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## Clinical Profile of Right Ventricular Myocardial infarction in inferior Wall Myocardial Infarction

Authors

### Dr Rajiv Girdhar<sup>1</sup>, Dr Mahesh Manish<sup>2</sup>, Dr Sudir Vigneshwar K.N.<sup>3</sup>, Dr Kruthika Sridharan<sup>4</sup> Dr Subhashree Jana<sup>5</sup>

<sup>1</sup>Assistant Professor in Cardiology Department <sup>2,3,5</sup>Intern in RRMCH <sup>4</sup>Post Graduate in Medicine Department

#### Introduction

Coronary artery disease is the commonest form of heart disease and the leading cause of morbidity and mortality throughout the world. Its prevalence among Indians has doubled during the past two decades<sup>1</sup>. Myocardial infarction is one of the most common diagnosis in hospitalized patients. Acute myocardial infarction is the single most important cause of morbidity and mortality in developed countries<sup>2</sup>. In developing countries, it follows infections. Now it is recognized as one of the major non-communicable public health problem. There is increased incidence of acute myocardial infarction in developing countries because of multiple factors like unhealthy food habits, stress factors, increase in habits like smoking and alcohol and rapid urbanization<sup>3,4</sup>. There is an advent of newer diagnostic techniques for the disease, but still ECG remains the pillar as it is non-invasive and easily available. Now right ventricular MI is diagnosed using right sided precordial leads (RPL) with introduction of RPL diagnosis of RVI has become easy and economical. RVMI is not uncommon in acute MI

and has its own therapeutic and prognostic implications. Management of RVMI differs from other MIs. The presence of RVI is known to increase the chances of cardiogenic shock, arrhythmias and conduction blocks<sup>1</sup>.

### Objectives

- 1. To study the frequency and clinical profile of right ventricular MI in patient with inferior wall MI.
- 2. To study prognosis in a patient admitted with right ventricular MI in patient among inferior wall MI.

#### Methodology

This study is based on analysis of 60 consecutive patients of Inferior wall myocardial infarction as proved by E.C.G. admitted from Aug 2017 to Aug 2019 to the ICCU of to Rajarajeswari Medical College and Hospital, Bengaluru. All the Patients were studied at the time of admission, during management in hospital and followed up in the hospital until recovery or death. Criteria Only patients with definite evidence of IMI in 12 lead

standard ECG were included in this study. For these patient's additional Right Precordial leads were taken at the time of admission and repeated at 12 hours, 24 hours and 48 hours. A detailed case history was taken and a detailed physical examination was done at the time of admission. For recording ECG 12 lead ECG (3 standard leads, 3 augmented limb leads, 6 precordial leads) machine was used. The recording was made at 25 mm/sec. Speed and 1 mv = 10 mm. Right precordial leads were applied on the areas of chest which the leads corresponded on the left. Criteria for diagnosing RVI ST elevation in II, III, avF, V1 and ST elevation in all are any one of the right procordial leads i.e. RV3, RV4, RV5, RV6 and associated mirror changes in the anterior leads. As Echo Cardiography and Coronary Angiography was not performed on all the patients in this study, so the reports of these investigations was not considered for the diagnosis of RVI. Inclusion Criteria All the patients with definite evidence of acute inferior wall myocardial infarction as proved• by 12 lead ECG along with right ventricular pericardial leads RV3, RV4, RV5, RV6 and associated mirror changes in the anterior leads. Exclusion Criteria \* ECG evidence of LBBB \* History of previous MI \* Cor pulmonale \* Suspected pulmonary embolism \* Associated pericardial disease. 44 \* Patients with chest pain of more than 24-hour duration, as ST elevation in RPLs is transient Emphasis was given to the examination of Jugular venous pulse, Kussumauls sign, blood pressure, S3 and S4 and systolic murmur of Tricuspid regurgitation. Continuous ECG monitoring was done to detect arrhythmias and conduction defects. Routine investigations like Random Blood Sugar, Urea, Creatinine, total Cholesterol and in most of the Creatinine phosphokinase, lactate cases dehydrogenase and SGOT were estimated. As Echo and Angiogram were done in very few patients, there were not considered for this study. of given. Routine treatment AMI was Complications were identified and treated accordingly.

### Results

Out of the total 170 cases of acute MI admitted in Rajarajeshwari medical college and hospital.

