Al-Azhar International Medical Journal

Volume 4 | Issue 12 Article 12

2023
Section: Cardiovascular

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How to Cite This Article

El-Dessouky, Mohammed Helmy; Allam, Ahmed Mohamed; Abdelgalil, Yasser; and ElGamal, Mohamed Saad (2023) "ST-T Changes as a Predictor of Coronary Artery Diseases in Patients with Increased Left Ventricular Mass Index," *Al-Azhar International Medical Journal*: Vol. 4: Iss. 12, Article 12.
DOI: https://doi.org/10.58675/2682-339X.2184

ORIGINAL ARTICLE

ST-T Changes as a Predictor of Coronary Artery Diseases in Patients With Increased Left Ventricular Mass Index

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Abstract

Objectives: **The purpose of this investigation is to assess the relationship between electrocardiographic ST-segment and T wave (ST-T) changes and coronary artery disease (CAD) and coronary lesions in individuals with increased left ventricular mass index (LVMI).**

Methods: **This retrospective cohort study was conducted on 160 patients with elevated LVMI (LVMI >115 g/m^2 in males and 95 g/m^2 in females), patients with hypertrophic cardiomyopathy (HOCM), patients with cardiac amyloidosis, individuals with aortic stenosis (AS), and individuals with athletic myocardial hypertrophy undergoing coronary angiography. Participants were split into two groups. Group I: 80 individuals with increased LVMI and positive strain pattern. Group II: 80 individuals with increased LVMI and no strain pattern.**

Results: **ST-T changes were significant predictors of significant/nonsignificant coronary angiography lesion T-wave inversion was the highest predictor (AUC: 0.726, $P < 0.001$) with an accuracy of 71.9 %, followed by downward sloping depression (AUC: 0.694, $P < 0.001$) with an accuracy of 62.2 % and horizontal depression (AUC: 0.602, P 0.024) with an accuracy of 54.8 %. The number of patients with significant angiography lesions was significantly higher in patients with positive strain patterns compared with those with no strain patterns.**

Conclusions: **Electrocardiographic ST-T changes (T-wave inversion, downward sloping depression, and to a lesser degree horizontal depression) can be used as significant predictors for significant/nonsignificant coronary artery disease angiographic lesions in patients with increased LVMI.**

Keywords: Coronary artery, Increased left ventricular mass index, Strain pattern

1. Introduction

Left ventricular mass (LVM), which is regularly determined by echocardiography, is the weight of the left ventricle (LV) and is assumed to indicate the cumulative impact of blood pressure on the heart. Because of its close relationship to body size, the LVMI links the LV mass to the body surface area (BSA).[1]

They are calculated as follows: LV Mass (g) = 0.8 X (1.04 ((PWd + IVSd + LVEDD)3 − LVEDD3)) + 0.6, LV mass index (g/m^2) = LV mass/BSA.

LVMI is increased in the elderly, obese, hypertensive (HTN), and diabetic mellitus (DM) patients. LVMI is also increased in a variety of cardiac problems including aortic valve stenosis (AS), aortic valve regurgitation, hypertrophic cardiomyopathy, and other problems. It can be a useful echocardiographic predictor of coronary artery disease (CAD) and coronary artery lesions.[2]

With an increase in LVMI, there is an elevation in muscle mass and therefore an elevation in QRS width and abnormal ST-segment and T wave (ST-T)

Accepted 15 August 2023.
Available online 22 January 2024

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https://doi.org/10.58675/2682-339X.2184

changes. The latter is referred to as 'LVH with repolarization abnormality' or 'LVH with strain'.[3]

Typical features of hypertrophy of the left ventricle consist of ST segment deviation in the reverse direction of the QRS complex (discordance) and inversion pattern of T wave, prominent U wave, left axis deviation, and left atrial enlargement[4]

Although there are plenty of electrocardiographic criteria to diagnose strain pattern with left ventricular hypertrophy it is often difficult to differentiate whether the strain pattern present in an electrocardiogram (ECG) is due to LVH or due to myocardial ischemia.[5]

LVH is a potentially curable cause of cardiac mortality in CAD. Nevertheless, LVH will have a significant cumulative effect on CAD if the condition is extremely prevalent.[6]

Coronary angiographic results associated with the existence of ST segment with T wave irregularities consist of significant coronary obstruction (<70 %), a greater number of affected vessels, and occlusion in the left anterior descending coronary artery.[7]

The purpose of this investigation is to assess the relationship between electrocardiographic ST-T changes and CAD and coronary lesions in patients with increased LVMI.

