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Prognostic significance of ST segment changes in lead aVR in patients with acute inferior myocardial infarction with ST segment elevation

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Abstract

Background: Patients with inferior wall ST segment elevation myocardial infarction (STEMI) are considered to be at lower risk than patients with anterior wall STEMI. Nonetheless, 30–40% of all acute inferior wall MI cases have a poor prognosis.

Aim: To assess the frequency of ST segment changes (elevation or depression) in lead aVR in inferior STEMI patients, and to determine the clinical course and short-term prognosis of such patients.

Methods: The study retrospectively analysed the records of 320 consecutive patients with inferior wall STEMI (206 males, 114 females, mean age 65.6 ± 11.1 years). Patients were divided into three groups based on treatment: group A, primary percutaneous coronary intervention (134 patients); group B, fibrinolytic therapy (96 patients); and group C, conservative treatment (no reperfusion therapy) (90 patients). The mean time from onset of pain to the first ECG for all patients was 6.1 h. The total number of in-hospital deaths was 29 (9.0%), comprising 11 (8.2%) in group A, seven (7.3%) in group B, and 11 (12.2%) in group C (NS). The mean maximum creatine phosphokinase was 2,021 ± 1,837 U/L in group A, 1,734 ± 1,581 U/L in group B, and 1,217 ± 981 U/L in group C ($p = 0.01$). The mean left ventricular ejection fraction was 50.2% ± 9.0%, 54.9 ± 8.6%, and 51.3% ± 9.7% for groups A, B and C, respectively (NS).

Results: ST segment changes in lead aVR were observed in 135 (42.2%) patients, comprising elevation in 47 (14.7%) patients and depression in 88 (27.5%) patients. The in-hospital mortality rates for patients with ST segment elevation, ST segment depression, and no ST segment changes were 27.7%, 16.5%, and 1.0%, respectively ($p < 0.001$). For group A, the in-hospital mortality rate was higher in patients with ST segment elevation than in patients with no ST segment changes (15.4% *vs* 1.2%, $p < 0.001$). For group B, the in-hospital mortality rates were 33.3%, 12.9%, and 0%, in patients with ST segment elevation, ST segment depression, and no ST changes, respectively ($p = 0.006$). For group C, the in-hospital mortality rate was higher in patients with ST segment elevation (32%) than in patients with ST segment depression (12.5%) and patients with no ST segment changes (2%, $p = 0.006$). Logistic regression analysis found that female gender, diabetes, hypertension, lower ejection fraction, and cardiogenic shock on admission were independent predictors of ST segment elevation.

Conclusions: ST segment changes in lead aVR occurred in approximately half of inferior wall STEMI patients. The presence of such ST segment changes was associated with a poorer prognosis during the hospital stay, and the changes were not associated with the type of reperfusion treatment.

Key words: inferior STEMI, lead aVR, ECG

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INTRODUCTION

ST segment elevation myocardial infarction (STEMI) of the inferior wall is considered to be associated with a lower risk of MI than STEMI of the anterior wall [1, 2]. Nevertheless, approximately 30% of patients with inferior wall STEMI experience complications during their hospital stay which are associated with a poor prognosis [3–6].

There is a need for non-invasive assessments that enable clinicians to identify whether an inferior STEMI patient is at a high risk of complications and/or adverse outcomes. ST segment changes in lead aVR are typically ignored in electrocardiograph (ECG) analysis [7, 8].

According to the European Society of Cardiology (ESC) guidelines, ST segment elevation in lead aVR is not useful for the diagnosis of MI. However, publications over the past ten years have indicated that aVR changes occur in acute coronary syndrome (ACS) [9, 10]. ST segment elevation in lead aVR in ACS could indicate stenosis of the left main coronary artery or three-vessel coronary artery disease (CAD), and is associated with a poor prognosis. ST segment changes in lead aVR can be useful in predicting the success of primary percutaneous coronary intervention (PCI) [11–15]. A Japanese study of inferior wall STEMI found that the grade of ST segment depression correlated with impaired reperfusion during PCI [16].

Despite the abovementioned studies, the prognostic significance of ST segment elevation in lead aVR in STEMI patients remains to be established. In particular, little is known about the incidence of ST segment changes in lead aVR in inferior wall STEMI patients, nor about the prognostic significance of such changes.

