# CORRELATION BETWEEN PRECORDIAL ST DEPRESSION AND LEFT ANTERIOR DESCENDING ARTERY DISEASE IN PATIENTS WITH ACUTE INFERIOR MYOCARDIAL INFARCTION AFTER THROMBOLYTIC THERAPY

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*Abstract - This study was conducted to determine whether precordial ST segment depression in admission electrocardiogram (ECG) in patients with acute phase inferior myocardial infarction (MI) eligible for thrombolytic therapy and subsequent angiographic evaluation, influences the incidence of left anterior descending artery (LAD) disease. 31 consecutively admitted patients were entered in our study. 20 patients had precordial ST segment depression (group A) and 11 patients were without ST segment depression (group B). After adjusting for baseline variables there was no significant difference in incidence of LAD and circumflex and 3 vessel disease between the two groups. Ejection fraction and wall motion abnormality were also identical. Group A had higher complication rate ($P < 0.05$) than group B. Right ventricular (RV), lateral and posterior MI that influence the degree of ST segment depression were meaningfully different between these groups. RV infarction was observed 63% in group B and 35% in group A ($P < 0.05$). Sum of lateral and posterior MI were 38% in group A and 9% in group B ($P < 0.05$).*

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*Key Words: Precordial leads, inferior MI, ST depression, LAD disease.*

## INTRODUCTION

Confusion abounds regarding the significance of precordial electrocardiographic ST segment depression in the setting of acute and evolving inferior myocardial infarction. The subject has been of intrest to clinicians. As an isolated finding in the context of elevated cardiac enzyme this pattern is regarded as pathogonomonic of anterior non Q wave myocardial infarction (1,2). If, on the other hand, anterior ST segment depression is associated with acute inferior infarction, significance of this precordial ST segment depression is much less certain. In some investigations this finding has been attributed to concomitant anterior myocardial ischemia and presence of left anterior descending coronary artery disease. Other studies have suggested that this effect is related to posterolateral, inferoposterior, inferoseptal acute myocardial infarction, myocardial ischemia, or benign reciprocal changes (3,4,5). These contradictory conclusions may be partially attributable to the differences between multiple methods of noninvasive and invasive assessement, various patient population, and type of evaluation. This report details angiographically proven findings in our study groups at the thrombolytic era.

The main purpose of the present study was to investigate the relation of precordial ST depression to LAD lesions in acute inferior MI. In addition we analyzed a few other clinical and angiographic variables.

## MATERIALS AND METHODS

Patient population: From 196 consecutively admitted patients in our coronary care unit with presentation of prolonged chest pain lasting 30 minutes, unrelieved by sublingual nitroglycerine and associated with typical electrocardiographic changes of acute inferior myocardial infarction and elevation of serum creatin kinase (CK), 31 patients (28 men and 3 women) who had indication for thrombolytic therapy and could undergo coronary angiography during a 4 weeks interval, were entered in our study. These patients divided in two groups in term of presence ST depression in their precardial leads (group A), and absence of these changes (group B). Patients with evidence of previous infarction, history of coronary bypass surgery, other kind of major cardiac disease, abnormal QRS complex, bundle branch blocks or

hypertrophied ventricles were excluded. All patients had received their routine thrombolytic, antiplatelet and anti-ischemic therapy. Electrocardiography: 12 lead ECG using a standard paper speed of 25mm/s was taken on admission and before thrombolytic therapy. ST segment was considered that part of the electrocardiogram from the J point (end of QRS complex) to the onset of T wave. ST segment was recognized depressed if there was > 1mm ST depression in at least two of contiguous leads.

Inferior myocardiol infaction: Recognized as ST elevation> 1mm in at least two of the three inferior leads (II - III - aVF).

Right ventricular infarction: Defined as ST elevation ≥ 1mm in at least 2 right precordial leads.

Posterior myocardiol infarction: Determined by R>S in lead $V_1$ and $V_2$ in association with other electrocardiographic and clinical findings of myocardial infarction.

Lateral MI: recognized as ST segment elevation > 1mm in aVL. $L_1$-$V_5$-$V_6$ leads.

Complications : Defined as pulmonary edema, ventricular fibrilation, sustained ventricular tachycardia, atrioventricular blocks that needed temporary pacemaker, postmyocardial infarction angina, extension of myocardial infarction, pericarditis and supraventricular arrhythmias that needed therapy. All these complications were defined and diagnosed on the basis of conventional presentation.

