Comparative Study between 12 and 15 Lead Electrocardiograms for Evaluation of Acute Posterior Myocardial Infarction

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Abstract:

Background: *The ECG diagnosis of acute posterior infarction has traditionally been based on the presence of ST segment depression on the pericardial chest leads. However, such ST segment depression is neither specific nor sensitive for the diagnosis of a posterior infarction. Detection of ST segment elevation in V7- V9 in 15 lead ECG can help in early diagnosis of acute Posterior myocardial infarction.*

Method: *Total of 100 patients Inferior myocardial infarction was evaluated by Electrocardiography in standard 12 lead and 15 lead. The patients were categorized into group I having posterior/ and or associated changes and group II having without posterior changes in ECG. Echocardiography and LVgraphy was done to evaluate the posterior wall movement.*

Result: *For diagnosis of posterior myocardial infarction, 43.3% sensitivity and 95.1% specificity was found in 12 lead ECG, whereas in 15 lead ECG it was 80.0% sensitivity and 95.1% specificity.*

Conclusion: *15 Lead ECGs (including V7-V9) more sensitive than 12 lead ECGs in diagnosis of acute posterior myocardial infarction. Patient with inferior myocardial infarction or anterior ischemia developed more complications if associated with acute posterior myocardial infarction. 15 lead ECGs can routinely be used in patients with ischaemic type of chest pain.*

Introduction:

Ischaemic heart disease is a major health problem throughout the world. In the western countries 50% of all death are due to cardiovascular diseases, half of these are attributed to myocardial infarction.[1] In Bangladesh coronary artery disease is the third largest cause of death today. One survey by Malik et al [2] detected the prevalence rate of IHD as 3.3/thousand and a multicentre study by Farid N et al [3] revealed prevalence of IHD as 14/thousand .

The diagnosis of acute myocardial infarction requires a joint evaluation of the clinical aspects, the ECG and the serum enzyme activity. ST Segment of the electrocardiogram (elevation in the leads overlying the infarction and depression in the reciprocal leads) is altered very early in the course of AMI and is one of the indirect markers of myocardial damage. Latter on pathological Q wave and T wave inversion occur. But in many instances, it (about 50% of patients with AMI) does not show diagnostic ECG changes.[4]

Moreover the standard 12 lead ECG is a relatively insensitive tool for detecting posterior myocardial infarction.[5] This a particularly true in the acute stage when prompt and accurate diagnosis of AMI is critical in determining the initiation of reperfusion therapy.[6] The major obstacle in the ECG diagnosis of posterior myocardial infarction lies in the absence of standard leads facing the posterior left ventricular wall, which results in failure to reveal ST segment elevation in a high proportion of patients with acute posterior infarction. Recently it was demonstrated that during acute inferior infarction, ST segment elevation in the posterior chest leads V7-V9 identifies those patients with concomitant posterior wall involvement.[7] It was shown that ST segment elevation is present solely in posterior chest leads in 3%-4% of all patients with AMI and in as many as 20% of Ami patients without ST segment elevation on the standard 12 lead.[8]

Shlomi M et al [8] showed that in acute posterior myocardial infarction ST segment elevation was present in lead V7 and V9 in 91% and in lead V8 in 100% patients. Prominent R-wave appeared in lead V1 in 9% and in lead V2 in 44%. In 25% patients, on admission, no pathologic ST segment changes were noted in standard 12 lead ECG and the ST segment elevation in leads V7 through V9 was the only pathologic ST segment change.

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The ECG diagnosis of acute posterior infarction has traditionally been based on the presence of ST segment depression on the pericardial chest leads.[9,10] However, such ST segment depression are neither specific nor sensitive for the diagnosis of a posterior infarction. These ECG change might be caused by at. Ischaemia [11] and therefore do not constitute an indication for thrombolytic therapy (TIMI-III B. Investigation 1994). Prominent R wave in V1-V2, which might be a manifestation of posterior myocardial infarction, do not enable the determination of the age of the infarction. Shlomi M demonstrates that Isolated with acute posterior myocardial infarction and moderate to severe MR were detected in 22% of such cases.[8] Thus ST segment elevation in leads V7-V9 identify infarctions that are potentially harmful, out of proportion to their size owing to their special location in the left ventricle and thereby their potential to cause MR. The presence of mitral regurgitation after acute myocardial infarction is associated with the development of hemodynamic deterioration of heart failure and poor prognostic outcome.

