Right Ventricular Involvement in Inferior Myocardial Wall Infarction: Incidence, Clinical Spectrum and In-Hospital Outcome

A M AKBAR M A NADEEM T WASEEM F MUJIB A H KHAN

Department of Medicine, Mayo Hospital, Lahore.
Correspondence to Dr. Muhammad Arif Nadeem

Abstract

Right ventricular infarction (RVI) complicating inferior wall myocardial infarction (MI) is common, associated with morbidity and mortality, and requires thrombolytic therapy. Electrocardiogram (ECG) through right precordial leads (V_3R to V_6R)) is a useful and convenient tool of diagnosing RVI. We studied 50 (38 males, 12 females) patients with acute inferior wall MI to evaluate the clinical course and in-hospital outcome with regard to the presence or absence of RVI as diagnosed by ST-segment elevation in leads V_3 to V_6R on ECG, which was repeated daily to follow for the reversion of ST-segment elevation. Continuous monitoring was done for 72 hours. All patients were managed with standard treatment protocol. In-hospital outcome of all patients was observed for any dysrrhythmia or cardiogenic shock. The mean age was 56 $\pm$ 11 years. Thirty-five (70%) patients were without RVI and 15 (30%) had it with no difference in demographic profile and previous history of ischemic heart disease (IHD). Fourteen percent patients died of inferior wall MI, 5.7% in group I and 33.3% in group II. In-hospital complications were more in RVI i.e. cardiogenic shock ($P<0.004$), complete atrioventricular (AV) block ($P<0.005$) and bradycardia ($P< 0.04$) but there was no difference for other dysrrhythmias. In-hospital mortality may be high upto 31% with RVI complicating inferior wall MI, we observed it as 33.3%. Five diagnostic procedures like autopsy, coronary angiography, echocardiography, haemodynamic measurements, echocardiography and ECG have shown that ST-segment elevation in lead V_4R had an overall sensitivity of 88%, 78% specificity and 83% accuracy in diagnosing RVI. We observed 30% patients having RVI with inferior wall MI as compared to 19-61% in other studies. We conclude that right ventricular infarction can be diagnosed reliably on the basis of ST-segment elevation of $\geq$01mm in right precardial leads V_3R to V_6R in patients with inferior wall MI to stratify further risks and their treatment.

Key Words: Right ventricular infarction, Inferior wall MI.

Acute Myocardial Infarction (AMI) is a leading cause of morbidity and mortality.[1] The urban South Asian populations are at an increased risk of premature coronary artery disease in younger population as compared to Western Countries.[2,3] Important changes have taken place in the management protocol of Acute MI during last decade e.g. rapid restoration of blood flow to the ischemic myocardium by the use of thrombolytic agents. The importance and urgency of reperfusion increases in proportion to the amount of myocardium at risk.

In acute anterior wall MI, the amount of ischemic myocardium corresponds to the number of chest leads showing QRS and ST changes and to the magnitude of ST elevation in these leads.[4] Right Bundle branch Block is another indicator of infarct size in acute anterior wall MI denoting occlusion of left anterior descending artery proximal to its first septal branch.[5]

Acute inferior wall MI is usually considered to have a more favorable prognosis than anterior wall infarction but this is not always true. Involvement of the right ventricle in inferior wall infarction is common and has important prognostic implications warranting use of thrombolytics.[6] Right ventricular infarction is associated with considerable morbidity and mortality. Detection of this condition requires high degree of suspicion in appropriate clinical setting. Right ventricular infarction should be suspected in all patients with inferior wall infarction, especially those who are hypotensive, have raised jugular venous pressure and clear lung fields on auscultation. Electrocardiogram (ECG) through right precordial leads is a useful and yet convenient tool for diagnosing right ventricular infarction (RVI).[7]

The proper management of these patients requires sustaining adequate right ventricular preload, reduction of right ventricular afterload and inotropic support when and where indicated. Early reperfusion with thrombolytic agents or direct angioplasty has also been shown to be beneficial. After initial hospitalization period, RVI does not have additional prognostic import.

Right ventricular infarction was first described about 65 years ago[8] but it was recognized as a distinct clinical entity only two decades ago.[9] Now it is recommended to record right precordial leads in all patients with inferior wall infarction.