2. Patients and methods

This retrospective cohort research was conducted on 160 patients with high LVMI (LVMI >115 g/m^2 in men and 95 g/m^2 in women) according to the American Society of Echocardiography, individuals with hypertrophic cardiomyopathy (HOCM), Patients with cardiac amyloidosis, patients with aortic stenosis (AS) and patients with athletic myocardial hypertrophy undergoing coronary angiography at the Faculty of Medicine Al-Azhar University from January 2022 to October 2022.

Each patient submitted signed informed permission, and the investigation had been authorized by the Committee of Medical Ethics.

Exclusion criteria were as follows: Patients who have been diagnosed as CAD, individuals with left bundle branch block, a state of cardiogenic shock, acute kidney injury, acute ST-elevated myocardial infarction (STEMI), glomerular filtration rate (GFR) of less than 30 ml/min either with or without pre-existing dialysis, and patients having contraindications to coronary angiography.

The following was performed on all individuals included in this study: a thorough history taking that includes: Patients' risk factors (sex, age, DM, HTN, and smoking), clinical assessment of signs and symptoms regarding the patient, clinical examination that consists of blood pressure (BP), heart rate (HR), body mass index (BMI), and BSA, laboratory investigation including: kidney function tests, patient coagulation profile, virology profile (hepatitis B surface antigen (HBsAg) and hepatitis C virus (HCV) antibodies), other investigations such as 12-lead resting surface electrocardiogram (ECG) (ST segment direction (Horizontal or down sloping), T wave abnormalities (flat or inverted T wave), presence of Sokolow-Lyon Voltage Criteria of LVH, two dimensional transthoracic echocardiography (TTE) using the 2D-TTE machine (Siemens ACUSON NX3 Ultrasound system), the following views were obtained according to the American society of echocardiology: parasternal long-axis view (M-mode of aorta/the left atrium), parasternal long-axis view (M-mode of the left ventricle), and parasternal short-axis view (M-mode at the level of papillary muscles).

Using the mentioned views, the following parameters were obtained: diastolic interventricular septum thickness (IVSd), diastolic left ventricular posterior wall thickness (LVPWd), left ventricular end-systolic dimension (LVESD), and left ventricular end-diastolic dimension (LVEDD).

LV mass could be calculated according to the following equation:

$$\mathrm{LV\,Mass\,(g)} = 0.8\mathrm{X}\left(1.04\left((\mathrm{LVPWd}+\mathrm{IVSd}+\mathrm{LVEDD})^3 - \mathrm{LVEDD}^3\right)\right)+0.6$$

After calculating BSA, LVMI can be calculated as follows:

$$\mathrm{LV\ Mass\ index}\ \left(\mathrm{g}/\mathrm{m}^2\right) = \mathrm{LV\ mass}/\mathrm{BSA}$$

Using the 2D-TTE machine, other views shall be obtained: Apical views of the heart and subcostal views of the heart.

These views were used to identify other echocardiographic parameters: assessment of diastolic function (using mitral valve inflow signals by PW Doppler estimating E/A and E/e' ratio) and assessment of right ventricular systolic function.

The patient also underwent percutaneous coronary angiography (significant CAD showing lesion greater than 50 % stenosis of the left main stem of greater than 70 % in the major coronary vessel).

Patients were split into two groups. Group I: 80 individuals with increased LVMI and positive strain pattern. Group II: 80 individuals with increased LVMI and no strain pattern.

2.1. Statistical analysis

SPSS v26 (IBM Corp., Chicago, IL, USA) was used for statistical analysis. Comparing both groups using an unpaired Student's t-test, the quantitative data were provided as the mean and the standard deviation (SD). When appropriate, qualitative data were provided as frequency and percentage (%) and analyzed using the Chi-square or Fisher's exact test. A two-tailed *P* value less than 0.05 was deemed to be highly significant.

Evaluation of diagnostic performance: Diagnostic specificity, sensitivity, negative predictive value (NPV), positive predictive value (PPV), and receiver-operating characteristic (ROC) curve analysis.

The analysis of ROC curves was used to assess the overall diagnostic performance of each test. A perfect test has a curve that runs from the lower left corner toward the upper left corner and then to the top right corner. The area under the curve (AUC) is used to evaluate the overall performance of a test (where an AUC greater than 50 % indicates acceptable performance and an AUC near 100 % indicates the greatest test performance). Less than 0.5 denotes a very weak model. A value of 0.5 indicates that the model predicts outcomes no better than random chance. Values more than 0.7 denote a good model. Values more than 0.8 denote a robust model.