The presence of ST segment elevation in leads V7-V9 may contribute to the triage of patients with chest pain and help in the early differentiation between patients with acute posterior myocardial infarction and those with anterior wall ischemia. This distinction may influence the decision making process regarding the treatment of patients with acute coronary syndrome. Currently, the indication for thrombolytic therapy requires the presence of ST segment elevation in the standard 12-lead ECG. However, because ST segment elevation is not seen on the standard 12 lead ECG in up to 50% of patients with posterior or circumflex-related infarction [12, 6] the presence of ST should be sought in leads V7-V9. This sub-group of AMI patients can be benefited from thrombolysis or PTCA. Thrombolytic therapy in MI also reduces the incidence of significant MR.

Objectives-
1. To find out the utility of doing V7-V9 chest leads in addition to conventional 12 lead electrocardiogram in patients with ischemic type of chest plain.

2. To evaluate the degree of association of posterior wall myocardial infarction with other wall infarction or ischemia.

3. To find out in-hospital outcome of patients with acute posterior myocardial infarction with or without other wall infarction.

Material & methods
This prospective study was performed on 100 selective patients admitted in the Department of Cardiology, NICVD, Dhaka on the basis of inclusion and exclusion criteria. during the period from July 2000 to December 2000

Inclusion criteria
Selected patients presenting with acute inferior myocardial infarction for the first time or anterior ischemia having.

A. Typical ischemic chest pain for more than 30 minutes with onset within preceding 24 hours.
B. ECG: ST segment elevation or depression contiguous e"2 leads in conventional 12 lead ECG with or without ST elevation in V7-V9.

Exclusion Criteria
a. Chest Pain persisting for less than 30 minutes or onset is more than 24 hours.
b. Patients who was got Streptokinase for this episode of myocardial infarction.
c. History and evidence of previous myocardial infarction.
d. Patient with anterior myocardial infarction or right ventricular infarction.
e. Pericarditis, Myocarditis, Cardiomyoparthy.
f. Patients with RBBB, RVH, LBBB, cardiac pacing.
g. Significant Valvular and Congenital heart diseases.

Electrocardiographic evaluation (ECG)
A standard 12 lead ECG were performed in all patients on admission and within 24 hours of admission. During arrhythmia or chest pain, repeat ECG was done. The Magnitude of ST-segment deviation was measured from the baseline (TP-segment). Significant ST-depression was defined as 1mm or greater horizontal or down-sloping ST-depression measured at 20ms after the J point. The ECG reports were carefully analyzed and the serial ECGs were compared to detect any complication like extension of infarction, pericarditis, arrhythmia, etc. In case of inferior AMI, right pericardial (V4R), leads were recorded on admission or within 2-3 hours of admission. Posterior myocardial infarction was diagnosed if a tall R-wave appeared in lead V1-2 with upright T-wave having R/S ration e"1.0. Particular attention was given to detect any "reciprocal change" in admission-ECG and predischarge-ECG.

15 Lead ECG
Patients having chest and back pain in addition to conventional 12 lead ECG, V7-V9 lead ECG was

done at admission, eight hours after and before discharge. Leads V7 through V9 was recorded on the same horizontal plane as lead V6 and in the posterior axillary line (lead V7), beneath the Scapular angle (V8) and on the paravertiral line (V9) respectively. Significant ST elevation of at least 0.5mm in two or more consecutive leads, based on the increased distance between the posterior chest wall and the heart and Q wave wider than 0.04 sec or deeper than one-quarter of amplitude of the succeeding R-wave in two or more consecutive leads was considered pathologic in leads V7 through V9.

Grouping of the patients

Patients were categorized into Group I and Group II on the basis of 15 lead ECG.

Group I : Patients with acute posterior myocardial infarction (APMI) in 15 lead ECGs (n=34)

Group II : Patients without AMPI in 15 lead ECGs (n=66)

Patients of these two groups were followed up during hospital stay. They were monitored through clinical examination (recurrent chest pain, heart failure, mitral regurgitation etc), ECG (arrhythmia or conduction disturbances), cardiac enzymes assay (CK-MB), echocardiogram, left ventriculography (wall motion abnormalities, ejection fraction and development of MR) and coronary angiogram (vessel involvement).

