IMPORTANCE OF 15 LEAD ECG
K. M. Bhargav

HOW TO CITE THIS ARTICLE:

ABSTRACT: BACKGROUND AND OBJECTIVES: Posterior wall myocardial infarction is not uncommon acute myocardial infarction and has got its own therapeutic and prognostic implications. Management of Posterior wall myocardial infarction differs from inferior wall myocardial infarction alone. The presence of posterior wall myocardial infarction is known to increase the incidence of cardiogenic shock, arrhythmias and conduction blocks in case of myocardial infarction. Hence the present study was taken up to find out the incidence, clinical profile and complications of posterior wall myocardial infarction in a rural hospital using simple non-invasive investigations like 15 lead electrocardiography and echocardiography. METHODS: A prospective study was conducted on 50 patients of inferior wall myocardial infarction out of 228 Acute Myocardial Infarction proved by ECG standard and posterior leads (v7, v8, v9) were taken at the time of admission and repeated as necessary. A detailed case history was taken and a detailed physical examination was done at the time of admission and during follow up. For recording ECG 12 lead (3 standard leads, 3 augmented limb leads, 6 precordial leads) machine was used. The recordings were made at 25 mm/sec. speed and 1mV=10mm. Posterior leads were taken by using 3 precordial leads fixing on the posterior axillary (v7), infrascapular (v8) and paraspinal (v9) regions all in a same line with the 5th ICS anteriorly. RESULTS: Out of 50 cases of inferior wall myocardial infarction (IWMI) studied only 13 (26%) had ST elevation in posterior leads indicating posterior wall myocardial infarction (PWMI). Our study showed that complications and mortality was higher in patients of IWMI with PWMI compared to IWMI without PWMI. Out of 50 patients 33 (66%) were males indicating a male predominance. Syncope was present in 18% of PWMI and 14% in overall IWMI. Palpitation was seen in 53% of PWMI and 21% of IWMI without PWMI. Smoking history was present in 14% of the patients with IWMI where as it was 42% in patients of PWMI. History of alcoholism was present in 22% of IWMI patients and 38% in the patients of PWMI and 6% in IWMI without PWMI. Diabetes was seen in 30% of PWMI patients where as it was 35% in patients IWMI without PWMI and 34% overall. Hypotension was present in 46% of PWMI and 16% of IWMI without PWMI. Ventricular fibrillation was seen in 8% of PWMI and 2.8% of patients of IWMI without PWMI. Heart blocks were seen in 15% of PWMI and in 10% of patients of IWMI without PWMI. Mortality was 23% in PWMI and 10% in patients of IWMI without PWMI. INTERPRETATION AND CONCLUSION: The incidence of mortality and complications can be reduced only when we are fully aware of diagnosis and complications that can occur in PWMI. So, in all cases of IWMI, PWMI should be looked for by using simple and specific investigation like Posterior leads ECG. It should be kept in mind that incidence of complications like hypotension, conduction defects and arrhythmias are very high in PWMI and hence early detection is important. KEYWORDS: Posterior wall myocardial infarction; Posterior leads; Inferior wall MI; Complete heart block; Syncope; Bradycardia; Hypotension.
INTRODUCTION: The most common cause of ST elevation myocardial infarction is a complete thrombotic occlusion in a major coronary artery causing transmural injury. Striking ST segment elevation is present in the inferior leads II, III, and aVF, suggesting that the right coronary artery is the infarct-related artery. Myocardial ischemia tends to be a regional event. When both ST segment elevation and depression are observed on the 12 lead ECG, the ST elevation is usually considered primary and the ST segment depression changes are considered reciprocal.

When the distal right coronary artery blood supply is inadequate, the posterior wall may become involved. Because there are no electrodes facing the posterior wall in the standard 12-lead ECG, reciprocal ST changes are represented as ST depression in leads V2 and V3. In addition, ST elevation is present in lead V6, indicating apical injury.

Posterior myocardial infarction refers to infarction of the posterobasal wall of the left ventricle. The diagnosis is often missed as the standard 12 lead ECG does not include posterior leads. The changes of posterior myocardial infarction are seen indirectly in the anterior precordial leads. Leads V1 to V3 face the endocardial surface of the posterior wall of the left ventricle.

