"CLINICAL AND ELECTROCARDIOGRAPHIC PROFILE OF INFERIOR WALL MYOCARDIAL INFARCTION WITH RIGHT VENTRICULAR INVOLVEMENT– ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY"

By

REG. NO.: BG0118011

Dissertation

Submitted to the
KAHER, Belagavi, Karnataka

In partial fulfillment
of the requirements for the degree of

DOCTOR OF MEDICINE (M.D.)
In
GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE

J. N. MEDICAL COLLEGE, NEHRU NAGAR,

BELAGAVI-590010

APRIL 2021

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

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LIST OF ABBREVIATIONS USED

IWMI	–	Inferior Wall Myocardial Infarction
RVMI	–	Right Ventricular Myocardial Infarction
RV	–	Right Ventricle
LV	–	Left Ventricle
ECHO	–	Echocardiography
BP	–	Blood Pressure
JVP	–	Jugular Venous Pressure
ECG	–	Electrocardiography
RCA	–	Right Coronary Artery
PTCA	–	Percutaneous Transluminal Coronary Angioplasty
AWMI	–	Anterior Wall Myocardial Infarction
ECMO	–	Extracorporeal Membrane Oxygenation
IABP	–	Intra Aortic Balloon Pump
NLR	–	Neutrophil Lymphocyte Ration
PLR	–	Platelet Lymphocyte Ratio
CHB	–	Complete Heart Block

ABSTRACT

Background and objectives

This study was aimed to characterize, the clinical and ECG features of patients who present with ECG findings suggestive of IWMI with RV involvement. Due to the differences in management protocols for patients with RVMI in comparison to those without, delineation of features suggestive of the same would prove beneficial.

Methodology

This one year cross sectional study was done from January 2019 to December 2019 in the Department of General Medicine, KLE's Dr. Prabhakar Kore Hospital and MRC. 40 patients who had the ECG findings suggestive of IWMI with RVMI were enrolled and their clinical characteristics were observed, along with a recording of ECG patterns and other laboratory parameters.

Results

Hypotension and raised JVP were the most commonly observed clinical findings. In the 12 lead ECG recordings, the concomitant presence of ST Elevation in Lead V1 and ST Depression in lead V2 were found to correlate statistically with the presence of RV Dysfunction. The presence of ST elevation in lead V4R was found to be the most consistent finding on the Right Sided ECG Leads. The occurrence of arrhythmias was common, and their presence correlated with mortality in the study population. Fluid resuscitation and inotropic support was provided to the larger majority of patients in the study population.

Conclusion and interpretation

Clinically, the subset of patients with IWMI and RV involvement present most commonly with hypotension. Raised JVP is a consistent finding in these patients. The patterns of ECG changes observed had good correlation with echocardiographic assessment of RV dysfunction.

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INTRODUCTION

Acute Coronary Syndrome (ACS) is an umbrella term, applied to patients in whom there is a suspicion or confirmation of myocardial ischemia or injury. It includes Non ST elevation MI (NSTEMI), ST elevation MI (STEMI) and Unstable Angina. It is caused by the sudden reduced flow of blood to the heart muscle. It is one of the leading causes of morbidity and mortality worldwide. The Asia-Pacific region, accounts for a significant proportion of the global burden of morbidity and mortality associated with ACS. This spectrum of diseases also imposes a significant economic burden on households. Survey data indicate that in India, the household health expenditure went up by 16.5% when one or more adult members of the household had cardiovascular diseases[1].

Coronary syndromes, and their association with sudden death are findings which date back to antiquity. They have been the subject of studies by experts from various disciplines throughout the history of human civilization. “Angina Pectoris” was a term coined by William Heberden, from the Greek ankhonē which means “strangling” and the Latin pectoris, meaning “chest “in 1768, in a paper titled “Some Account of a Disorder of the Breast”. But despite the recognition of the symptom of angina, and the description of atheromas in autopsy specimens, such as the description by the Italian anatomist GiovaniMorgagni, who described the lesions as “Hardening of Arteries”, physicians failed to correlate the pathological findings with the clinical symptoms. And as such, it remained as mainly a disease only of pathological interest.

William Osler was one of the first, to indicate that angina might not be a disease in itself, rather might be the component of a syndrome. It was in 1856, that Rudolf Virchow described the cellular basis of venothrombosis in pulmonary

embolism. It was only following this, that the implications of the pathological findings which had been described earlier began to be taken seriously. And in 1879, the pathologist Ludwig Hektoen, concluded that myocardial infarction was caused by coronary thrombosis, secondary to sclerotic changes in the coronaries. The pathological entity had finally received clinical correlation.

The history of myocardial infarction and the acute coronary syndrome is extensive, but up until the 20th century, myocardial infarction was thought of to be an entity confined to the left ventricle. The clinical consequences and the hemodynamic effects of the right ventricular infarction was largely unknown, with it being considered merely as a pathological entity. The first reports of the hemodynamic consequences of right ventricular infarction was published in 1974[2]. Several unique characteristics of the right ventricle accounts for the lower prevalence and recognition of right ventricular infarctions.

The recognition of the unique hemodynamic nature of right ventricular infarction, highlighted the need for its recognition and consideration, during the treatment of myocardial infarctions, because of the differences observed in the clinical presentation. The occurrence of isolated right ventricular infarction has been reported in autopsy specimens, but with an incidence of only 3% of all myocardial infarctions[4]. The incidence of right ventricular infarctions associated with inferior wall infarctions have on the other hand shown to be as high as 30%-50%[3]. Thus it is important to consider Right ventricular infarction in any case of Inferior wall myocardial infarction.

The clinical profile of right ventricular infarctions varies, not just in the hemodynamic profile, but also in terms of the increased incidence of arrhythmias[5].

Right ventricular involvement has also been found to be an independent predictor of morbidity and mortality in cases of ACS. Patients with right ventricular involvement have been found to have slower recovery and increased mortality[5]. This serves to add to the already heavy burden of ACS on the quality of life.

The differences in clinical presentation, also had therapeutic implications. Mandating that the suspicion of Right Ventricular involvement be entertained in all cases of inferior wall myocardial infarctions. The recognition of Right ventricular involvement is aided by clinical signs like hypotension, an elevated JVP, the presence of pulsusparadoxus and the presence of the Kussumal's sign. But none of the clinical signs of Right ventricular involvement are pathognomic. Hence the burden of diagnosis rests on the typical findings on the electrocardiogram, both the traditional 12 lead ECG and an ECG with right sided leads in place. An ECG with right sided precordial leads, can show extensive ST elevations in all the pre cordial leads from V1R to V6R. A sole ST segment elevation in lead V4R >1.0 mm is a reliable marker of an RV infarction, with 100% sensitivity, 87% specificity and 92% predictive accuracy [6, 7].

In view of the differences in management strategies suggest the need to identify this entity, which had hitherto been considered to be a rarity. Thus a study detailing the clinical profile and the specific electrocardiographic characteristics of patients with inferior wall myocardial infarction with involvement of the right ventricle, would help in better identifying these patients and according them the appropriate therapeutic options.

OBJECTIVES

The objective of this study is to analyze the clinical and electrocardiographic profile of patients presenting with the ECG signs of Inferior Wall Myocardial Infarction with involvement of the Right Ventricle.

REVIEW OF LITERATURE

Acute Coronary Syndrome, is one of the leading causes of death worldwide, and in India. The burden of non-communicable disease burden has been escalating in India over time. With a trend of earlier onset of NCDs, by almost a decade at >45years being observed in the Indian population, adding to the still persistent and significant communicable disease burden on the health care system[8]. As a part of the Global Burden of Disease, Risk Factors and Injuries (GBD) Study, in 2017 the Indian State-Level Disease Burden Initiative CVD Collaborators, analysed the prevalence and DALYs (Disease Adjusted Life Years) due to cardiovascular diseases and the major causes, during the period from 1990-2016[9]. Compared to a 15.2% contribution to total deaths and 6.9% of total DALYs in 1990, cardiovascular diseases contributed 28.1% of deaths and 14.1% of total DALYs in 2016.

Historically, myocardial infarction has been described as an affliction of the left ventricle, and the hemodynamic picture that has been described is predominantly of left ventricular involvement. Right ventricular involvement on autopsy specimens had been noted, but the consideration accorded to the entity was merely pathological. It was in 1974, in a paper detailing the clinical and hemodynamic profile of 6 patients who had acute myocardial infarction with predominant features of right ventricular involvement, that Cohn et al. provided an insight into the constellation of features that might accompany the infarction of the right ventricle. The involvement of the right ventricle was significantly associated with inferior wall infarction, in up to one third to half of the cases. And the same was found to be an independent predictor of hemodynamic instability and in hospital mortality in patients of inferior wall MI[10]. The outcomes in patients with RVMI were poorer, secondary to electrical and

hemodynamic disturbances. Therein lies the need for its early recognition and appropriate treatment.

