

Original Article:**A study of right ventricular infarction in inferior wall myocardial infarction**Asif Iqbal,¹ R. Muddarangappa,² S.K.D. Shah,² Sudha Vidyasagar¹¹Department of Medicine, Kasturba Medical College, Manipal, Karnataka, and²Department of Medicine, Sree Siddhartha Medical College, Tumkur, Karnataka**ABSTRACT****Background:** Right ventricular infarction (RVI) is frequently associated with inferior wall myocardial infarction (MI).**Methods:** This study was designed to identify the burden of RVI in patients presenting with inferior wall MI (n=50) by right precordial electrocardiogram (ECG) and comparing it with echocardiography (ECHO).**Results:** Their mean age was (54.5 ± 11.9 years); there were 42 males. ST elevation of greater than 1 mm in right precordial leads (RPL) suggestive of RVI was evident in 16 (32%) cases. Among the RPL (V₃R - V₆R) V₄R and V₅R showed sensitivity of 87.5%. The 12-lead ECG finding of ST-elevation greater than 1 mm in lead III and lead III/II greater than 1, had poor sensitivity (75%), specificity (88.2%) compared to ST- elevation of greater than 1 mm in any of the RPL (100%). Both the echocardiography criteria, namely right ventricular end-diastolic dimension (RVEDD) greater than 25 mm (92.3%) and the ratio of RVEDD to left ventricular end-diastolic dimension (RVEDD/LVEDD) greater than 0.7 (90%) indicating right ventricle (RV) dilatation was observed significantly more frequently in RVI group.**Conclusions:** RVI occurs in more than one-third of patients with acute inferior wall MI. All the patients with inferior wall MI should have RPL recorded as early as possible for evidence of RVI, of which V₄R, V₅R have the highest sensitivity.**Key words:** Right ventricular infarction, Electrocardiogram, Right precordial leads, Inferior wall myocardial infarctionIqbal A, Muddarangappa R, Shah SKD, Vidyasagar S. A study of right ventricular infarction in inferior wall myocardial infarction. *J Clin Sci Res* 2013;2:66-71.**INTRODUCTION**

Right ventricular infarction (RVI) was first recognized in a sub-group of patients with inferior wall myocardial infarction (MI) in whom right ventricle (RV) failure and elevated right ventricular filling pressure despite of relatively normal left ventricular (LV) filling pressure was observed. Post-mortem studies revealed that there is RV involvement in 19% to 51% of patients with acute inferior wall MI. RVI contributes significantly to haemodynamic instability, atrioventricular (AV) block, bradycardia requiring pacing support and in-hospital mortality and morbidity.¹

The diagnosis of RVI can be made from physical examination, echocardiography, first pass/equilibrium radionuclide ventriculography, technetium pyrophosphate myocardial scintigram, haemodynamic measurements and angiography. But, right precordial electrocardio-

gram (ECG) is most readily available simple, routinely performed investigation and is of prognostic value.¹

It is important to recognize RVI in patients presenting with inferior MI because, management of RVI differs substantially from the management of LVMI. Keeping this in mind, the present study was designed to study the prevalence of RVI among patients presenting with inferior wall MI using right precordial ECG and comparing it with echocardiography.

MATERIAL AND METHODS

Fifty patients presenting with inferior wall MI diagnosed on the basis of history, ECG, cardiac enzyme estimation and echocardiography^{2,3} admitted to intensive care unit (ICU) of Sree Siddhartha Medical College and Research Centre, Tumkur, during the period November 2007 to August 2009 were enrolled in the study. Institutional Ethics Committee approval was obtained to conduct the study.

Received: 30 October, 2012.

