Original Article

In-Hosptal Outcome and Angiographic Findings in Acute Inferior Myocardial Infarction with ST-Segment Elevation in Posterior Chest Leads (V_7, V_8, V_9) Following Thrombolytic Therapy

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Abstract:

Background: Electrocardiographic diagnosis of a posterior wall myocardial infarction is difficult to accomplish by the standard 12-lead ECG. Early detection of posterior wall involvement in an inferior myocardial infarction is of paramount importance for the therapeutic outcome. The aim of this study is to assess the role of ST segment elevation in posterior wall leads (V_7, V_8, V_9) on the admission ECG of acute inferior myocardial infarction, for the diagnosis of posterior wall myocardial infarction and the identification of infarct related artery as well as in-hospital outcome following thrombolysis.

Methods: A total of 90 patients with acute inferior MI were enrolled by purposive sampling. On the basis of ST segment elevation in posterior leads (V_7,V_8,V_9), study subjects were categorized into two groups: 45 patients of acute inferior MI with ST segment elevation in posterior leads as group I and 45 patients of acute inferior MI without ST segment elevation in posterior leads as group II. Coronary angiography was done during index hospital admission. Interpretation of coronary angiogram was done by visual estimation by two cardiologists to assess the severity of coronary artery disease. Severity of coronary stenosis was graded according to the number of major epicardial vessel with significant stenosis by vessel score and Friesinger score. After CAG, patients were evaluated for in hospital adverse outcome like heart block, cardiogenic shock, arrhythmia, and death.

Results: Patients of PMI and non PMI groups were similar in terms of age and sex. Smoking and dyslipidemia (p=0.05) were significantly higher in PMI group. Mean RBS and Troponin-I difference were significantly (p<0.05) higher in group I. Majority of patients had ejection fraction 45-55% in both groups. Patients in group I showed more normal LVEF, than group II, which was statistically significant. This study provided the evidence that the ST segment elevation in posterior leads associated with more left circumflex (LCX) and posterior left ventricular brass (PLVB) involvement. Majority of the patients had vessel score 2, Friesinger score 5-10 in group I and vessel score 1, Friesinger score 1-4 in group II.

Conclusion: ST segment elevation in posterior chest leads (V_7, V_8, V_9) were associated with more in-hospital adverse outcome than those who had inferior MI alone. This group of patients had more PLVB involvement. Recording of posterior precordial leads appear to be beneficial for risk stratification and to locate the site of lesion in patients admitted with acute inferior myocardial infarction. Since it is inexpensive method, it may be used in any hospital.

Key words: Myocardial Infarction, ECG, Thrombolytic Therapy, Angiography

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Introduction

The presentation of acute myocardial infarction is different depending on the coronary artery involved. Inferior wall myocardial infarction results from either right coronary (RCA) or left circumflex coronary artery (LCX) occlusion. Electrocardiographic diagnosis of a posterior wall myocardial infarction is difficult to accomplish by the standard 12-lead ECG, especially during the acute phase.[1] Although an infarction involving the posterior wall

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might occur as an isolated event, it is more often associated with an inferior myocardial infarction. [2]

Diagnosis of a posterior wall infarction in the acute setting is typically based on the ECG detection of ST segment depression in leads V_1 to V_3. [3] However, these changes are relatively insensitive and not specific for the diagnosis of an acute posterolateral infarction, since they may also represent inferoseptal infarction, anterior ischemia or non-Q wave myocardial infarction. [4] Taking into account the fact that the benefit of thrombolytic therapy is proportional to the amount of jeopardized myocardium, it becomes obvious that the early detection of posterior wall involvement in an inferior myocardial infarction is of paramount importance for the therapeutic outcome. Studies have shown that posterior ECG leads (V7, V8, V9) can identify patients with posterior wall infarction. [5]

Acute PMI has been reported to represent 15-20% of acute myocardial infarction – the vast majority occurring with acute infarction of the inferior or lateral wall of the left ventricle. The additional lead ECG, using left posterior chest leads, has increased the rate of isolated PMI diagnosis from "very rare" to a 3-11% range among all patients with AMI. [6] Rapid recognition of acute posterior myocardial infarction is of clinical importance for several reasons. Firstly, patients with acute inferior or lateral wall myocardial infarction who also have posterior involvement are experiencing a large sized infarct. Secondly, the use of acute therapies including treatments aimed at urgent revascularization may benefit patients with acute inferio-posterior myocardial infarction, more than patients with an isolated infarct of a single wall. Lastly, isolated, acute PMI, if not clinically recognized as a transmural infarction, likely will not receive appropriate therapy, including thrombolytic agent or urgent angioplasty. [6]

