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Association between ST-segment changes in lead aVR and angiographic findings, syntax score, short-term and intermediate outcomes in patients with acute coronary syndrome: A pilot study

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Original Article

Abstract

BACKGROUND: In this study, we aimed to investigate the prognostic implications of lead aVR ST-segment elevation in an initial electrocardiogram (ECG) in patients diagnosed with acute coronary syndrome (ACS). Furthermore, we evaluated the association between electrocardiographic changes in lead aVR and objective measures such as angiographic findings and Syntax score.

METHODS: This retrospective cohort study, conducted as a pilot study, encompassing both a retrospective cross-sectional analysis and a longitudinal follow-up, took place at Chamran Hospital from November 2017 to October 2019. A 6-month follow-up was conducted via phone interviews to assess patient outcomes.

RESULTS: During the study period, there were 76 admissions with the final diagnosis of acute coronary syndrome and lead aVR ST-segment elevation on ECG. ARB intake and the severity of right coronary artery stenosis were significantly higher in patients with STE-aVR ≥1.5 mm. The clinical pathway analysis and 6-month follow-up outcomes concerning ST-segment changes in lead aVR did not reveal statistically significant differences in the distribution of various intervention strategies and clinical events. The overall ST-change was a significant risk factor for 6-month follow-up angiography (OR: 1.10; 95% CI: 1.002 to 1.213) and was also significantly associated with any stenosis in the RCA territory (OR: 1.10; 95% CI: 1.004 to 1.21). There was no significant association between ST-change and other follow-up hospital and angiography outcomes.

CONCLUSION: The findings suggest that medication history, particularly with angiotensin receptor blockers, may shape the observed ST-segment changes in lead aVR. However, further investigation is needed to better understand the clinical implications of these trends.

Keywords: Lead aVR; ST elevation myocardial infarction; Acute coronary syndrome; Angiography; Syntax score; Outcome

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Introduction

Acute Coronary Syndrome (ACS) remains a critical cardiovascular condition associated with significant morbidity and mortality. Due to the time-sensitive nature of this cardiovascular emergency, prompt and accurate decision-making is paramount in emergency medicine, especially for ACS patients. ACS encompasses conditions such as unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI)[1].

Electrocardiogram (ECG) remains an indispensable tool in emergency medicine, providing valuable insights into the cardiac status of patients. The correlation observed in ECG findings plays a critical role in establishing criteria that accurately identify the specific location of a culprit lesion within a designated artery[2].

Lead aVR is a unipolar limb lead augmented for enhanced sensitivity. ST-segment elevation in lead aVR (STE-aVR), in combination with other repolarization changes, has been associated with severe coronary artery lesions in patients with unstable angina or STEMI[3-5]. It is strategically positioned on the lateral aspect of the right arm, directly facing the thinner wall of the right superior surface. This placement is designed to capture information from the right ventricle's outflow tract and the interventricular septum's basal portion[6]. Once underestimated by cardiologists, lead aVR was frequently regarded merely as a provider of reciprocal information, especially when juxtaposed with leads II, V5, and V6, commonly utilized for lead placement verification. Additionally, it was deemed non-adjacent to the other 11 electrocardiographic leads[5,6]. Nevertheless, contemporary research progressively revealed its potential to detect changes across diverse clinical contexts.

Although some studies have indicated a link between STE-aVR and obstruction in the proximal region of the left anterior descending artery (LAD) or three-vessel disease (3VD), other studies found no correlation between post-MI STE-aVR changes and the number of involved vessels or infarct location[6-9].

According to the American Heart Association recommendation, an ECG showing ST depression of more than 0.1 mV in about eight leads and STE-aVR should suggest ischemia occurring because of left main (LM) coronary artery or triple-vessel coronary artery obstruction[10]. Besides, it has been demonstrated that STE-aVR is linked to increased mortality risk[11]. Lead aVR, offering crucial short-term prognostic insights, signifies a worse prognosis in individuals experiencing a first NSTEMI. Considering its association with more advanced coronary artery disease (CAD), an early invasive strategy might offer particular benefits for patients demonstrating ST-segment elevation in lead aVR[11].

Since the diagnostic and prognostic significance of ST-segment depression and STE-aVR for detecting MI remains unclear, it would be beneficial to delineate the ECG characteristics of STEMI patients to identify associated angiographic findings[12]. This study aims to comprehensively understand the prognostic value and clinical implications of lead aVR in the ACS patient population. By elucidating the relationship between electrocardiographic changes in lead aVR and objective measures such as angiographic findings and SYNTAX score, the study seeks to contribute valuable insights that could inform risk stratification, guide therapeutic decisions, and potentially improve patient outcomes in the acute phase and beyond in ACS cases.

