*Original Article*

# Correlation of Sum of ST Segment Depression in Leads $V_1$ to $V_4$ in Acute Inferior Myocardial Infarction with Angiographic Severity of Coronary Artery Disease

Md. Badiuzzaman[1], A F Khabiruddin Ahmed[2], Mohammad Refatul Islam[3], Md. Durul Hoda[4],
Abdur Rashid[5], Faruk Ahmed[6], Iftekhar Alam[7]

## Abstract

**Background:** Inferior wall acute myocardial infarction accounts for 40 to 50% of all acute STEMI. Patients of acute inferior myocardial infarction with ST depression in lateral leads often have greater incidence of triple vessel disease and proximal RCA lesion but in case of patients of acute inferior myocardial infarction with ST depression in precordial leads (leads V1 to V4), is a subject of determination whether it is associated with more sever coronary artery disease. The aim of this study is to determine the association of sum of ST depression in precordial leads with the probability of concomitant severity of coronary artery disease in patients with acute inferior STEMI.

**Methods:** this cross sectional observational study was done at National institute of cardiovascular diseases (NICVD), Dhaka, Bangladesh. Total 90 patients of acute inferior myocardial infarction with precordial lead ST depression admitted at CCU within 12 hours of onset of chest pain were the study population. They were divided in two groups on the basis of sum of ST depression in precordial leads (Sum of ST depression d"4mm and >4mm), 36 patients in group I and 54 patients in group II. Coronary angiography was performed during the index hospitalization period. Gensini score and Reardon score were measured.

**Results:** Overwhelming majority of the patients was male (83.3% and 90.7%) with mean age of 51.0±9.7 and 51.0±9.9 years in group I and II respectively. Smoking, hypertension and diabetes mellitus were the most frequent risk factors in both groups. Serum troponin level was significantly high in group II (6.2±2.2 vs. 13.6±17.7, p <0.05). Sum of ST depression in precordial leads was 2.84±0.66 and 7.53±3.51 in group I and group II respectively, and was statistically significant. Consideration of mean of ST depression in individual leads shows significantly higher mean in leads V2, V3 and V4 among group II patients. SVD was more frequent in group I but group II patients had higher statistically significant incidence of DVD and TVD. Gensini score (20.26±13.0 vs. 36.98±16.9) and Reardon score (4.63±2.2 vs. 6.83±2.2) was high in group II patients. Positive correlation had been depicted between summation value of ST segment depression with that of Gensini score & Reardon score (Gensini score r=0.61 and Reardon score r= 0.52).

**Conclusion:** Involvement Triple vessel and double vessel disease was remarkably higher with increasing sum of ST segment depression in precordial leads. The severity of ST segment depression in terms of summation of ST depression in leads V1 to V4 is directly proportional to the extent and severity of coronary artery disease.

***Key words:*** *Inferior AMI, Sum of ST depression, Gensini score, Reardon score.*

1. Assistant Professor (Cardiology); Shaheed, Tajuddin Ahmed Medical College, Gazipur
2. Associate Professor (Cardiology); Shaheed Tajuddin Ahmed Medical College, Gazipur
3. Assistant Professor (Cardiology); Shaheed Tajuddin Ahmed Medical College, Gazipur
4. Assistant Professor (Cardiology); Shaheed Tajuddin Ahmed Medical College, Gazipur
5. Assistant Professor (Cardiology); Shaheed Tajuddin Ahmed Medical College, Gazipur
6. Assistant Professor (Cardiology); Shaheed Tajuddin Ahmed Medical College,Gazipur
7. Assistant Professor (Cardiology); National Institute of Cardiovascular Disease, Dhaka

**Address of Correspondence:** Dr. Md Badiuzzaman, Assistant Professor (Cardiology), Shaheed Tajuddin Ahmed Medical College, Gazipur. Tel: 01712636235.

