Annals of Cardiology

Open Access

Research Article

Global longitudinal strain as a non-CMR surrogate marker of myocardial fibrosis in hypertrophic cardiomyopathy

Sameer Purra[1]*; Imran Akram Khanday[2]; Gulnar Shah[3]; Waris Shah[4]

[1]Princpal Consultant and Director Cath lab, City Heart Superspecialist Hospital, HP, India.

[2]Head Structural and Pediatric division, City Heart Superspecialist Hospital, HP, India.

[3]Intensivist and Cardiac Anesthesiologist, City Heart Superspecialist Hospital, HP, India.

[4]Medical Officer and Research Analyst, City Heart Super speciality Hospital, HP, India.

Corresponding Author: Sameer Purra

Department of Cardiology, City Heart Superspecialist Hospital, HP, Green Street, Opposite Green Valley School, Elahibagh 190020, Srinagar, India.

Tel: 6005537883; Email: purrasameer79@gmail.com

Introduction

Hypertrophic Cardiomyopathy (HCM) is the most common inherited cardiac disorder and is characterized by unexplained left ventricular hypertrophy with marked phenotypic and clinical heterogeneity [1,2]. While many patients remain asymptomatic for prolonged periods, others are at increased risk of heart failure, atrial fibrillation, ventricular arrhythmias, and sudden cardiac death [2]. Accurate identification of myocardial substrates associated with adverse outcomes is therefore central to effective risk stratification in HCM [2,3].

Myocardial fibrosis represents a key pathological process in HCM and contributes to ventricular remodelling, diastolic dysfunction, microvascular ischemia, and arrhythmogenesis [3,4]. Late gadolinium enhancement (LGE) on Cardiac Magnetic Resonance (CMR) imaging provides a validated non-invasive means of detecting replacement fibrosis and has been consistently linked to ventricular arrhythmias and adverse prognosis, including sudden cardiac death [3-5]. However, routine access to CMR is constrained by cost, limited availability, and contraindications, particularly in resource-limited healthcare systems.

Speckle-tracking echocardiography allows sensitive assessment of myocardial deformation. Left Ventricular Global Longitudinal Strain (LV-GLS) reflects longitudinal shortening predominantly mediated by subendocardial fibres, which are preferentially affected by fibrosis and myocyte disarray in HCM [5,6]. Consequently, GLS impairment may occur early in the disease course and provide incremental prognostic information beyond conventional echocardiographic indices, even in the presence of preserved left ventricular ejection fraction [6,7].

Despite growing evidence supporting the prognostic relevance of GLS, its relationship with myocardial fibrosis assessed by LGE and its association with arrhythmic burden remain incompletely characterized, particularly in populations from resource-constrained regions. The present study was therefore designed to evaluate the relationship between LV-GLS and myocardial fibrosis assessed by LGE on CMR in patients with HCM, and to examine the association of GLS

Abstract

Background: Myocardial fibrosis is a major determinant of adverse outcomes in Hypertrophic Cardiomyopathy (HCM). Late Gadolinium Enhancement (LGE) on cardiac magnetic resonance (CMR) imaging is an established marker of fibrosis, but its routine use is limited in many clinical settings. Left Ventricular Global Longitudinal Strain (LV GLS) derived from speckle tracking echocardiography may serve as an accessible surrogate marker of myocardial fibrosis.

Aim: To evaluate the relationship between LV GLS and myocardial fibrosis assessed by LGE on CMR in patients with HCM, and to examine the association of GLS with arrhythmic burden and clinical outcomes.

Methods: This single centre observational study included 80 patients with HCM who underwent comprehensive clinical evaluation, transthoracic echocardiography with speckle tracking strain analysis, 24 hour Holter monitoring, and CMR with LGE assessment. Impaired GLS was defined as an absolute value < −15%. Multivariate logistic regression and Kaplan-Meier survival analyses were performed.

