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ORIGINAL ARTICLE

Electrocardiogram in Acute Myocardial Infarction: What to Expect?

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Abstract

Background: Cardiovascular diseases are the leading cause of death. The electrocardiogram (ECG) is an accessible and useful tool in the initial evaluation of acute coronary syndromes (ACS).

Objective: To identify and correlate electrocardiographic changes in different leads with the location of the intracoronary thrombus in the artery involved in the coronary event.

Methods: Retrospective and observational study conducted with 179 patients with ACS. The data were analyzed considering three diagnostic groups: unstable angina (n = 31), non-ST–elevation myocardial infarction (n = 86), and ST-elevation myocardial infarction of the anterior (n = 34) and inferior (n = 28) walls. Data obtained from ECG, coronary angiography, and transthoracic echocardiogram tests were analyzed and compared among the three groups.

Results: The sensitivity and specificity of the ECG in detecting the culprit coronary artery were 70.0% and 79.1%, respectively. The positive and negative predictive values for ECG location of the proximal segment of the left anterior descending (LAD) artery were 70.6% and 66.7%, respectively. Regarding the distal segment of the LAD, the positive and negative predictive values were 100.0% and 28.0%, respectively. With ECG analysis, we were able to identify the right coronary (RC) artery as the culprit artery in 88.9% of the cases, with positive and negative predictive values of 90.0% and 14.3%, respectively.

Conclusion: The ECG is an indispensable diagnostic method in ACS, even though it fails to locate the culprit artery accurately. (Int J Cardiovasc Sci. 2016;29(3):198-209)

Keywords: Myocardial Infarction / mortality; Electrocardiography / diagnosis; Acute Coronary Syndrome; Coronary Angiography.

Introduction

Cardiovascular diseases continue to be the leading cause of death in European countries and in the US.[1] In European countries, mortality at 30 days has been reported in 3.4% of the cases of non-ST–elevation myocardial infarction (non-STEMI) and in 6.4% of those with ST-elevation myocardial infarction (STEMI).[2]

The blood supply to the heart is established by three arteries: left anterior descending (LAD), right coronary (RC), and circumflex (CX).[3] Acute myocardial infarction (AMI) usually occurs when the coronary flow suddenly decreases after an obstruction or thrombotic occlusion in a coronary artery previously affected by atherosclerosis.

The diagnosis of AMI is established in the presence of at least two of the following three criteria (with mandatory plasma elevation of myocardial necrosis markers [MNM]): chest pain, electrocardiographic changes (ST segment and T wave), and/or elevation in MNM (creatine kinase [CK], creatine kinase MB [CK-MB], myoglobin, troponin).[4] Acute transmural ischemia caused by occlusion of a coronary artery is usually represented by an elevation in the ST segment.[5]

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The electrocardiogram (ECG) is undoubtedly the most accessible and useful tool for initial assessment, screening, and risk stratification in patients with a suspected acute coronary event, as well as an "indicator" of the therapy to be instituted in these patients.[5,6] The ECG performed in the acute phase is particularly useful in acute coronary syndromes (ACS) with ST-segment elevation, because it contains important information about the involved coronary artery.[7,8]

According to several studies, an accurate ECG analysis can determine if the AMI occurred due to occlusion of the LAD with sensitivity of 90.0% and specificity of 95.0%, and whether it occurred due to an occlusion in the RC or CX with sensitivity of 53.0% and specificity of 98%.[5] Rapid recognition of STEMI by the ECG is crucial for the indication of percutaneous coronary intervention with direct angioplasty. Although the blood flow can be reestablished in more than 85% of the cases, the prognosis depends on the capacity of the existing microcirculation in the affected tissue.[9] Cases of spontaneous reperfusion have been reported; in this situation, the patients have a more favorable outcome and the extent of the myocardial injury is decreased.[10]

This study had the main objective of identifying and correlating the electrocardiographic changes in different leads (especially ST-segment elevation) with the location of the intracoronary thrombus in the artery involved in the coronary event, as well as determining the sensitivity and specificity of the ECG. The secondary objective was to associate the "total" ST elevation (a sum of the ST-segment elevations in the various leads related to the culprit artery) with the elevation of the MNM and the corresponding necrotic area, and with the left ventricular systolic dysfunction observed on echocardiogram.

