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RESEARCH ARTICLE

aVR ST-segment changes and prognosis of ST-segment elevation myocardial infarction

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Abstract

Background: Clinical importance of aVR lead-related changes in predicting the prognosis of acute myocardial infarction remains uncertain. The present study aimed to assess the value of ST-segment changes in aVR lead and the outcome and sequels of the first episode of acute ST-segment elevation myocardial infarction.

Methods: This prospective cohort study was conducted on patients suffering first episode of ST-segment elevation myocardial infarction and underwent percutaneous coronary intervention. Information was collected through hospital-recorded files reading. The electrocardiogram (ECG) was taken from the patients upon entering the hospital and followed-up for 30 days to assess cardiovascular complications.

Results: In patients with anterior STEMI, with the use of multivariate analysis, admission aVR ST elevation ≥1 mm was found to be a strong and independent predictor of major cardiovascular adverse events (MACE) within 30 days of discharging (*P* value for trend .002). In patients with inferior (± RV) ST-segment elevation myocardial infarction (STEMI), with the use of multivariate analysis, admission aVR ST depression ≥1 mm was found to be a strong and independent predictor of MACE within 30 days of discharging (*P* value for trend .01).

Conclusion: In patients with anterior STEMI, admission aVR STE ≥1 mm was found to be a strong and independent predictor of MACE within 30 days of discharging. On the other hand, in patients with inferior STEMI, aVR ST depression ≥1 mm was found to be a strong and independent predictor of MACE within 30 days of discharging.

KEYWORDS
aVR ST-segment change, myocardial infarction, prognosis

1 | INTRODUCTION

Lead aVR on electrocardiogram as a unipolar and augmented limb lead can present valuable and specific information from the right upper portion of the heart. It is well established that ST-segment changes in the aVR lead are associated with three coronary vessels involvement or left main disease in patients suffering acute coronary syndrome (ACS).[1,2] These changes have been accepted as a main source for additional information to determine culprit lesions and thus for predicting poor prognosis especially in patients with ST-segment elevation myocardial infarction (STEMI).[3,4] The pathological change in ST segment in lead aVR is closely linked to anterior STEMI; however, these segmental changes among those with inferior STEMI remain inconsistent.[5] Additionally, some studies could only determine the value of ST-segment

changes in the aVR for differentiation of infarctions due to stenosis in left circumflex artery (LCX) and right coronary artery (RCA) arteries with no extra data on the influence of the site of occlusion in these arteries.[6] According to the literature, the occurrence of ST-segment elevation in lead aVR can be also very valuable to predict poor prognosis following coronary revascularization.[7,8] In this regard, it has been clearly shown that the grade of ST-segment changes in this lead significantly correlates with impaired reperfusion as well as coronary restenosis following percutaneous coronary intervention (PCI).[9,10]

Although in previous studies, the link between ST-segment elevation in the aVR lead and the risk for left main artery lesions or three-vessel coronary disease has been well understood, it is important to note that clinical importance of other lead-related changes such as ST-segment depression or T-wave inversion in the aVR in predicting the prognosis of myocardial infarction remains uncertain and requires further investigations. Hence, the present study aimed to assess the value of ST-segment changes in aVR lead and the outcome and sequels of the first episode of acute STEMI.

