Modern Medicine. 2015, Vol. 22, No. 3 : 204-212

Original Paper

The Evolution of Electrocardiographic Changes after Revascularization Therapy in Patients with ST Segment Elevation Myocardial Infarction

Magda Ruxandra Zaharia[1], Oliviana Geavlete[2], Alice Brînzea[3], Oana Tăutu[4], Gheorghița Aron[1], Ioan Tiberiu Nanea[5]

[1]"Carol Davila" University of Medicine and Pharmacy, Department of Internal Medicine, Saint John Hospital Bucharest, Romania
[2]"Carol Davila" University of Medicine and Pharmacy, Department of Cardiology, Institute of Emergency for Cardiovascular Diseases " Prof. C.C. Iliescu", Bucharest
[3]"Carol Davila" University of Medicine and Pharmacy, Pathophysiology Department II, Clinical Department 2, Bucharest, Romania
[4]"Carol Davila" University of Medicine and Pharmacy, Department of Cardiology, Clinical Emergency Hospital, Bucharest Romania
[5]"Carol Davila" University of Medicine and Pharmacy, Department of Cardiology, "Theodor Burghele" Hospital Bucharest, Romania

REZUMAT

Evoluția modificărilor electrocardiografice post-revascularizație la pacienții cu infarct miocardic acut cu supradenivelare de segment ST

Obiective: Scopul studiului de față a constat în estimarea necrozei miocardice inițiale și a calității reperfuziei prin prisma modificărilor electrocardiografice (ECG) la pacienții cu infarct miocardic acut cu supradenivelare de segment ST (STEMI).

Metodologie: S-a urmărit dinamica supradenivelării segmentului ST și a amplitudinii undei R atât la internare cât și după terapia de repefuzie (în 3 momente diferite). Au fost evaluate datele demografice, intervalul de la debutul durerii până la prezentare, comorbiditățile asociate, factorii de risc, parametri ecocardiografici, evenimentele cardiovasculare și decesul în timpul spitalizării.

Rezultate: Modificările magnitudinii supradenivelării segmentului ST din teritoriul infarctat s-au corelat în mod indirect dar de o manieră semnificativă cu modificările amplitudinii undei R atât la internare cât și în primele 48 de ore după revascularizație. Creșterea supradenivelării segmentului ST, întâlnită la pacienții prezentați tardiv, a fost asociată scăderii amplitudinii undei R, ambele fiind legate de evenimente cardiovasculare majore, fracție de ejecție a ventriculului stâng redusă și mortalitate crescută.

Corresponding author: Magda RuxandraZaharia, "Saint John" Hospital Bucharest, Internal Medicine Department Vitan-Bîrzesti Street no.13, sector 4, Bucharest, e-mail: magda_mirescu31@yahoo.com

Concluzii: Amplitudinea undei R la pacienții STEMI oferă informații suplimentare cu implicații clinice deosebite. Relația dintre segmentul ST și unda R ar putea reprezenta o provocare în estimarea perfuziei miocardice și a rezultatelor clinice.

Cuvinte cheie: supradenivelarea segmentului ST, unda R, STEMI, terapia de revascularizație

ABSTRACT

Objectives: The purpose of the study resides in estimating initial myocardial necrosis as well as reperfusion quality in ST segment elevation myocardial infarction(STEMI) patients involving electrocardiographic (ECG) changes.
Methodology: The dynamics of both ST segment elevation and R wave amplitude was followed in 146 STEMI cases upon admission and after reperfusion therapy, in 3 different moments. Demographics, the symptom onset to presentation interval, co-morbidities, traditional risk factors, echocardiography findings, cardiovascular events and mortality during hospitalization were assessed.
Results: The changes in ST-segment elevation magnitude and R-wave amplitude characterizing the infarction territory upon admission and up to 48 hours after revascularization were described as indirectly correlated in a significant manner.Therefore, the increased ST elevation found in late admission patients was associated with a decreased R wave amplitude, both being related to major cardiovascular events, poor left ventricular function and increased mortality.
Conclusions: The R wave amplitude in STEMI patients provides additional information with important clinical implications. The relationship between this parameter and the ST segment mayrepresent a challenge in estimating myocardial perfusion and eventually improved outcomes.

Key words: ST segment elevation, R wave, STEMI, revascularization therapy

INTRODUCTION

Despite the advanced technologies, the 12 leads electrocardiogram (ECG) remains an important investigation modality for providing a fast diagnostic of acute coronary syndromes (ACS). This method offers data concerning the presence, extension and severity characterizing the ischemic process (1). The ECG interpretation is still essential during the initial evaluation of patients admitted for ischemia suggestive symptoms (2).

Moreover, being a cheap, non-invasive and accessible technique, ECG continues to represent the gold-standard alternative for the differential diagnostic, for determining the appropriate treatment approach, for selecting patients susceptible of benefiting from reperfusion as well as regarding risk stratification (1).

