ORIGINAL ARTICLE

Positive T wave in lead aVR as an independent predictor for 1-year major adverse cardiac events in patients with first anterior wall ST-segment elevation myocardial infarction

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Background: Positive T wave in lead aVR has been shown to predict an adverse in-hospital outcome in patients with anterior wall ST-segment elevation myocardial infarction (STEMI). However, the prognostic value of positive T wave in lead aVR on a long-term outcome has not been fully explored.

Methods: We performed a retrospective analysis of 190 consecutive patients with first anterior wall STEMI who underwent an emergent coronary angiogram. Patients were divided into those with positive T wave > 0 mV and those with negative T wave ≤ 0 mV in lead aVR. Baseline and angiographic characteristics, and in-hospital revascularization procedures were recorded. In addition, in-hospital and 1-year major adverse cardiac events (MACE) including death, recurrent myocardial infarction, and target vessel revascularization were recorded.

Results: Among 190 patients, 37 patients (19%) had positive T wave and 153 patients (81%) had negative T wave in lead aVR. Patients with positive T wave had higher rate of left main disease defined as stenosis $\geq 50\%$ (11% vs. 2%, $p = .028$) than those with negative T wave. Patients with positive T wave had higher rate of 1-year MACE (38% vs. 13%, $p < .001$) driven by higher all-cause mortality (27% vs. 5%, $p < .001$). Positive T wave was an independent predictor for 1-year MACE (OR 2.74; 95% CI 1.04–7.15; $p = .04$).

Conclusion: Positive T wave in lead aVR was an independent predictor for 1-year MACE in patients with first anterior wall STEMI.

KEYWORDS

lead aVR, positive T wave, STEMI

1 | INTRODUCTION

Twelve-lead electrocardiogram provides fundamental information in the diagnosis of acute coronary syndrome (ACS). Among 12 leads in a standard electrocardiogram, lead aVR has been a lead of ignorance until recently when ST-segment deviations in lead aVR have been shown to be associated with the length of the occluded left anterior descending (LAD) artery and the site of the LAD artery occlusion in patients with first anterior wall ST-segment elevation myocardial

infarction (STEMI; Goto, Tamura, Kotoku, & Kadota, 2011; Kotoku, Tamura, Abe, & Kadota, 2009).

In addition to ST-segment deviations in lead aVR, T-wave amplitude in lead aVR has been evaluated in patients with prior anterior wall myocardial infarction (Shinozaki, Tamura, & Kadota, 2011). Shinozaki et al. showed that patients with positive T wave in lead aVR are more likely to have the occlusion of long LAD artery and severely reduced left ventricular ejection fraction. In addition, Torigoe et al. (2012) reported that positive T wave in lead aVR is associated with higher rate

of multivessel disease, proximal LAD occlusion, and lower left ventricular ejection fraction in patients with prior myocardial infarction.

Recently, Ayhan et al. (2013) reported that positive T wave in lead aVR is associated with a higher rate of multivessel disease and proximal LAD occlusion and predicts an adverse in-hospital outcome in patients with anterior wall STEMI. However, the prognostic value of positive T wave in lead aVR on a long-term outcome has not been fully investigated.

Thus, the aim of this present study was to clarify the long-term prognostic value of positive T wave in lead aVR in patients with first anterior wall STEMI.

2 | MATERIAL AND METHODS

A retrospective analysis was performed on patients with a diagnosis of first anterior wall STEMI who underwent an emergent coronary angiogram between January 2007 and December 2013 at our institution. Two researchers independently reviewed emergency department records, in-hospital admission records, and cardiac catheterization procedure records to identify patients with first anterior wall STEMI. Inclusion criteria were: (i) ST-segment elevation at least two adjacent precordial leads on admission electrocardiogram (≥ 0.20 mV in leads V_2 and V_3 , and ≥ 0.10 mV in other leads); (ii) having received an emergent coronary angiogram; and (iii) the presence of a culprit lesion in the LAD artery in a coronary angiogram. Exclusion criteria were: (i) previous myocardial infarction; (ii) previous coronary artery bypass grafting (CABG); (iii) left bundle branch block or ventricular paced rhythm; (iv) left main culprit lesion or isolated diagonal culprit lesion; and (v) insufficient data for analysis. Present study complied with the Declaration of Helsinki and was approved by the institutional review board of our hospital. Participants were contacted for telephone interview, and provided a verbal consent.