2 | MATERIALS AND METHODS

This prospective cohort study was conducted on patients suffering first episode of STEMI who were referred to Al-Zahra hospital in Shiraz between January 2018 and January 2019 and underwent PCI. This study was approved by the Ethics Committee of the Shiraz University of Medical Sciences (IR.SUMS.MED.REC.1398.552). In this regard, the changes in favor of bundle branch block, Wolff-Parkinson-White syndrome, or left ventricular hypertrophy on electrocardiogram, previous history of coronary artery bypass grafting or PCI, history of taking drugs that affect the electrocardiography patterns such as digoxin or history of acute liver or kidney failure were considered as the exclusion criteria. In this study, information was collected through hospital-recorded files reading, so that patients who were registered with the code 247 in the emergency and cathlab of the hospital were included in the study, and their files were examined to extract information. For each patient included in the study, general characteristics such as age, sex, previous history of hypertension, diabetes mellitus, hyperlipidemia, smoking, obesity, and family history of heart diseases as well as laboratory parameters were assessed. Furthermore, the ECG was taken from the patients upon entering the hospital using Cardiax software and the following information were extracted: (a) Max of ST elevation or the maximum voltage of ST elevation and its related lead (MAX STE), (b) sum of ST elevation or total voltage of ST elevation in leads that had ST elevation (SUM STE), (c) ST-wave in aVR lead or AVRST (ST elevation-depression voltage), (d) T-wave in aVR lead or AVRT (upright or invert wave voltage), (e) ST-segment resolution (percentage of normalization of ST elevation in repeated ECG), (f) MI territory (categorized as anterior, inferior, and inferior/right ventricle), (g) Selvester Score (which estimates the size and location of myocardial scar in the left ventricle and calculated based on based on Q- or R-wave duration, R- or S-wave amplitude and R/Q or R/S amplitude ratios as previously described [11]), and (h) Aldrich score (which was calculated using the following formula (anterior STEMI: $3 \times (1.5[\text{number of leads with ST}\uparrow] - 0.4)$ and inferior STEMI: $3 \times (0.6[\sum \text{ST}\uparrow \text{ II, III, aVF}] + 2.0)$). Then, all patients underwent echocardiography assessment to determine left ventricular ejection fraction (LVEF). After performing PCI procedure, the following coronary angiography parameters were also determined for all subjects: (a) Time to reperfusion (defined as duration between onset of pain and reopening of coronary occlusion or, if unsuccessful, to end of procedure), (b) door to device time (defined as duration between the time of entering emergency room and the time of angioplasty), (c) infarct-related artery, (d) final and initial TIMI flow (graded as 0 = absence of any antegrade flow beyond a coronary occlusion, 1 = faint antegrade coronary flow beyond the occlusion with incomplete filling of the distal coronary bed, 2 = delayed or sluggish antegrade flow with complete filling of the distal coronary bed, or 3 = normal flow which fills the distal coronary bed completely), (e) Thrombus Burden (graded as 0 = no thrombus, 1 = possibility of thrombus, 2 = small thrombus with diameter less than 1/2 of vessel diameter, 3 = moderate thrombus with diameter greater than 1/2 vessel diameter, 4 = large thrombus with diameter greater than 2 diameter of vessel diameter or 5 = unable to assess thrombus burden due to complete occlusion), (f) the place of culprit lesion (first, middle, or end of the vessel responsible for myocardial infarction), (g) use or not use of thrombosuction, (h) use or not use of GP IIB-IIIA, and (i) Syntax Score (calculated according to the guideline in the site of Syntaxscore.com). Finally, major cardiovascular adverse events (MACE) were defined as the occurrence of at least of the following cardiovascular complications; unstable angina, myocardial infarction, or cardiac death within 30 days of discharging as the study endpoint.

2.1 | Statistical analysis

Descriptive analysis was used to describe the data, including mean ± SD for quantitative variables and frequency (percentage) for categorical variables. Chi square test, independent *t* test, and Mann–Whitney *U*-test were used for comparison of variables. The relations of ST elevation measurements with MACE variables were evaluated using Pearson's correlation coefficient. The independence of associations was tested in multivariate analyses. For the statistical analysis, the statistical software IBM SPSS Statistics for Windows version 22.0 (IBM Corp. Released 2013, Armonk, New York) was used. *P* values < .05 were considered statistically significant.

3 | RESULTS

The patients were categorized based on the location of infarct as: 1, inferior MI (n = 128), 2, anterior MI (n = 227), and 3, inferior/right ventricle MI (n = 41). In the two first subgroups, the history of smoking was more prevalent in those with ST segment ranged −1 mm to +1 mm as compared to other subgroups of ST changes. Regarding other cardiovascular risk factors, in three subgroups according to the location of infarct, no difference was revealed in the risk factors between the groups with ST <−1 mm, ST ranged −1 mm and + 1 mm

TABLE 1 The association between demographic characteristics and ST pattern in aVR lead

