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ORIGINAL ARTICLE

Diagnostic Accuracy of 2D Speckle-Tracking Echocardiography for Detecting Coronary Artery Stenosis in Patients with Preserved Ejection Fraction

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ABSTRACT

Background: Two-dimensional speckle-tracking echocardiography can reveal subclinical myocardial dysfunction in patients with preserved left-ventricular ejection fraction. This study aimed to determine whether global and vessel-specific longitudinal strain metrics are associated with angiographically confirmed coronary artery stenosis.

Methods: In a retrospective cross-sectional study at Azadi Teaching Hospital, 90 adults with preserved LVEF ($\geq$ 55%) underwent resting 2D-STE and invasive coronary angiography. VMS/VMSR were computed for LAD, RCA, and LCX territories; GLS/GLSR were calculated for the left ventricle. Territory-level differences used linear regression with patient-clustered errors.

Results: Nearly half (48.9%) had stenosis. Territories with significant stenosis showed worse deformation, significant for LAD VMS, RCA VMSR, and LCX VMS. Territory-level discrimination was modest, best with RCA VMSR and LCX VMS. Patient-level global metrics were lower with significant stenosis. Age showed no correlation; hypertension impaired strain, and diabetes was associated with a borderline lower GLSR.

Conclusion: In patients with preserved EF, 2D-STE particularly territorial strain metrics maps to angiographic stenosis and provides incremental, localization-oriented information, while global indices show moderate discrimination.

Key words: Speckle-tracking echocardiography; Global longitudinal strain; Vessel-specific strain; Coronary artery stenosis; Preserved ejection fraction.

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INTRODUCTION

Myocardial strain imaging, particularly global longitudinal strain (GLS), has become a sensitive, reproducible marker of subclinical left-ventricular dysfunction that adds diagnostic information beyond ejection fraction [1].

Two-dimensional speckle-tracking echocardiography (2D-STE) quantifies myocardial deformation by tracking acoustic "speckles" throughout the cardiac cycle; appropriate acquisition and analysis require attention to methodological issues that can affect diagnostic performance [2].

Across clinical settings, 2D-STE has demonstrated utility, including coronary artery disease (CAD), heart failure, and inflammatory cardiomyopathies, often revealing dysfunction when conventional metrics appear normal [3].

Validation studies have shown that regional and global strain indices correlate with angiographic stenosis and with changes in regional blood flow under stress [4]. Moreover, concordance with advanced imaging modalities such as cardiac MRI supports the role of strain imaging as a cost-effective, widely available front-line tool [5].

Beyond global measures, territory-level (vessel-specific) strain provides incremental insight for detecting ischemia in patients with preserved ejection fraction, in whom wall-motion may remain normal [6]. Regional strain parameters can identify infarcted or ischemic segments and have been linked to subsequent ventricular recovery after myocardial infarction, underscoring their clinical relevance [7].

Territory-level (vessel-specific) strain abnormalities can be detected even when global systolic function and ejection fraction are preserved, as shown in studies using cardiac MRI feature tracking echocardiography; this supports early characterization of ischemic burden using regional deformation indices [8].

Similarly, in acute myocarditis with preserved EF, regional strain reductions are evident, showing that deformation imaging can detect dysfunction not captured by wall motion or EF alone [9]. In hypertension and heart failure with preserved ejection fraction HFpEF (clinical heart failure despite a normal or near-normal EF), strain mapping correlates with markers of microvascular dysfunction and interstitial fibrosis, providing complementary diagnostic information without the need for invasive testing or stress imaging [10].

Accordingly, this study aimed to determine whether two-dimensional speckle-tracking–derived myocardial strain parameters are associated with angiographically confirmed coronary artery stenosis in patients with suspected CAD. The primary objective was to evaluate the diagnostic performance of vessel-specific myocardial strain (VMS) and vessel-specific strain rate (VMSR) for detecting significant stenosis within the left anterior descending (LAD), right coronary (RCA), and left circumflex (LCX) territories. Secondary objectives were to assess the contribution of GLS and global longitudinal strain rate (GLSR) as markers of overall coronary disease burden and to examine the influence of cardiovascular risk factors—hypertension, diabetes mellitus, and smoking—on strain metrics.