Methods

Retrospective, observational study conducted with 179 patients admitted for ACS (unstable angina, non-STEMI, and STEMI) and who underwent 12-lead ECG, cardiac catheterization, and transthoracic echocardiography in a single research center between January 2008 and July 2008. The study was approved by the Ethics Committee for Health at *Centro Hospitalar e Universitário de Coimbra* (University and Hospital Center of Coimbra, Portugal). Given the nature of the study, a free and informed consent was not required.

The data were collected from medical charts and transferred to a clinical form for confidential assessment. The data analysis considered the distribution of the 179 patients into three diagnostic groups: unstable angina (ECG without changes or with ST/T changes but without ST elevation or increase in MNM; n = 31), non-STEMI (n = 86), and anterior wall (n = 34) and inferior wall (n = 28) STEMI.

The study excluded patients with complete left bundle-branch block, history of angioplasty with intracoronary stent placement, history of cardiac revascularization surgery, as well as patients transferred from other hospitals without having the first ECG carried out, or with an ECG with bad quality.

ECG analysis

All ECGs performed on admission to the emergency room were analyzed in search of changes in the ST segment, T-wave inversion, and presence or absence of pathological Q waves. The criteria applied were as follows:

Non-STEMI: ST-segment depression ≥ 0.05 mV in two contiguous leads, T-wave inversion ≥ 0.1 mV in two leads, R/S ratio > 1, and pathological Q waves (≥ 25% of the size of the subsequent R wave).[4]

STEMI: ST-segment elevation from the J point onwards ≥ 0.2 mV in the V2 and V3 precordial leads and ≥ 0.1 mV in the remaining leads.[4]

Figure 1 shows the orientation of the resultant vector in subendocardial ischemia in relation to the coronary obstruction and its repercussions on the ST segment.

Electrocardiographic criteria to identify the culprit artery in STEMI

To "identify" the LAD and establish its possible relationship to the location of the intracoronary occlusive thrombus, the following criteria were used: ST-segment elevation > 2 mV in V2, V3, and V4; ST-segment elevation in aVR and ST-segment elevation ≥ 2.5 mm in V1; depression or absence of change in the ST segment in the inferior leads (DII, DIII, and aVF) and in V5; abnormal Q wave (pathological) in aVL; wide Q wave from V4 to V6; ST-segment depression in aVR; complete right bundle-branch block.[3]

Figure 1
Resulting Vectors of subendocardial ischemia vs transmural ischemia and the effect on the ST-T segment.

To "locate" the occluded LAD segment (proximal or distal) by ECG, the following criteria were used: a) proximal occlusion: ST-segment elevation in V2, V3, and V4; ST-segment elevation in aVR, and ST-segment elevation ≥ 2.5 mm in V1; ST-segment depression in the inferior leads (DII, DIII, and aVF) and in V5; abnormal Q wave (pathological) in aVL; b) distal occlusion: ST-segment elevation in V2, V3, and V4; absence of ST-segment depression in the inferior leads; wide Q wave (pathological) from V4 to V6.[3]

In AMI of the inferior-posterior wall, the ST-segment elevation was used as the main criterion in the DII, DIII, and aVF leads (two contiguous leads). However, in this type of infarction, both the RC and the CX can be the culprit arteries.[3] In an attempt to "identify" with the ECG which of these arteries had occlusive thrombus, we used the criteria published by Gorgels et al.[3] To "locate" the RC: ST-segment elevation in DIII > DII; ST-segment depression in aVL > DI; ST segment elevated or isoelectric in V1, or isoelectric segment in V1 and ST depression in V2. To "locate" the CX: ST-segment elevation in DII > DIII; ST segment elevated or isoelectric in DI; ST-segment changes in V5 and V6; ST-segment depression in aVR > aVL.[3] Changes in the ST segment and T wave in the V4R lead were not evaluated in this study since they were not registered in most cases.

Figure 2 shows the orientations of the vectors arising from cases of occlusion of the LAD and their segments (A and B), as well as the orientations of the vectors arising from the injury current when the occlusion occurs in the RC or CX (C).

Coronary angiography and transthoracic echocardiography data were retrieved from patients' charts and databases storing these reports. To classify the left ventricular systolic function (LVSF) findings, we used the terms normal, slightly depressed, moderately depressed, and severely depressed.

Statistical analysis

The results are expressed as mean ± standard deviation (SD) or mean ± standard error of mean (SEM) for quantitative variables. For multiple comparisons, we used analysis of variance (ANOVA) followed by the Newman-Keuls method to correct the significance of the differences. For categorical variables, the chi-square test was used. P values < 0.05 were considered significant.