1.1 Pathophysiology

Early experimental models by Star et al, suggested that the right ventricle could effectively function as a mere conduit[11]. Thus, there was a prevalent belief that RV contraction was unimportant for the maintenance of circulation. This belief persisted until the publication of the report by Cohn et al. Global RV performance is determined by the RV free wall (RVFW) which is perfused by the RV branches of the Right Coronary Artery (RCA). Thus the main culprit artery that has been implicated in the RCA occlusion with disruption of flow to the RV branches.

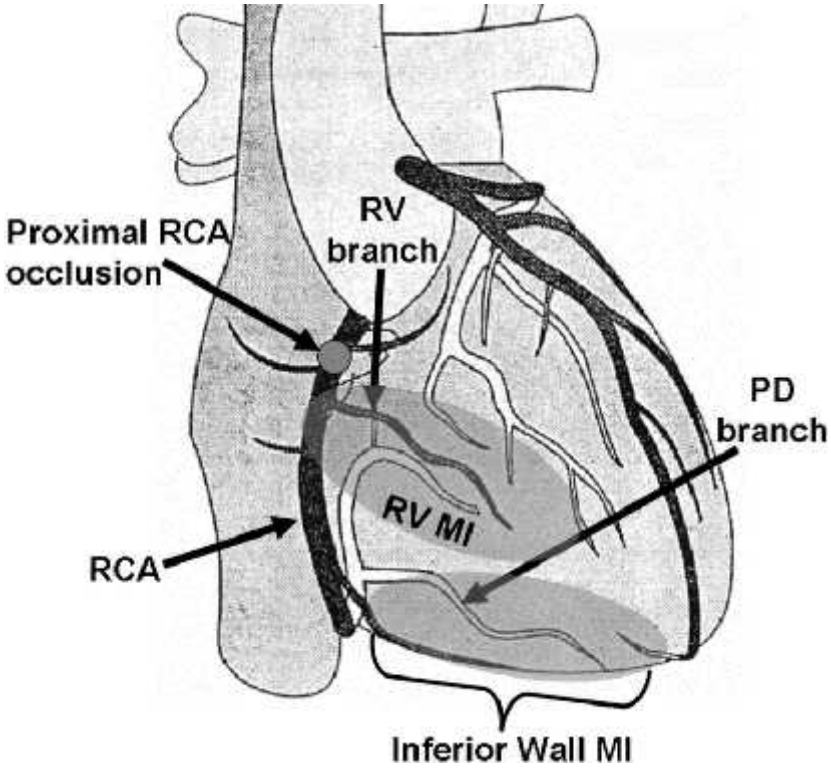

Figure 1: Blood Supply to the Inferior wall and the RV, Showing the culprit lesion for RVMI

The term RV infarction has been considered to be a misnomer to some extent, due to the stark differences in hemodynamic responses to ischaemic insult to the right ventricle, when compared to the same in case of left ventricular involvement[12-13]. Most patients represent spontaneous recovery of hemodynamic dysfunction and a recovery of the RV function, which occurs at a later juncture in time, with the incidence of chronic heart failure stemming from RV infarction being extremely rare[12]. Thus the myocardium despite the infarction is believed to remain viable.

Under normal physiological conditions, the RV contraction is generated by the contraction of the RVFW, generating a peristaltic wave of contraction from the apex to the outflow tract towards the septum. The septum is an important component, both architecturally and mechanically even under normal physiological conditions[12]. Compromise of the RCA circulation, results in RVFW dyskinesia and a severe reduction in RV global performance.

1.1.1 Ventricular Interdependence

It has been well documented that there occurs a decrease in LV function, both systolic and diastolic, in response to an acute RV failure. This phenomenon has been called the, "Ventricular Interdependence". The exact mechanism that underlies this process has been a question of debate.

The RV systolic dysfunction, leads to a diminished pre load delivery to the LV thereby reducing the cardiac output, despite adequate LV contractility. The previous notion of the RV acting merely as a conduit, were based on the assumption that, in spite of the absence of adequate RV contraction a pressure gradient could be generated from a congested systemic venous system and RA contractions[14]. But, later studies showed that, it is the interventricular septum that accounts for approximately

1/3rd of the systolic stroke work, even in normal physiological conditions. When RVFW function is compromised, a paradoxical inward motion of the septum into the RV is responsible for generating the RV systolic pressure.Decreased compliance of the RV, coupled with decreased contractility results in a diastolic pressure rise. This diastolic dysfunction is transmitted to the LV, probably by the inward movement of the septum. But recent studies on animal models by Chua et al in 2013, have suggested that an alteration in septal strain patterns, rather than mechanical obstruction could be the contributory factor for this phenomenon[15].

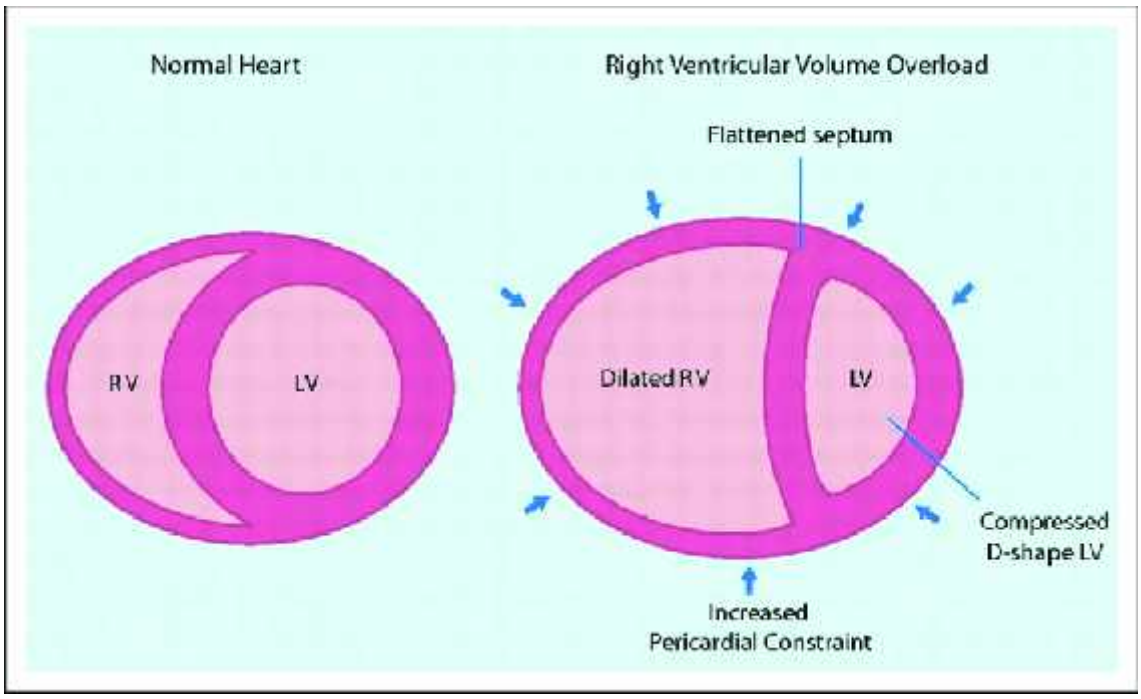

Figure 2: Mechanism of Ventricular Interdependence

1.1.2 Augmented RA Contractions

The RA plays a significant role in the hemodynamic changes associated with RVMI. The contractility of the same is segmented in an attempt to normalize the output of the right ventricle. A stiff and non-contractile right ventricle, imposes the

stress of increased pre load and after load on the right atrium, which enhances the RA contractility. This compensatory optimisation of RV performance is observed in cases of RVMI with intact RA perfusion. With more proximal occlusions, the branches to the RA are involved and this causes ischaemic depression of the RA function. The loss of the augmented RA contractility adversely affects the hemodynamic profile of RV infarctions[13]. Ischaemic atrial involvement has been noted to be more frequent on the right side than the left, found in upto 20% of RVMIs[16]

1.1.3 Oxygen Supply Demand

The oxygen supply demand profile of the Right Ventricle is more favourable than that of the left ventricle.

1. The myocardial mass of the RVFW is lesser than that of the left ventricle
2. The RV is subjected to a reduced preload as well as afterload as compared to the left ventricle

These two factors combine to result in a reduced myocardial oxygen demand of the right ventricle. This results in reduced oxygen extraction by the RV at rest, and gives the ventricular myocardium greater capability of extraction reserve during stress[17].

