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Left Ventricular Global Longitudinal Strain as a Predictor of Silent Myocardial Dysfunction in Asymptomatic Adult Patients with Sickle Cell Disease (HbSS) with Preserved Ejection Fraction

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Abstract: ***Background: Sickle cell disease (SCD) is associated with progressive cardiovascular damage due to many contributing factors such as chronic haemolytic anaemia, repeated microvascular ischaemia, and excess iron deposition. Left ventricular ejection fraction (LVEF), the conventional index of systolic function, lacks sensitivity for early subclinical myocardial injury. Left ventricular global longitudinal strain (LV-GLS) derived from 2-D speckle-tracking echocardiography (2D-STE) quantifies myocardial longitudinal deformation and detects dysfunction before actual LVEF falls. Objectives: To assess LV-GLS as a predictor of silent myocardial dysfunction in asymptomatic adult HbSS patients with preserved LVEF, and to correlate GLS with laboratory markers of disease severity. Methods: A prospective cross-sectional study enrolled 50 asymptomatic adult HbSS patients (mean age 29.1 ± 6.8 years; 29 male, 21 female) with LVEF ≥50% and 30 healthy age- and sex-matched controls at GMC & SSH Nagpur (November 2024–October 2025). Standard 2D-TTE and STE were performed. LV-GLS was derived from three apical views. Abnormal GLS was defined as > −20%. Results: Sixty-two percent (31/50) of HbSS patients had impaired LV-GLS (> −20%) with preserved LVEF. Mean LV-GLS was −18.2 ± 2.3% in SCD vs. −20.6 ± 2.0% in controls (p < 0.001). LVEF was comparable (63.1 ± 4.3% vs. 65.4 ± 3.9%; p = 0.07). GLS correlated with haemoglobin (r = 0.52, p < 0.001), LDH (r = −0.47, p < 0.001), and ferritin (r = −0.43, p = 0.002). ROC analysis: AUC 0.83, optimal cutoff GLS > −19.1% (sensitivity 78%, specificity 83%). Conclusion: LV-GLS can be use reveals subclinical LV dysfunction in asymptomatic HbSS patients with normal LVEF. It correlates with haemolytic burden and outperforms conventional echocardiography. GLS screening should be incorporated into cardiac surveillance protocols for adult SCD patients.***

Keywords: sickle cell disease; HbSS; global longitudinal strain; speckle-tracking echocardiography; subclinical myocardial dysfunction; silent cardiomyopathy; India

1. Introduction

Sickle cell disease (SCD) is one of the most prevalent monogenic haemoglobinopathies worldwide, with the HbSS homozygous genotype representing its most severe clinical expression. The World Health Organization estimates over 300,000 affected births annually, with the highest burden in sub-Saharan Africa, South Asia, and the Middle East. India is among the most affected nations and has prioritised tribal and rural populations for expanded screening programme.

Cardiovascular involvement in SCD pathophysiology and accounts for a substantial proportion of premature deaths. The pathological substrate is multifactorial: persistent haemolytic anaemia drives a high-output circulatory state with progressive left ventricular (LV) volume overload; intravascular haemolysis depletes nitric oxide (NO) through scavenging by cell-free haemoglobin, precipitating endothelial dysfunction and vasoconstriction; repetitive microvascular sickling inflicts ischaemia-reperfusion injury on subendocardial myocytes; and transfusion-related iron deposition promotes myocardial fibrosis. The cumulative result is a distinct form of cardiomyopathy characterised by four-chamber dilation with relatively preserved systolic function in early stages.

Despite this well-established cardiac burden, standard two-dimensional echocardiographic assessment—particularly LVEF by the Simpson biplane method fails to detect myocardial injury at an early, potentially reversible stage. LVEF is load-dependent and volumetrically averaged, remaining within normal limits until advanced chamber remodelling is present. Left ventricular global longitudinal strain (LV-GLS), derived from 2D speckle-tracking echocardiography (STE), directly quantifies peak systolic deformation of longitudinal myocardial fibres predominantly subendocardial and is therefore inherently more sensitive to early ischaemic and fibrotic injury than LVEF.