Results: LGE was present in 46.3% of patients. LV GLS was significantly reduced in patients with LGE compared with those without LGE (-12.1±2.38% vs. -16.4±1.95%; p<0.001). Impaired GLS was observed in 83.8% of LGE positive patients versus 25.6% of LGE negative patients (p<0.001). Non sustained ventricular tachycardia occurred more frequently in the LGE positive group (27.0% vs. 4.7%; p=0.013). On multivariate analysis, LV GLS emerged as the strongest independent predictor of LGE. During a median follow up of 30 months, impaired GLS was associated with significantly lower event free survival.

Conclusion: LV GLS is strongly associated with myocardial fibrosis detected by LGE and correlates with increased arrhythmic burden and adverse clinical outcomes in HCM. GLS represents a reliable, non invasive, and widely available tool that may enhance risk stratification, particularly in settings with limited access to CMR.

Keywords: Hypertrophic cardiomyopathy; Global longitudinal strain; Late gadolinium enhancement; Myocardial fibrosis; Ventricular arrhythmias; Echocardiography.

Article Details

Received: Sep 18, 2026

Accepted: Oct 12, 2026

Published: Oct 19, 2026

Annals of Cardiology - www.anncardiology.org

Purra S et al. © All rights reserved

Citation: Purra S, Khanday IA, Shah G, Shah W. Global longitudinal strain as a non-CMR surrogate marker of myocardial fibrosis in hypertrophic cardiomyopathy. Ann Cardiol. 2026; 3(2): 1026.

impairment with arrhythmic burden and clinical outcomes.

Materials and methods

Study design and population: This was a prospective and retrospective observational study conducted at a tertiary care centre. Consecutive patients with a confirmed diagnosis of Hypertrophic Cardiomyopathy (HCM) were enrolled between 2020 and 2023. The diagnosis of HCM was established based on echo-cardio graphic or Cardiac Magnetic Resonance (CMR) imaging evidence of unexplained left ventricular hypertrophy (maximum wall thickness ≥15 mm in adults), in the absence of other cardiac or systemic causes of hypertrophy.

Both symptomatic and asymptomatic patients, including those diagnosed incidentally or identified through family screening, were included. Patients with congenital heart disease, significant valvular heart disease, chronic kidney disease, pregnancy, contraindications to CMR or gadolinium contrast, prior thoracic irradiation or chemotherapy, prosthetic heart valves, or suboptimal echocardiographic image quality were excluded.

The study was approved by the institutional ethics committee, and written informed consent was obtained from all participants.

Clinical and electrocardiographic assessment: All patients underwent detailed clinical evaluation, including documentation of symptoms, functional status, and a three-generation family history with specific emphasis on hypertrophic cardiomyopathy and sudden cardiac death. Physical examination focused on detection of murmurs and signs of heart failure.

A standard 12-lead electrocardiogram was recorded in all patients and analysed for rhythm abnormalities, axis deviation, voltage criteria for left ventricular hypertrophy, ST–T changes, abnormal Q waves, and atrial abnormalities.

Echocardiography and strain analysis: Transthoracic echocardiography was performed using a GE Vivid E95 system (GE Healthcare, USA). Standard two-dimensional, Doppler, and colour Doppler images were acquired according to current recommendations. Left ventricular dimensions, wall thickness, ejection fraction, diastolic function parameters, presence of systolic anterior motion of the mitral valve, mitral regurgitation, and left ventricular outflow tract gradients were assessed.

Left Ventricular Global Longitudinal Strain (LV-GLS) was measured using two-dimensional speckle-tracking echocardiography with Echo PAC software. GLS was calculated by averaging peak systolic longitudinal strain values obtained from the apical two-, three-, and four-chamber views using a 16-segment left ventricular model. Impaired GLS was defined as an absolute value < −15%, based on prior literature.