Patients' demographic data, risk factors along with admission characteristics including hemodynamic parameters such as blood pressure, heart rate, and Killip classification were obtained. Laboratory data on admission including white blood cell count, hemoglobin, and creatinine kinase were recorded. Left ventricular ejection fraction was evaluated during hospital stay either with transthoracic echocardiogram or with left ventriculography.

2.1 | Electrocardiogram

Standard 12-lead electrocardiograms (25 mm/s and 10 mm = 1 mV) were obtained from all patients on arrival to emergency department and were reviewed by two independent reviewers in a blinded fashion. In the events of any discrepancy in the assessments, the two reviewers reached a consensus through discussion.

Positive T wave was defined as T-wave amplitude >0 mV, and negative T wave was defined as T-wave amplitude ≤ 0 mV in lead aVR. ST-segment deviations were measured at the J point. ST-segment elevation ≥ 0.20 mV in lead V_2 and V_3 , and ST-segment elevation ≥ 0.10 mV in other leads (except for lead aVR) were recorded. In addition, ST-segment elevation ≥ 0.05 mV and ST-segment depression

≥ 0.05 mV in lead aVR were recorded. The left ventricular hypertrophy (LVH) was diagnosed using Sokolow–Lyon voltage amplitude criteria $S_{V_1} + R_{V_5 \text{ or } V_6} \geq 3.5$ mV and Cornell voltage criteria $S_{V_3} + R_{aVL} \geq 2.8$ mV in men and ≥ 2.0 mV in women. In patients with left anterior hemi-block or right bundle branch block, LVH was defined as SIII + maximal precordial R + S ≥ 3.0 mV (Gertsch, Theler, & Foglia, 1988; Vandenberg, Sagar, Paulsen, & Romhilt, 1989). Negative T wave in lead V_5 and V_7 was defined as T-wave amplitude ≤ 0 mV.

2.2 | Coronary angiogram

All patients underwent an emergent cardiac catheterization. An independent cardiologist blinded to the clinical data reviewed coronary angiography, and the assessment was compared with the primary assessment by the treating cardiologist. In case of any discrepancy between the assessments, a third investigator made the final interpretation. Obstructive coronary artery disease (CAD) was defined as stenosis $\geq 50\%$ in the left main coronary artery and 70% in any other epicardial coronary arteries. Long LAD artery was defined as LAD artery supplying blood flow to more than one-fourth of the inferior wall of the left ventricle in the right anterior oblique projection. Coronary blood flow was graded according to TIMI criteria (TIMI Study Group, 1985). The site of the culprit lesion was determined by the angiographic appearance of thrombus or ulcerated plaque. Revascularization procedures including primary percutaneous coronary intervention (PCI) and CABG were performed at the discretion of the treating physician. A successful primary PCI was defined as a TIMI 3 flow after the procedure.

2.3 | Endpoints

The primary endpoint of this study was 1-year major adverse cardiac events (MACE) including death, recurrent myocardial infarction, and target vessel revascularization. The secondary endpoint was in-hospital MACE. Aforementioned clinical outcomes were obtained through outpatient follow-up, telephone interviews, and the Social Security Death Index.

2.4 | Statistical analysis

Patients were divided into those with positive T wave and those with negative T wave in lead aVR, and clinical variables were compared between the two groups. In addition, subgroup analysis was performed to evaluate the association between 1-year MACE and positive T wave in lead aVR after excluding patients with unsuccessful PCI (subgroup analysis 1) and patients without ST-segment elevation in lead V_1 (subgroup analysis 2) as our prior study revealed its predictive value for worse long-term outcomes in patients with anterior wall STEMI (Misumida et al., 2015).