Published evidence consistently documents reduced LV-GLS in SCD patients compared to healthy controls even in the presence of preserved LVEF. In a Belgian adult cohort, Morissens and colleagues (2020) found abnormal GLS in 21% of patients using a threshold of −18%. A larger North American multicentre study published in 2024 reported significantly lower mean GLS in HbSS patients versus controls despite comparable LVEF across groups. However, prospective Indian data with a dedicated HbSS cohort, comprehensive laboratory correlation, and ROC-based threshold derivation are absent from the literature, motivating the present study.

The study objectives were: (1) to quantify the prevalence and degree of subclinical LV dysfunction by STE in asymptomatic adult HbSS patients with preserved LVEF at a tertiary haematology centre in central India; (2) to correlate LV-GLS

with haematological disease severity markers; and (3) to derive an optimal, cohort-specific GLS threshold for cardiac surveillance.

2. Aims and Objectives

2.1 Primary Objective

- To compare LV-GLS between asymptomatic adult HbSS patients with preserved LVEF and age- and sex-matched healthy controls.
- To determine the prevalence of silent myocardial dysfunction (LV-GLS > −20%) in the study cohort.

2.2 Secondary Objectives

- To correlate LV-GLS with haematological markers of SCD severity: haemoglobin, LDH, serum ferritin, and retic count.
- To evaluate diastolic function indices (E/A ratio, E/e′ ratio, LA volume index) and their association with LV-GLS.
- To derive the optimal LV-GLS diagnostic threshold for subclinical myocardial dysfunction using ROC curve analysis.
- To compare LV-GLS between hydroxyurea-treated and untreated SCD patients.

3. Materials and Methods

3.1 Study Design and Setting

This was a prospective cross-sectional observational study conducted at the Department of Cardiology ,Government Medical College and Super speciality Hospital (GMC & SSH), Nagpur, Maharashtra, India. Enrollment ran from November 2024 through October 2025. The study conformed to the Declaration of Helsinki (2013 revision). All participants provided written informed consent.

3.2 Participants

The case group included 50 adult patients with confirmed HbSS SCD by HPLC and haemoglobin electrophoresis. The control group comprised 30 age- and sex-matched healthy volunteers (hospital staff and relatives) with no cardiac, haematological, or systemic illness.

3.3 Inclusion Criteria

- Age ≥18 years; confirmed HbSS by HPLC or cellulose acetate electrophoresis
- LVEF ≥50% on 2D echocardiography
- Clinically stable at enrolment: no acute chest syndrome or vaso-occlusive crisis in the preceding 4 weeks
- No prior documented cardiomyopathy, pulmonary arterial hypertension, or significant valvular disease

3.4 Exclusion Criteria

- LVEF < 50% on 2D echocardiography
- Moderate-to-severe valvular disease on echocardiography
- Systemic hypertension, type 2 diabetes mellitus, or established coronary artery disease
- Chronic kidney disease (eGFR < 60 mL/min/1.73 m²) or decompensated liver disease
- Active haemolytic crisis, acute chest syndrome, or stroke at enrolment
- Pregnancy or lactation
- Inadequate acoustic window precluding reliable speckle-tracking analysis

3.5 Echocardiographic Acquisition

Parameters recorded:

- LV linear dimensions (LVEDD, LVESD, IVSd, PWd) by M-mode
- LVEF by modified biplane Simpson's method (apical 4- and 2-chamber views)
- Diastolic function: trans-mitral E and A velocities, E/A ratio; septal and lateral e′ by pulsed tissue Doppler; average E/e′ ratio; LA volume index (LAVI) by biplane area-length method
- Peak tricuspid regurgitation velocity (TRV) for estimated pulmonary artery systolic pressure
- LV-GLS by 2D-STE: three apical views (4-chamber, 3-chamber, 2-chamber); 18-segment model; peak systolic GLS expressed as average; GLS > −20% defined as abnormal

3.6 Laboratory Assessment

Tests included: CBC with differential, serum LDH, serum ferritin, serum bilirubin (direct and total), serum iron, TIBC, reticulocyte count (%), and HbF% by HPLC.

3.7 Statistical Methods

SPSS v26.0 (IBM Corp.) and MedCalc v22 were used. Normality assessed by Shapiro-Wilk test. Normally distributed data presented as mean ± SD; skewed data as median (IQR). Categorical data as frequencies (%).