## Introduction:

In the era of thrombolytic therapy and primary percutaneous coronary intervention (PCI), it is increasingly important to identify patients with an acute myocardial infarction (AMI) who are likely to develop complications during the early phase of infarction. This is especially true for inferior wall STEMI with ST depression in anterior leads[1].In inferior wall AMI; the infarct related artery can be either the RCA or the LCX[2]. Acute inferior infarction is accompanied by ST segment depression in the precordial chest leads in approximately one half of the patients suffering a first, inferior infarction[3]. Reasons of precordial ST depression is thought to be multi-factorial in terms of infarct size, peri-infarction ischemia, extension of infarction, concomitant LAD or LCX involvement and collateral vessels stenosis[4].

Hospital mortality in patients with ST depression (9.2%) is as twice as high as that in those without ST depression (4.6%)[5]. Coronary angiography (CAG) unveils more frequent left coronary artery disease in patients with precordial ST depression than without it[6]. Anterior reciprocal ST depression accompanying acute Inferior myocardial infarction is known to be attributable to the co-existent LAD & LCX lesion [7]and disease with lower LVEF[8] and henceforth associated with poor prognosis. The prognosis of inferior AMI is adversely affected when it is associated with proximal occlusion of RCA which leads to right ventricular infarction (RVI) with hypotension, high degree AV conduction disturbances, sinus bradycardia and ventricular tachycardia[9].

The magnitude of precordial ST segment depression (sum of leads V1 to V4) adds significant independent prognostic information after adjustment for clinical risk factors[10]. The risk of 30-day mortality increases by 36% for every 0.5 mV of precordial ST segment depression[10]. Therefore, assessment of the magnitude of precordial ST segment depression is useful for risk stratification in patients with an inferior myocardial infarction. Magnitude of sum of ST depression also known as Sigma ST (total degrees of ST segment depression in leads V1 to V4 ) depression in the acute stage is significantly greater in patients with LAD lesion[11]. Patients of acute inferior myocardial infarction with ST depression in lateral leads often have greater incidence of triple vessel disease and proximal RCA lesion. But in case of ST depression in leads V1 to V4, it is subject of determination whether it is associated with single, double or triple vessel disease and severity of CAD.

So in the abovementioned context, magnitude of ST depression play a paramount importance in assessing number of vessels involved. The greater the ST depression in leads V1 to V4 the more is the probability of concomitant double or even triple vessel disease. Therefore, in this study we attempted to unearth the association between the magnitude of ST depression with the coronary artery severity as determined by number of vessels involvement and severity of the lesions.

## Method:

This was cross sectional observational study, carried out in the Department of Cardiology, National Institute of Cardiovascular Disease (NICVD), Dhaka, during the period of January 2011 to December 2011. Sampling method was purposive sampling. Patients with acute inferior myocardial infarction with ST depression of >1mm in at least two contiguous leads of $V_1$ to $V_4$ in 12 leads admission ECG, who had undergone thrombolytic therapy, in CCU of NICVD were included in the study. A total 90 subjects were included in the study on the basis of pre defined inclusion and exclusion criteria. Previous myocardial infarction, previous revascularization, valvular heart diseases and associated posterior myocardial infarction and right ventricular myocardial infarction and ST depression in leads $V_5$ and $V_6$ were excluded.

The measure of ST segment depression at 0.08 second after the J point in the reciprocal leads had been recorded for the qualification of ST segment depression from $V_1$ to $V_4$ precordial leads in mille meter (mm). Magnitude of ST depression was calculated by summation of ST depression in leads $V_1$ to $V_4$. This summation resulted value was expressed in mm. On the basis of sum of ST depression in precordial leads e.g. summation of ST depression V1 to V4 precordial leads patients were divided in two groups. In group I 36 patients with sum of ST depression at or less than 4mm was included and rest of the 54 patients made up the group II whose sum of ST depression was more than 4mm. Coronary angiography was performed during the hospital stay.