Time is crucial during ST segment elevation myocardial infarction (STEMI). Time between the onset of symptoms and presentation or treatment can, at best, determine the duration of the infarct, but it cannot provide information about how much of the myocardium has been affected. Early restoration of patency and adequate anterograde reflow in the infarct-related artery has been the primary objective of treatment strategies in STEMI patients. The ECG signs of reperfusion constitute a valuable marker with regard to the micro-vascular flow as well as to the subsequent prognostic (3).

A detailed analysis of the ST segment' s elevation pattern may influence the therapeutic decisions concerning the use of reperfusion techniques (3). In acute myocardial infarction (AMI), the surface ECG may quantify the risk of major adverse events by estimating the size of the affected myocardial area. This aspect could help selecting patients that would benefit the most from reperfusion therapy (Hein J.J. Wellens, 2002).

The main objective of immediate risk assessment after admission is to offer a reliable stratification of AMI cases as well as to identify high risk patients and to predict complications (4,5,6). Ultimately, establishing the categories of patients marked by an

increased risk of major cardiovascular events secondary to AMI remains remarkably important with regard to prevention (7). The highest risk occurs during the early post-AMI period (8) and some ECG changes may be useful from this perspective (9).

Acute myocardial ischemia can affect all electrical activation components 2 by modifying the segments and waves of both depolarization and especially repolarization (QRS complex and ST-T changes, respectively) (H. Engblom, 2011) (10). Typically, the most significant ECG modifications induced by myocardial ischemia (secondary to the lesion current generated by voltage gradients between ischemic and non-ischemic areas) are related to the ST segment (11).

The anomalies characterizing the ST segment and the T and Q waves were recommended as relevant for the standard definitions by the present diagnostic and treatment guidelines along with clinical profile, biomarkers and cardiac catheterization outcomes (12,13). On the other hand, the actual value of these changes concerning the prediction of short as well as long term mortality and morbidity remains rather debatable (14).

The purpose of the present study resides in estimating myocardial reperfusion quality as well as the quantification of salvaged myocardium after revascularization therapy, correlated with ECG dynamics in STEMI patients. The analyzed changes imply both ST segment elevation magnitude and R wave amplitude as expressions of the ischemic lesions and loss of myocardial mass.

MATERIALS & METHODS

The trial was based on a retrospective analysis which included 146 STEMI patients admittedfor chest pain (prolonged pain over 20 minutes with retrosternal or epigastric location, not favorably responding to nitroglycerine) and ECG changes in accordance with the present STEMI diagnosis guidelines (7). The latter parameter refers to ST segment elevation measured at J point in 2 contiguous leads ($\geq$ 0.25 mV in men aged below 40 and $\geq$ 0.2 in those over 40; $\geq$ 0.15 mV in V2-V3 leads and/or $\geq$ 0.1 mV in other leads for women). Subsequent to angiography, solely patients with TIMI 0 and 1 flow were selected.

Patients with Q wave on the initial ECG, history of myocardial infarction, intra-ventricular conduction disturbances, left ventricular hypertrophy, Killip grade IV heart failure, echocardiographically visible ventricular aneurysm and angiographically established TIMI 2-3 flow were excluded from the study.

The selected series was analyzed with regard to demographics, medical history, associated co-morbidities, previous outpatient therapyandthe number of hospital days. The assessed features consisted in the time passed from the onset of chest pain to first medical exam, ECG upon hospital admittance and after revascularization therapy in the first 24 and 48 hours as well as 5 days. There were determined the STEMI location, magnitude of the ST segment elevation as well as the R wave amplitude at the specified moments.

In accordance with the European guideline for STEMIdiagnosis and management (7), the ST segment elevation was measured at J point above the isoelectric line. Moreover, the R wave amplitude was represented by the vertical distance from baseline (defined as two P-R consecutive intervals) to peak, in every ECG lead.

Echocardiography upon admittance and after revascularization was performed in all cases. There were assessed parameters such as the presence of ventricular aneurysm, left ventricular ejection fraction (LVEF – using Simpson biplane method), the segmental kinetics' anomalies and associated valvulopathies (e.g. mitral regurgitation). Specific cardiac biomarkers like troponin (Tn), creatine-kinase (CK), isoenzyme creatine kinase-MB (CK-MB)were determined. Angiography was applied at 60-120 minutes from diagnostic in all patients identifying the infarct-related artery. Mostincluded patients underwent percutaneous coronary intervention (PCI) with stent implantation and subsequent successful reperfusion (final TIMI-3 flow). The clinical evolutions of patients after revascularization therapy as well as the associated cardiovascular events during hospitalization were traced.

The statistical analysis was performed using the IBM SPSS Statistics 20.0 software at a significance level of $p \leq 0.05$, while the ANOVA or Kriskal-Wallis test (depending on the normality of continuous data distribution assessed by Kolmogorov-Smirnov test) and Chi-square test were applied. Binary logistic regression was used in order to assess the association between ECG changes (ST-segment elevation and R-wave amplitude variations in the infarction territory) and the target variables.The values were presented as mean $\pm$ s.d.

(standard deviation) for continuous data and absolute numbers (percent) for categorical data.

