Original Article

Prediction of the Culprit Vessel using Territorial Longitudinal Strain in Non-ST-segment Elevation Myocardial Infarction

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Abstract

Objectives: We evaluated the role of speckle tracking derived territorial longitudinal strain (TLS) to predict the culprit artery in non-ST elevation myocardial infarction (NSTEMI). **Methods:** We evaluated 211 NSTEMI patients scheduled for invasive coronary angiography. Transthoracic conventional and speckle tracking echocardiography was done to assess the left ventricular systolic function, wall motion score index (WMSI), territorial WMSI, and TLS. Coronary angiography within 24 h of hospital admission was used to evaluate the culprit vessel. **Results:** The culprit vessel was most commonly left anterior descending (LAD) "36.5%" and left circumflex (LCX) "35.1%" followed by right coronary (RCA) "28.4%." Multivessel disease patients had higher WMSI and lower global longitudinal strain (GLS) compared to single- and 2-vessel disease ($P < 0.001$). TLS was significantly lower in the culprit vessel territory than in normal and stenosed nonculprit vessels. Moreover, TLS-LCX and TLS-RCA were markedly lower in the stenosed nonculprit territory than normal vessels. Adding TLS to WMSI significantly improved the prediction of the culprit vessel ($P < 0.001$). TLS-LAD, TLS-LCX, and TLS-RCA were independent predictors of the culprit vessel. The optimal cutoff value of TLS-LAD, TLS-LCX, and TLS-RCA was −15.65% (96.1% sensitivity and 100% specificity), −15.65% (94.6% sensitivity and 98.5% specificity), and −16.6% (100% sensitivity and 98.7% specificity), respectively. **Conclusions:** TLS is an independent predictor for identifying the culprit vessel in patients with NSTEMI. Therefore, it should be evaluated during echocardiographic assessment of those patients.

Keywords: Culprit artery, non-ST elevation acute myocardial infarction, speckle tracking, echocardiography, territorial longitudinal strain

Introduction

Acute coronary syndrome (ACS) is a term that describes a group of conditions including unstable angina, ST-segment elevation myocardial infarction (STEMI), and non-ST elevation myocardial infarction (NSTEMI). Nearly more than 7 million people in the world are diagnosed to have ACS.[1] NSTEMI is a major cause of morbidity and mortality, and its incidence increased compared to STEMI in the past two decades.[2] It occurs due to rupture or fissure of coronary atherosclerotic plaques, leading to thrombosis and ischemia.

Management of NSTEMI depends mainly on revascularization using percutaneous coronary intervention (PCI) or coronary artery bypass surgery. Early detection and management of NSTEMI patients have been associated with improved outcomes.[3]

Conventional echocardiography is essential in ACS patients as it is generally used to evaluate the left ventricular (LV) function. It can also evaluate LV regional wall motion abnormalities. Speckle tracking echocardiography (STE) has been widely used as a noninvasive and available tool for evaluating global and regional LV function. STE has the advantage of being relatively operator-independent with lower interobserver variability.[4] Prior studies suggest that using two-dimensional global (GLS) and territorial longitudinal strain (TLS) in assessing LV systolic function may improve echocardiography's diagnostic and prognostic accuracy.[5] STE can help in detecting NSTE-ACS patients who had transmural damage, which had a significant impact on the prognosis.[6] Moreover, it has an important role

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in noninvasively predicting multivessel coronary artery disease in patients with NSTE-ACS. Therefore, we aimed to assess the role of TLS to detect the culprit artery in NSTEMI patients.

Methods

Study population

This single-center, prospective study was conducted at Benha University Hospital. It included NSTEMI patients admitted to our coronary care unit from April 2023 to March 2024. NSTEMI was diagnosed by an increase and decrease in cardiac biomarkers with at least one value above the 99 percentile of the URL in the presence of clinical symptoms or ECG findings suggestive of myocardial ischemia.[2] We excluded patients with prior myocardial infarction, myocardial revascularization, persistent ST-segment elevation, bundle branch block, significant valvular heart disease, AF with rapid ventricular rate, and poor echocardiographic window.

