thus, LV GLS may be a useful tool for identifying a cohort of HFpEF patients with more overt myocardial dysfunction who are at risk for worse outcomes following a hospitalization for HF. Whether therapies targeting myocardial function or energetics would be more or less effective in acute HFpEF based on the presence of abnormal LV GLS requires further study.

Acknowledgement

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Conflict of interest

None declared.

Supporting information

Additional Supporting Information may be found online in the supporting information tab for this article.

Figure S1. Unadjusted Kaplan-Meier Curves: All-cause survival at 1 year.

Figure S2. Unadjusted Kaplan-Meier Curves: All-cause survival or rehospitalization at 1 year.

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