A value of 1 indicates that the model precisely identifies which group members will and will not experience a particular outcome. The suggested significance level was *P* less than 0.05.

3. Results

No substantial variation in baseline demographic data and clinical data existed among the studied groups (Table 1).

LVM and LVMI were substantially greater in group I compared with patients in group II. However, LVEF was substantially reduced in group I compared with patients in group II (Table 2).

Patients with significant coronary angiography lesions were substantially greater in group I contrasted to group II, but there was no substantial variation among the two groups in the existence of nonsignificant lesions. Normal patients without any coronary angiography lesions were substantially lower in group I contrasted to group II. There was no substantial variation in the number of participants with ectasia between both groups. Patients with tortuosity were substantially greater in group I contrasted with group II. The number of patients with horizontal and downward sloping depression and T-wave inversion was significantly higher in group I compared with group II. However, the number of patients with upward sloping ST-segment changes was significantly higher in group II compared with group I (Table 3).

T-wave inversion (AUC: 0.726, *P* value < 0.001), downward sloping depression (AUC: 0.694, *P* value < 0.001), and horizontal depression (AUC: 0.602, *P* value 0.024) were significant predictors of significant/nonsignificant coronary angiography lesions with an accuracy of 71.9 %, 62.2 %, and 54.8 %.

Table 1. Baseline demographic data and clinical data of the groups under the study.

	Group I (n = 80)	Group II (n = 80)	*P* value
Age (y)			
Mean ± SD	59.04 ± 7.28	57.1 ± 9.61	0.153
Range	41–72	43–80	
Sex			
Male	45 (56 %)	52 (65 %)	0.332
Female	35 (44 %)	28 (35 %)	
HTN			
Yes	66 (83 %)	59 (74 %)	0.251
No	14 (18 %)	21 (26 %)	
DM			
Yes	38 (48 %)	32 (40 %)	0.426
No	42 (53 %)	48 (60 %)	
HF			
Yes	4 (5 %)	2 (2.5 %)	0.682
No	76 (95 %)	78 (97.5 %)	
Chest pain			
Yes	59 (73.75 %)	51 (63.75 %)	0.232
No	21 (26.25 %)	29 (36.25 %)	
AS			
Yes	8 (10 %)	5 (6.25 %)	0.564
No	72 (90 %)	75 (93.75 %)	
HOCM			
Yes	3 (3.75 %)	0	0.245
No	77 (96.25 %)	80 (100 %)	
Smoking			
Yes	43 (54 %)	39 (49 %)	0.635
No	37 (46 %)	41 (51 %)	

AS, Aortic stenosis; DM, Diabetes mellites; HF, Heart failure; HOCM, hypertrophic obstructive cardiomyopathy; HTN, Hypertension; SD, Standard deviation.

Table 2. Echo parameters of the groups under the study.

	Group I (n = 80)	Group II (n = 80)	*P* value
LVM (g)			
Mean ± SD	323.09 ± 81.29	258.88 ± 36.21	<0.001*
Range	217–546	169–354	
LVMI (g/m^2)			
Mean ± SD	168.43 ± 38.39	144.23 ± 23.16	<0.001*
Range	117–261	94–195	
LVEF (%)			
Mean ± SD	59.33 ± 7.96	65.3 ± 5	<0.001*
Range	39–78	54–77	

LVEF, Left ventricular ejection fraction; LVM, Left ventricular mass; LVMI, Left ventricular mass index; SD, Standard deviation.

Table 3. Coronary angiography lesions, ST-T changes, ectasia and tortuosity of the groups under the study.

	Group I (n = 80)	Group II (n = 80)	P value
Coronary Angiography Lesions			
Normal	12 (15 %)	49 (61 %)	<0.001[a]
Non-significant	24 (30 %)	21 (26 %)	0.725
Significant	44 (55 %)	10 (13 %)	<0.001[a]
Ectasia			
Yes	7 (9 %)	4 (5 %)	0.534
No	73 (91 %)	76 (95 %)	
Tortuosity			
Yes	53 (66 %)	38 (48 %)	0.025[a]
No	27 (34 %)	42 (52 %)	
ST-segment changes			
Horizontal depression	45 (56 %)	4 (5 %)	<0.001[a]
Downward sloping depression	35 (44 %)	6 (8 %)	<0.001[a]
Upward sloping	0	38 (47.5 %)	<0.001[a]
No ST changes	0	32 (40 %)	<0.001[a]
T-wave changes			
Inversion	80 (100 %)	4 (5 %)	<0.001[a]
Normal	0	76 (95 %)	

[a] Statistically significant.