Severity of coronary artery disease was assessed by Gensini score and Reardon score. For severity of CAD the Gensini score system was used. The reduction in the lumen diameter and the angiographic appearance

118     Correlation of Sum of ST Segment Depression in Leads $V_1$ to $V_4$
Badiuzzaman M et al.

Bangladesh heart j Vol. 37, No. 2
July 2022

of concentric lesions and eccentric plaques were evaluated (reductions of 25%, 50%, 75%, 90%, 99%, and complete occlusion are given Gensini scores of 1, 2, 4, 8, 16, and 32, respectively). Each principal vascular segment was assigned a multiplier in accordance with the functional significance of the myocardial area supplied by that segment, e.g.: the left main coronary artery, X5; the proximal segment of left anterior descending coronary artery (LAD), X2.5; proximal segment of the circumflex artery, X2.5; the mid-segment of the LAD, XI.5; the right coronary artery, the distal segment of the LAD, the posterolateral artery and the obtuse marginal artery X1.0 and others XO.5. The Gensini score was expressed as the sum of all scores of all coronary arteries. This score therefore, places emphasis on the severity of stenosis, while including some of the extent of CAD21.

In Reardon score system we divided coronary arteries into four parts (Left main, left anterior descending, right coronary, left circumflex artery).Each part divided to segments. Atherosclerotic lesion of each segments was scored (Normal: 0 point, <50%:1point, 50-74%:2 point, 75-99%:3 point, 100%:4 point).Total scores were calculated by sum of each segment score.

All data were analyzed through SPSS 21 software system. Pearson's correlation coefficient was used for correlation study (for r value) and level of significance was carried out by Pearson's correlation T test (for p value)and where appropriate student T test and chi square test were used ($p=<0.05$). This study was approved by ethical review committee of NICVD and written informed consent was taken from all participants.

**Results:**

There was male predominance in between the groups among the study population (Figure 1). Smoking, hypertension, diabetes mellitus and family history of coronary heart disease was the conventional risk factors present in both groups (Table I).  In table II, mean age, BMI, hemodynamic parameters and biochemical parameters were similar in  both groups except serum troponin level which was significantly high in group II patients.  In table III, mean values of ST depression was high in group II and the changes in precordial lead V2, V3, V4 and sum of ST depression was significantly high in group II($p=<0.05$). Coronary angiographic profile as depicted in table IV, SVD was common in group I whereas, prevalence of DVD and TVD was significantly high in group II. Severity of CAD measured by Gensini score and Reardon score was high in group II patients (Table V). Correlation between sum of ST segment depression and Gensini score and Reardon score revealed positive correlation. ($r=0.61$ & $r=0.52$ respectively) which depicted below in figure 2.
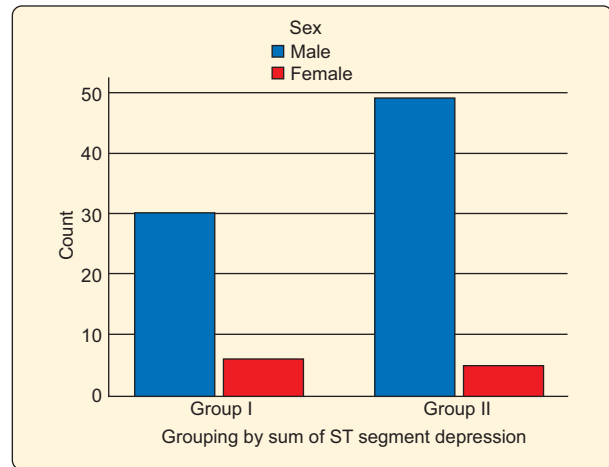