The RVFW also receives perfusion that is nearly homogenous during both systole and diastole. The underlying factors facilitating this are the reduced thickness of the RVFW, and lower pressures during systole and diastole[18].

The likelihood for development of collaterals to the RCA is more[19], and this along with other variables in the circulatory pattern, allow for a greater anatomic reserve capacity in maintaining perfusion.

These characteristics, both physiological and structural are contributory to the observed rarity of occurrence of right ventricular infarcts and the speedier recovery in case of such an occurrence.

2. Clinical Features

There does not appear to be any pathognomonic clinical signs that accurately predict the occurrence of right ventricular involvement in inferior wall myocardial infarction. But as a hemodynamic consequence of right ventricular infarction, hypotension has been seen as a frequent occurrence in patients who have involvement of the right ventricle[2].

Other clinical signs suggestive of right ventricular infarction have been noted in several studies, including, elevated jugular venous pressure with the concomitant presence of clear lung fields, Kussumal's sign and a tricuspid regurgitation murmur[2021]. In a study conducted by Sinha et al. in 1998, it was observed that hiccups and giddiness were common symptoms. A possible explanation for these findings were proposed to be the activation of the BezoldtJarisch reflex[22].

No observable difference was noted in the occurrence of other symptoms suggestive of acute coronary syndrome, in patients with RVMI compared to those without it.

3. **Electrocardiography**

The 12 lead electrocardiogram is of vital importance in diagnosing acute myocardial infarction. The standard 12 lead system consists of electrodes placed at different points on the body that records the electrical impulses originating from the heart. Any flow of current towards a positive electrode is recorded as a positive

deflection, while the corollary holds true for electrical activity that is towards a negative electrode[23].

3.1. **The ECG in Acute Coronary Syndrome**

The ECG remains the only modality that remains capable of diagnosing STEMI. It defines the timing of onset of the coronary event and can also help in localizing the culprit lesion. The ECG also remains one of the most important modalities that helps in prediction and identification of several potential complications of ACS.

Complete occlusion of an artery by a thrombus, in the absence of adequate collateral flow causes irreversible necrosis within 6 hours of arterial occlusion. The necrotic changes during the first 6 hours are not visible microscopically, and do not incite an adequate response in cardiac biomarkers. But ECG changes are dramatic during this period and serve as the primary modality for diagnosis of acute STEMI[23].

Of the ECG changes to occur, hyperacute T waves overlying the areas of ischemia. This is concomitantly accompanied or followed immediately by ST segment elevation. The presence of symptoms of chest discomfort in concordance with ST segment elevation is suggestive of an acute process.

In acute IWMI, the ST elevations are seen in the leads II, III and aVF. The ECG of these patients can be further characterized by the presence or absence of changes are not seen in upto 50% of patients with IWMI[24]. The presence of these reciprocal changes was found to have a correlation with mortality, an in hospital mortality rate of 3.2% in those patients who did not have reciprocal changes was noted when compared to 4.7% in those patients with reciprocal changes. Additionally,

the patients with reciprocal changes have been found to have larger infarctions and an increased frequency of complications post infarction[25].

3.4.1 The ECG in RVMI

The involvement of RV in IWMI can be detected in the ECG, by using the right sided precordial leads in addition to the standard 12 lead ECG. Thus there is a recommendation to examine the right sided leads in all patients who have the ECG changes of acute IWMI.

ST elevations in the right sided leads from V3R to V6R are diagnostic of RVMI. Of the right sided leads V4R was the one found to perform the best with regards to sensitivity and specificity[26]. The recommendation for obtaining a right sided ECG holds true even for patients without the hemodynamic features of RV involvement, in view of the pathophysiological differences which contribute to a longer period of hospitalization in those patients who do have involvement of the RV[27].

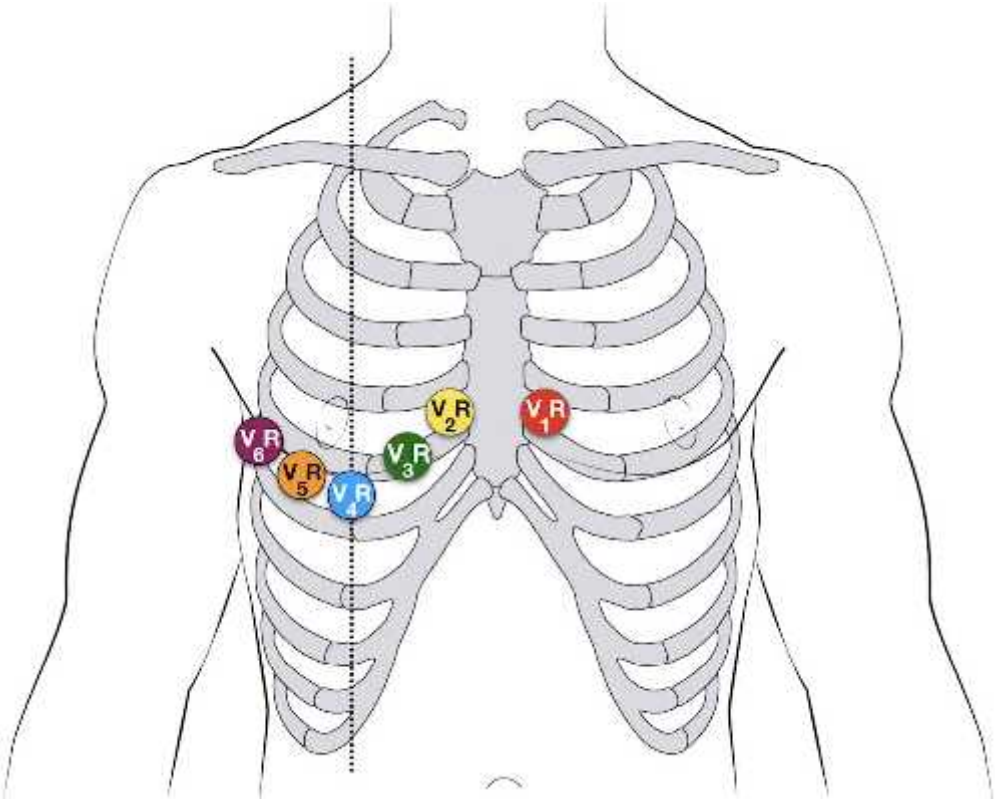

Figure 3: Lead Placement for all Right Sided Leads

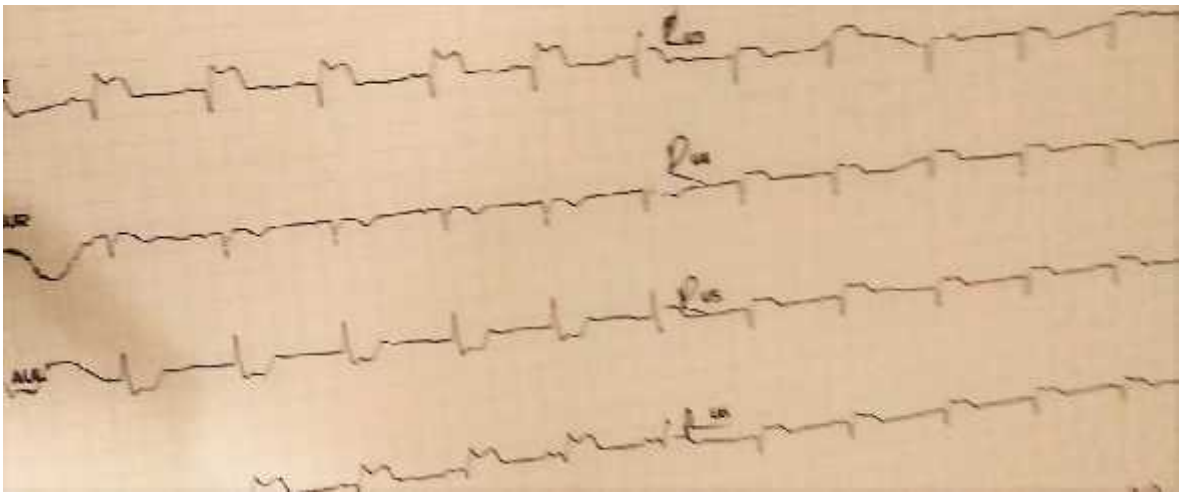

Figure 4: ECG Tracing showing ST Segment elevations in Right Sided Precordial Leads

3.4.2. The 12 Lead ECG in RVMI

The requirement of placement of additional leads to characterize RVMI, prompted some investigators to assess the viability of the 12 Lead ECG and findings

on the same as a surrogate for the right sided leads. A 2005 study by Moye et al, put forth the analysis that ST elevations of greater magnitude in lead III as compared to lead II was suggestive of RVMI[3]. In 2018, Johanna et al, analysed the significance of changes in the precordial leads and in lead 1, with reference to RVMI. In concordance with several other studies they did not find any significance for the presence of ST depressions in lead 1 in identifying RVMI. Some sensitivity was attached to the finding of ST elevation in lead V1, and a possible role for the presence or absence of concomitant ST depression in lead V2 in modifying the sensitivity of the finding[26].