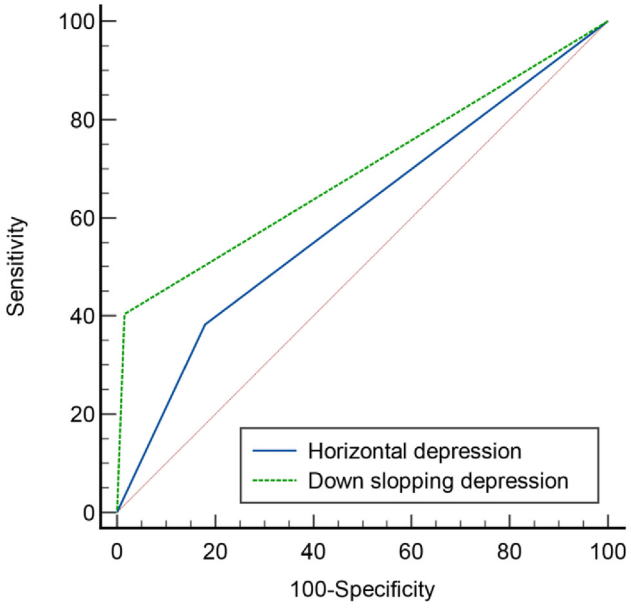

Fig. 1. Receiver-operating characteristic curve analysis of horizontal and downward sloping depression for prediction of coronary angiography lesions.

Upsloping was not a significant predictor of coronary angiography lesions. It showed a sensitivity of 21.2 %, specificity of 72.1 %, and an accuracy of 40.6 %. The strain pattern (AUC: 0.754, P value < 0.001) was a significant predictor of significant/nonsignificant coronary angiography lesions (Table 4 and Figs. 1 and 2).

Downward sloping depression (AUC: 0.706, P value < 0.001), horizontal depression (AUC: 0.593, P value 0.032), and T-wave inversion (AUC: 0.666, P value < 0.001) were significant predictors of significant coronary angiography lesions with an accuracy of 77.5 %, 66.3 %, and 61.8 %. Strain pattern (AUC: 0.683, P value < 0.001) was a significant predictor of significant coronary angiography lesions with an accuracy of 64.3 % (Table 5).

4. Discussion

Coronary heart disease (CHD) and CVD (HT, DM) are both linked to electrocardiographic (ECG) alterations (T-wave inversion and ST segment depression).[8,9] Cardiovascular mortality is at risk due to LVH. An ECG is an insensitive tool for detecting ventricular hypertrophy, which is diagnosed by echocardiogram (Echo) in 20 % of instances and in 50 % of cases of enlarged left ventricular mass (LVM). According to reports, patients with HT may also have LVH, which is associated with a high cardiovascular death rate.[8] Conventional echocardiography is the most popular method for measuring the shape and function of the left ventricle in HTN individuals, and it is widely accessible.[9]

According to the data of this study, it is noted that no statistically substantial variation existed among the two groups concerning age and gender.

A similar investigation was carried out in the Iraqi Center for Heart Diseases Hospital by Raheem et al., which was a cross-sectional study conducted examine the impact of electrocardiographic strain pattern of the left ventricle in individuals with HTN as an indicator for the development of CAD and cerebral vascular accident.[10]

Table 4. Receiver-operating characteristic curve analysis of strain pattern and ST-T changes for prediction of significant/nonsignificant coronary angiography lesions.

	Accuracy	Sensitivity	Specificity	PPV	NPV	AUC	P value
Strain pattern	73.1 %	68.7 %	80.3 %	85 %	61 %	0.754	<0.001[a]
Horizontal depression	54.8 %	38 %	82 %	78 %	45 %	0.602	0.024[a]
Downward sloping depression	62.2 %	40 %	98 %	98 %	50 %	0.694	<0.001[a]
Upward sloping	40.6 %	21.2 %	72.1 %	55.3 %	36 %	–	–
T-wave inversion	71.9 %	69.7 %	75.4 %	82.1 %	60.5 %	0.726	<0.001[a]

[a] Statistically significant.