Echocardiography

Transthoracic echocardiography was done using EPIQ equipment (Philips, Erlangen, Germany). Images were obtained with simultaneous ECG recording of the patients in the left lateral position. Images were digitally stored for offline analysis. All echocardiographic measurements were evaluated according to the American Society of Echocardiography guidelines.[7] Modified Simpson's method was used to assess LV ejection fraction (LVEF) by measuring LV end-diastolic and end-systolic volumes at the apical 4- and 2-chamber views. We used the LV 17-segment model to assess the wall motion score index (WMSI). Wall motion was classified as (1) normal, (2) hypokinetic, (3) akinetic, and (4) dyskinetic. WMSI is the result of dividing the sum of each segment score by the number of segments. Territorial WMSI is a result of dividing the sum of segment scores by the number of segments supplied by this territory.

We used two-dimensional STE to evaluate LV myocardial deformation. GLS and TLS were evaluated from the apical views (apical 4-, 2-, and 3-chamber). The software automatically tracked the LV myocardium; however, we corrected the endocardial border manually to improve the image quality. GLS was measured as the average of 17 segments strain measurements. TLS was calculated as the average of the segmental systolic strain within each territory. Apex, basal, mid, and apical anterior, basal, mid, and apical septal segments were related to LAD territory. Apical lateral, basal and mid anterolateral, basal and mid inferolateral segments were related to LCX territory. Basal and mid inferoseptal, basal, mid, and apical inferior segments were related to RCA territory [Figure 1].[8]

Coronary angiography

Coronary angiography was done within the first 24 h of hospital admission. Significant coronary artery disease (CAD) was considered if the luminal stenosis exceeds 50% in the left main (LM) coronary artery or 70% in a major epicardial vessel.[9] The culprit vessel was identified and managed by a skilled and qualified invasive cardiologist according to the guidelines.

Statistical analysis

We used SPSS 20 (SPSS Inc., Chicago, IL, USA) for statistical evaluation. Normally distributed data are expressed as mean ± standard deviation (SD) and compared using Student's *t*-test. Categorical variables are presented as absolute frequencies (percentages) and compared using Fisher's exact and Chi-square tests. Univariate and multivariate logistic regression analyses were done to detect the independent variables for predicting the culprit vessel. A value of $P < 0.05$ indicated statistical significance.

Results

During our study period, 211 NSTEMI patients were evaluated with a mean age of 53.44 ± 6.6 years. The baseline clinical and echocardiographic data are presented in Table 1. The culprit vessels were LAD (36.5%) and LCX (35.1%) followed by RCA (28.4%). The conventional echocardiographic parameters showed that most patients had normal LV volumes (LVESV = 45.26 ± 4.7 ml and LVEDV = 101.37 ± 2.25 ml) and preserved LVEF (53.01 ± 2.86%). WMSI was significantly higher in patients with multivessel than in single and 2-vessel diseases.