RESULTS

General characteristics of the study sample

The study sample was divided into 3 subgroups according to the time passed from pain onset to the first medical contact as following: Group 1 (less than 3 hours) – 24 patients; Group 2 (between 3 and 6 hours) – 57 patients; Group 3 (6 to 12 hours) – 65 patients; (**Fig. 1**).

There was no statistically significant difference between the 3 study subgroups regarding demographics, cardiovascular risk factors or medical history. The only exception was constituted by diabetes mellitus, which was more frequent among Group 3 cases (**Table 1**).

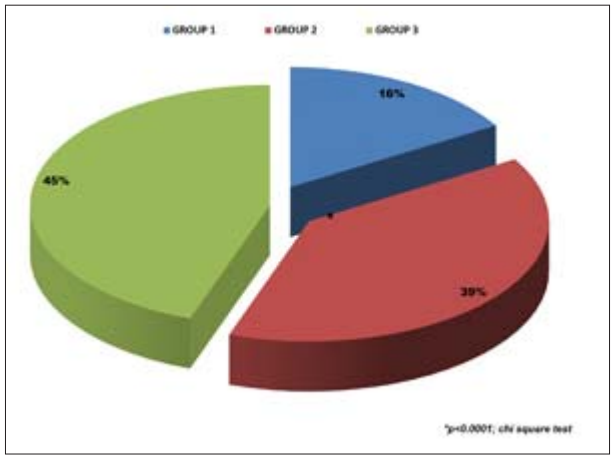

Figure 1. Study subgroups according to time from symptom onset to first medical contact

Infarction related characteristics

Although Killip classes I-III were statistically similar among the 3 study subgroups, the associated ventricular arrhythmias and intra-ventricular conduction disorders were statistically more frequent among late admission STEMI patients. Both the anterior and inferior STEMI locations were recorded in statistically similar proportions among the 3series. Moreover, there was no significant difference among study arms regarding the infarction-related artery. Furthermore, similar mean values of Tn, CK and CK-MB were recorded upon admission. Significantly lower LVEF levels were found in the delayed presentation (6 – 12 hours) STEMI cases. (**Table 2**)

Table 1. General characteristics of the 3 study subgroups

	Group 1 N = 24	Group 2 N = 57	Group 3 N = 65	p
Age (years)	53.4±12.7	56.8±13.5	60.5±14.9	NS*
Gender				
Females	2 (7.1)	12 (42.9)	14 (50.0)	NS**
Males	22 (18.6)	45 (38.1)	51 (43.2)	NS**
CV risk factors				
Smoking	17 (21.3)	33 (41.3)	30 (37.5)	NS**
Obesity	3 (12.5)	12 (50.0)	9 (37.5)	NS**
HTA	22 (17.9)	47 (38.2)	54 (43.9)	NS**
DM	11 (28.2)	9 (23.1)	19 (48.7)	0.017**
Dyslipidemia	17 (20.0)	32 (37.6)	36 (42.4)	NS**
Medication	6 (13.6)	12 (27.3)	26 (59.1)	NS**
β-blockers	4 (23.5)	5 (29.4)	8 (47.1)	NS**
ACEi	4 (16.7)	9 (37.5)	11 (45.8)	NS**
Antiplatelet	4 (22.2)	5 (27.8)	9 (50.0)	NS**
Statins	4 (36.4)	3 (27.3)	4 (36.4)	NS**
Nitrates	1 (14.3)	3 (42.9)	3 (42.9)	NS**

HTA: arterial hypertension; DM: diabetes mellitus; CV: cardio-vascular; ACEi: angiotensin-converting-enzyme inhibitor; *ANOVA; **chi square test; NS: non-statistically significant differences (p≥0.05).

Table 2. Infarction-related characteristics of the study subgroups

	Group 1 N = 24	Group 2 N = 57	Group 3 N = 65	p
Killip 1	12 (12.9)	35 (37.6)	46 (49.5)	NS*
Killip 2	11 (23.4)	19 (40.4)	17 (36.2)	NS*
Killip 3	1 (16.7)	3 (50.0)	2 (33.3)	NS*
Associated phenomena	5 (7.4)	28 (41.2)	35 (51.5)	0.019*
STEMI localization				
Anterior	10 (13.7)	26 (35.6)	37 (50.7)	NS*
Inferior	14 (19.2)	31 (42.5)	28 (38.4)	NS*
IRA				
LAD	10 (13.7)	26 (35.6)	37 (50.7)	NS*
RCD	14 (22.2)	24 (38.1)	25 (39.7)	NS*
CX	0 (0.0)	6 (75.0)	2 (25.0)	NS*
Other	0 (0.0)	1(100.0)	0 (0.0)	NS*
Tn (ng/ml)	0.7±1.9	0.6±0.8	0.8±1.6	NS**
CK-MB	71.7± 82.3	106.5± 149.7	147.4± 307.1	NS**
LVEFad	43.7±6.5	44.2±6.7	41.5±6.6	0.048**

STEMI: ST-segment elevated myocardial infarction; IRA: infarction related artery; LAD: left anterior descending artery; RCD: right coronary artery; CX: circumflex artery; Tn: cardiac troponin; CK-MB: izoenzymecreatin kinase; *chi square test; LVED ad: left ventricular ejection fraction on admittance; **Kruskal-Wallis test; NS: non-statistically significant differences (p≥0.05).

