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ORIGINAL ARTICLE

Reciprocal ST segment changes in acute inferior myocardial infarction: Clinical, hemodynamic and angiographic implications

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KEYWORDS

Coronary artery disease;
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Abstract *Objective:* To investigate the clinical significance of reciprocal ST segment depression on the presenting electrocardiogram in patients with acute inferior myocardial infarction.
Design and setting: A prospective, randomized, controlled single center study done in the critical care department, Cairo university Hospital.
Subjects: Forty consecutive patients with acute inferior myocardial infarction were enrolled in this study divided into two groups, 20 patients with reciprocal ST depression (group 1) and 20 patients without such depression (group 2).
Interventions: All patients were investigated with serial ECG, cardiac biomarkers, echocardiography and coronary angiography.
Results: There was no significant difference in the proportion of coronary disease risk factors in patients in group 1, versus those in group 2. Patients in group 1 had significant higher degree ST elevation (in inf. Leads) than patients in group 2, higher levels of peak total CPK and CKMB was also seen. In addition patients in group 1 developed complication more frequently than those in group 2. Although no statistically significant difference between the two groups was seen as regard the ejection fraction sought by echocardiography, it did show a higher incidence of mitral regurge in group 1 [14 (70%)] versus 6 (30%) in group 2 with *P* value of 0.01. In group 1 left anterior descending artery lesions was significantly more frequent than in group 2 with P value < 0.001, also multivessel disease was significantly more frequent in group 1.
Conclusion: The significance of reciprocal ST depression on the electrocardiogram during the course of inferior MI remains uncertain, opinion is divided as to whether it is a benign electrical

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phenomenon or a sign of a greater myocardial necrosis and more frequent left coronary artery disease, from our study we support the latter opinion. This simple ECG finding may be used to differentiate high risk patients for a more aggressive approach.

1. Introduction

Acute inferior MI in its early stages is usually accompanied by ST segment depression in the precordial leads.

Patients with ST elevation in one myocardial zone often have concurrent ST depression in other myocardial zones. Such ST depression may represent pure ''mirror image'' reciprocal changes or may be indicative of acute ischaemia due to coronary artery disease in non-infarct related arteries (''ischaemia at a distance'').[1]

Interest in reciprocal ECG changes in acute inferior MI has yielded many findings, which have led to broadly differing inferences. Some studies have concluded that reciprocal changes represent an electrophysiological phenomenon related with an injury at the infarct site. Others assume that reciprocal changes are associated with more extensive ischemic area or larger infarction.[2,3]

1.1. Inferior wall myocardial infarction

It accounts for 40–50% of all acute myocardial infarction and are generally viewed as having a more favorable prognosis than anterior infarction, however, 50% of patients suffering acute inferior infarction would have complications that will substantially alter that favorable outcome.[4]

In inferior acute myocardial infarction, the leads showing the greatest magnitude of ST elevation are, in descending order: leads III, aVF, and II. The vast majority (80–90%) of patients with ST elevation in these ''inferior'' leads has an occlusion of the right coronary artery; however, an occlusion of the left circumflex artery can produce a similar ECG pattern.[5] In addition to ST elevation in the inferior leads II, III, and aVF, reciprocal ST depression in lead aVL is seen in almost all patients with acute inferior myocardial infarction.[6]

1.2. Diagnosis of inferior infarction extending to contiguous myocardial zones

1.2.1. Right ventricular myocardial zone

When right ventricular infarction occurs, it almost always occurs in the setting of inferior acute myocardial infarction.

Multiple investigators have found that ST elevation in lead V4R is diagnostic of right ventricular infarction with sensitivities and specificities well over 90%.[7]

1.2.2. Lateral apical myocardial zone

In patients with acute inferior myocardial infarction, ST elevation in leads V5 and V6 is thought to indicate extension of the infarct to the lateral aspect of the cardiac apex; however, there is as yet no direct evidence for this.[8]

1.2.3. Posterior myocardial zone

In patients with acute inferior myocardial infarction, ST depression in leads V1–V3 has been shown by numerous investigators to indicate a larger infarction with extension of the injury to the posterolateral and/or the inferoseptal wall.[9]

