Clinical Profile of Right Ventricular Infarction

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Abstract- Coronary artery disease usually involves the left ventricle and acute myocardial infarction is almost always associated with hemodynamic evidence of predominant left ventricular dysfunction although the right ventricle may be involved in a reasonable proportion of patients dying with myocardial infarction. The lesion is usually accompanied by left ventricular infarction.

Involvement of the right ventricle is a common sequel of acute inferior myocardial infarction, especially after proximal right coronary artery occlusion. Patients clinically presenting with hypotension, elevated jugular venous pulse (JVP), and occasionally shock, all in the presence of clear lung fields should raise the suspicion of RVI. The ST-segment elevation of ≥0.1mV in the right precordial leads V4R is a readily available electrocardiographic sign used for diagnosis of (RVI) Right Ventricular Infarction Electrocardiography is recognized as the most simple and readily available diagnostic tool for identification of RVI.ECG with the right precordial leads (V3R to V4R) should be a routine part of the initial evaluation of patients with clinical suspicion of acute inferior myocardial infarction for early recognition and treatment of right ventricular infarction.

Index Terms- Right Ventricular Infarction, Kussmals Sign, ST Elevation Myocardial Infarction, Right Pre Cardial Leads

I. INTRODUCTION

In current Indian scenario coronary artery disease (CAD) is the most common non communicable disease. It is estimated that CAD will affect more than 65 million people by the year 2015. ST - elevation myocardial (STEMI) is the most dreaded complication of CAD[1] Acute myocardial infarction (AMI) is a clinical syndrome that results from occlusion of a coronary artery with resultant death of cardiac myocytes in the region supplied by that artery. When these patients present in emergency, they have ST elevation in ECG and so are referred as ST elevation myocardial infarction (STEMI). Depending on the distribution of the affected coronary artery, AMI can produce a wide range of clinical sequel, varying from a small, clinical silent region of necrosis to a large area of infarcted tissue resulting in cardiogenic shock and death. AMI is the leading cause of death in the developed and developing countries, including India.[2] Right ventricular infarction (RVI) as assessed by various diagnostic methods accompanies inferior-posterior wall myocardial infarction (MI) in 30 to 50% of patients. Recognition of the syndrome of RVI is important as it defines a significant clinical entity, which is associated with considerable immediate morbidity and mortality and has a well-delineated set of priorities for its management. Patients may clinically present with hypotension, elevated jugular venous pulse (JVP), and occasionally shock, all in the presence of clear lung fields. The ST-segment elevation of ≥0.1mV in the right precordial leads V4R is a readily available electrocardiographic sign used for diagnosis of RVI Right Ventricular Infarction [3]The triad of hypotension, elevated jugular venous pressure (JVP), and clear lung fields has been recognized as marker of RVI in acute inferior-posterior wall MI[4 5 6] Pulses paradoxus (decrease in size, or even momentary disappearance of the pulse during inspiration) and Kussmaul's sign[7] (an inspiratory increase in JVP) have also been reported in patients with RVI. The presence of elevated JVP and Kussmaul's sign in the setting of an acute inferior wall infarction indicate a hemodynamically significant RVI[6] (sensitivity = 88% and specificity= 100%), particularly when it is associated with significant damage to the left ventricle and/or interventricular septum Elevated JVP alone was found to be more sensitive (88%) but less specific (69%) Therefore, a careful bedside examination of the jugular venous pulse serves as an important diagnostic tool in determining the severity of RVI and raising the clinical alert of its acute hemodynamic consequences and the caution for the judicious use of drugs like morphine and nitrates.

Auscultation may reveal a right-sided S3 and S4.[7] Tricuspid regurgitation may be identified because of dilatation of right ventricular chamber, which may be severe when related to papillary muscle dysfunction [8, 9].

Electrocardiogram Electrocardiography is recognized as the most simple and readily available diagnostic tool for identification of RVI The right ventricular involvement can be diagnosed with a predictive accuracy well above80% by the presence of ST-segment elevation of ≥1 mm in the right-sided precordial lead, V4R, in the presence of an acute inferior or inferioposterior MI.,[10,11] The ST-segment elevation in V4R is a strong independent predictor of major complications and in-hospital mortality[12].

II. MATERIAL OF STUDY

A : Selection of Patients :

The present study was conducted on 116 patients admitted to ICCU M.G.M Hospital Warangal with acute myocardial infarction, out of which 35 patients are of acute inferior myocardial infarction fulfilled the following criteria and 16 cases are diagnosed as RVI.

