

Study of Right Ventricular Involvement in Acute Inferior Myocardial Infarction in Hospitalized Patients.

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Abstract:

A comparative cross sectional study was done in department of cardiology in Sylhet MAG Osmani Medical College Hospital during the period of July 2004 to June 2006 to compare in hospital (7 days) morbidity and mortality of inferior myocardial infarction (IMI) with or without right ventricular involvement (RVI). 70 patients were included in this study and divided into two groups. Group-I (IMI with RVI) includes 31 cases and group-II (IMI without RVI) includes 39 controls. ≥ 1 mm elevation of ST segment in V4R in ECG in the setting of acute IMI were taken as cases and only acute IMI were taken as controls. Each patient was treated with conventional treatment of myocardial infarction. Nitrates and diuretics were avoided in cases of RVI. Every patient was evaluated by clinical, ECG and echocardiographic examination.

RVI in acute IMI was 44.28%. Major risk factors are almost equally distributed between groups ($p>0.05$). History of Syncope was 12.9% in cases but none in controls ($p<0.05$). Heart failure and cardiogenic shock were observed to be considerably higher in cases (32.1% and 56.7%) than those in controls (13.5% and 12.8%) respectively ($p<0.05$ and $p<0.001$). Though statistically is not significant, recurrent angina or MI, arrhythmia and mortality were higher in RVI group. A significant proportion of cases (45.8%) tend to be associated with reduced right ventricular ejection fraction (RVEF) ($<50\%$) compared to none of their control counterpart ($p<0.001$). 95.8% of cases had wall motion abnormality in comparison to only 2.7% in controls ($p<0.001$). Patients with raised jugular venous pressure (JVP) had significantly worse in-hospital outcome in terms of arrhythmia, cardiogenic shock and mortality compared to IMI without raised JVP.

RVI in acute IMI has worse prognosis in terms of morbidity and mortality (25%). Therefore, RVI should be diagnosed accurately and early on the basis of ECG criteria. An early and prompt management is necessary for better outcome.

Introduction:

Coronary Artery Disease (CAD) is a worldwide health epidemic. It is estimated that 30 percent of all deaths can be attributed to cardiovascular disease¹. In Bangladesh, the prevalence of CAD was estimated as 3.3/1000 in 1976² and 13/1000 in 2004³ indicating four folds increase of the disease in last two decades.

IMI associated with right ventricular involvement defines a high-risk subset with a mortality rate of 25 to 30 percent, as opposed to an overall mortality of about 6 percent in IMI⁴. In our country the prevalence of RVI is increasing day by day. The increased morbidity and mortality of RVI may be due to I) Late arrival in hospital, II) Lack of awareness of physician about RVI, III) Lack of awareness about risk factors for heart disease, IV) Inadequate diagnosis, V) Inadequate treatment.

The initial description of the clinical syndrome of right ventricular failure in patients of RVI was given in 1974⁵. Since then RVI has recognized more frequently and continues to be a diagnostic and therapeutic challenge. Acute myocardial infarction (AMI) involving only the RV is a rare event. RVI in the setting of an acute IMI is much more common. Its presence defines a high-risk subgroup of patients with acute IMI who should be considered high-priority candidates for reperfusion. Ischemia or infarction of the right ventricle results in decreased RV compliance, reduce filling, and decreased RVEF. In turn, these changes lead to diminished left ventricular filling and drop in cardiac output (CO) that could result in systemic hypotension and shock. Frequent accompaniments may include atrial infarction, sinus bradycardia, atrial fibrillation, and atrioventricular block (A-V block). Hence the presence of RVI should raise a clinical alert for its potential immediate life-threatening consequences⁵.

The incidence of RVI is variable depending on the criteria used for detection^{5,6}. Autopsy studies⁵ suggest that right ventricular infarction accompanies fatal IMI in 24 to 34 percent of cases. Noninvasive studies^{7,8} suggest that RVI occurs in more than 30 percent of patients with acute inferior or inferior-posterior left ventricular MI. Anatomic evidence of RVI is more common than expected hemodynamic

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pattern⁷. RVI in acute IMI can be diagnosed clinically, electrocardiographically (ECG), echocardiographically, radionuclide studies and angiographically. The classic symptoms that are associated with inferior or posterior wall infarction: hypotension, raised JVP and clear lung fields are strong indicators of RVI⁹.

Right-sided ECG evaluation of all patients with IMI at the time of presentation is currently the gold standard in the diagnosis of RVI. ST elevation ≥ 1 mm in right chest lead V4R is 70% sensitive and nearly 100% specific for RVI¹⁰. Echocardiographic findings include - RV dilation, hypokinesia or akinesia of free wall of RV may be observed. The work carried out in this study was to look at the presentation and early outcomes of noninvasive management of RVI complicating IMI in the current era in the setting of a peripheral teaching hospital.