On the other hand, there was no significant difference among subgroups regarding revascularization therapy, PCI with stent implantation being the reperfusion method used in the majority of cases (group 1: 22 cases (15.9%); group 2: 55 cases (39.9%); group 3: 61 cases (44.2%); p = 0.650).

Patients' evolution during hospitalization

After a similar mean number of hospitaldays, there was no significant difference between the 3 series regarding both LVEF on discharge and cardiovascular events during hospitalization (recurrent angina, left ventricular failure, arrhythmias and death). Severe ischemic mitral regurgitation was significantly more frequent among the subgroup 3' patients (Table 3).

The evolution of ST segment elevation after myocardial revascularization

Upon admission, the highest ST-segment elevation in the infarction territory was recorded among late presentation patients. Both at 24 and 48 hours after revascularization, the ST-segment elevation was statistically similar among the 3 study subgroups. The same situation was also noticed at 5 days after revascularization. (Table 4)

By comparison to the ST-segment elevation measured on admission, at 24 hours after revascularization, a ST-segment elevation decrease was recorded in 85 cases (72%), an increase in 12 patients (10.2%), and a stationary evolution of the respective parameter in the remaining 21 cases (17.8%). At 48 hours after revascularization, a ST-segment elevation decrease was found in 96 cases (81.4%), a reduction of the parameter in question in 7 patients (5.9%), while in the remaining 15 cases (12.7%), a stationary outcome was found. Further along this line, 5 days from revascularization, a reduction in ST-segment elevation was recorded in 114 cases (90.5%), an increase in 5 cases (4%), and stationary results in the remaining 7 cases (5.6%).

There werestatistically significant differences among the 3 subgroups regarding variations in ST-segment elevation after myocardial reperfusion at 24 and 48 hours as well as 5 days, the majority of cases with a decrease of this feature being identified as part of subgroup 1. Also, the majority of patients with increased ST-segment elevation was described in the third study arm. (Fig. 2)

R-wave amplitude evolution in the infarct territory after myocardial revascularization

Both during admission as well as 24 hours after myocardial reperfusion, the mean R-waveamplitude value characterizing the infarction territory was similar among the 3 subgroups. Statistically significant differences between study arms were noticed at 2 and 5 days after revascularization, as the lowest R-wave amplitude was recorded among late admission patients. (Table 5)

By comparison to the R-wave amplitude at presentation, no significant differences were discovered concerning the evolution of this parameter at 24 and 48 hours after myocardial revascularization. As of

Table 3. Evolution during hospitalization of the 3 study subgroups

	Group 1 N = 24	Group 2 N = 57	Group 3 N = 65	p
LVEFdis	43.8±6.3	46.1±6.3	44.5±5.8	NS**
Mitral regurgitation				
Grade 1	9 (13.2)	35 (51.5)	24 (35.3)	0.032
Grade 2	14 (20.9)	20 (29.9)	33 (49.3)	0.032
Grade 3	1 (9.1)	2 (18.2)	8 (72.7)	0.032
CV events	3 (8.1)	15 (40.5)	19 (51.4)	NS**
Recurrent angina	1 (9.1)	3 (27.3)	7 (63.6)	NS**
LV failure	2 (11.8)	9 (52.9)	6 (35.3)	NS**
Arrhythmias	0 (0.0)	3 (60.0)	2 (40.0)	NS**
Death	1 (9.1)	4 (36.4)	6 (54.5)	NS**
Hospitalization (days)	7.88±62.7	8.1±2.6	7.9±2.7	NS**

LVEFdis: left ventricular ejection fraction on discharge; CV: cardiovascular; LV: left ventricle; **Kruskal-Wallis test; NS: non-statistically significant differences (p≥0.05)

Table 4. ST-segment elevation variation in the infarct territory across the 3 study arms

	Group 1 N = 24	Group 2 N = 57	Group 3 N = 65	p
STadm	1.8±0.9; 1.4-2.2	2.4±1.4; 1.9-2.9	2.7±0.9; 2.4-3.0	0.031*
ST24h	1.7±1.6; 1.0-2.4	1.7±1.4; 1.4-2.1	1.3±0.7; 1.1-1.5	NS**
ST48h	1.5±1.6; 0.9-2.2	1.14±1.0; 0.8-1.4	1.1±0.6; 0.9-1.2	NS**
ST5d	1.2±1.6; 0.6-1.9	0.9±0.9; 0.7-1.2	0.9±0.7; 0.7-1.1	NS**

Values: presented as mean ± s.d. (standard deviation); 95% – confidence interval for meanST; ST – segment elevation in mm; adm: upon admission; 24h: 24 hours after revascularization; 48h: 48 hours after revascularization; 5d: 5 days after revascularization; *ANOVA test; **Kruskal-Wallis test; NS: non-statistically significant differences (p≥0.05).