1. Patients presenting with typical or atypical chest pain showing evidence of inferior infarction on ECG with window period of 1 hr to 48 hrs.
2. Patients showing evidence of congestive heart failure were excluded. Continuous bed side ECG monitoring done.

B. Method of Study:

Patients presenting with chest pain suggestive of myocardial infarction who showed ECG evidence of acute inferior infarction changes were assessed, by detailed history and physical examination as out lined in pro forma.

Inferior myocardial infarction is diagnosed by ST segment elevation >1mm, or QS with ST segment elevation in two or more leads of L II, III, AVF. For all inferior wall infarction cases right sided leads V_3R, V_4R are taken at the time of admission and daily. Investigations given in table form

Clinical features of 16 patients with Right Ventricular Infarction

Case	IP No	Age (Yr)	PR (mt)	BP (MmHg)	Infarct Site	Estimated CVP (cm H20)	Kussmaul's Sign	Right		Complications
								S3	S4	
1	9886	55	60	130/90	Inferior	N	-			-
2	10194	60	35	110/70	Inf– Post		+	+	-	Partial RBBB
3	11147	47	41	120/90	Inferior		+	+	-	2nd degree AV block
4	11637	60	-	-	Inferior		+	-	-	Junctoinal rhythem + episodes +
5	12318	73	100	150/100	Inf – Post	N	-	-	+	
6	14390	70	60	110/60	Inferior		+	+	+	Multiple ectopics
7	147241	65	60	90/60	Inferior	N	-	-	-	
8	15030	70	65	160/100	Inferior	N	-	-	-	Junctionalatrailectopics +
9	15576	50	65	110/70	Inferior		+	+	-	RBBB
10	17121	50	88	120/70	Inferior		+	-	+	VF
11	18751	65	100	110/70	Inferior	N	-	+	-	-
12	19610	45	68	120/70	Inf – Post	N	-	+	-	-
13	20637	60	88	100/80	Inferior	N	+	+	-	-
14	21914	62	71	90/70	Inferior		+	-	+	-
15	22286	50	50	130/90	Inferior	N	-	-	-	-
16	22962	47	65	100/70	Inferior	N	-	-	-	