Ambulatory rhythm monitoring: All patients underwent 24-hour ambulatory electrocardiographic monitoring to assess for atrial fibrillation, Non-Sustained Ventricular Tachycardia (NSVT), sustained ventricular tachycardia, and other clinically relevant arrhythmias.

Cardiac magnetic resonance imaging: CMR was performed using a 1.5-Tesla scanner (Siemens Avant, Germany). Cine images were acquired using steady-state free precession sequences in standard long- and short-axis planes. Late gadolinium enhancement imaging was performed 15 minutes after intravenous administration of gadolinium-based contrast (0.1 mmol/kg), using phase-sensitive inversion recovery sequences in matching planes.

Presence and distribution of LGE were assessed qualitatively. Left ventricular volumes, ejection fraction, hypertrophy pattern, diastolic function, systolic anterior motion, and left ventricular outflow tract gradients were evaluated.

Follow-up and outcomes: Patients were followed for a median duration of 30 months. Clinical outcomes assessed during follow-up included all-cause mortality, ventricular arrhythmias, appropriate Implantable Cardioverter-Defibrillator (ICD) therapies, and development of heart failure symptoms.

Statistical analysis

Continuous variables are expressed as mean ± standard deviation and categorical variables as frequencies and percentages. Comparisons between groups were performed using the independent Student's t-test for continuous variables and the chi-square test or Fisher's exact test for categorical variables, as appropriate.

Multivariate logistic regression analysis was conducted to identify independent predictors of LGE. Variables included in the model were selected based on clinical relevance and univariate analysis. Event-free survival was assessed using Kaplan–Meier analysis, and comparisons were made using the log-rank test. A p-value <0.05 was considered statistically significant. Statistical analysis was performed using SPSS version 20.0 (IBM Corp., USA).

Results

Study population and baseline characteristics: A total of 80 patients with hypertrophic cardiomyopathy were included in the final analysis. The mean age of the study population was 46.9±12.2 years, with a male predominance (76.3%). Late gadolinium enhancement on cardiac MRI was present in 37 patients (46.3%). Baseline demographic characteristics, clinical presentation, family history, and management strategies are summarized in (Table 1).

There were no statistically significant differences between LGE-positive and LGE-negative groups with respect to age, sex distribution, presenting symptoms, or family history of sudden cardiac death. Most patients were managed medically, predominantly with beta-blockers. Implantable cardioverter-defibrillator implantation was more frequent among LGE-positive patients, although this did not reach statistical significance (43.2% vs. 27.9%, p=0.152).

Electrocardiographic findings: Electrocardiographic abnormalities were common in the study cohort. The most frequent ECG finding was T-wave inversion (55%), followed by left axis deviation (43.7%) and electrocardiographic criteria for left ventricular hypertrophy (38.7%). Abnormal Q waves were observed in 20% of patients. There were no significant differences in ECG findings between patients with and without LGE on CMR (Table 2).

Echocardiographic characteristics: On transthoracic echocardiography, asymmetric septal hypertrophy was the most prevalent morphological pattern, present in 87.5% of patients. Systolic anterior motion of the mitral valve was observed in 42.5%, and left ventricular outflow tract obstruction (gradient >30 mmHg) was documented in 16.2% of patients.

Patients with LGE demonstrated a significantly higher prevalence of asymmetric septal hypertrophy and systolic anterior motion compared with those without LGE (p=0.034 and p=0.017, respectively). Other conventional echocardiographic parameters, including left ventricular dimensions, ejection fraction, and diastolic indices, were comparable between the two groups (Tables 2 and 3).

Global longitudinal strain analysis: The mean LV global longitudinal strain for the entire cohort was -14.4±2.68%. LV-GLS was significantly reduced in patients with LGE compared with those without LGE (-12.1±2.38% vs. -16.4±1.95%; p<0.001) (Table 4).