Methods

Study design and ethics

This retrospective cohort study, conducted as a pilot study, encompassing both a retrospective cross-sectional analysis and a longitudinal follow-up, took place at Chamran Hospital from November 2017 to October 2019. A 6-month follow-up was conducted via phone interviews to assess patient outcomes. The study received approval from the Ethical Committee of Isfahan University of Medical Sciences (IR.MUI.MED.REC.1399.161). It was conducted according to the ethical principles of the Helsinki Declaration and with patient data confidentiality maintained.

Patients

The research involved adults aged 18 and older who arrived at the emergency department with ACS and displayed STE-aVR on their initial ECG upon admission. STE-aVR was precisely examined twice to reduce possible inaccuracies—initially by the attending physician at the hospital and subsequently by our team. Exclusion criteria included patients who

refused to participate and those with incomplete ECG, diagnostic angiography, or medical records, particularly those with over 20% missing data.

Data collection

The study utilized specific file numbers from the medical records instead of patients' names to ensure patient anonymity. The variables were categorized into different groups to provide clarity. The patient history covered details such as admission date, age, gender, marital status, smoking history, and addiction history. Additionally, it encompassed patients' chief complaints, including but not limited to cardiogenic shock, chest pain, epigastric pain, dyspnea, sweating, nausea, vomiting, headache, palpitations, hand pain, and stereotypical symptoms. Furthermore, it incorporated patients' past medical history, comprising conditions like diabetes, hypertension, hyperlipidemia, chronic kidney disease (CKD), and heart failure (HF), along with any previous cardiological procedures such as percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG). Lastly, it addressed patients' medication history, detailing the use of various drugs such as aspirin (ASA), statins, beta-blockers, angiotensin-converting enzyme inhibitors (ACEI), clopidogrel, angiotensin II receptor blockers (ARB), calcium channel blockers (CCB), anticoagulants, proton pump inhibitors, nitrates, diuretics, and antidepressants.

Initial Admission Evaluations and ECG analysis

Initial admission evaluations consisted of data on systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), troponin (Trop), blood urea nitrogen (BUN), creatinine (Cr), ECG readings, ACS type including unstable angina, NSTEMI, and STEMI, and any concomitant arrhythmia including ventricular fibrillation (VF)/ventricular tachycardia (VT), premature ventricular contraction (PVC), sinus tachycardia, and others.

The study team evaluated the ECGs across all 12 leads, focusing on lead aVR. They identified the magnitude and direction of ST changes, measured in millivolts (mV) with a precision of 0.25. Furthermore, the study team estimated the type of ST depression, including its specific locations such as lateral, anterior, anterolateral, inferior, inferolateral, and diffuse. STE-aVR was categorized into two groups: ≥1.5 millimeters (mm) and <1.5 mm. This categorization was based on clinical guidelines and previous research indicating that ST-segment changes in lead aVR greater than 1.5 mm may indicate significant cardiac pathology. Additionally, Doppler echocardiography findings, such as ejection fraction (EF), were included in this group. EF was divided into categories: under 40%, 40-50%, and over 50%.

Coronary angiography and SYNTAX score analysis

Angiographic findings included the presence of vessel obstructions, thrombolysis in myocardial infarction (TIMI) flow in the vessels (ranging from 0 to 3), and the percentage of initial stenosis (ranging from 0 to 100) for five main territory coronary vessels and graft coronary vessels. These territories included the LM territory (including the LM artery), ramus territory (including the ramus artery), LAD territory (including the LAD artery and diagonal artery), left circumflex coronary (LCX) territory (including the LCX artery and obtuse marginal (OM1-3) arteries), and right coronary artery (RCA) territory (including the RCA, posterior descending artery (PDA), and posterior left ventricular (PLV) artery). In addition, the diagnosis (from no disease to 3VD and left main [3VD + LM]) was extracted. The SYNTAX score was calculated based on the SYNTAX score I calculator.

Clinical outcomes and Follow-up study

Clinical pathways included pharmacotherapy, PCI, CABG, patient-requested discharge, and instances of mortality. In cases where PCI was performed, data regarding the involved vessels, such as LM, LAD, LCX, Ramus, RCA, OM1, OM2, OM3, PDA, Diagonal, PLV, and graft arteries, were also recorded. Follow-up evaluation involved documenting rehospitalization events, identifying the underlying causes, recording accompanying procedures, and noting instances of mortality.