1.3. What about reciprocal ST segment depression?

Anterior precordial ST-segment depression (APSTD) is common in the setting of inferior myocardial infarction.[10]

The occurrence of precordial (V1–V4) ST segment depression during inferior ST elevation associated with acute inferior myocardial infarction has long been clinically recognized but the underlying mechanism and significance are debated.[11] Such electrocardiographic abnormalities have been attributed to benign reciprocal alteration.[12]

Reciprocal electrocardiographic patterns are recorded from two sets of leads with opposite lead axis orientation. One pattern is considered to directly reflect underlying pathology whereas the other is produced by viewing the myocardial event or lesion from the opposite perspective. Thus myocardial injury on the posterior wall would produce ST segment elevation in lead over the posterior wall and body surface while producing reciprocal ST depression on the anterior cardiac surface. The ST segment elevation may termed primary or intrinsic, reflecting damage to the myocardium directly under the exploring electrode, or termed reciprocal, secondary or extrinsic reflecting events in cardiac regions remote from that electrode. Such reciprocal or secondary effects are a direct biophysical consequence of cardiac electrical activity.[13]

1.4. Mechanism of reciprocal changes during acute inferior myocardial infarction

Mechanisms proposed to explain anterior precordial ST segment depression include simple reciprocal changes,[14] extension of larger IMIs to the posterior or posterolateral wall[15] and concurrent anterior ischemia.[16]

1.5. Sequela of patients with reciprocal ST depression: reciprocal ST segment depression and complications

Study on 108 patients of acute MI by Kumar[17] has shown the incidence of reciprocal ST segment depression in ECG in 58.3% patients with inferior MI. Those showing reciprocal changes had higher (65% vs. 15.5%) incidence of complications such as dysrhythmias, conduction defects, hypotension, left ventricular failure which was more conspicuous in inferior infarction. There was a higher incidence of complications whenever ST segment depression was 2 mm or more and there was steep rise incidence of complications whenever the ST segment depression persisted for 2 days and beyond.[17]

1.6. Reciprocal ST segment depression and left ventricular function

Berland et al.[3] have demonstrated lower ejection fraction in inferior infarction when precordial ECG changes were present.

2. Aim of the work

The aim of our study was to determine the significance of the reciprocal ST segment changes in the early stage of acute inferior wall myocardial infarction and whether it is truly reciprocal or represents ischemia at a distance revealed by echocardiography and coronary angiography.

3. Patients and methods

3.1. Patients

The study was conducted to 40 patients with acute inferior myocardial infarction.

The patients were divided into two groups:

Group 1: consisted of 20 patients with at least 1 mm of ST segment depression in at least one of leads V1 to V4, lead I or aVL.
Group 2: consisted of 20 patients without precordial ST segment depression or a depression < 1 mm volt.

3.2. Methods

All patients were subjected to the followings:

Full history taking with special stress on: age, sex, coronary risk factors (HTN, DM, dyslipidemia, history of IHD or +ve family history of IHD.
Clinical evaluation and follow up of hemodynamics.
Serial ECG.
Serial CPK and CKMB.
Echocardiography.
Coronary angiography.

3.2.1. ECG

A 12 leads resting ECG was recorded on admission as well as V3R and V4R then before and after infusion of streptokinase or before and after primary PCI then after 2 h and 12 h during the first day then once daily during the hospital stay.

All ECGs were examined for.

Inferior myocardial infarction is diagnosed on detection of ST segment elevation ($\geqslant$0.1 mv above the TP segment measured 80 ms after the *J*-point) with new Q wave in (leads II, III and AVF)[18]
Any associated ST segment depression: site and degree of depression.
Right ventricular infarction is diagnosed by ST segment elevation in right precordial leads (V3R, V4R).[7]
Any evidence new conduction abnormalities, e.g., occurrence of heart block, bundle branch block, trifasicular block or dysrhythmias.

3.2.2. Biological Follow-up

CK enzyme ± MB fraction were measured on admission then after 12 h then every 12 h for the first 48 h, then once a day, diagnosis is made on finding at least twice the normal elevation in serum CPK and MB fraction.[18]

3.2.3. Echocardiography

Ejection fraction (%).
Diastolic dysfunction.
Regional wall motion abnormalities.
Mitral regurge.