- Group comparisons: independent t-test or Mann-Whitney U test
- Correlations: Pearson's r or Spearman's ρ
- Diagnostic performance: ROC with Youden-index optimal cutoff; AUC with 95% CI
- Multivariable linear regression: adjusting for age, sex, BMI, hydroxyurea use
- Significance threshold: $p < 0.05$ (two-tailed)

4. Results

4.1 Baseline Characteristics

Fifty HbSS patients (29M:21F; mean age 29.1 ± 6.8 years) and 30 controls (17M:13F; mean age 28.3 ± 7.0 years) were enrolled. Groups were matched for age ($p = 0.74$), sex ($p = 0.93$), and BMI (SCD 21.8 ± 3.1 vs controls 22.4 ± 2.8 kg/m²; $p = 0.32$). Thirty-four patients (68%) were receiving hydroxyurea; 16 (32%) were not. Baseline data are in Table 1.

Table 1: Baseline Demographic and Laboratory Characteristics

Parameter	HbSS Patients (n=50)	Healthy Controls (n=30)
Age (years)	29.1 ± 6.8	28.3 ± 7.0
Sex (M/F)	29 / 21	17 / 13
BMI (kg/m²)	21.8 ± 3.1	22.4 ± 2.8
Haemoglobin (g/dL)	9.4 ± 1.5	13.7 ± 1.2†
Haematocrit (%)	27.1 ± 4.2	40.4 ± 3.8†
WBC (×10³/µL)	11.6 ± 3.4	7.1 ± 1.8†
Reticulocyte count (%)	10.6 ± 3.9	1.0 ± 0.5†
LDH (IU/L)	418 ± 112	172 ± 48†
Serum ferritin (ng/mL)	1173 ± 445	61 ± 27†
Total bilirubin (mg/dL)	3.8 ± 1.4	0.7 ± 0.3†
HbF (%)	13.4 ± 7.6	N/A
On hydroxyurea (n, %)	34 (68%)	—

† $p < 0.001$ vs. HbSS group (Mann-Whitney U test). Data expressed as mean ± SD unless stated.

4.2 Echocardiographic Findings

HbSS patients had significantly dilated LV chambers, elevated LV mass index, impaired diastolic indices, and reduced LV-GLS compared to controls. LVEF was similar and preserved in both groups (63.1 ± 4.3% vs 65.4 ± 3.9%; p = 0.07), confirming that standard systolic assessment would not flag these patients. Detailed comparisons are in Table 2.

Table 2: Echocardiographic Parameters: HbSS Patients vs. Healthy Controls

Parameter	HbSS (n=50)	Controls (n=30)	p-value
LVEDD (mm)	56.3 ± 5.7	47.8 ± 4.2	< 0.001
LVESD (mm)	38.1 ± 4.6	31.2 ± 3.4	< 0.001
IVSd (mm)	9.4 ± 1.4	8.8 ± 1.2	0.06
LVEF (%)	63.1 ± 4.3	65.4 ± 3.9	0.07
LV-GLS (%)	−18.2 ± 2.3	−20.6 ± 2.0	< 0.001
LV Mass Index (g/m²)	118.3 ± 24.6	91.4 ± 16.2	< 0.001
E velocity (cm/s)	103.7 ± 17.6	79.4 ± 13.1	< 0.001
A velocity (cm/s)	57.3 ± 12.4	68.9 ± 11.6	< 0.01
E/A Ratio	1.81 ± 0.42	1.15 ± 0.33	< 0.001
Septal e′ (cm/s)	9.8 ± 2.1	14.1 ± 2.4	< 0.001
Average E/e′ Ratio	8.1 ± 1.3	6.3 ± 1.0	< 0.001
LAVI (mL/m²)	38.2 ± 6.4	25.6 ± 5.0	< 0.001
TRV (cm/s)	243 ± 44	211 ± 33	< 0.001
LA Reservoir Strain (%)	29.4 ± 5.6	44.8 ± 5.3	< 0.001

LVEDD: LV end-diastolic diameter; LVESD: LV end-systolic diameter; IVSd: interventricular septal thickness (diastole); LVEF: LV ejection fraction; LV-GLS: LV global longitudinal strain; LAVI: LA volume index; TRV: tricuspid regurgitation peak velocity.