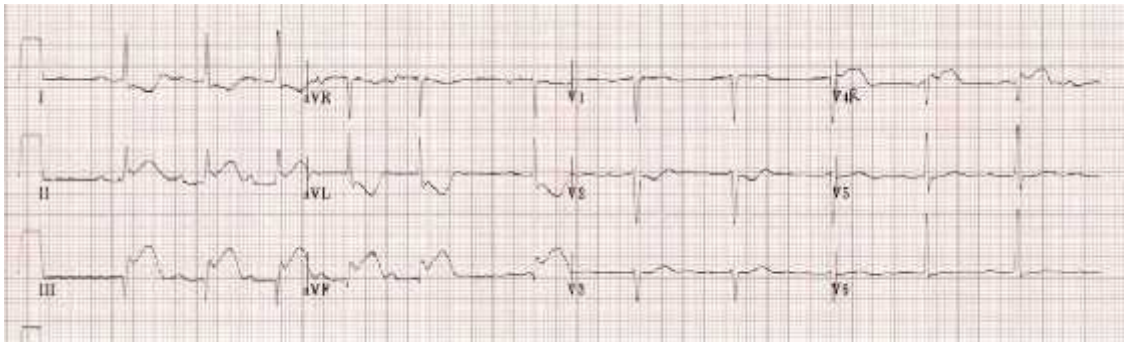

Figure 5: IWMI with RVMI, with V4R along with the precordial leads

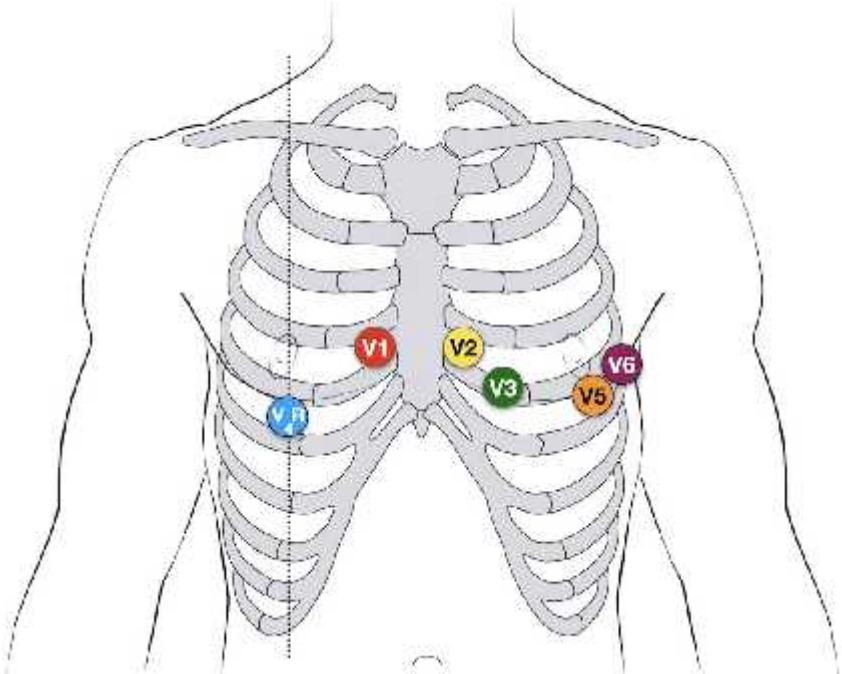

Figure 6: Placement of V4R along with the standard precordial leads.

4. Echocardiography

2D echocardiography is an imaging modality that can demonstrate several features of infarction of the right ventricle. It can aid by both demonstrating the direct effects of RV infarction in RV dyskinesis, as well as the indirect effects to the hemodynamic sequelae of RV infarction. But, the use of echocardiography in the evaluation and diagnosis of RVMI is considered a challenge due to several factors, one of which is the complexity of the RV in a geometrical sense[29], and another occurs in the form of the often transient nature of the RV dysfunction occurring due to ischemia.

The RV is a crescent shaped chamber, which adds its own unique difficulty to the quantification of the chamber size. The systolic function of the RV can be assessed by various parameters, which include among others;

a) RIMP- Right Ventricular Index of Myocardial Performance
b) TAPSE- Tricuspid Annular Plane Systolic Excursion
c) S'- Tissue Doppler of the Free Lateral Wall

Of these aforementioned modalities the TAPSE is most commonly used[30]. The ease of procuring this measurement makes it a viable marker, some limitations do exist in its use, one of which is the fact that it remains a one dimensional measurement. A 2015 recommendation by the European Association of Cardiovascular Imaging has suggested that a TAPSE of <1.7cm would represent RV systolic dysfunction[31].

5. Coronary Angiography

The gold standard for diagnosis of RVMI remains angiographic demonstration of coronary artery blockade. The culprit artery is the right coronary artery, with an occlusion proximal to the RV branches in majority of the cases. The infundibular region is frequently spared due to separate ostia to the arterial branches supplying this area.

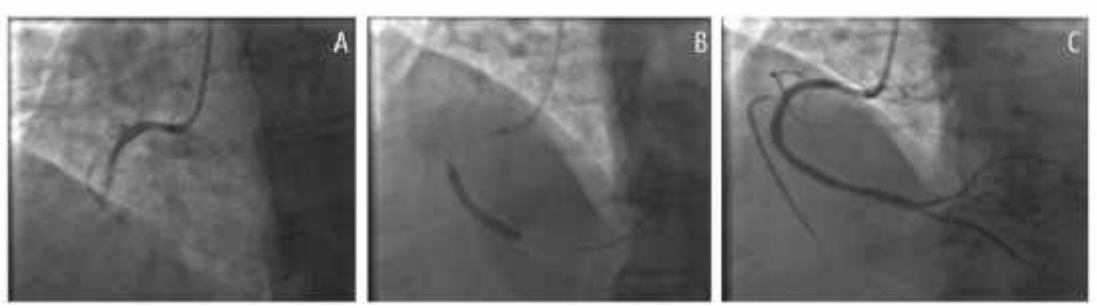

Figure 7: Cardiac Catheterization images

6. Cardiac Imaging

Even in the absence of extensive evaluation, Cardiac Magnetic Resonance remains one of the standard imaging techniques. Late enhancement noted on Gadolinium scans have been shown to have a sensitivity comparable to or better than that of 2D ECHO.

7. Management and Challenges

The therapeutic approaches to RVMI includes the following options[32];

a) Emergent Revascularization

b) Maintaining the adequacy of the RV preload

c) Rhythm optimization

d) Ionotropic or mechanical support

7.1 Reperfusion

The timing and completeness of reperfusion stratagems, either by way of thrombolytic agents or percutaneous revascularization influence the prognosis of patients with RVMI[33]. Incomplete attempts at the same have been associated with worse outcomes with respect to mortality and complications[34]

7.2 Maintenance of RV Preload

An important facet which distinguishes the therapy of RVMI from that of its left sided counterpart is the need for intravenous fluid resuscitation. The need for the same is rooted in the physiology of hemodynamic changes that accompany RVMI[35, 36]. The degree of LV involvement was a significant contributor to the hemodynamic changes in RVMI, hence great care is to be exercised in avoiding overzealous fluid resuscitation[37]. Studies have shown the significant influence of a CVP guided approach to fluid resuscitation, on the process of clinical decision making[38].

7.3. Optimization of Rhythm

The contribution of heart rate to cardiac output is derived predominantly from the heart rate. Hence in this setting maintenance of AV synchrony assumes paramount importance[39]. The requirement of pacemaker insertion, albeit temporary might occur in a number of patients who present with rhythm abnormalities as a consequence of RV infarction, most significantly AV bocks[40]. The procedure helps to maintain the cardiac output in these patients.

7.4. Ionotropic Support

The interdependence of the ventricular systems confers a unique importance to this modality of treatment. Combining ionotropic support with fluid resuscitation serves as a bulwark in our armoury to compensate for the systolic compromise of the RV[41]. Dobutamine, has been found to be an ideal candidate for this purpose. Along

with serving the purpose of augmenting the RV systolic function, the ionotropic agent plays a role in reducing the resistance of the pulmonary vasculature as well as negating the detrimental effects of an increased RV afterload. The utility of Dobutamine is particularly pronounced in cases of RVMI with the involvement of the interventricular septum.