Coronary Angiography: Selective coronary angiography with multiple views and left ventricular angiography were carried out in study patients and were reviewed by at least two observer blinded to the results of clinical and electrocardiographic findings. The number and severity of coronary stenosis, ejection fraction and any abnormal region of wall motion were determined by visual inspection.

Significant coronary artery stenosis was defined as >70% in diameter. Stenosis and the numbers of ventricular segments with abnormal motion were estimated using a scoring system described by Rogers et al. (6,7)

Statistical methods: Data are expressed as mean ±standard deviation. The t-test was used to determine differences between continous variables. Chi squre test was used to assess differences in the categorial variables.

# RESULTS

The patients were divided into two groups. Group A with ST depression in precordial leads included 20 patients (19 male + 1 female), and group B without ST segment depression included 11 patients (9 male + 2 female). Therfore 95% of group A and 81% ofgroup B were men and the differences were not significant (P=NS).

Mean age for group A was 54 ± 6.4 and for group B 53.3 ± 9 years (P =0.4)

The mean time interval from the begining of chest pain to hospital admission was 216 ± 112 minutes and 210 ± 120 minutes respectively for group A and B (P=0.2).

**Table 1.** Frequency of complications in study subjecs

| Event | Group A | Group B | Pvalue |
|---|---|---|---|
| VT or VF | 2 | 0 | NS |
| AV Block | 0 | 1 | NS |
| SVT | 4 | 0 | NS |
| Post MI angina | 1 | 0 | NS |
| Extension of MI | 2 | 0 | NS |
| Total | 9 | 1 | < 0.05 |

VT: Ventricular tachycaradia
VF: Ventricular fibrillation
AV: Atrio Ventricular
SVT: Supraventricular tachycardia
MI: Myocardial infarction

The mean time interval from admission to angiography was 8.2 ± 4 day for group A and 11.9 ± 7.6 for group B (P = 0.1).

ECG assessment: ST elevation measured in mm in inferior leads (II - III - avf), and related data was analyzed, the proportion was similar in both groups.

**Table 2.** Number and frequency of narrowed coronary artery in study groups (Group A, n=20) (Group B, n = 11)

| Vessel | N (%) | N (%) | Pvalue |
|---|---|---|---|
| LM | 0 (0) | 0 (0) | NS |
| 3VD | 2 (10) | 1 (9) | NS |
| 2VD | 8 (40) | 6 (55) | NS |
| 1VD | 10 (50) | 4 (36) | NS |

Complications: The incidence of total complication rate in group A was more than group B. Comparison between specific subset of complications was not feasible statistically because of small number of these subgroups. Totally, complication was observed among 45% of group A and 9% of group B (P < 0.05).

**Table 3.** Angiographic characteristics of patients

| Vessel | Group A N (%) | Group B N (%) | Pvalue |
|---|---|---|---|
| LM | 0 (0) | 0 (0) | - |
| LAD | 8 (40) | 5 (45) | NS |
| CX | 7 (35) | 3 (27) | NS |
| RCA | 15 (75) | 8 (72) | NS |

Right ventricular infarction: 7 patients (35%) in group A and 7 patients in group B (65%) had RV infarction (P <0.05). Patients who had posterior MI or lateral MI in association with RVMI were not entered

**Table 4.** Ejection fraction in study subjects

| Group A (n = 20) | | | Group B (n = 11) | | |
|---|---|---|---|---|---|
| LVEF % | n (%) | Cumulative frequency (%) | LVEF % | n (%) | Cumulative frequency (%) |
| 30 | 1 (5) | 5 | 35 | 1 (9) | 9 |
| 35 | 1 (5) | 10 | 40 | 2 (18) | 27 |
| 40 | (4 (20) | 30 | 45 | 2 (18) | 45 |
| 45 | 2 (10) | 40 | 50 | 3 (27) | 73 |
| 50 | 4 (20) | (60) | 55 | 2 (19) | 92 |
| 55 | 6 (30) | 90 | 60 | 1 (9) | 100 |
| 60 | 2 (10) | 100 | | | |
| Mean EF % (47.7 ± 8.4) | | | Mean EF % (47.3 ± 7.4) | | |