Purpose of Study

The presence of acute ST-segment elevation in right precordial leads V_3R to V_6R is considered to be highly reliable in diagnosing right ventricular infarction when compared with the gold standard of hemodynamic measurement or autopsy. Patients with acute inferior wall myocardial infarction will be studied to evaluate the clinical course and in-hospital outcome with regard to the presence or absence of right ventricular infarction as diagnosed by ST-segment elevation in the right precordial leads V_3R to V_6R.

Inclusion Criteria

1. Adult patients of either sex.

2. Chest pain or any angina equivalent symptom (dyspnea, profuse sweating with apprehension or sinking of heart) lasting for more than 30 minutes.
3. Acute inferior wall myocardial infarction on ECG (ST segment elevation ≥0Imm in at least 2 of leads II, III and aVF.
4. Increase in the scrum Creatine Kinase (CK) level to more than twice the normal value with raised MB fraction measured within 24 hours of admission.

Exclusion Criteria

Those patients were not included who were admitted 24 hours after the onset of symptoms, because right precordial electrocardiography loses its sensitivity by that time.[9]

Materials and Method

We studied 50 consecutive patients with acute inferior wall myocardial infarction. Every patient was considered for thrombolytic therapy as it results in rapid and significant recovery of right ventricular performance. All convention therapeutic measures were employed. All patients were specifically examined to assess the presence or absence of clinical signs of right ventricular infarction including jugular venous distension above the level of the clavicle with patient sitting at 45°, clear lung fields on auscultation, arterial hypotension, S4 gallop and Kusmaul's sign. CK and its MB fraction were determined in every patient within 24 hours after admission.

An ECG including right ventricular precordial leads V_3R to V_6R were systematically recorded. Right ventricular involvement was diagnosed when an ST-segment elevation of ≥0Imm in leads V_3R to V_6R was present. Right precordial electrocardiogram was repeated daily in patients having ST-segment elevation in V_3R to V_6R until reversion of this elevation to baseline and in the absence of reversion until the patient was discharged.

Continuous cardiac monitoring was done in every patient for at least 72 hours in the absence of any rhythm disturbance. In the presence of any rhythm abnormality cardiac monitoring was continued for as long as the patient stayed in the hospital. All patients were managed with standard treatment strategies and kept under observation in hospital for at least 7 days.

In-hospital outcome was studied for all patients in terms of mortality and major in-hospital complications including ventricular fibrillation, sustained ventricular tachycardia (lasting for more then 30 seconds or producing hemodynamic intolerance), cardiogenic shock, second degree (Mobitz II) and third degree atrioventricular block. All complications were analyzed according to whether they occurred less then 24 hours after admission or at any time during the hospitalization.

Two groups were made out of these 50 patients:

Group I (RV -ve): Patients having inferior wall myocardial infarction without any ECG evidence of right ventricular infarction.

Group II (RV +ve): Patients having inferior myocardial infarction with ECG evidence of right ventricular infarction.

Patients in each group were further characterized with regard to age, sex, initial blood pressure, history of myocardial infarction, use of thrombolytic therapy, coronary risk factors and in case of right ventricular infarction a lack of response to volume loading as well as a requirement for intravenous catecholamines for hemodynamic support.

Results

There were 50 patients (38 males, 12 females) with inferior wall myocardial infarction (table 1). The mean age was 56 ± 11 years.

Table No. 1: Clinical characteristics of 50 patients with acute inferior wall myocardial infarctions.

Sex M/F	38/12 76%/ 24%
Age	
Mean ±sd	56±11
Range	36-80
History of previous MI	4(8%)
Thrombolytic therapy	24(48%)
Hours since onset of symptoms	9 (median)
Presenting complaints	
Typical chest pain	46 (92%)
Atypical presentation	04 (08%)

There were 35 (70%) patients without right ventricular involvement (Group I RV -ve) and 15 (30%) patients with right ventricular involvement (Group II RV +ve). There were no significant differences between the two groups with regard to age, sex, atherosclerotic risk factors, duration of symptoms, history of previous MI and percentage of patients who received thrombolytic therapy (table 2).

Table 2: Clinical characteristics of patients in group I (RV -ve) and group II (RV +ve)

	GROUP I (RV -ve) n=35	GROUP II (RV +ve) n=15	SS =
AGE (Years)	54±10.6	61 ± 10.4	NS
SEX M/F	27/8	11/4	NS
DURATION of SYMPTOMS (hrs)	10	8	NS
SHOCK at PRESENTATION	2 (5.7%)	5(33.3%)	P<0.02
HISTORY of MI	3(8.5%)	1(6.6^)	P<0.43
THROMBOLYTIC THERAPY	16 (45.7%)	9 (53.3%)	P<0.21

MI = Myocardial Infarction

NS = Not Significant

SS = Statistical significance was calculated by using Fisher exact probability test.