The present study examined ST segment changes (elevation and depression) in lead aVR in inferior wall STEMI patients. The study determined the frequency of ST segment changes in such patients, and the relationship between ST segment changes and short-term clinical outcomes.

METHODS

Study groups

We retrospectively analysed the records of 320 consecutive patients (206 males, 114 females, mean age 65.6 ± 11.1 years) who presented with inferior wall STEMI at the Department of Internal Medicine (Gorlice, Poland) and the Department of Cardiology and Hypertension (Krakow, Poland) between 2003 and 2006. Patients were divided into three groups based upon the treatment they had received: group A, invasive treatment with primary PCI (81 males, 53 females, mean age 66.1 ± 10.6 years); group B, fibrinolytic therapy (77 males, 19 females, mean age 62.7 ± 12.3 years) because PCI of less than 90 min was not possible or because written informed consent for invasive treatment was not provided; and group C, conservative treatment without reperfusion therapy (48 males, 42 females, mean age 67.8 ± 10.9 years) owing to late presentation of symptoms, duration of infarct pain more or less than 12 h but with contraindication(s) for fibrinolytic therapy, or because written informed consent for invasive treatment was not provided.

Myocardial infarction and definition of end-points

Demographic parameters and risk factors were analysed for all groups (Table 1). Myocardial infarction was defined based on ESC/American College of Cardiology (ACC) guidelines [1]: (1) duration of pain more than 30 min; (2) a necrotic myocardial marker (troponins or myocardial fraction of creatinine phosphokinase [CK-MB]) at least twice the upper limit; or (3) ST segment elevation in two continuous leads II, III, or aVF of at least 1 mm (60 ms) after the J point.

The duration of infarction pain was defined as the time from initiation of pain to the time of first medical contact. Death and cardiogenic shock were recorded, and the composite end-point was defined as death or cardiogenic shock not ending with death. Heart failure was diagnosed based on the presence of pulmonary rales, pulmonary oedema, chest X-ray, third heart sound, or peripheral oedema. The presence of a second- or third-degree atrio-ventricular block (AVB II/III degree), paroxysmal atrial fibrillation (PAF), sustained ventricular tachycardia (VT), and ventricular fibrillation (VF) was also noted.

ECG analysis

The first ECG was analysed at a paper speed of 25 mm/s, and the time from onset of pain to ECG was recorded. ST segment changes (elevation or depression) were measured manually at 60 ms after the J point, and a magnitude of 1 mm was considered significant. The magnitude of ST segment

Table 1. Patient clinical and demographic characteristics

	Group A (n = 134)	Group B (n = 96)	Group C (n = 90)	All patients (n = 320)	P*
Age [years]	66.1 ± 10.6	62.7 ± 12.3	67.8 ± 10.9	65.5 ± 11.3	NS
Gender (women/men)	81/53	77/19	48/42	206/114	NS
Hypertension	83 (61.9%)	40 (41.7%)	36 (40%)	159 (49.7%)	NS
Hypercholesterolaemia	91 (67.9%)	50 (52.1%)	49 (54.4%)	190 (59.3%)	NS
Smoking	45 (33.6%)	50 (52.1%)	28 (31.1%)	123 (38.4%)	NS
Obesity	24 (17.9%)	14 (14.6%)	4 (4.44%)	42 (13.1%)	NS
Diabetes mellitus	25 (18.7%)	17 (17.7%)	12 (13.3%)	54 (16.9%)	NS

*p — group A *vs* group B *vs* group C

changes in lead aVR was relative to the TP segment as a baseline. The characteristics of ST segment changes in the II, III, aVF, and aVR leads were analysed.

Statistical analysis

Categorical variables are expressed as numbers or percentages of patients, and continuous variables as medians ± SD. For comparison of categorical variables, the χ^2 test or the χ^2 test with Yates' correction if appropriate was used. The Mann-Whitney test was used for comparison of numerical variables. All clinical and demographic variables underwent logistic regression analysis to identify independent predictors of ST segment elevation. Odds ratios and 95% confidence intervals were calculated. A two-tailed p value of less than 0.05 was considered to indicate significance. Analyses were performed using Statistica PL software (StatSoft 8.0, StatSoft Polska, Krakow, Poland).

