Section: Anatomy

Original Research Article

STUDY OF RIGHT VENTRICULAR INFARCTION: ECHO CARDIO GRAPHIC EVIDENCE AMONG PATIENTS WITH INFERIOR WALL MYOCARDIAL INFARCTION IN SOUTH KARNATAKA POPULATION

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Abstract

Background: Cardiovascular disease is a major health problem in developed as well as developing countries. The rising prevalence of chronic diseases like hypertension type IIDiabetes Meletus, obesity, and an unhealthy lifestyle have led to coronary artery disease, which leads to myocardial infarction. **Materials and Methods:** Out of 225 CVD patients, 75 had MI with abnormal segmental elevation and 150 had normal segmental elevation. Both groups I and II were compared with the ECG, echocardiography Blood examination included a troponin-T level, and significant results were noted. **Result:** Kussmaul's signs Echocardiography and ECG were compared in both groups except LVED, and LV all the parameters had significant p values ($p<0.001$). **Conclusion:** Right ventricular MI complicating inferior is wall identified by ECG. Hence, early echocardiography will help to determine the presence or absence of RVMI, and the cardiologist can predict the prognosis of both groups and treat them efficiently.

INTRODUCTION

Cardiovascular disease (CVD) is a significant problem in developing countries.[1] Over the past 40 years, the mortality rate of coronary heart disease has dropped by two-thirds. This decline is the result of improved identification and management of risk factors as well as advances in treating CVD and myocardial infarction. But in underdeveloped countries, CVD remains the leading cause of mortality andmorbidities.[2]

Myocardial infarction and unstable angina can occur with or without ST-segment elevation. These conditions are brought on by unstable coronary plaques, abnormal blood clotting, and constriction of blood vessels.

This results in subendocardial or transmural ischemia which ultimately ends in myocardial infarction. ST elevation myocardial infarction (STEMI) is life-threatening unless immediate coronary intervention is done.[3] Inferior wall myocardial infarction occurs from coronary artery occlusion, with the resultant decrease in perfusion to that region of myocardium. In 80% of cases, the inferior myocardium is supplied by the posterior descending branch of the right coronary artery.[4] Approximately 40%–50% of all MIs involve the inferior wall. Hence, an attempt is made to evaluate the cardiac functions with the help of echocardiography to rule out the severity of MI and treat it accordingly.

MATERIALS AND METHODS

225 patients aged between 25 to 60 years of age were admitted to the ICU due to symptoms of myocardial infarction at Pradeep's cardiac care center, BGSMCH Hospital and Sapthagiri Institute of Medical Sciences and research Center Bangalorewere studied.

Inclusive Criteria

Patients having chest pain suggestive of angina lasting for more than thirty minutes positive Troponin-T test after six hours of the onset of chest pain. ECG evidence of evolving acute myocardial infarction (MI) in inferior wall leads. II, III, and a VF in the form of new Q waves or ST segment abnormalities were selected for study.

Exclusion Criteria

Patients with previous decimated abnormal ventricular function left bundle branch block, atrial

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fibrillation, COPD, already under treatment were excluded from the study.

Method: Echocardiographic evaluation was done during admission. Each diastolic dimension was obtained for both ventricles in the minor axis. The end diastolic right ventricle/left ventricle dimensional ratio was calculated. Elevation of the ratio suggested relative dilatation of the right ventricle. Real-time two-dimensional echocardiography was performed through standard para-sternal long and short axis views as well as four chamber views from apical and sub-costal positions. Wall motion was qualitatively classified as normal (limited systolic inward motion), akinetic (lack of systolic inward motion), or dyskinetic (outward motion of the endocardium during systole).

Volumes at end diastole and end systole were estimated for the left ventricle and right ventricle by using single-plane Simpon's rule. Right ventricular enlargement was considered to be present when the end diastolic ratio between the right and left ventricles was greater than 0.5. In an attempt to relate abnormal wall motion to global function, the injection fraction (EF) was estimated for these volumes.

$$\text{Ejection infarction} = \frac{\text{End diastrolic} + \text{Eng diastolic (volume)}}{\text{End diastolic volume}} \text{X } 100$$

The though none of dimensional echocardiography patients with evidence of abnormal segmental wall motion of the right ventricular free wall was classified in group I (75), and the rest of the patients in group II (150). These two groups will be compared for clinical profile, ECG, and echocardiographic parameters.

Patients in Group I with RVMI were compared with those in Group II without RVMI so as to determine the significance of age-related smoking mellitus (type II DM) in addition to echocardiographic parameters.

The duration of the study was from June 2022 to April 2024.