Data were expressed as either a number (percentage) or median [interquartile range]. For continuous variables, Shapiro–Wilk test was used to check the normality of the distribution. Continuous variables were compared using either Student's *t* test or Wilcoxon rank sum test. Dichotomous variables were compared using the chi-squared test or Fisher's exact test.

TABLE 1 Demographic, laboratory, and hemodynamic data

	Positive T wave (n = 37)	Negative T wave (n = 153)	p-Value
Demographics			
Age (years)	67 [54–76]	59 [51–67]	.012
Male	30 (81)	124 (81)	1
Hypertension	23 (62)	69 (45)	.062
Diabetes mellitus	13 (35)	36 (24)	.15
Hyperlipidemia	15 (41)	71 (46)	.52
Chronic kidney disease (eGFR < 60 mL min ⁻¹ 1.73 m ⁻²)	8 (22)	24 (16)	.29
Family history of coronary artery disease	4 (11)	24 (16)	.45
History of coronary artery disease	7 (19)	17 (11)	.27
Current smoker	10 (27)	60 (39)	.17
Previous percutaneous coronary intervention	5 (14)	15 (10)	.55
Interval from symptom onset to presentation (hours)	6 [3–10]	2 [1–4]	<.001
Hemodynamic and laboratory data			
Systolic blood pressure (mm Hg)	129 [114–147]	134 [121–150]	.39
Diastolic blood pressure (mm Hg)	82 [71–99]	85 [72–95]	.7
Heart rate (beat/minute)	95 [86–108]	82 [72–95]	.001
Hemoglobin (g/L)	14.3 [12.7–15.2]	14.8 [13.5–15.6]	.16
White blood cell count (10 ⁹ /L)	12.2 [9.5–14.2]	11.5 [9.3–13.4]	.44
eGFR (mL min ⁻¹ 1.73 m ⁻²)	76 [59–90]	81 [64–95]	.27
Peak creatinine kinase (U/L)	2557 [932–5,054]	2875 [916–4,731]	.78
Killip class 3–4 on admission	7 (19)	19 (12.4)	.3
Left ventricular ejection fraction (%)	35 [25–40]	35 [30–45]	.17

eGFR, estimated glomerular filtration rate.

Data are expressed as a number (percent) or median [interquartile range].

Univariate and multivariate analyses were performed to assess predictors for 1-year MACE. Following variables were evaluated first in a univariate model: age (per decade increase), left ventricular ejection fraction (per 1% increase), Killip class 3–4 on admission, door to balloon time (per 1-min increase), peak creatinine kinase (per 100 IU/L increase), postprocedure TMI flow 3, right bundle branch block, atrial fibrillation, ST-segment elevation ≥ 0.05 mV in lead aVR, ST-segment depression ≥ 0.05 mV in lead aVR, and positive T wave in lead aVR, and absence of ST-segment elevation in lead V₁. Significant variables with a *p* value <.10 in the univariate analysis were then entered into a multivariate logistic regression analysis using backward stepwise elimination. A significance level of 0.10 was required to allow a variable to remain in the model. Two-sided *p* values <.05 were considered statistically significant. All statistical analyses were performed with R software (version 3.0.1).

3 | RESULTS

A total of 190 patients with first anterior wall STEMI were included in final analysis. Of the 190 patients, 37 patients (19%) had positive T wave and 153 patients (81%) had negative T wave in lead aVR on presenting electrocardiograms.

3.1 | Baseline, hemodynamic, and laboratory data as well as electrocardiographic findings

Baseline demographic and clinical characteristics are summarized in Table 1. Patients with positive T wave were older than those with negative T wave. Interval from symptom onset to presentation was significantly longer in patients with positive T wave than those with negative T wave. Peak creatinine kinase was comparable between the two groups. Electrocardiographic findings were presented in Table 2. Patients with positive T wave in lead aVR were more likely to have ST-segment elevation and negative T wave in lead V₅ and V₆ than those with negative T wave in lead aVR. Patients with positive T wave were less likely to have ST-segment elevation in lead V₁. No significant difference was seen in the rate of electrocardiographic LVH between the two groups.