**Fig.-1:** *Distribution of the study subjects by gender among the groups.*

**Table I**
*Risk factor analysis of the study population*

| Characteristics | Group I | Group II | P value |
| --- | --- | --- | --- |
| | Frequency (%) | Frequency (%) | |
| Smoking | 22(24.4%) | 39(43.3%) | 0.269ns |
| Hypertension | 21(23.3%) | 37(41.1%) | 0.323ns |
| Diabetes | 14(15.5%) | 23(25.5%) | 0.726ns |
| Dyslipidaemia | 4(4.4%) | 9(10.0%) | 0.463ns |
| Family H/O IHD | 18(20.0%) | 30(33,3%) | 0.605ns |

**Table II**

*Age, hemodynamic parameters, biochemical variables and ejection fraction of study population.*

| Characteristics | Group I | Group II | P value |
|---|---|---|---|
| | Mean ± SD | Mean ± SD | |
| Age (in years) | 51.03±9.3 | 52.09±9.9 | 0.983[ns] |
| BMI(kg/m$^2$) | 26.67±8.73 | 29.58±7.72 | 0.145[ns] |
| Hemodynamic parameters | | | |
| Heart rate (bpm) | 77.9±10.6 | 78.1±14.2 | 0.480[ns] |
| Systolic BP (mmHg) | 112.4±18.0 | 117.5±18.2 | 0.0936[ns] |
| Diastolic BP (mmHg) | 76.3±11.6 | 78.7±11.3 | 0 .309[ns] |
| Biochemical variables | | | |
| RBS (mmol/L) | 7.39±2.54 | 8.55±4.14 | 0.514[ns] |
| S. Creatinine (mg/dl) | 1.08±0.34 | 1.02±0.24 | 0.257[ns] |
| Troponin-I (ng/ml) | 6.2±12.2 | 13.6±17.7 | 0.003[s] |
| Ejection fraction (%) | 53.39±6.7 | 53.78±7.0 | 0.985[ns] |

**Table III**

*Distribution of mean ST depression in individual precordial leads and the sum of ST depression among the groups.*

| ST depression in precordial leads(mm) | Group I | Group II | P value |
|---|---|---|---|
| | Mean ± SD | Mean ± SD | |
| V1 | 0.56±0.62 | 1.34±0.97 | 0.330[ns] |
| V2 | 1.54±0.76 | 2.89±1.75 | 0.028[s] |
| V3 | 1.09±0.30 | 2.48±1.33 | 0.001[s] |
| V4 | 1.10±0.38 | 2.08±1.35 | 0 .022[s] |
| Sum of ST depression | 2.84±0.66 | 7.53±3.58 | 0.001[s] |

**Table IV**

*Coronary angiographic profile of the study population (n=160).*

| Number of involved vessels | Group I(n=36) | | Group II(n=54) | | p value |
|---|---|---|---|---|---|
| | n | % | n | % | |
| None | 02 | 5.5% | 00 | 0.0% | 0.228[ns] |
| SVD | 10 | 27.7% | 08 | 14.8% | 0.169[ns] |
| DVD | 13 | 36.1% | 22 | 40.7% | 0.022[s] |
| TVD | 11 | 30.5% | 23 | 42.6% | 0.026[s] |
| LM | 00 | 0.0% | 01 | 1.8% | 0115[ns] |

**Table V**

*Severity of CAD among the study groups by Gensini and Reardon score*

| Severity of CAD | Group I (n=36) | Group II (n=54) | p Value |
|---|---|---|---|
| | Mean ± SD | Mean ± SD | |
| Gensini Score | 20.26 ±13.0 | 36.98±16.9 | 0.428[ns] |
| Reardon Score | 4.63±2.21 | 6.83±2.20 | 0.744[ns] |