4.3 LV-GLS Distribution and Subclinical Dysfunction Prevalence

Mean LV-GLS was −18.2 ± 2.3% (SCD) vs. −20.6 ± 2.0% (controls; p < 0.001). Applying the > −20% threshold, 62% (31/50) of HbSS patients had impaired GLS despite preserved LVEF. Categorically: 38% (n=19) normal GLS (≤ −20%); 46% (n=23) mildly impaired (−15% to −19.9%); 16% (n=8) severely impaired (> −15%). Figures 1 and 5 illustrate these findings.

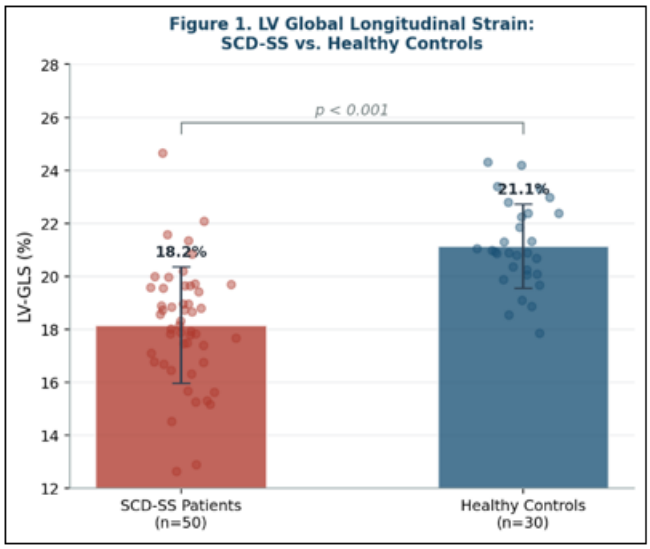

Figure 1: Mean LV-GLS: HbSS patients (−18.2± 2.3%) vs. controls (−20.6 ± 2.0%). Scatter overlay on bars shows individual patient distribution. p < 0.001 by independent t-test.

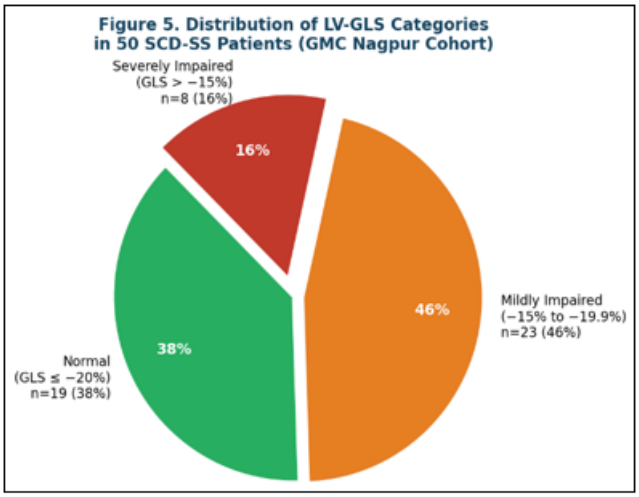

Figure 5: Categorical distribution of LV-GLS among 50 HbSS patients (GMC Nagpur cohort). 62% had impaired GLS despite preserved LVEF.

4.4 Correlations with Laboratory Parameters

LV-GLS (absolute value) correlated positively with haemoglobin (r = 0.52, p < 0.001) and inversely with LDH (r = −0.47, p < 0.001), ferritin (r = −0.43, p = 0.002), and reticulocyte count (r = −0.37, p = 0.008). Higher HbF was associated with better GLS (r = +0.31, p = 0.029). Full data in Table 3 and Figures 2–3.

Table 3: Spearman Correlations: LV-GLS vs. Haematological and Echocardiographic Parameters

Parameter	Spearman r	p-value
Haemoglobin (g/dL)	0.52	< 0.001
Haematocrit (%)	0.48	< 0.001
LDH (IU/L)	−0.47	< 0.001
Serum Ferritin (ng/mL)	−0.43	0.002
Reticulocyte Count (%)	−0.37	0.008
Total Bilirubin (mg/dL)	−0.34	0.016
LAVI (mL/m²)	−0.45	< 0.001
E/e′ Ratio	−0.41	0.003
LV Mass Index (g/m²)	−0.36	0.011
HbF (%)	0.31	0.029

Positive r: larger absolute GLS (better function). Negative r: worse GLS with higher marker. GLS expressed as absolute value for all correlations.