Location of MI	Characteristics	ST < −1	ST: −1 to +1	ST > +1	*P*
Inferior MI	Female gender (%)	20	20.3	0	.19
	Age, year	61.1 ± 5.1	58.6 ± 12	-	.49
	Family history	4	28	0	.26
	hypertension	3	49	0	.66
	Diabetes mellitus	5	38	0	.23
	Smoking	9	55	0	.001
	hyperlipidemia	2	36	0	.25
	Obesity	1	14	0	.05
Anterior MI	Female gender	9.1	26.7	0	.73
	Age, year	52.55 ± 12.36	56.58 ± 12.18	55.5 ± 13.7	.29
	Family history	4	49	2	.09
	hypertension	3	67	3	.22
	Diabetes mellitus	3	47	1	.23
	Smoking	9	103	3	.009
	hyperlipidemia	4	58	3	.63
	Obesity	1	22	1	.73
Inferior/RV MI	Female gender	50	29.7	0	.72
	Age, year	69.5 ± 17	56.78 ± 10.57	-	.36
	Family history	0	8	0	.71
	hypertension	1	16	0	.08
	Diabetes mellitus	1	9	0	.59
	Smoking	2	20	0	.72
	hyperlipidemia	2	7	0	.36
	Obesity	0	4	0	.71

Abbreviations: MI, myocardial infarction; RV, right ventricle.

and ST > +1 mm (Table 1). In terms of laboratory parameters, as shown in Table 2, the serum level of triglyceride was only different in anterior MI patients due to ST changes in the aVR lead so that in patients with ST ranged −1 mm and + 1 mm was significantly lower than other groups with different ST patterns. However, ST-segment changes had no effect on patients' LVEF.

In inferior MI subgroup, mean SUM STE was significantly high in the patients with ST <−1 mm as compared to those with ST ranged −1 mm and + 1 mm ($P = .003$). In anterior MI subgroup, MAX STE was significantly higher in patients with ST < −1 mm ($P < .001$), while ST-segment resolution was significantly the lowest in those with ST ranged −1 mm and + 1 mm ($P = .04$). Also, in the subgroups with inferior/right ventricle MI, SUM STE was significantly higher in the patients with ST < −1 mm as compared to those with ST ranged −1 mm and + 1 mm ($P = .04$). Mean Aldrich score was also lower, and also ST-segment resolution was higher in those with ST < −1 mm ($P = .05$) as compared to other ST subgroups (Table 3). As indicated in Table 4, none of the angiographic findings was significantly associated with ST-segment changes in the aVR lead. There was no significant association between the site of infarct in each type of vessel involvement and ST-segment changes in the aVR lead (Table 5).

With regard to the patients' prognosis, in anterior MI subgroup (Figure 1), reinfarction, and cardiac death were significantly higher in patients with ST >+1 mm than those with other ST patterns. Also, in inferior/right ventricle MI subgroup, reinfarction and cardiac death occurred more in those with aVR ST depression ≥1 mm. In anterior MI subgroup, three coronary vessels involvement was found more in those with ST >+1 mm. The use of integrilin in anterior MI, inferior MI, and inferior/right ventricle MI subgroups was reported to be 80%, 75%, and 75%, respectively, with no difference.

In 25 consecutive patients with first episode of STEMI, we prospectively evaluated admission ECG for aVR lead ST elevation ≥1 mm and aVR lead ST depression ≥1 mm.

In patients with anterior STEMI, with the use of multivariate analysis, admission aVR ST elevation ≥1 mm was found to be a strong and independent predictor of MACE within 30 days of discharging (*P* value for trend .002).

In patients with inferior (± RV) STEMI, with the use of multivariate analysis, admission aVR ST depression ≥1 mm was found to be a strong and independent predictor of MACE within 30 days of discharging (*P* value for trend .01).