As these leads record from the opposite side of the heart instead of directly over the infarct, the changes of posterior infarction are reversed in these leads. The R waves increase in size, becoming broader and dominant, and are associated with ST depression and upright T wave. This contrasts with the q waves, ST segment depression and T wave inversion seen in acute anterior wall myocardial infarction. Ischemia of the anterior wall of the left ventricle also produces ST segment depression in V1 to V3, and this must be differentiated from posterior myocardial infarction.

The use of posterior leads V7 to V9 will show ST segment elevation in patients with posterior infarction. These additional leads therefore provide valuable information, and they help in identifying the patients who may benefit from urgent reperfusion therapy. Therefore the following study is taken to know the importance of 15 lead ECG in inferior wall myocardial infarction patients. Therefore the following study is undertaken to know the importance of 15 lead ECG recording in the sub group of patients.

METHODOLOGY:
Sample Size: The sample size was 50 patients.

Study Design: One year cross sectional study.

Study Population: Fifty patients with acute inferior myocardial infarction of all ages admitted RMMCH, Chidambaram constitutes the study population.

Inclusion Criteria: All patients of acute inferior wall myocardial infarction, defined as ECG changes, in patients with prolonged chest pain, of ST segment elevation in leads, II, III, aVF, ≥ 1 mm.

Patients of all age groups were included, from the time of admission to time of discharge in the hospital.

Exclusion Criteria:
- Severely ill patients.
- Pregnant women.
- Morbid obesity.
- With on-going stroke.
- Electrolyte imbalance.
- Patient on digoxin.
- Anti-arrhythmic and anti-depressants.
- COPD

**Data Collection:** 50 consecutive patients with acute inferior wall myocardial infarction were included in the study. Their detailed history, including duration of chest pain, associated symptoms, presence of risk factors, past history of coronary artery disease was obtained as per the proforma. It was followed by detailed physical examination.

Then their 15 lead electro cardiograph was taken. All patients were subjected to echocardiogram within first 24 hrs of admission. The diagnosis of myocardial infarction was confirmed by measuring their serum CKMB levels.

**15 LEAD ECG:**

**PROCEDURE:**

- The 15 lead ECG was obtained by using a standard 12-lead ECG machine. After taking a 12-lead electrocardiograph, ECG V7, V8, V9 were recorded by placing the usual precordial leads (V1 to V6) over the additional areas posterior axillary (v7), infrascapular (v8), and paraspinal (v9) regions. Subsequently patients were divided into two groups
- IWMI only: Isolated inferior wall MI only, when ST segment elevation ≥1 mm was seen only in leads, II, III and aVF.
- IWMI with PWMI – Inferior wall MI with posterior wall MI when ST segment elevation ≥ 1 mm in leads II, III, aVF, V7 to V9.
- Patients were also classified on the basis of L.V. ejection fraction as assessed by an 2D echocardiogram. Patients with LVEF ≤ 45% were classified as with systolic LV dysfunction, those with LVEF = 50% as borderline systolic LV function and Those with LVEF ≥ 55% as normal systolic LV function.

**Statistical Method:** Statistical analysis was done using Chi-square test. It was considered significant if p<0.05.

**RESULTS:**

<table>
<thead>
<tr>
<th>Total No. of Acute MI</th>
<th>No. of IWMI among Acute MI</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence in all groups (n)</td>
<td>227</td>
<td>118</td>
</tr>
</tbody>
</table>

**Table 1: Incidence in all groups**

<table>
<thead>
<tr>
<th>Total No. of IWMI</th>
<th>No. of PWMI in IWMI</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence of PWMI in IWMI (n)</td>
<td>50</td>
<td>13</td>
</tr>
</tbody>
</table>

