



**ORIGINAL RESEARCH PAPER**

**Cardiology**

**CORRELATION BETWEEN ELECTROCARDIOGRAM (ECG) AND CORONARY FINDING IN PATIENTS WITH ACUTE ST ELEVATION MYOCARDIAL INFARCTION**

**KEY WORDS:** ECG; culprit artery; STEMI

<b>Sanoussi Hamza*</b>	Cardiology Department "B", IBN SINA hospital, Rabat, Morocco. *Corresponding Author
<b>Hasni Mohammed Ali</b>	Cardiology Department B, IBN SINA hospital, Rabat, Morocco.
<b>Kourireche Najla</b>	Cardiology Department "B", IBN SINA hospital, Rabat, Morocco.
<b>Lakhal Zouhair</b>	Cardiology Department, Military hospital, Rabat, Morocco.

**ABSTRACT**

**OBJECTIVES:** The aim of this work is to expose the important role of the ECG in predicting the culprit artery in STEMI.  
**METHODS:** Retrospective study of 100 patients admitted to the military hospital, Rabat, Morocco, with the diagnosis of STEMI from January 2013 to December 2014. We differentiate anterior and inferior MI. In inferior MI patients were enrolled in two groups depending on culprit artery: Right Coronary Artery (RCA group, N:24) and Left Circumflex artery (LCx group, N: 17).  
**RESULTS:** The mean age was 60.3 ± 9.2 years with extremes ranging from 31 years to 83 years, and 91% were male. 41% of patients had at least two cardiovascular risk factors. Smoking was the predominant cardiovascular risk factor and diabetes affected half of patients. Almost 60% of patients were admitted < 6 hours following the onset of pain. The localization of MI was anterior in 55%. LAD was the culprit lesion in 98% of cases. The sum of ST segment elevation was found in leads V1, V2, V3 and V4. The antero-septal localization was related to mid-LAD lesion in 67% of cases, and the extended anterior myocardial infarction was related to proximal LAD lesion in 73% of cases. In inferior MI, ST segment elevation was more marked in lead DIII than DII in 88% of RCA group versus 6% only in LCx group. ST segment depression was more important in lead aVL than DI in 91% of RCA group against 6% only in LCx group. Both criteria were positive in 83% of patients in the RCA group and 0% in the LCx group, while both criteria were negative in 88% of LCx group and 0% in RCA group (p<0.001).  
**CONCLUSIONS:** In anterior MI, LAD is almost always the culprit artery, ECG analysis help to identify the segment involved: proximal, mid or distal. In inferior MI, ECG studies can allow to identify the culprit lesion with much more precision.

**INTRODUCTION**

For a long time, the electrocardiogram was considered an essential element of the initial diagnostic procedure for patients suffering from chest pain. Indeed, it provides more information about the exact location of the lesion, the prediction of the infarct size and the prognosis estimation the increased use of primary percutaneous coronary interventions (PCI) in patients with ST elevation makes this information less useful. However, even with immediate coronary angiography, the identification of the infarct site and the culprit coronary artery remains sometimes difficult. In some patients, more than one lesion can be found, making the determination of the acute lesion difficult. In other cases, complete occlusion of the bifurcation branches of the coronary arteries may go unnoticed.

The purpose of this study is to study the correlation between electrical and angiographic data and to judge the reproducibility of the electrocardiogram in the identification of the culprit lesion.

**MATERIAL AND METHODS**

This is a retrospective study of 100 patients initially admitted to the emergency department of the military hospital, Rabat, Morocco, with the diagnosis of STEMI from January 2013 to December 2014. We included patients with anterior and inferior myocardial infarction (MI). In patients with inferior MI, two groups were enrolled depending on culprit artery two groups depending on culprit artery: Right Coronary Artery (RCA group, N:24) and Left Circumflex artery (LCx group, N: 17).

The enrolled patients met the following criteria:  
 Have chest pain suggesting acute coronary syndrome (ACS) less than 24 hours.