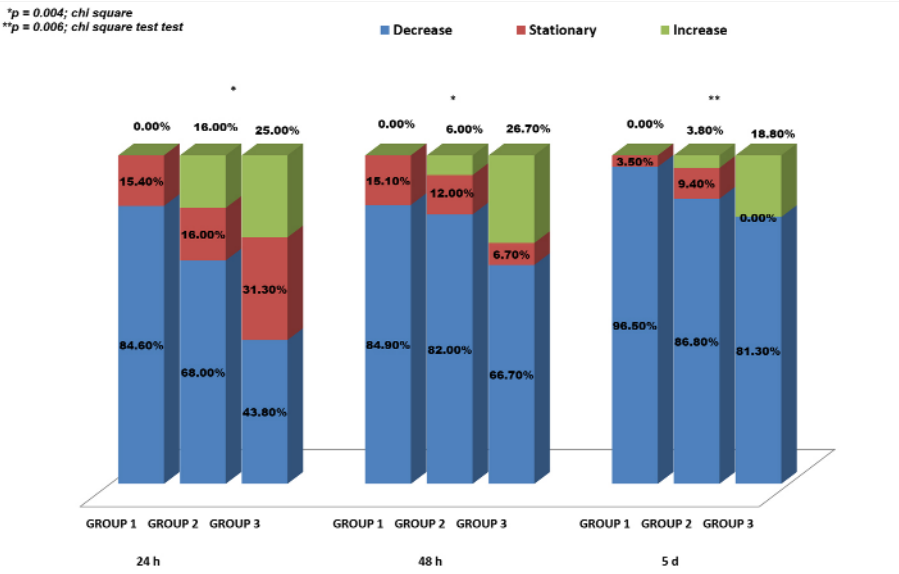

Figure 2. Evolution of ST-segment elevation in the infarcted territory after myocardial reperfusion

Table 5. R-wave amplitude variation in the infarction territory across the 3 study subgroups

	Group 1 N = 24	Group 2 N = 57	Group 3 N = 65	p
Radm	5.9±3.2; 4.6-7.3	5.7±3.4; 4.8-6.6	5.4±3.4; 4.6-6.3	NS*
R24h	3.1±1.7; 2.4-3.8	2.5±1.8; 2.0-2.9	2.4±1.6; 2.0-2.8	NS**
R48h	3.3±1.7; 2.5-3.9	2.5±2.1; 1.9-2.9	2.4±1.8; 1.9-2.9	0.003**
R5d	4.6±4.2; 2.7-6.4	2.6±2.3; 1.9-3.2	2.3±1.8; 1.8-2.8	0.003**

Values: presented as mean ± s.d. (standard deviation); 95% – confidence interval for meanR; Rwave– amplitude in mm; adm: on admission; 24h: 24 hours after revascularization; 48h: 48 hours after revascularization; 5d: 5 days after revascularization; *ANOVA test;**Kruskal-Wallis test;
NS: non-statistically significant differences (p≥0.05)

such, the proportion of cases with decreased, increased and stationary R-wave amplitude was statistically similar in the 3 series. At 5 days after reperfusion, the highest proportion of decreased R wave amplitude cases affecting the infarction territory was discovered among subgroup 3 cases. Last but not least, the highest proportion of increased R wave amplitude was emphasized among the early admitted STEMI patients (**Fig. 3**).

Correlations between ECG changes after myocardial revascularization

The changes in ST-segment elevation and R-wave amplitude characterizing the infarction territory at both 24 hours and 48 hours after revascularization

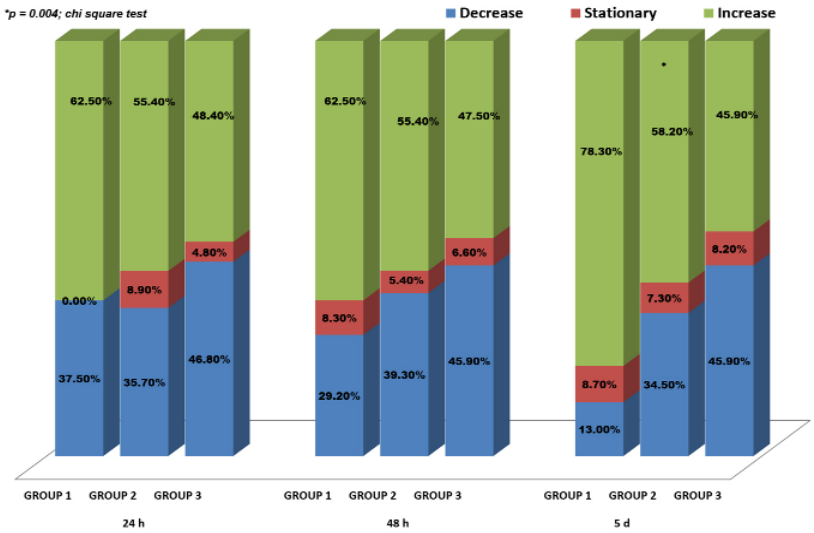

Figure 3. Evolution of R-wave amplitude in the infarction territory after myocardial reperfusion

were described as indirectly correlated (24h: rs = -0.312; rs2 = 0.097; p = 0.001; 48h: rs = -0.178; rs2 = 0.031; p = 0.045). On the other hand, at 5 days, the 2 variables were independent from each other (rs = -0.114; rs2 = 0.012; p = 0.207).