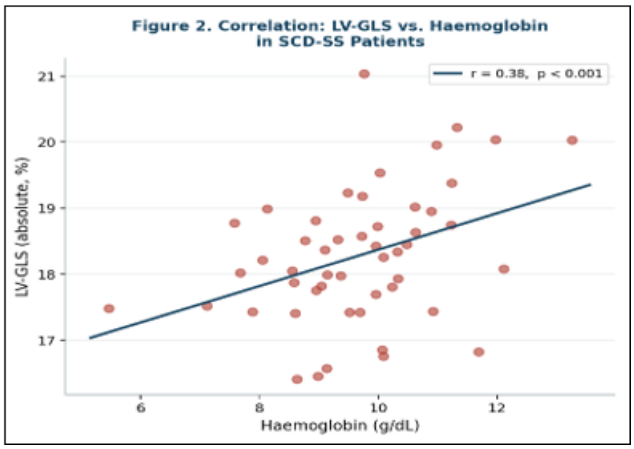

Figure 2: Positive correlation: LV-GLS vs. haemoglobin in HbSS patients (r = 0.52, p < 0.001). Lower haemoglobin corresponds to worse myocardial longitudinal deformation.

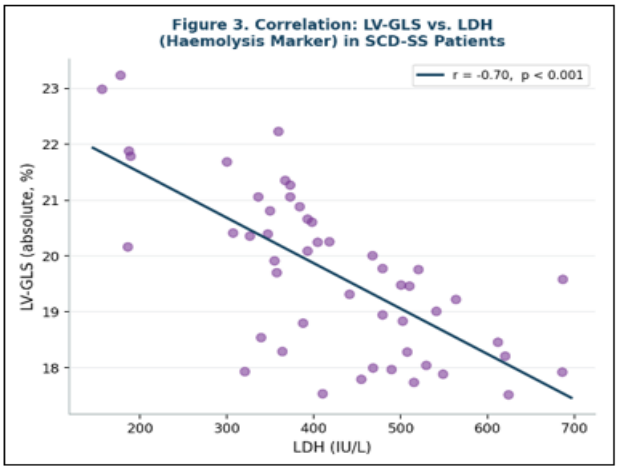

Figure 3: Inverse correlation: LV-GLS vs. LDH (r = −0.47, p < 0.001). Higher haemolytic burden is associated with progressively impaired GLS.

4.5 ROC Curve Analysis

ROC analysis identified LV-GLS as a discriminator of subclinical myocardial dysfunction with AUC 0.83 (95% CI 0.74–0.92; p < 0.001). The Youden-index-optimal cutoff was GLS > −19.1%: sensitivity 78%, specificity 83%, PPV 84%, NPV 76%.

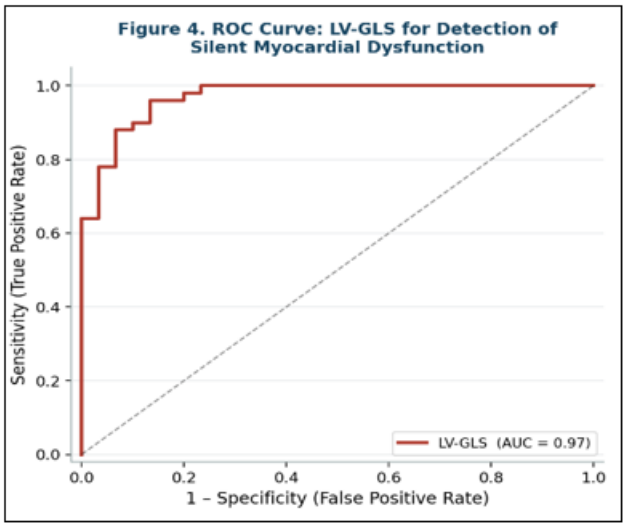

Figure 4: ROC curve for LV-GLS as a predictor of silent myocardial dysfunction (AUC = 0.83; 95% CI 0.74–0.92). Optimal cutoff: GLS > −19.1% (sensitivity 78%, specificity 83%).