Overall, 52.5% of patients had impaired GLS (defined as LV-GLS < −15%). Among patients with LGE, 83.8% had reduced GLS, whereas only 25.6% of patients without LGE demonstrated impaired GLS (Figure 3). This highlights a strong association between myocardial fibrosis on CMR and subclinical systolic dysfunction assessed by strain imaging.

Holter monitoring and arrhythmic events: Twenty-four–hour ambulatory ECG monitoring revealed atrial fibrillation in 18.7% of patients and Non-Sustained Ventricular Tachycardia (NSVT) in 15%. Sustained ventricular tachycardia was documented in two patients.

NSVT was significantly more frequent in the LGE-positive group compared with the LGE-negative group (27.0% vs. 4.7%; p=0.013). No significant difference was observed in the prevalence of atrial fibrillation between the two groups (Table 5).

Cardiac magnetic resonance findings: On CMR, asymmetric septal hypertrophy was present in 86.3% of patients, and diastolic dysfunction was identified in 81.3%. Left ventricular ejection fraction was preserved in all patients. Obstructive physiology on CMR was noted in 18.8% of patients, with no significant association with LGE status.

Patients with LGE were more likely to have asymmetric septal hypertrophy and systolic anterior motion on CMR, while diastolic dysfunction was more prevalent in the LGE-negative group (Table 3).

Multivariate analysis: Multivariate logistic regression analysis was performed to identify independent predictors of LGE, including asymmetric septal hypertrophy, LV-GLS, NSVT, systolic anterior motion, and diastolic dysfunction. LV-GLS emerged as the strongest independent predictor of LGE {OR per 1% worsening of GLS = 1.48 (95% CI 1.22–1.79; p<0.001)}. Other variables were not independently associated with LGE (Table 6).

Clinical outcomes and follow-up: During a median follow-up period of 30 months, adverse clinical events were observed predominantly in patients with impaired GLS. Three patients died, six received appropriate ICD shocks, two experienced sustained ventricular tachycardia, and six developed heart failure symptoms.

Kaplan–Meier survival analysis demonstrated significantly lower event-free survival among patients with reduced LV-GLS compared with those with preserved strain (p=0.006) (Figure 3), underscoring the prognostic relevance of GLS in this population.

Table 1: Baseline clinical characteristics of the study population.

Parameter	Total (n=80)	LGE present (n=37)	LGE absent (n=43)	p value
Age, years	46.9±12.2	45.1±13.4	48.4±11.8	0.245
Male sex, n(%)	61(76.3)	29(78.4)	32(74.4)	0.678
Chest pain, n(%)	53(66.3)	24(64.9)	29(67.4)	0.808
Palpitations, n(%)	12(15.0)	5(13.5)	7(16.2)	0.975
Syncope, n(%)	19(23.7)	8(21.6)	11(25.5)	0.678
Dyspnea, n(%)	41(51.2)	21(56.8)	20(46.5)	0.361
Fatigue, n(%)	7(8.8)	2(5.4)	5(11.6)	0.558
Family history of SCD, n(%)	7(8.8)	4(10.8)	3(6.8)	0.081
Family history of HCM, n(%)	10(12.5)	7(18.9)	3(7.0)	0.204
Systolic murmur, n(%)	13(16.2)	6(16.2)	7(16.3)	0.994
Clinical heart failure, n(%)	8(10.0)	4(10.8)	4(9.3)	0.823
ICD implanted, n(%)	28(35.0)	16(43.2)	12(27.9)	0.152
Post alcohol septal ablation, n(%)	6(7.5)	3(8.1)	3(7.0)	1.000

Values are mean±SD or n(%). SCD: Sudden Cardiac Death; HCM: Hypertrophic Cardiomyopathy; ICD: Implantable Cardioverter-Defibrillator.

Table 2: Echocardiographic characteristics.