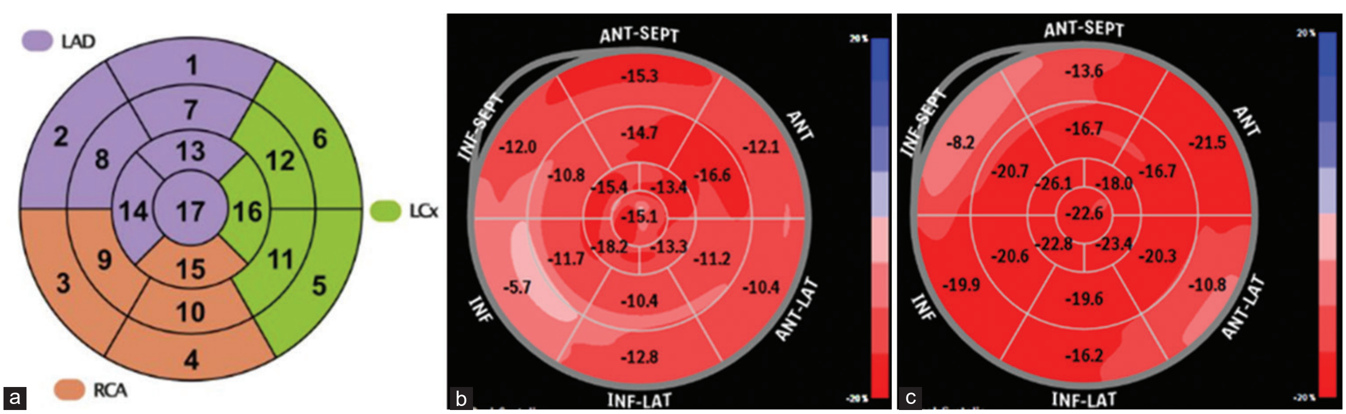

Figure 1: (a) Left ventricular myocardial segments with the corresponding blood supply. (b and c) Bull's eye of left ventricular strain of two patients. (b) patient with 3-vessel disease (LVGLS = −12.9%) and (c) patient with 2-vessel disease (LVGLS = −18.7%)

Table 1: Clinical characteristics and echocardiographic

Characteristics	All patients (n=211)
Age (years)	53.44±6.6
Males	151 (71.6)
HTN	112 (53.1)
DM	124 (58.8)
Dyslipidaemia	142 (67.3)
BMI	31.69±3.43
Smoking	125 (59.2)
Family history	70 (33.2)
ST segment deviation	103 (48.8)
Cardiac arrest	6 (2.8)
Killip class	
1	153 (72.5)
2	58 (27.5)
GRACE score	109.72±11.29
HS-troponin (ng/L)	147.83±63.77
Number of diseased vessels	
Single vessel	103 (48.8)
2-vessels	73 (34.6)
3-vessels	35 (16.6)
Culprit vessel	
LAD	77 (36.5)
LCX	74 (35.1)
RCA	60 (28.4)
LVEDV (mL)	101.37±2.25
LVESV (mL)	45.26±4.7
LVEF (%)	53.01±2.86
E/A	0.76±0.15
E/E`	9.82±1.13
LAVI (mL/m^2)	32.93±1,82
TAPSE (mm)	19.08±1.05
S` (cm/s)	11.91±1.07
PASP (mmHg)	29.78±1.62

Values are presented as *n* (%) or mean±SD. SD=Standard deviation, HTN=Hypertension, DM=Diabetes mellitus, BMI=Body mass index, GRACE: Global registry of acute coronary events, LAD=Left anterior descending, LCX=Left circumflex, RCA=Right coronary artery, LVEDV=Left ventricular end-diastolic volume, LVESV=Left ventricular end-systolic volume, LVEF=Left ventricular ejection fraction, LAVI=Left atrial volume index, TAPSE=Tricuspid annular plane systolic excursion, PASP=Pulmonary artery systolic pressure, GRACE=Global registry of acute coronary events

However, GLS was significantly lower in multivessel disease patients than in single and 2-vessel disease. Moreover, GLS was significantly lower in patients with 2-vessel than single-vessel disease. TLS of the RCA territory (TLS-RCA) was significantly lower in multivessel disease patients than single vessel disease [Table 2].

WMSI can differentiate between the culprit and both normal and stenosed non-culprit vessels. However, it could not differentiate normal from nonculprit stenosis. TLS in all territories (TLS-LAD, TLS-LCX, and TLS-RCA) was significantly lower in the culprit vessel territory than in both stenosed nonculprit (−14.11 ± 4.99 vs. −17.29 ± 0.94%, P < 0.001; −13.78 ± 5.97 vs. −16.38 ± 0.94%, P < 0.001; −14.75 ± 1.00 vs. −17.76 ± 0.51, P < 0.001; respectively) and normal vessels (−14.11 ± 4.99 vs. −17.90 ± 0.60%, P < 0.001; −13.78 ± 5.97 vs. −17.57 ± 3.60%, P < 0.001; −14.75 ± 1.00 vs. −18.05 ± 0.45, P < 0.001; respectively). Moreover, in LCX and RCA territory, TLS-LCX (−16.38 ± 0.94 vs. −17.57 ± 3.60%, P < 0.001) and TLS-RCA (−17.76 ± 0.51 vs. −18.05 ± 0.45%, P < 0.001) were significantly lower in the stenosed nonculprit territory than in normal vessels [Table 3]. Figure 2 demonstrates that TLS was significantly reduced in the territorial supply by the culprit artery.