**Table 2: Incidence of PWMI in IWMI**
### IWMI without PWMI (37) | PWMI (13) | Total | P Value
--- | --- | --- | ---
**AGE IN YEARS**
20-29 | 0 | 0 | 0.001 (P<0.01)
30-39 | 3(8%) | 0 | 3(6%)
40-49 | 2(5%) | 4(30%) | 6(12%)
50-59 | 13(35%) | 5(38%) | 18(36%)
60 and above | 19(51%) | 4(30%) | 23(46%)
Mean | 7.40 | 2.60 | 0.001 (P<0.01)
SD | 8.20 | 2.40 | 
**SEX**
Male | 24(64%) | 9(69%) | 33(66%)
Female | 13(36%) | 4(31%) | 17(34%)
Mean | 18.50 | 6.50 | 
SD | 7.77 | 3.53 | 
**RISK FACTORS**
Diabetes | 13(35%) | 4(30%) | 17(34%)
Hypertension | 11(30%) | 2(15%) | 13(26%)
Smoking | 4((11%) | 3(23%) | 7(14%)
Alcohol | 6(16%) | 5(38%) | 11(22%)
Mean | 8.50 | 3.50 | 0.001 (P<0.01)
SD | 4.20 | 1.29 | 

Table 3: Age Incidence, Sex Incidence, Incidence of Risk Factors

P<0.01 Significant

### Symptoms

<table>
<thead>
<tr>
<th>Symptom</th>
<th>PWMI (n=13)</th>
<th>IWMI without PWMI (37)</th>
<th>IWMI (n=50)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest Pain</td>
<td>10(76%)</td>
<td>35(94%)</td>
<td>45(90%)</td>
<td>0.001 (P&lt;0.01) significant</td>
</tr>
<tr>
<td>Syncope/giddiness</td>
<td>0</td>
<td>7(18%)</td>
<td>7(14%)</td>
<td></td>
</tr>
<tr>
<td>Palpitation</td>
<td>7(53%)</td>
<td>8(21%)</td>
<td>15(30%)</td>
<td></td>
</tr>
<tr>
<td>Sweating</td>
<td>8(61%)</td>
<td>9(24%)</td>
<td>17(34%)</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>6.25</td>
<td>14.75</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>4.34</td>
<td>13.52</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Physical findings

<table>
<thead>
<tr>
<th>a. Pulse</th>
<th>PWMI (n=13)</th>
<th>IWMI without PWMI (37)</th>
<th>IWMI (n=50)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Normal(60-100/min)</td>
<td>9(69%)</td>
<td>33(99%)</td>
<td>42(84%)</td>
<td>0.001 (p&lt;0.01) significant</td>
</tr>
<tr>
<td>2. Bradycardia (&lt;60/min)</td>
<td>2(15%)</td>
<td>1(2%)</td>
<td>3(6%)</td>
<td></td>
</tr>
<tr>
<td>3. Tachycardia(&gt;100/min)</td>
<td>2(15%)</td>
<td>3(8%)</td>
<td>5(10%)</td>
<td></td>
</tr>
</tbody>
</table>
b. Blood pressure

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>Normotensive (100-140/60-90) mm of hg</th>
<th>Hypotension (&lt;100/&lt;60) mm of hg</th>
<th>Hypertension (&gt;140/&gt;90) mm of hg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4(31%)</td>
<td>6(46%)</td>
<td>3(23%)</td>
</tr>
<tr>
<td></td>
<td>29(78%)</td>
<td>6(16%)</td>
<td>2(5%)</td>
</tr>
<tr>
<td></td>
<td>33(66%)</td>
<td>12(24%)</td>
<td>5(10%)</td>
</tr>
</tbody>
</table>

Mean: 4.33, 12.33
SD: 2.73, 14.61

ECG Showing Arrhythmias

<table>
<thead>
<tr>
<th></th>
<th>SVT/AF</th>
<th>Ventricular Ectopic</th>
<th>Ventricular Tachycardia</th>
<th>Ventricular Fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>2(15%)</td>
<td>1(7%)</td>
<td>1(7%)</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>4(10%)</td>
<td>1(2.7%)</td>
<td>1(2.7%)</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>6(12%)</td>
<td>1(2%)</td>
<td>2(4%)</td>
</tr>
</tbody>
</table>

Mean: 1.00, 1.25
SD: 0.82, 1.89

Conduction blocks

<table>
<thead>
<tr>
<th></th>
<th>First Degree AV Block</th>
<th>Second Degree AV Block</th>
<th>Complete Heart Block</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1(7%)</td>
<td>1(7%)</td>
</tr>
<tr>
<td></td>
<td>3(8%)</td>
<td>1(2.7%)</td>
<td>1(2%)</td>
</tr>
<tr>
<td></td>
<td>3(6%)</td>
<td>2(4%)</td>
<td>2(4%)</td>
</tr>
</tbody>
</table>