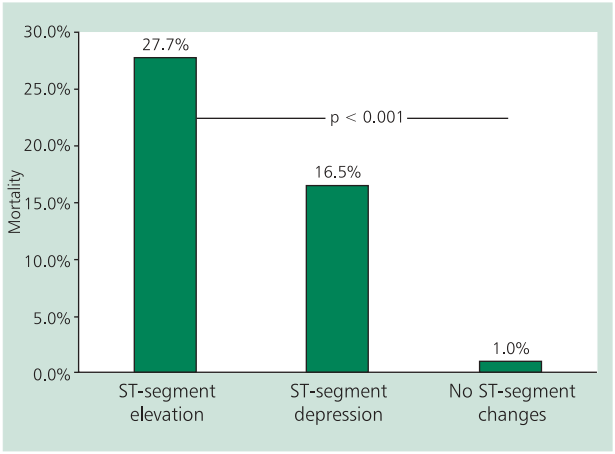

Figure 1. The relationship between ST segment changes in lead aVR and in-hospital mortality

RESULTS

Of the total of 320 patients, 230 underwent reperfusion therapy (72%, 160 males). One hundred and thirty four patients underwent primary PCI treatment (group A), 96 patients underwent fibrinolytic therapy (group B), and 90 patients underwent conservative treatment (group C). The time from pain onset to first medical contact was 6.1 ± 3.5 h for the overall 320 patients, 4.0 ± 3.7 h for group A, 3.5 ± 2.8 h for group B, and 8.6 ± 4.5 h for group C ($p < 0.04$ for group A *vs* group C and for group B *vs* group C).

During hospitalisation, 29 (9.0%) deaths occurred, with 11 (8.2%) in group A, seven (7.3%) in group B, and 11 (12.2%) in group C ($p > 0.05$). The maximal creatine phosphokinase (CPK) levels in groups A, B, and C were 2,021 ± 1,837 U/L, 1,734 ± 1,581 U/L, and 1,217 ± 981 U/L, respectively ($p = 0.01$ for group A *vs* group C).

There were ST segment changes in lead aVR in 135 (42.2%) patients. Those changes comprised elevation in 47 (14.7%) patients and depression in 88 (27.5%) patients.

The in-hospital mortality rates of patients with ST segment elevation, ST segment depression, and no ST segment changes were 27.7%, 16.5%, and 1.0%, respectively ($p < < 0.001$; Fig. 1). The percentage of patients with ST segment elevation, ST segment depression, and with no ST segment change who reached the composite end-point (death or cardiogenic shock) was 31.9%, 27.0%, and 3.2%, respectively ($p < 0.001$).

Patients with ST segment depression in lead aVR had higher CPK levels than patients with ST segment elevation in lead aVR (max. CPK: 2,375 ± 2,012 *vs* 1,342 ± ± 1,342 U/L, $p = 0.003$), and higher CPK than patients with no changes in lead aVR (max. CPK: 2,375 ± 2,012 *vs* 1,563 ± 1,422 U/L, $p = 0.0045$). Logistic regression analysis identified age, infarct localisation, ejection fraction, cardiogenic shock on admission, VF/VT, AVB III degree, diabetes mellitus and ST segment changes (elevation or depression) in lead aVR as independent predictors of in-hospital mortality (Table 2).

Table 2. Logistic regression analysis of independent predictors of in-hospital mortality

Variable	95% confidence interval	Odds ratio	P
Gender	0.77–4.1	1.78	0.17
Age	1.01–1.09	1.05	0.017
Ventricular tachycardia/ventricular fibrillation	1.51–11.75	4.2	0.006
Diabetes	1.08–5.83	2.53	0.032
AV block III degree	1.35–8.16	3.31	0.009
Cardiogenic shock	8.55–52.58	21.2	0.00000
Heart failure	0.95–4.63	2.1	0.06
Creatine phosphokinase	0.99–1.00	0.99	0.83
Left ventricular ejection fraction [%]	0.85–0.98	0.92	0.01
ST segment changes in lead aVR	5.46–101.61	23.56	0.000003

Outcomes according to therapy and ST segment changes

In group A, 51 (38%) patients had ST segment changes in lead aVR, comprising elevation in 13 (9.7%) patients and depression in 38 (28.3%) patients; there was no ST segment change in 83 (61.9%) patients. The in-hospital mortality rates were higher for ST segment elevation patients and for ST segment depression patients than for patients with no ST segment changes in lead aVR (Fig. 2).