But the propensity for a hypotensive response and a systemic vasodialatory effect coupled with a propensity for arrhythmias could be potential pitfalls restricting the use of this agent.

Milrinone, Levosimedan, Norepinephrine, are other agents which can be used to support the failing RV. Milrinone enables the reduction of the degree of afterloading on the RV, but at the expense of a concomitant reduction of preload and supplementing the deleterious effects of hypotension. Levosimedan has been granted approval only in Europe. It acts via activation of ATP sensitive potassium channels located in the pulmonary vascular bed, enabling a reduction of the afterload. It is a calcium sensitizing agent which has been shown to have efficacy in augmenting the RV contractions[42].

7.5 Mechanical Ventricular Support

In accordance to the degree of support required by the patient, the modalities of mechanical support that can be used are as follows;

1- Direct RV Support

2- Indirect RV Support

3- Biventricular Support

7.5.1. Direct RV Support

Several devices are commercially available for this purpose, of these the sole device currently having approval for this indication is the Impella RP[43]. The RECOVER – RIGHT trial had a cohort of 30 patients who were treated with the device, and a 83.3% positive outcomes were noted in those who received the device based mechanical support[44]. Other devices available are the TandemHeart or the ProtekDuo, these have the additional benefit of being capable of being outfitted with an oxygenator, which could prove efficacious in cases of hypoxemia.

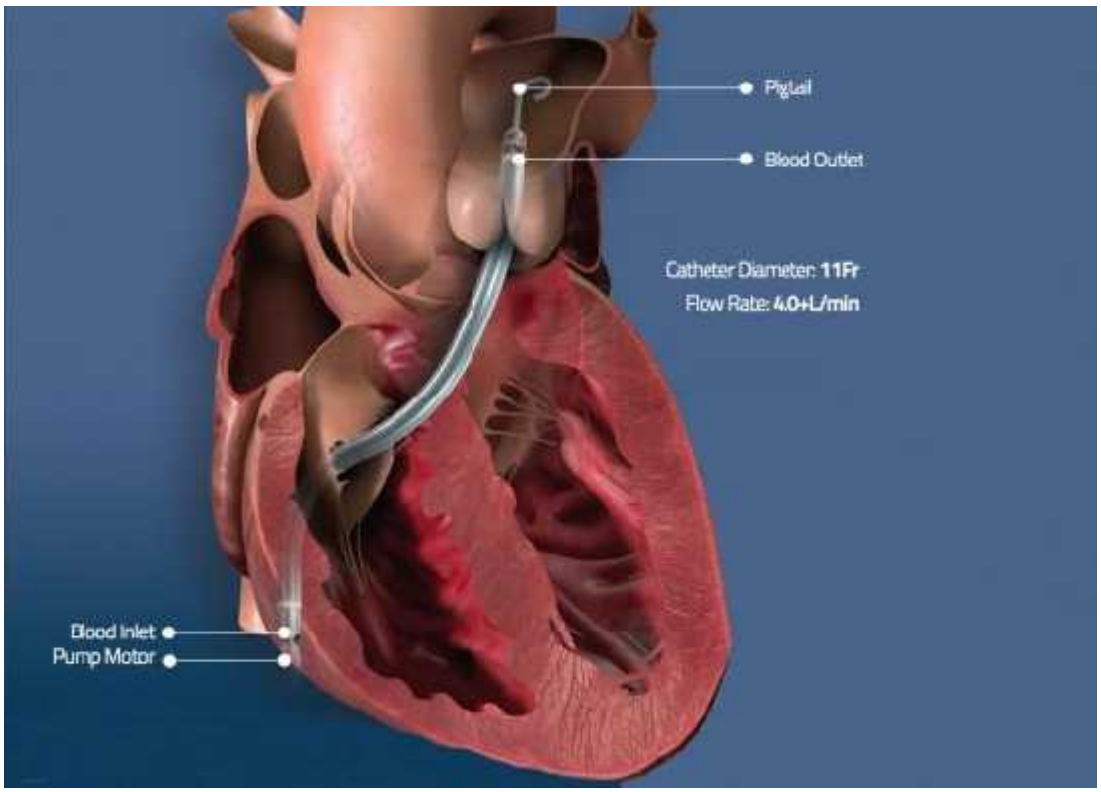

Figure 8: Placement of the Impella RP device.

7.5.2. Indirect RV Support

The means for providing indirect RV support which are currently in use are the ECMO[45] and IABP.

The use of the ECMO device can be detrimental in conditions of biventricular failure, where the supplemental effects of an increased LV afterload would have adverse consequences. In such situations the VA ECMO can be considered in conjunction with the use of another modality such as the IABP or the ImperllaRP. A 2014 study by McNamara et al, on the impact of IABP on hypotension and outcomes in RVMI, found that the use of IABP produced a survival rate of 81%[46]. The reduction of LV afterload with IABP, would of particular benefit in patients who also have concomitant LV failure.

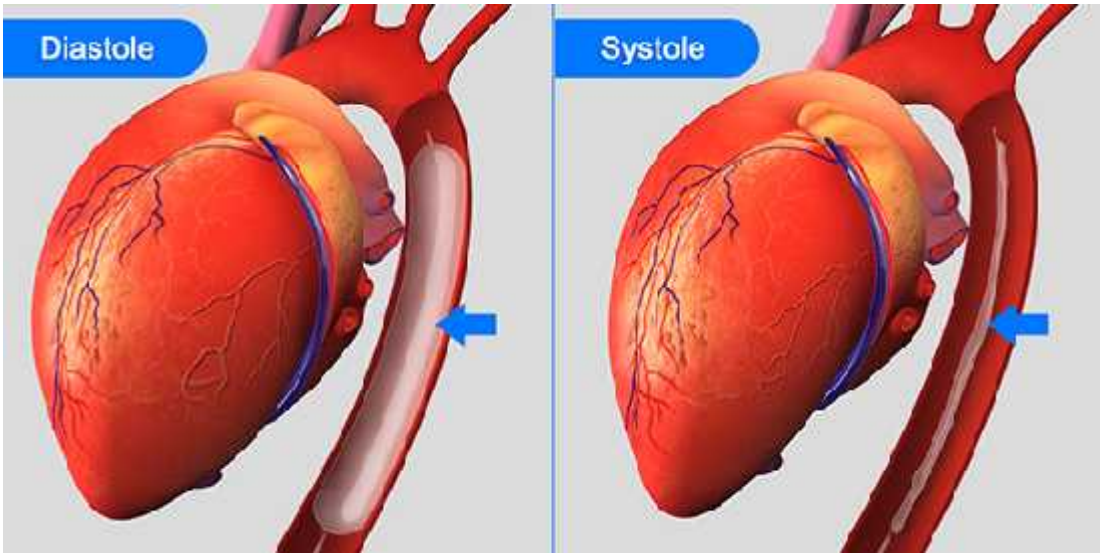

Figure 9: IABP Device, placement and action

7.5.3. Biventricular Support

LVAD or Direct RV support with indirect means of support used in combination might prove to be of benefit in certain sub populations of patients[47]