3.2.4. Coronary angiography

Number of the lesions.
Site of the lesions.
Degree of the lesions (%).
Intervention done.

3.3. Treatment

All patients, on admission, received thrombolytic therapy or primary PCI. All patients received routine coronary care therapy such as oxygen, aspirin, analgesia and sedation. Other drugs such as nitrates, beta blocker, and calcium antagonist were added whenever needed. Complications were treated as required.

3.4. Exclusion criteria

Patients with history of ECG evidence of previous myocardial infarction.
Patients whose ECG show ventricular paced beats.

4. Results

4.1. Clinical results

It showed that there is no significant statistical difference in both studied groups according to the major risk factors including (age, sex, DM, HTN, smoking, dyslipidemia, history of IHD or +ve family history of IHD).

4.1.1. ECG

Degree of ST elevation in II, III and aVF on admission.

Table 1 shows the distribution of both studied groups according to the degree of ST segment elevation in II, III and aVF on admission. It shows a significant statistical difference in both studied groups with *P* value < 0.001.

Table 1 Degree of ST elevation in II, III and AVF in mv.

	Group 1 ($N = 20$)	Group 2 ($N = 20$)	P-value
ST elevation (mean + SD)	0.72 + 0.25	0.41 + 0.14	< 0.001
Range	0.3–1.4	0.25–0.85	

Table 2 ST elevation in right chest leads on admission.

	Group 1 ($N = 20$)	Group 2 ($N = 20$)	P-value
ST elevation in Rt chest leads in mV (mean + SD)	9 (45%)	1 (5%)	0.003

Table 3 Correlation between the degree of ST elevation and depression on admission.

	ST elevation (mv)	ST depression (mv)	P-value
Group 1	0.68 ± 0.33	0.72 ± 0.25	0.009
r value	0.56		

Table 2 shows the distribution of both studied groups according to the degree of ST segment elevation in right chest leads on admission. It shows a statistically significant difference in both studied groups with P-value of 0.003.

4.1.2. Correlation between the degree of ST elevation and depression on admission

Table 3 shows that there is a significant correlation between the degree of ST segment depression and the degree of ST segment elevation on admission ($r = 0.565$, P-value 0.009) in patients with reciprocal ST depression (Fig. 1).

4.2. Biological results

Table 4 shows the distribution of both studied groups according to the total CPK and CKMB. It shows that the serum peak total CPK was significantly higher in group 1 (2755.45 ± 1015.73) than in group 2 (2108.35 ± 1015.19) with P-value of 0.037, also It shows that the serum peak CKMB was significantly higher in group 1 (343.60 ± 124.12) than in group 2 (257.25 ± 113.46) with P-value of 0.05 (Fig. 2).

4.3. Complications

Nineteen patients (95%) in group 1 developed complications during their hospital course including (arrhythmias, cardiogenic shock, mitral regurge, RV infarction, LV dysfunction and heart block) versus 10 patients (50%) in group 2 who developed such complications showing significant statistical difference in both groups with P-value < 0.01.

There is no significant statistical difference between both studied groups according to arrhythmias, cardiogenic shock, LV dysfunction or heart block.

In contrast a significant statistical difference was seen between the two studied groups according to the development of MR or RV infarction during the acute phase with P-value of 0.011 and 0.013, respectively.

4.4. Echocardiography

Table 5 shows the distribution of both studied groups according to ejection fraction. It shows no significant statistical difference in both groups with P-value of 0.257.

Table 6 shows the distribution of both studied groups according to echo-findings including (Diastolic Dysfunction, RWMAs, Mitral regurge). It shows no significant statistical difference in both groups according to diastolic dysfunction and RWMAs but shows significant statistical difference in both groups according to Mitral regurge as 14 patients (70%) in group 1 developed Mitral regurge with only six patients (30%) in group 2 developed Mitral regurge with P-value of 0.011.

