



Journal Homepage: - www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/11714

DOI URL: <http://dx.doi.org/10.21474/IJAR01/11714>



RESEARCH ARTICLE

TO STUDY INCIDENCE AND CLINICAL OUTCOMES OF RVMI IN PATIENTS WITH ACUTE INFERIOR WALL MI , A PROSPECTIVE OBSERVATIONAL STUDY

Sameer Anand¹, Owais Ahmed², Suhail Masood Khan³ and Rajveer Bainiwal²

1. Registrar General Medicine, GMC Jammu.
2. DNB Cardiology, GMC Jammu.
3. DNB Urology, GMC Jammu.

Manuscript Info

Manuscript History

Published: September 2020

Key words:-

Inferior Wall Myocardial Infarction,
Right Ventricular Infarction,
Electrocardiography

Abstract

Background: Recognition of the syndrome of RVMI is important , as it is associated with major complications and in hospital mortality after acute IWMI. RV leads are often not done routinely as there is less awareness about RVMI

Aims And Objective: To study incidence and clinical outcomes of RVMI in patients with acute inferior wall MI using right precordial ECG leads

Materials And Methods: It was a prospective observational study, patients admitted with inferior wall MI to hospital from October 2019 to September 2020 underwent a twelve leads ECG ; and additional right precordial leads V3R, V4R, V5R, V6R.

Results: Out of 150 patients of acute IWMI admitted to CCU , 45 patients had RVMI (30%). Mortality was 5 times higher in patients with IWMI and RV extension , Complications like cardiogenic shock , various arrhythmia , AV block, VT were higher in RVMI.

Conclusion: RVMI is very common with inferior wall MI and It can be easily diagnosed by a simple bedside routine 12 lead ECG along with right sided ECG leads . Early diagnosis of RVMI can prevent fatal complications and high in-hospital mortality. Therefore right sided ECG should be taken routinely in all patients of IWMI.

Copy Right, IJAR, 2020.. All rights reserved.

Introduction:-

In 1974, Cohn for the first time described potentially serious and unique hemodynamic consequences of right ventricular infarction.¹ It has also been shown that right ventricular infarction occurs most commonly in association with inferior myocardial infarction or inferoposterior myocardial infarction^{2,4}. Although isolated right ventricular infarction had been described in autopsy reports as less than 3% of all acute myocardial infarction,² the incidence of right ventricular infarction associated with IWMI has been shown to be as high as 30%–50%^{3,4,5}.

It results primarily from occlusion of the right coronary artery and infrequently from involvement of the left anterior descending artery and occasionally in inferoposterior left ventricular infarction also.^{2,4}

Right ventricular myocardial infarction can lead to diminished right sided stroke volume with concomitant right ventricular dilatation and septal changes. The potential hemodynamic derangement associated with right ventricular

infarction renders the patients unusually sensitive to diminished ventricular preload. These two circumstances can result in a severe decrease in right and, secondarily, left ventricular output resulting in a clinical triad of hypotension and jugular venous pressure distension in the presence of clear lung fields.^{6,7,8}

It is known that ST segment elevation of 0.1 mV or greater in one or more of right precordial leads V4R to V6R is highly sensitive (90%) and specific (91%) in identifying acute right ventricular infarction.^{9,10}

A subtle clue to the presence of hemodynamically significant right ventricular infarction is a marked sensitivity to preload-reducing agents such as nitrates, morphine, or diuretics. Prompt fluid therapy may abort the vicious cycle set in motion by right ventricular infarction, which if treated in conventional way or neglected tends to lead to true cardiogenic shock.^{11,12}

Recognition of the syndrome of RVMI is important as it identifies a significant clinical entity, which is associated with and is an independent predictor of major complications and in-hospital mortality after acute inferior MI.^{12,13}

It is associated with increased risk of death, shock, ventricular tachycardia or fibrillation and atrioventricular block (AVB), and a higher mortality rate for the first month post MI in patients with RVMI but without hemodynamic impairment. These complications in RVMI may be due to the increased parasympathetic tone, sinoatrial (SA) node dysfunction, atrioventricular (AV) nodal dysfunction.^{4,13,14}

Aims And Objective:-

To study incidence and clinical outcomes of RVMI in patients of acute inferior wall MI using right precordial ECG leads. This study was done in a tertiary care hospital in north India from October 2019 to September 2020.

Objectives-

1. To study the incidence of right ventricular infarction in patients of acute inferior myocardial infarction using right precordial electrocardiography.
2. To study the clinical profile of patients of RVMI in acute IWMI.
3. To study the incidence of complications in patients of acute IWMI with and without RVMI.
4. To compare the mortality in patients of IWMI with and without RVMI.
5. To study the response of specific therapy in patients with right ventricular infarction.