Statistical analysis: ECG and echocardiographic parameters were compared by the t test. The statistical analysis was carried out using SPSS software. The ratio of males and females was 2:1.

RESULTS

[Table 1] Comparison of detection of right ventricular infarction by echocardiography and presence of Kussmaul's sign in both groups Echocardiography has assessed 75+ve (right ventricular infarction) and 150-ve (no right ventricular infarction); the same findings were observed in Kussmaul's sign.

[Table 2] Comparison of Echocardiographic Parameter Profiles in Both Groups

- REVD–25–14 (±4.10) in group I, 16.0 (±4.10) in group II, t test was 14.1 and p<0.001 (p value was highly significant).
- LVED: 45.80 (±7.60) in group I, 46.70 (±6.8) in group II, t test was 0.86 and p>0.38
- RVED/LEVD: 55.12 (± 11.1) in group I, 39.5 (± 8.2) in group II; t test was 10.8 and p<0.001 (p value was highly significant).
- RV: 43.12 (± 13.40) in group I, 26.40 (±10.15) in group II; t test was 9.69 and p<0.001 (p value was highly significant).
- LV: 69.22 (± 20.21) in group I, 65.15 (± 21.24) in group II; t test was 1.4 and p>0.16 (p value was insignificant).
- RV/LV: 65 (±20) in group I, 42 (±16) in group II, t test was 8.6 and p<0.001) (p value was highly significant).
- RVEF: 39.98 (± 5.20) in group I, 51.8 (± 6.40) in group II; t test was 14.8 and p<0.001.
- LVEF: 59.28 (± 9.20) in group I, 55.18 (± 6.50) in group II; t test was 3.45 and p<0.001 (p value was highly significant).

[Table 3] Comparative study of detection of right ventricular infarction by ECG and echocardiographic study; 75 had +ve (positive) and 150 had -ve (negative) findings.

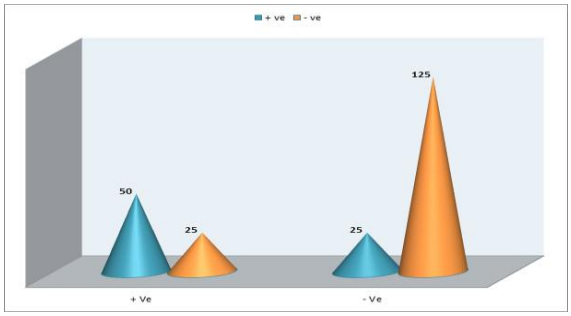

Figure 1: Comparison of detection of Right ventricular infarction by Echocardiography and presence of Kussmaul's signs in both groups Echocardiography

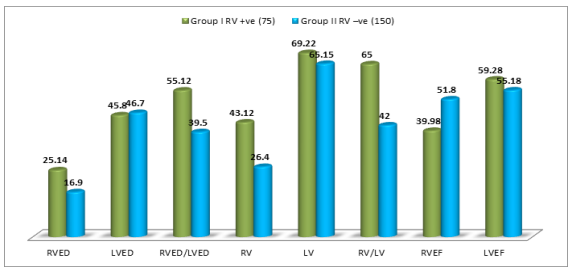

Figure 2: Comparison of Echocardiographic parameters profile of both group of patients

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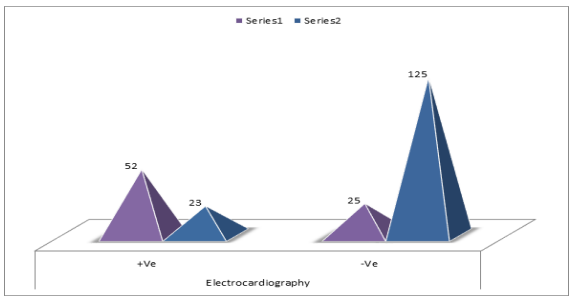

Figure 3: Comparative study of detection of Right ventricular infarction by Echocardiography and Electrocardiography

Table 1: Comparison of detection of Right ventricular infarction by Echocardiography and presence of Kussmaul's signs in both groups Echocardiography.