In group B, 43 (44.8%) patients had ST segment changes in lead aVR, comprising elevation in nine (9.4%) patients and depression in 34 (35.4%) patients; there was no ST segment change in 53 (55.2%) patients. The in-hospital mortality rate for patients with ST segment elevation was higher than that for patients with ST segment depression and for patients with no ST segment changes (Fig. 3). The percentage of patients who reached the composite end-point (death or cardiogenic shock) was greater in patients with ST segment elevation (33.3%) than in patients with ST segment depression (25.8%) or patients with no ST segment changes (3.6%, $p = 0.002$ for all).

In group C, 41 (45.6%) patients had ST segment changes in lead aVR, comprising elevation in 25 (27.8%) patients and depression in 16 (17.8%) patients, and no ST segment changes were observed in 49 (54.4%) patients. The in-hospital mortality rate of patients with ST segment elevation was higher than that for patients with ST segment depression and for patients with no ST segment changes (Fig. 4). The percentage of patients who reached the composite end-point was greater in patients with ST segment elevation (36%) than in patients with ST segment depression (25%) and patients with no ST segment changes in lead aVR (4%, $p = 0.002$ for all).

Clinical parameters and ST segment changes in lead aVR

Logistic regression analysis showed that the independent predictors of ST segment elevation in lead aVR were female gender, diabetes, hypertension, lower ejection fraction, and cardiogenic shock on admission (Table 3).

DISCUSSION

The use of fibrinolytics to treat inferior wall STEMI is associated with a mortality rate of 2–9% [17–19]. However, almost all large clinical trials have shown that fibrinolytic treatment of inferior wall MI has no effect on mortality [3, 17–19]. In ECG analysis, all leads except aVR are considered for recognition of MI and localisation of STEMI [20].

ST segment depression in lead aVR

Several studies have examined ST segment changes in lead aVR in inferior wall STEMI [16, 21–25]. Senaratne et al. [25] found that patients with inferior wall STEMI more com-

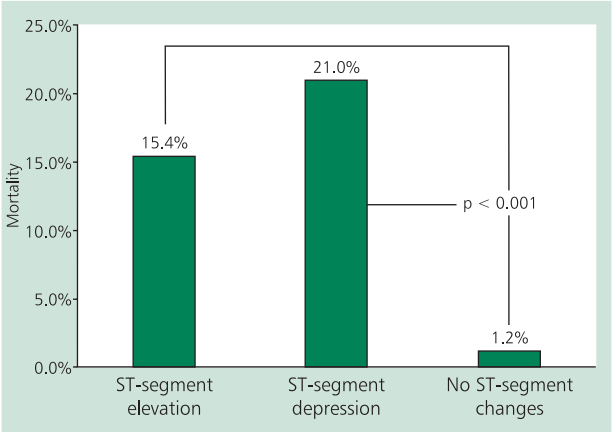

Figure 2. The relationship between ST segment changes in lead aVR and in-hospital mortality of patients treated with primary percutaneous coronary intervention (group A)

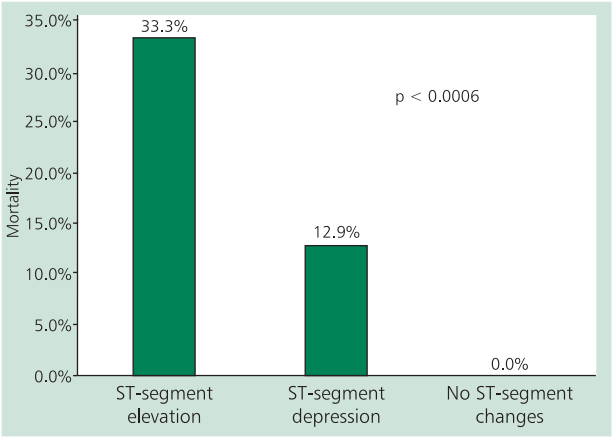

Figure 3. The relationship between ST segment changes in lead aVR and in-hospital mortality of patients treated with fibrinolysis (group B)