**Table 1:** Incidence in all groups

	Total No. of All AMI	No. of IMI among AMI	Percentage
Incidence in all groups (n)	170	50	29.4%

The incidence of IMI among all the cases of AMI was 29.4%

#### Table 2: Incidence of RVI in IMI

	Total No. of IMI	No. of RVI in IMI	Percentage
Incidence of			
RVI in IMI (n)	50	15	30%

In our study group of IMI RVI incidence was 30 %. So the incidence of RVI in all cases of AMI was 10 %.

	RRMCH
Cases of all Acute MI from	170
Aug2017 to Aug2019	

#### Table 3: Age Incidence

Age in years	IMI without	RVI	Total
	<b>RVI</b> $(n = 35)$	(n = 15)	(n = 50)
21 – 30	2 (5.7%)		2 (4%)
31 – 40	4 (11.4%)	1 (6.6%)	5 (10%)
41 – 50	8 (22.8%)	4 (26.6%)	12 (24%)
51 - 60	10 (28.5%)	8 (53.3%)	18 (36%)
61 and above	11 (31.4%)	2 (13.3%)	13 (26%)

Our study showed a peak incidence of RVI in the age group of 51 - 60 years but the peak incidence of IMI was in the age group of 61 years above.

### Table 4: Sex Incidence

Sex	Total Incidence in IMI = n	IMI without RVI = n	RVI = n
Male	43 (86%)	31 (88.5%)	12 (80%)
Female	7 (14%)	4 (11.5%)	3 (20%)

Our study showed a very high incidence of IMI and as well as RVI in males compared to females. This may be due to association of many risk factors which is more common in males.

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Table 5. Inclucite of Risk I actors					
	IMI without	RVI	Total		
Risk Factors	<b>RVI</b> $(n = 35)$	(n = 15)	(n = 50)		
Diabetes	9 (25.7%)	2 (13.3%)	11 (22%)		
Hypertension	17 (48.5%)	6 (40%)	23 (46%)		
Smoking	28 (80%)	11 (73.3%)	39 (78%)		
Family History	14 (40%)	7 (46.6%)	21 (42%)		
Alcohol	7 (20%)	5 (33.3%)	12 (24%)		

### **Table 5:** Incidence of Risk Factors

Our study shows percentage of various risk factors associated with MI. In most of cases multiple risk factors co-existed.

Table 6: Symptomatology at Presentation

Symptoms	RVI	IMI without	IMI
	(n = 15)	RVI (n=35)	( <b>n</b> = <b>50</b> )
Chest Pain	15 (100%)	34 (97.14%)	49 (98%)
Syncope	7 (46.6%)	2 (5.7%)	9 (18%)
Palpitation	1 (6.6%)	2 (5.7%)	3 (6%)
Sweating	10 (66.6%)	28 (80%)	38 (76%)
Angina Pai within 24 hrs.	n 4 (26.6%)	6 (17%)	10 (20%)

In our study chest pain was the commonest symptom followed by sweating. Syncope was essentially an important presenting symptom in RVI. Palpitation was the least presenting symptom in IMI.

**Table 7:** Physical findings at presentation

Physical Finding	IMI	RVI	IMI without			
	(n = 50)	(n = 15)	RVI (n=35)			
a. Pulse:						
Normal(60 –						
100)Bradycardia	39 (78%)	6 (40%)	33 (94.2%)			
(< 60)	8 (16%)	7 (46.6%)	1 (2.8%)			
	3 (6%)	2 (13.3%)	1 (2.8%)			
Blood Pressure						
Normotensive (100- 140/60-90) Hypotensive (<100/<60)	22 (49%) 14 (28%) 14 (28%)	2 (13.3%) 10 (66.6%) 3 (20%)	20 (57.1%) 4 (11.4%) 11 (31.4%)			
JVP						
Normal Elevated	12 (24%) 7 (14%)	10 (66.6%) 6 (40%)	2 (5.7%) 1 (2.8%)			
rt Sounds S3/S4	4 (8%)	2 (13.3%)	2 (5.7%)			
e.Tricuspid	7 (14%)	2 (13.3%)	5 (14.2%)			
regurgitation murmur						
f. Respiratory Crepitations	15 (30%)	7 (46.6%)	8 (22.8%)			
Hupstansian algusted IVD Producerdia and						

Hypotension elevated JVP, Bradycardia and Kussumauls sign were increasingly associated with RVI when compared to IMI without RVI.

