In Hospital Complications and Mortality of Patients of Inferior Wall Myocardial Infarction with Right Ventricular Infarction

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ABSTRACT

This is a cross-sectional study of patients with inferior wall Myocardial Infarction (MI), who attended emergency and got admitted in ICU/CCU of TU Teaching Hospital, Maharajgunj and Bir Hospital, Kathmandu, Nepal during November 1999 to October 2000. This study was undertaken to compare the in-hospital complications and mortality of patients of inferior wall myocardial infarction with or without associated right ventricular infarction.

Total 53 consecutive patients with acute inferior wall myocardial infarction were enrolled in the study. Right ventricular infarction was determined by the presence of ST elevation of more than 0.1 mv in V_4R. All the patients of inferior wall myocardial infarction were divided into two groups. Group A consisted of patients of inferior wall MI with right ventricular infarction and group B consisted of patients of inferior wall MI without right ventricular infarction. In-hospital complications and mortality of group A were compared with group B.

Among 20 patients of group A and 33 patients of group B, incidence of cardiogenic shock was significantly higher in patients of group A compared to group B ($p=0.05$). Ten patients of group A developed third degree AV Block compared to only one in group B; the incidence of which was significantly higher ($p<0.001$). Sinus nodal dysfunction, manifested by junctional rhythm was found in six patients of group A compared to only two patients of group B ($p<0.05$). Mortality was found higher in patients of group A, but it was not statistically significant. Two patients of group A expired on first day whereas only one patient of group B expired on the eighth day of admission.

In hospital complications especially cardiogenic shock, complete A-V block and junctional rhythm are significantly higher in inferior wall MI when associated with RV infarction.

Key words : *Antrioventiricular block, Cardiogenic shock, Inferior Wall Myocardiar Infarction, Right Ventricular Infarction*

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INTRODUCTION

Right Ventricular (RV) Infarction complicates 30-50% of Inferior Wall Myocardial Infarction (MI). Most of the RV myocardial infarction result from occlusion of the right coronary artery. RV Infarction may lead to decreased RV compliance, reduced filling and diminished right sided stroke volume with concomitant RV dilatation and septal changes.[1] These changes can result in reduced left ventricular filling and contractile function with a concomitant fall in cardiac output. The net effect is low left sided filling pressure despite clinical signs of high pressure in the right side.[2] RV infarction should be suspected in any patient with an acute inferior wall myocardial infarction with evidence of distended neck veins, hypotension and clear lungs.[3] A considerable number of patients with RV infarction develop severe right sided failure and cardiogenic shock.[2,3,4] Creamer et al have concluded in their study that probable mechanism of the shock associated with RV infarction is concomitant severe Left Vetricular (LV) dysfunction.[5] Gold Stein et al concluded in his study that RV infarction occurs in 50% of patients with acute inferior wall MI and may result in severe haemodynamic compromise associated with poor clinical outcome.[4] Patients with RV infarction have higher incidence of cardiogenic shock, complete heart block, right ventricular free wall rupture, cardiac tamponade, pulmonary embolism, atrial and supraventricular tachycardia and atrial fibrillation.[6,7,8,9] We selected this study to know the common complications that we come across in our set up and compare the mortality between the two groups.

MATERIALS AND METHODS

This is a cross sectional descriptive study done during November 1999 to December 2000. Total 53 patients with confirmed acute inferior wall myocardial infarction who attended emergency and got admitted in Intensive Care Unit (ICU)/Coronary Care Unit (CCU) of TU Teaching Hospital, Maharajgunj and Bir Hospital were enrolled in the study. Right Ventricular Infarction was suspected by suggestive history, clinical findings and was confirmed by electrocardiogram (ECG) and echocardiogram. Patients with history of chest pain and/or epigastric pain with or without radiation, nausea, vomiting, diaphoresis, dizziness and syncope with increased jugular venous pressure, kussmaul's sign, hypotension, right ventricular gallop (S_3 or S_4), clear lungs, hepatomegaly, peripheral oedema were given high suspicion for RV infarction. RV infarction was confirmed by ST elevation of more than1 mv in V_4R with normal chest X-ray and typical rise of cardiac enzyme. A Standard 12 lead electrocardiogram with four right sided precordial leads V_3R –V_4R was taken on admission and routinely at 24, 48, and 72 hours after onset of chest pain. In addition a 12 lead ECG was done routinely after clinical stability before discharge from the hospital. The pre-discharge ECG was analysed for evidence of true posterior infarction. All patients of inferior wall MI were divided into two groups. Group – A consisted of patients of inferior wall MI with confirmed right ventricular infarction and Group – B consisted of patients of inferior wall MI without right ventricular infarction. All patients were kept on continuous ECG monitoring at least for 72 hours to detect rhythm and conduction disturbances. Diseases mimicking RV Infarction like, cardiac tamponade, constrictive pericarditis, acute pulmonary embolism, restrictive cardiomyopathy, patients with previous MI, associated anterior wall MI, chronic lung disease, pulmonary hypertension, pericardial effusion, left or right ventricular hypertrophy, bundle branch block, inter ventricular conduction defects or fascicular block were excluded from the study. All patients were admitted for eight days and meticulous care was given to judge presentation and complications developed in the whole course. Shock was defined as a state of severe failure of tissue perfusion characterized by systolic blood pressure of less than 90 mm of Hg , a low cardiac output and signs of poor tissue perfusion such as oliguria, cold extremities and poor cerebral function.Patients having other causes of shock like sepsis, hypovolaemia were not included in the study.