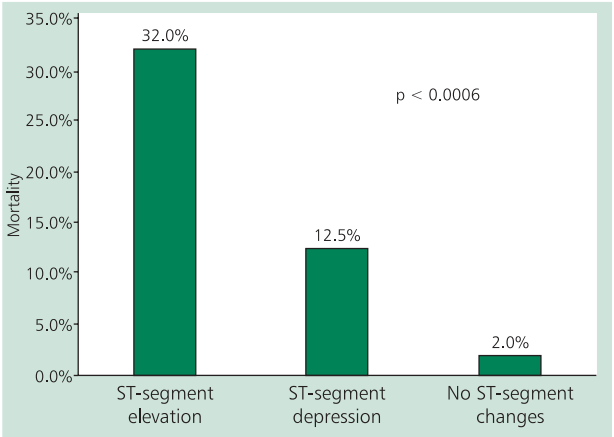

Figure 4. The relationship between ST segment changes in lead aVR and the mortality of patients not treated with reperfusion therapy (group C)

Table 3. Logistic regression analysis of independent predictors of ST segment elevation in lead aVR

Variable	95% confidence interval	Odds ratio	P
Gender	2.1–53.2	10.8	0.003
Smoking	0.07–1.75	0.35	0.20
Obesity	0.04–3.5	0.41	0.41
Diabetes	1.12–13.44	4.7	0.032
Hypertension	1.04–125.24	11.4	0.043
Cardiogenic shock	1.2–184.19	14.9	0.032
Heart failure	0.53–6.36	1.8	0.32
Creatine phosphokinase	0.99–1.00	1.0	0.89
Left ventricular ejection fraction [%]	0.88–1.00	0.94	0.84
Multivessel coronary disease	0.17–2.10	0.61	0.43

monly presented with ST segment depression in lead aVR than patients with anterior wall STEMI (20.6% *vs* 5.8%, $p < 0.05$). In the present study of inferior wall STEMI, we found that ST segment depression was present in 26.6% of patients.

We found that patients with ST segment depression in lead aVR had a higher CPK level compared to patients with no ST segment depression in lead aVR (2,375 ± 2,012 *vs* 1,563 ± 1,422 U/L, $p = 0.004$). Senaratne at al. [25] found that although patients with inferior STEMI (181 patients) with ST segment depression in lead aVR appeared to have a higher CPK level compared to patients with no ST segment depression in lead aVR, statistical analysis showed that the levels were not significantly different (2,196 ± 368 U/L *vs* 1,566 ± 151 U/L, $p = 0.79$). It may be that the difference would have become significant if a larger number of patients had been used in that study.

In the present study, patients with ST segment depression in lead aVR had higher rates of death (16.5% *vs* 1.0%), composite end-point (27.0% *vs* 3.2%) and VF (12.1% *vs* 4.8%) than patients with no ST segment changes in lead aVR ($p < 0.001$ for all). Moreover, that trend was independent of the type of therapy.

ST segment depression in lead aVR is poorly characterised. Recent publications suggest that this phenomenon reflects ischaemia or injury in the region of the apex and/or the infero-lateral area [16, 21, 22, 24, 25]. Those areas are supplied with blood from the postero-lateral branch of the circumflex artery or an AV branch of the right coronary artery. In a study of patients diagnosed with inferior wall STEMI within 6 h of the onset of pain, Kosuge at al. [16] reported that ST segment depression in lead aVR was associated with impaired coronary perfusion despite thrombolysis in myocardial infarction grade 3 (TIMI-3) after primary PCI.

One study reported that the magnitude of ST segment depression in lead aVR was a strong predictor of impaired myocardial perfusion after successful primary PCI according to multivariate regression analysis [24]. ST segment depression in lead aVR could indicate impaired tissue perfusion, which is associated with poor prognosis even in patients with TIMI-3 flow after primary PCI [24]. It is likely that patients in the study by Kosuge et al. [24], as with the patients in our present study, had ST segment depression in lead aVR that correlated with a larger area of MI (hence the higher CPK levels), an event that is associated with a range of complications.