Results

Non-STEMI predominated (48.0%) in the study population (n = 179), followed by anterior wall AMI (19.0%), unstable angina (17.3%), and inferior wall AMI (16.9%). Table 1 shows the demographic characteristics and cardiovascular risk factors of the patients.

The mean age of the patients was 61.2 ± 11.9 years and did not differ significantly among the four studied groups. However, the mean age of women with anterior wall STEMI was significantly lower than that of women

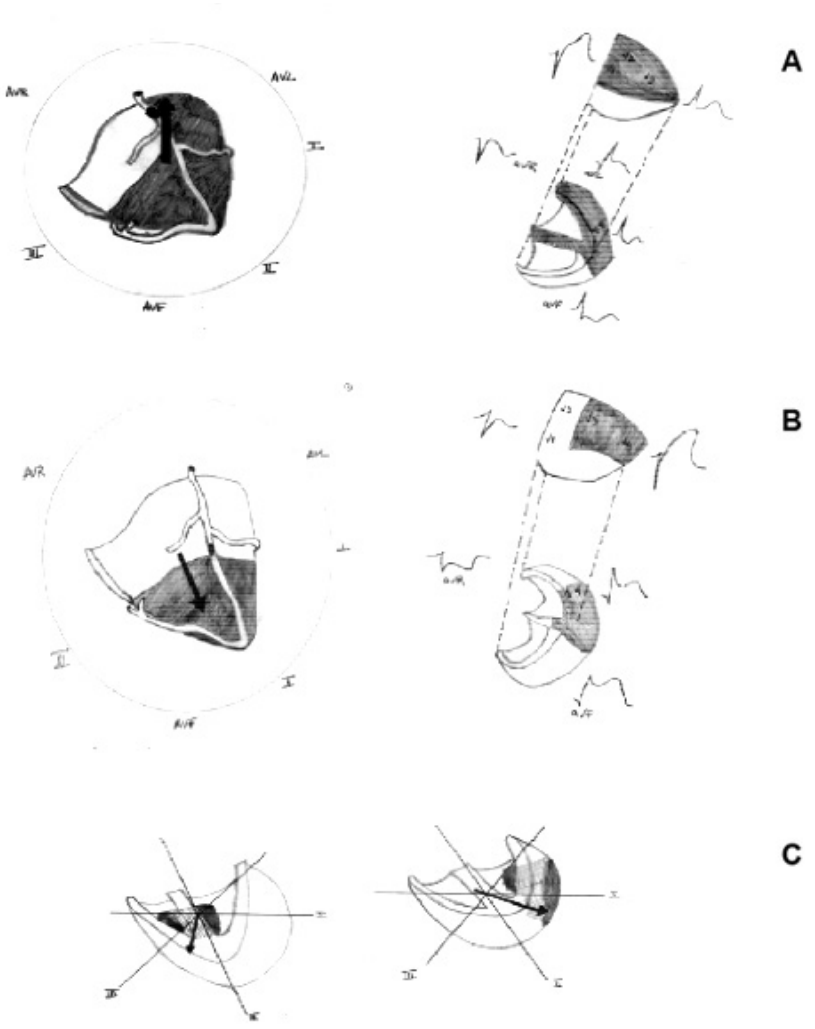

Figure 2
a) Vectors resulting from occlusion of the proximal left anterior descending.
b) Vectors resulting from the occlusion of the distal anterior descending.
c) Vectors resulting from injury current when the CD occlusion or Cx (C).

with unstable angina or non-STEMI ($p < 0.05$). Among men, the age of those with inferior wall STEMI was significantly lower than that of men with non-STEMI ($p < 0.05$). We also observed that the mean age of the men with inferior wall AMI was significantly lower than that of women with unstable angina or non-STEMI ($p < 0.05$).

Regarding cardiovascular risk factors, diabetes mellitus was present in 20.9 to 32.2% of the patients. There was no significant difference among the four groups regarding the proportion of patients with diabetes, hypertension, dyslipidemia, or smoking.

Regarding coronary angiography, 20 patients with non-STEMI had "total" occlusion of one of the main coronary arteries or one of their branches (23.3%). There were no significant differences among the four groups regarding the number of arteries with significant stenosis or occlusion.