A significant elevation of the ST segment at the J point in two contiguous leads with the cut-points of ≥0.2 mV in leads V2 and V3 ≥0.05 mV in the right and basal leads ≥0.1mV in the other leads, left bundle branch block (LBBB).

Benefit from coronary angiography in the acute phase.

**Data has been collected:**

On the basis of the hospital medical file filled out by the treating physicians.

Based on the register of the catheterization room.

All patients received pre-treatment with aspirin (loading dose: 200 mg) and clopidogrel (loading dose: 300 or 600 mg) All procedures were performed under effective intravenous heparin anticoagulation according to standard regimens.

Reperfusion therapy was indicated in all patients with symptoms of <12 h duration and persistent ST-segment elevation or (presumed) new LBBB. Primary PCI was indicated if there was evidence of ongoing ischaemia, even if symptoms may have started >12 h.

When Primary PCI couldn't be performed in <12 h of symptoms onset, Thrombolysis was indicated. Successful thrombolysis criteria was defined as the presence of at least two of the following criteria at 2 hours after thrombolytic treatment: (1) significant relief of pain (a 5-point reduction on a 1 to 10 subjective scale), (2) > or =50% reduction of sum of ST segment elevation, and (3) abrupt initial increase of creatine kinase levels.

**RESULTS**

**1. Clinical characteristics:**

Clinical characteristics are summarized in (Table 1). The mean age was 60.3 ± 9.2 years with extremes ranging from 31 years to 83 years, and 91% were male. 41% of patients had at least two cardiovascular risk factors. Smoking was the predominant cardiovascular risk factor and diabetes affected half of patients.

The patient delay varied between a minimum of 1H and a maximum of 24H with an average of 5H. Almost 60% of patients were admitted < 6 hours following the onset of pain.

88% presented with typical chest pain. This pain was inaugural in 77% of cases. No patient was in cardiogenic shock at presentation, only one patient presented with a acute heart failure Killip 3

**Table1: Clinical characteristics.**

Clinical Characteristics	Value
Age	60,3+/-9,2
<b>Sexe</b>	
Male	91%
Female	9%

Cardiovascular risk factors	
Smoking	74,4%
Diabetes	46,3%
Hypertension	26,8%
Dyslipidemia	15,9%
Hereditary CAD	1,4%
Patient delay	
≤ 3 H	27%
3 H<H<6 H	32%
6 H≤H< 12 H	23%
≥12 H	18%
Clinical presentation	
Typical chest pain	88%
Acute heart failure	7%
<b>Values are mean SD or n (%).</b>	
<b>CAD: Coronary Artery Disease</b>	

**2 ECG and angiographic findings:**

The ECG and angiographic findings are summarized in (Table 2). 99% of patients presented with ST segment elevation and one patient presented with recent complete LBBB.

MI was anterior in 55% of cases of which almost half was an extensive anterior. 43% of MI were inferior. 10 patients presented with inferior MI extended to the right ventricle (RV). Arrhythmias are mainly represented by atrial fibrillation, ventricular and supraventricular extrasystoles.

All patients underwent coronary angiography in the acute phase of the MI. A monotoncular lesion was found in 47% of cases, involving the left anterior ascending artery (LAD) in 51%.

**Table2: Electrical and angiographic findings**

Characteristics	Value (%)
ECG territory	
Antero-septal	29%
Extensive anterior	24%
Inferior	26%
Infero-basal	15%
Inferior + Extension to RV	10%
Lateral	2%
Coronary artery	
LM	1%
LAD	52%
LCx	15%
RCA	24%
<i>RV : Right Ventricle, LM : Left Main coronary artery, , LAD :Left anterior descending artery, LCx : Left Circumflex, RCA : Right Coronary Artery,</i>	

**3. Revascularization strategy:**

Thrombolysis was performed in 56% of the cases, with a success rate of 75%. Rescue angioplasty concerned only 5 patients. Only 14 patients underwent primary PCI. The rest of the patients received conventional medical treatment.