Statistical analysis

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 the statistical methods. Data were expressed in frequency, percentage, mean and standard deviation as applicable. Comparison between groups was done by standard 't' test, chi-square test, and fisher's exact test as applicable. The sensitivity and specificity of 12 lead and 15 lead ECGs were calculated by comparing with echocardiography and left ventriculography. Data were analyzed but using computer based SPSS program. Probability less than 0.55 were considered as significant.

Results:

Total of 100 patients were evaluated by Electrocardiography in standard 12 lead and 15

Table-I
Clinical presentation of the study patients

Characteristics	Group-1 (n=34)	Group-II (n=66)	Total (N=100)	p value
Back pain	15 (44.1)	4 (6.1)	19 (19.0)	0.001^S
Dyspnoea	19 (55.9)	17 (25.8)	36 (36.0)	0.003^S
Sweating	25 (73.5)	45 (68.2)	70 (70.0)	0.580^{NS}

N.B. Figure in parenthesis indicate percentages
p value reached from chi-square analysis

Table-II
Risk factors of the study population

Risk factors	Group-I (n=34)	Group-II (n=66)	Total (N=100)	p value
Smoking habit	18 (52.9)	37 (56.1)	55 (55.0)	0.766^{NS}
Hypertension	10 (29.4)	22 (33.3)	32 (32.0)	0.690^{NS}
Dyslipidaemia	10 (29.4)	10 (15.2)	20 (20.0)	0.091^{NS}
Family H/O IHD	8 (23.5)	13 (19.7)	21 (21.0)	0.656^{NS}
*Composite risk factors	1.6±1.0	1.4±1.0	1.5±1.0	0.526^{NS}

N.B. Figure in parenthesis indicate percentages
P value reached from chi-square analysis
*p value reached from unpaired student's test

Table III

Electrocardiographic diagnosis made by 12 and 15 lead of the study population

ECG findings	12-Lead	15 –Lead	Final diagnosis
Inferior MI with anterior ischemia	33 (33.0)	26 (26.0)	26 (26.0)
Anterior ischemia	31 (31.0)	25 (25.0)	25 (25.0)
Posterior MI and inferior MI with anterior ischemia	10 (10.0)	16 (16.0)	16 (16.0)
Inferior myocardial infarction	15 (15.0)	15 (15.0)	15 (15.0)q
Posterior MI with anterior ischemia	9 (9.0)	15 (15.0)	15 (15.0)
Posterior MI with inferior MI	2 (2.0)	3 (3.0)	3 (3.0)

lead. The patients were categorized into group I having posterior/ and or associated changes in other electrocardiographic lead and group II having without posterior changes in ECG. The mean (±SD) age of the patients was 51.9±9.0 years. The mean age (±SD) for group I was 53.7±7.4 and for group II was 50.9±9.6 years. It was evident that 87.0% were male and 13.0% were female.

Table-I shows the clinical characteristics of the patients. It was found that cent percent had complaints of chest pain followed by sweating (70.0%), dyspnoea (36.0%) and back pain (19.0%).

Table-II Shows the selected risk factors associated with cardiovascular disease. The composite mean

(±SD) was 1.5(±1.0). Out of 100 patients 55.0% were smoker followed by hypertension (32.0%), family history of ischemic heart disease (21.0%), dyslipidaemia (20.0%) and diabetes mellitus (19.0).

Table-III shows the electrocardiography diagnosis made by 12 and 15 lead. In 12 lead, highest percentages were IMI with anterior ischemia (33.0%) followed by anterior ischemia only (31.0%), IMI (15.0%), IMI, PMI with anterior ischemia (10.0%), PMI with anterior ischemia (9.0%) and IMI and PMI (2.0%). Similar pattern of ECG findings were found in 15 lead, where 26.0% was IMI with anterior ischemia followed by anterior ischemia only (25.0%), IMI and PMI with anterior ischemia (16.0%), IMI (15.0%), PMI with anterior ischemia and PMI with IMI (3.0%)
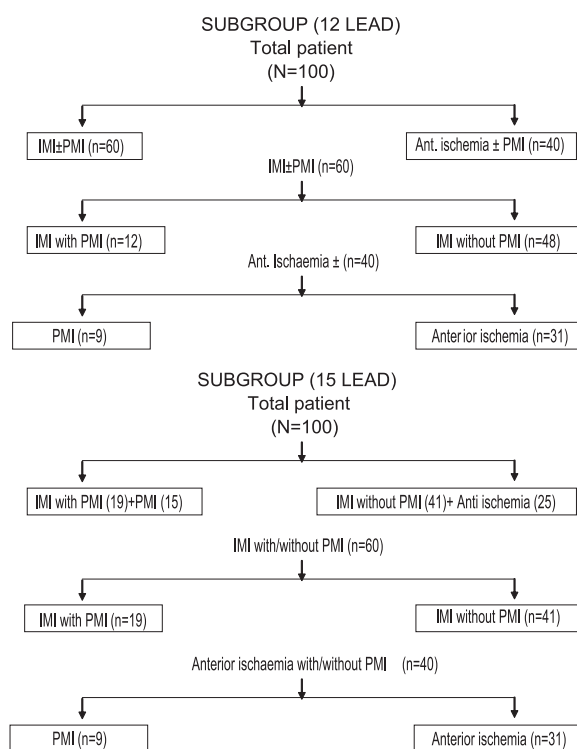

