

## Correlation between Left Ventricular Speckle Tracking and Coronary Angiography in Patients with Suspected Coronary Artery Disease

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### Abstract

**Background:** To evaluate the efficacy of speckle tracking echocardiography (STE) in detecting the existence, degree, and intensity of coronary artery affection in patients with suspected coronary artery disease (CAD). **This study aimed to** Investigate the ability of STE to identify the existence, size, and intensity of coronary artery affection in individuals with a suspected diagnosis of CAD. **Methods:** This cross-sectional study enrolled 200 candidates with suspected COD. Patients underwent STE and coronary angiography. Global longitudinal peak systolic strain (GLPSS) was measured and linked with coronary angiography findings for every subject. **Results:** ROC analysis was done for GLPSS in predicting single-vessel affection. It revealed a significant AUC of 0.713, with confidence interval of 95 percent ranging from 0.555-0.871 (P = 0.013). The best cutoff was  $\leq -18$ , at which specificity and sensitivity were 53.8 and 83 percent. ROC analysis was done for GLPSS in predicting multi-vessel affection. It revealed a significant AUC of 0.908 (P < 0.001). The best cutoff was  $\leq -11$ , at which specificity and sensitivity were 89.6 percent and 87.7 percent. **Conclusion:** Two-dimensional speckle tracking echocardiography predicts the existence, size, and degree of CAD with high sensitivity and specificity.

**Keywords:** Global Longitudinal Strain, CAD, stable angina, Coronary angiography

### 1. Introduction

The diagnosis and assessment of coronary artery disease involves clinical evaluation, identifying risk factors for atherosclerosis, and specific cardiac examinations as various stress tests modalities and coronary imaging (1).

non-invasive identification of patients with CAD is medical dilemma; Coronary angiography revealed that more than half of the individuals had normal or non-obstructive CAD (2).

The diagnosis of CAD using echocardiography rely on the identification of aberrant left ventricular (LV) wall motion and evaluation of LVEF. CAD individuals with no MI history have no aberrant LV wall movements at rest (3).

strain can be used in assessing myocardial contraction myocardial viability either at rest or with stress (4).

It is more appropriate to use 2D-STE than conventional 2D echocardiography in evaluating the regional and global myocardial function and measuring myocardial damage, vitality, and moderate alterations of myocardial ischemia (5).

Speckle tracking echocardiography is a semi-automatic method, so it gives great intra-observer and inter-observer reproducibility (6).

"strain" and "strain rate" scanning need one cardiac cycle for offline processing and interpretation (7).

Longitudinal strain gives an accurate quantitative measurement of myocardial distortion within every LV section, enabling early diagnosis of systolic dysfunction in preserved LVEF individuals (8).

Using STE longitudinal strain, CAD may be detected and risk stratified with high precision and consistency. Strain and SR are uniformly distributed throughout heart, therefore minor variations in either metric indicate cardiac dysfunction. While strain scanning has the potential to play a role in diagnosis and therapy of nearly any cardiac illness, its most important function is to identify IHD (9).

Measuring cardiac strain and strain rate are more recent indicators with the ability to circumvent these constraints. Strain and Strain rate reflect the amplitude and rate of myocardial distortion, respectively (10).

### 2. Aim of the work

That research aims to detect validity of LV speckle tracking in suspected CAD individuals and correlates these findings with coronary angiographic results.

### 3. Patients and methods

This single center, cross sectional research concluded 200 individuals with suspected coronary artery disease from January 2021 to November 2022 at cardiology department, Benha university. All participants completed a written informed consent, and the local ethics committee authorized the research.

**Inclusion criteria were** individuals with typical angina pain, critical ECG differences patients, positive non-invasive imaging individuals (resting echo, stress ECG, stress echo) and age more than 18 years old.