Corresponding Author: Dr Sudha Vidyasagar, Professor and Head, Department of Medicine, Kasturba Medical College, Manipal, Karnataka 574105 India. **e-mail:** vsagar33@yahoo.com

Patients with associated anterior and lateral wall MI, proved by ST elevation in anterior and lateral leads in ECG and wall motion abnormalities by echocardiography, a history of previous MI; ECG evidence of left bundle branch block (LBBB); cor-pulmonale; suspected pulmonary embolism; associated pericardial disease; and patients not consenting to participate were excluded from the study. RVMI was diagnosed based on the criteria listed in Table 1.

In all patients, a detailed history was taken and careful physical examination was done with special reference to haemodynamic parameters like jugular venous pulse (JVP), Kussmaul's sign, hypotension, presence of third and fourth heart sounds and cardiac murmur. They were also evaluated for coronary risk factors like smoking, hypertension, diabetes mellitus, obesity, alcohol and hypercholesterolaemia.

In all of them 12-lead ECG, cardiac enzyme assay and echocardiography were carried out. The 12-lead ECG recordings were made at 25 mm/sec speed and 1 mV=10mm setting. Right precordial leads (RPL) were applied on the right side of chest on the areas to which the leads corresponded on the left. Lead II ECG monitoring was done during the stay in ICU for identification of arrhythmias and conduction blocks. Echocardiography was done in all patients and compared with ECG findings. Coronary angiography was done in selected patients who could afford the procedure.

Other laboratory investigations included random plasma glucose, fasting lipid profile, blood urea nitrogen, along with serum lactate dehydrogenase (LDH) and serum aspartate aminotransferase (AST), CKMB (immunoinhibition method; Erba creatine kinase-MB kit, Transasia biomedical Ltd.); TROP I (lateral flow immunochromatographic test, Instant view trop I whole blood/serum kit, Alfa scientific design). All patients with inferior wall MI were divided into RVI and non RVI group based on right precordial ECG findings.

Table 1: Criteria for diagnosing RVI

ST segment elevation > 1mm in at least one of the right precordial leads
RV dilatation by 2D-echocardiography is given by RVEDD > 25 mm and RVEDD /LVEDD > 0.7

RVI=right ventricular infarction

RVEDD=right ventricular end-diastolic dimension

LVEDD=left ventricular end-diastolic dimension

Source: references 2,3

Statistical analysis

Descriptive statistical analysis was carried out and results on continuous measurements have been presented on mean \pm standard deviation (SD). Results on categorical measurements have been presented in number (%). Significance was assessed at 5 % level of significance. Chi-square, Fisher's exact tests were used to compare categorical variables. Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV) and accuracy were computed for 12-lead ECG and RPL, considering ST elevation greater than 1 mm in any of the RPL as 'gold standard'.

RESULTS

Of the 50 cases with inferior wall MI, 16 (32%) cases had evidence of RVI. Their mean age was 54.5 ± 11.9 years; there were 42 (84%) males. Most of the patients (30%) were in the age group of 51 - 60 years. Smoking was the major risk factor in 41 (82%) patients followed by hypertension in 22 (44%). They presented with chest pain (100%), sweating (86%) and syncope (2%) in RVI group. Comparison of physical examination findings in patients with and without RVI is shown in Table 2.

ST elevation of greater than 1 mm in one of the RPL indicating RVI was evident in 16 cases, of which 6 cases showed ST elevation in all 4 leads, namely, $V_3R - V_6R$ (Figure 1). ST-elevation was most commonly seen in lead V_4R and V_5R (87.5%) (Figure 2). In addition to RPL, ST elevation of 1 mm or more in lead III, ratio III/II greater than 1 was found in the 12-lead ECG in 14 patients (58.3%) in RVI group and 10 patients (41.7%) in non-RVI group. Echocardiography criteria³ of RVI, suggestive

Table 2: Comparison of physical examination findings at presentation in patients with and without RVI