Parameter	Total (n=80)	LGE present (n=37)	LGE absent (n=43)	p value
Asymmetric septal hypertrophy, n(%)	70(87.5)	36(97.3)	34(79.1)	0.034
Systolic anterior motion, n(%)	34(42.5)	21(56.8)	13(30.2)	0.017
Mitral regurgitation, n(%)	15(18.7)	7(18.9)	8(18.6)	0.971
LVOT gradient >30 mmHg, n(%)	13(16.2)	5(13.5)	8(18.6)	0.538
LV end-diastolic diameter(mm)	42.8±7.2	42.4±6.4	43.1±5.4	0.598
LV end-systolic diameter(mm)	25.9±6.8	25.7±7.4	26.1±5.9	0.788
Interventricular septum(mm)	20.8±5.0	20.1±6.1	21.4±6.6	0.365
Posterior wall thickness(mm)	11.0±3.0	11.4±4.2	10.7±3.9	0.445
LVEF(%)	73.1±8.2	73.5±6.2	72.8±7.5	0.654
E/e' ratio	16.1±6.3	17.1±5.7	15.2±4.9	0.114
BNP(pg/mL)	143.3± 63.5	147.4± 72.3	139.7±69.1	0.628
LV GLS(%)	**−14.4±2.7**	**−12.1±2.4**	**−16.4±2.0**	**<0.001**

LVOT: Left Ventricular Outflow Tract; LVEF: Left Ventricular Ejection Fraction; GLS: Global Longitudinal Strain.

Table 3: Cardiac magnetic resonance findings.

Parameter	Total (n=80)	LGE present (n=37)	LGE absent (n=43)	p value
Asymmetric septal hypertrophy, n(%)	69(86.3)	36(97.3)	33(76.7)	0.007
Systolic anterior motion, n(%)	28(35.0)	18(48.6)	10(23.3)	0.018
Normal LVEF(>50%), n(%)	80(100)	37(100)	43(100)	1.000
Diastolic dysfunction, n(%)	65(81.3)	26(70.3)	39(90.7)	0.041
Mitral valve abnormality, n(%)	8(10.0)	5(13.5)	3(7.0)	0.549
LVOT gradient >30 mmHg, n(%)	15(18.8)	6(16.2)	9(20.1)	0.590

LGE: Late Gadolinium Enhancement; CMR: Cardiac Magnetic Resonance.

Table 4: Comparison based on GLS impairment.

Variable	Impaired GLS (n=40)	Preserved GLS (n=40)	p value
Presence of LGE, n(%)	31(77.5)	6(15.0)	<0.001
Max LV wall thickness (mm)	21.0±4.1	18.3±3.6	0.006
Left atrial diameter (mm)	44.1±5.3	40.2±4.6	0.001

Impaired GLS defined as absolute LV GLS < −15%.

Table 5: Holter monitoring and arrhythmic burden.

Arrhythmia	LGE present (n=37)	LGE absent (n=43)	p value
NSVT, n(%)	10(27.0)	2(4.7)	0.013
Frequent PVCs, n(%)	15(40.5)	9(20.9)	0.048
Sustained VT, n(%)	1(2.7)	1(2.3)	1.000
Atrial fibrillation, n(%)	6(16.2)	3(7.0)	0.190

NSVT: Non-Sustained Ventricular Tachycardia; PVC: Premature Ventricular Complex; VT: Ventricular Tachycardia.

Table 6: Multivariate logistic regression for predictors of LGE.

Variable (per unit increase)	Odds ratio	95% CI	p value
LV GLS (per 1% worsening)	1.48	1.22–1.79	<0.001
Max LV wall thickness (mm)	1.11	1.02–1.21	0.010
Left atrial diameter (mm)	1.07	1.01–1.14	0.020

GLS entered as a continuous variable. CI: Confidence Interval; LGE: Late Gadolinium Enhancement.