**Exclusion criteria** were serum creatinine >2 mg/dL, Poor echocardiographic window, Rhythm other than sinus rhythm, Significant valvular heart disease, Hypertrophic or idiopathic restrictive cardiomyopathy, Patients with previous cardiac surgery, pulmonary hypertension, prior history of PCI, prior history of chronic obstructive lung disease or recent pulmonary embolism.

**All patients were subjected to** complete demographic data involving age, gender, and cardiovascular risk factors. Systolic and diastolic BP, Heart rate, resting 12-lead ECG, Ischemic changes, Associated arrhythmias and conduction defects.

Conventional Trans-thoracic echocardiography was done by a comprehensive traditional echocardiographic assessment utilising PHILIPS Affiniti 50, USA apparatus. LVEF, LV end-diastolic volume and LV end-systolic volume were assessed utilizing modified Simpsons method.

All patients were examined to detect wall motions abnormalities by regional wall motion score index, using 17 segments model by American Society of Echocardiography. Every section was assessed by semi-quantitative score (normo-kinetic = 1, hypokinetic = 2, akinetic = 3, dyskinetic = 4) and worldwide wall motion score index will be determined by averaging regional scores.

2D ST Strain Analysis was performed by 3 straight cardiac rounds were captured in the apical four, apical two, and apical three chamber images at high frame rates (>70 frames/sec). Using a semi-automated technique, three locations were found (basal septal, basal lateral and apical) (11,12).

All patients had coronary angiography throughout one week following speckle tracking echo by an experienced cardiologist who was masked to the echocardiographic data using the Judkins method. Substantial stenosis is described as stenosis of 50% in the left main artery and 70% in the right coronary, left anterior ascending, and circumflex arteries. Multivessel CAD is described as severe stenosis in two or more vessels. severity coronary artery disease will be assessed by Gensini score being calculated by assigning a severity score to every coronary stenosis as follows: 1 point for 25 percent narrowing, 2 points for 26–50 percent narrowing, 4 points for 51–75 percent narrowing, 8 points for 76–90 percent narrowing, 16 points for 91–99 percent narrowing, and 32 points for complete blockage. Every lesion score is then expressed in equation that accounts for the significance of the lesion's location in the coronary circulation (2.5 for the proximal section of the left anterior declining coronary artery, and 5 for the left main coronary artery, 2.5 for the proximal section of the circumflex artery and 1.5 for the middle portion of the left anterior declining coronary artery, 1.0 for the right coronary artery, distal section of the left anterior declining coronary

artery, posterolateral artery, and obtuse marginal artery, and 0.5 for other parts) (13).

#### Statistical analysis

Version 28 of SPSS was used for data administration and statistical analysis (IBM, Armonk, New York, United States). Means and standard deviations were used to summaries quantitative data. As numbers and percentages, categorical data were summed up. Quantitative data were compared according to coronary affection using one-way ANOVA. Pairwise analyses were performed in case of a critical total influence. All pairwise analyses were modified for multiple comparisons. Correlation analyses were done using Pearson's correlation. ROC analysis was done for GLPSS to predict single-vessel and multi-vessel disease. Areas Under Curve (AUC) had 95 percent assurance intervals, best cutoff points, and diagnostic indices were measured. Multinomial logistic regression analysis was performed to predict coronary affection. Odds ratios had 95 percent assurance intervals were measured. All statistical tests had two outcomes. P values below 0.05 were deemed statistically meaningful.

#### 4. Results

Patients' general characteristics, Echo and coronary angiography findings of evaluated individuals were shown in **Table 1**.

A critical correlation was seen among diabetes and coronary affection ( $P = 0.024$ ); Diabetes was higher in those with multi-vessel affection (66.7%) compared to those with single-vessel affection (52.8%) or normal coronaries (30.8%). Additionally, hypertension appeared a critical correlation with coronary affection ( $P = 0.004$ ); Dyslipidemia showed a similar association with coronary affection ( $P < 0.001$ ). No critical changes were seen concerning age ( $P = 0.778$ ), gender ( $P = 0.781$ ), BMI ( $P = 0.474$ ), family history ( $P = 0.336$ ), and smoking ( $P = 0.622$ ).