These correlations remained the same after adjustment for the 3 study subgroups.Therefore, the increased ST elevation found in subgroup 3 patients was associated with a decreased R wave amplitude, both being related to major cardiovascular events, poor left ventricular function and increased mortality (24 and 48 hours after revascularization).

DISCUSSIONS

ST segment elevation remains the most widely acknowledged ECG manifestation of acute transmural ischemia (2). In STEMI patients, ST segment characteristics offer early information, reproducible while aiming to establish the location of both the infarction related artery and the affected region by coronary occlusion (11).

The AMI management is specifically targeted to determine the culprit artery flow. ST segment analysis was also validated as apatency marker for epicardial artery as well as tissue myocardial perfusion (15). However, the normal epicardial flow does not always correlate with the microvascular perfusion of myocardic tissue (16).

We found the most significant ST segment elevation upon admission in patients presented late from the symptom onset (group 3), involving a higher risk of major events (arrhythmias, intraventricular conduction disorders, low LVEF) and a consequently negative outcome. Moreover, this category of cases emphasized a higher prevalence of diabetes mellitus which could explain the late presentation due to prolonged specific symptoms' onset.

Additionally, the ST segment elevation magnitude on admission and up to 48 hours after reperfusion-wasinversely correlated with R wave amplitude in late admission patients, a result susceptible of establishing the loss of myocardial mass. These ECG changes were also associated with post–infarction complications, lower LVEF and death during hospital stay.

From another perspective, the present result showed that the R wave could represent a negative prognostic outcome given the eventual association with ST segment analysis in high risk late admission patients.

ST segment elevation changes after reperfusion therapy

The spontaneous evolution of ECG modifications in STEMI patients became different after reperfusion therapies. As far as the ST segment elevation was concerned, the literature data underlined some aspects with regard to its' variability. The resolution of the ST segment elevation was considered a surrogate endpoint that could suggest the vessel recanalization, tissue perfusion restoration and myocardial salvage with prognostic implications (17).

Sometimes, it may offer more complex detailsby comparison to the angiographic parameters, as it estimates the reperfusion quality together with the metabolic and electrical activity resumption in the perfused area (18). ST segment elevation decrease after angioplasty is an independent predictor of LV function recovery (19).

The presented study demonstrated a ST segment elevation resolution in 72% of the total number of patients during the first 24 hours after reperfusion, 81.4% in 48 hours and 90.5% at 5 days after the revascularization. These outcomes are in fact related to the patients' ratio in which both the recanalization of the occluded vessel as well as a better coronary microcirculatory perfusion was achieved, with a progressive improvement from the revascularization moment.

The resolution degree is also a rather important feature, as the literature data indicated that patients with complete ST segment resolution (over 70%) at 24 hours after revascularization were characterized by a significantly lower prevalence of microvascular obstruction 20 when compared to the partial (30-70%) or absent (≤ 30%) resolution, thus presenting a better prognosis while concomitantly maintaining LV function (21,22).

The persistent ST segment elevation after reperfusion therapy may express a lack of response in the microcirculation of affected area (the „no-reflow" phenomenon), with persistent ischemic damage associating a high morbi-mortality, even in cases of successful coronary angioplasty (23,24). These patients were described as marked by a high prevalence of early post-infarction complications (such as arrhythmias, pericarditis, tamponade and congestive heart failure), impaired ventricular remodeling, late hospitalization for heart failure and mortality (25). Therefore, microvascular detection, prevention and treatment could have an important impact over PCI outcomes.

Despite obtaining a TIMI 3 flow, the present trial substantiated a persistent ST segment elevation after 24 hour in 17.8% of the studied cases, 12.7% at 48 hours and 6% at 5 days post-revascularization. The most probable cause was constituted by an impaired tissueperfusion associating an insufficient LV function recovery, an increased infarction size as well as a quite poor prognosis.

The increase in ST segment elevation after reperfusion therapy may indicate the presence of a progressive myocardial infarction or an inefficient reperfusion therapy (26). The ST segment elevation ascension could express supplementary ischemic myocardial damage having a negative effect upon the LV function recovery (27).

The outcomes of the study in question confirmed an increase in ST segment elevation in 10.2% of all cases during the first 24 hours after reperfusion, 5.9% in 48 hours and 4% at 5 days after reperfusion. The specific change was observed during the first 24 hours from reperfusion in patients admitted between 3 and 6 hours from the symptom onset (group 2). On the other hand, at 2 and 5 days, respectively, the increase was noticed in patients who presented early (group 1), thus showing the evolution of ischemia despite revascularization.