3.2 | Angiographic characteristics and revascularization procedures

Angiographic characteristics are presented in Table 3. Door to balloon time was longer in patients with positive T wave than those with negative T wave. There was a trend toward higher rate of multivessel

TABLE 2 Electrocardiographic characteristics

	Positive T wave (n = 37)	Negative T wave (n = 153)	p-Value
Electrocardiographic findings			
ST-segment elevation in lead aVR	9 (24)	44 (29)	.59
ST-segment depression in lead aVR	3 (8)	16 (10)	1
ST-segment elevation in lead V ₄	30 (81)	111 (73)	.29
ST-segment elevation in lead V ₅	17 (46)	42 (27)	.029
ST-segment elevation in lead V ₆	7 (19)	13 (8.5)	.079
Negative T wave in lead V ₅	10 (27)	4 (3)	<.001
Negative T wave in lead V ₆	10 (27)	3 (2)	<.001
ST-segment elevation in lead I and aVL	8 (22)	36 (24)	.81
ST-segment elevation in lead II, III, and aVF	3 (8)	10 (7)	.73
Atrial fibrillation	5 (14)	7 (5)	.06
Right bundle branch block	4 (11)	19 (12)	1
Absence of ST-segment elevation in lead VI	14 (38)	28 (18)	.01
Left ventricular hypertrophy	7 (19)	15 (10)	.12

Data are expressed as a number (percent).

disease and the occlusion of long LAD artery prior to the first diagonal branch. Patients with positive T wave had higher rate of left main disease than those with negative T wave.

With respect to revascularization procedures, patients with positive T wave had a lower rate of in-hospital primary PCI. Patients with positive T wave had a lower rate of successful primary PCI than those with negative T wave.

3.3 | Endpoints

Short- and long-term clinical outcomes are summarized in Table 3. Although it did not reach a statistical significance, there was a trend toward for a higher rate of in-hospital MACE in patients with positive T wave. Patients with positive T wave had a higher rate of 1-year MACE driven by a higher all-cause mortality.

In a subgroup analysis after excluding patients with unsuccessful PCI (subgroup analysis 1), positive T wave in lead aVR remained significantly associated with a higher rate of 1-year MACE (35% vs. 13%, $p = .018$). In addition, the presence of positive T wave in lead aVR was significantly associated with a higher rate of 1-year MACE (30% vs. 10%, $p = .013$) in a subgroup analysis after excluding patients without ST-segment elevation in lead V₁ (subgroup analysis 2).

Multivariate logistic regression analysis revealed that positive T wave in lead aVR was an independent predictor for 1-year MACE after adjusting other clinical variables (odds ratio 2.74; 95% confidence

TABLE 3 Angiographic characteristics, revascularization procedures, and in-hospital and 1-year outcomes

	Positive T wave (n = 37)	Negative T wave (n = 153)	p-Value
Angiographic findings			
Door to balloon time (min)	75 [63–91]	57 [42–76]	.007
Multivessel disease	25 (68)	81 (53)	.11
Three-vessel disease	12 (32)	35 (23)	.23
Left main disease	4 (11)	3 (2)	.028
Long LAD artery occlusion prior to the first diagonal branch	9 (24)	22 (14)	.14
In-hospital revascularization			
Primary PCI	30 (81)	149 (97)	.001
Emergent CABG	3 (8)	2 (1)	.25
TIMI flow 3 after primary PCI	20 (67)	120 (81)	.093
In-hospital staged PCI	3 (8)	14 (9)	1
In-hospital outcomes			
In-hospital MACE	5 (14)	8 (5)	.14
1-year MACE	14 (38)	20 (13)	<.001
1-year all-cause mortality	10 (27)	8 (5)	<.001
1-year recurrent myocardial infarction	2 (5)	5 (3)	.62
1-year target vessel revascularization	2 (5)	7 (5)	.69

LAD, left anterior descending; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; MACE, major adverse cardiac events. Data are expressed as a number (percent) or median [interquartile range].

interval 1.04–7.15; $p = .04$). Other independent predictors for 1-year MACE were age (per decade increase), left ventricular ejection fraction (per 1% increase), Killip class 3–4 on admission, and absence of ST-segment elevation in lead V₁ (Table 4).