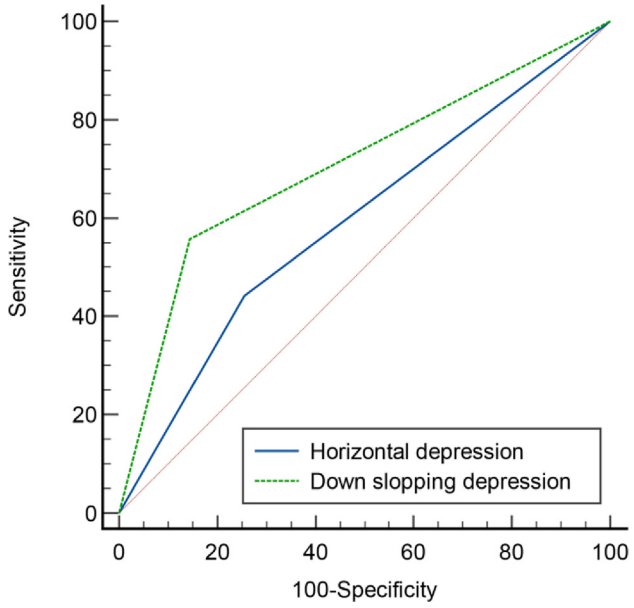

Fig. 2. Receiver-operating characteristic curve analysis of horizontal and downward sloping depression for prediction of significant coronary angiography lesions.

Among the participants, 301 (75.1 %) were reported to have LV strain pattern and 100 (24.9 %) had normal ECG findings. The mean age of participants was 60.07 ± 10.83 years, which is older than our study.[10]

Okin and colleagues conducted different research to examine the relationship between ECG strain and elevated LV mass, regardless of the relationship to CHD. In all, 132 individuals (15 %) had strain, and those with CHD had it more often than those without it (29 %, 51/175 vs. 11 %, 81/711, $P < 0.001$). Individuals without CHD who had typical strain had much greater systolic pressures, a lower level of high-density lipoprotein (HDL) cholesterol, and much greater levels of creatinine in their serum than those without strain. In addition, they were more likely to be male and African-Americans. Individuals with evident CHD who had strain were comparable to individuals without strain in terms of systolic pressure, gender, levels of serum potassium, and sodium. However, they have elevated serum creatinine, a slight reduction in HDL cholesterol, and a higher incidence of DM.[11]

Our study showed that no statistically substantial variation existed among both groups concerning medical history such as DM, HTN, and smoking. Raheem et al. noted that all included patients were hypertensive.[10]

In their investigation on heterogeneous clinical people, Devereux and Reichek observed that LV mass rose linearly with an increase in the occurrence of ST depression accompanying asymmetric T waves and that LV strain performed just in addition to other single or combination ECG-LVH criteria.[12]

It is interesting to note that in that research, LV strain and increasing LV wall thickness were not substantially correlated. ECG-LVH and Echo-LVH were both identified by Sundström et al. as independent indicators for fatalities in older men.[13]

Regarding angiographic findings, our study showed that LVM and LVMI were substantially greater in group I contrasted to patients in group II. But LVEF was significantly lower.

In accordance with our findings, Okin and colleagues reported the following: ECG strain was linked to thicker LV walls, more LV mass, and LV mass index for both BSA or height in individuals either with or without CHD. LV interior dimensions were also larger in individuals with strain than in individuals without CHD. In the two patient groups, strain was linked to an elevated relative wall thickness as well as a larger rise in LV wall thickness compared with cavity dimensions. Although individuals with strain had reduced LV mid-wall efficiency than would be expected for the ESS level, as evidenced by reduced stress-corrected mid-wall reduction, the strain was linked to comparable LV efficiency at the endocardium, as indicated by the fractional shortening, and with comparable amounts of circumferential ESS. Only among individuals without CHD was electrocardiographic strain linked to an elevated wall mass–stress–heart rate product.[11]

Similar to our findings, Roman and colleagues reported decreased LVEF in individuals with strain contrasted to individuals without strain.[14]

Table 5. Receiver-operating characteristic curve analysis of strain pattern and ST-T changes for prediction of significant coronary angiography lesions.