R wave amplitude changes after reperfusion therapy

As far as the R wave was concerned, the available literature confirmed the fact that its'amplitude, as a marker of myocardial damage severity, is directly related with the myocardial viability degree (28). Several studies demonstrated a significant correlation between imagistic myocardial viability and the R wave voltage in AMI cases (29). Ischemic progression up to transmural lesions development, otherwise a time dependent phenomenon, was associated with myocardial activity reduction and R wave amplitude significant decrease (29,30).

R wave changes on surface ECG were described both during ischemia as well as during AMI (31), since adecrease amplitude in anterior AMI was observed while brief transmural ischemia episodes determined a significant increase in precordial leads (32).

An "in vivo" study underlined correlations between a reduced R wave amplitude and increased myocardial infarction size both during the early as well as late ischemia stages. This is a remarkably relevantclinical aspect, making the R wave an early and easy predictor for minimizing the development of a severe AMI (32).

After a successful revascularization in such patients, an increase in R wave amplitude wasconsequently observed (29,30). Although the reversibility of the R wave voltage gathered a substantial amount of interest, the electrophysiological mechanisms behind its' variations still remained unknown.

The outcomes of the present study showedthat the R wave recorded the lowest amplitude in late admission patients (group 3). By comparison to the R wave amplitude at admission, 5 days after reperfusion, the highest amplitude increase was observed in early presented patients (group 1), thus suggesting the presence of myocardial viability as well as the improvement of ventricular function. On the other hand, the most important amplitude decrease as detected in late presentation patients (group 3) correlated with low LV function and a significantly higher incidence of associated events (ventricular arrhythmias and conduction disturbance) and death during hospitalization.

CONCLUSIONS

Surface ECG remains a landmark while evaluating the initial myocardial necrosis as well as the evolution after revascularization therapy. ST segment elevation was emphasized as the most important marker of myocardial reperfusion, associated with the myocardial tissue damage, functional recovery, frequency of congestive heart failure and adverse outcome. However, supplementary data, other than the conventional ST-T analysis, is definitely required to be evaluated.

Reversible depolarization changes in the R wave also appear, although they are less understood and usually not considered while determining the clinical decision. The R wave demonstrates dynamic changes during both coronary artery occlusion as well as after revascularization, thus correlating with the extent and severity of the necrosis.

The combination of ST segment and R wave parameters is rather useful while aiming to establish the quality of reperfusion in the affected perfusion bed and extent of the injured myocardium. This feature is also most likely to improve the predictive accuracy in patients undergoing PCI.

The present study found that changes in ST segment elevation and R wave amplitude in the infarction-related territory leads were indirectly

correlated upon admission and up to 48 hours after revascularization. These findings emphasized the fact that the R wave amplitude in STEMI cases could bring additional information with important clinical implications.

Further studies are required in order to examine whether assessing the relationship between ST segment and R wave in STEMI patients allows a superior prediction of myocardial perfusion improvement and clinical outcomes.

Conflict of interest

None declared.

