



CLINICAL HEMODYNAMIC STUDY OF RECIPROCAL ST SEGMENT CHANGES IN ACUTE INFERIOR MYOCARDIAL INFARCTION

Medicine

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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 medicine department of JLNMC, Bhagalpur, Bihar.

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 regurg in group 1 [14 (70%)] versus 6 (30%) in group 2 with P value of 0.01. In group 1 left ante-rior 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 phenomenon or a sign of a greater myocardial necrosis and more frequent left coronary artery dis-ease, from our study we support the latter opinion. This simple ECG finding may be used to differ-entiate high risk patients for a more aggressive approach.

KEYWORDS

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" reci-procal changes or may be indicative of acute ischaemia due to coronary artery disease in non-infarct related arteries ("ischaemia at a distance").

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.

AIM OF THE STUDY

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.

MATERIAL AND METHODS

Material

The study was conducted to 40 patients with acute inferior myocardial infarction at Medicine Department of Jawaharlal Nehru Medical College and Hospital, Bhagalpur, Bihar from March 2020 to February 2021.

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.

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.

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 (P0.1 mv above the TP segment measured 80 ms after the J-point) with new Q wave in (leads II, III and AVF) 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).

Any evidence new conduction abnormalities, e.g., occurrence of heart block, bundle branch block, trifascicular block or dysrhythmias.

Biological Follow-up CK enzyme $\pm$ 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

Echocardiography

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

Coronary angiography

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

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.

Exclusion Criteria

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

RESULTS**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).

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 Right Chest	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.

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.

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.

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.

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.

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 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.
7. 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.

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.

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 (n=20)	Group 2 (n=20)	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		

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 Su "kru" et al. 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. showed no significant differences between patients with reciprocal changes and patients without as regards age, sex, HTN, DM, dyslipidemia or smoking.

Lembo et al. 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 studies²² using the CK levels or CKMB, as indicators of the necrotic myocardial mass.

Gibelin et al. 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 Su "kru" et al. 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. 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-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. 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 groups P-value 0.257.

Su "kru" et al. demonstrated no significant difference between patients with reciprocal ST depression and those with-out with regard to the echocardiographic parameters. As regards to ejection fraction (%) there was no significant statistical difference between patients with and patients with-out reciprocal ST depression.

Zoghi et al. found a significant difference between patients with reciprocal ST depression ($49\% \pm 19\%$) and patients without ($52\% \pm 15\%$) as regards EF (%) with P-value < 0.001.

Gibelin et al. 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. 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.

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 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. 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.

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.