Table 2: Wall motion score index and speckle tracking data according to the number of diseased vessels

	1 vessel (n=103)	2 vessels (n=73)	3 vessels (n=35)	P
WMSI	1.03±0.06	1.03±0.07	1.10±0.09^{a,b}	<0.001*
WMSI LAD	1.03±0.08	1.05±0.08	1.09±0.15^{a,b}	0.005*
WMSI LCX	1.03±0.07	1.05±0.08	1.10±0.15^{a,b}	0.002*
WMSI RCA	1.02±0.06	1.03±0.07	1.08±0.14^{a,b}	<0.001*
GLS	−17.05±0.53	−16.79±0.38^{a}	−15.98±0.13^{a,b}	<0.001*
TLS-LAD	−16.61±3.64	−16.24±4.00	−16.01±1.66	0.62
TLS-LCX	−16.14±5.82	−16.24±3.98	−15.57±1.21	0.78
TLS RCA	−17.33±1.37	−16.89±1.48	−16.40±2.17^{a}	0.007*

*P<0.05, [a]Siginificant from 1 vessel, [b]Significant from 2 vessels. WMSI=Wall motion score index, LAD=Left anterior descending, LCX=Left circumflex, RCA=Right coronary artery, GLS=Global longitudinal strain, TLS=Territorial longitudinal strain

Adding TLS to WMSI significantly improved the predictive value to detect the culprit vessel, compared to using WMSI alone [Figure 3]. Univariate and multivariate logistic regression analyses were done to detect the independent variables and predict the culprit vessel. TLS-LAD, TLS-LCX, and TLS-RCA were independent predictors of the culprit vessel [Table 4].

A receiver operating characteristic (ROC) curve analysis was done to evaluate the diagnostic accuracy of the various techniques used to predict the culprit vessel. It revealed that TLS and GLS can accurately predict the culprit vessel with improved AUC for TLS in the different territories. AUC was 0.996 (95% confidence interval (CI): 0.991–1.001; P < 0.001), 0.98 (95% CI: 0.962–0.999; P < 0.001), and 0.999 (95% CI: 0.998–1.001; P < 0.001) for TLS-LAD, TLS-LCX, and TLS-RCA, respectively. The optimal cutoff value of TLS-LAD, TLS-LCX, and TLS-RCA was −15.65% (96.1% sensitivity and 100% specificity), −15.65% (94.6% sensitivity and 98.5% specificity), and −16.6% (100% sensitivity and 98.7% specificity), respectively [Table 5 and Figure 4].

Discussion

ACS carries significant morbidity and mortality. NSTEMI represents 70% of the presentation of ACS.[10] Detecting the culprit vessels in NSTEMI patients, especially if multiple vessels were affected, represents a challenge. Intravascular imaging could be complementary to conventional angiography, unveiling the culprit lesion through the detailed analysis of the