Fig.-1: *Flow chat showing patient distribution in two subgroups*

Tale-IV

In hospital complications of the study population

Complication	Group-I(N=34)	Group-II(n=66)	Total(N=100)	P value
Recurrent Chest Pain	1 (2.9)	1 (1.5)	2 (2.0)	0.0629[NS]
Heart Block	04	09	13	0.0860[NS]
Heart failure	08	07	15	0.009[S]
Arrhythmia	07	06	13	0.147[NS]
Mechanical complication (Mitral regurgitation)	10	06	16	0.002[S]
Death	07	02	09	0.004[S]

*p value reached from chi-square analysis; NS = Not significant (p>0.05).

Table-IV Shows the in-hospital complications of Acute Myocardial Infarction in terms of recurrent chest pain, heart failure, arrhythmia, heart block, and death. 23.5% had heart failure followed by 20.6% death, 20.5% arrhythmia, 11.8% heart block and 2.9% had recurrent chest pain in group I, whereas 13.6% heart block followed by heart failure (10.6%), arrhythmia (9.1%), death (3.0%) and 1.5% had recurrent chest pain in group II.

Significant in-hospital complications were found among the group I in heart failure and death (p<0.05), but no statistically significant difference was found in terms of recurrent chest pain, arrhythmia and heart block between two groups (p>0.05). It was found that 37.0% had mitral regurgitation among the group-I, whereas it was 9.4% in group II and the difference was statistically significant (p<0.05).

Table-V

Wall motion abnormality by Echocardiography among the study population

Wall motion	Group-1 (n=27)	Group-II (n=64)	Total (N=91)	P value
Anterior				
Normal	3 (11.1)	15 (23.4)	18 (19.8)	0.134[NS]
Hyperkinesias	24 (88.9)	45 (77.3)	69 (75.8)	
Akinesia	0 (0.0)	4 (6.2)	4 (4.4)	
Posterior Wall				
Normal	0 (0.0)	64 (100.0)	64 (70.3)	0.001[S]
Hyperkinesias	16 (59.2)	0 (0.0)	16 (17.6)	
Akinesia	11 (40.8)	0 (0.0)	11 (12.0)	
Inferior Wall				
Normal	15 (51.8)	25 (39.1)	40 (43.9)	0.324[NS]
Hyperkinesias	11 (40.7)	34 (53.1)	45 (49.4)	
Akinesia	1 (3.7)	5 (7.8)	6 (6.6)	
Lateral Wall				
Normal	26 (96.3)	62 (96.8)	88 (96.7)	1.00[NS]
Hyperkinesias	1 (3.7)	2 (3.2)	3 (3.3)	
Akinesia	0 (0.0)	1 (1.6)	1 (1.1)	
Septal Wall				
Normal	27 (100.0)	63 (98.4)	90 (98.9)	1.00[NS]
Hyperkinesias	0 (0.0)	1 (1.6)	1 (1.1)	
Apical Wall				
Normal	25 (92.6)	56 (87.5)	81 (89.9)	1.00[NS]
Hyperkinesias	2 (7.4)	8 (12.5)	10 (11.0)	

P value reached from chi-square analysis/Fisher's exact test

Table VI

Echocardiographic mean percent of ejection fraction

Patients	N	Mean	SD	P value
Group-I	27	49.5	6.4	0.001S
Group-II	64	54.5	5.9	
Total	91	52.8	6.5	

P value reached from unpaired student's test
S = Significant (p<0.05)