Table 1
Demographic data and cardiovascular risk factors in the study population

	Unstable angina (n = 31)	Non-STEMI (n = 86)	STEMI		p
			Anterior (n = 34)	Inferior (n = 28)	
Age (years) (mean ± SD)	64.0 ± 12.5	63.0 ± 11.2	58.2 ± 11.5	56.2 ± 12.0	> 0.05
Male sex	61.6 ± 13.3	62.1 ± 11.4	59.9 ± 11.0	54.5 ± 10.4	< 0.05
Female sex	69.1 ± 9.2	66.0 ± 10.3	51.3 ± 11.6	61.1 ± 15.5	< 0.05
Cardiovascular risk factors n (%)					
Diabetes mellitus	10 (32.2)	18 (20.9)	10 (29.4)	7 (25.0)	ns
Hypertension	19 (61.3)	51 (59.3)	16 (47.1)	9 (23.1)	ns
Smoking	7 (22.6)	30 (34.9)	18 (52.9)	13 (46.4)	ns
Dyslipidemia	19 (61.3)	53 (61.6)	14 (41.2)	10 (35.7)	ns
Number of cardiovascular risk factors n (%)					
1	6 (19.4)	12 (14.0)	9 (26.5)	12 (42.9)	< 0.001
2	11 (35.5)	18 (20.9)	14 (41.2)	7 (25.0)	ns
3	9 (29.0)	13 (15.1)	7 (20.6)	2 (7.1)	ns
4	1 (0.03)	1 (0.03)	0	1 (0.03)	ns
Absence of cardiovascular risk factors	1	1	0	1	ns

SD: standard deviation; AMI: acute myocardial infarction; STEMI: ST-elevation myocardial infarction; non-STEMI: non-ST–elevation myocardial infarction.
(1) ANOVA with Newman-Keuls correction; (2) Chi-square test

Table 2 shows the relationship between obstructive/occlusive coronary disease and ST segment changes. Since occlusion of an epicardial artery or one of its branches was not observed in any of the 31 cases with unstable angina, these patients were not included in the analysis. Of the patients with non-STEMI, 76.6% did not have occlusion of any main coronary artery, and 23.3% had occlusion of one or more arteries or one of their branches. Among patients with STEMI, 70.0% had occlusion of one or more arteries or one of their branches ($p < 0.0001$).

Table 3 shows the association between LVSF and MNM. Rates of slightly or moderately depressed LVSF did not differ significantly in the four groups. However, the rate of normal LVSF was significantly lower in patients with anterior wall STEMI, who also had severe systolic left ventricular dysfunction more frequently.

Figure 3 shows the progression of the plasma concentrations of CK-MB and troponin I during the first 48 hours of hospitalization. In a comparison between Figure 3 and the data in Table 3, we observed that patients with anterior wall AMI had an area under the curve (AUC) *versus* time elapsed between the onset of symptoms and 48 hours (AUC_{0-48}) for MNM significantly higher than patients with other ACS. However, after Newman-Keuls correction, the differences in anterior and inferior AMI were no longer significant; the same was observed when we analyzed the maximal concentration (Cmax) of troponin values.

As shown in Figure 3, there were no significant differences when we compared the sum of the ST-segment elevations from V2 to V4 and the degree of systolic left ventricular dysfunction. A thorough ECG analysis allowed the localization of the intracoronary occlusive thrombus and its relationship with the

Table 2
Occlusive versus non-occlusive coronary disease in the study population

	Non-STEMI (n = 86)	STEMI	
		Anterior (n = 34)	Inferior (n = 28)
Artery not occluded, but with ≥ 70% stenosis (n)			
Total (n)	68	9	9
LAD	12	6	
CX	8		2
RC	6	1	6
CT	1	2	
LAD + CX	6		
CX + RC	4		
LAD + RC	8		
LAD + CX + RC	9		
Absence of disease	12		1
Occlusion of an artery other than the culprit one	6		
With occlusion of the culprit artery on ECG			
Total (n)	18	23	19
LAD	4	21	2
CX	5	1	2
RC	7		15
CT		1	
Two occluded arteries	2		

RC: right coronary; CX: circumflex artery; LAD: left anterior descending artery; CT: left common trunk; ECG: electrocardiogram; AMI: acute myocardial infarction; non-STEMI: non-ST–elevation myocardial infarction; STEMI: ST-elevation myocardial infarction.
Chi-square test: $p < 0.0001$

culprit artery and corresponding involved ventricular wall. Table 4 shows the sensitivity, specificity, and positive (PPV) and negative (NPV) predictive values of the presence in the ECG of ST-segment elevation in the identification of the occlusive intracoronary thrombus.