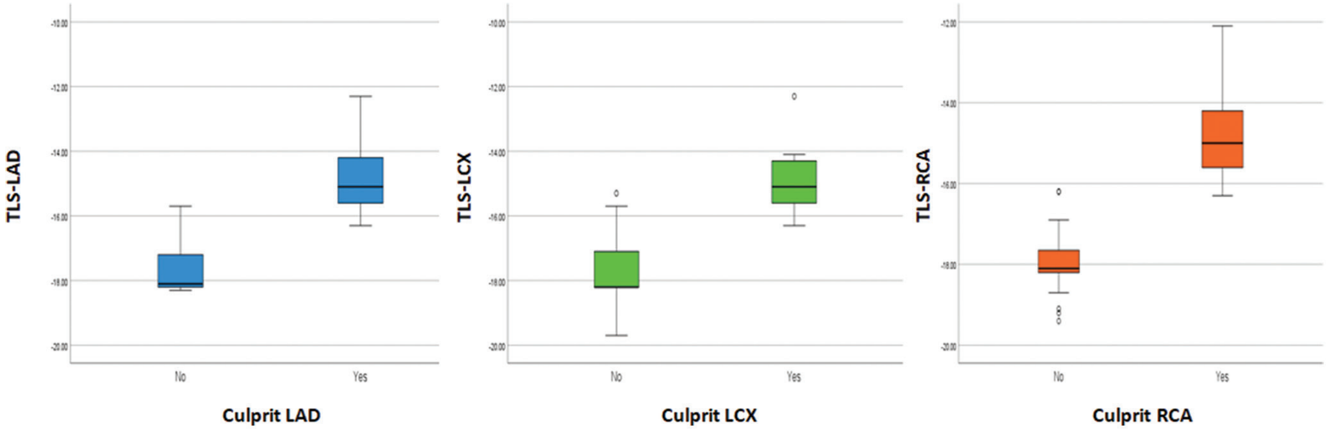

Figure 2: Box plots of the territorial longitudinal strain (TLS) compared to the culprit vessel. TLS-LAD: Territorial longitudinal strain-left anterior descending, TLS-LCX: Territorial longitudinal strain-left circumflex artery, TLS-RCA: Territorial longitudinal strain-right coronary artery

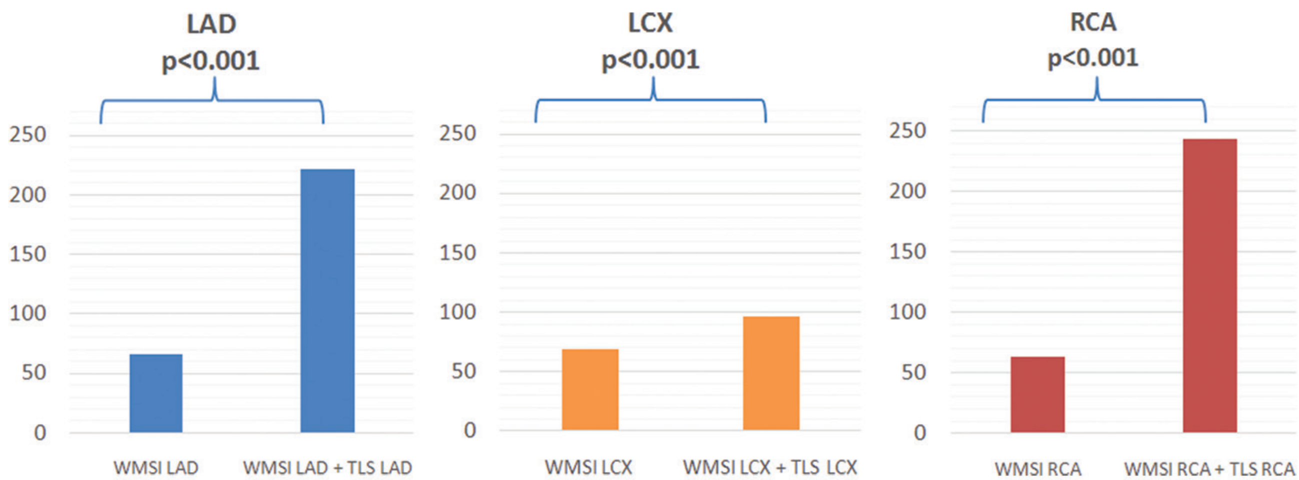

Figure 3: Territorial longitudinal strain + wall motion score index have an added value in predicting the culprit vessel. LAD: Left anterior descending, LCX: Left circumflex, RCA: Right coronary artery, WMSI: Wall motion score index, TLS: Territorial longitudinal strain

Table 3: Coronary-specific WMSI and territorial longitudinal strain in relation to the culprit vessel