4 | DISCUSSION

Our present study revealed that positive T wave in lead aVR was an independent predictor for 1-year MACE in patients with first anterior wall STEMI.

Positive T wave in lead aVR has been reported to be associated with an occlusion of long LAD artery and lower left ventricular ejection fraction in patients with prior anterior wall myocardial infarction (Shinozaki et al., 2011). Shinozaki et al. theorized that an occlusion of long LAD artery, which supplies the apical, inferior, and lower lateral regions of the left ventricle, would result in widespread myocardial ischemia and severe left ventricular dysfunction. Torigoe et al. (2012) evaluated patients with prior myocardial infarction and reported that patients with positive T wave in lead aVR were more likely to have multivessel disease. In addition, Ayhan et al. (2013) reported that positive T wave in lead aVR is associated with a higher rate of multivessel disease and

TABLE 4 Univariate and multivariate analyses to assess predictors for 1-year major adverse cardiac events

	Univariate analysis			Multivariate analysis		
	Odds ratio	95% Confidence interval	p-Value	Odds ratio	95% Confidence interval	p-Value
Clinical variables						
Age (per decade increase)	1.66	1.24–2.26	.001	1.59	1.14–2.27	.008
Left ventricular ejection fraction (per 1% increase)	0.95	0.91–0.98	.002	0.96	0.93–0.99	.046
Killip class on admission 3–4	4.5	1.81–11.0	.001	3.59	1.21–10.60	.02
Door to balloon time (per 1 min increase)	1	0.99–1.02	.5			
Peak CK (per 100 IU/L increase)	1.02	1.00–1.03	.006			
Postprocedure TIMI flow 3	0.53	0.24–1.20	.12			
Right bundle branch block	4.58	1.78–11.6	.001	2.59	0.82–8.00	.099
Atrial fibrillation	3.67	1.03–12.3	.036			
ST-segment elevation in lead aVR	2.01	0.83–5.66	.15			
ST-segment depression in lead aVR	1.96	0.52–12.7	.39			
Absence of ST-segment elevation in lead VI	3.77	1.69–8.37	.001	2.67	1.03–6.89	.041
Positive T wave in lead aVR	4.05	1.78–9.15	.001	2.74	1.04–7.15	.04

MACE, major adverse cardiac events; CK, creatinine kinase; TIMI, thrombolysis in myocardial infarction.

proximal LAD occlusion in patients with anterior wall STEMI, suggesting that positive T wave is associated with extensive CAD which leads to widespread myocardial ischemia and adverse clinical outcomes.

Although the exact mechanism of the positive T wave in lead aVR remained to be a subject for further investigation, it has been suggested that concomitant myocardial ischemia in the apical, inferior, and lower lateral walls results in inversion of T-wave vector to those injured regions due to the delayed repolarization, thus result in a positive T wave in lead aVR (Ayhan et al., 2013; Shinozaki et al., 2011).

In our present study, patients with positive T wave in lead aVR were more likely to have ST-segment elevation and negative T wave in lead V_5 and V_6 , suggesting lateral wall ischemia. In addition, patients with positive T wave in lead aVR tended to have multivessel disease and an occlusion of long LAD artery prior to the first diagonal branch. We speculate that concomitant lateral wall ischemia due to either diagonal branch involvement or multivessel disease would result in the higher MACE in our present study. This theory is supported by higher prevalence of abnormal Q waves in lead I, V_5 , and V_6 in patients with positive T wave in lead aVR in Shinozaki's study (Shinozaki et al., 2011). In addition, the association between positive T wave in lead aVR and lateral wall ischemia has been reported in patients with ischemic cardiomyopathy (Al-Zaiti, Fallavollita, Canty, & Carey, 2015).