Materials and method:

This prospective observational study was conducted in the department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh . Objective of the study was to evaluate in-hospital outcome and angiographic findings in acute inferior myocardial infarction with ST segment elevation in posterior leads (V_7, V_8, V_9,) following thrombolytic therapy. Considering inclusion and exclusion criteria 90 patient of acute inferior myocardial infarction with or without ST segment elevation in posterior leads (V_7, V_8, V_9,) treated with thrombolytic therapy who subsequently underwent coronary angiogram were included in this study by purposive sampling . 45 patients were in group I with acute inferior myocardial infarction with ST-segment elevation in (V_7, V_8, V_9) leads and 45 patients were in group II with acute inferior myocardial infarction without ST-segment elevation in (V_7, V_8, V_9) leads. 12 lead ECG with right precordial and posterior leads (V_7, V_8, V_9). Within index hospital admission, the enrolled patients underwent coronary angiography (CAG), and coronary artery lesions were correlated with ECG findings. CAG was analysed by visual estimation. Angiographic severity of coronary artery disease was assessed by Vessel score and Friesinger score.

Vessel Score: This is number of vessels with a significant stenosis (for left main coronary artery 50% or greater and for others 70% or greater reduction in luminal diameter). Left main coronary artery will be scored as single vessel disease. [7]

Score 0 = no vessel involvement.

Score 1 = single vessel involvement.

Score 2 = double vessel involvement.

Score 3 = triple vessel involvement.

Friesinger score: Friesinger index is a score ranges from 0 to 15. Each of the three main coronary arteries is scored separately from 0 to 5. [8]

Score 0: no arteriographic abnormality.

Score 1: trival irregularities (lesion from 1-29%).

Score 2: localized 30-68 luminal narrowing.

Score 3: Multiple 30-68% luminal narrowing of same vessel.

Score 4: 69-100% luminal narrowing without 100% occlusion of proximal segments.

Score 5: Total obstruction of proximal segment of a vessel.

Statistical methods:

The collected data were checked and coded manually and then entered into computer. The numerical data obtained from the study were analyzed and significance of difference were estimated by using statistical methods.

The data obtained were expressed in frequency, percentage, and mean ± standard deviation as applicable. Comparison between groups was done by chi-square test, Student's test or others as applicable. Computer based SPSS (Statistical package for social science) program was used for data analysis. All p values of < 0.05 were accepted as statistically significant.

Results

Mean age was found 51.64±8.28 and 51.22±9.11 years in group I and II respectively ($p>0.05$) . it was observed that in group I, maximum 19(42.2%) patients age

belonged to 41-50 years and in group II 17(37.8%) patients age belonged to 51-60 years. Male were predominant in both groups, 40(88.9%) in group I and 39(86.7%) in group II. It was observed that chest pain, shortness of breath, syncope, nausea, vomiting and sweating had 86.7% vs. 77.8%, 48.9% vs. 53.3%, .7% vs. 2.2%, 24.4% vs. 20.0%, 26.7% vs. 24.4% and 60.0% vs. 60.0% in group I and group II respectively. Back pain was significantly higher (55.6%) in group I than group II (6.7%). Regarding risk factors smoking, HTN, DM, dyslipidaemia and obesity was 33(73.3%) vs. 19(42.2%), 20(44.4%) vs.19(42.2%), 20(44.4%) vs. 24(53.3%), 32(71.1%) vs. 19(42.2%) and 5(11.1%) vs. 2 (4.4%) in group I and II respectively. Smoking and Dyslipidemia difference was statistically significant (p<0.05). Mean RBS, creatinine and Troponin I was found 11.17±3.87 vs. 9.57±3.6 mmol/L, 1.04±0.17 vs. 1.07±0.26 mg/dl and 55.16±48.97 vs. 28.8±29.54 ng/ml in group I and II respectively. Mean RBS and Troponin-I difference was statistically significant (p<0.05) between two groups. Majority patients had ejection fraction 45-54 in both groups, which was 26(57.8%) in group I and 25(55.6%) in group II. Normal wall motion was found 15(33.3%) in group I and 9(20.0%) in group II. Inferior wall motion abnormality more marked, 38 (84.4%) in group II than 12(26.7%) in group I. while, Inferior and posterior wall motion abnormality more marked, in group I, 18(40.0%) than group II, 4(8.9%). Mitral regurgitation was found 12(26.7%) in group I and 5(11.1%) in group II. Ejection fraction, and wall motion abnormality were statistically significant (p<0.05) between two groups (Table I).