ST segment elevation in lead aVR

No previous study has examined ST segment elevation in lead aVR in inferior wall STEMI. However, there have been studies concerning the prognostic significance of ST segment changes in lead aVR in patients with non ST segment elevation myocardial infarction (NSTEMI) or anterior wall STEMI [13, 15, 26–29]. In our present study, we observed higher rates of death (27.7% *vs* 1.0%, $p < 0.001$), composite end-point (1.9% *vs* 3.2%, $p < 0.001$), VF (12.8% *vs* 4.8%, $p < 0.045$) and III degree AVB (21.3% *vs* 8.5%, $p < 0.01$) in patients with ST segment elevation compared to patients with no ST segment changes. Those trends were observed regardless of the type of reperfusion therapy. In our study population, three-vessel disease occurred in 33% of patients with ST segment elevation in lead aVR. The poor prognosis of the present patients who had ST segment elevation may be partly due to this high incidence of three-vessel CAD.

What influences ST segment elevation in lead aVR?

Our study found an association between hypertension and ST segment elevation in lead aVR (Table 1). Similarly, Rostoff and Piwowarska [26] showed that patients with ACS ST segment elevation in lead aVR were more likely to have hypertension. We also found that ST segment elevation was associated with female gender, diabetes, lower ejection fraction, and cardiogenic shock on admission (Table 1). Recent publications showed that ST segment elevation in lead aVR was

associated with three-vessel CAD or with left main disease, both of which predispose patients to cardiogenic shock in MI. The present study also found that diabetes and decreased left ventricular ejection fraction were associated with ST segment elevation, and both of those factors are well-known indicators for cardiogenic shock in MI. We found that female patients with ST segment elevation had poorer prognoses than male patients. This may be because women experience MI at an older age than men, and age is associated with an increased risk of death. Alternatively, women often have more accompanying disorders (e.g., hypertension, diabetes and obesity) that are risk factors.

Clinical significance of ST segment changes in lead aVR

The present study found that analysis of ST segment changes in lead aVR in inferior wall STEMI patients provided information that would assist in the classification of patient risk and prognosis. In particular, our study showed that patients with ST segment elevation or depression have a poor prognosis. Importantly, we also found that the incidence of ST segment changes in lead aVR did not depend on the reperfusion strategy (i.e., mechanical *vs* pharmacological). Based on these findings, we would classify a patient with inferior wall STEMI as being at high risk when an ECG shows ST segment changes (elevation or depression) in lead aVR. We suggest that such patients should be treated with primary PCI. It should be noted that even if the invasive therapy is successful, such patients still have an increased risk of death due to acute MI.

Limitations of the study

We found that mortality rates were similar for the two reperfusion groups. This may have been due to: (1) the large number of complications that occurred during hospitalisation, including cardiogenic shock, in the primary PCI group; (2) the time from onset of pain to treatment being only 3.5 h for the fibrinolytic group, which could be associated with their favourable prognosis; and/or (3) the sample sizes being too small to detect significant differences. Furthermore, the study only included inferior wall STEMI patients; as large clinical trials have shown, there is no benefit of fibrinolytic therapy over placebo, and no benefit of primary PCI over fibrinolysis during the hospital stay or in terms of long-term mortality in these patients [30, 31].

CONCLUSIONS

ST segment changes in lead aVR were present in approximately half of all inferior wall STEMI patients, with depression being more common than elevation. Those ST segment changes were found to be associated with a poor prognosis during the hospital stay. Finally, patient outcomes were not influenced by the type of reperfusion treatment.

Conflict of interest: *none declared*

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Znaczenie prognostyczne zmian odcinka ST w odprowadzeniu aVR u chorych z zawałem ściany dolnej z uniesieniem odcinka ST

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Streszczenie

Wstęp: Zawał serca z uniesieniem odcinka ST (STEMI) ściany dolnej uznaje się za zawał niskiego ryzyka, mimo że 30–40% zawałów ściany dolnej to zawały wysokiego ryzyka związane z gorszym rokowaniem. Wyniki ostatnich badań sugerują, że obecność uniesienia odcinka ST w odprowadzeniu aVR wiąże się z przebiegiem klinicznym zawału serca bez uniesienia odcinka ST.