Statistical analysis

Descriptive statistics were employed to summarize the characteristics. Continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on the data distribution. Categorical variables were presented as frequencies and percentages. Based on expected cell frequencies, the association between STE-aVR and each categorical variable was examined

using a chi-square test or Fisher's exact test. Mean or median values between the two groups of STE-aVR were compared for continuous variables using a t-test or Mann-Whitney U test, depending on the data distribution. Multivariate logistic regression with a logit link function was utilized to investigate the association between the ST-segment changes in Lead aVR and the follow-up outcomes (rehospitalization, angiography, CABG, and mortality), in-hospital outcomes (PCI, CABG, medical therapy, and mortality), and angiography outcomes (presence of obstruction in LM vessel, Ramus vessel, LAD territory, LCX territory, and RCA territory). Effect size determination based on different outcomes was extracted by reporting odds ratios (ORs) and 95% confidence intervals (CI) in logistic regression analysis. All statistical analyses were conducted using the SPSS software package. Statistical significance was defined as a p-value below 0.05.

Results

Demographic and characteristics

In this retrospective cohort study of 76 ACS patients with STE-aVR on ECG, 39 had STE-aVR ≥1.5 mm,

Table 1. General and demographics data in patients with ST-segment elevation in lead aVR based on lead aVR ST-segment elevation <1.5mm and ≥1.5mm.

Demographic Variables	ST-Segment Changes in Lead aVR < 1.5mm (n=37)	ST-Segment Changes in Lead aVR ≥ 1.5mm (n = 39)	p-value
Age (mean + SD)	64.49±11.80	66.49±10.11	0.429
Gender n, (%) of female	13 (35.1)	22 (56.4)	0.063
Marital Status n, (%) of married	36 (97.3)	39 (100)	0.487[f]
History of Smoking, n (%)	1 (2.7)	0 (0)	0.690
History of Addiction, n (%)	5 (13.5)	3 (7.7)	0.475[f]
Chief Complaints, n (%)			
- Cardiogenic Shock	1 (2.7)	1 (2.6)	1.0[f]
- Chest Pain	32 (86.5)	35 (92.1)	0.480[f]
- Epigastric Pain	4 (10.8)	2 (5.3)	0.430[f]
- Dyspnea	20 (54.1)	18 (47.4)	0.563
- Sweating	22 (59.5)	23 (60.5)	0.925
- Nausea	16 (43.2)	11 (28.9)	0.197
- Vomiting	11 (29.7)	9 (23.7)	0.554
- Headache	0 (0)	3 (7.9)	0.240
- Palpitations	1 (2.7)	0 (0)	0.493
- Hand Pain	0 (0)	1 (2.6)	1.000
- Stereotypical Symptoms	1 (2.7)	2 (5.3)	1.000
Past Medical History, n (%)			
- Diabetes	11 (29.7)	9 (23.1)	0.510
- Hypertension	15 (40.5)	23 (59.0)	0.108
- Hyperlipidemia	8 (21.6)	4 (10.3)	0.174
- Chronic Kidney Disease	6 (16.2)	13 (33.3)	0.085
- Heart Failure	5 (13.5)	8 (20.5)	0.418
- End-stage renal disease	0 (0)	4 (10.3)	0.116[f]
- Ischemic heart disease	8 (21.6)	15 (39.5)	0.094
Medication history, n (%)			
- Aspirin	15 (40.5)	19 (48.7)	0.474
- Statins	10 (27.0)	12 (30.8)	0.719
- Beta-Blockers	13 (35.1)	14 (35.9)	0.945
- Angiotensin-Converting Enzyme inhibitors	3 (8.1)	5 (12.8)	0.503
- Clopidogrel	4 (10.8)	4 (10.3)	1.000[f]
- Angiotensin II Receptor Blockers	3 (8.1)	13 (33.3)	**0.007**
- Calcium Channel Blockers	2 (5.4)	1 (2.6)	0.610[f]
- Anticoagulants	1 (2.7)	0 (0)	0.487
- Proton Pump Inhibitors	5 (13.5)	8 (20.5)	0.418
- Nitrates	5 (13.5)	8 (20.5)	0.418
- Diuretics	2 (5.4)	7 (17.9)	0.154
- Antidepressants	1 (2.7)	3 (7.7)	0.615

f: p-values are resulted from fisher's exact test.

and 37 had STE-aVR <1.5 mm. A higher proportion of females was observed in the STE-aVR ≥1.5 mm group (p = 0.063), and marital status, smoking, and addiction history showed no significant associations (Table 1). Regarding clinical symptoms, exertional chest pain was the most common symptom but was not significantly linked to ST-segment changes. ARB intake was significantly higher in patients with STE-aVR ≥1.5 mm (p = 0.007), unlike other medications. STE-aVR showed no significant differences in hemodynamics, laboratory parameters, ST depression types, ACS types, arrhythmias, or ejection fraction categories (Table 2).