A comparison between the location estimated by ECG and by catheterization of the occluded LAD segment identified three cases in which the ECG criteria indicated a mixed location (proximal/distal). Therefore, these data were not included in the analyses of sensitivity, specificity, PPV, and NPV of the ECGs with ST-segment elevation in the identification of the occluded LAD segment in anterior wall AMI shown in Table 5.

In patients with anterior wall AMI, 11.7% had additional ST elevation in aVR, 26.5% had ST elevation in V1, 8.8% presented a complete right bundle-branch block, 20.1% had pathological Q waves from V4 to V6, and 5.9% showed ST depression in aVL. Table 5 also shows the data related to the involvement of the RC or CX in inferior wall STEMI. For the purposes of the calculations, obstructions of 90–95% were considered as

indicators of the location of the STEMI. If this fact was excluded and only cases of occlusion were considered, the sensitivity of the ECG to identify the occlusion of the proximal LAD would decrease to 50.0%, specificity would remain at 61.5%, and PPV and NPV would reduce to 61.5% and 50.0%, respectively.

Regarding the culprit artery implicated in inferior wall AMI, the criteria for ECG located the RC as the artery involved in 88.9% of the cases (in 24 out of 28 cases, the dominant artery was the RC); in two cases, it was not possible to locate the involved artery, and in only one case (3.7%) the occluded artery was the CX.

Table 3
Left ventricular systolic function (acute phase) and myocardial necrosis markers

	Unstable angina (n = 25)	Non-STEMI (n = 62)	STEMI		p
			Anterior (n = 24)	Inferior (n = 24)	
Left ventricular systolic function on TTE					
Normal	21 (84.0)	35 (56.5)	2 (8.3)	19 (79.2)	< 0.007
Slightly depressed	2 (8.0)	14 (22.6)	3 (12.5)	4 (16.7)	ns
Moderately depressed	2 (8.0)	7 (11.3)	6 (25.0)	1 (4.2)	ns
Severely depressed		6 (9.7)	13 (54.2)	0 (0.0)	< 0.001
CK-MB		43.6 ± 6.7	217.9 ± 37.0	119.7 ± 23.0	< 0.001
CK-MB AUC		78.05	456.7	251.1	0.06
Troponin I		9.8±1.8	81.2 ± 15.7	57.2 ± 17.5	< 0.001
Troponin I AUC		19.07	168.4	94.64	0.02

Values are expressed as mean ± standard deviation / frequency and percentage
AUC: Area under the curve for plasma concentration versus time (up to 48 hours); SD: standard deviation; AMI: acute myocardial infarction; TTE: transthoracic echocardiography; non-STEMI: non-ST–elevation myocardial infarction; STEMI: ST-elevation myocardial infarction.
(1) Chi-square test; (2) ANOVA followed by Newman-Keuls correction.

Discussion

The main goal of this study was to evaluate the potential of the 12-lead ECG in predicting the culprit artery when performed in the acute phase of an ACS. Another objective, strongly related to the first one, was to determine whether the ST elevation could predict if the artery was obstructed or occluded. These facts have clear clinical relevance since identification of an occlusion requires invasive treatment.[4]

We included all cases of ACS, including those with unstable angina, because the underlying pathophysiological mechanism in all involves, in most cases, rupture of an unstable plaque with resultant obstruction or occlusion of the artery.[11] The possibility of the initial ECG analysis in identifying the occluded arterial segment could have a significant clinical impact, with proper stratification and within the ideal time.[12]

In this population, non-STEMI (48.0%) were the most frequent events. This can be explained by the fact that the studied patients had an advanced mean age (61.2 years), therefore more time to develop a collateral vascular network. This justification is supported by Levin et al.[13] who stated that this collateral vascular network does not develop *de novo*; an anastomotic network would already exist in the normal heart and would not be used until a need for that arose, as in a serious obstruction in one of the main coronary arteries.[14]