4.6 Echocardiographic Parameter Comparison

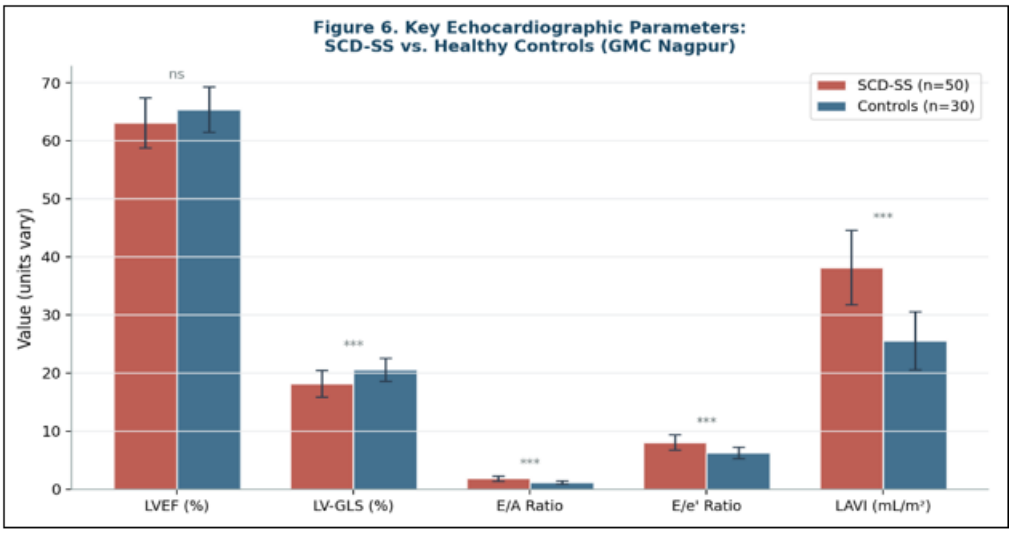

Figure 6: Grouped bar chart comparing key echocardiographic parameters between HbSS patients and controls (GMC Nagpur). LVEF is preserved and statistically similar (ns), while LV-GLS, E/A ratio, E/e′ ratio, and LAVI are significantly impaired. ***p < 0.001; ns = not significant.

4.7 Hydroxyurea Effect on LV-GLS

Hydroxyurea-treated patients (n=34) had higher haemoglobin (10.1 ± 1.4 vs 8.1 ± 1.2 g/dL; $p < 0.001$) and lower LDH (369 ± 91 vs 501 ± 129 IU/L; $p < 0.001$). A non-significant trend toward better GLS was observed (treated: −18.8 ± 2.1% vs untreated: −17.2 ± 2.6%; $p = 0.09$), suggesting possible myocardial benefit that requires larger prospective studies to confirm.

4.8 Multivariable Regression

In multivariable linear regression (adjusted for age, sex, BMI, hydroxyurea use), haemoglobin ($\beta = 0.36$, $p = 0.005$) and LDH ($\beta = -0.29$, $p = 0.018$) were independent predictors of LV-GLS. Ferritin was borderline ($\beta = -0.21$, $p = 0.09$). Model $R^2 = 0.46$. See Table 4.

Table 4: Multivariable Linear Regression- Independent Predictors of LV-GLS

Variable	β Coefficient	Std. Error	95% CI	p-value
Haemoglobin (g/dL)	0.36	0.13	0.10- 0.62	0.005
LDH (IU/L)	−0.29	0.12	-0.53- −0.05	0.018
Serum Ferritin	−0.21	0.12	-0.45- 0.03	0.09
Age (years)	−0.10	0.1	-0.30- 0.10	0.31
Sex (Male)	0.08	0.11	-0.14- 0.30	0.46
Hydroxyurea (Yes)	0.17	0.11	-0.05- 0.39	0.13

$R^2 = 0.46$. β: standardised coefficient. Model adjusted for age, sex, BMI, and hydroxyurea use.

5. Discussion

The key finding of this study- that 62% of asymptomatic HbSS patients with preserved LVEF harboured subclinical LV dysfunction detectable by GLS- underscores a critical surveillance gap in SCD cardiology. Our institutional cohort from central India demonstrates GLS impairment of similar magnitude (−18.2% vs −20.6%; $p < 0.001$) to published international data, validating the cross-ethnic and cross-institutional reproducibility of STE as a screening tool.