Characteristics	LAD no stenosis (*n*=89)	LAD stenosis not culprit (*n*=45)	LAD culprit (*n*=77)	*P*
WMSI-LAD	1.01±0.03	1.02±0.04	1.11±0.13[a,b]	<0.001*
TLS-LAD	-17.90±0.60	-17.29±0.94	-14.11±4.99 a,b	<0.001*
	LCX no stenosis (*n*=108)	**LCX stenosis not culprit (*n*=29)**	**LCX culprit (*n*=74)**	***P***
WMSI-LCX	1.01±0.02	1.04±0.05	1.11±0.13[a,b]	<0.001*
TLS-LCX	-17.57±3.60	-16.38±0.94[a]	-13.78±5.97[a,b]	<0.001*
	RCA no stenosis (*n*=89)	**RCA stenosis not culprit (*n*=62)**	**RCA culprit (*n*=60)**	***P***
WMSI-RCA	1.01±0.03	1.01±0.03	1.10±0.12[a,b]	<0.001*
TLS-RCA	-18.05±0.45	-17.76±0.5[a]	-14.75±1.00 [a,b]	<0.001*

*Indicates significant *P* value, [a]significant from no stenosis, [b]significant from stenosis not culprit. WMSI: wall motion score index, LAD: left anterior descending, LCX: left circumflex, RCA: right coronary, TLS: territorial longitudinal strain

vessel wall and lumen, which could not be available in all cath. Labs. Therefore, other more available yet sensitive imaging modalities, such as STE, are needed.

Prior studies focused on the evaluation of speckle tracking's role in identifying the presence of CAD in NSTE-ACS patients.[11] However, our study aimed to detect the role of global and TLS in predicting the culprit vessel in NSTEMI patients and if they can differentiate multivessel from single or 2-vessel CAD, as it has an essential role in guiding the management of NSTEMI patients.

In our study population, the culprit vessels were most commonly LAD and LCX, followed by RCA. In a study to detect the culprit

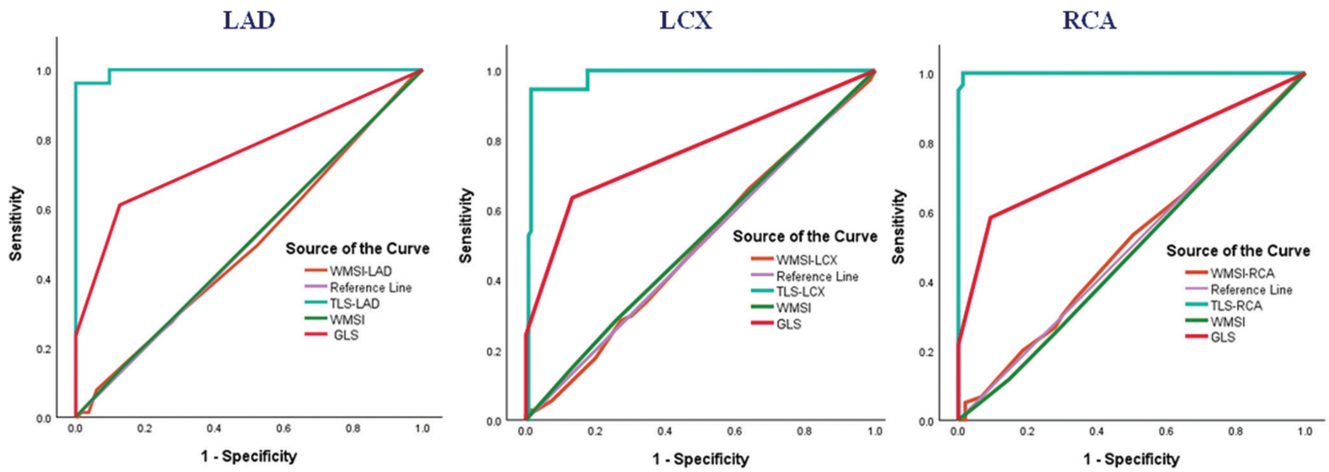

Figure 4: Receiver operating characteristic analysis of the diagnostic accuracy of the various techniques to predict the culprit vessel. LAD: Left anterior descending, LCX: Left circumflex, RCA: Right coronary artery

Table 4: Univariate and multivariate logistic regression analysis to predict the culprit vessel