Angiographic findings

The angiographic analysis showed no significant differences in stenosis severity between patients with STE-aVR ≥1.5 mm and <1.5 mm in the LM, ramus, LAD, diagonal, LCX, OM, and graft territories, nor in TIMI flow and PCI rates. However, RCA stenosis severity differed significantly, with higher significant stenosis in patients with the STE-aVR ≥1.5 mm group (p = 0.007). The type of vessel disease was significantly associated with STE-aVR, showing a higher prevalence of 3VD in the STE-aVR ≥1.5 mm group (p = 0.004). SYNTAX scores did not

Table 2. Examination, laboratory, and clinical data in patients with ST-segment elevation in lead aVR based on lead aVR ST-segment elevation <1.5mm and ≥1.5mm.

Clinical Variables	ST-Segment Changes in Lead aVR < 1.5mm (n=37)	ST-Segment Changes in Lead aVR ≥ 1.5mm (n=39)	p-value
Examination and labs (mean ± SD)			
- Systolic blood pressure (mmHg)	132.65±27.26	143.51±31.12	0.110
- Diastolic blood pressure (mmHg)	81.65±15.79	87.08±20.28	0.199
- Heart rate	86.46±23.03	86.23±15.97	0.960
- Troponin	1.72±0.45	1.83±0.38	0.263
- Blood urea nitrogen	33.69±13.17	37.78±17.02	0.349
- Creatinine	1.14±0.25	1.2±0.32	0.415
ST Depression Type, n (%)			**0.315f**
- Lateral	3 (8.1)	2 (5.1)	
- Anterior	3 (8.1)	0 (0)	
- Anterolateral	9 (24.3)	7 (17.9)	
- Inferior	0 (0)	1 (2.6)	
- Inferolateral	4 (10.8)	3 (7.7)	
- Diffuse	18 (48.6)	26 (66.7)	
ACS type, n (%)			0.216[f]
- Unstable angina	9 (34.6)	4 (14.8)	
- NSTEMI	14 (53.8)	19 (70.4)	
- STEMI	3 (11.5)	4 (14.8)	
-- extensive	0 (0)	1 (3.7)	
-- inferior	0 (0)	1 (3.7)	
-- posterior	1 (3.8)	0 (0)	
-- lateral	1 (3.8)	0 (0)	
-- antroseptal	1 (3.8)	1 (3.7)	
Arrhythmia, n (%)			0.287[f]
- none	19 (73.1)	16 (59.3)	
- VF/VT	1 (3.8)	1 (3.7)	
Non sustained VT	1 (3.8)	0 (0%)	
- PVC	4 (15.4)	2 (7.4)	
- PAC	0 (0)	1 (3.7)	
- Sinus tachycardia	0 (0)	3 (11.1)	
EF, n (%)			0.610
- EF = 0-40%	12 (32.4)	13 (33.3)	
- EF = 40-50%	10 (27.0)	14 (35.9)	
- EF = 50-70%	15 (40.5)	12 (30.8)	

Abbreviations: ACS: acute coronary syndrome; NSTEMI: non–ST elevation myocardial infarction; STEMI: ST-segment elevation myocardial infarction; VF: Ventricular fibrillation; VT: Ventricular tachycardia; PVC: premature ventricular contractions; PAC: premature atrial contractions; EF: ejection fraction.
f: p-values are resulted from fisher's exact test.

differ significantly between groups (Supplementary Table 1).

Clinical outcomes

Rehospitalization was higher in the STE-aVR ≥1.5 mm group (41.7% vs. 24.3%) during the 6-month follow-up, but this was not statistically significant (Table 3). ST-change in lead aVR was a significant risk factor for RCA stenosis, with a 10% increase in odds per unit increase in ST-change (OR: 1.10; 95% CI: 1.004 to 1.21). ST-change in lead aVR had a significant association with angiography in the 6-month follow-up (OR: 1.10; 95% CI: 1.002 to 1.213). No other significant associations were found with hospital or follow-up outcomes (Table 4).