Mean: 0.67, 1.33
SD: 0.57, 1.52

Echo LV ejection fraction%

<table>
<thead>
<tr>
<th></th>
<th>&lt;45</th>
<th>50</th>
<th>&gt;55</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6(46%)</td>
<td>7(54%)</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>3(8%)</td>
<td>6(12%)</td>
</tr>
<tr>
<td></td>
<td>34(92%)</td>
<td>10(20%)</td>
<td>34(68%)</td>
</tr>
</tbody>
</table>

Mean: 4.33, 12.33
SD: 3.78, 18.82

Table 4: Clinical features at presentation and complications

DISCUSSION: Differentiating small and large inferior wall myocardial infarction without and with posterior wall myocardial infarction respectively is important both in acute management and in long term prognostication. The purpose of this study was to examine the value of 15 lead electrocardiograms in identifying the PW infarction in patients with IWMI. We also studied the clinical features of posterior wall infarction in patients with IWMI.

The echocardiographic features, particularly LV systolic function were estimated in patients with associated PWMI. Studies by Stewart et al have already confirmed a close relationship between electrocardiographic changes and more extensive PW infarction in terms of standard 12-lead ECG and the more extensive 15-lead ECG. Currently, routine performance of the 15-lead ECG is recommended to identify the posterior wall myocardial infarction easily.
INCIDENCE: Our study consisted of 50 patients of acute inferior wall myocardial infarction as proved by ECG, who were admitted to RMMCH. Additionally posterior leads were taken. In this series of 15-lead ECG taken from 50 IWMI patients, the overall incidence of ST elevation in the posterior chest leads (V7-V9) was 26%. 2 patients among the 50 had both RVI and PWMI associated with IWMI. 2 patients had inferolateral wall myocardial infarction with posterior wall myocardial infarction. In studies by Matzecky et al PWMI in patients with IWMI was around (27-53%).

RISK FACTORS: In Dittrich et al study the maximum incidence of MI was below 60 years (75.26%) and 24.74% in the patients aged above 60 years. Here the study was done in 820 patients of MI. In Senthil kumar et al study, the peak age of incidence was seen in age group of 40–54 years. Our study showed a peak incidence and percentage in the age group of 60 and above for IWMI without PWMI. The percentage was more in the age group of 50-59 years for the PWMI. So our report is similar when compared with these two groups with respect to age.

Kannel W.B. et al in 26 years follow up of a group of males and females aged between 35 – 84 years found the incidence to be 66% males and 34% in females. Our study shows 66% in males and 34% in females. This clearly indicates a male predominance and it might be due to higher associated risk factors in male.

Masaharu Ishihara et al showed an incidence of Cigarette Smoking to be present in 90% of patients with MI and Framingham Study shows 86%. Our study showed 24% of PWMI patients to be smokers and none of the females in this study group were smokers. Our study shows a lower incidence as it is done in a village locality and many of the Indian women are non-smokers. This has to be clarified in the futures studies in this region.

In Dittrich study, the incidence of diabetes was 17.5%. Our study shows an incidence of 34%, which shows higher incidence than that of Dittrich study. This shows that Indian population is more vulnerable to the diabetes mellitus and its complications. This was also more common in women than in the men according to our study. Usually these risk factors are not present singly. More than 2 risk factors are present for many cases.

CLINICAL FEATURES: Chest pain is the commonest symptom (90%) followed by sweating and palpitation which were (34%) and (30%) respectively. Palpitation was essentially an important presenting symptom in PWMI occurring in around 61% of the individuals. Syncope is the least presenting symptom in IWMI. In studies conducted by Senthil kumar et al, the incidence of chest pain was 98% followed by sweating 80% and palpitation 80%. The least presenting symptom is syncope which is 2% in their study. This shows that our study had similar findings when compared to Senthil kumar et al study.

COMPLICATIONS: Zahida fida et al reported an incidence of bradycardia in 29% patients of IWMI. Our study shows 12% incidence. Whereas bradycardia in IWMI without PWMI in our study is 15%. The little difference here we observe may be due to small study groups in both the studies.