120    Correlation of Sum of ST Segment Depression in Leads $V_1$ to $V_4$
Badiuzzaman M et al.

Bangladesh heart j Vol. 37, No. 2
July 2022



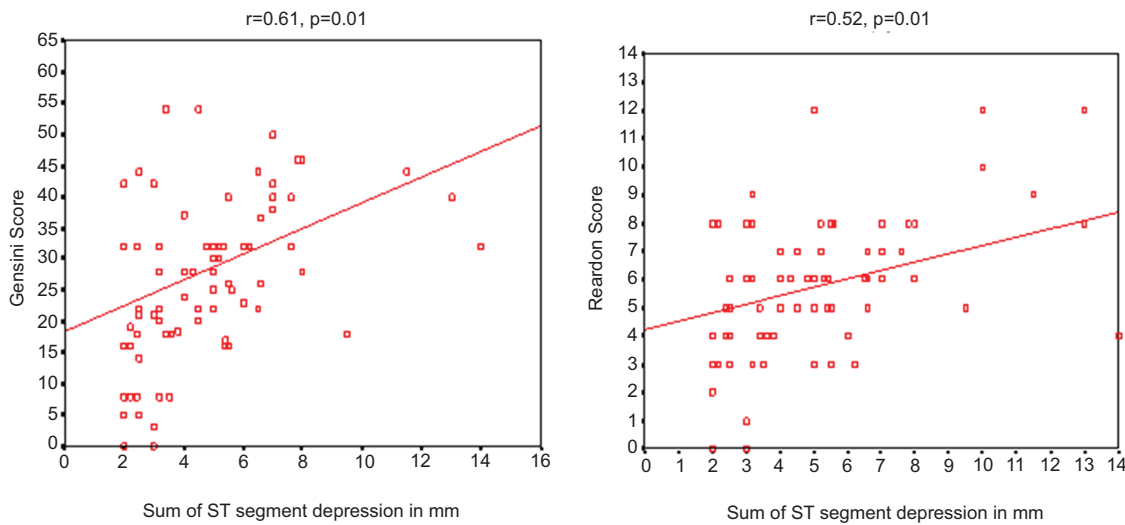**Fig.-2:** *Correlation between sum of ST segment depression and Gensini score & Reardon score.*

## Discussion

In this study there was male predominance in both group, similar distribution was reported by Fiol et.al. in their study[12]. Conventional risk factors of CAD similarly present in both groups, in studies by Fiol et.al. and Karapýnar et.al. distribution was similar except smoking[12, 13]. Mean age was 51.03±9.3 and 52.09±9.9 years in group I and II respectively. Karapýnar et.al.in their study mean age was 54±5 years, which was similar with this study[13]. Serum troponin level was significantly high in group II, other hemodynamic, biochemical variables and ejection fraction was similar in between the groups. Roubin et.al. had increased level of CKMB and troponin in patients with anterior and lateral ST depression in precordial leads[14].

ST depression in precordial leads were significantly high in group II patients and 40% with <4mm ST depression were in group I, similarly Nishian K et.al. had 40% patient with <4mm ST depression and rest had more precordial lead ST depression[11]. Mean value of sum of ST segment depression was 2.84±0.6 and 7.53±3.58 in group I and II, which was much higher than the previous similar study by Karapýnar et.al.[13].

Coronary angiographic profile revealed prevalence of DVD and TVD was significantly high in group II. LM involvement was only 1.8%, all patients had sum of ST depression > 4 mm. Roubin et.al. and David et.al. also reported comparable finding in their studies[14, 15].

Mean value of Gensini score 20.26 ±13.0 and 36.98±16.9 in between the groups, in terms of severity in group II patients Karapýnar et.al. found Gensini score 31.2 ± 30.2, who had more severe CAG lesion[13]. Mean value of Reardon score was 4.63±2.21 and 6.83±2.20 which was little lower for group I patients and similar with group II patients as asserted in the aforementioned study[13].

Correlation test unveiled a positive linear correlation between magnitude of sum of ST depression in leads V1 to V4 with the angiographic score of Gensini and Reardon (r =0.61, p=0.01 for Gensini and r=0.52, p=0.01 for Reardon). Karapýnar et.al found significant correlation between reciprocal ST depression and disease extension (r=0.68 for Gensini score, r= 0.88 for Reardon score, p<0.05 for both).These data provide strong evidence that precordial ST-segment depression during acute inferior infarction is a marker for larger infarction as a result of either ischemia at a distance due to the presence of multi-vessel disease or a greater amount of myocardium supplied by the infarct-related artery.