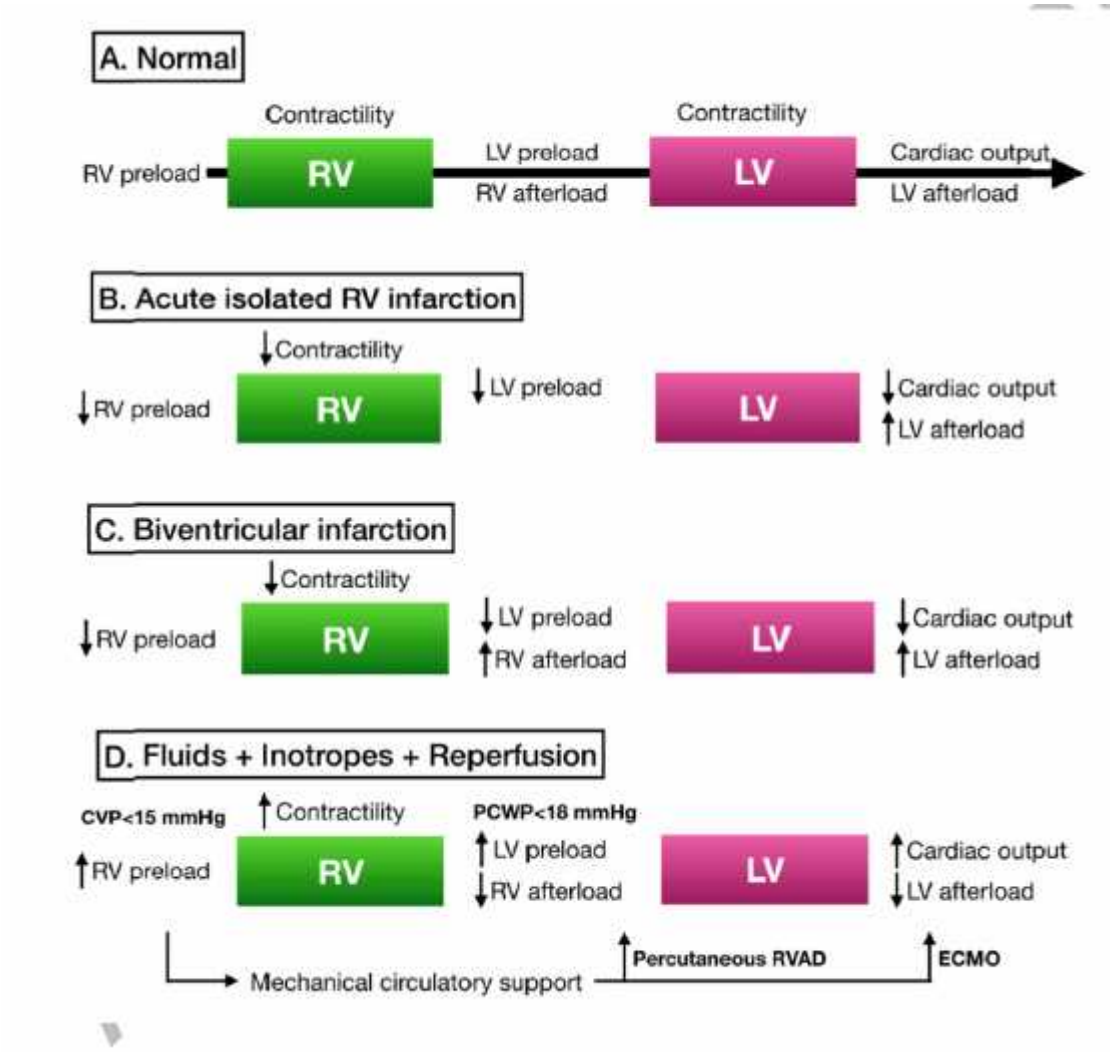

Figure 10: Schema of Hemodynamic Changes in RVMI and effects of treatment

7.6 Complications

Several investigators have, over the years, outlined and characterized the myriad complications that occur in cases of RVMI. These complications occur both as a consequence of the hemodynamic properties of RV compromise and due to the alterations in blood flow itself. The presence of conduction abnormalities was a

common occurrence in cases of RV involvement, due to aberrations in blood to the AV nodal arteries. The occurrence of high degree AV blocks was documented at a higher frequency and there was an association established with prognosis.Other arrhythmias were also documented, including ventricular tachycardias and atrial fibrillation. One possible mechanism of atrial fibrillation was the concomitant distention of atria. Subsequently other complications have also been described, including ventricular septal rupture and tricuspid regurgitation.

Complications of RVMI
1. Atrioventricular blocks
2. Arrhythmias (bradyarrhythmia or tachyarrhythmia)
3. Vasovagal symptoms
4. Hypotension
5. Cardiogenic shock
6. Ventricular septal defect
7. Pericarditis +/- pericardial effusion
8. RV thrombus
9. Tricuspid regurgitation
10. Pulmonary hypertension
11. Right heart failure
12. Atrial fibrillation

8. Prognosis

RV involvement serves as a predictor of mortality, independent of other factors[48]. And the most significant contributor to the adverse outcome was the presence of refractory cardiogenic shock. There was also an association with a multitude of in hospital complications including arrhythmias.

But, post discharge the patients of RVMI had relatively good prognosis. And as has been observed in studies, the RV functional eventually returns to normalcy in a larger proportion of patients[49].

METHODOLOGY

Study site

This study was conducted in the Department of General Medicine, KLE's Dr. Prabhakar Kore Hospital, Belagavi.

Study designand duration

The current study was a one year Cross-sectional study.

Study period

The study was conducted from January 2019 to December 2019.

Study population

All admitted cases of ECG proven Inferior Wall Myocardial Infarction, with concomitant right ventricular infarction were considered in the study population.

Sample size

The study included a total of 40 patients of Inferior Wall Myocardial Infarction with Right Ventricular Infarction.

Sampling procedure

The sample size was calculated by the followingformula:

Sample size (n) = $4\ PQ/D^2$

P = Prevalence of the disease

Q= 100- P

D = Absolute error taken as 15%

(P = 30; Q = 70; D=15)

$n = 4 \times 30 \times 70 / 15^2$

$n = 37.33 \approx 40$

All eligible patients were recruited in this study consecutively by convenient sampling till the sample size was reached.

Selection criteria

Inclusion Criteria

- All ECG proven cases of Inferior Wall Myocardial Infarction with Right Ventricular involvement, above the age of 18 years who were admitted in KLE's Dr Prabhakar Kore Hospital and MRC

Exclusion criteria

- Old cases of myocardial infarction
- Concomitant Involvement of the anterior wall
- Patients with a known history of COPD

Ethical clearance

The study was approved by the Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belagavi prior to the commencement.

Informed consent

The patients fulfilling the selection criteria were briefed about the study and those who expressed their willingness to participate in the study were enrolled after obtaining a written informed consent (Annexure-I).

Data collection

On admission, the demographic data of the patients along with relevant history of current illness and past medical history were documented. Further these patients underwent clinical examination followed by investigations.

Investigations

Patients were subjected to following investigations.

- A standard 12 Lead ECG
- ECG using the Right Sided Precordial Leads
- 2D Echocardiography
- Hemogram- Haemoglobin, Neutrophil Lymphocyte Ratio, Platelet Lymphocyte Ratio
- Renal Parameter- Serum Creatinine, Serum Sodium, Serum Bicarbonate

Procedure

At the time of admission, patients were assessed for symptoms and signs of Acute Myocardial Infarction.

The diagnosis of Inferior Wall Myocardial Infarction with Right Ventricular involvement was made on the basis of:

1. Symptom profile characteristic of acute coronary syndrome.
2. ST segment changes in Leads 2, 3 and VF
3. ST Elevation greater than 1mm in at least one of the right sided leads from V1R to V6R.

All relevant data for patients who fit the eligibility criteria was recorded in a structured proforma.

Statistical methods

The data obtained was coded and entered into Microsoft excel spreadsheet and data was analyzed using SPSS version 21 and Statistica 12. The categorical data was expressed in terms of rates, ratio and percentage and the continuous data was expressed in terms of mean ± standard deviation. The association between the outcome, clinical and demographic characteristics was tested using chi-square test. Co-relation among the continuous and categorical variables were compared using Spearman's rank method. A probability (p) value of ≤ 0.05 was considered as statistically significant when the confidence interval is 95%. To nullify the impacts posed by small data sets, confidence interval is varied till 80%, and thus the corresponding p-value is chosen for appropriate comparison.

DISCUSSION

The involvement of the Right Ventricle in Acute Myocardial Infarction, was an entity which received recognition much later, due to the characterization of MI as being a predominantly Left Ventricular entity. But ever since its recognition, the involvement of the RV in myocardial infarction, has been associated a more protracted hospital course due to a variety of associated complications.

The treatment of IWMI associated with RVMI also varies from others. Hence there is a need for characterization of this entity by itself.

The present study aimed to determine the clinical and electrocardiographic characteristics of patients presenting with IWMI with RV involvement.

Age and Gender

The majority of patients in the present study fell into the 60-80 years age group, with 22 patients 50% falling into this age bracket. The oldest patient was 85 years old and the youngest patient was 29 years old.

The mean age of the study population was 59.23 years SD 13.0.

The study population was divided among 65% males and 35% female patients. The most common age group among male patients was 50-60 years. Maximum number of patients fell into the 60-70 years age group among females. The mean age among female patients was 65.21 years with SD 10.02. The mean age among males was 59.23 years SD 13.90.

No correlation between the severity of symptoms and outcome with either the age or gender was demonstrated in the present study.

This absence of correlation goes against the findings of a study by Bueno et al in 1997, wherein an increased mortality was demonstrated in the elderly population[50]. And yet another 2017 study by Obradovic et al, which found an increased incidence of RV involvement in women compared to men in contrast to the present study[51]. But these findings were in concordance with a 2013 study by Asif Iqbal et at, where there was a male preponderance among patients[52].

Risk Factors

Taking comorbidities into consideration, there was an even distribution of hypertensive and non-hypertensive patients.

Considering the gender wise distribution, 13% among the male patients were hypertensive and 8% per of females were hypertensive. 8% per of male patients and 3% of female patients were diabetic in the study population.18 % of male patients and 13% of female patients had both the comorbidities and 28% of male patients and 13% of the female patients had neither.