Kussmaul's Sign	+ Ve	- Ve
+ ve	50	25
- ve	25	125

+ve = Right ventricular infarction
-ve = No Right ventricular infarction

Table 2: Comparison of Echocardiographic parameters profile of both group of patients

Parameters	Group I RV +ve (75)	Group II RV –ve (150)	t test	p value
RVED	25.14 (±4.12)	16.90 (±4.10)	14.1	P<0.001
LVED	45.80 (±0.60)	46.70 (±6.82)	0.86	p>0.38
RVED/LVED	55.12 (± 11.1)	39.5 (±8.2)	10.8	P<0.001
RV	43.12 (±13.40)	26.40 (±10.15)	9.69	P<0.001
LV	69.22 (±20.21)	65.15 (±21.24)	1.4	p>0.16
RV/LV	65 (±20)	42 (±16)	8.6	P<0.001
RVEF	39.98 (±5.20)	51.8 (±6.40)	14.8	P<0.001
LVEF	59.28 (±9.20)	55.18 (±6.50)	3.45	P<0.001

RVED = Right ventricular end dimension LVED = Left ventricular end dimension
RV = Right ventricular end dimension volume LV = Left ventricular end dimension volume

Table 3: Comparative study of detection of Right ventricular infarction by Echocardiography and Electrocardiography

Electrocardiography		
Electrocardiography	+Ve	-Ve
	52	25
	23	125

+Ve = Right ventricular Infarction
-Ve = No Right ventricular Infarction

DISCUSSION

The present study of right ventricular infarction Echocardiographic evidence among patients with inferior wall myocardial infarction in the south Karnataka population. In the comparison of detection of right ventricular infarction by echocardiography and the presence of Kussmaul's sign, 75 patients were +Ve (they had an infarction of the right ventricular wall) and receiving 150 did not have a right ventricular infarction [Table 1]. In comparison of echo graphic parameter profiles in both groups, except LVED (left ventricular and diastolic dimension) and LV (left ventricular end diastolic dimension), all the parameters have significant p values (p<0.001) [Table 2]. In the comparative study of detection of right ventricular infarction by echocardiography and electrocardiography, 75 patients had right ventricular infarction and 150 had no right ventricular infarction [Table 3]. These findings are more or less in agreement with previous studies.[5-7]

Diagnosis of RV infarction by physical examination depends on the triad of hypertension, venous distention, and clear lung fields in the setting of inferior wall myocardial infarction. Hypovolemia, or hypervolemia, also creates problems in the diagnosis by physical examination.

The risk factors were smoking, hypertension, diabetes mellitus, obesity, and clinical presentation with RVMI, which had 7 to 8.5% of hospital deaths. Advanced age was found to be a risk factor in the present study.[8] It is also reported that a patient with right ventricular infarction who also had a complete heart block presented with syncope. Such episodes of syncope are classically described as stokes Adams attack.[9]

In right ventricular infarction, right chest leads recognition of infarction signals is easy because only the presence of ST elevation of 1 mm or more is required to diagnose right ventricular infarction, but inferior wall leads a difference between ST elevation in two leads must be interpreted. Thus, right

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precordial leads appear to be a better indicator of right ventricular infarction (RV).[10]

The RV has unique characteristics that make it less susceptible to MI. As the RV myocardium is thin and has a lower oxygen requirement, the LV ECG is mandatory for the investigation of MI. ST segment elevation ≥ 1 mm correlates with proximal RCA occlusion and strongly predicts hospital mortality. In an echocardiographic study, RV dilatation, RV free wall impairment, diastolic reversed septal curvature, systolic paradoxical motion, and decreased tricuspid annular plane systolic excursion (TAPSE), which correlates with RV ejection fraction and is an excellent measure of RV systolic function, Hence, an echocardiographic study is an ideal tool to diagnose and confirm RVMI. It is reported that inferior wall MI patients who also have RV myocardial involvement are at risk of death, cardiogenic shock, and arrhythmia. The inferior wall MI patients treated with volume replacement because they are hemodynamically unstable should be managed with volume loading to maintain adequate RV preload, as preload determines RV output. Diuretics must be avoided even in the absence of hypotension, as this will lead to a worsening of symptoms. A spectrum of disease, from asymptotic mild right ventricular dysfunction to cardiogenic shock, has been recognized in RVI. All patients with RVI of the inferior wall had V4R and V5R, which had the highest sensitivity. This diagnosis can be further confirmed by echocardiography for RV dilatation, thus helping to prevent complications and mortalities.[11]

CONCLUSION

In the present study, it is concluded that right ventricular myocardial infarction is common after MI, occurring in about one-third of the population. ECG and echocardiography are the gold standard techniques for detecting RVMI apart from physical signs and Troponin-T elevation. This study demands that such clinical trials be conducted in a large number of patients in high-tech cardiac centers to confirm the present significant results because the factors that initiate ischemia and infarction are still unclear as coronary arteries are functional end arteries.

Limitation of study: Owing to the tertiary location of the research center, the small number of patients, and the lack of the latest techniques, we have limited findings and results.

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