Materials And Methods

It was a prospective observational study, patients admitted with inferior wall MI to hospital from October 2019 to September 2020 underwent a twelve leads ECG; and additional right precordial leads V3R, V4R, V5R, V6R.

All ECG's were recorded in 25mm/second 10 mV setting. The points on chest wall used for recordings chest leads were marked with a skin pencil so that same points could be used serially in a given patient. Serial ECG's were taken on admission, at the end of 6 h, end of 12 h and daily till ST segment became isoelectric in right precordial leads. ECG's were also repeated whenever patient complained of chest pain. Patients were continuously monitored. The diagnosis of acute inferior wall myocardial infarction was made as typical history of chest pain, ST segment elevation in leads II, III and avF and by development of pathological q waves in the above mentioned leads. Tall 'R' waves in V1 & V2 and increased serum cardiac enzymes Troponin-T[qualitative]). The diagnosis of right ventricular myocardial infarction will be made as by the criteria of ST segment elevation of 0.1 mV or more in one or more of the right precordial leads (V3R, V4R, V5R & V6R) in those patients who satisfied the criteria for an inferior wall myocardial infarction.

Patients were classified into two groups

Group A: Inferior wall infarction with right ventricular infarction.

Group B: Inferior wall infarction without right ventricular infarction.

Their clinical course were studied and compared. The clinical course, and ECG analysis was compared in both the groups during their entire hospital stay. Complications viz ventricular tachycardia, AV blocks, hypotension, cardiogenic shock and deaths in both the groups were analysed and compared.

Inclusion Criteria

1. All patients with definite evidence of acute inferior wall myocardial infarction as proved by 12 lead ECG along with right precordial leads and chest pain of duration less than 24 hours were considered in our study.

Exclusion Criteria

1. History of chest pain of more than 24 h duration.
2. Patients whose initial ECG's showed an anteroseptal or anterior wall myocardial infarction will be also excluded because these infarctions, may produce an anteriorly oriented ST vector which may also cause ST segment elevation in the right precordial leads. For the same reason patients with pericarditis, left bundle branch block were excluded.
3. Patients with chronic lung disease, cor pulmonale were also excluded because they may be associated with a right ventricular dysfunction.
4. Patients with previous history of a myocardial infarction were also excluded to avoid a false positive result for right precordial electrocardiography.

Following clinical parameters were considered for comparison viz

1. Hypotension was defined as systolic arterial blood pressure <90 mm of Hg or which has declined by at least 30 mm of Hg below previous level.
2. Right heart failure was considered when the hepatojugular reflux or abdominal compression test will be positive. JVP more than 4 cm in 45° position with pedal oedema and systemic venous congestion.
3. Ventricular tachycardia
4. AV block 2° and higher
5. Death
6. Cardiogenic shock

Left heart failure was considered when bilateral pulmonary rales was present on inspiration; Dullness on percussion over lung bases and on auscultation S3 and S4 were present. Peripheral pulse shows pulsus alternans. Chest X-ray shows Kerley B lines. Present study deals with clinical profile of right ventricular infarction as diagnosed by right precordial electrocardiography in patients of acute inferior wall myocardial infarction. The results obtained were analysed for their statistical significance using fishers exact test and unpaired 't' test.

Results:-

150 patients of acute inferior wall MI were admitted to CCU, from October 2019 to September 2020 and their serial 12 lead ECGs along with right sided leads RV3, RV4 were taken at the time of admission, then every six hourly and thereafter whenever the patient complained of chest pain. Patients were continuously monitored during hospital stay and their serial ECGs, clinical course and response to the treatment was analysed.

Out of 150 patients admitted with IWMI, 45 patients had RVMI (30%) and 105 patients were without RVMI (70%) in IWMI. Incidence of RVMI was more in females(40%) as compared to males(20%). 28% hypertensive patients had RV involvement while in diabetics the incidence was 36%.Tobacco and positive family history contributed significantly in IWMI with incidence being 42% and 32.2% respectively. Maximum number of patients presented with chest pain (90%) followed by perspiration(35%),dyspnoea(28%),palpitations(20.%)and syncope(10%).Raised JVP was an important sign in RVMI (65%) as compared to patients without RVMI(30%). There was five times increase in mortality in patients of IWMI with RVMI as compared to in no RVMI group. There was 6% mortality in the thrombolysed group compared to 18% in non thrombolysed group in IWMI, suggesting that mortality was higher in non thrombolysed group in IWMI. In RVMI also mortality was higher in non thrombolysed (50%) patients compared to thrombolysed patients(13%).There was a drastic increase in mortality in patients of RVMI who present late after the onset of symptoms, from nil presenting within 7 hours to 37.5% within 8-12 hours and 50% after 12 hours.