Discussion

The present study aimed to explore the diagnostic significance of STE-aVR among ACS patients. Additionally, it aimed to study the correlation between this elevation and angiographic findings, SYNTAX score, and short-term and intermediate outcomes. In this investigation, our discernment of medication history, particularly the use of ARBs, emerged as a potential factor influencing the observed ST-segment alterations in lead aVR. The study underscored the

Table 3. Follow-up data in patients with ST-segment elevation in lead aVR based on lead aVR ST-segment elevation <1.5mm and ≥1.5mm.

	ST-Segment Changes in Lead aVR <1.5mm (n=37)	ST-Segment Changes in Lead aVR ≥1.5mm (n=39)	p-value
In-hospital clinical pathway, n (%)			
- Pharmacotherapy	7 (18.9)	5 (12.8)	0.466
- PCI	16 (43.2)	14 (35.9)	0.513
- CABG	6 (16.2)	8 (20.5)	0.629
- Patient-Requested Discharge	8 (21.6)	12 (30.8)	0.365
- In-hospital mortality	2 (5.4)	5 (12.8)	0.432[f]
6-month Follow-up, n (%)			
- Rehospitalization Events	9 (24.3)	15 (41.7)	0.278[f]
- Accompanying Procedures (angiography and CABG)	7 (20.6)	15 (44.1)	0.092[f]
- Mortality, n (%)	4 (11.8)	3 (8.8)	1.000

Abbreviations: PCI: percutaneous coronary intervention; CABG: coronary artery bypass grafting.
f: p-values are resulted from fisher's exact test.

Table 4. Association between ST-segment change and follow-up, in hospital, and angiography outcomes by the logistic regression model.

Outcomes		Odds ratio	95% CI Lower	Upper	P-value
Follow-up Outcomes					
Rehospitalization		1.02	.962	1.086	0.479
Procedure	**CABG** [a]	0.98	0.878	1.070	0.653
	Angiography [a]	**1.10**	**1.002**	**1.213**	**0.044***
Mortality		0.98	0.882	1.094	0.743
In hospital Outcomes					
PCI Therapy		1.04	0.985	1.103	0.139
CABG Therapy		1.00	0.939	1.072	0.922
Medical Therapy		0.93	0.842	1.031	0.172
Death		1.07	0.998	1.154	0.057
Angiography Outcomes					
LM Vessel		1.02	0.965	1.080	0.469
Ramus Vessel		1.02	0.951	1.086	0.636
Territory LAD		1.02	0.940	1.108	0.627
Territory LCX		1.07	0.976	1.169	0.150
Territory RCA		**1.10**	**1.004**	**1.21**	**0.042***

Abbreviations: PCI: percutaneous coronary intervention; CABG: coronary artery bypass grafting; LM: Left Main; LAD: Left Anterior Descending; LCX: Left Circumflex Coronary; RCA: Right Coronary Artery; CI: confidence interval.
* p-value < 0.05 is statistically significant.
[a] p-values are resulted from multinomial logistic regression.

necessity for a nuanced comprehension of clinical presentations linked to ST-segment changes in lead aVR and the severity of CAD. The results imply an association between STE-aVR and more severe CAD, notably prevalent in patients with 3VD.

Furthermore, our findings indicated a potential correlation between RCA stenosis and elevated ST-segments in lead aVR, emphasizing the potential impact of medication history, particularly ARBs, on the observed alterations in this electrocardiographic parameter. The comprehensive examination of clinical variables, including hemodynamic parameters, laboratory outcomes, ST depression types, ACS classification, arrhythmias, and EF, demonstrated an absence of significant differences among patients with varying STE-aVR. These outcomes suggest that the influence of these clinical variables on the presence of ST-segment changes in lead aVR may not be substantial. This underscores the imperative for additional investigations and larger sample sizes to validate these findings. The angiographic findings spanning different coronary territories and vessel diseases offer valuable insights into their association with STE-aVR. Although several territories exhibited no significant differences, noteworthy associations were observed in the RCA territory regarding vessel disease severity. This underscores the necessity for further exploration of these specific coronary parameters.

The underlying mechanisms behind ST-segment alterations in lead aVR are intricate and not completely elucidated. Proposed mechanisms for ST-segment elevation in lead aVR encompass transmural basal septal ischemia, transmural ischemia of the right ventricular outflow tract, and reciprocal changes leading to ST-segment depression in lateral leads[13].