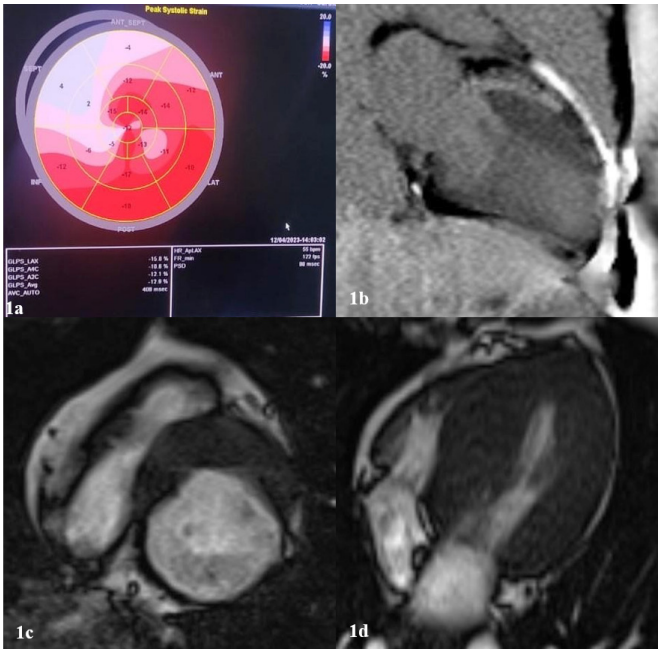


Figure 1: **(a)** Representative speckle-tracking echocardiography image demonstrating reduced global longitudinal strain in a patient with hypertrophic cardiomyopathy.

(b,c,d) Cardiac magnetic resonance images showing late gadolinium enhancement in the interventricular septum.

Showing a patient with HCM. **(a)** Decreased LV GLS most pronounced in septal area. **(b)** LGE Cardiac MRI image showing enhancement in the septum correlating with the decreased LV GLS in that area. **(c)** Cine short axis cardiac MRI image showing septal hypertrophy. **(d)** Cine 2 chamber cardiac MRI image showing hypertrophied LV.

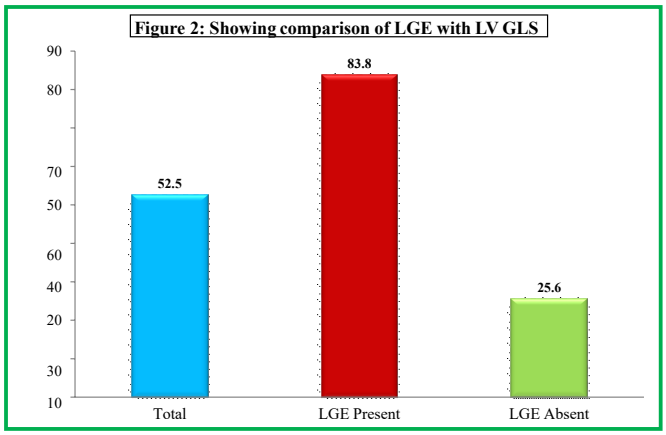


Figure 2: Comparison of LV-GLS values between LGE-positive and LGE-negative groups. Kaplan-Meier curve demonstrating event-free survival stratified by GLS impairment.

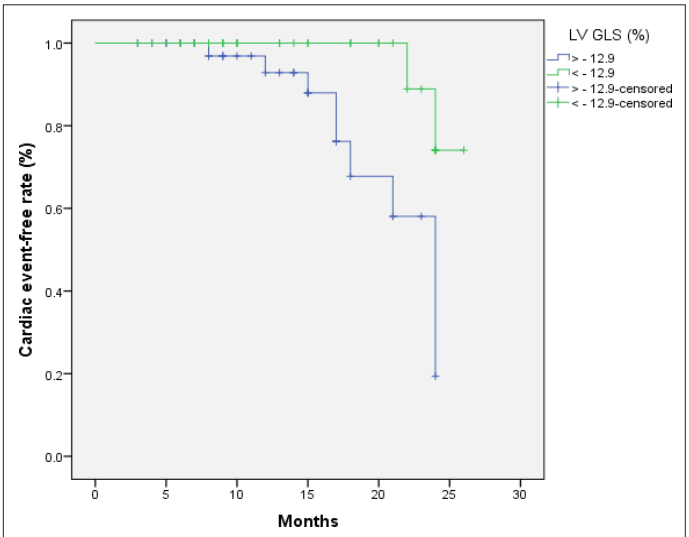


Figure 3: Cardiac event-free rate (%).