Discussion:-

Most patients were in the age range of 41-80 years,with slightly more patients falling in the age group 41-60 years in both RVMI+ and RVMI- groups. This was not significant $P >>0.05$ so this may be a chance association. This may be due to non consecutive selection of patients. It shows that right ventricular myocardial infarction does not show any preponderance to a particular age group.

The mean age of our study population was 59.7 years which correlated very well with the study of **Mehta et al**¹³.

In the present study population there were 97 male patients (65%) and 53 female patients (35%). Incidence of RVMI was found to be more in female population 36 % vs 22 % in males. Since ($p > 0.05$) this correlation was statistically not significant

So distribution of RVMI did not show any sexual predilection.

Incidence of RVMI in inferior wall MI :

In the present study RVMI was diagnosed in 45 patients out of 150 patients of IWMI.

Incidence of RVMI is variable depending upon the criteria used to diagnose it so in many other studies its incidence varies from 10 % to 50% .^{4,11,13,15} In the present study we used ECG criteria as the sole method to diagnose RVMI while most other studies have used echocardiography, right heart catheterisation and technetium scan to diagnose RVMI^{3,13,1} and because ST elevations in lead V4R mostly persist upto 12 hours after the MI, some patients are likely to be missed.

Mortality in IWMI patients

In the present study we followed the clinical course of 150 hospitalised IWMI patients. Out of 150 patients we had 12 deaths leading to 8% mortality. Out of 12 deaths, 8 were diagnosed to have RVMI leading to mortality of 18% in RVMI group whilst 4 deaths occurred in non RVMI group leading to 4.2 % mortality in that group.

Mortality was significantly higher in RVMI group $P < 0.05$.

Therefore presence of right ventricular infarction in IWMI qualifies as high risk group. But mortality rate varies among different studies due to different management protocols and facilities available in different centres.

Complications

Cardiogenic shock:

Out of 45 patients with RVMI, 9 patients had cardiogenic shock accounting for 20% incidence in that group while only 3 patients out of 105 patients had cardiogenic shock (3%). This was significant ($P = 0.04$).

Mehta et al¹³ also found 6.9 % incidence of shock in RVI patients compared to 5.5% incidence without RVMI.

AV blocks: In the present study, we evaluated the incidence of 2° and higher blocks in patients with and without RVMI. Out of 45 patients with RVMI 11 had AV blocks which is 24% incidence in that group, whilst 12 patients of IWMI without RVMI had AV blocks accounting 12.6% incidence. So incidence of AV blocks was found higher in RVMI group.

$P = 0.01$, therefore there is significant association between AV blocks and incidence of RVMI.

Mehta et al¹³ also showed similar results in their study with 21% incidence in RVMI group compared to 9% incidence in non RVMI group.

Bueno et al¹⁴ in 1998 found AV blocks in 25% of patients of RVMI and 6.7% patient of IWMI without RVMI.

Ventricular tachycardia:

Out of 45 patients with RVMI, 5 had Ventricular Tachycardia which is 12% incidence in that group whilst 4 patients of IWMI without RVMI had Ventricular Tachycardia accounting for 4% incidence. So incidence of VT was found to be higher in RVMI group. Although $p > 0.05$ incidence was found to be twice that of patients without RVMI

Mehta et al¹³ also found increased incidence of VT/VF (three times) in patients with RVMI. They attributed increased mortality in RVMI group to increased arrhythmias.

Hypotension : It is one of clinical hallmark of RVMI. Out of 45 patients with RVMI 18 patients had hypotension during their hospital stay which is equal to 40% incidence whilst only 12 patients in IWMI without RV involvement had hypotension accounting for 12.6% incidence $P = 0.006$ which was very significant. Therefore hypotension was much more common in patients with RVMI.

Mehta et al¹³ reported hypotension in 29% patients with RVMI compared to 19.3% in patients of IWMI without RV involvement.

Hypotension in RVMI occurs due to impaired filling of left ventricle secondary to impaired right ventricular cardiac output and decreased LV filling pressures therefore volume loading in case of RVMI is an important therapeutic approach to prevent cardiogenic shock.

Conclusion:-

Right ventricular myocardial infarction occurs in a significant number of patients with inferior wall myocardial infarction. Extent of right ventricular involvement varies from patient to patient and ranges from myocardial stunning to full blown infarction.