Certain studies have suggested a link between STE-aVR and blockages in the proximal segment of the LAD or the presence of 3VD[8,9]. In NSTEMI patients, STE-aVR ≥0.5 mm was a useful predictor of LM/3VD (sensitivity 78%, specificity 86%). Furthermore, they found that STE-aVR was the strongest predictor of adverse events at 90 days in NSTEMI subjects[14,15]. In another study, the sensitivity and specificity were reported as 77% and 65%, respectively, with a cutoff point of 0.5 mm for detecting LM/3VD. However, when a higher cutoff point (>1.5 mm) was used, sensitivity decreased to 14%, while specificity increased to 98%[16]. A case report of a 51-year-old man with chest pain also underscores the significance of lead aVR as an electrocardiographic indicator for LM or multi-vessel disease[17]. Due to its correlation with more severe CAD, an early invasive approach could be particularly beneficial for patients exhibiting STE-aVR. However, other studies found no correlation between post-MI STE-aVR changes and the number of vessels involved or infarct location[10,11]. Harhash et al. noted that STE-aVR is correlated with acute coronary thrombotic occlusion in only 10% of all cases[18].

In a cross-sectional study with 472 subjects, reported by Sheibani et al., there was no significant association between STE in lead aVR and angiography results in patients presenting with ACS[19]. In addition, a single cohort study conducted by Nabati et al. revealed no significant difference between the two groups (STE-aVR compared to non-STE-aVR) concerning LM/3VD. However, they noted that STE in lead aVR was associated with the severity of atherosclerosis, although their sample size was small[20]. Alherbish et al. observed that STE-aVR, while not able to show injury to the myocardium, was still correlated with a previous history of CAD. They also noted that STE-aVR in inferior MI is correlated with a two-fold increase in mortality rates[21]. Kukla et al. also showed a significant association between STE-aVR and poor prognosis of inferior wall STEMI. They noted that such changes are correlated with higher in-hospital mortality regardless of the treatment[22]. A systematic review and meta-analysis that included 52,175 patients reported that the likelihood of in-hospital and 90-day mortality was elevated in patients with ACS and STE-aVR[8]. Lead aVR was found to be a significant predictor of short-term prognosis in the study of 525 subjects with a first acute MI and no STE-aVR. The indication of a poorer outcome associated with aVR-STE appears to be linked to more severe CAD, which underscores the potential benefits of an early invasive approach for patients presenting with this particular electrocardiographic finding[11]. These studies concluded that lead aVR provides crucial short-term prognostic insights, indicating a poorer prognosis in individuals with a first NSTEMI. By contrast, it has been reported that the presence of aVR-STE did not significantly change the number

of in-hospital mortalities[7]. Furthermore, a separate study with a substantial sample size (n = 15,315) found no significant association between STE-aVR and 30-day mortality in the adjusted model[23].

Adar et al. delineated the connection between ST-segment shifts in lead aVR and coronary complexity among patients with ACS. Their findings suggested that in ACS patients, the presence of ST-segment elevation or depression ≥ 1 mm in the lead aVR could signify coronary complexity[24]. Our study showed no correlation between STE-aVR and SYNTAX score.

Conclusion

In summary, our results suggest that medication history, particularly involving ARBs, might influence the observed STE-aVR. Examining the clinical pathway and 6-month follow-up results concerning STE-aVR did not uncover statistically significant variations in the distribution of different intervention strategies and clinical events. These results imply consistency in management and outcomes across both groups, underscoring the necessity for further research to identify potential indicators of distinct clinical pathways and long-term outcomes. However, further investigation is warranted to fully grasp these trends' clinical implications.

Limitations and strength

The study has several limitations that should be considered when interpreting the results. Firstly, its single-center nature may restrict the generalizability of the findings. Secondly, being retrospective introduces the possibility of bias and constrains the establishment of causality. Thirdly, the relatively small sample size might curtail the study's statistical power. Lastly, the study did not delve into the long-term prognostic implications of ST-segment changes in lead aVR, warranting further investigation. The suggestion for future studies with a longer follow-up period, larger sample sizes, and multi-center designs enhances the potential for capturing more comprehensive and extended-term outcomes.

Some strengths were identified in our study, including the novel contribution to existing literature through an emphasis on medication history, specifically with ARBs. Insights gained from the findings shed light on the potential role of medication history, particularly with ARBs, in influencing observed ST-segment changes in lead aVR. Given the established correlation between ST-segment changes in lead aVR and poor prognosis in MI patients, the exploration of the risks and benefits of ARBs in HF patients, especially those more susceptible to MI, emerges as a pertinent clinical consideration. Furthermore, evaluating the relationship between STE-aVR and coronary complexity, as assessed by the SYNTAX score, contributes depth to the understanding of ACS.

Conflict of interests

The authors declare no conflict of interest.