STATISTICAL METHOD

Data were expressed as mean standard deviation (SD). Discrete variables were analysed by using x^2 test (Yates corrected); for cases where expected cell size was less than five, Fisher's exact text was used. Comparisons of continuous variables were made by using student's t-test. $P \leq 0.5$ was taken as statistically significant.

RESULTS

We compared the incidence of various complications between the two groups. Eight patients of group A

Table 1. Comparison of complications and mortality in patients of inferior wall MI with and without RV infarction.

S.N.	Complications	Group A (20)	Group B (33)	P-Value
1.	Cardiogenic Shock	8 (40%)	4 (12%)	0.05
2.	Sinus Bradycardia	6 (30%)	9(27.27%)	(NS)
3.	First Degree A-V Block	8 (40%)	10 (30%)	(NS)
4.	Second Degree A-V Block	6 (30%)	6(18.18%)	(NS)
5.	Complete Heart Block	10(50%)	1 (5%)	<0.001
6.	Sino-Atrial Block	1 (5%)	1 (3%)	(NS)
7.	Sinus Tachycardia	7 (35.1%)	12 (36%)	(NS)
8.	Junctional Rhythm	6 (20%)	2 (6%)	<0.05
9.	Atrial Fibrillation	2 (10%)	2 (6.06%)	(NS)
10.	Atrial Premature Contraction	1 (5%)	1 (3.03%)	(NS)
11.	Premature Ventricular Contraction	3 (15%)	6 (18%)	(NS)
12.	Ventricular Tachycardia	2 (10%)	2 (6%)	(NS)
13.	Ventricular Fibrillation	2 (10%)	1 (3%)	(NS)
14.	Mortality	2 (10%)	1 (3.03%)	(NS)

NS = Not significant

developed cardiogenic shock compared to only four in group B; the incidence of which was significantly higher (P≤0.05). There was no significant difference in the incidence of sino-atrial block, first and second degree AV block between the two groups. Ten patients of group A developed third degree AV block, which was significantly higher compared to patients of group B (P≤0.001). Sinus nodal dysfunction manifested by junctional rhythm developed in six patients of group A compared to only two in group B (P≤0.05). The incidence of sinus bradycardia, sinus tachycardia, atrial fibrillation, atrial premature contraction, premature ventricular contraction, ventricular tachycardia, ventricular fibrillation was not significantly different between the two groups. There was no evidence of pericarditis, pericardial effusion, mural thrombus, pulmonary embolism and rupture of ventricular septum in any of our patients. The mortality between the two groups was not statistically significant. Two patients of group A died on the first day. One had complete heart block as well and both died of cardiogenic shock. Only one patient of group B expired on the eighth day.

DISSCUSSION

Incidence of RV infarction ranges from 10-50% depending on the series.[10] The potential haemodynamic derangement associated with right ventricular infarction render the afflicted patient unusually sensitive to diminished pre-load (i.e. volume) and loss of atrio-ventricular synchrony. These two circumstances can result in a severe decrease in right and, secondarily, left ventricular output.[11] A subtle clue to the presence of haemodynamically significant RV infarction is a marked sensitivity to preload reducing agents such as nitrates, morphine and diuretics.[12] Patients with haemodynamically significant RV infarction may develop hypotension, jugular vein distension and occasionally shock, all in presence of clear lung fields.[2] A high incidence of hypotension and cardiogenic shock has been documented in RV infarction.[1,2,3]

In our study eight patients of group A presented with cardiogenic shock compared to only four in group B (P=0.05). Patients with RV Infarction associated with inferior wall myocardial infarction have much higher rates of significant hypotension, bradycardia requiring pacing support and in-hospital mortality than isolated inferior wall infarction.[13] The conduction disturbances in the acute inferior wall myocardial infarction are usually located at the AV node.[14,15] So all forms of AV block occurs 2-4 times more frequently in acute inferior wall infarction as compared to anterior wall MI.[16] This is explained by the fact that 90% of the blood supply of the AV node is by the right coronary artery. Incidence of high degree AV block has been found to be between 33-66% of inferior wall MI with right ventricular infarction.[17] In our study we found 50% incidence of complete heart block, which is statistically significant. This result seems to be high but considering the mode of blood supply to the AV node it is not as high as can be expected. Further most of the RV infarction results from occlusion of the right coronary artery proximal to

the acute marginal branch, which is above the origin of the AV nodal artery. An explanation could therefore be that dual or collateral blood supply to the AV node is functionally common. Zehender et al concluded in their study that when inferior wall MI is complicated by RV infarction, the in-hospital mortality may be as high as 31% as compared with 6% for patients without RV involvement.[18] Our study showed mortality of 10% in group A as compared to 3.03% in group B. Among the two deaths in patients with RV infarction, one had bilateral crackles in the chest, which is unusual in dominant RV infarction indicating more extensive damage of left ventricle. So prognosis of RV infarction could be related to the extent of LV damage.Low incidence of other complications in our study could be because of small number of patients enrolled in the study. Treatment of RV infarction with haemodynamic compromise is aimed at increasing preload by fluid resuscitation to Central Venous Pressure (CVP) 16-20, avoidance of nitrates, diuretics, morphine; maintaining atrio-ventricular synchrony by AV sequential pacing for complete heart block, prompt cardioversion for atrial fibrillation, judicious use of ionotropic agents and reducing right ventricular afterload by intra aortic balloon counter pulsation, vasodilators (sodium nitroprusside), reperfusion by thrmobolytic agents or direct angioplasty.[19]

CONCLUSION

Because of the pathophysiology of right ventricular infarction, its management differs substantially from the routine management of left ventricular infarction. So early and accurate diagnosis of RV infarction is imperative. Cardiogenic shock, complete heart block and junctional rhythm are the most common complications found in our study.

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