The overall in-hospital mortality in patients with inferior wall myocardial infarction was 14%. However it was 5.7% in patients without right ventricular involvement, while in patients with right ventricular involvement it was

33.3% (P<0.02). Prevalence of in-hospital complications was significantly higher in group 2 (RV +ve) as compared to the patients in group 1 (RV -ve). These complications included cardiogenic shock (P<0.004), complete AV block (P<0.005), bradycardia (P<0.04). There was no significant difference between two groups as regards the prevalence of ventricular fibrillation, sustained ventricular tachycardia and second degree AV block. It was noticed that presence or absence of shock at the time of presentation had a predictive value for RVI or no RVI respectively: sensitivity 33%, specificity 94%, positive predictive value 71%, negative predictive value 77% and diagnostic accuracy 76% (table 3).

Table No.3: Shock at Presentation – Predictive nd Diagnostic Value for RV in Inferior Wall Mi.

	Shock Present	Shock Absent
RV +ve (n=15)	5	10
Rv -ve (n=35)	2	33

Sensitivity = 33% Specificity = 94%
+ve Predictive value = 71% -ve Predictive value = 77%.
Diagnostic accuracy = 76 %

Mortality within first 24 hours was significantly higher in group 2, as compared to group 1 (P <0.02). After first 24 hours, there was no significant difference of mortality between two groups. Moreover, patients in group 2 had higher requirement for volume loading (P< 0.002) and inotropic support (P< 0.01) as compared to those in group 1 (table 4).

Table No. 4 in-hospital course of patients in group I (RV -ve) and Group II (RV +ve)

	Group I (RV-ve) n= 35 (70%)	Group II (RV +ve) n= 15 (30%)	SS=
DEATHS	2(5.7%)	5(33.3%)	P < 0.02
SHOCK	3 (8.6%)	7 (46.7%)	P <0.004
VF	1(2.8%)	1(6.7%)	P < 0.42
SUSTAINED VT	1(2.8%)	2(13.3%)	P < 0.19
COMPLETE AV BLOCK	2 (5.7%)	6(40%)	P < 0.005
2^0 TYPE II AV BLOCK	1(2.8%)	1(6.7%)	P < 0.42
2^0 TYPE I AV BLOCK	2(5.7%)	3(20%)	P > 0.13
BRADYCARDIA	5(14.3%)	6(40%)	P < 0.04
VOLUME LOADING	2(5.7%)	7 (46.7%)	P < 0.002
REQUIREMENT for INOTROPES	6(17.1%)	8 (53.3%)	P < 0.01

Some patients had more than one major complication
VF= Ventricular Fibrillation
VT= Ventricular Tachycardia
AV= Atrioventricular
SS= Statistical significance was calculated by using Fisher exact probability test.

Discussion

Inferior wall myocardial infarction is usually considered to have a better both short and long term prognosis than anterior wall myocardial infarction. However it is associated with higher mortality and morbidity when complicated by right ventricular infarction. In-hospital mortality may be as high as 31% compared to 6% with inferior wall myocardial infarction without right ventricular involvement.

The diagnosis of right ventricular infarction can be made from physical examination, Doppler and 2D echocardiography, scintigraphy, right ventricular angiography and hemodynamic measurement but right precordial electrocardiography is the most readily available, simplest and most objective technique.
ST-segment elevations and Q waves in the right precordial leads (V_3R to V_6R) have previously been shown to have a diagnostic accuracy for right ventricular infarction greater than 80%.[7] Five diagnostic procedures like autopsy, coronary angiography, echocardiography, hemodynamic measurements and ECG have shown that the ST-segment elevation in lead V_4R had an overall sensitivity of 88%, a specificity of 78% and a diagnostic accuracy of 83% for the diagnosis of right ventricular involvement during acute inferior wall myocardial infarction (table 9).