In group I majority 17(37.8%) patients had normal RCA and in group II 31(68.9%) patients had mid RCA. The difference was statistically significant (p<0.05). Majority patients had normal LAD in both groups, which was 26(57.8%) in group I and 24(53.3%) in group II. The difference was not statistically significant (p>0.05). In group I, majority 29(64.4%) patients had mid LCX and in group II 27(60.0%) patients had normal LCX. The difference was statistically significant (p<0.05). PLVB more involved in group I 34(75.5%) than group II 15 (13.3%) (Table II). In group I, 71.1% patients had right coronary dominant vessel, 17.8% had left coronary dominant vessel and 11.1% had co-dominant vessel. In group II, most patients (95.5%) also had right coronary dominant vessel, 4.4% had co-dominant vessel (Table III).

In group I majority 22(48.9%) patients had vessel score 2 and in group II 25(55.6%) patients had vessel score 1. In group I majority 22(48.9%) patients had 5-10(moderate) friesinger score and in group II 23(51.1%) patients had 1-4 (mild) friesinger score. The difference was statistically significant (p>0.05) (Table IV). Regarding patent IRA of the study patients it was observed that in RCA, TIMI (2, 3) was found 39 in group I and 22 in group II, it also found in LCX, TIMI (2,3) was found 34 in group I and 43 in group II. This signifies that patent coronary artery more involved in group I than group II. So group I is more benefited from thrombolytic therapy (Table V). More than two third (66.7%) patients had complication in group I and 10(22.2%) patients in group II. No complication patients was found 15(33.3%) in group I and 35(77.8%)

Table-I

Characteristics of study population (n= 90)

Variables	Group I(n=45)		Group II(n=45)		pvalue
Age (years)	51.64±8.28		51.22±9.11		0.819^{ns}
RBS (mmol/L)	11.17±3.87		9.57±3.6		0.045^{s}
Creatinine (mg/dl)	1.04±0.17		1.07±0.26		0.999^{ns}
Troponin –I (ng /ml)	55.16±48.97		28.81±29.54		0.002^{s}
	n	%	n	%	
Male	40	88.9	39	86.7	0.747^{ns}
Female	5	11.1	6	13.3	
Smoking	33	73.3	19	42.2	0.001^{s}
HTN	24	53.3	22	48.9	0.673^{ns}
DM	20	44.4	24	53.3	0.398^{ns}
Dyslipidemia	32	71.1	19	42.2	0.005^{s}
F/H of IHD	12	26.7	10	22.2	0.623^{ns}
Obesity	5	11.1	2	4.4	0.217^{ns}
EF ≥ 55%	17	37.8	3	6.7	0.001^{s}

in group II. Among complication, post infarct angina was found 8(17.8%) in group I and 3(6.7%) in group II. Majority (6.7%) patients had A/V block in group II and 2(4.4%) in group I. Re-infarction was found 2(4.4%) n group I but not found in group II. Atrial fibrillation (AF) was found 6(13.3%) in group I and 2(4.4%) in group II. Killip II heart failure was 7(15.6%) in group I and 2(4.4%) in group II. Cardiogenic shock was 5(11.1%) in group I and 1(2.2%) in group II. Complication and no complication difference was statistically significant ($p<0.05$), as both arrhythmias, heart failure were significantly higher in group I than in group II (Table VI).

Table-II

Distribution by site of coronary artery lesion (n=90)

Site	Group I(n=45)		Group II(n=45)		Pvalue
	n	%	n	%	
RCA					
Normal	17	37.8	5	11.1	
Proximal	8	17.8	1	2.2	
Mid	15	33.3	31	68.9	0.001[s]
Distal	5	11.1	8	17.8	
LAD					
Normal	26	57.8	24	53.3	
Proximal	8	17.8	8	17.8	0.771[ns]
Mid	11	24.4	12	26.7	
Distal	0	0	1	2.2	
LCX					
Normal	7	15.6	27	60.0	
Proximal	2	4.4	9	20.0	
Mid	29	64.4	9	20.0	0.001[s]
Distal	7	15.6	0	0.0	
OM	0	0.0	0	0.0	

Table-III

Distribution of coronary dominance between two groups (n=90)

Dominant Vessel	Group I(n=45)		Group II(n=45)	
	n	%	n	%
Right dominant	32	71.1	43	95.5
Left dominant	8	17.8	0	00.0
Co-dominant	5	11.1	2	4.4