### **Table 8:** ECG Findings of RVI

	Changes in RPLs	No. of patients of RVI (n=15)	Percentage
1.	Changes in only one RPL	0	0
2.	In only two leads	7	46.6
3.	In all the four leads	8	53.3
4.	In number of patients ST <sup>†</sup> in RV4	15	100
5.	Associated ST1 in V1	9	60

In our study ST segment, ST of RV4 was elevated in all the 15 cases of RVI, ST elevation in all four leads (RV3, RV4, RV5 and RV6) was in 8 cases, ST elevation in any lead in 7 cases and ST of V1 was elevated in 9 cases.

#### Table 9: Showing Clinical Course

		<b>RVI</b> (n = 15)	IMI without RVI (n = 35)	<b>Total</b> (n = 50)
1.	Complicated	11	18 (51.4%)	29 (58%)
2.	Uncomplicated	(73.3%) 4 (26.6%)	17 (48.5%)	21 (42%)

Complications were significantly higher in RVI than in IMI without RVI in our study. This clearly indicated that patients with RVI were prone to develop some complication.

Table 10: Showing Arrhythmia's

	Type of	RVI	IMI without	Total
	Arrhythmias	(n=15)	<b>RVI</b> (n=35)	(n =50)
1.	SVT/AF	0	0	0
2.	Ventricular Ectopics	2 (13.3%)	4 (11.4%)	6 (12%)
3.	Ventricular	1 (6.6%)	1 (2.8%)	2 (4%)
	Tachycardia			
4.	Ventricular	4 (26.6%)	1 (2.8%)	5 (10%)
	fibrillation			

The incidence of VF was significantly high in cases of RVI and it was a major cause for mortality. However, the incidence of it was very low in IMI without RVI. Ventricular Ectopics were seen at a similar incidence in both the groups. And most of them were transient which disappeared without any medication or causing any major problem.

	Table 11: Snowing conduction blocks						
	Conduction Block	RVI	IMI without	Total			
		(n=15)	<b>RVI</b> (n=35)	(n =50)			
1.	First Degree AV Block	0	3 (8.5%)	3 (6%)			
2.	Second Degree AV Block	1 (6.6%)	1 (2.8%)	2 (4%)			
3.	Complete Heart Block	7 (46.6%)	2 (5.7%)	9 (18%)			

Table 11. Showing conduction blocks

Our study shows the incidence of conduction block to be significantly high in cases of RVI. Complete Heart Block was commonly seen in the RVI and few of them it became normal that too after medication.

Table 12: Showing total incidence of Mortality after thrombolysis

	No. of Patients $(n = 50)$	Mortality (n = 9)
With Streptokinase	32 (64%)	2 (18.1%)
Without Streptokinase	18 (36%)	7 (81.9%)

Our study shows a high incidence of mortality in non thrombolysed patients which proves the benefit of thrombolysis.

Table 13: Showing incidence of Mortality Total death in the study = 9

Death in RVI group = 7 Death in IMI without RVI group = 2

	<b>RVI</b> (n = 15)	IMI without RVI (n=35)
Mortality	7 (77.78%)	2 (22.22%)

Mortality is significantly high in RVI were as it is lower in IMI without RVI.

Table 14: Showing Mortality incidence in Thrombolysed v/s non thrombolysed patients

	$\frac{\text{RVI}}{(n=15)}$	Mortality (n = 7)	IMI without $RVI (n = 35)$	Mortality $(n = 2)$
Thrombolysed	4 (26.6%)	1 (25%)	28 (80%)	1 (3.5%)
Non	11 (73.3%)	6 (54%)	7 (20%)	1 (14%)
Thrombolysed				

This report clearly shows the benefit of thrombolysis as the mortality in the nonthrombolysed group is very high.

#### Discussion

Our study consisted of 50 consecutive patients of AIMI as proved by ECG, who were admitted to ICCU of RRMCH. Additional RPLs were taken.