MATERIALS AND METHODS

This retrospective cross-sectional study included 90 consecutive adults (30–75 years) evaluated for suspected coronary artery disease (CAD) at Azadi Teaching Hospital, Kirkuk, Iraq. As part of routine care, all patients underwent transthoracic echocardiography in the echocardiography unit and invasive coronary angiography in the hospital catheterization laboratory. Eligibility required preserved left-ventricular ejection fraction (LVEF $\geq$ 55%), no prior coronary revascularization, and no known non-ischemic structural heart disease. Demographics, cardiovascular risk factors, echocardiographic strain metrics, and angiographic findings were abstracted from the medical record by investigators blinded to the other modality's results.

Two-dimensional speckle-tracking echocardiography (2D–STE) was acquired from standard apical views on commercial ultrasound systems with optimized frame rates. Endocardial borders were traced manually; tracking was reviewed segment-by-segment, and segments with inadequate visualization or suboptimal tracking were excluded *a priori*. Global longitudinal strain (GLS) and global longitudinal strain rate (GLSR) were calculated for the left ventricle. Vessel-specific longitudinal strain (VMS) and vessel-specific strain rate (VMSR) were computed as the mean of segments assigned to the left anterior descending (LAD), right coronary (RCA), and left circumflex (LCX) territories using the standard 18-segment model. For presentation and analysis, strain and strain-rate were handled as absolute magnitudes (i.e., reported as positive values); thus, smaller magnitudes indicate worse deformation. All strain analyses were performed offline with vendor-neutral software by operators blinded to angiography.

Coronary angiography was performed using standard techniques with multiple projections of the LAD, RCA, and LCX. Stenosis severity was determined by expert visual estimation (no QCA). A lesion was classified as a *significant stenosis* when estimated luminal narrowing was $\geq$ 70% in any major epicardial coronary artery or $\geq$ 50% in the left main coronary artery, reflecting widely accepted diagnostic criteria. Lesions below these thresholds were considered *non-significant*. For territory-level analyses, each vessel territory (one label per vessel per patient) was dichotomized as *significant* (S) or *non-significant* (NS) according to the most severe lesion within that territory. All assessments relied on expert visual interpretation, consistent with real-world clinical practice. Statistical analyses were conducted in SPSS v26. Continuous variables are reported as mean ± SD and categorical as *n* (%); all tests were two-sided at $\alpha = 0.05$ without adjustment for multiple comparisons. Territory-level comparisons used linear regression with patient-clustered (Huber–White) standard errors, modeling strain as absolute magnitudes; the coefficient (β) estimates the (significant stenosis − non-significant stenosis) mean difference, with negative values indicating worse deformation in territories with significant stenosis. Discrimination was assessed with ROC curves and AUCs using cluster bootstrapping; scores were inverted to preserve directionality, and positive/negative predictive values were reported at prespecified thresholds reflecting study sample prevalence. Patient-level GLS/GLSR differences were tested with Welch's *t*-test, with nonparametric bootstrap AUCs and predictive values at prespecified cut-offs. Associations between age and strain used Pearson correlation; comorbidity effects (hypertension, diabetes, smoking) were examined with unadjusted independent-samples *t*-tests (Welch's correction as needed). The primary outcome was the territory-level association of vessel-specific strain/strain-rate (VMS/VMSR) with angiographic stenosis in the LAD, RCA, and LCX; the secondary outcome was the patient-level diagnostic performance of global strain/strain-rate (GLS/GLSR) for any-vessel stenosis. The study complied with the Declaration of Helsinki and was approved by the Kirkuk Health Directorate Research Committee (document no. 425; July 10, 2025). Given the retrospective design and use of de-identified data, the requirement for individual informed consent was waived. Data were stored on secure, access-restricted systems.

RESULTS

Ninety adults with preserved LVEF (≥ 55%) were included (mean age 59.2 ± 7.3 years). Hypertension, diabetes mellitus, and smoking were present in 42/90 (46.7%), 30/90 (33.3%), and 10/90 (11.1%), respectively; all had normal regional wall motion on resting echocardiography (Table 1).

Table 1. Baseline study sample characteristics (*n* = 90)

Variable	Mean ± SD	*n*	%
Age, years	59.2 ± 7.3	–	–
Hypertension	–	42	46.7
Diabetes mellitus	–	30	33.3
Smoking	–	10	11.1
Preserved EF (≥ 55%)	–	90	100.0

Data are mean ± SD or counts and percentages.
Abbreviations: EF, ejection fraction.

Angiographic classification

Any-vessel *significant* stenosis was present in 44/90 (48.9%) patients; per-territory counts are shown in Table 2.

Table 2. Angiographic classification by coronary territory (*n* = 90 per artery)

Artery	Territories, *n*	S	NS
LAD	90	36	54
RCA	90	24	66
LCX	90	20	70

Abbreviations: LAD, left anterior descending artery; RCA, right coronary artery; LCX, left circumflex artery; S, significant stenosis (≥ 70% epicardial or ≥ 50% left main); NS, non-significant stenosis.