Discussion

Principal findings: The present study demonstrates a robust and clinically meaningful association between left Ventricular Global Longitudinal Strain (LV-GLS) and myocardial fibrosis detected by Late Gadolinium Enhancement (LGE) on Cardiac Magnetic Resonance (CMR) imaging in patients with Hypertrophic Cardiomyopathy (HCM). The principal findings are threefold: (1) LV-GLS is significantly impaired in patients with LGE compared with those without fibrosis; (2) impaired GLS is strongly associated with increased ventricular arrhythmic burden, particularly non-sustained ventricular tachycardia; and (3) GLS emerges as an independent predictor of myocardial fibrosis and adverse clinical outcomes. These observations reinforce the concept that myocardial deformation indices provide functional insight into disease severity beyond conventional structural parameters [3-6].

Pathophysiological link between GLS and myocardial fibrosis: Myocardial fibrosis is a hallmark of HCM and plays a central role in ventricular stiffening, impaired relaxation, microvascular ischemia, and electrical instability. Replacement fibrosis detected by LGE has been consistently associated with ventricular arrhythmias and sudden cardiac death, with several large studies and meta-analyses demonstrating a dose-response relationship between LGE burden and adverse outcomes [3,4]. Fibrosis predominantly affects the subendocardial and mid-myocardial layers, regions that are critical for longitudinal shortening. Consequently, LV-GLS-which largely reflects

subendocardial fibre function-serves as a sensitive functional marker of fibrotic myocardial involvement [5,6].

In the present cohort, patients with LGE exhibited markedly impaired GLS despite relatively preserved left ventricular ejection fraction, highlighting the dissociation between conventional systolic indices and true myocardial contractile performance in HCM. These findings are in line with prior reports demonstrating that strain abnormalities often precede overt systolic dysfunction and may represent an early manifestation of adverse myocardial remodelling [5-7].

Comparison with previous studies: Several investigators have explored the relationship between myocardial deformation and fibrosis in HCM. Saito et al. demonstrated that GLS impairment correlates with both the presence and extent of LGE, and that reduced GLS is associated with worse functional capacity [5]. Chan et al. and Weng et al. established LGE as a powerful prognostic marker for sudden cardiac death and ventricular arrhythmias, providing the pathological substrate upon which functional abnormalities such as impaired GLS may develop [3,4]. Our study extends these observations by confirming the strong association between GLS and LGE in a real-world cohort and by emphasizing the potential role of GLS as a practical surrogate marker of fibrosis, particularly in clinical settings where routine CMR is not readily available.

GLS and arrhythmic risk stratification: Ventricular arrhythmias remain a major cause of morbidity and mortality in HCM. Fibrotic tissue creates regions of slow conduction and electrical heterogeneity, facilitating re-entry and malignant ventricular arrhythmias. In our study, non-sustained ventricular tachycardia was significantly more frequent in patients with impaired GLS, supporting the mechanistic link between myocardial deformation abnormalities, fibrosis, and arrhythmogenesis. These findings are consistent with prior studies demonstrating that reduced GLS is associated with ventricular arrhythmias and adverse outcomes, independent of left ventricular ejection fraction [6-8].

Importantly, current risk stratification algorithms for sudden cardiac death rely heavily on clinical and structural parameters, which may not fully capture the functional consequences of myocardial disease. GLS may therefore provide incremental prognostic information and could be particularly useful in patients with intermediate risk profiles, where decision-making regarding implantable cardioverter-defibrillator therapy remains challenging.