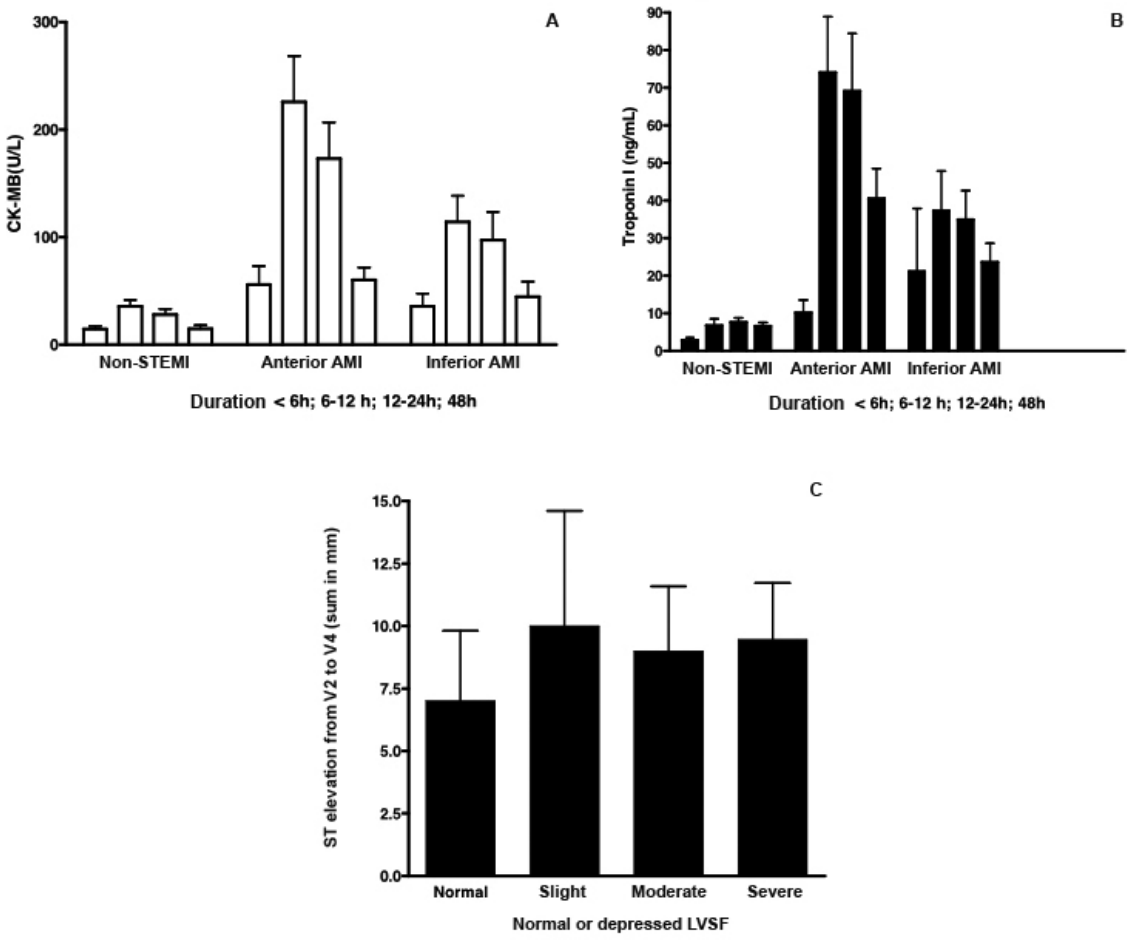

Figure 3
a) Evolution of plasma concentrations of CK-MB.
b) Evolution of plasma concentrations of troponin I.
c) ST-T segment elevation comparisons in patients with AMI of the anterior wall with systolic left ventricular function.

Table 4
Sensitivity, specificity, positive and negative predictive values of non-STEMI and STEMI

	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Non-STEMI + STEMI of the anterior and inferolateral walls	70.0	79.1	70.0	79.1

AMI: acute myocardial infarction; PPV: positive predictive value; NPV: negative predictive value; non-STEMI: non-ST–elevation myocardial infarction; STEMI: ST-elevation myocardial infarction.

Table 5
Sensitivity, specificity, positive and negative predictive values of ST elevation in identifying the occluded segment of the LAD in anterior wall AMI and inferior wall AMI of the RC and CX documented by coronary angiography

	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Occluded LAD segment located by ECG				
Proximal	75.0	61.5	70.6	66.7
Distal	36.4	100.0	100.0	28.0
Occluded artery in inferior AMI (RC or CX) located by ECG				
CD	75.0	33.3	90.0	14.3
CX	ND	ND	ND	ND

LAD: left anterior descending artery; RC: right coronary artery; CX: circumflex artery; AMI: acute myocardial infarction; ND: not determined (n = 1); PPV: positive predictive value; NPV: negative predictive value; ECG: electrocardiogram.