The other risk factors we had taken into consideration was a history of alcohol consumption and a history of smoking.12 patients, 30% of our study population gave a history of alcohol consumption, all 12 patients were male. 9 male patients were smokers. 3 of these patients had a history of both smoking and alcohol consumption.The present study did not find any statistically significant correlation between lifestyle habits and adverse outcomes.

Symptomatology

The most common symptom occurring in 92% of the patients was chest pain. The mean duration of chest pain in our patients was 6.46 hours +/- 3.74 hours. 100% of the male patients and 87.5% of female patients had presented with chest pain.The second most common symptom was diaphoresis which occurred in 75% of patients 69.23% male and 85.71% Syncope occurred in 2per of our patients. 15.38% in males and 21.42% in females.Chest pain was also present in all patients included in a study conducted by Asif Iqbal et al in 2013, but the same study also reported a higher prevalence of syncope as a symptom among patients of IWMI with RVMI[52]. There were no significant differences observed in the symptomatology of patients presenting with infarctions of the Right Ventricle and those without with reference to the symptom complex consisting of chest pain, diaphoresis and palpitations in an earlier study done by Sinha et al in 1988[53].

A more recent study done in 2005 by Chockalingam et al, showed a similar percentage of syncope occurring in patients of RVMI to that seen in the present study[54].

ECG Characteristics

All patients included in the study had the classical ECG signs of inferior wall MI, which is ST Elevations in leads 2, 3 and aVF. In addition we looked at the findings on the Right sided leads V3R, V4R, V5R and V6R. Additional findings on the standard 12 lead ECG which were taken into consideration were:

1- ST Depression in Lead 1

2- ST Elevation in Lead V1

3- ST Depression in Lead V2

4- Concomitant presence of ST Elevation in Lead V1 and ST Depression in Lead V2

5- The presence of ST Elevation in Lead 3 greater than that in Lead 2

Among the right sided leads, the most consistent finding was ST Elevation in Lead V4R, occurring in 100% of the patients.

The second most common observation was ST Elevation in lead V5R occurring in 77.5% of the patients. A combination of ST Elevation in Lead V3R and Lead V4R was seen in 62.5% of the study population, while 47.50% had ST elevations in all the right sided leads.

The combination of ST Elevation in Lead V3R and V4R was found to have statistically significant correlation with the presence of RV Dysfunction on 2D ECHO p value 0.014.

The frequency of occurrence of ST elevation being the maximum in lead V4R was in concordance with studies conducted by Chhapra et al in 2013[55].

The ECG patterns, in addition to the ST Elevations in lead 2,3 and aVF observed in the standard 12 lead ECG were, ST Depression in Lead 1 in 85% of patients, ST Elevations in V1 in 57% of patients. 77% of the patients observed had ST elevations of greater magnitude in Lead 3 than in Lead 2.

Moye et al in a 2005 study had stated that the presence of ST elevation in Lead III more than that in lead II was highly suggestive of RVMI, and this was in concordance with our findings. Evidence for the presence of ST depression in lead I signifying a RCA occlusion was shown by Chia et al in a 2000 study[56].

There was no statistically significant correlation between the presence of ST Elevation in Lead V1 and the presence of RV Dysfunction on 2D ECHO. But when the ST Depression in V2 was considered in combination with ST Elevation in V1, the presence of both in combination had a significant correlation with the presence of RV dysfunction with a p value of, 0.007.

Multiple studies have shown that a concomitant presence of ST elevation in lead V1 and ST depression lead V2 was sensitive for the detection of RVMI. Bischof et al had tested a hypothesis, wherein the sensitivity of the presence of ST depression in lead V1 was postulated to increase if considered in the absence of ST depression in lead V2, but there was no statistical significance in their study.

Physical Examination

Having set a cut of less than or equal to 100/60mmHg for hypotension, 52 % of the study population was found to fall into that category. 11 patients, i.e 21% of the study population were in the normotensive range and 8 patients had an initial blood pressure recording of greater than 120/80mmHg

72 % of the patients had a raised JVP on examination.

On systemic examination, basal crepitations were observed in 55% of the patients.

The classic triad of symptoms associated with RVMI, which are Hypotension, Raised JVP and Clear Lung fields were observed in only 13 patients, 32.5% of the study population.

These findings were in line with those of Chockalingam et al, Asif Iqbal et al and Chhapra et al, who all observed hypotension to be a common manifestation of IWMI with RVMI, along with raised JVP.

The present study had a larger percentage of patients with basal crepitations than the other studies, but the overall percentage of patients who had the classical triad of symptoms was comparable to the aforementioned studies.

Laboratory Findings

Haemoglobin

55% of the study population was non anaemic, with a mean haemoglobin of 13.054 g/dl SD 1.89 among the male patients and 11.964 g/dl SD 1.345 among the female patients.

No significant correlation could be found with the presence of anaemia and unfavourable outcomes in the study population. In contrast to several larger studies which assessed the risk of baseline anaemia on mortality in patients of STEMI[57].

NLR

The neutrophil lymphocyte ratio was calculated for all patients in study. 37% of patients fell into the 3-6 category, with 33% comprising the 6-9 category. 13% had a NLR greater than 9, while 17% had the NLR within the normal ranges.

There was a positive correlation observed in the present study between the NLR and mortality.Studies conducted by Núñez et al and Akpek et al, among others have in recent years showed a clear relationship between the NLR and in hospital adverse outcomes in patients of STEMI[58-59]. There was a relationship between NLR and the presence of RV Dysfunction demonstrated by Yalak et al, the present study

could not demonstrate a significant correlation in the 95 confidence interval, but was able to do so in the 90 percent confidence interval[64].

PLR

The Platelet Lymphocyte Ratio was also calculated for all patients, the cut off for male patients was a normal range of 36-149, with 62% falling into this range and 38% having a PLR above the cut off. While there was an even distribution among females. No correlation was observed between this value and the eventual outcome. A 2014 study by Kurtul et al, had contrasting results where they showed an association between PLR and the complexity of atherosclerosis[60].

Renal Parameters

The mean serum creatinine in the study population was 1.2595 mg/dl with SD 0.566. The mean value of serum sodium was 134.375 mEq/L with SD 5.708. 45% of our patients had a serum sodium of less than 135.The mean bicarbonate levels were 17.875 mmol/L SD 3.508. The present study could not find any correlation between the bicarbonate levels and the degree of LV Dysfunction or RV Dysfunction.Base excess was shown to be a good prognostic marker in a study by Lazzeri et al in 2010[61], but in the present study was not able to derive such a correlation.With respect to potassium levels, 73% had it within the range of 3.5 to 5 mEq/L. 25% had potassium values greater than 5 mEq/L and 2% had hypokalemia with levels less than 3.5 mEq/L.

Treatment

50% of the patients received ionotropic support. The most common inotrope used was Dobutamine. 1 patient received the combination of dopamine, dobutamine

and noradrenaline. Several comprehensive reviews of patients with IWMI and RVMI have shown Dobutamine to be the inotrope of choice in view of cardiogenic shock[32].

65% of the patients received IV crystalloid support in view of hypotension. Which has again been shown to be a difference in the treatment approach to patients with RVMI compared to those without[32].57% of our patients were thrombolysed, the most common agent used for thrombolysis was Streptokinase.

Complications

53% of the patients had arrhythmias on admission, Sinus tachycardia and 1st Degree AV block were the most common among these patientsComplications were more commonly reported in IWMI with RVMI in several studies done previously 57.65% was observed by Garg et al. Klein et al reported a complicated course in 59 of the patients in their 1981 study.4 hours after admission 61 percentage of our patients had arrhythmias recorded on their ECG tracings. The most common among these patients was sinus bradycardia, followed by 1st Degree AV Blocks and Sinus tachycardia.

A higher percentage of AV blocks was most commonly observed in studies by Chhapra et al and Garg et al.Normal Sinus rhythm was seen to be restored in 68 of our patients 24 hours post admission

2D ECHO

All patients included in the study had varying degrees of LV Dysfunction, the mean EF in our study population was 42.75% SD 4.99. The presence of LV Dysfunction did not have a statistically significant correlation with the hypotension observed in our patients.

75% of the patients had RV Dysfunction on 2d ECHO. TAPSE was the parameter used to asses RV Dysfunction in the present study, the mean TAPSE was 1.28 cm SD 0.12. TAPSE has a high sensitivity and specificity for the identification of RV Dysfunction as demonstrated in a study by Gopalan Nair et al[62]

Outcome

53% of the patients were managed on medical therapy and did not undergo invasive procedures. 32% of the patients underwent successful PTCA. 15% of the study population, 6 patients expired.67% of the patients who expired had hypotension on admission compared to 52% of the whole study population.