4.5. Coronary angiography

After performing coronary angiography for all patients in the study the following statistical results were found:

1. No significant statistical difference in both studied groups as regard of presence of normal coronaries with P value: 1.000.
2. No significant statistical difference in both studied group as regard the presence of coronary ectasia with P value: 1.000.
3. As regard to the presence of right coronary lesion no significant statistical difference was found between the two studied groups (P value: 1.000).
4. Also, no significant statistical difference (SSD) was found as regard to the presence of left circumflex coronary artery lesion between the two studied groups. P value: 0.204.
5. A significant statistical difference with a P value of 0.001 was found between the two studied groups as regard the presence of LAD lesion as 14 patients (70%) in group 1 had a LAD lesion while four patients (20%) in group 2 had a LAD lesion.
6. Also there was a significant statistical difference between both studied groups as regard the degree of LAD lesion with P value < 0.001. As 10 patients out of twenty in group 1 had significant LAD disease (Stenosis > 50%), nine patients of those had total LAD occlusion and only one with a lesion of 50–70%. Four other patients in this group 1 had insignificant LAD lesion less than 50% stenosis.

In contrast in group 2 there was only four patients out of twenty showing LAD lesion, two of those patients had significant LAD lesion showing stenosis of more than 50%, and two more patients with a less than 50% stenosis.

4.6. Correlation between the degree of ST segment depression on admission and the degree of LAD lesion

Table 7 shows that there is no correlation between the degree of ST segment depression on admission and the degree of LAD lesion ($r = 0.069$, P-value 0.772) in patients with reciprocal ST depression (Fig. 3).

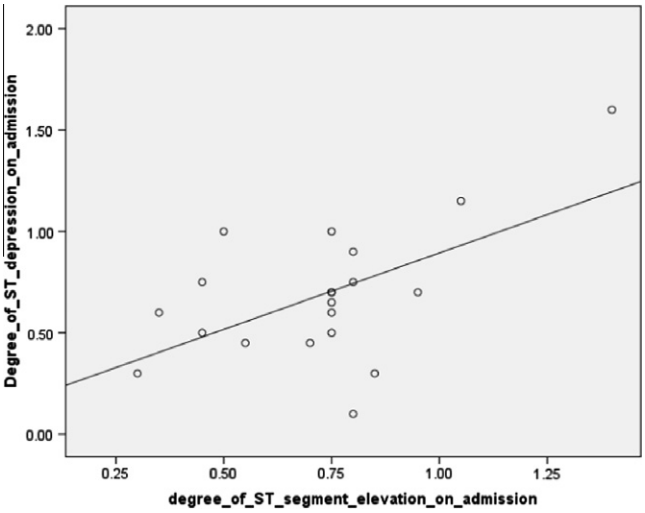

Figure 1 Correlation between the degree of ST elevation and depression in group 1 on admission.

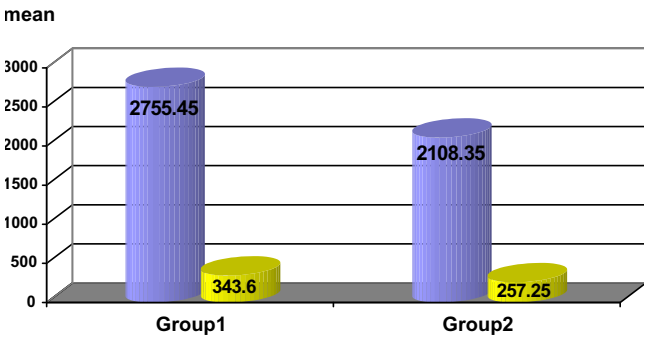

Figure 2 Total CPK and CKMB.

5. Discussion

This study was conducted upon 40 patients with acute inferior myocardial infarction aiming to determine if the reciprocal ST segment changes usually seen in this settings are truly reciprocal or represents ischemia at a distance, patients were divided into two groups, group 1 consisted of 20 patient with at least 1 mm ST segment depression in at least one of the lead V1 to V4, lead I or aVL and group 2 consisted of 20 patients without precordial ST segment depression.

In the current study there was no significant statistical difference in both studied groups according to the major risk factors including (age, sex, DM, HTN, smoking, dyslipidemia, history of IHD or +ve family history of IHD). These results were consistent with the results of Sükrü et al.[19] whom reported that in the comparison of patients with and those without reciprocal changes, there were no significant differences in age, gender, and risk factors for atherosclerosis such as hypertension, smoking, and diabetes mellitus.