Almost 75% of the patients underwent angioplasty of the culprit lesion. A drug eluting stent (DES) was implanted in 66% of cases. 11 patients underwent coronary artery bypass grafting (CABG).

**4. Correlation between ECG and coronary angiography:**

**4.1. Anterior MI:**

55% of all MI were anterior. LAD was the culprit lesion in 98% of cases. In two cases, coronary angiography was normal. The sum of ST segment elevation was found in leads V1, V2, V3 and V4. The antero-septal localization was related to mid-LAD lesion in 67% of cases, and the extended anterior myocardial infarction was related to proximal LAD lesion in 73% of cases.

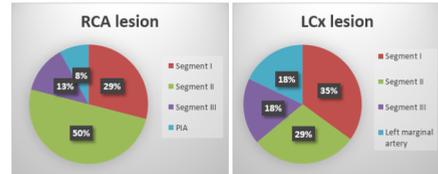
The mirror image in the inferior territory was observed in 30% of cases. Proximal LAD lesion was noted in 67% and 75%, respectively, for the group with anterior MI and inferior mirror image vs without mirror image.

**Table 3: Correlation between ECG and coronary angiography in anterior MI.**

Leads	Nb (%)	Culprit lesion	
		Proximal LAD Nb (%)	Mid-LAD Nb (%)
V1, V2, V3	12 (22%)	4 (33%)	8 (67%)
V1, V2, V3, V4	14 (26%)	8 (57%)	6 (43%)
V1, V2, V3, V4, V5, V6	4 (7%)	2 (50%)	2 (50%)
V1, V2, V3, V4, V5, V6, DI, aVL	22 (41%)	16 (73%)	6 (27%)
V3, V4, V5, V6, DI, aVL	1	0	1

**4.2. Inferior MI:**

43% of all MI in our study were inferior. The RCA was the culprit artery in 56% of cases and the LCX in 40% of cases. Coronary angiography was normal in 2 patients (4%) (Figure 1).



**Figure 1: RCA and LCx lesions in inferior MI**

**Table 4 shows the ECG criteria predicting RCA or LCx lesion in inferior MI**

ECG Criterion	RCA (n=24)	LCx (n=17)	p value
1) ST - ≥1mm DI	12 (50%)	3 (17%)	0,035
2) ST-≥1mm aVL	17 (71%)	3 (17%)	0,001
3) ST- aVL> DI	22 (91%)	1 (6%)	<0,001
4) ST+ DIII> DII	21 (88%)	1 (6%)	<0,001
5) ST-≥1mm V1 and/or V2	11 (46%)	7 (41%)	0,767
6) ST+≥1mm V5 and/or V6	6 (35%)	6 (35%)	0,475
7) ST isoelectric or (+) DI	2 (8%)	11 (64%)	<0,001
8) ST+ ≥1mm V4r	10 (41%)	0	0,002
9) ST+≥1mm V7 V8	5(21%)	6(29%)	0,476
Both Criterion (3) + Criterion (4) positive	20(83%)	0	<0,001
Both Criterion (3) + Criterion (4) negative	0	15(88%)	<0,001

The sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of these different electrocardiographic criteria are presented in Tables 5 and 6.

**Table 5 : Sensitivity, specificity PPV and NPV of ECG criteria when RCA is the culprit lesion in inferior MI.**

ECG criterion	Sensitivity	Specificity	PPV	NPV
ST- DI	50%	83%	80%	53%
ST- aVL	71%	83%	85%	67%
ST- aVL>DI	92%	94%	96%	89%
ST- DIII>DII	88%	94%	96%	84%
ST+ V4r	42%	100%	100%	55%
ST- aVL>DI and ST- DIII>DII	83%	100%	100%	81%

**Table 6 : Sensitivity, specificity PPV and NPV of ECG criteria when LCx is the culprit lesion in inferior MI.**

ECG criterion	Sensitivity	Specificity	PPV	NPV
ST isoelectric or (+) in DI	65%	92%	85%	79%
Criterion A	82%	71%	67%	85%
Criterion B	88%	100%	100%	92%

Criterion A= Absence ST- in aVL lead ; Criterion B= ST- in lead aVL> in lead DI and ST+ in lead DIII> in lead DII : absent.