Cel: Celem pracy było ustalenie, czy zmiany odcinka ST w odprowadzeniu aVR mają związek z rokowaniem krótkoterminowym i przebiegiem klinicznym STEMI ściany dolnej w zależności od metody leczenia reperfuzyjnego.

Metody: Do analizy retrospektywnej włączono 320 kolejnych chorych z zawałem ściany dolnej, 206 mężczyzn i 114 kobiet, w średnim wieku 65,6 ± 11,1 roku. Chorych podzielono na 3 grupy: Grupa A — leczona inwazyjnie za pomocą pierwotnej angioplastyki wieńcowej (PCI) (n = 134; 81 mężczyzn i 53 kobiet, śr. wiek 66,1 ± 10,6 roku); Grupa B — leczona fibrynolitycznie (n = 96; 77 mężczyzn i 19 kobiet, śr. wiek 62,7 ± 12,3 roku); Grupa C — bez leczenia reperfuzyjnego (n = 90; 48 mężczyzn i 42 kobiety, śr. wiek 67,8 ± 10,9 roku). Leczenie reperfuzyjne zastosowano u 230 (72%) chorych. Czas od początku wystąpienia bólu zawałowego do wykonania EKG wynosił 6,1 h. Podczas obserwacji szpitalnej stwierdzono łącznie 29 (9,0%) zgonów, w grupie A — 11 (8,2%), w grupie B — 7 (7,3%), a w grupie C — 11 (12,2%) (p = NS). Maksymalne stężenie kinazy fosfokreatynowej wynosiło 2021 ± 1837 j./l w grupie A, 1734 ± 1581 j./l w grupie B oraz 1217 ± 981 j./l w grupie C (p = 0,01). Frakcja wyrzutowa lewej komory wynosiła odpowiednio: 50,2% ± 9,0%; 54,9% ± 8,6% i 51,3% ± ± 9,7% (p = NS).

Wyniki: Zmiany odcinka ST w odprowadzeniu aVR obserwowano łącznie u 135 (42,2%) chorych, w tym uniesienie odcinka ST u 47 (14,7%), a obniżenie odcinka ST u 88 (27,5%) pacjentów. Śmiertelność była istotnie wyższa w grupie osób z uniesieniem odcinka ST w odprowadzeniu aVR (13 chorych; 27,7%) w porównaniu z grupą z obniżeniem odcinka ST (14 chorych; 16,5%) oraz z grupą bez zmian odcinka ST w aVR (2 chorych; 1%); p < 0,001. W grupie A zanotowano znamiennie większą śmiertelność u chorych z uniesieniem odcinka ST w aVR w porównaniu z pacjentami bez zmian odcinka ST w odprowadzeniu aVR (15,4% *v.* 1,2%; p < 0,001). W grupie B śmiertelność wewnątrzszpitalna wynosiła: 33,3%, 12,9% i 0% (p = 0,006), w grupie C — 32,0%; 12,5% i 2% (p = 0,006), odpowiednio u chorych z uniesieniem odcinka ST, obniżeniem odcinka ST i brakiem zmian odcinka ST w odprowadzeniu aVR. W analizie regresji wieloczynnikowej niezależnymi predyktorami uniesienia odcinka ST były: płeć żeńska, cukrzyca, nadciśnienie tętnicze, niższa frakcja wyrzutowa, wstrząs kardiogenny przy przyjęciu.

Wnioski: Zmiany odcinka ST w odprowadzeniu aVR, zarówno uniesienie, jak i obniżenie, są częste w STEMI ściany dolnej i występują łącznie u prawie połowy chorych. Obecność zmian odcinka ST w odprowadzeniu aVR w zawale ściany dolnej wiąże się z gorszym rokowaniem w trakcie hospitalizacji niezależnie od rodzaju terapii i rodzaju zastosowanego leczenia reperfuzyjnego.

Słowa kluczowe: zawał serca ściany dolnej, odprowadzenie aVR, elektrokardiogram

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