The incidence of RVI in such cases ranges from 10-50%, depending on the series¹¹. Another three studies abroad showed incidence of RVI were, 54%,^{4,12} 37%¹⁰. In Bangladesh it is found that RVI was 40%^{13,14,15}.

The principal cause of RVI is atherosclerotic proximal occlusion of the right coronary artery¹⁶. The etiology and pathogenesis are similar to that of left ventricular infarction.

Clinical recognition of acute RVI is extremely important, as it causes decreased RV compliance, reduced filling, and stroke volume followed by diminished LV filling and cardiac output^{17,18,19,20,21}. The triad of hypotension, elevated JVP, and clear lung fields has been recognized as marker of RVI in acute inferior-posterior wall MI^{22,23,24}.

Volume loading helps to maintain the CO and BP of the patient^{25,26}. Response to reperfusion depends on the duration of the preceding ischemia. Early reperfusion leads to prompt improvement and subsequent recovery of RV free wall contraction and global RV function without any scar formation.²⁵

After fluid loading, a trial of intravenous dobutamine is reasonable to increase contraction strength. Temporary AV pacing in symptomatic bradycardia may be needed.^{25,26} Fibrinolytic therapy ideally should be started within 30 minutes of presentation²⁷. Early reperfusion using thrombolytic therapy or primary percutaneous transluminal coronary angioplasty (PTCA) may reduce infarct size and improve the short-term survival in many of these patients²⁸. When IMI is complicated by RVI, however, the in-hospital mortality may be as high as 31 %, as compared with 6% for patients with inferior myocardial infarction^{4,26}. There are conflicting data, that right ventricular dysfunction after a myocardial infarction is an independent risk factor for higher long-term mortality^{26,29,30,31}.

Aims and objectives:

To see in hospital morbidity and mortality of patients of IMI with or without RVI. To record clinical presentation, syncope, dyspnoea and in hospital prognostic variables- raised JVP, heart failure, cardiogenic shock, arrhythmias, recurrent angina, cardiac biomarkers (CK-MB, Troponin-I), RWMA, LVEF, RVEF by echocardiography and death between patients of IMI with or without RVI.

Patients and methods:

This was a comparative cross sectional study. Sample was taken by using Cochran's formula. All IHD patients admitted within 12 hours of chest pain in CCU in Sylhet MAG Osmani Medical College hospital from 1st July 2004 – 30th June 2006 were included as study population. All patients were followed up for 7 days in CCU.

Inclusion criteria:

All IMI patients on the basis of WHO criteria, irrespective of age and sex within 12hrs of chest pain were included.

Exclusion criteria:

MI other than acute IMI, NSTEMI, UA, heart failure, valvular HD, history of cardiac intervention and cardiac surgery.

Selection of cases and controls:

IMI with RVI patients were taken as cases (n = 31) and only IMI as controls (n = 39).

Variables:

Heart rate, Blood pressure (BP), Jugular venous pressure (JVP), recurrent angina, heart failure, arrhythmia (Complete heart block, Ventricular tachycardia, Atrial tachycardia, Supra ventricular tachycardia-SVT, Atrial fibrillation, Ventricular fibrillation), cardiogenic shock, hospital stay (days) and mortality, Troponin-I and CK-MB, Fasting blood sugar, Lipid profile, ECG, EF, RWMA were investigated.

Ethical consideration:

The study was approved by ethical committee of Sylhet MAG Osmani Medical College.

Observations and results:

A total number of 70 patients were included in the study. They were divided into two groups. Group-I (IMI with RVI) includes 31 cases, group-II (IMI without RVI) includes 39 controls. The following results were obtained.

Clinical and haemodynamic presentation:

Table I compares the mode of clinical and haemodynamic presentation between groups. In the control group, approximately 64% had dyspnoea compared to 39% in the case group. In control group approximately 97% exhibited chest pain compared to 93% in the case. In contrast, about 13% of the cases had syncope as opposed to none of the controls. Majority of both cases and controls complained of chest pain. The mean basal systolic and diastolic blood pressures were found to be significantly dropped in case group compared to their control counterpart. More than half (51.6%) of the cases exhibited raised jugular venous pressure compared to none in the controls. However, the groups did not differ with respect to basal heart rate.