Zahida fida et al reported hypotension in 22% and our study shown 24% in cases of IWMI. In PWMI the incidence is 46%. This shows that PWMI causes hemodynamic derangements. Our study supports the evidence of Zahida fida et al study.
Zahida fida et al studies have reported Heart Blocks in 11% of the cases where as our study showed 12%. In our study of complete heart block in PWMI was 7%. This underscores the importance of peculiar blood supply and hemodynamic instability of these cases.

MORTALITY: Out of 50 patients in the study group 7 persons died, 4 had inferior wall and other 3 had posterior wall infarction associated with IWMI. It is generally believed that female patients have poor prognosis but studies by Roberto Malacrida, et al reveal only subtle variation in mortality between the genders that too particularly during the early phases. Our study shows 42% mortality in women. In posterior wall MI the mortality in women is 66%.

The results of the present study highlight the utility of 15 lead ECG in identifying associated posterior wall infarctions. The study also identifies the common clinical manifestations and complications of the associated infarctions.

SUMMARY:

- Our study was based on 50 patients of IWMI proved by ECG admitted to RMMCH from September 2012-september 2014.
- Peak age incidence was in 60 and above year’s age group (46%).
- Out of 50 patients 33 were males indicating a male predominance.
- Diabetes was seen in 30% of PWMI with IWMI and 35% in patients IWMI without PWMI.
- Hypertension was present in 15% PWMI with IWMI and 29% in IWMI without PWMI.
- Smoking was a risk factor in 14% of the patients of IWMI and 42% in patients of PWMI with IWMI.
- History of alcoholism was present in 38% in patients of PWMI with IWMI and 6% in IWMI without PWMI.
- Chest pain was the presenting complaint in 90% of IWMI and in 76% of PWMI with IWMI alone.
- Syncope/giddiness was present in 18% in IWMI without PWMI where as it is 14% in overall IWMI. Palpitation was seen in 53% of PWMI with IWMI and 21% of IWMI without PWMI.
- Hypotension was present in 46% of PWMI with IWMI and 16% of IWMI without PWMI. Hence hypotension is an important physical finding in inferior wall myocardial infarction patients.
- Ventricular fibrillation was seen in 8% of PWMI with IWMI and 2.8% of patients of IWMI without PWMI, 4% of overall IWMI.
- Very frequently heart blocks were seen in inferior wall myocardial infarction (15% of PWMI with IWMI and in 10% of patients of IWMI without PWMI).
- Mortality was 23% in PWMI with IWMI and 10% in patients of IWMI without PWMI.

CONCLUSION: Acute inferior wall myocardial infarction is complicated by posterior wall infarction and is a well-known entity. Posterior wall infarction makes the hemodynamics of the patient unstable. Symptoms cannot exactly predict whether the inferior wall myocardial infarction is complicated by co existing posterior wall myocardial infarction or right ventricle myocardial infarction. This explains the importance of 15 lead ECG in these patients to diagnose the condition.

Early diagnosis means careful management for avoidance of complications like hypotension, Complete Heart Block etc., which are common in these patients, by using inotropes, volume loading and IV atropine and readiness for the thrombolysis and their complications. To conclude, by taking a
little extra effort and doing 15 leads while taking conventional leads, mortality in inferior wall myocardial infarction may be reduced by diagnosing PWMI early.

REFERENCES:
1. Matzesky S. Significance of ST segment elevation in posterior chest leads in patients with acute inferior myocardial infarction application for thrombolytic therapy. JACC. 1988 march 1; 31(506).
Fig. 2: Age incidence

Fig. 3: Sex incidence
Fig. 4: Incidence of risk factors

Fig. 5: Arrhythmias
### AUTHORS:

1. K. M. Bhargav

### PARTICULARS OF CONTRIBUTORS:

1. Post Graduate Student, Department of Medicine, Rajah Muthiah Medical College and Hospital, Anamali Nagar, Chidambaram, Cuddalore District, Tamilnadu.

### NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. K. M. Bhargav,
1-5-525,
Balaji Colony, Tirupati-517502,
Chittor District, Andhra Pradesh.
Email: bhargavkm2002@gmail.com

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