Our previous study has shown that in patients with anterior wall STEMI, the absence of ST-segment elevation in lead V_1 is associated with lateral wall involvement leading to adverse clinical outcomes (Misumida et al., 2015). In our present study, although patients with positive T wave in lead aVR were less likely to have ST-segment elevation lead V_1 , the presence of positive T wave in lead aVR remained significantly associated with 1-year MACE after excluding patients without ST-segment elevation in lead V_1 (subgroup analysis 2), suggesting that positive T wave in lead aVR is not merely reflective of the concomitant ST-segment changes in lead V_1 .

In addition, our study also has shown that patients with positive T wave in lead aVR have higher rate of left main disease than those with negative T wave. Previous studies reported the association between ST-segment elevation in lead aVR and left main disease in patients with ACS (Atie et al., 1991; Hori et al., 2000; Yamaji et al., 2001). In our study, the rates of ST-segment elevation in lead aVR were comparable between patients with positive T wave and those with negative T wave in lead aVR. In ACS populations, patients presenting with left main disease are at increased risks of adverse events (Figueras, Domingo, & Hermosilla, 2005; Figueras & Lidón, 1993; Kosuge et al., 2011). Although, left main disease was only observed in total seven patients (13%) of our cohort (four patients with positive T wave and three patients with negative T wave), three of four patients with both positive T wave in lead aVR and left main disease died within 1-year follow-up. This higher rate of left main disease would in part explain the increased MACE in patients with positive T wave in lead aVR.

A previous study has shown that positive T wave in lead aVR predicts increased in-hospital adverse cardiac events in patients with anterior wall STEMI (Ayhan et al., 2013). Our present study added the long-term prognostic value of positive T wave in lead aVR, while no statistically significant difference was observed in the rate of in-hospital MACE between patients with positive T wave and those with negative T wave. This discrepancy can be explained by different definitions of positive and negative T wave in lead aVR in the two studies. Ayhan et al. defined (i) positive T wave ≥ 0.10 mV and (ii) negative T wave < -0.10 mV in contrast to (i) positive T wave > 0 mV and (ii) negative T wave ≤ 0 mV in our present study. Tan, Engel, Myers, Sandri, and Froelicher (2008) reported that mortality increases as T-wave amplitude become less negative in general population. In addition, Okuda et al. (2011) also reported that as T-wave amplitude in lead aVR becomes less negative, there is a trend for higher mortality in patients with heart failure. The higher cutoff of ≥ 0.10 mV for positive T wave and lower cutoff of < -0.10 mV

for negative T wave in Ayhan's study would result in comparing very contrastive populations leading to significant difference in the rate of in-hospital adverse events between patients with positive T wave and those with negative T wave in their study.

One thing we should note in our present study is that patients with positive T wave had a longer interval between symptom onset to presentation and door to balloon time which would lead to a lower rate of successful primary PCI and subsequent adverse outcomes. However, in a subgroup analysis after excluding patients with unsuccessful PCI (subgroup analysis 1), positive T wave in lead aVR remained significantly associated with higher rate of 1-year MACE.

Our study has several limitations. First, our study was subject to the usual constraints associated with a retrospective observational study. However, we reviewed all patients who underwent cardiac catheterization, thus limiting our selection bias. Second, our study included relatively small number of patients. Third, the optimal cutoff for positive T wave has not been determined; thus, further studies are necessary to determine the optimal cutoff to predict short- and long-term adverse events in this first anterior wall STEMI population. Finally, we evaluated only initial presenting electrocardiograms. T-wave positivity can be influenced by revascularization procedures. Thus, it would be of clinical interest to see if (i) positive T wave becomes negative after revascularization and (ii) persistent positive T wave even after revascularization predicts worse clinical outcomes in patients with first anterior wall STEMI.

5 | CONCLUSIONS

We conclude that positive T wave in lead aVR on admission electrocardiography is an independent predictor for 1-year MACE in patients with first anterior wall STEMI.

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None.

DECLARATION OF INTEREST

We have no conflicts of interest to declare.

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