Table-IV

Comparison of coronary angiographic severity between two groups (n=90)

Coronary angiographic severity	Group I(n=45)		Group II(n=45)		P value
Vessel score	n	%	n	%	
Score 0	4	8.9	5	11.1	0.500[ns]
Score 1	15	33.3	25	55.6	0.033[s]
Score 2	22	48.9	12	26.7	0.029[s]
Score 3	4	8.9	3	6.7	0.500[ns]
Friesinger score					
0 (Normal)	3	6.7	3	6.7	0.661[ns]
1-4 (Mild)	13	28.9	23	51.1	0.031[s]
5-10 (Moderate)	22	48.9	14	31.1	0.085[s]
11-15 (Severe)	7	15.6	5	11.1	0.535[ns]

Table-V

Distribution according to patent infarct related artery (IRA) (n=90)

Patent IRA	TIMI 0,1(n=45)		Pvalue	TIMI 2,3 (n=45)		P value
	Group I	Group II		Group I	Group II	
RCA	06	23	0.001[s]	39	22	0.020[s]
LCX	11	02		34	43	

Table-VI

Distribution of in-hospital outcome (n=90)

Hospital outcome	Group I(n=45)		Group II(n=45)		P value
	n	%	n	%	
Complication	30	66.7	10	22.2	0.001[s]
No complication	15	33.3	35	77.8	
Post infarction angina	8	17.8	3	6.7	0.107[ns]
A/V block	3	6.7	6	13.3	0.242[ns]
1^0 HB	1	2.2	2	4.4	
2^0 HB	0	0.0	1	2.2	
CHB	2	4.4	3	6.7	
Re-infarction	2	4.4	0	0.0	0.247[ns]
Arrhythmias	11	24.4	2	4.4	0.006[s]
Atrial fibrillation (AF)	6	13.3	2	4.4	
Ventricular tachycardia (VT)	3	6.7	0	0.0	
Ventricular fibrillation (VF)	2	4.4	0	0.0	
Heart failure Killip I	122	26.74.4	20	4.40.0	0.003[s]
Killip II	5	11.1	2	4.4	
Killip III	4	8.9	0	0.0	
Killip IV	1	2.2	0	0.0	
Cardiogenic shock	5	11.1	1	2.2	0.101[ns]
Death	0	0	0	0.0	

Discussion:

The mean age was found 51.64±8.28 years in group I and 51.22±9.11 years in group II. Maximum (42.2%) patients were in 5th decade in group I and most (37.8%) of the group II patients were in 6th decade. However, no statistical significant mean age difference was found between two groups of patients (p>0.05). Similarly, another study [9] showed the mean age was found 50±8 years in group I and 53±5 years in group II, which is closely resembled with the current study.

Among the studied patients, male were predominant in both groups, 88.9% in group I and 86.7% in group II and male to female ratio was 7.2:1, which is consistent with the result of a study [10] where the percentage of male patient were 73.68% and 84.6% in group I and group II respectively.

The important risk factors in studied patients were, history of smoking (73.3%) in group I and (42.2%) in group II, followed by hypertension (44.4%) and (42.2%) in group I and group II respectively. DM was found (44.4%) in group I and (53.3%) in group II. Dyslipidemia was found (71.1%) and (42.2%) in group I and group II respectively. Family history of IHD was found (26.7%) in group I and (22.2%) in group II. Obesity was found (11.1%) in group II. Contraceptives was found (2.2%) in group II but not found in group I. Smoking and Dyslipidemia difference were significantly (p<0.05) higher in group I but others risk factor were almost similar between two groups. Studies done by others also reported similar data.[10, 11, 12]

In this study it is found that, extensive MI presenting with ST segment elevation in the posterior leads were associated with more favourable effect from thrombolytic

therapy administration. In particular, group I patients showed more normal LVEF, 15(33.3%) than group II, 9(20.0%). This may be due to a patent IRA (TIMI flow 2,3) after successful thrombolytic therapy. In patients with an inferior infarction, posterolateral involvement is associated with the development of significant mitral regurgitation. In this study mitral regurgitation found 12 (26.7%) in group I and 5 (11.1%) in group II. Another study found moderate or severe mitral regurgitation 22.0% in their study patients.[12]

The clinical importance of ST segment elevation in posterior leads has not yet been clarified, even though ECG findings can identify a subset of high risk patients with left circumflex occlusion responsible for a larger MI and a more complicated clinical course. In general, its prognostic implications are not widely known, since a study [12] found that ST segment elevation in posterior leads is combined with a larger MI and a more complicated clinical course (reinfarction, heart failure, mortality), whereas some study [13] failed to show any difference in the incidence of ST segment elevation in posterior leads, in acute MI patients with or without a complicated clinical course.