Territory-level strain (primary analysis)

Vessel-specific longitudinal strain (VMS) and strain rate (VMSR) were lower in regions with significant stenosis across vessels. In patient-clustered models, stenosis was associated with significantly lower RCA VMSR and LCX VMS; LAD VMS showed a borderline reduction (Table 3, panel A).

Diagnostic performance by territory (ROC)

Discrimination was modest overall, with the highest AUCs for RCA VMSR and LCX VMS (Table 3, panel B).

Global strain (secondary analysis)

For the patient-level endpoint (any-vessel stenosis), GLS and GLSR were lower in patients with significant stenosis and showed moderate ROC performance (Table 4).

Age and comorbidity effects

Age was not correlated with any strain index (Table 5). In exploratory, unadjusted comparisons, hypertension and diabetes were associated with more impaired strain, whereas smoking was not (Table 6). These comorbidity findings did not change the primary territory-level associations.

Table 3. Territory-level strain: means with clustered comparisons (Panel A) and ROC performance (Panel B)

(A) Means and clustered comparisons by stenosis status

Vessel	Metric	NS mean ± SD (n)	S mean ± SD (n)	β (S−NS)	95% CI	*p*
LAD	VMS (%)	12.50 ± 3.93 (54)	10.96 ± 2.94 (36)	−1.541	−3.14 to 0.05	0.056
LAD	VMSR (s^{-1})	1.48 ± 0.61 (54)	1.28 ± 0.39 (36)	−0.202	−0.410 to 0.007	0.058
RCA	VMS (%)	14.25 ± 3.45 (66)	13.35 ± 3.19 (24)	−0.897	−2.372 to 0.577	0.233
RCA	VMSR (s^{-1})	1.64 ± 0.40 (66)	1.43 ± 0.23 (24)	−0.216	−0.352 to −0.080	**0.002**
LCX	VMS (%)	10.63 ± 4.28 (70)	8.68 ± 3.13 (20)	−1.946	−3.565 to −0.327	**0.019**
LCX	VMSR (s^{-1})	1.22 ± 0.50 (70)	1.19 ± 0.44 (20)	−0.031	−0.256 to 0.193	0.784

(B) ROC performance and operating points

Vessel	Predictor	AUC (95% CI)	Cut-off	Sens	Spec	PPV	NPV
LAD	VMS	0.603 (0.483–0.716)	≤ 13.8 %	0.92	0.32	0.48	0.85
LAD	VMSR	0.561 (0.440–0.670)	≤ 1.4 s^{-1}	0.75	0.42	0.47	0.71
RCA	VMS	0.568 (0.451–0.694)	≤ 17.8 %	1.00	0.18	0.31	1.00
RCA	VMSR	0.668 (0.554–0.785)	≤ 1.7 s^{-1}	0.92	0.36	0.34	0.92
LCX	VMS	0.639 (0.504–0.754)	≤ 9.5 %	0.75	0.59	0.34	0.89
LCX	VMSR	0.509 (0.366–0.646)	≤ 1.6 s^{-1}	0.90	0.19	0.24	0.87

Panel A: β from linear regression with patient-clustered standard errors; negative β indicates smaller absolute magnitude (worse deformation) in stenotic territories. Means/SDs are descriptive; *n* reflects analyzable territories after speckle-tracking quality control.
Panel B: ROC, receiver-operating characteristic; AUC, area under the curve; CI, confidence interval; **Sens = sensitivity**; **Spec = specificity**; PPV, positive predictive value; NPV, negative predictive value. Cut-offs were prespecified; predictive values reflect study-sample prevalence.
Abbreviations (both panels): GLS, global longitudinal strain; GLSR, global longitudinal strain rate; VMS, vessel-specific longitudinal strain; VMSR, vessel-specific strain rate; LAD, left anterior descending artery; RCA, right coronary artery; LCX, left circumflex artery.