It represents a high risk subgroup of IWMI with distinct clinical presentation comprising of raised JVP and hypotension with clear lung fields which requires a high degree of clinical suspicion.

However it can be easily diagnosed with considerable accuracy by a simple bedside routine 12 lead ECG along with right sided ECG leads. Since ECG changes are transient in right sided ECG, it is difficult to diagnose RVMI after 12 hours. So ECG should be supplemented by urgent routine echocardiography to improve diagnostic yield.

Frequent accompaniments of RVMI include atrial infarction, sinus bradycardia, ventricular tachycardia, atrial fibrillation, atrioventricular block and cardiogenic shock. Patients with RVI who are hemodynamically unstable should be managed with volume loading to maintain adequate right ventricular preload. Early recognition and prompt reperfusion with thrombolytic therapy or coronary angioplasty, rate and rhythm control, and inotropic support may also be warranted since patients with RV infarction have high incidence of complications and in-hospital mortality.

Patients who survive the acute phase have no long-term consequences, and complete recovery over a period of weeks to months is the rule in majority of patients, suggesting right ventricular "stunning" rather than irreversible necrosis.

Therefore right sided ECG should be taken routinely in all patients of inferior wall MI

References:-

1. Cohn JN, Guha NH, Broder MI, Lima CJ: Right ventricular infarction: Clinical and hemodynamic features. *Am J Cardiol* 1974 FEB;33(2):209-14.
2. Menown IB, Allen J, Anderson JM, et al. Early diagnosis of right ventricular or posterior infarction associated with inferior wall left ventricular acute myocardial infarction. *Am J Cardiol* 2000; 85: 934-938.
3. Kinch JW, Ryan TJ. Right ventricular infarction. *N Engl J Med* 1994; 330:1211.
4. Khan, S., Kundi, A. and Sharieff, S. (2004) Prevalence of Right Ventricular Myocardial Infarction in Patients with Acute Inferior Wall Myocardial Infarction. *International Journal of Clinical Practice*, 58, 354-357.
5. Anderson HR, Falk E, Nielson D. Right ventricular infarction: frequency, size and topography in coronary heart disease. *J Am Coll Cardiol* 1987; 10: 1223-1232.
6. Brookes C, Ravn H, White P, Moeldrup U, Oldershaw P, Redington A: Acute right ventricular dilatation in response to ischemia significantly impairs left ventricular systolic performance. *Circulation* 1999 AUG 17;100(7):761-7.
7. Goto Y, Yamamoto J, Saito M, Haze K, Sumiyoshi T, Fukami K, Hiramori K: Effects of right ventricular ischemia on left ventricular geometry and the end-diastolic pressure-volume relationship in the dog. *Circulation* 1985; 72:1104-1114.
8. Goldstein J.A. Pathophysiology and management of right heart ischemia. *J Am Coll Cardiol*. 2002;40:841. [PubMed].
9. Braat SH, Gorgels AP, Bär FW, Wellens HJ. Value of the ST-T segment in lead V4R in inferior wall acute myocardial infarction to predict the site of coronary arterial occlusion. *Am J Cardiol* 1988; 62:140.
10. Klein HO, Tordjman T, Ninio R, Sareli P, Oren V, Lang R, Gefen J, Puzner C, Di Segni E, David D, Kaplinsky E: The early recognition of right ventricular infarction: Diagnostic accuracy of the electrocardiographic V4R lead. *Circulation* 1983;67:558-565.
11. Right Ventricular Infarction-Diagnosis and Treatment SHOWKAAT. H kn, M.D., AND ASSAD MOVAHED MD.D., , *Clin. Cardiol*. 23,473-482 (2000).
12. Carter T, Ellis K. Right ventricular infarction. *Crit Care Nurse*. 2005;25: 52-4, 56-8, 60-2.
13. Impact of Right Ventricular Involvement on Mortality and Morbidity in Patients With Inferior Myocardial Infarction Shamir R. Mehta, MD, FACC,*† John W. Eikelboom, MBBS, FRACP,† Madhu K. Natarajan, MD, FACC,*† *Journal of the American College of Cardiology* Vol. 37, No. 1, 2001 2001 by the American College of Cardiology ISSN 0735-1097/01.

14. In-Hospital Outcome of Elderly Patients With Acute Inferior Myocardial Infarction and Right Ventricular Involvement by Héctor Bueno, Ramón López-Palop, Javier Bermejo, José L. López-Sendón, and Juan L. Delcán *Circulation* Volume 96(2):436-441 July 15, 1997.
15. Iqbal 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.