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 $\geq 1$ 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 $\geq 1$ 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 $\geq 1$ 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 $\geq 1$ 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. 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. 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). 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 sequel 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 study, 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 AVRT (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: \(3 \times (1.5 \cdot \text{number of leads with ST}^+ \cdot 0.4)\) and inferior STEMI: \(3 \times (0.6 \cdot \text{ST}^+ \cdot II, III, aVF + 2.0)\)). 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 thrombosisulation, (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
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 $/C0_{-1}$ 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 ($P = .05$) 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 $\geq 1$ 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 $\geq 1$ mm and aVR lead ST depression $\geq 1$ mm. In patients with anterior STEMI, with the use of multivariate analysis, admission aVR ST elevation $\geq 1$ 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 $\geq 1$ 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 | 12.59 |     |
|                |            | LDL               | 37     | 84.89 | 37.85 |     |
|                |            | EF                | 37     | 45.59 | 7.19  |     |

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  | 18.17993 |       |
|                  |            | 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  | .18|
|                |                 | Final TIMI flow             | 118    | 2.98   | 0.184  | .32|
|                |                 | SYNTAXSCORE                 | 113    | 17.6841| 9.23126| .49|
|                |                 | Time to reperfusion         | 116    | 365.09 | 327.387| .74|
|                |                 | Door to device Time         | 118    | 94.77  | 99.551 | .59|
| 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| 13.8217| .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  | .18|
|                |                 | Final TIMI flow             | 210    | 2.96   | 0.256  | .59|
|                |                 | SYNTAXSCORE                 | 194    | 19.5593| 9.21928| .59|
|                |                 | Time to reperfusion         | 206    | 414.83 | 478.283| .79|
|                |                 | Door to device time         | 206    | 107.70 | 110.873| .59|
|                | ST > +1 mm      | Initial TIMI flow           | 6      | 0.67   | 1.033  | .36|
|                |                 | Final TIMI flow             | 6      | 3.00   | 0.000  | .36|
|                |                 | SYNTAXSCORE                 | 5      | 21.6000| 9.05124| .71|
|                |                 | Time to reperfusion         | 6      | 413.33 | 581.848| .59|
|                |                 | Door to device time         | 6      | 99.17  | 39.550 | .59|
| 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| .71|
|                |                 | Door to device time         | 4      | 93.25  | 28.206 | .59|
|                | −1 mm < ST < +1 mm| Initial TIMI flow         | 37     | 0.38   | 0.794  | .59|
|                |                 | Final TIMI flow             | 37     | 2.95   | 0.229  | .59|
|                |                 | SYNTAXSCORE                 | 36     | 18.7778| 9.86729| .59|
|                |                 | Time to reperfusion         | 37     | 453.19 | 879.971| .59|
|                |                 | Door to device time         | 37     | 106.54 | 157.130| .59|

### 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)|
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. 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. 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. In 2012, Kukla et al 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. 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 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 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. However, the presence of ST elevation $\geq 1$ 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 $\geq 1$ 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 $\geq 1$ 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 $\geq 1$ 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 $\geq 1$ mm was found to be a strong and independent predictor of MACE within 30 days of discharging.

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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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