Table 4. Global diagnostic performance (any-vessel significant stenosis vs non-significant stenosis)

Predictor	AUC (95% CI)	Cut-off	Sens	Spec	PPV	NPV
Group means						
Non-significant stenosis (n = 46): GLS 12.07 ± 3.19%, GLSR 0.92 ± 0.29 s^{-1}						
Significant stenosis (n = 44): GLS 10.43 ± 2.65%, GLSR 0.79 ± 0.23 s^{-1}						
Welch *t*-tests (S−NS)						
GLS: −1.653 percentage points (95% CI −2.790 to −0.516), p = 0.004						
GLSR: −0.132 s^{-1} (95% CI −0.205 to −0.059), $p < 0.001$						
ROC and operating points						
GLS	0.644 (0.520–0.753)	≤ 11.6 %	0.74	0.48	0.57	0.67
GLSR	0.630 (0.514–0.738)	≤ 1.0 s^{-1}	0.91	0.33	0.56	0.79

Abbreviations: GLS, global longitudinal strain; GLSR, global longitudinal strain rate; ROC, receiver-operating characteristic; AUC, area under the curve; CI, confidence interval; **Sens = sensitivity**; **Spec = specificity**; PPV, positive predictive value; NPV, negative predictive value.

Table 5. Correlation of age with strain indices

Parameter	*r*	*p*
GLS	−0.019	0.862
GLSR	−0.027	0.805
LADVMS	−0.019	0.857
LADVMSR	−0.085	0.426
RCAVMS	0.064	0.551
RCAVMSR	0.101	0.348
LCXVMS	0.024	0.823
LCXVMSR	−0.014	0.895

Pearson correlations.
Abbreviations: GLS, global longitudinal strain; GLSR, global longitudinal strain rate; VMS, vessel-specific longitudinal strain; VMSR, vessel-specific strain rate; LAD, left anterior descending artery; RCA, right coronary artery; LCX, left circumflex artery.

Table 6. Exploratory associations between comorbidities and global / vessel-averaged strain metrics

Comorbidity	Metric (unit)	Present, mean±SD	Absent, mean±SD	*t*	*p*
Hypertension	GLS (%)	10.5 ± 3.1	11.9 ± 3.6	−2.21	0.030
Hypertension	V-GLS (%)	12.3 ± 4.0	14.5 ± 3.8	−2.78	0.007
Diabetes	GLSR (s^{-1})	0.82 ± 0.27	0.93 ± 0.29	−1.96	0.054
Diabetes	V-GLSR (s^{-1})	1.25 ± 0.44	1.45 ± 0.51	−2.12	0.038
Smoking	GLS (%)	11.0 ± 3.3	11.2 ± 3.4	−0.31	0.758
Smoking	V-GLS (%)	13.1 ± 4.2	13.6 ± 4.0	−0.58	0.564

Values are mean ± SD. Independent-samples *t*-tests (Welch's correction when variances were unequal); exploratory, unadjusted analyses.
Abbreviations: GLS, global longitudinal strain; GLSR, global longitudinal strain rate; V-GLS, vessel-averaged longitudinal strain (composite across LAD, RCA, LCX per subject); V-GLSR, vessel-averaged strain rate.

DISCUSSION

Subclinical ischemia may escape detection on resting echocardiography when LVEF is preserved, and wall motion appears normal. Two-dimensional speckle-tracking echocardiography (2D-STE) quantifies deformation (GLS/GLSR and vessel-specific strain/strain rate), providing a sensitive and noninvasive method for detecting CAD and localizing culprit territories [11].

In this study of adults with preserved LVEF evaluated for suspected CAD, deformation abnormalities measured by 2D–STE were mapped to angiographic disease. Territory-level analyses showed more impaired mechanics in regions with significant stenosis—most clearly for RCA VMSR and LCX VMS, with a borderline reduction for LAD VMS—while patient-level GLS/GLSR were lower in those with any-vessel stenosis and yielded only moderate discrimination. These global findings accord with prior work demonstrating that impaired GLS tracks CAD burden: Koulaouzidis et al. (2025) [12] reported diagnostic GLS ranges around −16.25% to −14.45%; Fiorillo et al. (2021) [13] found lower GLS in peripheral artery disease patients with concomitant CAD, supporting asymptomatic risk screening; Bar et al. (2022) [14] observed stepwise GLS decline with increasing vessel involvement and proposed a −18.3% cut-off for single-vessel disease; Al-Amin et al. (2020) [15] described inverse correlations between GLS and Gensini scores in NSTEMI; and Mawla et al. (2022) [16] showed reasonable accuracy for severe lesions using a −15.9% threshold. Variability in vessel-specific signals—including the weaker LAD association here—likely reflects differences in coronary dominance, loading conditions, image quality, and analytic workflow across studies.

Our regional findings reinforce the value of territorial strain for localization. Guaricci et al. (2022) [17] showed that coronary-specific deformation can disclose the culprit vessel in NSTE–ACS, particularly when combined with wall-motion scoring.