**DISCUSSION**

There is a correlation between ischemic territories, ECG and occluded artery.

The anterior infarction is invariably related to LAD occlusion. The ST segment vector being directed to the left and lateral, ST segment elevation is expressed in leads V1, V2, V3 and V4 [1,2]. Aldrich et al. reported similar results concerning the maximum ST segment elevation in patients with anterior myocardial infarction due to LAD occlusion. The ranking in descending order showed: V2, V3, V4, V5, aVL, V1, V6. Thus, among 68 patients with anterior myocardial infarction, ST segment elevation was present in lead V2 in 99% of the cases. Maximum elevation was found in leads V2 and V3 [3].

ST segment elevation and depression in the ECG inform about the proximal or distal location of LAD occlusion. In fact, proximal LAD occlusion above the first septal and diagonal branches may involve the basal part of the left ventricle, the anterior and lateral walls and the interventricular septum; The ST segment vector is directed up and to the left so the ST segment elevation concerns leads V1 to V4, DI, aVL and often aVR, in addition ST segment depression in leads DII, DIII, aVF and often V5 [4,5].

Our study reported similar results. Indeed, ST segment elevation in lead aVL was found in 72% of proximal LAD occlusion.

When LAD occlusion is located between the first septal and the first diagonal branch, the basal part of the inter ventricular septum is spared, ST segment vector is directed to aVL, ST segment elevation appears in lead aVL but not in lead V1. In distal LAD occlusion, below both the first septal and first diagonal branches, the basal part of the left ventricle is not involved, and the ST segment vector is oriented further down. Thus, the ST segment is not elevated in leads V1, aVR or aVL and is not depressed in leads II, III, or aVF [35].

Englen et al. reported that ST segment elevation in lead aVR and an ST elevation in V1  $\geq$  2.5 mm, complete RBBB, or a ST segment depression in V5 strongly predicts a proximal LAD occlusion above the first septal. An elevation in leads V4-V6 is associated with a occlusion after the first septal artery [6].

In our study, we reported two cases of anterior MI with RBBB, the two patients had a proximal LAD occlusion above the first septal artery.

In inferior MI, the amplitude of ST segment elevation is in descending order: DIII, aVF, and DII. In the majority (80 to 90%) of cases, ST segment elevation in inferior leads is related to RCA occlusion, but LCx lesion can also produce similar electric modifications [7].

Cooksey et al. found that lead DIII is oriented towards the right inferior segment, while DII is oriented towards the left one, this can explain that lead DIII is more often influenced by RCA lesions and lead DII by LCx lesions [8].

These data were supported by Zimetbaum et al. who have demonstrated that RCA occlusion is responsible of ST segment elevation in lead DIII > DII with a PPV of 97% [9].

Chia et al reported that this criterion has a sensitivity of 97%, a specificity of 90%, a PPV of 97% and a NPV of 90% in RCA occlusion [10].

Birnbaum et al. reported that ST segment depression in the lead aVL was one of the early sign of inferior MI [11].

Hasdai et al found that ST segment depression  $\geq$  1 mm in lead aVL, was suggestive of RCA occlusion with a sensitivity rate of 100%, but a specificity rate of 38%. On the other hand, the absence of this criterion predicts LCx occlusion with a sensitivity rate of 86% and a specificity rate of 100% [12].