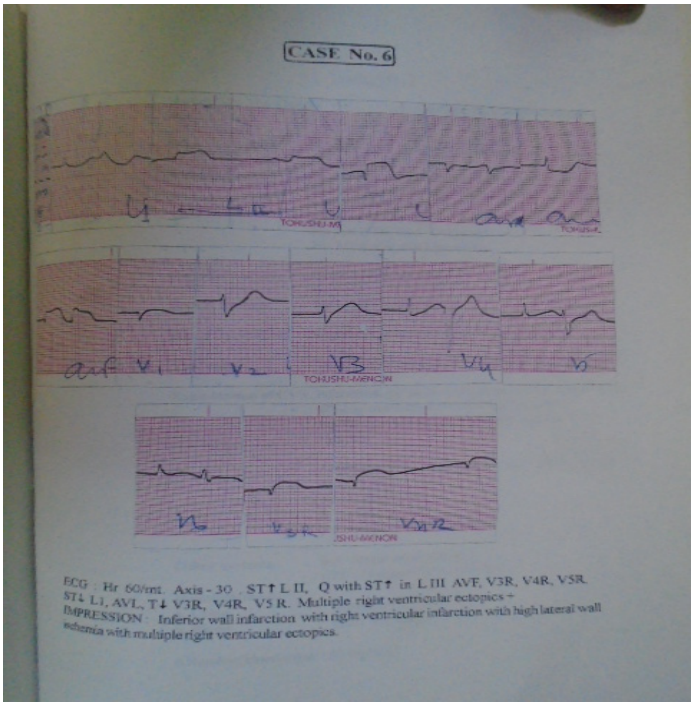
CHART SHOWING INVESTIGATIONS – TREATMENT – PROGNOSIS

S.No	IP No	Urine		Blood			Electrolytes		Enzmyes	Lipid profile					Treatment			
		Albumin	Sugar	ESR 1st hr mm	Blood sugar mg%	Blood Urea mg%	Sr Na+ meq / 1	Sr K+ meq/1	Ast Units / 1	Sr. Choleer mg/dl	HDL mg/dl	Trigly mg/dl	LDL mg/dl	Srer mg/dl	Heparin units	LMH	Strept units (million)	Prognosis
1	9880	-	-	10	112	32	140	3.5	189	176	46.6	362	57.4	0.3	-		1.5	Discharged
2	10194	-	-	15	98	40	130	3.3	117	146	40	344	37.4	0.8	-		1.5	Discharged
3	11147	-	-	12	92	24	141	3.7	136	256	53	396	120	0.7	-		1.5	Discharged
4	11637	-	-	-	-	-	-	-	-	-	-	-	-	-	-		-	Expired
5	12318	-	-	5	84	20	130	3.8	27	180	52	390	135	1.2	-		1.5	Discharged
6	14390	-	++	12	200	40	133	4	90	260	-	-	-	1.4	-	clexane	-	Expired
7	14721	-	-	15	140	20	130	3.8	40	230	50	256	129	1.2	-	clexane	-	Discharged
8	15030	+	-	14	92	24	138	3.8	390	216	55	170	125	1	-	clexane	-	Discharged
9	15576	-	-	20	100	28	132	3.2	160	200	50	216	117	0.8	-	clexane	-	Discharged
10	17121	-	++	18	200	28	142	4.7	78	160	40.3	180	120	0.7	Heparin	-	-	Expired
11	18751	-	-	18	120	28	133	4.2	110	232	40	300	132	0.7	-	clexane	-	Discharged
12	19610	-	-	10	128	20	130	3.8	129	176	40	180	106	0.7	Heparin	-	-	Discharged
13	20637	-	-	20	124	20	132	3.8	122	266	73	299	133.7	0.8	-	-	1.5	Discharged
14	21914	-	-	15	100	26	134	3.3	163	192	50.6	180	119	1.2	-	clexane	1.5	Discharged
15	22286	-	-	18	128	40	132	4	102	260	46	230	174	1.3	-	-	1.5	Discharged
16	22962	-	-	20	128	22	130	3.8	238	184	38.8	190	109	0.8	-	-	1.5	Discharged

ELECTROCARDIOGRAPHIC SIGNS OF RIGHT VENTRICULAR INFARCTION

Sign	Serial No. of Patients	Total
ST Segment abnormalities ST segment elevation in leads V_4R V_3R V_1R	 4,5,6,11,15 1,4,6,11 6,12	 5 4 2
Decreased ratio of ST segment Depression in V2 to ST segment Elevation in a VF	4, 5,6	3
QRS complex abnormalities Qs in leads V_3R V_4R QR in leads V_3R V_4R	1,3,5,6,7,8,9, 10,11,13,16 2,12	 13 2
Conduction disturbance Second and third degree AV block Right bundle branch block	 3 2,9	 1 2

ECG Changes	NO. of Patients
Total No. of RVMI Total No. of ST Segment abnormalities ST ↑ V4R ST ↑ V3R ST ↑ V1 Ratio of ST V2 ST AVF QS in leads V3 R V4R QR in leads V3R V4R 2^{nd}& 3^{rd} degree AV block Right bundle branch block	16 7 5 4 2 3 13 2 1 2



III. RESULTS

In the present study of 116 acute myocardial infarction cases anterior wall infarction 56 (48%), inferior wall infarctions 35 (30%), lateral wall infarctions 13 (11%), septal wall infarctions 12 (10%). Out of 35 inferior wall infarctions right ventricular infarctions are 16 (45.7%).

Age and Sex: In India the coronary artery disease is more common between the age group of 40-70 yrs. In the present study the age incidence is 40-75 yrs. The age incidence is higher in females. Out of 16 right infarctions 4 are females and attained menopause long back. The male: Female ratio 16:4. Mean age is 57. 2 patients presented with previous history of infarction. One patient presented with shock (case No. 4) and expired on the same day. Another (case 9) has RBBB. She was treated with clexane and she survived.

The clinical features reflect the extent of myocardial damage. The bed side diagnosis has been based on recognition of severe right ventricular dysfunction with right sided heart failure.

In our study Jugular venous pressure raised in 7 patients (43%) Kussmauls sign positive in 8 patients (50%) right sided 3rd sound 43% right sided 4th sound present in 4 patients (25%). Clinical findings are shown in the table 3.

Electrocardiogram: (ECG): Various authors studied ECG changes in acute inferior infarction. ECG changes produced by right ventricular involvement during inferior myocardial infarction may not be detectable in traditional right precordial leads V1 to V2 and perhaps only seen in patients with major right ventricular involvement or with clock wise rotation of heart. ECG findings indicated right ventricular damage exceeding 25% or reaching lateral margin of the right ventricular free wall. ECG changes are shown in table V & VI.