It was observed that post infarct angina found 17.8% in group I and 6.7% in group II. A/V block was 6.7% and 13.3% in group I and group II respectively. In 43 patients of acute inferior MI, [14] showed that overall conduction disturbance were 58.14%. Of them 53.48% were intraventricular conduction disturbance. Re-infarction was 4.4% in group I but not found in group II.. Arrhythmia was 24.4% in group I and 4.4% in group II. Mitral regurgitation was 26.7% in group I and 11.1% in group II. Heat failure was 26.7% and 4.4% in group I and group II respectively. It was statistically significant. Cardiogenic shock was 11.1% in group I and 2.2% in group II in this study but this was not significant.

According to the CAG finding: 17 (37.8%) patients had normal RCA in group I, and 5(11.1%) in group II. 7(15.6%) patients had normal LCX in group I and 27(60.0%) in group II. The difference was statistically significant ($p<0.05$). In group I, angiographically significant lesion of RCA found in 28(62.2%) patients and of LCX found in 38(84.4%) patients. PLVB more involved in group I patients 14(31.1%) compared to group II patients. This indicates that patients with ST elevation in posterior leads had more PLVB involvement. The difference was statistically significant ($p<0.05$). Meanwhile, group II had more, angiographically significant lesion in RCA, 40 (88.9%) patients than LCX, which is found in 18 (40.0%) patients only and more PLVB involvement.

Coronary angiogram was performed in all study population during index hospital admission. Coronary angiographic severity was assessed by vessel score and Friesinger score. The possible vessel score ranges from zero to three vessel disease. [7] In group I majority (48.9%) patients had vessel score 2 and in group II 55.6% patients vessel score 1. In group I majority (48.9%) patients had friesinger score 5-10 (moderate) and in group II 51.1% patients had friesinger score 1-4 (mild). Another author observed that, Friesinger score 0-4 indicated less extensive disease and Friesinger score e" 5 indicated extensive coronary atherosclerosis. The vessel score and friesinger score were statistically significant.[15]

Regarding the patent IRA of the study patients, it was observed that in RCA, TIMI (2, 3) was found 39 patients in group I and 22 patients in group II, it also found in LCX, TIMI (2,3) was 34 patients in group I and 43 patients in group II. This signifies that patents coronary artery more involved in group I than group II. So group I is more benefited from thrombolytic therapy. Another author [10] showed patent IRA in RCA, TIMI (2, 3) 52(54.74%) in group I and 54(83.08%) in group II. In LCX, TIMI (2,3) was found 41(43.16%) in group I and 8(12.3%) patients in group II, which support the current study. [12]

About the hospital outcome more than two third (66.7%) of patients had complications in group I and 22.2% patients in group II. In this study, more than one complication developed in single patient. Another study showed 63.0%patients had complications in group I and 38.0% in group II, which is similar with the current study. Complications were significantly higher in group I, which were arrhythmias and heart failure. [9]

In general, the 12-lead ECG is less sensitive in identifying left circumflex occlusion. Huey, et al., 1988 found that 52% of patients with acute MI from left circumflex disease did not show any ST segment elevation, while other investigators reported that acute left circumflex occlusion either does not bring about any changes at all in the standard 12-lead ECG [16] or generates only ST depression in the precordial leads (Jacobs, et al., 2000). Scintigraphic studies showed that thallium myocardial perfusion defects in posterolateral segments are relatively specific for left circumflex occlusion (Newman, et al., 1983). Thus, posterior leads may contribute to the regional diagnosis of an acute inferior MI.[13,12] 1998 study claims that an increase in posterior lead sensitivity from 57.7% to 59.7% could lead to a beneficial clinical outcome. In another study [17] showed that the criterion of ST segment e"0.5 mm in the 15 lead ECG (12 classic and

V_7, V_8, V_9) can improve the sensitivity of the diagnosis of acute coronary syndromes attributed to left circumflex occlusion by at least 94%.

Conclusion:

From this study, it may be concluded that patients with ST segment elevation in posterior chest leads (V_7, V_8, V_9) were associated with more in-hospital adverse outcome than those who had inferior MI alone. This group of patients had more PLVB involvement.

Recording of posterior precordial leads appear to be beneficial for risk stratification and to locate the site of lesion in patients admitted with acute inferior myocardial infarction. Since it is inexpensive method, it may be used in any hospital.

Conflict of interest: None.

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