Table-VII

Coronary Angiography results of the study population

CAG/vessel	Group-I(n=18)	Group-II(n=42)	Total(N=60)	p value
Single vessel	6 (33.3)	15 (35.7)	21 (35.0)	0.982NS
LAD	-	6 (14.3)	6 (10.0)	
RCA	1 (5.5)	7(16.7)	8 (13.3)	
LCX	5 (27.8)	2 (4.8)	7 (16.7)	
Double vessel	9 (50.0)	20 (47.6)	29 (48.3)	
LAD+LCX	4 (22.2)	5 (11.9)	9 (15.0)	
LAD+RCA	-	9 (21.4)	9 (15.0)	
RCA+LCX	5 (27.8)	6 (14.3)	11 (18.3)	
Triple vessel	3 (16.7)	7 (16.7)	10 (16.7)	

P value reached from chi-square analysis
NS = Not significant (p>0.05)

Table-VIII

Lesion in of LCX among the study population

Vessel	Group-I(n=18)	Group-II(n=42)	Total(N=100)	P value
LCX with or without other vessel	17(94.4)	20 (47.6)	37 (61.7)	0.001S
Without LCX	1 (5.6)	22 (52.4)	23 (38.3)	

P value reached from chi-square analysis

Table IX

Dominant vessel in CAG among the study population

Dominant vessel	Group-I(n=18)	Group-II(n=42)	Total(N=60)	P value
Right dominant	9 (50.0)	37 (88.1)	46 (76.7)	0.001S
Left dominant	9 (50.0)	4 (11.9)	13 (23.3)	

P value reached from chi-square analysis
S = Significant (p<0.05)

Table X

Wall motion abnormalities by LV graphy among the study population

WMA by LV graphy	Group-I(n=18)	Group-II(n=42)	Total(N=60)	P value
Anterolateral	2 (11.1)	4 (9.5)	6 (10.0)	0.001S
Anterobasal	0 (0.0)	5 (11.9)	5 (8.3)	
Apical	0 (0.0)	5 (11.9)	5 (8.3)	
Poster basal	6 (33.3)	3 (7.1)	9 (15.0)	
Inferior	0 (0.0)	22 (52.4)	22 (36.7)	
Poster lateral	10 (55.6)	3 (7.1)	13 (21.7)	
Septal	0 (0.0)	0 (0.0)	0 (0.0)	

S = Significant (p<0.05)

Table XI

*Sensitivity and specificity analysis of diagnostic evaluation of 12-Lead and 15-Lead
ECG compared with Echocardiography.*

Test	12 Lead			15 Lead		
	Positive	Negative	Total	Positive	Negative	Total
Positive	13(43.3)	3(4.9)	16(17.6)	24(80.0)	3(4.9)	27(29.7)
Negative	17(56.7)	58(95.1)	75(82.4)	6(20.0)	58(95.1)	64(70.3)
Total	30(100.0)	61(100.0)	91(100.0)	30(100.0)	61(100.0)	91(100.)
Sensitivity	43.3%		80.0%			
Specificity	95.1%			95.1%		

Table XII

*Sensitivity and specificity analysis of diagnostic evaluation of 12-lead, 15-lead and Echocardiography
compared with Ventriculography*

Test	12 Lead			15 Lead			Echocardiography		
	+ve	-ve	Total	+ve	-ve	Total	+ve	-ve	Total
Positive	7(35.0)	4(10.0)	11(18.3)	15(75.0)	3(7.5)	18(30.0)	18(90.0)	2(5.0)	20(33.3)
Negative	13(65.0)	36(90.0)	49(81.7)	5(25.0)	37(92.5)	42(70.0)	2(10.0)	38(95.0)	40(66.7)
Total	20(100.0)	40(100.0)	60(100.0)	20(100.0)	40(100.0)	60(100.0)	20(100.0)	40(100.0)	60(100.0)
P value	001S		0.001S		0.001S				
Sensitivity	35.0%		75.0%		90.0%				
Specificity	90.0%			92.5%			95.0%		

Table-V shows the wall motion abnormality detected by echocardiography. It was evident that no statistically significant difference in wall motion was found in group I and group II except posterior wall (p<0.05)

Table-VI shows the echocardiographic mean percent of ejection fraction of the study patients. The mean percent of ejection fraction was 49.5±5.9 in group II. It was found that the mean percent of ejection fraction was significantly lower in group-I than group-II (p<0.05).