In the present study out of 35 inferior infarction cases 16 patients had ECG changes in V3R V4R representing involvement of right ventricle. ST segment abnormalities are present in 7 cases (44%) QRS complex abnormalities are present in 15 (93.8%). Conduction disturbance are present in 3 cases (18%). One patient presented with second degree AV block with the wenckebach phenomenon (case 3). One patient presented with partial RBBB (Case 2). One patient presented with RBBB (case 9). Other ECG abnormalities are junctional rythm in 1 case : atrial ectopics in one case, ventricular ectopics in 2 cases.

2D Echo: 12 patients undergone 2D Echo on 7th day 10 patients showed inferior wall hypokinesia. One patient with ejection fraction 42% with MR^{2+} expired on 9th day of admission. 3 patients developed mitral regurgitation.

Prognosis: out of 16 patients of right ventricular infarction 7 patients and treated with streptokinase (48%) 6 are treated with clexanae (38%). 2 patients are treated with Heparin (12.4%) 3 patients (18.7%) expired out of 16 admissions.

IV. DISCUSSION

Although right ventricular infarction (RVI) was described earlier in the autopsy studies, [13,14,15] Cohn et al.4 in 1974 gave the initial description of the clinical syndrome of right ventricular failure in patients of RVI. Since then RVI has been recognized more frequently and continues to be a diagnostic and therapeutic challenge[15].

The natural history of dominant RVI may be favorable because the right ventricle is very resistant to ischemia and usually recovers. Most patients, even those with substantial RV dysfunction, spontaneously improve in 48 to 72 hours after the acute event. Patients in shock may benefit from angioplasty of the occluded right coronary artery or from temporary use of an RV assist device; however, many of these patients have associated significant LV dysfunction, which complicates the picture. The overall balance between the extent of RV and LV dysfunction is a major determinant of long-term outcome, and the majority of patients with RVI and significant hemodynamic compromise usually have evidence of extensive biventricular infarction and cardiogenic shock. The *Clinical Practice Guidelines* recommend the following:

Class I

1. Patients with inferior STEMI and hemodynamic compromise should be assessed with a right precordial V4RR lead to detect ST-segment elevation and an echocardiogram to screen for RV infarction. (See the ACC/AHA/ASE [American Society of Echocardiography] 2003 Guideline Update for the Clinical Application of Echocardiography.) (Level of Evidence: B)
2. The following principles apply to therapy for patients with STEMI and RV infarction and ischemic dysfunction:

a. Early reperfusion should be achieved if possible. (Level of Evidence: C)
b. Atrioventricular synchrony should be achieved, and bradycardia should be corrected. (Level of Evidence: C)
c. RV preload should be optimized, which usually requires initial volume challenge in patients with hemodynamic instability provided the jugular venous pressure is normal or low. (Level of Evidence: C)
d. RV afterload should be optimized, which usually requires therapy for concomitant LV dysfunction. (Level of Evidence: C)
e. Inotropic support should be used for hemodynamic instability not responsive to volume challenge. (Level of Evidence: C)[16]

V. CONCLUSION

ECG evidence of RVMI in inferior wall myocardial infarction is 45.7% in our study. Majority of cases of RVMI observed in age group 40-75 years a mean age of 57 years. There is increased risk of MI in postmenopausal women. Most common modifiable risk factors found in our study are smoking (62%), dyslipidemia (50%), Hypertension (50%), Prevenon of which decreases risk of CAD. Common signs of RV dysfunction are raised JVP (43%) Kussmaul's sign (50%), right sided 3rd heart sound (43%), right sided 4th sound (25%). Bradycardia (43.8%) and hypotension are reserved with IV atropine and IV fluids. ECG Changes: QRS abnormalities in 93.8%, ST segment abnormalities in 43.8%, conduction disturbance in 35% of patients noted. Thrombolysis is beneficial in right ventricular

infarction patients. Mortality is high in patients presenting with shock. Patients treated with streptokinase are 48%, Clexane 38% Heparin 2%. Mortality in patients with right ventricular infarction is 18.7%.A 12 lead ECG with the right precordial leads (V3R to V4R) should be a routine part of the initial evaluation of patients with clinical suspicion of acute inferior myocardial infarction for early recognition and treatment of right ventricular infarction.

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