Physical findings	With RVI (n=16)	Without RVI (n=32)	p-value
Pulse (/min)			
<60	3	2	0.377
60-90	11	26	
>90	2	6	
Systolic blood pressure (mm Hg)			
Low (<90)	3	2	0.398
Normal (91-140)	12	30	
High (>140)	1	2	
Tachypnoea	4	9	1.000
Jugular Venous Pulse			
Elevated	4	3	0.190
Kussumaul's sign positive	5	0	0.002
Basal crepitations	1	3	1.000

RVI= right ventricular infarction

of RV dilatation was observed in both the groups. Thirteen patients showed right ventricular end-diastolic dimension, (RVEDD) greater than 25 mm indicating RV dilation of which 10 (92.3%) had evidence of RV infarction. Also, 9 of the 10 patients with ratio of RVEDD to left ventricular end-diastolic dimension, (RVEDD/LVEDD) greater than 0.7 had evidence of RVI. Table 3 shows the sensitivity, specificity, PPV, NPV and accuracy of 12-lead ECG criteria and RPL in diagnosing RVI.

Comparison of the sensitivity of right precordial leads (V_3R - V_6R) in diagnosing RVI in the present study and other published studies is shown in Table 4.

Complications like sinus bradycardia, atrioventricular block, asystole and multiple ventricu-

lar premature complexes (VPCs) in patients with and without RVI is shown in Table 5.^{1,4-9}

DISCUSSION

In the present study, RVI was evident in 32% of cases with inferior wall MI. A similar prevalence of RVI of 30%¹⁰ and 37%¹¹ has been reported in other studies in patients with inferior wall MI. RVI is less common since RV is less susceptible to ischaemia as oxygen demand is significantly lesser because of its smaller muscle mass^{12,13} and coronary perfusion in right ventricle occurs in both systole and diastole.¹⁴

Most of the cases (46.7%) of RVI occurred in the age group of 51 - 60 years in the RVI group compared to non-RVI that was commonly (84.6%) seen in the age group of 41-50 years. In the present study 84% were males. Similar

Table 3: Diagnostic yield of 12-lead ECG and right precordial leads in diagnosing RVI

ECG Criteria	Sensitivity	Specificity	PPV	NPV	Accuracy	p-value
ST \geq 1mm in lead III, in lead III/II>1	75.0	88.2	75.0	88.2	84.0	<0.001
RV ₃	37.5	100	100	77.3	80.0	<0.001
RV ₄	87.5	100	100	94.4	96.0	<0.001
RV ₅	87.5	100	100	94.4	98.0	<0.001
RV ₆	75.0	100	100	89.5	92.0	<0.001

* ST elevation > 1 mm in any of the right precordial leads was considered as 'gold standard'

ECG= electrocardiogram; RVI= right ventricular infarction; PPV= positive predictive value; NPV= negative predictive value