Also, the study by Zoghi et al.[20] showed no significant differences between patients with reciprocal changes and patients without as regards age, sex, HTN, DM, dyslipidemia or smoking.

Lembo et al.[21] found that patients with reciprocal ST depression were older than patients without reciprocal ST depression (67 ± 9 vs. 59 ± 8; *P*-value < 0.01).

Results of the current study showed that serum peak total CPK was significantly higher in group 1 (2755.45 ± 1015.73) than in group 2 2108.35 ± 1015.19) with *P*-value of 0.037. Also serum peak CKMB was significantly higher in group 1 (343.60 ± 124.12) than in group 2 (257.25 ± 113.46) with *P*-value of 0.05.

Patients with inferior myocardial infarction who had ST segment depression in their precordial leads during the acute phase sustained more extensive myocardial necrosis than patients without reciprocal change, as shown by serum total CK and CKMB levels. These results concur with numerous previous studies[22] using the CK levels or CKMB, as indicators of the necrotic myocardial mass.

Gibelin et al.[23] demonstrated that the serum peak total CK was significantly higher (1835 ± 940) in patients with reciprocal ST depression than in patients without (875 ± 305, *P*-value < 0.01). The same differences were found for peak CKMB (269 ± 102) in patients with reciprocal ST depression than in patients without (95 ± 35, *P*-value < 0.01).

In contrast Sükrü et al.[19] showed no significant statistical difference (*P* = 0.08). As mean peak serum creatine kinase level was 2605 ± 995 IU/L in patients with reciprocal ST depression and 2194 ± 1128 IU/L in those without reciprocal ST depression.

In the current study the degree of ST segment elevation in the inferior leads on admission was significantly higher in group 1 (0.72 ± 0.25) than in group 2 (0.41 ± 0.14) with *P*-value < 0.001. These results were consistent with the results of Wasserman et al.[14] who reported that the prevalence of ST segment depression increase with increase inferior ST segment elevation.

By comparing the degree of ST segment depression on admission and the degree of LAD lesion in patients with reciprocal ST depression we found no correlation between the degree of ST segment depression on admission and the degree of LAD lesion (*r* = 0.069, *P*-value 0.772). In contrast by comparing the degree of ST segment elevation and the degree of ST segment depression on admission in group 1 there was a significant correlation between the two variants (*r* = 0.565, *P*-value 0.009). Lew et al.[24] revealed a close correlation between pre-

Table 4 Total CPK and CKMB.

	Group 1		Group 2		*P*-value
	Range	Mean + SD	Range	Mean + SD	
CPK	1190–4820	2755.45 + 1015.73	890–4770	2108.35 + 1015.19	0.04
CKMB	190–655	343.60 + 124.12	110–480	257.25 + 113.46	0.05

CPK, creatine phosphokinase; S, significant; CKMB, creatine kinase MB fraction.

Table 5 Ejection fraction.

	Group 1 ($N = 20$)	Group 2 ($N = 20$)	*P*-value
Std. deviation (mean + SD)	59.10 + 7.24	61.8 + 6.89	0.3
Range	42–74	45–68	

Table 6 Other Echo-Findings.

	Group 1	Group 2	*P*-value
Diastolic Dysfunction	16 (80%)	16 (80%)	1.00 NS
RWMAs	18 (90%)	14 (70%)	0.235 NS
Mitral regurge	14 (70%)	6 (30%)	0.011 S

RWMAs, regional wall motion abnormalities.

Table 7 Correlation between LAD lesion and ST depression in group 1.

	ST depression (mv)	LAD lesion	*P*-value
Group 1	51.5 ± 40.97	0.69 ± 0.34	0.77
r value	0.069		

cordial ST segment depression and inferior ST segment elevation ($r = 0.89$).

This current study showed that 19 patients (95%) in group 1 developed complications during their hospital course including (arrhythmias, cardiogenic shock, mitral regurge, RV infarction, LV dysfunction and heart block) versus 10 patients (50%) in group 2 who developed such complications showing significant statistical difference in both groups with *P*-value < 0.01.

Salcedo et al.[16] in their study of 45 patients with acute inferior wall infarction reported that 12 of the 13 patients who developed complications during their hospital stay showed precordial ST segment depression (3 patients died, 5 had ventricular fibrillation, and 4 had left ventricular failure).