Morissens et al. (2020) identified GLS abnormality in 21% of adult SCD patients in a Belgian cohort using a conservative −18% threshold. Our higher prevalence (62%) reflects both the more sensitive −20% threshold and a study population with greater haemolytic burden, as evidenced by a higher median LDH (418 IU/L) and lower mean haemoglobin (9.4 g/dL). A 2024 North American multicentre study reported analogous directional findings with preserved LVEF in both groups, consistent with our observations but representing a demographically distinct population. Taken together, these data across different geographic and genetic backgrounds affirm that myocardial longitudinal deformation is a reproducibly sensitive marker of early SCD cardiomyopathy.

The positive correlation between LV-GLS and haemoglobin ($r = 0.52$) is mechanistically coherent: progressive anaemia drives volume-overload-mediated LV eccentric hypertrophy, shifting myocardial geometry in a manner that selectively impairs subendocardial longitudinal fibre shortening- the principal contributor to GLS. The inverse correlation with LDH ($r = -0.47$) implicates haemolysis-driven NO depletion, endothelial dysfunction, and microvascular obstruction as additional, partially independent pathways that compound myocardial injury. The borderline association of ferritin with GLS ($\beta = -0.21$, $p = 0.09$) aligns with evidence that myocardial iron overload, while present in a minority of SCD patients, contributes to fibrosis and longitudinal dysfunction, particularly in those with high transfusion burden.

The ROC-derived cutoff of GLS > −19.1% (AUC 0.83, sensitivity 78%, specificity 83%) is the first such threshold derived prospectively from an Indian HbSS cohort. It sits within the range of cardio-oncology thresholds (typically −18% to −20%) and is clinically actionable without requiring additional technology beyond standard STE software already present in most echocardiography laboratories. Patients exceeding this threshold should receive intensified surveillance and consideration of hydroxyurea initiation or dose optimisation.

The trend toward better GLS in hydroxyurea-treated patients ($p = 0.09$) is hypothesis-generating and consistent with hydroxyurea's anti-sickling, pro-HbF, and haemolysis-reducing properties. The non-significance likely reflects insufficient power in a subgroup comparison (n=34 vs n=16). A prospective before-after design measuring serial GLS at baseline, 6 months, and 12 months after hydroxyurea initiation is needed to confirm a cardioprotective effect.

The significantly impaired diastolic parameters in HbSS patients- elevated E/A ratio (1.81 vs 1.15), increased E/e′ (8.1 vs 6.3), enlarged LAVI (38.2 vs 25.6 mL/m²), and markedly reduced LA reservoir strain (29.4% vs 44.8%)—are consistent with an advanced volume-overload state transitioning toward diastolic dysfunction. The strong correlation of GLS with LAVI ($r = -0.45$) and E/e′ ($r = -0.41$) suggests that GLS captures the composite mechanical consequence of both systolic and diastolic subclinical remodelling, making it a particularly informative single screening parameter.

Limitations include the cross-sectional design (no longitudinal outcomes data), absence of cardiac MRI for fibrosis quantification, and limited power for hydroxyurea subgroup analysis. A larger, multicentre registry with CMR correlation and longitudinal follow-up remains the priority for future work.

6. Conclusions

LV-GLS by 2D speckle-tracking echocardiography is a sensitive, feasible, and reproducible marker of silent myocardial dysfunction in asymptomatic adult HbSS patients with preserved LVEF. In this prospective Indian institutional cohort, 62% of patients demonstrated impaired GLS despite normal LVEF, a finding that conventional echocardiography would entirely miss. LV-GLS independently correlates with haemoglobin and LDH, reflecting the additive cardiac toll of chronic anaemia and haemolytic burden.

A GLS threshold of > −19.1% provides strong, cohort-derived diagnostic utility (AUC 0.83, sensitivity 78%, specificity 83%) and is readily implementable in clinical practice. We advocate the systematic integration of GLS measurement into periodic cardiac surveillance protocols for all adult HbSS

patients. Prospective longitudinal trials are warranted to determine whether GLS-guided therapeutic escalation translates into improved long-term cardiovascular outcomes in this population.

Declarations

Conflict of Interest
The authors declare no conflicts of interest.

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