It was observed that about one third (35.0%) had single coronary artery involvement in CAG finding, 48.3% had double blood vessel lesion and only 16.7% had triple vessel lesion. No statistically significant difference was found between group I and group II (p>0.05). (Table VII)

Table X shows the ventriculographic findings of the study patients. It was found that 88.9% had poster basal and poster lateral lesion whereas 14.2% had posterior lesion in group II group and 76.2% had other site lesion. A statistically significant difference was found between group I and group II in ventriculographic findings indicating group I had posterior myocardial infarction (p<0.05).

Table XI shows the sensitivity and specificity analysis of 12 and 15 lead compared with echocardiography. Regarding diagnosis of posterior myocardial infarction, 43.3% sensitivity and 95.1% specificity was found in 12 lead, whereas in 15 lead it was 80.0% sensitivity and 95.1% specificity.

Table XII shows the sensitivity and specificity analysis of 12, 15 lead and echocardiography compared with ventriculography. The sensitivity were 35.5%, 75.0% and 90.0% in 12 lead, 15 lead and echocardiography and specificity was 90.0%, 92.5% and 95.0% in 12 lead, 15 lead and echocardiography.

Discussion:

Diagnosis of acute posterior myocardial infarction is often missed on routine 12 lead ECGs . Boden et al [13] suggested that pericardial ST segment depression with an upright T wave is an early

electrocardiographic sign suggestive of posterior myocardial infarction .

Agarwal et al.[14] found that PMI often have anterior ST segment depression and are considered to have unstable angina with ischemia in the anterior descending coronary artery distribution or even left main coronary related infarction. Recording of posterior chest leads (V7-V9) particularly in patients with anterior ST segment depression is an easy method to diagnose posterior infarction. This may help the clinicians in managing the patients. Shah and Berman [15] demonstrate that patients with a first inferior myocardial infarction who had concomitant pericardial ST depression had more frequent and severe reduction of LVEF than do those without precordial ST segment depression. Patients with such ST depression had higher peak serum CK-MB level and more frequent in-hospital complications than did patients without such ST depression. Pericardial ST segment depression in a setting of first acute infarction involving the inferior wall represent more extensive ischemic injury to the left ventricle most, likely due to posterior myocardial infarction.

About 43 percent patients with pericardial ST segment depression did have anterolateral wall motion abnormalities, but these occurred only in patients with very extensive poster lateral wall motion abnormalities. Prognostic significance of pericardial ST segment depression was studied by various authors. Krone et al.[16] conducted studies to determine whether pericardial ST segment depression had any prognostic significance. They indicated that pericardial ST segment depression in acute inferior myocardial infarction predicted poor short term and long-term prognosis and had a hospital mortality rate of 13 % compared with 4 percent in patients without ST depression. Most of the patients with anterior ischemia as evidenced by 12 lead ECGs were the mirror image of posterior myocardial infarction evidence by 15 lead ECGs.

The mean (±SD) age of this study population was 51.9±9.0 years. The majority of the patients (44$) were in the age group 40-50 years. Similar findings wes Gibson et al.[17] who showed mean age as 52 years. In the Bangladeshi population, Rahim [18] reported a mean age of 51 years (range 23-75 years) among the patients with myocardial infarction. When the mean age was compared between two

groups i.e. in group I and group II, showed no statistically significant difference (p>0.5). In the present study, there were 13 % female.

Among the risk factors, Cigarette smoking was the commonest, 55 % seen in the study patients, 32 % had history of hypertension and 19 % had diabetic. On comparison of the risk factors (including lipid profile) in the two groups, no statistically significant difference was found (p>0.05).

Cardiac enzyme (CK-MB) was elevated both on admission and six hours after admission in group I and the enzyme level between two groups was statistically significant (p<0.001). This finding was comparable to the findings of Gellman and Saltrups [19] that showed that peak. CK-MB level of patients with pericardial ST segment depression (1779 U/L) was statistically higher (p<0.001) than without ST depression (1090 U/L). The higher level of peak CK levels to the larger infarcts seen in the patients with pericardial ST segment depression. Matetzky S et al.[7] showed in PMI, Creatine Kinase (CK) was elevated twice the upper normal limit. The mean CK-value was 663±314 IU/L. The upper normal range was 90 IU/litre).