## Conclusion

This study revealed that severity of ST segment depression in terms of summation of ST depression in leads V1 to V4 is directly proportional to the extent of coronary artery disease as reflected by the r value of Gensini score and Reardon score. ST segment depression in leads V1 to V4 during acute inferior myocardial infarction is associated with frequent multi-vessel disease and more severe CAD.

## References

1.  Bimbaum Y, Herz I, Sclarovsky S. Prognostic significance of precordial ST segment depression on admission electrocardiogram in patients with inferior wall myocardial infarction. J Am CollCardiol1996 ;28 : 313-318.

2.  WellensHJ. The value of the right precordial leads of the electrocardiogram. N Engl J Med 1993; 340: 381-383.

3.  Gelman JS, Saltups A. Precordial ST segment depression in patients with inferior infarction Clinical implications. Br Heart J1982 ; 48 : 560-565.

4.  Dewhurst NG, Muir AL. Clinical significance of reciprocal ST segment depression in acute myocardial infarction. Relative contributions of infarct size and ischemia at a distance. Am J Med 1985;78:765.

5.  Ishikawa K, Kanamasa K, Morishita M. Clinical characteristics of precordial ST-segment depression in acute inferior myocardial infarction. J Cardiol1991; 21(2): 221-8.

6.  Gibelin P, Gilles B, Baudouy M. Reciprocal ST segment changes in acute inferior myocardial infarction: clinical, haemodynamic and angiographic implications. *EurHeartJ*1986;7 (2): 133-139.

7.  Mirvis DM. Physiologic bases for anterior ST segment depression in patients with acute inferior wall myocardial infarction. Am J Cardiol 1988; 116: 1308-1322.

8.  Wasserman AG, Ross AM, Bogaty D. Anterior ST segment depression during acute inferior myocardial infarction: Evidence for the reciprocal change theory. Am Heart J 1983; 105: 516-20.

9.  Majumder A A S, Haque A, Haque M. Right ventricular involvement in acute myocardial infarction: Course, complication and management- an experience at a teaching hospital. Journal of Institute of Post-graduate Medicine & Research 1996; 11 : 22-25.

10. Eric D. Peterson, William R. Hathaway, K. Michael Zabel, Karen S. Pieper, Christopher B. Granger, Galen S. Wagner, Eric J. Topol, Eric R. Bates, Maarten L. Simoons, Robert M. Califf, Prognostic significance of precordial ST segment depression during inferior myocardial infarction in the thrombolytic era: Results in 16,521 patients, Journal of the American College of Cardiology, Volume 28, Issue 2, 1996, Pages 305-312.

11. Nishian K, Naruse H, Kawakami K. Clinical significance of precordial ST segment depression during acute inferior myocardial infarction. J Cardiol 1988; 18(1): 67-77.

12. Fiol M, Carrillo A, Cygankiewich I. Value of electrocardiographic algorithm based on ups and downs of ST in assessment of a culprit artery in evolving inferior wall acute myocardial infarction. Am J Cardiol2004 ;94: 709 -714.

13. Karapýnar, H., Yanartaþ, M., Karavelioðlu, Y., Kaya, Z., Kaya, H., Pala, S., Yýlmaz, A. Importance of Reciprocal ST Segment Depression in the Extensive Coronary Artery Disease. European Journal of General Medicine, 2010. 7(1), pp. 88-91.

14. Roubin GS, Shen WF, Nicholson M. Anterolateral ST segment depression in acute inferior myocardial infarction: Angiographic and clinical implications. Am Heart J1984; 107 :1777-1782.

15. David T. Cardiogenic shock complicating acute coronary syndromes.Lancet 2000; 356: 749-756.