	Accuracy	Sensitivity	Specificity	PPV	NPV	AUC	*P* value
Strain pattern	64.3 %	76.7 %	59.8 %	41.2 %	87.5 %	0.683	<0.001[a]
Horizontal depression	66.3 %	44.2 %	74.4 %	38.8 %	78.4 %	0.593	0.032[a]
Downward sloping depression	77.5 %	55.8 %	85.5 %	58.5 %	84 %	0.706	<0.001[a]
T-wave inversion	61.8 %	76.7 %	56.4 %	39.3 %	86.8 %	0.666	<0.001[a]

[a] Statistically significant.

Individuals with significant coronary angiography lesions were substantially greater in group I contrasted to group II, but there was no substantial variation between the two groups in the existence of nonsignificant lesions. Normal patients without any coronary angiography lesions were substantially lower in group I contrasted to group II.

No substantial variation existed in the number of individuals with ectasia between both groups. Patients with tortuosity were substantially higher in group I contrasted with group II.

Strain pattern is a substantial predictor of coronary angiography lesion (AUC: 0.754, P value < 0.001). It has a sensitivity of 68.7 %, specificity of 80.3 %, PPV of 85 %, and NPV of 61 %.

In line with our findings, Raheem et al. noted that patients with LV strain are associated with coronary findings of CAD.[10]

This result was in line with a study conducted in the United States,[15] which came to the conclusion that LVH and its criteria, such as the criteria of high voltage and strain pattern of LV, are strongly linked to and regarded as a prognostic indicator for CAD.

In accordance with our findings, another investigation, the ALLHAT Study,[16] found that LVH is associated with high-risk CV mortality in individuals with HT regardless of therapy. According to the several forms of research, LVH identified by ECG-LVH, and echocardiography (echo-LVH) is associated with a high risk of cardiovascular disease. Leigh and colleagues observed that ECG-LVH and ECHO-LVH are both CVD predictors. As the detection of LVH by ECG was independent of LV architecture, this suggests that ECG-LVH is a significant electrophysiological marker for cardiac alterations.[17]

It has been shown that the existence of the strain pattern of LV on the 12-lead resting ECG is linked to worse cardiac systolic performance. Like our findings, Okin et al.[11] showed that strain was linked to a greater incidence of LVH determined by LV mass/BSA among individuals either with or without CHD in this group of patients chosen based on ECG LVH by the Cornell product and/or the Sokolow-Lyon voltage.

Additional analyses based on sex-specific LV mass/height 2.7 partitions demonstrated a greater incidence of LVH among individuals with ECG strain, regardless of whether they had CHD (92 % vs. 84 %, $P = 0.226$) or did not have CHD (82 % vs. 70 %, $P = 0.039$).

Regardless of the existence or lack of evident CHD, individuals who had strain had a lower probability of having concentric remodeling or normal LV geometry, a similar incidence of eccentric LVH, and a much greater chance of having concentric LVH than individuals without strain on the rest ECG. Patients with CHD who had strain had a higher prevalence of depressed LV contractility as measured by stress-corrected mid-wall shortening less than 89.2 %, while individuals without CHD had no differences in prevalence based on either the existence or lack of strain.[11]

In 923 white individuals with HTN without clinically evident CHD, Schillaci and colleagues showed poor sensitivity (16 %) as well as elevated specificity (98 %) of strain for LVH, but they did not look at variations in structure and mass of LV in relationship with the strain, which is similar to our findings away from low sensitivity.[18] Roman and colleagues showed that strain was connected to higher LV mass in a sample of 40 individuals with severe isolated aortic regurgitation but no substantial obstructive CHD at angiography which is similar to our findings.[14]

Limitations of the study include that it was a single-center study, and the results may differ elsewhere. There was no follow-up for outcomes such as CAD and mortality. Drugs were not used as an intervention to measure the effects on strain patterns. No correlation analysis was done between the ST-T pattern and coronary angiography. Further clinical studies are needed with multicenter cooperation to validate our findings. We recommend further follow-up for outcome assessment, further interventional studies are required to find the efficacy of drugs and correlation analysis between ST-T and coronary angiography.

4.1. Conclusions

Electrocardiographic ST-T changes (T-wave inversion, downward sloping depression, and to a lesser degree horizontal depression) can be used as significant predictors for significant/non-significant CAD angiographic lesions in patients with increased LVMI.

Financial support and sponsorship

Nil.

Source(s) of support

Nil.

Authors' contribution

M H E-D: idea formulation, data collection, analysis, and writing. A M A: supervision, writing, and revision. Y A: supervision, writing, and revision.

M S E: data collection, supervision, writing, and revision.

Conflicts of interest

Nil.

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