In this study, cent percent of the study population presented with the complains of chest pain. One fifth of cases (19.0%) complaints of back pain in association with retrosternal pain. This presentation (back pain) was statistically significant (p<0.001). Menown A B et al[20] mentioned that posterior wall involvement may present with pain radiating to the back. But they did not mention how many percent patients with posterior well infarction were suffered.

In this study, highest percentage were inferior myocardial infarction with anterior ischaemia 33.0 % followed by anterior ischemia 31.0 % inferior myocardial infarction (MI) with posterior myocardial infarction (PMI) with anterior ischemia 10 %, IMI and PMI 2 % and IMI 15 % were detected by using 12 lead ECGs.

Similarly in 15 lead ECGs- similar pattern of ECG findings were found, where 26 % was IMI with anterior Ischemia, followed by anterior Ischemia 25 % IMI and PMI with anterior Ischemia 16% , PMI with anterior ischemia 3 % and PMI with IMI 3 % were detected.

Cases RE et al.[21] in their study, by using additional three leads (V7-V9) of patients with either because of tall R waves in V1 and/or V21 or because of clinical suspicion of posterior infarction had shown that out of 250 patients 110 patients (44%) showed evidence of new or old posterior myocardial infarction consisting of ST segment elevation and / or board Q waves in elad V7, V8, V9 in 27 them (10.8%) only these were diagnostic of infarction. In 83 others (33.2%) signs of infarct also were present in inferior (LII LIII aVF) or occasionally in high lateral leads as well. Gibson RS et al. showed only 5 percent posterior myocardial infarction was associated with IMI without ST segment depression and 26 percent PMI associated with IMI with anterior Ischemia patients. [17]

Matetzky S et al. [22] demonstrated that 53 percent had significant ST segment elevation in posterior chest leads V7-V9 of patients with inferior myocardial infarction. Matetzky S et al.[7] showed, ST segment elevation was present as many as 20% of AMI patients without ST elevation on the standard 12 lead ECGs. Boden WE et al. [10] showed that non Q-wave anterior AMI patients progressed to posterior AMI (approximately 30% of all Q wave infarction).

The ECGs findings of the present study was comparable with the above studies abroad in this study, 31.3% IMI patients were associated with PMI and 37.5 % anterior ischemia associated with PMI. Sensitivity and specificity for diagnosis of acute PMI by conventional 12 lead as compared with 2D Echocardiography and LV graphy which was 43.3 % sensitivity and 95.1% specificity compared with echocardiography whereas it was 35.0% sensitivity and 90.0 % specificity compared with LV graphy. On the contrary, in 15 lead, 80.0% sensitivity and 95.1 % specificity compared with echocardiography, this was 75.0 % specificity compared with echocardiography, this was 75.0 % sensitivity and 92.5% % specificity compared with LV graphy. So, compared with conventional 12 leads, 15 lead could be more sensitive and specific (p<0.05) tool for the diagnosis of PMI. Matetzky et al [7] showed the sensitivity was 44 % and specificity was 97 %in 12 lead ECG, whereas 91% sensitivity and 97 % specificity in 15 lead ECG compared with echocardiography. On follow up a total 36 % patients were presented with complains of shortness

of breath. Among them 19 % from case group and the difference between the groups in the presentation of shortness of breath was statistically significant (p<0.003). The clinical evidence of heart failure was graded using Killips classification. 33.5 % patients from group I and 10.6 percent from group I had clinical evidence of heart failure. Patients in group I more often had heart failure class III and IV than in group II (14.7% vs. 3%). This difference was statistically significant (p<0.001). Matetzky S et al. [22] compared heart failure in the patients with or without ST segment elevating in the posterior chest leads. They showed that patients having St segment elevation more often had heart failure (11% vs. 5%) than patients without St segment elevation.

Arrhythmia (13 %) and conduction disturbances (13 %) were seen in the study patients. 20.5 %of the patient in group I had arrhythmias, premature ventricular contraction being the most common (11.8%). Conduction disturbances were seen in 11.7 % in group I and 10.3 % in group II. There were no statistically significant difference was found between two the groups (p>0.05). Death rate was higher in group I (20.6 %) than group II (3 %). This was statistically highly significant (p<0.001). The mortality rate was a bit higher than other study showed by Matetzky S et al [22] where the rate 6.5 %. This difference may be due to small sample size.