4 | DISCUSSION

ECG, as an available noninvasive method, has been used worldwide for more than 70 years to diagnose ischemic heart diseases. Of the 12 leads studied in the ECG, the aVR lead can be considered the most

TABLE 2 The association between laboratory parameters and ST pattern in aVR lead

Location of MI	ST pattern	Parameter	Number	Mean	SD	*P*
Inferior MI	ST < −1 mm	WBC	10	9130.00	1734.006	.20
		PLT	10	240 300.00	51 372.388	.92
		TG	10	111.30	36.788	.30
		CHOL	10	157.20	35.668	.48
		HDL	10	41.90	9.539	.74
		LDL	10	91.50	26.588	.18
		EF	10	45.50	6.852	.58
	−1 mm < ST < +1 mm	WBC	118	9438.14	2905.695	
		PLT	118	216 628.64	65 219.658	
		TG	118	134.53	72.341	
		CHOL	118	153.37	40.423	
		HDL	118	37.69	17.515	
		LDL	118	94.28	39.028	
		EF	118	46.31	7.768	
Anterior MI	ST < −1 mm	WBC	11	9590.91	3225.044	.72
		PLT	11	212 818.18	55 450.551	.36
		TG	11	170.36	187.896	.03
		CHOL	11	169.09	55.616	.13
		HDL	11	37.36	14.988	.09
		LDL	11	102.00	20.712	.64
		EF	11	36.82	10.313	.56
	−1 mm < ST < +1 mm	WBC	210	10 156.90	3615.346	
		PLT	210	216 089.04	71 312.093	
		TG	210	121.62	63.823	
		CHOL	210	159.41	46.701	
		HDL	210	39.65	12.769	
		LDL	210	96.78	39.695	
		EF	210	39.43	8.648	
	ST > +1 mm	WBC	6	8400.00	1779.888	
		PLT	6	177 000.00	42 951.135	
		TG	6	171.33	119.323	
		CHOL	6	193.83	72.411	
		HDL	6	33.33	4.082	
		LDL	6	128.50	61.497	
		EF	6	39.17	5.845	
Inferior/RV MI	ST < −1 mm	WBC	4	9100.00	2483.277	.42
		PLT	4	142 500.00	62 045.682	.65
		TG	4	135.75	46.536	.90
		CHOL	4	160.00	40.825	.94
		HDL	4	41.00	13.491	.64
		LDL	4	124.25	54.689	.56
		EF	4	43.75	4.787	.20
	−1 mm < ST < +1 mm	WBC	37	9302.70	2897.651	
		PLT	37	205 630.00	61 141.301	
		TG	37	126.24	54.616	
		CHOL	37	142.43	50.252	

TABLE 2 (Continued)

Location of MI	ST pattern	Parameter	Number	Mean	SD	*P*
		HDL	37	38.54	12.591	
		LDL	37	84.89	37.856	
		EF	37	45.59	7.190	

Abbreviations: CHOL, cholesterol; EF, ejection fraction; HDL, high-density lipoprotein; LDL, light-density lipoprotein; myocardial infarction; PLT, platelet; RV, right ventricle; TG, triglycerides; WBC, white blood cell.

TABLE 3 The association between electrocardiogram (ECG) findings and ST pattern in aVR lead

Location of MI	ST pattern	Parameter	Number	Mean	SD	*P*
Inferior MI	ST < −1 mm	MAX STE	10	0.3200	0.09189	.45
		SUM STE	10	1.1150	0.57400	.003
		SELVESTE score	10	31.8000	17.38965	.06
		Aldrich score	10	7.2330	0.72170	.13
		ST resolution	10	69.5090	28.05080	.09
	−1 mm < ST < +1 mm	MAX STE	118	0.2504	0.40051	
		SUM STE	118	0.6335	0.47749	
		SELVESTE score	118	31.0169	14.90138	
		Aldrich score	117	12.8773	66.49912	
		ST resolution	117	70.6233	34.46874	
Anterior MI	ST < −1 mm	MAX STE	11	0.6182	0.33710	< .001
		SUM STE	11	2.1955	1.04987	.06
		SELVESTE score	11	28.9091	16.28161	.13
		Aldrich score	11	24.1455	6.75224	.09
		ST resolution	11	60.4400	26.23850	.04
	−1 mm < ST < +1 mm	MAX STE	209	0.3364	0.20823	
		SUM STE	210	1.1410	1.93170	
		SELVESTE score	207	37.6522	18.78738	
		Aldrich score	205	20.0532	18.41310	
		ST resolution	206	47.0281	32.90065	
	ST > +1 mm	MAX STE	5	0.4200	0.27749	
		SUM STE	5	1.2000	1.07005	
		SELVESTE score	6	29.0000	16.17405	
		Aldrich score	6	13.2500	10.87543	
		ST resolution	6	76.0000	29.28310	
Inferior/RV MI	ST < −1 mm	MAX STE	4	0.3500	0.12910	.25
		SUM STE	4	1.2250	0.62383	.04
		SELVESTE score	4	45.7500	8.61684	.57
		Aldrich score	4	7.4850	0.47339	.05
		ST resolution	4	89.5750	12.50557	.05
	−1 mm < ST < +1 mm	MAX STE	37	0.2851	0.15849	
		SUM STE	37	0.7473	0.41832	
		SELVESTE score	37	38.0022	18.17893	
		Aldrich score	35	8.3617	4.50099	
		ST resolution	35	56.2509	32.34821	