Clinical implications: From a clinical perspective, GLS offers several advantages. It is widely available, reproducible, and can be obtained during routine echocardiographic evaluation without additional cost or patient burden. In regions with limited access to CMR, GLS may function as a pragmatic surrogate marker of myocardial fibrosis, enabling early identification of high-risk patients who may benefit from closer surveillance, advanced imaging, or intensified risk-modifying strategies. Furthermore, serial GLS assessment may allow longitudinal monitoring of disease progression and therapeutic response, potentially detecting functional deterioration before overt structural changes occur.

Future directions: Future multicentre studies with larger populations and longer follow-up are warranted to validate the prognostic role of GLS for hard clinical endpoints such as sudden cardiac death and appropriate implantable cardioverter-defibrillator therapies. Integration of quantitative LGE burden with strain parameters may further refine risk prediction models. Additionally, the role of GLS in guiding therapeutic interventions and monitoring treatment response in HCM represents an important area for future research.

Limitations

The single-centre design and modest sample size may limit generalizability. LGE assessment was qualitative, and quantitative fibrosis burden was not measured. Longer-term follow-up is required to establish the prognostic value of GLS for hard endpoints such as sudden cardiac death.

Conclusion

LV-GLS is strongly associated with myocardial fibrosis detected by LGE and correlates with increased arrhythmic burden and adverse clinical outcomes in HCM. GLS represents a reliable, non-invasive, and widely available surrogate marker of myocardial fibrosis that may enhance risk stratification, particularly in settings with limited access to CMR.

Declarations

Ethical approval: The study protocol was reviewed and approved by the Institutional Ethics Committee of City Heart Super Speciality Hospital, HP (Approval No: #43/2021). The study was conducted in accordance with the Declaration of Helsinki.

Consent to participate: Written informed consent was obtained from all participants or their legally authorized representatives prior to enrolment

Patients' consent form: Consent for participation and use of anonymized clinical data for research and publication purposes was obtained from all patients.

Funding: This study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

References

1. Maron BJ, Gardin JM, Flack JM, Gidding SS, Kurosaki TT, et al. Prevalence of hypertrophic cardiomyopathy in a general population of young adults. Circulation. 1995; 92: 785-789.

2. Elliott PM, Anastasakis A, Borger MA, Borggrefe M, Cecchi F, et al. 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy. European Heart Journal. 2014; 35: 2733-2779.

3. Chan RH, Maron BJ, Olivotto I, Pencina MJ, Assenza GE, et al. Prognostic value of quantitative contrast-enhanced cardiovascular magnetic resonance for the evaluation of sudden death risk in hypertrophic cardiomyopathy. Circulation. 2014; 130: 484-495.

4. Weng Z, Yao J, Chan RH, He J, Yang X, et al. Prognostic value of late gadolinium enhancement in hypertrophic cardiomyopathy: A meta-analysis. JACC: Cardiovascular Imaging. 2016; 9: 1392-1402.

5. Saito M, Okayama H, Yoshii T, Hiasa G, Sumimoto T, et al. Clinical significance of global longitudinal strain in patients with hypertrophic cardiomyopathy. Journal of the American Society of Echocardiography. 2012; 25: 587-594.

6. Sengupta PP, Narula J. Cardiac strain as a universal biomarker: Interpretation and clinical utility. JACC: Cardiovascular Imaging. 2014; 7: 534-545.

7. Tower-Rader A, Mohananey D, To A, Lever HM, Popović ZB, et al. Prognostic value of global longitudinal strain in hypertrophic cardiomyopathy: A systematic review. Journal of the American Heart Association. 2019; 8: 011148.

8. Debonnaire P, Thijssen J, Leung M, Joyce E, Katsanos S, et al. Global longitudinal strain predicts clinical outcome in patients with hypertrophic cardiomyopathy. Heart. 2017; 103: 1663-1669.