In the, current study Ejection fraction was lower in group 1 (59.10 ± 7.24) than in group 2 (61.8 ± 6.89) but with no significant statistical difference in both studied groups1 *P*-value 0.257.

Sükrü et al.[19] demonstrated no significant difference between patients with reciprocal ST depression and those without with regard to the echocardiographic parameters. As regards to ejection fraction (%) there was no significant statistical difference between patients with and patients without reciprocal ST depression.

Zoghi et al.[20] found a significant difference between patients with reciprocal ST depression (49% ± 19%) and patients without (52% ± 15%) as regards EF (%) with *P*-value < 0.001.

Gibelin et al.[23] demonstrated that left coronary artery disease was significantly more frequent (84%) in patients with reciprocal changes as regards the left anterior descending artery or left circumflex artery. Also multivessel disease was more frequent (71%) in patients with reciprocal changes.

In contrast Peterson et al.[15] found that the frequency of left anterior descending coronary artery disease did not vary by the presence or distribution of precordial ST segment depression, nor did the frequency of multivessel coronary artery disease.

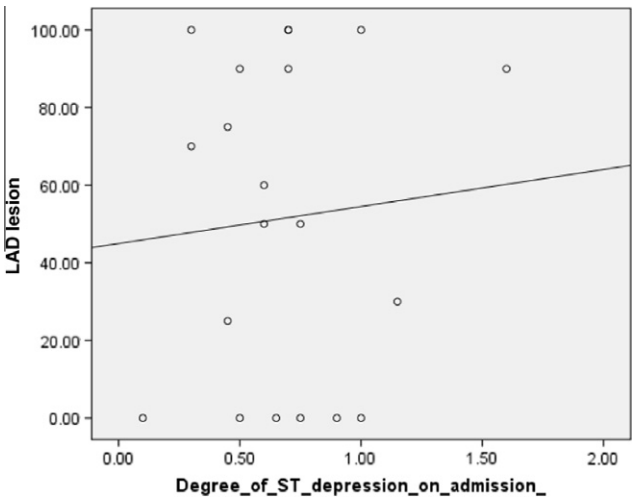

Figure 3 Correlation between LAD lesion and ST depression in group 1.

In our study, LAD lesion was significantly more frequent in group 1. Fourteen patients (70%) in group 1 had LAD lesion with only four patients (20%) in group 2 had LAD lesion showing significant statistical difference in both studied groups with *P*-value < 0.001.

Also, there was a significant statistical difference between both studied groups as regard the degree of LAD lesion with *P* value < 0.001. As 10 patients out of twenty in group 1 had significant LAD disease (stenosis > 50%), nine patients of those had total LAD occlusion and only one with a lesion of 50–70%. Four other patients in this group 1 had insignificant LAD lesion of less than 50%.

In contrast in group 2 there was only four patients out of twenty showing LAD lesion, two of those patients had significant LAD lesion showing stenosis of more than 50% and two more patients with a less than 50% stenosis.

Also multivessel disease was significantly more frequent in group 1. Ten patients (50%) in group 1 had three -vessel disease (LAD, LCX and RCA) with only one patient (5%) in group 2 had three- vessel disease showing significant statistical difference in both studied groups with *P*-value 0.001.

Zoghi et al.[20] concluded that the presence of precordial ST segment depression during acute inferior myocardial infarction may not always be only an electrical phenomenon. The presence of anterior precordial ST segment depression during an acute inferior myocardial infarction correlates with presence of multivessel CAD. In these patients, the presence of an anterior precordial ST segment depression of greater magnitude than ST segment elevation in the inferior leads increase the likelihood of multivessel disease.

6. Conclusion

The significance of reciprocal ST depression on the electrocardiogram (ECG) during the course of inferior myocardial infarction (MI) remains uncertain, opinion is divided as to whether it is a benign electrical phenomenon or a sign of a greater myocardial necrosis and more frequent left coronary artery disease, from our study we support the latter opinion.

Recommendation

So, we recommend that this simple ECG finding is used to differentiate high risk patients for a more aggressive approach, fortunately in the early hours of infarction in those high risk patients.

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