REFERENCES

1. Assali AR, Herz I, Vaturi M, et al. Electrocardiographic criteria for predicting the culprit artery in inferior wall acute myocardial infarction. *Am J Cardiol* 1999;84:87–9.
2. Berger PB, Ryan TJ. Inferior myocardial infarction: high-risk subgroups. *Circulation* 1990;81:401–11.
3. Berland J, Cribier A, Bahar P, Letac B. Anterior ST segment depression in inferior myocardial infarction: Correlation with results of intracoronary thrombolysis. *Am Heart J* 1986;111:481–8.
4. Bimbaum Y, Drew BJ. Anatomy and prognosis myocardial infarction: correlation with coronary the electrocardiogram in ST elevation acute Postgrad. *Med J* 2003;79:490–504.
5. Bimbaum Y, Sclarovsky S, Mager A, et al. ST segment depression in aVL: a sensitive marker for acute inferior myocardial infarction. *Eur Heart J* 1993;14:4–7.
6. 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.
7. Elik SC, Yilmaz R, Baykan M, Orem C, Erdo T C. Are reciprocal changes a consequence of "ischemia at a distance" or merely a benign electrical phenomenon? A pulsed-wave tissue Doppler echocardiographic study. *ANE* 2003;8(4):302–7.
8. Gelman JS, Saltups A. Precordial ST segment depression in patients with inferior myocardial infarction: clinical implications. *Br Heart J* 1982;48:560–5.
9. Gibelin P, Gilles B, Baudouy M, Guarino L, Morand. Reciprocal ST segment changes in acute inferior myocardial infarction: clinical, hemodynamic and angiographic implications. *Eur Heart J* 1986;7:133–9.
10. Gibson MS, Michael C, Angeja BG, Murphy SA, Marble SJ, Barron HV, et al. Cannon MD for the TIMI Study Group: Precordial ST-Segment Depression in Inferior Myocardial Infarction is Associated with Slow Flow in the Non-Culprit Left Anterior Descending Artery. *J Thromb Thrombolysis* 2002;13(1):9–12.
11. Hasdai D, Sclarovsky S, Solodky A, et al. Prognostic significance of maximal precordial ST-segment depression in right (V1 to V3) versus left (V4 to V6) leads in patients with inferior wall acute myocardial infarction. *Am J Cardiol* 1994;74:1081–4.
12. Hlatky MA, Califf RM, Lee KL, Pryor DB, Wagner GS, Rosati RA. Prognostic significance of precordial ST segment depression during inferior acute myocardial infarction. *Am J Cardiol* 1985;55:325–9.
13. Kumar H. Prognostic significance of reciprocal changes in acute myocardial infarction. *J Assoc Physicians India* Sept 1992;40(9):630–1.
14. Lembo NJ, Starling MR, Dell' Italia LJ, et al. Clinical and prognostic importance of persistent precordial (V1–4) electrocardiographic ST segment depression in patients with inferior transmural myocardial infarction. *Circulation* 1986;74:56–63.
15. Lev AS, Maddah J, Shah PK, et al. Factors that determine the direction and magnitude of precordial ST segment deviations during inferior wall myocardial infarction. *Am J Cardiol* 1985;55:883–8.
16. Marriott JHL. Myocardial infarction. In: Marriott JHL, editor. *Practical electrocardiography*. Baltimore: Williams & Wilkins; 1972. p. 226–53.
17. Mirvis DM. Physiologic bases for anterior ST segment depression in patients with acute inferior wall myocardial infarction. *Am Heart J* 1988;116:1308–22.
18. Nagahama Y, Sugiura T, Takehana R, Tarumi N. PQ segment depression in acute Q wave inferior wall myocardial infarction. *Circulation* 1995;91:641–4.
19. Peterson ED, Hathaway WR, Zabel KM, et al. Prognostic significance of precordial ST segment depression during inferior myocardial infarction in the thrombolytic era: results in 16,521 patients. *Am Coll Cardiol* 1996;28:305–12.
20. Salcedo JR, Baird MG, Chambers RJ, Beanlands DS. Significance of reciprocal ST segment depression in anterior precordial leads in acute inferior myocardial infarction: concomitant left anterior descending coronary artery disease? *Am J Cardiol* 1981;48:1003–8.
21. Wasserman AG, Ross AM, Bogaty D, Richardson DW, Hutch-inson RG, Rios JC. Anterior ST segment depression during acute inferior myocardial infarction: evidence for the reciprocal change theory. *Am Heart J* 1983;106:516–20.
22. Wong CK, Freedman SB, Bautovich G, et al. Mechanism and significance of precordial ST-segment depression during inferior wall acute myocardial infarction associated with severe narrowing of the dominant right coronary artery. *Am J Cardiol* 1993;71:1025–30.
23. Zehender M, Kasper W, Kauder E, et al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. *N Engl J Med* 1993;328:981–8.
24. Zoghi M, Gu'gu'n C, Yavuzgi O, Tu'rko Iu I, Ku'ltu'say H, Akilli A, et al. The angiographic correlation between ST segment depression in non infarcted leads and the extent of coronary artery disease in patients with acute inferior myocardial infarction: A clue for multivessel disease. *Can J Cardiol* 2003;19:67–71.