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Author's Contributions

Concept = AA and MS; Design = AA and RZ; Literature search = HM, BD, SMM, and RAB; Data acquisition = AA, RZ, BD, SMM, and RAB; Data analysis = BD and RAB; Statistical analysis = BD and RAB, Manuscript preparation = HM, BD, and SMM; Manuscript editing and manuscript review = AA, MS, RZ, and RAB.

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Supplementary Table 1. Angiographic findings and syntax score in patients with ST-segment elevation in lead aVR based on lead aVR ST-segment elevation <1.5mm and ≥1.5mm.

Angiographic Findings	ST-Segment Changes in Lead aVR <1.5mm (n=37)	ST-Segment Changes in Lead aVR ≥1.5mm (n=39)	p-value
LM artery territory			
- Stenosis			0.134[f]
-- 0-49%	26 (70.3)	29 (74.4)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	5 (13.5)	1 (2.6)	
-- 90-99%	5 (13.5)	9 (23.1)	
-- 100%	1 (2.7)	0 (0)	
- TIMI			0.497
-- 0	1 (2.7	0 (0)	
-- 1	0 (0)	1 (2.6)	
-- 2	0 (0)	2 (5.1)	
-- 3	36 (97.3)	36 (92.3)	
- PCI; n (%)	0 (0)	1 (2.6)	1.000[f]
Ramus artery territory			
- Stenosis			0.814[f]
-- 0-49%	32 (86.5)	32 (82.1)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	1 (2.7)	2 (5.1)	
-- 90-99%	2 (5.4)	4 (10.3)	
-- 100%	2 (5.4)	1 (2.6)	
- TIMI			1.000[f]
-- 0	2 (5.4)	3 (7.7)	
-- 1	0 (0)	0 (0)	
-- 2	2 (5.4)	2 (5.1)	
-- 3	33 (89.2)	34 (87.2)	
- PCI; n (%)	0 (0)	0 (0)	-
LAD Territory; n (%)			
LAD artery			
- Stenosis			0.464[f]
-- 0-49%	11 (29.7)	8 (20.5)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	8 (21.6)	5 (12.8)	
-- 90-99%	15 (40.5)	20 (51.3)	
-- 100%	3 (8.1)	6 (15.4)	
- TIMI			0.713[f]
-- 0	4 (10.8)	3 (7.7)	
-- 1	2 (5.4)	3 (7.7)	
-- 2	8 (21.6)	5 (12.8)	
-- 3	23 (62.2)	28 (71.8)	
- PCI	7 (18.9)	3 (7.7)	0.186[f]
Diagonal artery			
- Stenosis			0.756[f]
-- 0-49%	23 (62.2)	20 (51.3)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	1 (2.7)	2 (5.1)	
-- 90-99%	10 (27.0)	12 (30.8)	
-- 100%	3 (8.1)	5 (12.8)	
- TIMI			1.000[f]
-- 0	3 (8.1)	3 (7.7)	
-- 1	0 (0)	1 (2.6)	
-- 2	3 (8.1)	4 (10.3)	
-- 3	31 (83.8)	31 (79.5)	
- PCI	1 (2.7)	2 (5.1)	1.000[f]
LCX Territory; n (%)			
LCX artery			
- Stenosis			0.084[f]
-- 0-49%	17 (45.9)	8 (20.5)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	3 (8.1)	6 (15.4)	
-- 90-99%	14 (37.8)	23 (59.0)	
-- 100%	3 (8.1)	2 (5.1)	
- TIMI			0.221[f]