#### Table 2

**Regarding the clinical parameters according to coronary affection:** EF significantly differed between levels of coronary affection ( $P < 0.001$ ). It was substantially lower in those with single ( $49 \pm 4$ ) or multiple vessel disease ( $41 \pm 6$ ) than normal coronaries ( $53 \pm 4$ ). Additionally, it was significantly higher in single-vessel disease than in multi-vessel disease individuals. RWMSI showed a significant difference between the levels of coronary affection ( $P < 0.001$ ). It was substantially higher in single ( $1.5 \pm 0.3$ ) or multiple vessel disease ( $2.1 \pm 0.3$ ) than normal coronaries ( $1.3 \pm 0.3$ ) individuals. Additionally, it was significantly lower in single-vessel disease than multi-vessel disease patients. ESV revealed a significant difference according to coronary affection ( $P < 0.001$ ). It was substantially higher in

single ( $68 \pm 8.3$ ) or multiple vessel disease ( $87.4 \pm 12.6$ ) patients than normal coronary patients ( $61.3 \pm 8.3$ ). Additionally, it was significantly lower in single-vessel than multi-vessel diseases.

EDV significantly differed between coronary affection levels ( $P < 0.001$ ). It was substantially lower in single-vessel ( $130 \pm 9$ ) or normal coronary ( $125 \pm 9$ ) diseases than multi-vessel disease ( $147 \pm 7$ ). GLPSS showed an overall critical change among coronary affection levels ( $P < 0.001$ ). It was substantially lower in single ( $-15 \pm 3$ ) or multiple vessel ( $-9 \pm 4$ ) diseased individuals than normal coronary individuals ( $-17 \pm 3$ ). Additionally, it was significantly higher in single-vessel disease than in multi-vessel disease individuals. Gensini score significantly differed between levels of coronary affection ( $P < 0.001$ ). It was substantially higher in single ( $63 \pm 3$ ) or multiple vessel disease ( $76 \pm 10$ ) than normal coronary individuals ( $55 \pm 7$ ). Additionally, it was significantly lower in single-vessel diseased than multi-vessel diseased individuals **Table 3**.

GLPSS revealed a critical positive association with EF ( $r = 0.965$ ,  $P < 0.001$ ). In contrast, it revealed significant negative correlations with RWMSI ( $r = -0.953$ ,  $P < 0.001$ ), ESV ( $r = -0.947$ ,  $P$

$< 0.001$ ), EDV ( $r = -0.761$ ,  $P < 0.001$ ), and Gensini score ( $r = -0.936$ ,  $P < 0.001$ ). **Table 4**

ROC analysis was done for GLPSS in predicting single-vessel affection. It revealed a significant AUC of 0.713, with a 95 percent assurance interval ranging from 0.555-0.871 ( $P = 0.013$ ). Best cutoff was  $\leq -18$ , at which specificity and sensitivity were 53.8 percent and 83 percent.

#### Figure 1

ROC analysis was done for GLPSS in predicting multi-vessel affection. It revealed a significant AUC of 0.908 ( $P < 0.001$ ). Best cutoff was  $\leq -11$ , at which specificity and sensitivity were 89.6 percent and 87.7 percent. **Figure 2**

Multinomial logistic regression analysis was done to predict coronary affection (single and multi-vessel). The model was built clinically, including GLPSS and all factors that might contribute to coronary affection. GLPSS was an independent predictor for single coronary affection (OR = 0.8, 95% CI = 0.647 – 0.989,  $P = 0.04$ ) and multi-vessel affection (OR = 0.487, 95% CI = 0.376 - 0.632,  $P < 0.001$ ), controlling for age, gender, BMI, diabetes, hypertension, dyslipidemia, family history, and smoking. **Table 5**