It is also noteworthy that the age of men with inferior wall AMI was significantly lower than that of women with unstable angina or non-STEMI. This difference seen among women can be explained by cardioprotective hormonal factors.[15]

Other relevant data from this study were related to coronary angiography: 20 patients with non-STEMI had "total" occlusion of one of the main coronary arteries or one of their branches (23.3%), and none of them showed ST-segment elevation in the first ECG performed. These results are similar to those published by Wang et al.,[16] which showed that 27.0% of the patients had occlusion of an artery related to the AMI. These patients presented, more frequently, occlusion in one of the arteries that irrigate the inferolateral wall of the left ventricle. Also in this study, 77.8% of the occluded arteries in patients with non-STEMI irrigated the inferolateral wall (RC and/or CX). Therefore, these data should serve as a warning, especially since these patients are regarded as having a similar presentation to STEMI and, thus, benefit from myocardial revascularization procedures.

Ripa et al.[17] have suggested that ST depression in V1–V3 leads can be considered equivalent to ST elevation, thus translating into a CX occlusion. This was observed in only two (2.3%) cases in the present study in which the CX was the occluded artery.

We also observed that the AUC for the plasma concentrations of CK-MB and troponin I *versus* time was higher in patients with anterior wall STEMI than in those with other ACS, which is a result of greater cardiomyocyte loss with a consequent loss of myocardial contractile function. Correlating this fact with echocardiographic data, we found that the AUC of anterior wall AMI was larger in patients with anterior wall AMI with moderately or severely depressed LVSF, although this finding did not show any statistical significance. The mean CK-MB and troponin Cmax values were significantly greater in anterior wall AMI than in those of the inferior wall ($p < 0.01$) or non-STEMI ($p < 0.001$). The non-significant differences found for the troponin Cmax and AUC are probably related to the fact that the plasma concentrations of this MNM were only analyzed within 48 hours, since the correlation with LVSF is stronger for troponin levels assessed within 72 hours.[18]

Although there were no significant differences observed in the present study between the sum of the millimeters of the ST elevations in the anterior wall leads (V2, V3, and V4) and LVSF, Kurisu et al.[19] demonstrated an association between a Σ ST $\geq$ 10 mm and LVSF depression in patients with anterior wall AMI and total LAD occlusion.

According to Blanke et al.,[20] AMI due to occlusion of the LAD can be identified by ECG with a high degree of sensitivity (90.0%) and specificity (95.0%), while identification of the AMI by occlusion of the RC or CX using the ECG shows lower sensitivity and specificity (53.0% and 98.0%, respectively).

The sensitivity and specificity of the ECG to detect the involved coronary artery were 70.0% and 79.1%, respectively. A study published by Menown et al.[21] documented lower sensitivity (56.0%) and greater specificity (94.0%). However, Rude et al.[22] obtained

higher sensitivity (81.0%) and lower specificity (69.0%) rates. These differences can be attributed to the size of the study population and the use of different criteria for interpretation of the ECG. Another factor that could have influenced the ECG was the variation in the evolution time: in this study and in that by Rude et al.,[22] the ECGs were performed in the emergency room, while in the study by Menown et al.,[21] they were performed both in pre-hospital services and in the emergency room. These differences can also be attributed to anatomic variations of the coronary arteries, different dimensions of these arteries, and exact localization of the involved arterial territory, which may have a different electrocardiographic translation even with the same occlusion location, *i.e.*, the ECG patterns may not be the same in similar occlusions. The presence of severe stenosis in a non-culprit coronary artery can also cause remote changes in the ECG, a fact that has not been sufficiently studied yet. Most papers published on the correlation between the ECG pattern and the culprit artery were conducted in patients with a one-vessel disease. In this study, of the 62 patients who had ST elevation, more than 25% had double- or triple-vessel disease.

If cases with critical stenosis in this study were excluded from the analysis and only those of occlusion were considered, the sensitivity of the ECG to identify the occlusion of the proximal LAD would reduce to 50.0%, the specificity would remain at 61.5%, and the PPV and NPV would reduce to 61.5% and 50.0%, respectively. Interestingly, Tierala et al.[24] found different values (sensitivity 95.0%, specificity 97.0%, PPV 96.0%, and NPV 96.0%), probably due to the use of different criteria.

Despite the ECG having shown ST elevation from V2 to V4 in 33% of cases in this study (indicating, with a high probability, occlusion of the LAD that was not later confirmed), performing catheterization at an early stage allowed for suitable reperfusion treatment in those patients. However, this study also showed that six patients (four with non-STEMI and two with inferior wall AMI) had no ST changes from V2 to V4, despite the occluded LAD.