	Univariate OR	*P*	Multivariate OR	*P*
LAD-culprit				
WMSI-LAD	5.3 (4.6–9.9)	<0.001*	0.85 (0–16.5)	0.97
TLS-LAD	7.9 (7.4–69.7)	<0.001*	72.5 (6.9–758.8)	<0.001*
LCX-culprit				
WMSI-LCX	5.9 (3.6–9.7)	<0.001*	1.7 (0–3.2)	0.88
TLS-LCX	2.7 (2.1–69.7)	0.03*	1.9 (1.3–18.7)	0.031*
RCA-culprit				
WMSI-RCA	5.9 (3.6–9.7)	<0.001*	1.7 (0–3.2)	0.88
TLS-RCA	2.7 (2.01–22.5)	0.03*	1.9 (1.3–18.7)	0.031*

*indicates significant *P* value. WMSI=Wall motion score index, LAD=Left anterior descending, LCX=Left circumflex, RCA=Right coronary artery, TLS=Territorial longitudinal strain

Table 5: Receiver operating characteristic curve analysis to predict the culprit vessel

Variable	Cut-off	AUC	Sensitivity (%)	Specificity (%)	*P*
LAD-culprit					
TLS-LAD	−15.65	0.996	96.1	100	<0.001
GLS	−16.35	0.757	61	87.3	<0.001
LCX-culprit					
TLS-LCX	−15.65	0.98	94.6	98.5	<0.001
GLS	−16.8	0.767	63.5	86.8	<0.001
RCA-culprit					
TLS-RCA	−16.6	0.999	100	98.7	<0.001
GLS	−17.2	0.755	58.3	90.7	<0.001

LAD=Left anterior descending, LCX=Left circumflex, RCA=Right coronary artery, TLS=Territorial longitudinal strain, AUC=Area under the curve

lesions' anatomic distribution in patients with normal ECG non-ST segment elevation myocardial infarction, the LAD was the most common culprit artery, followed by LCX and RCA.[12]

According to our results, the conventional echocardiographic parameters showed that most patients had normal LV volumes. WMSI was significantly increased in multivessel disease patients compared to single and 2-vessel disease. LVEF and WMSI are routinely used to assess patients with ACS; however, we cannot depend on these parameters to detect subtle ischemia. During myocardial ischemia, the subendocardial fibers are first affected even before symptom onset, ECG changes, and wall motion. However, speckle tracking measures strain and can detect changes in the subendocardium, which WMSI cannot assess, mainly evaluating the radial fibers. Radial and circumferential fibers may not be affected or even show compensatory hyperkinesis during subendocardial ischemia.[4]

In our study, GLS was significantly lower in multivessel disease patients than in single and 2-vessel disease patients. Moreover, GLS was significantly lower in patients with 2-vessel compared to single-vessel disease. Therefore, we can efficiently use GLS to differentiate multivessel from 2- and single-vessel disease. Furthermore, it can differentiate between single and 2-vessel CAD. Mghaieth Zghal *et al*.[5] assessed NSTEMI patients and found an association between GLS and CAD severity and lesions' complexity. Similarly, Tibaldi *et al*.[13] reported that LVGLS measurement in NSTE-ACS patients is important in predicting the presence of severe coronary disease. The number of coronary arteries involved has a direct relationship with the degree of LVGLS reduction.

Savage *et al*.[14] assessed the prognostic role of WMSI in STEMI patients. They reported that WMSI can superiorly predict 12-month mortality over LVEF in STEMI patients managed with primary PCI. Giridharan *et al*.[15] recruited patients with unstable angina and normal ejection fraction. They measured their echocardiographic GLS, revealing that GLS worse than −16.1% represents a high probability of having obstructive multivessel CAD. Similarly, Sourabh *et al*.[16] revealed that GLS was very accurate in detecting severe CAD in NSTE-ACS.