In our study 30% of the patients with inferior wall myocardial infarction also had right ventricular involvement. Many studies have shown that right ventricular infarction complicates 19 to 51% of inferior wall myocardial infarction. In a recent study done by Cohen and co-workers, almost 61% of the patients with acute inferior myocardial infarction have right ventricular involvement, as determined by coronary angiography.[9]. In another study carried by Zehender et al. patients with inferior wall MI, more than half (54%) had right ventricular involvement as diagnosed by ST segment elevation in lead V_4R, the in-hospital mortality after inferior myocardial infarction was 19% and major complications occurred in 47% of patients.[10] While patients with ST-segment elevation in lead V_4R had a higher in-hospital mortality 31% Vs 6% (P>.001), and a higher incidence of major complications 64% Vs 28% (P<0.001), as compared to patients without ST-segment elevation in lead V_4R.

Our study showed that patients with acute inferior wall MI had an in-hospital mortality of 14%. Right ventricular involvement as diagnosed by ST-segment elevation of $\geq$0I mm in V_3R to V_6R, was shown to be an independent prognostic indicator of the in-hospital course. The presence of right ventricular infarction had increased in-hospital mortality from 5.7% to 33.3% (P<0.02) and significant difference of mortality was observed during first 24 hours after admission as compared with the remaining period of hospital stay.
The use of ST-segment elevation in lead V_3R to V_6R for the diagnosis of right ventricular infarction is known to lose specificity in the presence of any heart disease that may induce ST-segment elevation in lead V1 such as pericardial disease, acute pulmonary embolism, left anterior fascicular block and acute anterior wall MI (present in 10% of patients with right ventricular

involvement).[12] The diagnostic accuracy of right precordial ST-segment elevation is considered to be greatest during the first 10 hours after an acute infarction, which emphasizes the need for an electrocardiographic assessment as soon as possible after admission to the hospital.[10]

Conclusion

Right ventricular infarction can be diagnosed reliably on the basis of an ST-segment elevation of ≥01mm in the right precordial leads V_3R to V_6R, soon after the onset of infarction, and this elevation indicates a significantly increased risk of major complications and in-hospital death. Recording lead V_3R to V_6R helps to stratify patients with acute inferior wall myocardial infarction into high and low risk groups, and is essential for making timely and correct therapeutic decisions in this potentially high risk group of patients.

References

1) Pasternak RC, Braunwald E, Sobel BE. Acute myocardial infarction. In: HBJ International edition. Braunwald Heart disease: A text book of cardiovascular medicine. WB Saunders 1992, 4th edition: 1200-91.
2) Virk Z, Khan AA, Bokhari AM. Ischemic heart disease in the young population (35 years): A clinical profile. PJC 1995; 6: 63-66.
3) Yousuf M, Alsandi, D Abdul Aziz. Pattern of cardiovascular problems among Haj Pilgrims. Specialist 1996, 12(2): 127-129.
4) Bar FW, Vermeer F, De Zwaan C, et al: Value of admission electrocardiogram in predicting out come of thrombolytic therapy in acute myocardial infarction: a randomized trial conducted by Netherlands inter-university cardiology institute. Am J Cardiol 1987; 59:6-13.
5) Lie KI, Wellens HH, Schuilenburg RM, Becker AE, Durrer D. Factors influencing prognosis of bundle branch block complicating acute antero-septal infarction. Circulation 1974; 50; 935-41.
6) Wellens HJJ. Right ventricular infarction (Editorial). N Eng J Med 1993; 328: 1036-1038.
7) Andres P. Michaelides, Zoi D. Psomadaki, Polychronis E. Dilaveris, Dimitris J. Richter, George K. Andrikopoulos, Konstanaina D. Aggeli, et al. Improved detection of Coronary Artery Disease by Exercise Electrocardiography with the use of right precordial leads. N. Engl J Med 1999;340:340-5.
8) Kinch JW, Ryan TJ. Right ventricular infarction. N Engl J Med 1994; 330: 1211-7.
9) Cohn JN, Guiha NH, Broder MI, Limas CJ. Right ventricular infarction: clinical and hemodynamic features. Am J Cardiol 1974; 33:209-14.
10) Zehender M, Kasper W, Kauder E et al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. N Eng J Med 1993; 328: 981-8.
11) Cohen A, Guyon P, Chauvel C et al. Relations between Doppler tracings of pulmonary regurgitation and invasive hemodynamics in acute right ventricular inarction complicating inferior wall left ventricular infarction. Am J Cardiol 1995; 75: 425-30.
12) Coma-Canella I, Lopez-Sendon J. Ventricular compliance in ischemic right ventricular dysfunction. Am J Cardiol 1980; 45:555-61.