#### Table 15: Comparison of Incidence of RVI in all ٦*1*1

MI	

Study	Percentage of RVI in all MI
Cabin and Setaro study	13
Present Study	9.3

Cabin and Setaro J. reported an incidence of RVI in 13% of all cases of MI they studied in 1992. Our study shows 9.3% (only ECG proven) of RVI in all cases of MI. Our reported incidence of RVI is comparable to that of Cabin and Setarostudy.<sup>5</sup>

#### Table 16: Comparison of RVI in IMI

Study	Percentage of RVI in all MI
Gertz et al	32
Present Study	30

Our study based on ECG shows an incidence of 30% and Gertz et al reported necropsy analysis of IMI as a part of TIMI study to have had RVI in 30% of patients. So our report tallied with that of Gertz et al study.<sup>6</sup>

Study	Age of peak incidence of AMI
Dittrich H. Griffin	75.26% in below 60 years
et al Study	
Isreli Heart Study	34% in 45 – 54 years' age
Present Study	36% in 51 – 60 years' age

In Dittrich et al study the maximum incidence of MI was below 60 years (75.26%) and 24.26% in the patients aged above 60 years. Here the study was done in 820 patients MI. In Isreli Heart Study the peak age incidence was seen in age group of 45 – 54 years. Our study of IMI had a peak incidence of IMI in 36% in the age group of 51 -60 years and RVI of 53.3% in the same age group. So our report is similar and compared with these two groups with respect toage.<sup>7</sup>

 Table 18: Comparison of Sex incidence

Study	Males	Females
Chinnaiah et al	72%	28%
Kannel W B., et al	66%	34%
Present Study	86%	14%

Chinnaiah et al reported an incidence of 72% in males and Kannel W.B. et al in a 26 years follow up of a group of males and females aged between 35 - 84 years found the incidence to be 66%. Our study shows a higher incidence i.e., 86% in males. This clearly indicates a male predominance and it might be due to higher associated risk factors like smoking stress factors and alcoholism. Kannel et al study might be showing a lower male predominance because of other associated risk factors in females of the west which is not present in the females of ourstudy.<sup>8,9</sup>

**Table 19:** Comparison of Smoking as a risk factor

 in MI

Incidence of Smoking	Percentage
Masaharu Ishihara et al	90
Framingham Study	86
Present Study	78

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 78% of IMI patients to be smokers and none of the females in this study group weresmokers.<sup>10</sup>

So this high incidence of smoking associated with MI clearly proves that tobacco smoking is a very important risk factor for the development of MI.

 Table 20: Comparison of incidence of diabetes in

 MI

Incidence of diabetes	Percentage
Kaul et al	5
Dittrich et al	17.5
Present Study	22

Kaul et al noted the incidence of 5% of diabetes in MI. This may be due to the fact that his study consisted of young patients. In Dittrich Study it was 17.5%. Our study shows an incidence of 22%, which matches with that of Dittrichstudy.<sup>11</sup>

**Table 21:** Comparison of Risk of Hypertension in

 MI

Risk of Hypertension in MI	Percentage
Framingham study	40
North Karella Project	29
Present study	46

There is little difference between our study and Framingham study with that of North Karella Project and this difference is not very significant. The high incidence of hypertension in our study may be because we had a very limited study group over a short period.<sup>12</sup>

Usually these risk factors are not present singly. Multiple risk factors present together and this increases the risk.

Chest pain is the commonest symptom in AMI and it was present in 98% of our study group and in 100% of patients of RVI. Syncope was a very prominent symptom to be present with RVI that is in 46.66%. It was also present in 18% of IMI without RVI. This indicates a higher incidence of conduction defects in cases of RVI, which is the cause of syncope.

**Table 22:** Comparison of incidence ofbradycardia

Study	Incidence of bradycardia
Braat SH et al	48%
Mohan et al	66%
Present study	46.6%

Braat reported an incidence of bradycardia in 48% patients of RVI and Mohan et al in 66%. While out study shows 46.6% incidence. Whereas bradycardia in IMI without RVI in our study was 2.8%. Our study correlates with the reported incidences of other two groups. This mainly due to the involvement of the A.V. Node.<sup>13</sup>

Table	23:	Comparison	of	incidence	of
hypoter	nsion				

Study	Incidence of Hypotension
Shah et al	52%
Mohan et al	55%
Present study	66.6%

Shah et al reported hypotension in 52% and Mohan et al in 55% and our study shown 66.6% in cases of RVI. This is comparatively high when compared with 11.4% of hypotension in IMI without RVI. This proves that RVI causes significant hemodynamic derangements.<sup>13</sup> **Table 24:** Comparison of incidence of elevatedJVP

Study	Incidence of elevated JVP
Dell' Italia et al	88%
Present study	66.6%

Dell' Italia showed that raised JVP was present in 88% of the cases and 66.6% in our study. Dell' Italia et al reported the specificity of raised JVP to the 69% in cases RVI. This shows that JVP is an important clinical indicator for RVI and it also proves RV is an important part of circulation.