Although many studies usually go for GLS when using echocardiographic speckle tracking, we also used TLS, which has more accuracy than GLS, even in predicting the infarct size and extent.[17] According to our study, we can depend on TLS to predict the culprit vessel in NSTEMI patients as it can differentiate the culprit from stenosed nonculprit and normal vessels. Moreover, in LCX and RCA territory, TLS-LCX and TLS-RCA were markedly lower than normal vessels in the stenosed nonculprit territory. Caspar *et al*.[11] reported that TLS has a good localizing value in detecting significant stenosis in the perfusion territories.

Our study revealed that TLS added to WMSI significantly improves the predictive value of evaluating the culprit vessel compared to using WMSI only. This finding proves that combining one of the conventional echocardiographic parameters like WMSI with easy yet sensitive imaging modalities such as GLS and TLS not only gives an idea of the severity of CAD in NSTEMI patients but also makes a very good prediction of the culprit artery in these patients. This finding was similar to Guaricci *et al*.,[18] who evaluated the role of strain echocardiography in predicting the culprit vessel in patients with NSTE-ACS. They reported that TLS added to WMSI enabled them to identify the culprit vessel accurately; however, they did not mention a cut-off value for each artery-specific TLS to label it as the most probable culprit artery.

Our study's univariate and multivariate logistic regression analysis revealed that TLS-LAD, TLS-LCX, and TLS-RCA are independent predictors for predicting the culprit vessel. Multivariate analysis showed that TLS-LAD has a very accurate predictive value. Atici *et al*.[19] revealed that TLS-LAD had a better predictive value of significant CAD in NSTEMI than TLS-CX and TLS-RCA. Their rationale for this finding was that the LAD supplies a larger area of the myocardium and that the poor echogenicity sometimes makes the visualization of RCA and LCX difficult. On the other hand, we detected this difference only when applying multivariant regression analysis, making TLS, especially if combined with WMSI, remarkably accurate in detecting the culprit artery in NSTEMI patients but with greater accuracy if the culprit artery was the LAD.

We used the ROC curve to evaluate the diagnostic accuracy of the various techniques to predict the culprit vessel. It revealed that TLS and GLS can accurately predict the culprit vessel with improved AUC for TLS in the different territories. The optimal cutoff value of TLS-LAD, TLS-LCX, and TLS-RCA was −15.65%, −15.65%, and −16.6%, respectively. Caspar *et al*.[11] when applied an ROC curve analysis to detect the diagnostic performance of TLS in predicting coronary stenosis in the LAD, LCX, or RCA, revealed that the optimal threshold for the average TLS in LAD segments to predict LAD stenosis was −19.4%, yielding a sensitivity of 91% and a specificity of 86%. The optimal threshold for the average TLS in LCX segments to predict LCX stenosis was −16.36%, with 87% sensitivity and 81.4% specificity, while the optimal threshold for the average TLS in RCA segments to predict RCA stenosis was −18%, yielding 82% sensitivity and 78% specificity. It is worth mentioning that these cutoffs represent stenosed coronary arteries and do not specify the culprit artery as they aim to diagnose the presence of CAD. Furthermore, the studied group included patients presented with NSTEMI and unstable angina; these differences could explain their better TLS cutoffs than we revealed in our study.

Conclusions

TLS is an independent predictor for identifying the culprit vessel in patients with NSTEMI. A cut-off value of −15.65% can be used for TLS-LAD and TLS-LCX (96.1, 94.6% sensitivity; 100 and 98.5% specificity, respectively) and −16.60% for TLS–RCA (100% sensitivity and 98.7% specificity) to predict the culprit artery. Therefore, it should be evaluated during the echocardiographic assessment of those patients.

Limitations

Our study has many limitations. First, we studied a small number of populations. Therefore, further studies on a large scale of patients are needed to generalize the data. Second, we could not exclude the inter- and intra-observer variability as echocardiography is an observer-dependent technique. Third, we did not assess the prognostic role of global and TLS in the studied patients. Fourth, the impaired TLS of stenosed nonculprit arteries, which mostly had borderline lesions, should be compared to fractional flow reserve guidance in these arteries to detect if a TLS could be used in guiding the management of those subsets of patients.

Ethical statement

The study was approved by the institutional Ethics Committee of Benha Faculty of Medicine, Approval No. RC12-4-2023.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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