TABLE 4 The association between angiography findings and ST pattern in aVR lead

Location of MI	ST pattern	Parameter	Number	Mean	SD	*P*
Inferior MI	ST < −1 mm	Initial TIMI flow	10	0.80	1.135	.13
		Final TIMI flow	10	2.90	0.316	.42
		SYNTAXSCORE	10	16.6500	9.84900	.99
		Time to reperfusion	10	190.10	78.340	.61
		Door to device time	10	96.80	56.391	.37
	−1 mm < ST < +1 mm	Initial TIMI flow	118	0.43	0.842	
		Final TIMI flow	118	2.98	0.184	
		SYNTAXSCORE	113	17.6841	9.23126	
		Time to reperfusion	116	365.09	327.387	
		Door to device Time	118	94.77	99.551	
Anterior MI	ST < −1 mm	Initial TIMI flow	11	0.18	0.603	.92
		Final TIMI flow	11	3.00	0.000	.30
		SYNTAXSCORE	11	24.2273	13.82817	.48
		Time to reperfusion	10	267.00	221.349	.74
		Door to device Time	11	80.73	35.497	.18
	−1 mm < ST < +1 mm	Initial TIMI flow	210	0.43	0.811	
		Final TIMI flow	210	2.96	0.256	
		SYNTAXSCORE	194	19.5593	9.21928	
		Time to reperfusion	206	414.83	478.283	
		Door to device time	206	107.70	110.873	
	ST > +1 mm	Initial TIMI flow	6	0.67	1.033	
		Final TIMI flow	6	3.00	0.000	
		SYNTAXSCORE	5	21.6000	9.05124	
		Time to reperfusion	6	413.33	581.848	
		Door to device time	6	99.17	39.550	
Inferior/RV MI	ST < −1 mm	Initial TIMI flow	4	0.00	0.000	.72
		Final TIMI flow	4	3.00	0.000	.36
		SYNTAXSCORE	4	13.7500	3.59398	.71
		Time to reperfusion	4	358.75	312.633	.08
		Door to device time	4	93.25	28.206	.59
	−1 mm < ST < +1 mm	Initial TIMI flow	37	0.38	0.794	
		Final TIMI flow	37	2.95	0.229	
		SYNTAXSCORE	36	18.7778	9.86729	
		Time to reperfusion	37	453.19	879.971	
		Door to device time	37	106.54	157.130	

TABLE 5 The site of coronary vessel involvement in terms of ST pattern in aVR lead

Artery	Site of Involvement	ST < −1	ST: −1 to +1	ST > +1
LAD	Proximal	5 (45.5)	76 (41.5)	3 (60.0)
	Middle	5 (45.5)	102 (55.7)	1 (20.0)
	Distal	1 (9.1)	5 (2.8)	1(20.0)
RCA	Proximal	3 (23.1)	39 (34.5)	42 (33.3)
	Middle	7 (53.8)	45 (39.8)	52 (41.3)
	Distal	3 (23.1)	29 (25.7)	32 (25.4)
LCX	Proximal	2 (100)	28 (68.3)	30 (69.8)
	Middle	0 (0.0)	3 (7.3)	3 (7.0)
	Distal	0 (0.0)	10 (24.4)	10 (76.8)