In cases where the ECG showed inferior wall AMI, no impact occurred on the therapeutic strategy. However, in cases of non-STEMI (two cases of occlusion of the first diagonal, one case of occlusion of the middle LAD, and one case of occlusion of the distal LAD), the most appropriate reperfusion therapy was not performed. In these last two cases of non-STEMI involving the LAD, an annulment of the vectors may have occurred if the LAD was too long and extended and surrounded the apex, thereby irrigating the inferoapical and anterosuperior walls. Thus, as shown by Sasaki et al.,[25] no ST elevation occurs in anterosuperior leads (DI and aVL) or inferior leads (DII, DIII, and aVF) since both vectors of the injury current cancel each other out.

With the criteria used for anterior wall AMI,[3] this study found greater sensitivity for the detection of an occlusion in the proximal LAD than in the distal LAD (75.0% and 36.4%, respectively). The specificity, in contrast, was higher for the distal LAD (100.0%) than for the proximal LAD (61.5%). The PPV and the NPV for proximal occlusion of the LAD were 70.6% and 66.7%, respectively. Eskola et al.,[26] in the identification of proximal occlusion of the LAD in the DANAMI-2 trial obtained a sensitivity of 94.0%, specificity of 49.0%, PPV of 85.0%, and NPV of 71.0%.

Of the 32 patients with anterior wall STEMI (V2 to V4), only 11.7% showed aVR elevation (which may explain the lower estimated sensitivity). Zhong-qun et al.[27] and Tong-Wen et al.[28] have shown that ST elevations in aVR and V1 were more prevalent in patients with occlusions proximal to the septal branch (41.7%) than in those with occlusions distal to this branch (4.9%).

It is also important to mention that we considered 90–95% obstructions as indicators of STEMI for the results presented above. In this case, when performing the ECG we considered as though there had been an occlusion of a major artery by vasospasm added to the obstruction by the atheromatous and thrombus plaque and/or before the occurrence of endogenous fibrinolysis. Therefore, when the ECG was performed, there could have actually been a total occlusion, which was relieved after administration of nitrates. Another explanation for the presentation of STEMI in the absence of documented angiographic occlusion is the occurrence of endogenous fibrinolysis.[10] Regarding the culprit artery involved in inferior wall AMI, the electrocardiographic criteria identified the RC as the involved artery in 88.9% of the cases. According to Tierala et al.,[24] many cases of occluded CX are not diagnosed by ECG. In this study, 14.3% of the AMIs of the interior wall showed an involvement of the CX by angiography, and 5.8% (n = 5) of the patients diagnosed with non-STEMI by ECG and MNM had occluded CX as an explanation for the ACS. Of the 28 cases with ST-segment elevation in the inferior wall, the criterion of ST-segment elevation in DIII > DII allowed a correct identification of the RC occlusion by angiography in 71.4% of cases.

The electrocardiographic criteria used in this study allowed us to estimate the sensitivity and specificity of the detection of an RC occlusion at 75.0% and 33.3%, respectively. It was not possible to analyze the V3R and V4R leads in cases of inferior AMI, which could significantly point out to the RC as being the culprit artery.[3,29]

Study limitations

Some limitations can be pointed out: the retrospective and observational nature of the study; the small sample size; the analysis of only the first ECG performed by the patient; the absence of evaluation in the V3R and V4R leads; the lack of knowledge about the time elapsed between the onset of pain and the first ECG; the subjective analysis of the LVSF; and the registration of the MNM only in the first 48 hours.

Conclusion

In this study, when the ECG revealed an ST elevation, an indicator of potential coronary occlusion, the occlusion was confirmed by coronary angiography in 42 cases (70%). On the other hand, the ECG failed to indicate a possible coronary occlusion in 18 cases in which the occlusion was detected during angiography. Thus, the sensitivity and specificity of the ECG to detect an occluded coronary artery were 70% and 79.1%, respectively. With a careful analysis of the ECG, we were able to identify a LAD occlusion correctly in 65.6% of the patients and an RC occlusion in 71.4% of the cases. Despite these several limitations identified, the 12-lead ECG is, and will continue to be, an essential diagnostic method, at least in a first approach to patients with chest pain and/or suspected ACS.

Contribution

Conception and design of the research:Ferreira ARPA, Silva MV. Acquisition of data:Ferreira ARPA. Analysis and interpretation of the data: Ferreira ARPA. Statistical analysis: Ferreira ARPA, Silva MV. Writing of the manuscript:Ferreira ARPA. Critical revision of the manuscript for intellectual content: Ferreira ARPA, Silva MV, Maciel J.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

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