REFERENCES

1. Antman EM, Cohen M, Bernink PJ, et al. The TIMI risk score for unstable angina/non-ST elevation MI: A method for prognostication and therapeutic decision making. JAMA. 2000;284(7):835-842.
2. Birnbaum Y, Wilson JM, Fiol M, de Luna AB, Eskola M, Nikus K. ECG diagnosis and classification of acute coronary syndromes. Ann Noninvasive Electrocardiol. 2014;19:4-14.
3. Zimetbaum PJ, Josephson ME. Use of the electrocardiogram in acute myocardial infarction. N Engl J Med. 2003;348(10):933-940.
4. Das MK, Khan B, Jacob S, Kumar A, Mahenthiran J. Significance of a fragmented QRS complex versus a Q wave in patients with coronary artery disease. Circulation. 2006;113(21):2495-2501.
5. Das MK, Michael MA, Suradi H, et al. Usefulness of fragmented QRS on a 12-lead electrocardiogram in acute coronary syndrome for predicting mortality. Am J Cardiol. 2009;104(12):1631-1637.
6. Carey MG, Luisi AJ, Baldwa S, et al. The Selvester QRS Score is more accurate than Q waves and fragmented QRS complexes using the Mason-Likar configuration in estimating infarct volume in patients with ischemic cardiomyopathy. J Electrocardiol. 43(4):318-325.
7. Steg PG, James SK, Atar D, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J. 2012;33:2569-2619.
8. De Araïjo Gonɳalves P, Ferreira J, Aguiar C, Seabra-Gomes R. TIMI, PURSUIT, and GRACE risk scores: sustained prognostic value and interaction with revascularization in NSTE-ACS. Eur Heart J. 2005;26(9):865-872.
9. Chatterjee S, Changawala N. Fragmented QRS complex: a novel marker of cardiovascular disease. Clin Cardiol. 2010;33(2):68-71.
10. Correa R, Arini PD, Correa LS, Valentinuzzi M, Laciar E. Novel technique for ST-T interval characterization in patients with acute myocardial ischemia. Comput Biol Med. 2014;50:49-55.
11. Nikus K, Pahlm O, Wagner G, et al. Electrocardiographic classification of acute coronary syndromes: a review by a committee of the International Society for Holter and Non-Invasive Electrocardiology. J Electrocardiol. 2010;43(2):91-103.
12. Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. Circulation. 2012;126(16):2020-2035.
13. Nakamura N, Gohda M, Satani O, et al. Myocardial salvage for ST-elevation myocardial infarction with terminal QRS distortion and restoration of brisk epicardial coronary flow. Heart Vessels. 2009;24(May 2002):96-102.
14. Ari H, Hetinkaya S, Ari S, Koca V, Bozat T. The prognostic significance of a fragmented QRS complex after primary percutaneous coronary intervention. Heart Vessels. 2012;27:20-28.
15. Weston P, Johanson P, Schwartz LM, Maynard C, Jennings RB, Wagner GS. The value of both ST-segment and QRS complex changes during acute coronary occlusion for prediction of reperfusion-induced myocardial salvage in a canine model. J Electrocardiol. 2007;40(1):18-25.
16. Ito H, Maruyama A, Iwakura K, et al. Clinical implications of the "no reflow" phenomenon. A predictor of complications and left ventricular remodeling in reperfused anterior wall myocardial infarction. Circulation. 1996;93(2):223-228.
17. De Lemos J a, Braunwald E. ST segment resolution as a tool for assessing the efficacy of reperfusion therapy. J Am Coll Cardiol. 2001;38(5):1283-1294.
18. Birnbaum Y, Ware DL. Electrocardiogram of acute ST-elevation myocardial infarction: The significance of the various "scores." J Electrocardiol. 2005;38(2):113-118.
19. Santoro GM, Antoniucci D, Valenti R, et al. Rapid reduction of ST-segment elevation after successful direct angioplasty in acute myocardial infarction. Am J Cardiol. 1997;80(6):685-689.
20. Husser O, Bodv V, Sanchis J, et al. The sum of ST-segment elevation is the best predictor of microvascular obstruction in patients treated successfully by primary percutaneous coronary intervention. Cardiovascular magnetic resonance study. Rev espaǫola Cardiol. 2010;63(10):1145-1154.
21. Vaturi M, Birnbaum Y. The use of the electrocardiogram to identify epicardial coronary and tissue reperfusion in acute myocardial infarction. J Thromb Thrombolysis. 2000;10(2):137-147.
22. Rodrvguez-Palomares JF, Figueras-Bellot J, Descalzo M, et al. Relation of ST-segment elevation before and after percutaneous transluminal coronary angioplasty to left ventricular area at risk, myocardial infarct size, and systolic function. Am J Cardiol. 2014;113(4):593-600.
23. Nable JV, Brady W. The evolution of electrocardiographic changes in ST-segment elevation myocardial infarction. Am J Emerg Med. 2009;27(6):734-746.
24. Klaus RS, Karl W, Uwe Z, Ulrich T, Schroder. Extent of ST-segment deviation in a single electrocardiogram lead 90 min after thrombolysis as a predictor of medium-term mortality in acute myocardial infarction. Lancet. 2001;358(9292):1479.
25. Niccoli G, Burzotta F, Galiuto L, Crea F. Myocardial no-reflow in humans. J Am Coll Cardiol. 2009;54(4):281-292.
26. D. Goldwasser JCAB de LMF-SAC. Clinical and Imaging Correlations and Prognostic Implications. In: Futura, ed. The Surface Electrocardiography in Ischaemic Heart Disease. Blackwell Publishing Ltd.; 2008:19-128.
27. Tatu Chiţoiu G. Dinamica segmentului ST ξn evaluarea prognosticului pacienţilor cu STEMI şi tratament de reperfuzie. In: Electrocardiograma Ξn Reperfuzia Miocardică. Editura Me.; 2014.
28. Tsai T-H, Sun C-K, Chung W-J, et al. Prognostic value of R-wave voltage in patients with anterior wall ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. Int Heart J. 2010;51(5):325-330.
29. Isobe S, Okada M, Ando A, et al. Clinical significance of changes in electrocardiographic R-wave Voltage on Chest Leads in Patients with acute anterior myocardial infarction. J Electrocardiol. 2002;35(3).
30. Isobe S, Takada Y, Ando A, et al. Increase in electrocardiographic R-waves after revascularization in patients with acute myocardial infarction. Circ J. 2006;70(November):1385-1391.
31. Sinno MCN, Kowalski M, Kenigsberg DN, Krishnan SC, Khanal S. R-wave amplitude changes measured by electrocardiography during early transmural ischemia. J Electrocardiol. 2008;41(5): 425-430.
32. Sun X, Cai J, Fan X, et al. Decreases in Electrocardiographic R-Wave Amplitude and QT Interval Predict Myocardial Ischemic Infarction in Rhesus Monkeys with Left Anterior Descending Artery Ligation. PLoS One. 2013;8(8).