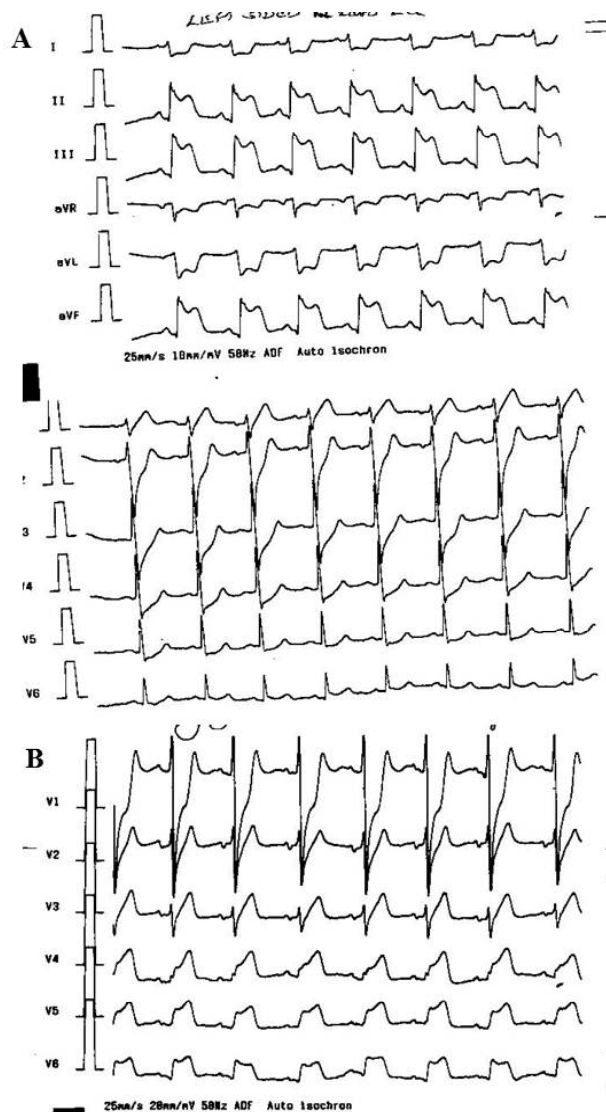


Figure 1: Electrocardiogram in a patient with inferior wall MI with right ventricular involvement showing (A) ST-elevation in leads II, III, aVF, lead III/II >1 (B) ST-elevation in right precordial leads (V₃R - V₆R) indicating right ventricular involvement

male preponderance (86%) was reported in another study.¹⁵ Among the 42 male patients in our study 13 (31 %) had RVI while only 3 of the 8 women (37.5%) had evidence of RVI; however, this difference did not attain statistical significance. In another study,¹⁶ it was observed that prevalence of RVI increased as age advanced and prognostic effect of RVI was independent of gender. All the patients in the study presented with chest pain. Prevalence of RVI was associated more with vomiting (41.6%), breathlessness (40%) and syncope (100%). A patient with RVI who also had com-

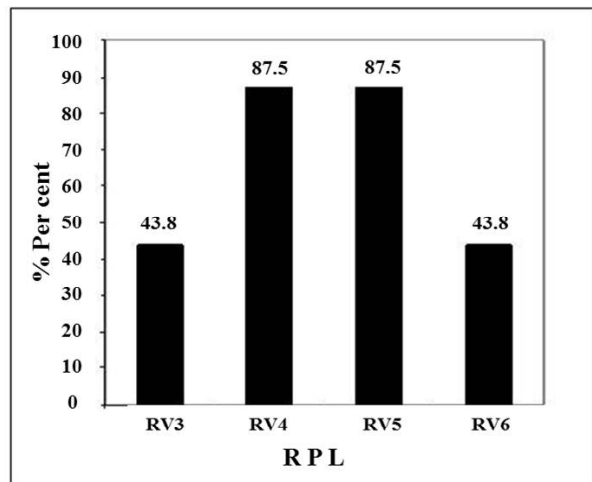


Figure 2: Evidence of ST elevation > 1 mm in individual RPL in patients with right ventricular infarction RPL=right precordial leads

plete heart block presented with syncope. Patients with RVI and complete heart block have been observed to present with episodes of syncope classically described as Stokes-Adams attack.¹⁷ In the present study among RPL, V₃R had sensitivity of 37% and lead V₄R and V₅R showed sensitivity of 87.5% for identifying ST elevation greater than 1 mm suggestive of RVI (Table 3). In most studies (Table 4)^{1,4-9} lead V₄R was found to be the most sensitive among all RPL in detecting RVI (Table 4). An ST segment which is higher in lead V₄R than in leads V₁ to V₃ offers the highest specificity and efficiency in diagnosis.² Comparing the reliability of 12-lead ECG criteria with that of RPL in diagnosing RVI, we found that 12-lead ECG finding of ST elevation greater than 1 mm in lead III, ST elevation in lead III/II greater than 1, had poor sensitivity (75%), specificity (88.2%), PPV(75%), NPV (88.2%) and accuracy (84%). In a study¹⁹ evaluating the reliability of 12-lead ECG criteria in the diagnosis of RVI, it was found that the right sided chest leads had a higher specificity and PPV than inferior leads, but poor sensitivity, NPV, and accuracy. However, the differences were not statistically significant. In right chest leads recognition of infarction signals is easy because only the presence of ST elevation of 1 mm or more is re-

quired to diagnose RVI but in inferior leads a difference between ST elevation in two leads must be interpreted. Thus right precordial leads appear to be a better indicator of RVI than 12-lead ECG.