**Table (1)** General characteristics, Echo and coronary angiography findings of the studied patients

<b>Age (years)</b>	62 ±11
<b>Gender</b>	
Males	126 (63)
Females	74 (37)
<b>BMI</b>	35 ±3
<b>Diabetes mellitus</b>	114 (57)
<b>Hypertension</b>	129 (64.5)
<b>Dyslipidemia</b>	126 (63)
<b>Family history</b>	87 (43.5)
<b>Smoking</b>	95 (47.5)
<b>Echo and coronary angiography findings</b>	
<b>EF (%)</b>	46 ±7
<b>RWMSI</b>	1.8 ±0.4
<b>ESV (ml)</b>	75.4 ±14.4
<b>EDV (ml)</b>	136 ±12
<b>Coronary affection</b>	
Normal	13 (6.5)
Single vessel affection	106 (53)
Multi-vessel affection	81 (40.5)
<b>LAD affection</b>	151 (75.5)
<b>RCA affection</b>	111 (55.5)
<b>LCX affection</b>	42 (21.0)
<b>GLPSS</b>	-13 ±4
<b>Gensini score</b>	68 ±10

Data are presented as mean ±SD or number (percentage)

**Table (2)** General characteristics according to levels of coronary affection

	<b>Normal (n = 13)</b>	<b>Single vessel (n = 106)</b>	<b>Multivessel (n = 81)</b>	<b>P-value</b>
<b>Age (years)</b>	63 ±11	62 ±11	61 ±10	0.778

<b>Gender</b>				
Males	9 (69.2)	68 (64.2)	49 (60.5)	0.781
Females	4 (30.8)	38 (35.8)	32 (39.5)	
<b>BMI</b>	36 ±4	35 ±3	35 ±3	0.474
<b>DM</b>	4 (30.8)	56 (52.8)	54 (66.7)	<b>0.024</b>
<b>HTN</b>	6 (46.2)	60 (56.6)	63 (77.8)	<b>0.004</b>
<b>Dyslipidemia</b>	8 (61.5)	53 (50)	65 (80.2)	<b>&lt;0.001</b>
<b>Family history</b>	6 (46.2)	41 (38.7)	40 (49.4)	0.336
<b>Smoking</b>	7 (53.8)	47 (44.3)	41 (50.6)	0.622

Data are presented as mean ±SD or number (percentage); Significant P-values are marked in bold.

Table (3) Clinical parameters according to levels of coronary affection

	Coronary affection			P-value
	Normal	Single vessel	Multivessel	
<b>EF</b>	53 ±4 <sup>a</sup>	49 ±4 <sup>b</sup>	41 ±6 <sup>c</sup>	<b>&lt;0.001</b>
<b>RWMSI</b>	1.3 ±0.3 <sup>a</sup>	1.5 ±0.3 <sup>b</sup>	2.1 ±0.3 <sup>c</sup>	<b>&lt;0.001</b>
<b>ESV (ml)</b>	61.3 ±8.3 <sup>a</sup>	68 ±8.3 <sup>b</sup>	87.4 ±12.6 <sup>c</sup>	<b>&lt;0.001</b>
<b>EDV (ml)</b>	125 ±9 <sup>a</sup>	130 ±9 <sup>a</sup>	147 ±7 <sup>b</sup>	<b>&lt;0.001</b>
<b>GLPSS</b>	-17 ±3 <sup>a</sup>	-15 ±3 <sup>b</sup>	-9 ±4 <sup>c</sup>	<b>&lt;0.001</b>
<b>GENSINI score</b>	55 ±7 <sup>a</sup>	63 ±4 <sup>b</sup>	76 ±10 <sup>c</sup>	<b>&lt;0.001</b>

Data are presented as mean ±SD or number (percentage); EF: Ejection fraction; RWMSI: Regional wall motion score index; ESV: End systolic volume; EDV: End diastolic volume; GLPSS: Global longitudinal peak systolic strain; Different small letters between any pair indicate statistical significance; Significant P-values are marked in bold.