Angiographic Findings	ST-Segment Changes in Lead aVR <1.5mm (n=37)	ST-Segment Changes in Lead aVR ≥1.5mm (n=39)	p-value
-- 0	3 (8.1)	2 (5.1)	
-- 1	0 (0)	2 (5.1)	
-- 2	7 (18.9)	13 (33.3)	
-- 3	27 (73.0)	22 (56.4)	
- PCI	3 (8.1)	7 (17.9)	0.311[f]
OM 1 artery			
- Stenosis			0.447[f]
-- 0-49%	27 (73.0)	23 (59.0)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	1 (2.7)	4 (10.3)	
-- 90-99%	8 (21.6)	11 (28.2)	
-- 100%	1 (2.7)	1 (2.6)	
- TIMI			0.785[f]
-- 0	2 (5.4)	1 (2.6)	
-- 1	0 (0)	0 (0)	
-- 2	3 (8.1)	5 (12.8)	
-- 3	32 (86.5)	33 (84.6)	
- PCI	1 (2.7)	2 (5.1)	1.000[f]
OM 2 artery			
- Stenosis			0.917[f]
-- 0-49%	32 (86.5)	35 (89.7)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	1 (2.7)	1 (2.6)	
-- 90-99%	3 (8.1)	2 (5.1)	
-- 100%	1 (2.7)	1 (2.6)	
- TIMI			0.555[f]
-- 0	2 (5.4)	1 (2.6)	
-- 1	0 (0)	0 (0)	
-- 2	2 (5.4)	1 (2.6)	
-- 3	33 (89.2)	37 (94.9)	
- PCI	0 (0)	0 (0)	-
OM-3 artery			
- Stenosis			-
-- 0-49%	37 (100)	39 (100)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	0 (0)	0 (0)	
-- 90-99%	0 (0)	0 (0)	
-- 100%	0 (0)	0 (0)	
- TIMI			0.487[f]
-- 0	1 (2.7)	0 (0)	
-- 1	0 (0)	0 (0)	
-- 2	0 (0)	0 (0)	
-- 3	36 (97.3)	39 (100)	
- PCI	0 (0)	0 (0)	-
RCA Territory; n (%)			
RCA artery			
- Stenosis			**0.007[f]**
-- 0-49%	17 (45.9)	5 (12.8)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	3 (8.1)	11 (28.2)	
-- 90-99%	10 (27.0)	12 (30.8)	
-- 100%	7 (18.9)	11 (28.2)	
- TIMI			0.244
-- 0	4 (10.8)	10 (25.6)	
-- 1	1 (2.7)	3 (7.7)	
-- 2	4 (10.8)	2 (5.1)	
-- 3	28 (75.7)	24 (61.5)	
- PCI; n (%)	5 (13.5)	6 (15.4)	0.817
PDA artery			
- Stenosis			0.056[f]
-- 0-49%	32 (86.5)	35 (89.7)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	0 (0)	3 (7.7)	
-- 90-99%	13.5	1 (2.6)	
-- 100%	0 (0)	0 (0)	
- TIMI			0.228[f]

Angiographic Findings	ST-Segment Changes in Lead aVR <1.5mm (n=37)	ST-Segment Changes in Lead aVR ≥1.5mm (n=39)	p-value
-- 0	1 (2.7)	0 (0)	
-- 1	0 (0)	0 (0)	
-- 2	1 (2.7)	0 (0)	
-- 3	35 (94.6)	39 (100)	
- PCI; n (%)	0 (0)	0 (0)	-
PLV artery			
- Stenosis			0.367[f]
-- 0-49%	33 (89.2)	37 (94.9)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	0 (0)	1 (2.6)	
-- 90-99%	2 (5.4)	0 (0)	
-- 100%	2 (5.4)	1 (2.6)	
- TIMI			0.163[f]
-- 0	2 (5.4)	0 (0)	
-- 1	0 (0)	1 (2.6)	
-- 2	1 (2.7)	0 (0)	
-- 3	34 (91.9)	38 (97.4)	
- PCI; n (%)	2 (5.4)	0 (0)	0.234[f]
Graft Territory			
- Stenosis			0.494[f]
-- 0-49%	37 (100)	37 (94.9)	
-- 50-69%	0 (0)	0 (0)	
-- 70-89%	0 (0)	0 (0)	
-- 90-99%	0 (0)	0 (0)	
-- 100%	0 (0)	2 (5.1)	
- TIMI			0.494[f]
-- 0	0 (0)	2 (5.1)	
-- 1	0 (0)	0 (0)	
-- 2	0 (0)	0 (0)	
-- 3	37 (100)	37 (94.9)	
- PCI; n (%)	0 (0)	1 (2.6)	1.000[f]
Type of vessel disease; n (%)			**0.004[f]**
- none	4 (10.8)	0 (0)	
- 1VD	6 (16.2)	1 (2.6)	
- 2VD	5 (13.5)	3 (7.7)	
- 3VD	12 (32.4)	27 (69.2)	
- 1VD+LM	0 (0)	1 (2.6)	
- 2VD+LM	2 (5.4)	0 (0)	
- 3VD+LM	8 (21.6)	7 (17.9)	
Syntax score			0.225
- Low (0-16)	10 (30.3)	5 (14.3)	
- Intermediate (17-22)	5 (15.2)	9 (25.7)	
- High (23-40)	18 (54.5)	21 (60.0)	

Abbreviations: TIMI: The Thrombolysis in Myocardial Infarction (TIMI) Score; PCI: percutaneous coronary intervention; LM: Left Main; LAD: Left Anterior Descending; LCX: Left Circumflex Coronary; RCA: Right Coronary Artery; OM: Obtuse Marginal; PDA: Posterior Descending Artery; PLV: Posterior Left Ventricular. VD: vessel disease.