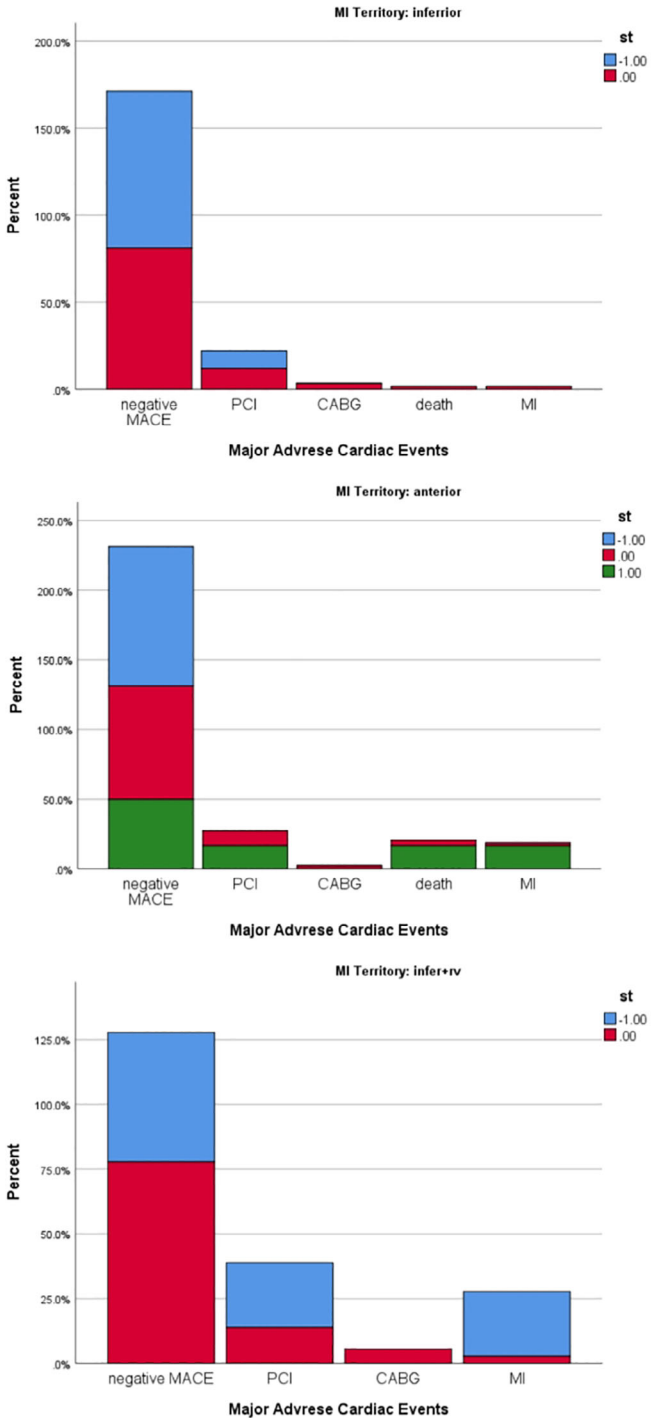

FIGURE 1 The rate of major adverse cardiovascular events according to ST pattern in inferior, anterior, and inferior/right ventricle myocardial infarction (MI) subgroups

forgotten part because it is not considered as a mirror image of other leads. Over the past few decades, this lead has re-emerged as an important part of the ECG among cardiologists. ST-segment changes can be considered the most important ECG finding in the diagnosis and evaluation of MI. The aVR lead is a good reference for what happens in the upper right part of the heart.[11] The last thoracic lead (V6) is located in the axillary midline, and a V7 lead in the axillary dorsal line can show extensive ischemia of the heart apex more clearly. This finding indicates the importance of the mirror image in aVR, which is mostly a reflection of ischemia in the apex of the heart, and shows the mirror image of the V7 lead more than other leads. This means that ST depression more in the aVR lead indicates more ST elevation not only in V5 and V6 but also in V7.[12] The use of ST-segment, T-wave, and Q-wave in the aVR lead to evaluate the current or past status of previous or current MI patients has been suggested in various studies.[13] In 2012, Kukla et al[14] reported that the changes in the aVR lead occurred in half of the patients with MI and were significantly associated with poor disease prognosis.