Table (4) Correlation between GLPSS and other parameters

	GLPSS	
	r	P
<b>Age (years)</b>	0.048	0.497
<b>BMI</b>	0.016	0.819
<b>EF</b>	.965	<b>&lt;.001</b>
<b>RWMSI</b>	-.953	<b>&lt;.001</b>
<b>ESV ml</b>	-.947	<b>&lt;.001</b>
<b>EDV ml</b>	-.761	<b>&lt;.001</b>
<b>GENSINI score</b>	-.936	<b>&lt;.001</b>

r: Correlation coefficient; EF: Ejection fraction; RWMSI: Regional wall motion score index; ESV: End systolic volume; EDV: End diastolic volume; GLPSS: Global longitudinal peak systolic strain Significant P-values are marked in bold.

Table (5) Multivariate logistic regression analysis to predict coronary affection

		OR (95% CI)	P-value
<b>Single vessel</b>	Age (years)	0.987 (0.932 - 1.045)	0.649
	Gender	1.141 (0.305 - 4.264)	0.844
	BMI	0.911 (0.76 - 1.091)	0.312
	DM	2.45 (0.637 - 9.426)	0.193
	HTN	1.581 (0.401 - 6.231)	0.513
	Dyslipidemia	0.522 (0.138 - 1.976)	0.339
	Family history	0.965 (0.23 - 4.046)	0.962
	Smoking	0.826 (0.217 - 3.149)	0.779
	GLPSS	0.8 (0.647 - 0.989)	<b>0.040</b>
	<b>Multi-vessel</b>	Age (years)	0.986 (0.92 - 1.056)
Gender		1.278 (0.278 - 5.869)	0.752
BMI		0.954 (0.767 - 1.186)	0.669
DM		3.954 (0.806 - 19.402)	0.09
HTN		1.94 (0.392 - 9.608)	0.417
Dyslipidemia		1.169 (0.249 - 5.49)	0.843
Family history		1.333 (0.256 - 6.944)	0.733

Smoking	1.416 (0.305 - 6.581)	0.657
GLPSS	0.487 (0.376 - 0.632)	<b>&lt;.001</b>

OR: Odds ratio; 95% CI: 95% Confidence interval; Significant P-values are marked in bold.