Echocardiographic evaluation of wall motion abnormalities showed that posterior wall was hyperkinetic as well as akinetic in patients in group I. Inferior wall hyperkinesias was in 40.7% and akinesia 3.7 %, lateral wall was 3.7 % hyperkinesias in group I of patients. It was evident that no statistically significant wall motion difference was found in between two groups except posterior wall, had significant wall motion abnormality (p<0.05), Matetzky S et al [7] had performed echocardiographic study in the patients with posterior myocardial infarction and showed that left ventricular posterior wall motion abnormality was the most common regional dysfunction, which was evidenced in 97 % patients. Inferior and lateral wall motion abnormalities were found in 47 % and 41 % respectively. The posterior wall was also the most severely abnormal being akinetic or dyskinetic in

61 %. Ejection fraction by echocardiography showed the moderate to severe LV dysfunction in group-I and mild to moderate in-group-II. Matetzky S et al.[7] found similar result by echocardiography. 89 Among the mechanical complications the incidence of mitral regurgitation was more 38.9 % in the patients of group I and only 11.9 % in group II by LV graphy. This was statistically highly significant (p<0.001). Matetzky S et al.[7] was detected moderate to severe MR in 22 % patient and mild MR in an additional 47 % patients having posterior myocardial infarction.

Total 60 of the patients underwent coronary angiography. Among them 18 patients from group I and 42 patients from group-II. In group-I single vessel involvement LCX (5(27.8%), RCA 1(-5.5%), double vessel involve RCA+LCX 5(27.8%), LAD+LCX 4(22.2%). Triple vessel involved only 3(16.7%).

Among the total 18 patients, LCX involvement was in 17 patients (94.4%), Among the patients from group II, single vessel involvement was 15 (35.7%), double vessels 20(47.6%) and triple vessels 7(16.7%). Among the total 42 patients from group II, LCX involvement was 20(47.6%). Significant difference was found for involvement of LCX between the two groups. It was comparable with the study by Matetzky S et al [7] where it was shown that 80 % patients had mid-portion LCX lesion and 20 % patients had lesion in the first marginal branch of the circumflex coronary artery. Casas RE et al [21] had shown that LCX involvement was more in patients with inferior myocardial infarction associated with posterior myocardial infarction (out of 21 patients LCX involvement in 17 patients) than in patients without involvement of PMI (out at 23 patients – LCX involvement in II patients). It was statistically significant (p<0.05).

The ejection fraction, wall motion abnormality and mechanical complications were studied during left ventriculography at RAO and LAO projection. The mean (±SD) percent of ejection fraction of the group I was 47.3±4.2 and group II 53.9±5.3. The difference between the groups was statistically highly significant (p<0.001). The findings of this study corroborate with that found by Matetzky S et al. [7] and this showed that LVEF was lower among the patients with posterior myocardial infarction (44±5%). The wall motion abnormalities on the

ventriculography showed the patients in group I had hypokinesia of poster basal and posterior wall motion abnormality in group II. Mitral regurgitation as evidenced by passes of dy from LV to LA during systole during the procedure was detected 38.9% group I and only 11.9% in group II. It was comparable with the study Matetzky et al.[7] who showed that moderate to serve MR was detected 22 % in patients with posterior myocardial infarction.

Conclusion:

15 Lead ECGs (including V7-V9) more sensitive than 12 lead ECGs in diagnosis of acute posterior myocardial infarction. Patient with inferior myocardial infarction or anterior ischemia developed more complications if associated with acute posterior myocardial infarction. 15 lead ECGs can routinely be used in patients with ischaemic type of chest pain.

A significant number of anterior ischemia patients actually mirror image of acute posterior myocardial infarction as evidenced by ST segment elevation in V7-V9 in 15 lead ECGs. The identification of latter ECG pattern will enable this sub group of AMI patients to benefit from early thrombolysis or direct PTCA rather than conventional treatment of ischemia.

15 lead ECGs can be helpful in the early diagnosis of acute posterior myocardial infarction in patients with ischemic type of chest pain without change in ST segment in 12 Lead ECGs and thereby facilitation prompt and accurate treatment.

Limitation of the study

The study was limited by small sample size. Yet, this study is the first attempt to emphasize on proper and prompt diagnosis of acute posterior myocardial infarction by simple 15 lead ECGs, and early detection of complications following acute posterior myocardial infarction, which may reduce the short and long term morbidly and mortality. Thus this study may serve as an impetus for future studies on acute posterior myocardial infarction on a larger scale in our country.

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