P = 0.3

in this analysis because of their enhancing influence in precordial ST depression. 8 (38%) patients in group A and only 1 (9%) in group B showed posterolateral MI (P<0.05). Associated RV MI was not etered in this subgroup of patients. Angiography: Left main artery was normal in all of the patients. 13 (42%) patients showed more than 70% stenotic lesion in left anterior descending (LAD)artery. In 10 (32%) patients significant (> 70%) lesion was present in left circumflex artery. 23 (47%) patients had significant right coronary artery lesion. Table 2 shows angiographic characteristics of patients according to number and frequency of the involved vessel. The number of single vessel two vessel, and 3 vessel disease among both groups were relatively similar. Despite our prediction the proportion of patients with significant LAD stenosis not only was not higher in group A but in relative comparision it was even more likely in group B. Of course this difference was not significant and meaningful (P = 0.1) (Table 3). Mean ejection fraction was 47.4 ± 8.4 in group A and 46.3 ± 74 in group B (P = 0.3) (Table 4).

## DISCUSSION

Several theories have been propounded about the pathogenesis of ST segment changes. The mechanism still remains controversial. One of the more accepted hypotheses is "current of injury theory" (8,9). On the basis of this principle, a lead oriented to injured surface will reflect a raised ST segment and leads facing external surface will reflect reciprocal ST segment depression. During acute myocardial infarction according to this time honoured hypothesis, ST segment has two distinguished categories: first, ST elevation regarded as evolving Q wave or non Q wave MI and second ST depression that generally came to be regarded as subendocardial ischemia (10,11). Despite this classic feature, ST segment depression may not indicate solely myocardiol ischemia or eventual non Q wave MI (3,5); in this context anterior ST segment depression is more problematic when associated with inferior MI. It has been suggested that depression of the ST segment in leads $V_1$ - $V_4$ in the presence of an inferior infarction may indicate ischemia secondary to significant stenosis of LAD (12). Indeed the magnitude of ST depression is influenced by many factors including the size and exact location of the infarct and the presence of left anterior descending coronary artery disease, or three vessel disease (13).

The purpose of this study was to determine the relation of this kind of ST segment depression with the presence of LAD or 3 vessel disease in our patients during thrombolytic era. In some of the previous studies a meaningful correlation has been shown between ST depression and disease of LAD, because patients selected for angiography were more complicated or were high risk or refered patients, consequently the incidence of LAD or 3 vessel disease was considered to be high. For example Salcedo et el have reported LAD lesion in 70% of patients and 3 vessel disease in 33% of patients (4). In our study the frequency of LAD disease was (42%) and that of 3 vessel disease (10%), and there was no significant difference between the two groups.

Few studies have been able to show that precordial ST segment depression can predict patient outcome. Ishikawa et al (14) reported that complication rates in the 17 patients with ST segment depression in leads V1 - to V4 were higher than other groups. Our study revealed that overall complication rate was higher in 21 patients with precordial ST depression which gives support to their findings. RV MI had lower occurance in patients with ST segment depression, in contrast lateral and posterior MI were more frequent in patients with ST depression (P = 0.05). Similar result was obtained in A.S. Lew study in 61 patients by comparison of thallium scan and angiographic findings (15); and this study also confirmed the findings in 25 patients who were evaluated by radionuclide imaging and reported by Goldberg et al (16). They proposed that precordial ST depression is purely an ECG consequence of ST elevation in inferior leads and RV infaction attenuates it whereas posterior or lateral MI increases the magnitude of ST depression.

Ejection fraction was slightly worse in group A but it was not statistically significant in our study. Similar findings but with significant differences have been reported in other studies; however in some of those studies, EF has been measured in acute phase when the myocardium has been in the stunning phase (17-23), while in our study patients the angiography and EF evaluation have been most often performed a few weeks following acute phase.

In conclusion, in this study we evaluated the patients with acute inferior MI with and without precordial ST segment depression on admission ECG and we did not find significant differences between the two groups in relation to frequency of LAD and 3 vessel disease. Complication rate was greater in patients with ST depression and as previous studies we suggest that RV infarctions attenuate the depth of ST depression while posterolateral infarction intensifies it. At the end, it should be stressed that we had some important limitations. First of all our patients belonged to a single center and achieving the best results needs multicenter investigations and analysis in greater number of patients. Secondly, we could not enter all the patients in invasive evaluation and the findings can not be generalized for patients who did not receive thrombolytic therapy and could not undergo angiographic procedure. Finally the effect of concomitant dynamic changes of coronary vessels is a potential limitation that is not possible to be determined.

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