In this study, we showed that in patients with anterior MI, MAX STE was significantly higher in patients with ST < −1 mm and significantly lower in patients with ST > +1 mm. Also, ST-segment resolution was significantly lower in patients −1 mm < ST < + 1 mm. Regarding the anatomy of coronary artery involvement and its relationship with changes in the aVR lead, no significant relationship was found in our study. However, in anterior MI patients, ST elevation in aVR was associated with greater three coronary vessels involvement. This finding has also been reported in the study of Beyranvand et al.[15] We showed in this study that in patients with inferior MI, SUM STE was significantly higher in patients with ST < −1 mm. Such patients had also slightly higher Selvester score. Additionally, in patients suffering inferior plus right ventricle MI patients, SUM STE was significantly higher in patients with ST < −1 mm compared to patients with −1 mm < ST < + 1 mm. Aldrich score was also lower in patients with ST < -1 mm. However, in the present study, disease prognosis was independent to ST-segment changes in aVR lead, indicating low powerfulness of such changes in predicting poor prognosis. The latter findings were however in contrary to some other studies. Wong et al[16] evaluated ST-segment elevation in aVR among a large group of patients with fibrinolytic AMI. Among all patients with normal intraventricular conduction, ST elevation in aVR was associated with a higher 30-day mortality even independent of concomitant ST-segment changes in other ECG leads. This association was strong for anterior AMI patients with a cutoff point greater than 1.5 mm and for inferior AMI patients with a cutoff point greater than 1 mm and were associated with an approximately 2.5-fold increase in 30-day mortality. Also, the study by Harhash et al[5] showed that only 10% of patients with ST elevation in aVR with ST diffuse depression had acute thrombotic coronary occlusion. This is much less than the standard STEMI population, which accounts for 65% to 85% of cases of acute coronary obstruction on immediate coronary angiography. Meanwhile, these patients with ST elevation and ST diffuse depression in aVR had fivefold higher in-hospital mortality compared with the standard STEMI population (65). In our study, in patients with anterior MI, reinfarction, and more death occurred in patients with ST > + 1 mm, but this difference was not statistically significant. Also, in patients with inferior plus right ventricle MI, reinfarction occurred more in patients with ST < −1 mm, but this difference was not also statistically significant that the discrepancy between our finding and other reports might be due to smaller sample size employed in our

cohort or even the genetically differences across the populations. In this regard, in a similar study among Iranian population, a significant relationship between ST-segment changes in the aVR lead and the number of vessels involved in angiography, infarct location, and fractional ejection was not reported.[15] However, the presence of ST elevation> +1 mm in the aVR lead was associated with an eightfold increase in the risk of in-hospital mortality. It should be noted that in this study, the existence of different number of cases in different groups can be a reason for a number of factors not being significant, and it is predicted that by increasing the number of cases and matching the number of cases in each subgroup. But still in our society, changes in the piece will not be a reason for its value in predicting the consequences of the disease.

In patients with anterior STEMI, with the use of multivariate analysis, admission aVR STE ≥1 mm was found to be a strong and independent predictor of MACE within 30 days of discharging. On the other hand, in patients with inferior (± RV) STEMI, with the use of multivariate analysis, admission aVR ST depression ≥1 mm was found to be a strong and independent predictor of MACE within 30 days of discharging.

5 | CONCLUSION

In patients with anterior STEMI, admission aVR STE ≥1 mm was found to be a strong and independent predictor of MACE within 30 days of discharging. On the other hand, in patients with inferior STEMI, aVR ST depression ≥1 mm was found to be a strong and independent predictor of MACE within 30 days of discharging.

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CONFLICT OF INTEREST

The authors have stated explicitly that there are no conflicts of interest in connection with this article.

AUTHOR CONTRIBUTIONS

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Revised the manuscript: Amir Aslani, Pooyan Dehghani.

All authors have read and approved the final version of the manuscript.

Mani Hassanzadeh had full access to all of the data in this study and takes complete responsibility for the integrity of the data and the accuracy of the data analysis.

TRANSPARENCY STATEMENT

The corresponding author affirms that this manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

This study was approved by the Ethics Committee of the Shiraz University of Medical Sciences (IR.SUMS.MED.REC.1398.552).

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