In the present study, among patients in whom RVI was confirmed by echocardiography criteria,³ RPL evidence of RVI was evident in more than 90 percent of cases. In another study²⁰ of 42 inferior wall MI patients, 16 had an echocardiography evidence suggestive of RV dilatation; this included 12 with V_4R evidence of RVI.

Cardiogenic shock was the cause of death in 2 cases in our study of which 1 had evidence of RV involvement. It has been observed that inferior wall MI patients who also have RV myocardial involvement are at risk of death, cardiogenic shock and arrhythmia.²¹ Among the 8 patients with inferior wall MI treated with volume replacement, 6 had evidence of RVI in the present study. Patients with RVI who are haemodynamically unstable should be managed

with volume loading to maintain adequate RV preload as preload determines RV output. Any interventions that reduce the preload, like diuretics, nitrates and vasodilators should be avoided even in the absence of hypotension as this will lead to worsening of symptoms. This treatment strategy would differ from therapy for pump failure caused by acute left ventricular infarction due to need for maintaining the RV preload in acute RV infarction.²²

Our observations suggest that RVI occurs in more than one-third of patients with acute inferior wall MI. A spectrum of disease from asymptomatic mild right ventricular dysfunction to cardiogenic shock has been recognized. All the patients with inferior wall MI should have right sided precordial leads recorded as early as possible for evidence of RVI, of which V_4R and V_5R have highest sensitivity. The diagnosis can be further confirmed by echocardiography for RV dilatation thus helping in preventing the complications and mortality associated with it.

Table 4: Comparison of sensitivity of leads V_3R and V_4R in the diagnosis of RVI

Study	Year	Sensitivity in leads%		
		V_3R	V_4R	V_5R
Braat et al ⁴	1983	69	93	–
Klein et al ⁵	1983	–	83	–
Anderson et al ⁶	1983	37	33	–
Morgen et al ⁷	1984	–	100	–
Braat et al ⁸	1984	–	80	72
Gupta et al ⁹	1990	88.8	100	–
Zehender et al ¹	1993	–	88	72
Present study		37	87.5	87.5

RVI=right ventricular infarction

Table 5: Comparison of complications in patients with and without RVI

Complications	With RVI (n=16)	Without RVI (n=34)	p-Value
Multiple VPC	1	1	NS
1° heart block	2	6	NS
2° heart block	1	0	0.320
Complete heart block	1	1	0.542
Asystole	2	0	0.098
Sinus tachycardia	0	1	1.000
Sinus bradycardia	3	2	0.311

VPC=ventricular premature contractions; RVI=right ventricular infarction; VF=ventricular fibrillation