In our study, ST segment elevation was more marked in lead DIII

than DII in 88% of the RCA group versus 6% only in LCx group. ST segment depression was more important in lead aVL than DI in 91% of RCA group against 6% only in LCx group. Both criteria were positive in 83% of patients in RCA group and 0% in the LCx group, while both criteria were negative in 88% of the group and 0% in RCA group. This means that when both criteria are positive: LCx is not the culprit artery; when both are negative: RCA is not the culprit artery ( $p < 0.001$ ).

ST segment depression in lead aVL was related to RCA occlusion with a sensitivity rate of 71% and a specificity rate of 83%. After measurement of ST depression DI/aVL ratio the sensitivity and specificity rate were 92% and 94% respectively. The PPV was 96% and the NPV was 89%.

We concluded that ST segment elevation in lead DIII > DII or ST segment depression more marked in aVL than in DI are sensitive and specific markers for the RCA occlusion. The combination of these two criteria increases the sensitivity and specificity at a value that reaches 90% and more. If both criteria are negative, occlusion is related to LCx with a PPV of 100%.

**CONCLUSION**

In anterior MI, LAD is almost the exclusive the culprit artery, ECG analysis help to identify the segment involved: proximal, mid or distal. In inferior MI, ECG studies allow to predict the culprit lesion with much more precision.

**REFERENCES:**

1. Blanke H, Cohen M, Schlueter GU, et al. Electrocardiographic and coronary arteriographic correlations during acute myocardial infarction. *Am J Cardiol* 1984; 54:249-55.
2. Y Birnbaum, B J Drew, The electrocardiogram in ST elevation acute myocardial infarction: correlation with coronary anatomy and prognosis. *Postgrad Med J* 2003; 79:490-504.
3. Aldrich HR, Hindman NB, Hinohara T, et al. Identification of the optimal electrocardiographic leads for detecting acute epicardial injury in acute myocardial infarction. *Am J Cardiol* 1987; 59:20-3.
4. Birnbaum, Y. (1993). Prediction of the level of left descending coronary artery obstruction during anterior wall acute myocardial infarction by the admission electrocardiogram. *Am J Cardiol*, Vol. 72, pp 823-826.
5. Vasudevan K, Manjunath CN, Srinivas KH, Prabhavathi, Davidson D, Kumar S, Yavagal ST, Electrocardiographic localization of the occlusion site in left anterior descending coronary artery in acute anterior myocardial infarction. *Indian Heart J*. 2004 Jul-Aug; 56(4):315-9.
6. Engelen, D.J. (1999). Value of electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute myocardial infarction. *J Am Coll Cardiol*, Vol. 34, pp 389-395, 0735- 1097.
7. Braat SH, Brugada P, den Dulk K, et al. Value of lead V4R for recognition of the infarct coronary artery in acute inferior myocardial infarction. *Am J Cardiol* 1984; 53:1538-41.
8. Cooksey JD, Dunn M, Massie E. Inferoposterior myocardial infarction. *Clinical Vetrocardiography and Electrocardiography*. Chicago: Year Book Medical Publishers, 1977:391-427.
9. Zimetbaum PJ, Krishnan S, Gold A, et al. Usefulness of ST-segment elevation in lead III exceeding that of lead II for identifying the location of the totally occluded coronary artery in inferior wall myocardial infarction. *Am J Cardiol* 1998; 81:918-19.
10. Chia BL, Yip JW, Tan HC, Lim YT. Usefulness of ST elevation III/II ratio and ST deviation in lead I for identifying the culprit artery in inferior wall acute myocardial infarction. *Am J Cardiol* 2000; 86:341-3.
11. Birnbaum Y, Sclarovsky S, Mager A, Strasberg B, Rechavia E. ST segment depression in aVL: a sensitive marker for acute inferior myocardial infarction. *Eur Heart J* 1993; 14:4-7.
12. Hasdai D, Birnbaum Y, Herz I, et al. ST-segment depression in lateral limb leads in inferior wall acute myocardial infarction: implications regarding the culprit artery and the site of obstruction. *Eur Heart J* 1995; 16: 1549-53.