Segmental strain retains diagnostic value when EF is preserved and wall motion appears normal, as reported by El-sayed et al. (2023) [18]. In acute settings, Zghal et al. (2020) [19] identified the occluded artery in most NSTEMI cases using territorial strain, while in unstable angina Bajracharya et al. (2020) [20] noted progressive strain deterioration with increasing stenosis severity. Consistent with our pattern, arteries with ≥ 70% stenosis exhibit significantly lower VMS/VMSR, as confirmed by Chaichuum et al. (2022) [21]. Collectively, these data position territorial strain as a sensitive adjunct for detecting ischemia and guiding diagnostic pathways when global indices are equivocal.

Comorbidity signals in our participants were directionally consistent with cardiometabolic injury to myocardial mechanics: hypertension and diabetes were associated with more impaired deformation, whereas smoking was not. Prior evidence supports additive adverse effects Li et al. (2020) [22] demonstrated that type 2 diabetes worsens deformation and perfusion in hypertension; Tadic et al. (2020) [23] reported greater impairment when hypertension and diabetes coexist than with either alone; and even in otherwise healthy individuals, smoking-related inflammation is linked to abnormal strain, as shown by Yaman et al. (2019) [24] and Mandraffino et al. (2020) [25]. Elnoamany et al. (2020) [26] similarly found that combined HTN/DM depresses strain and strain-rate more than either condition alone. Together with our data, these studies underscore the sensitivity of speckle-tracking to early cardiometabolic myocardial involvement.

Age was not correlated with deformation indices in this preserved-EF study sample contrasting with reports from HFpEF populations where aging, remodeling, and microvascular dysfunction relate to worse strain Lan et al. (2024) [27]; Shukla and Mohan (2023) [28]; and Yahia et al. (2023) [29] with smaller or context-dependent effects in selected subgroups, as noted by Park et al. (2022) [30] and Serezhina and Obrezan, 2021 [31]. This discrepancy highlights the importance of clinical context and exclusion of confounding pathology when interpreting age–strain relationships.

Overall, our results support 2D-STE—particularly territorial strain measures—as a practical adjunct to standard evaluation for suspected CAD, offering sensitivity suitable for ruling out CAD and potential territorial localization while acknowledging only moderate global diagnostic performance.

This study has important limitations. Its retrospective, single-center design with a modest sample (especially few smokers) limits power, generalizability, and susceptibility to selection and information bias. Coronary stenosis was determined by visual angiography (no QCA, FFR/iFR), and vessel assignment by the 18-segment model ignores anatomic variation/dominance. Only resting 2D-STE in patients with preserved EF was examined, without stress imaging or longitudinal outcomes, so causal inference and prognostic significance cannot be established.

Segments with poor tracking were excluded (potential informative missingness), inter/intra-observer reproducibility and vendor effects were not assessed, multiple territory-level tests were unadjusted for multiplicity, and PPV/NPV reflect the cohort's 49% prevalence. Future work should be prospective and multicenter, use standardized acquisition with blinded core-lab analysis, incorporate stress echocardiography or perfusion CMR and quantitative/physiologic coronary assessment (QCA, FFR/iFR), employ CTA-based territorial mapping, report reproducibility and vendor harmonization, model non-linearity and key confounders, pre-specify/validate diagnostic cut-offs, and test incremental and prognostic value over clinical risk scores.

CONCLUSION

In adults with preserved LVEF, 2D-speckle tracking showed clear territory-level correspondence with angiographic stenosis—most pronounced for RCA VMSR and LCX VMS—while GLS/GLSR provided moderate patient-level discrimination. Hypertension and diabetes were associated with more impaired deformation, whereas age and smoking were not. These findings support 2D-STE as a practical adjunct for evaluating suspected CAD, warranting prospective, multi-center validation and standardized diagnostic-performance testing.

ETHICAL DECLARATIONS

- ### Ethics Approval and Consent to Participate

Ethical approval was granted by the Kirkuk Health Directorate Research Committee (document no. 425; July 10, 2025).

- ### Consent for Publication

Non.

- ### Availability of Data and Material

The datasets are available from the corresponding author upon reasonable request.

- ### Competing Interests

The authors declare that there is no conflict of interest.

- ### Funding

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- ### Use of Generative Artificial Intelligence

The authors declare that no generative AI tools were used in the preparation, writing, or editing of this manuscript.

- ### Authors' Contributions

Khalil S. Ahmed was responsible for the literature review, design of the study, collection of the data, statistical analysis, and writing the manuscript. The author read and approved the final version of the manuscript.

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