REFERENCES

1. Zehender M, Kasper W, Kauder E, Schonthaler M, Geibel A, Olschewski Met.al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction *N Engl J Med* 1993; 328:981-8.
2. Schamroth L, Schamroth C. Myocardial infarction. In: Schamroth C, editor. An introduction to electrocardiography. 7th edition, Oxford: Blackwell Science; 2003.p.150-3.
3. Feigenbaum H, Armstrong WF, Thomas Ryan T. Feigenbaum's echocardiography. 6th edition. Philadelphia: Lippincott Williams and Wilkins; 2005.p.465 -7.
4. Braat SH, Brugada P, deZwaan C, Coenegracht JM, Wellens HJJ. Value of electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction. *Br Heart J* 1983;49:368-72.
5. Klein HO, Tordjman T, Ninio R, Sareli P, Oren V, Lang R, et al. The early recognition of right ventricular infarction: diagnostic accuracy of the electrocardiographic V4R lead. *Circulation* 1983;67:558-65.
6. Anderson HR, Falk E, Nielson D. Right ventricular infarction: frequency, size and topography in coronary heart disease: a prospective study comprising 107 consecutive autopsies from a coronary care unit. *J Am Coll Cardiol* 1987;10:1223-32.
7. Morgen T, Albert E, Silvestri F, Pandullo C, Della Mea MT, Camerini F. Right precordial ST and QRS changes in the diagnosis of right ventricular infarction. *Am Heart J* 1984;108:13-8.
8. Braat SH, Brugada P, deZwaan C, Coenegracht JM, Wellens HJ. Right ventricular involvement with acute inferior wall myocardial infarction identifies high risk of developing atrioventricular nodal conduction disturbance. *Am Heart J* 1984;107:1183-7.
9. Gupta MC, Singh R, Wahal PK, Jain VK, Singh MM, Goyal HM, et al. Right precordial mapping. Diagnostic sensitivity and specificity of leads V3R to V6R in different intercostals spaces in cases with right ventricular infarction. *J Assoc Physicians India* 1990;38:327-9.
10. Malla RR, Sayami A. In hospital complications and mortality of patients of inferior wall myocardial infarction with right ventricular infarction. *J Nepal Med Asso* 2007;46:99-102.
11. Chockalingam A, Gnanavelu G, Subramaniam T, Dorairajan S, Chockalingam V. Right ventricular myocardial infarction: presentation and acute outcomes. *Angiology* 2005;56:371-6.
12. Kusachi S, Nishiyama O, Yasuhara K, Saito D, Haraoka S, Nagashima H. Right and left ventricular oxygen metabolism in open-chest dogs. *Am J Physiol* 1982; 243:H761-6.
13. Lee FA. Hemodynamics of the right ventricle in normal and disease states. *Cardiol Clin* 1992;10:59-67.
14. Hess DS, Bache RJ. Transmural right ventricular myocardial blood flow during systole in the awake dog. *Circ Res* 1979;45:88-94.
15. Bueno H, Lopez-Palop R, Perez-David E, Garcia-Garcia J, Lopez-Sendon JL, Delcan JL. Combined effect of age and right ventricular involvement on acute inferior myocardial infarction prognosis. *Circulation* 1998;98:1714-20.
16. Ferguson JJ, Diver DJ, Boldt M, Pasternak RC. Significance of nitroglycerine-induced hypotension with inferior wall acute myocardial infarction. *Am J Cardiol* 1989;64:311-4.
17. Khan S, Kundi A, Sharieff S. Prevalence of right ventricular myocardial infarction in patients with acute inferior wall myocardial infarction. *Int J Clin Pract* 2004; 58:354-7.
18. Cintron GB, Hernandez E, Linares E, Aranda JM. Bedside recognition, incidence and clinical course of right ventricular infarction. *Am J Cardiol* 1981;47:224-7.
19. Anderson HR, Falk E, Nielsen D. Right ventricular infarction diagnostic accuracy of electrocardiographic right chest leads V3R to V7R investigated prospectively in 43 consecutive fatal cases from a coronary care unit. *Br Heart J* 1989;61:514-20.
20. Candell-Riera J, Figueras J, Valle V, Alvarez A, Gutierrez L, Cartadellas J, et al. Right ventricular infarction: relationship between ST segment elevation in V4R and hemodynamic, scintigraphic and echocardiographic findings in patients with acute inferior myocardial infarction. *Am Heart J* 1981;101:281-7.
21. Mehta SR, Eikelboom JW, Natrajan MK, Diaz R, Yi C, Gibbons RJ, et al. Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. *J Am Coll of Cardiol* 2001;37:37-43.
22. Haji SA, Movahed A. Right ventricular infarction--diagnosis and treatment. *Clin Cardiol* 2000;23:473-82.