Table I. Comparison of clinical presentation between groups (n = 70)

Clinical presentation	Group		p-value
	Case (n=31)	Control (n=39)	
Dyspnoea	12	25	<0.05
Chest pain	29	38	NS
Syncope	4	00	<0.05
Heart rate	70.16	66.69	NS
Systolic BP	80.37	103.60	<0.01
Diastolic BP	52.0	69.49	<0.001
Raised JVP	16	00	<0.001

Distribution of risk factors:

Table II shows some difference in the distribution of risk factors between groups but these are not statistically significant

Table II. Distribution of risk factors between the groups (n = 70)

Risk factors	Group		p-value
	Case (n=31)	Control (n=39)	
Smoking	25	27	NS
HTN	7	12	
DM	5	7	
Dyslipidaemia	18	28	
Overweight, Obese	16	12	
Family history of IHD	2	6	

Distribution of cardiac markers:

Table III demonstrates that the mean serum troponin-I level was almost same in both cases and controls. The serum CK-MB, was found higher in case group compared to the control group, which is statistically significant

Table III. Comparison of cardiac-markers between groups (n = 70)

Cardiac markers	Group		p-value
	Case (n=31)	Control (n=39)	
Troponin-I (ng/ml)	32.67 (SD 28.29)	32.90 (SD 32.40)	NS
CK-MB (U/L)	88.65 (SD 54.65)	70.09 (SD 37.08)	<0.001

Ventricular ejection fraction:

No significant difference of LVEF between the groups within 72 hours of admission. On the contrary, over 45% of the cases exhibited reduced RVEF compared to none of the control group.

Clinical findings:

All the 5 in-hospital outcome variables are higher in IMI with RVI group. Heart failure and cardiogenic shock were observed to be significantly higher in case group (Table-IV).

Table IV. Comparison of in-hospital outcome between groups (n = 70)

In hospital outcome	Group		p-value
	Case (n=31)	Control (n=39)	
Recurrent angina/MI	8	6	NS
Heart failure (Killip class)	9	5	<0.05
Arrhythmias	10	11	NS
Cardiogenic shock	17	5	<0.05
Mortality	8	4	NS

Discussion:

The right coronary artery (RCA) provides the predominant blood flow to the right ventricle, supplying the lateral wall through the acute marginal branches; in the majority of patients, it also supplies the posterior wall and posterior inter ventricular septum through the posterior descending artery.

Identification of RVI in CCU was based on clinical findings and electrocardiographic changes. Clinically patients have the features of raised JVP, hypotension and clear lung bases. Right sided chest leads are useful for diagnosis. It is now established that ST elevation of ≥ 1 mm in V4R is highly specific as well as sensitive¹⁰. In our study ST elevation of ≥ 1 mm in V4R was observed in all cases.

The age, sex, BMI and risk factors of the sampled population were similar with the previous study^{32,10,33,5,34}. Necropsy series revealed evidence of RVMI were 14% to 60% in deaths from IMI^{32,10}. Clinically, RVMI complicates about 50% 10 of acute IMI on the basis of an ST-segment elevation of ≥ 0.1 mV in lead V4R. Bangladeshi showed RVI were 30% to 40%^{13,14,15} which is very much similar to our present study (44.28%).

Biomarkers was found higher in case group compared to the control group, which indicates more myonecrosis in RVI³⁵.

The classical triad of hypotension, raised JVP and clear lung fields occurred in majority of these patients n =16 (51.6%) of RVI. The diagnosis of RVMI can be made clinically if these features are present in patients with IMI. We followed the standard modality of treatment, most of

the patients responded well except eight who developed shock, arrhythmia and succumbed to death.

Over one-quarter of the cases and controls (27.3% of the cases and 25.7% of the controls) had complete heart block in our study which is almost similar to one previous study in Bangladesh¹⁴ but greater than several other studies.^{10,15,33}

Heart failure (Killip class I, II, III & IV) and cardiogenic shock were observed to be considerably higher in case group than those in control group ($p < 0.05$ and $p < 0.001$ respectively), which is higher than three previous studies,^{10,15,33} which might be due to lack of PCI and cardiac pacing.

Occurrence of HF, shock and CHB in IMI was associated with more mortality in RVI. RVMI significantly increases the risk of major complications and in-hospital death up to 31% compared to only about 6% in IMI per se^{1,4,31}. In present study the mortality were 25.8% and 10.3% respectively, with or without RVI in acute IMI, which is higher than previous Bangladeshi studies, as 13%^{13,15,36}. Other studies in abroad showed the mortality were 16%^{10,33}. The relatively higher mortality in our study possibly be due to delayed arrival in CCU, lack of atrio-ventricular sequential pacing and PCI.

Study Limitation:

The study was done on a small series and sample was not randomized. Lack of PCI and pacing facilities may complicate the outcomes. Follow up was not possible to see the long term outcomes.

Conclusion and Recommendation:

RVI in acute IMI has worse in hospital morbidity and mortality (25%). Clinical presentation, ECG and Echo are attributable to non invasive diagnosis of RVI. ST segment elevation in right side lead (V4R) have been found to have high predictive value for recognition of RV infarction in this series. Therefore, the study concludes that RVI can be diagnosed accurately on the basis of ST segment elevation of ≥ 1 mm in the lead V4R soon after the onset of infarction. And early and prompt management of RVI may yield better prognosis of these patients.

Further randomized case control study needed with larger sample in the centres where interventional facilities in the form of primary PCI are available and intermediate and long term follow-up are possible to compare impact of right ventricular involvement in IMI.

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