In our study ECG was the only (12 lead standard ECg + RPLs) investigative device used to prove RVI as other techniques were not affordable or available.

In our study in patients with RVI, ST elevation in only one RPL was not found. But RV4 was elevated in all the 15 cases of RVI. Croft et al in 1982 was the first to report that ST elevation of 1 mm or more in one or more of RV4 to RV6 was 90% sensitive and 91% specific for RVI. It is now generally agreed that 1 mm ST elevation in RV3 to RV6 or only in RV4 in highly specific and sensitive for diagnosing RVI.<sup>14,15</sup>

Chou et al in 1981 had proposed that ST elevation in V1 might suggest RVI and our study of RVI in 15 patients showed that there was STS elevation in V1 in 9 cases (60%). This is significant finding.<sup>14</sup>

**Table 25:** Comparison of incidence of CHB inRVI

Study	Incidence of CHB in RVI
Lloyd et al	31%
Present study	46.6%

Lloyd et al have reported completed Heart Block in 31% of the cases where as our study showed 46.6%. in our study of complete heart block in IMI without RVI was 5.7%. This shows a significant risk in patients of RVI to develop complete heart block than in patients of IMI without RVI. This is because of the involvement of the AVnode.<sup>14</sup>

The incidence of VF in RVI was 26.6% where as in IMI without RVI was 2.8% in our study. Cinca

et al reported an incidence of 4% of patients developing VF during thrombolysis. His study included all cases of MI. Our study has only IMI and RVI. So incidence of VF in our study is very high.<sup>16</sup>

The incidence of VT in RVI was 6.6% and 2.8% in IMI without RVI in our study. Lopez and Sendon et al have reported very high incidence of VT and VF in cases of RVI who were catheterized (Swan Ganz Catheter) or were applie dpace makers. This might be due to the irritation by pacemaker or catheter of the injured RV.<sup>17</sup>

Mortality rates, particularly, in RVI, is higher than compared to IMI without RVI. In our study the mortality in RVI was 46.6%. Whereas it was only 5.7% in IMI without RVI. In thrombolysed patients the mortality was significantly low (25%) compared with non thrombolysed patients (54%). Most of the cases of RVI were not suited for thrombolysis of the associated complications. In these patients the death was high. Zehender et al have reported a high incidence of complications and mortality in patients who were not candidates for thrombolytic therapy. Castaigne et al in their study of mortality after thrombolysis reported an incidence of 4% where as in our study it was 18%.

### Conclusion

The incidence of mortality and complications can be reduced only when we are fully awareof the diagnosis and the complications that can occur in RVI.

So in all cases of IMI, RVI should be looked for by using simple and specific investigation like RPLs of ECG. Clinically RVI can be suspected when there is bradycardia, irregular pulse, hypotension and elevated JVP with clear lungs in a setting of Acute MI. ECG is a very simple investigative tool. The Advantage of ECG is it is easily available, noninvasive, cost effective, specific and sensitive.

The incidence of complications like hypotension, conduction defects and arrhythmias are very high in RVI. Hypotension occurs because of mechanical pump failure and can be corrected just

by volume loading and occasionally drugs may be required to raise the BP. Conduction defects like I degree, II degree and complete heart block are commonly seen. They are usually transient resolving with a short period of time but may be causing serious hemodynamic prolonged derangements and can cause death. Atropine should be given initially, if it does not improve, injection Isoprenaline should be given and still ifthere is no improvement, cardiac pacing should be done. Arrhythmias occurs commonly in RVI. Ventricular ectopic are the commonest and they usually do not cause problem so as to require treatment. If they are recurring regularly. Injection Lidocaine is given. Ventricular Tachycardia and Ventricular Fibrillation are seen commonly in RVI. VF is life threatening and should be reverted by DC cardioversion. The mortality rate in RVI is its association very high due to with complications. So RVI should be carefully searched for and the complications should be anticipated and necessary interventions should be undertaken as early as possible.

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