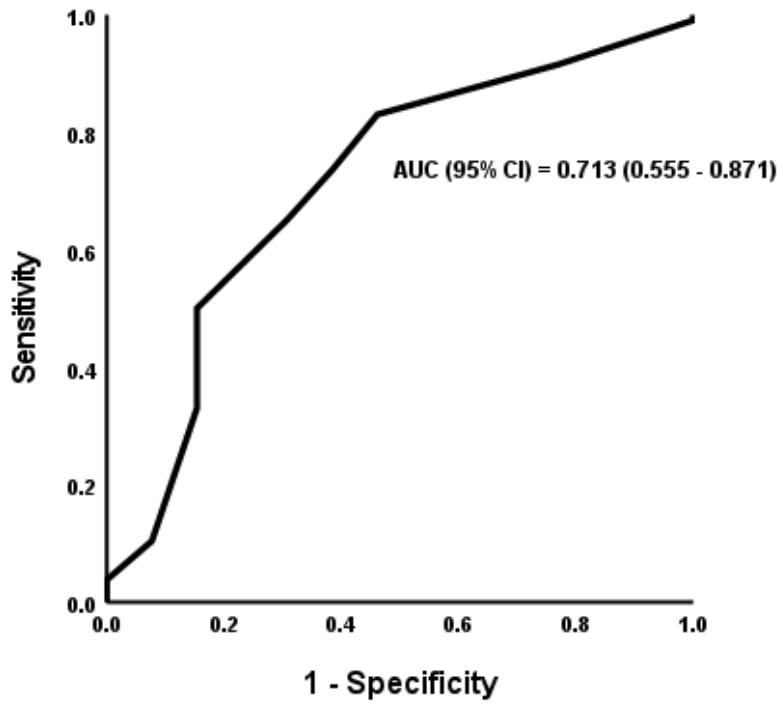


Fig. (1) ROC analysis of GLPSS to predict single coronary affection

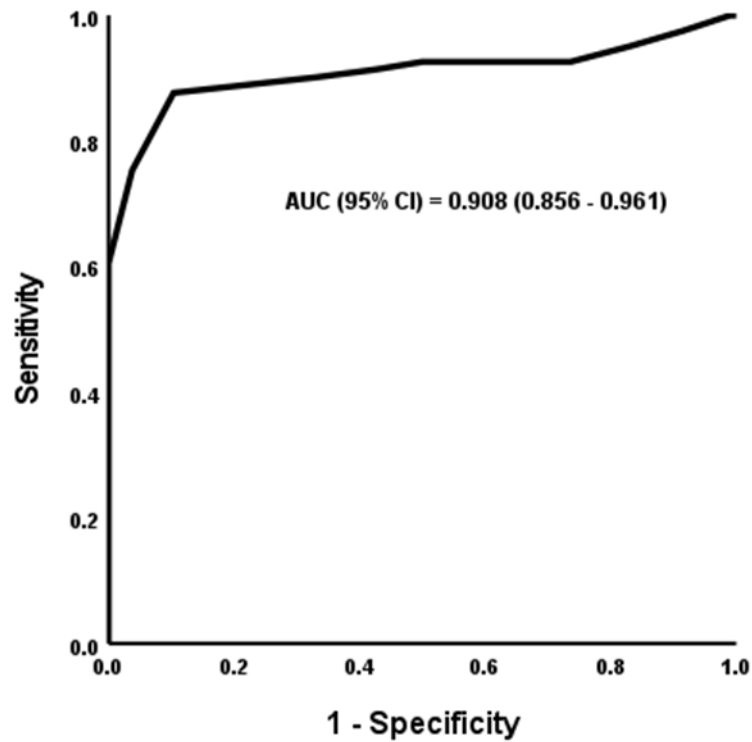


Fig. (2) ROC analysis of GLPSS to predict multi-vessel coronary affection

## 5. Discussion

In this research, the GLPSS value by STE was used to identify the existence and intensity of CAD in individuals with stable CAD. The GLPSS score was negatively correlated with CAD intensity.

In current investigation, 200 individuals were evaluated, More than half of the patients (53%) had single-vessel affection. More than one-third (40.5%) had multi-vessel affection, while only 6.5% had normal coronaries. This may be related to the fact that the questionnaire used at our institution was more sensitive than particular, providing us with a powerful control group. Mean age of studied patients was  $62 \pm 11$  years, that was NEARLY like mean population age  $60 \pm 12$  revealed by Montgomery et al 5. Consistent with previously published research, we identified a substantially greater proportion of patients with advanced age, male gender, BMI, diabetes, and smoking in the CAD group than in the non-CAD group. (5,14,15)

In this research, GLPSS was critically lower in single ( $-15 \pm 3$ ) or multiple vessel disease ( $-9 \pm 4$ ) than normal coronary individuals ( $-17 \pm 3$ ). Additionally, whereas in research by Gaibazzi et al 16 found  $-22 \pm 1.5$  (SVD),  $-19.4 \pm 2.4$  (DVD) and  $-18 \pm 2.3$  (TVD) in CAD patients and Radwan et al 17 reported GLPSS value of  $-15.13 \pm 0.64$  (SVD),  $-12.25 \pm 0.9$  (DVD) and  $-9.1 \pm 1.94$  (TVD), that validates our study's finding of an inverse relationship among GLPSS value and CAD intensity.

In single vessel affection this study found cutoff was  $\leq -18$ , at which specificity and sensitivity were 53.8 percent and 83 percent. In multivessel affection this study found cutoff was  $\leq -18$ , at which sensitivity and specificity were 83% and 53.8%, respectively

Moustafa et al 14 reported cutoff GLPSS value for SVD, DVD, TVD, high syntax score ( $> 16$ )  $-18.44$  (specificity 95.1 percent, sensitivity 90 percent);  $-17.35$  (specificity 88.9 percent, sensitivity 90 percent);  $-15.33$  (specificity 72 percent, sensitivity 63 percent);  $-13.75$  (specificity 91 percent, sensitivity 80 percent) respectively that validates the current study's SVD cutoff value is  $> -20$  with 70.27 percent specificity and sensitivity of 79.69 percent, DVD ( $-18\%$ ) with specificity of 86.49 percent and sensitivity of 77.70 percent, TVD ( $> -16\%$ ) with 98.20 percent specificity and sensitivity of 81.82 percent and high syntax ( $> 22$ )  $> -16$  with specificity 83.33 percent and sensitivity 76.7 percent. This could be the result of observer and vendor variation.

Abdelrazek et al 15 determined that the GLPSS cut-off value for high syntax score ( $\geq 22$ ) was  $-16.5$  (specificity 91 percent, sensitivity 93 percent), which is comparable to the present study's GLPSS cut-off value of  $-16$ , which has a sensitivity

of 81.82 percent and a specificity of 98.20 percent for syntax score  $\geq 22$ .

SYNTAX score is utilized to assess the difficulty of coronary revascularization lesions. The majority of research have shown that longitudinal strain corresponds with the existence and severity of CAD, although correlations between GLPSS and Syntax score are few.

Tanaka et al. (18) found a modest relationship among SYNTAX scores and the amount of stress-induced myocardial ischemia as evaluated by myocardial SPECT ( $r = 0.647$ ,  $P < 0.0001$ ) people without a history of MI. The majority of these strong connections were predicated on individuals with a low SYNTAX score ( $r = 0.580$ ,  $P < 0.0001$ ), although this link was shown to be negligible for individuals with a moderately high SYNTAX score ( $r = -0.033$ ). Dogdus M. et al. 19 classified serious CAD as a Gensini score  $\geq 20$ , and he found that GLS cutoff for severe CAD was  $-10$ . (specificity 92.9 percent, sensitivity 88.9 percent). We identified a negative association among GLPSS and syntax score in our research ( $r = 0.534$ ,  $P < 0.000$ ), demonstrating that CAD intensity had a greater impact on GLPSS.

In prediction of coronary affection GLPSS was an independent predictor for single coronary affection (OR = 0.8, 95% CI = 0.647 – 0.989,  $P = 0.04$ ) and multi-vessel affection (OR = 0.487, 95% CI = 0.376 - 0.632,  $P < 0.001$ ), according to Moustafa et al. 14 The segmental LPSS threshold for detecting a dysfunctional LAD artery was  $_{18.3}$  with 91.1 percent specificity and 90 percent sensitivity. The threshold value of segmental LPSS for detecting a dysfunctional LCX artery was  $_{19.3138}$ , with a sensitivity of 95 percentage points and a specificity of 80 percent. The segmental LPSS detection threshold for diseased RCA artery was 18.085, with a sensitivity of 72.9 percent and a specificity of 78.8 percent.

## 6. Limitation

The proportion of persons participating in our trial was undeniably limited and not randomly assigned. For the purpose of comparing the value of GLPSS with the existence and disease severity, we employed solely coronary angiography. Gensini score was not assessed by Intra Vascular Ultrasound (IVUS). In the current investigation, radial, transverse, circumferential, and synchronous strain analyses were not performed.

## 7. Conclusion

In suspected stable AP individuals, 2D STE provides a high sensitivity and specificity for predicting the existence, extent, and intensity of coronary artery disease.

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None to be declared

### Author contribution

Authors contributed equally in the study.

### Conflicts of interest

No conflicts of interest

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