Among the patients who expired, none had normal sinus rhythm on the ECG tracing on admission, the most common arrhythmias noted were sinus bradycardia and sinus tachycardia. 4 hours following admission 50% of these patients had sinus bradycardia on their ECG

RV Dysfunction was present in 5 out of the 6 mortalities in our study population. ST elevations in the right sided leads V4R and V5R were present in all 6 patients, so was ST depression in lead V2.

There was a significant association between the presence of arrhythmias on admission and at 4 hours with mortality in our patients. A similar finding was observed by Varun Kumar et al. An older study by Ramires et al, did not find a significant association between mortality and AV blocks[63].

CONCLUSION

The present study attempted to characterize the clinical features and ECG patterns in patients of IWMI with ECG findings suggestive of RVMI. Hypotension and raised JVP were found to be common clinical manifestations. But the classically described clinical triad of hypotension, raised JVP and clear lung fields was not a consistent finding.

The ECG pattern observed most consistently on the special right sided precordial leads was the presence of ST Elevation in lead V4R. Along with the ST elevations on the right precordial leads, specific patterns in the standard 12 lead ECG was also observed, these included ST depressions in lead 1, ST elevation of greater magnitude in lead 3 as compared to lead 2, ST elevations in lead V1 and ST depressions in lead V2. Of these observed ECG patterns, the ST elevations in right sided leads, V3R and V4R alone or all leads from V3R to V6R were found to correlate well with the 2D ECHO proven presence of RV dysfunction. Of the ECG findings on the standard 12 lead ECG, the concomitant presence of ST elevation in lead V1 and ST depression in lead V2 correlated well with the presence of RV Dysfunction.

The clinical course of patients were also complicated by the presence of arrhythmias, with AV blocks and Sinus Bradycardia being observed in majority of the patients. The observed distribution of arrhythmias varied from that on admission to that observed 4 hours after admission. Within 24 hours of admission most of our patients were found to have reverted back to a normal sinus rhythm. But, the presence of arrhythmias on ECG tracings on admission and after 4 hours, did correlate with mortality in our study population.

Among the laboratory parameters observed in our study population, the most significant correlation was found between the NLR and mortality, the same also correlated with the presence of RV Dysfunction, albeit at wider confidence intervals, and larger studies are needed to validate the correlation. Our study could find no correlation between variables such as age, gender or lifestyle habits of patients and adverse outcomes. All of our patients had varying degrees of LV Dysfunction on admission, but the hypotension which was observed in the study population was found to be independent of the degree of LV Dysfunction, highlighting the independent role of RV Dysfunction in causing hypotension in this specific population. The treatment of a large majority of patients, involved the judicious use of inotropic agents, the most preferred being Dobutamine at our center and the use of IV fluids. The ECG patterns observed, were seen to have good correlation with the presence of RV dysfunction and could guide in predicting the same.

The study population was limited to only 40 patients, hence larger studies are needed to validate the significance of this correlation between the echocardiographic confirmation of RV Dysfunction and the specific patterns of ECG changes.

SUMMARY

The present study to characterize the clinical and electrocardiographic features of IWMI with RV involvement was able to summarise the same as follows;

1- Hypotension is a common presentation of this subset of patients with IWMI and RV involvement. And the hypotension was demonstrated to be independent of the degree of LV Dysfunction.

2- Raised JVP was also seen in a majority of the patients in the present study.

3- The classically described triad of findings was not a common finding, and this was in concordance with several other studies.

4- Several of the patterns observed in the ECG tracings in the patient population correlated well with the 2D ECHO assessment of RV dysfunction.

5- Both the 12 Lead ECG as well as the Right sided leads were found to have patterns that correlated with the 2D ECHO findings

6- Patients frequently developed arrhythmias, and the spectrum of arrhythmias had an observable change from that observed on admission to after 4 hours of admission in the present study.

7- The presence of arrhythmias, also had a correlation with a complicated course and mortality in the patients considered under the present study.

8- The present study was not able to show any significant correlation between demographic patterns and outcomes.

9- A correlation between the NLR and mortality was also demonstrated in the present study, which could prove to be a cheap and easily available investigation helpful in risk stratification

10- The present study also attempted to ascertain the correlation between the NLR and the degree of RV dysfunction in the study population. A correlation was

found at wider confidence intervals. Larger studies are required to validate this finding

To summarise, the present study shows that ECG tracings can serve as a good marker for the identification of RV Dysfunction in patients with IWMI and features suggestive of RVMI. Both the Right sided ECG leads and a combination of several patterns in the standard 12 lead ECG could serve this purpose.

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ANNEXURE- I

ETHICAL COMMITTEE CERTIFICATE

K L E ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed to be University)

Accredited 'A' Grade by NAAC (CGPA 3.64) | Placed in Category 'A' by MHRD (India)

JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website http://www.jnmc.edu
E Mail dome_a@jnmc.edu

Phone (+91-0831) Office 2472550
Principal 2471701
Fax No (+91-0831) 2470759

Ref: MDC /DOME/ 19 **Date: 24/11/2018**

To,
REG. NO.: BG0118011

PG student in Medicine,
J N Medical College,
BELAGAVI

Sub: Institutional Ethical Clearance for the study

With reference to the above, we wish to inform you that your proposed research project titled **"CLINICAL AND ELECTROCARDIOGRAPHIC PROFILE OF INFERIOR WALL MYOCARDIAL INFRACTION, WITH RIGHT VENTRICULAR INVOLVEMENT – A ONE YEAR CROSS SECTIONAL STUDY AT KLE'S DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J N Medical College, Belagavi

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J N Medical College, Belagavi

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ANNEXURE II – CONSENT

TITLE OF RESEARCH STUDY: "Clinical And Electrocardiographic Profile Of Inferior Wall Myocardial Infarction With Right Ventricular Involvement– One Year Hospital Based Cross-Sectional Study."

Principal Investigator:

Dr. _____

Post Graduate Student

Department of General Medicine

Jawaharlal Nehru Medical College

Belagavi – 590010

Guide:

Dr. _____

MD (General Medicine)

Department of General Medicine,

Jawaharlal Nehru Medical College

Belagavi – 590010

Introduction and Purpose: -

The present study is conducted among patients with Inferior wall Myocardial Infarction with Right Ventricular Involvement in KLE's Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum and they will be investigated to characterize their clinical features, and the associated electrocardiographic changes

Procedure:

If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood samples for the necessary investigations.

Risk and Benefits:

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations. It may cause swelling, pain, redness (rarely happens) at the site from where the blood is drawn.You may also be benefitted by these investigations and you will be part of this study which is going to be useful to others in the future.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study. If you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy:

Does not apply to this research

Financial incentives for participation:

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results:

The results of the study would be forwarded to the KAHER, Belagavi as part of requirement towards the completion of MD degree, review and publishing.

In case of the queries during study or in future you may contact following persons,

1. Dr. Roopa Bellad,
   Chairman,
   J.N.M.C Ethical Committee
   for Human Research

2. Dr. _____
   MD (General Medicine),
   Dept of General Medicine,
   JNMC, Belgaum.

3.Dr. _____
Investigator,
PG in General Medicine,
JNMC, Belgaum.

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me, and have had all the questions answered.

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name: ……...

Signature / Left thumb impression ……...

of the participant:

Name of the legally authorized ……...

representative / guardian:

Signature / Left thumb impression: ……...

Witness name: ……...

Signature / Left thumb impression: ……...

Investigator's name and signature: ……...

Date:

Place:

‘

ANNEXURE III - PROFORMA

A- Patient Details

Name:		**Age:**		**Gender:**		**IP Number:**	

B- Presentation

Sl No.	Symptom	Yes/No
a)	Chest pain	
b)	Palpitations	
c)	Dyspnoea	
d)	Diaphoresis	
e)	Syncope	

Chest pain- If yes

a) Onset :
b) Duration :
c) Radiation :

C- Vitals

Sl No.	Parameter	Observation
a)	BP	
b)	Pulse	
c)	Saturation	
d)	JVP	

D- Personal History

Sl No.	Parameter	Yes/No
a)	Smoking	
b)	Alcohol consumption	
c)	Diet	

E- Comorbidities

Sl No.	Parameter	Observation
a)	Hypertension	
b)	Diabetes Mellitus	

F- Systemic Examination

G-

Sl No.	CVS	Observation
a)	S1 S2	
b)	Murmurs	

Sl No.	Respiratory	Observation
a)	Air Entry	
b)	Basal Crepitations	

H- ECG

a) 12 Lead ECG-

Sl No.	Lead	Observation
a)	2	
b)	3	
c)	AVF	
d)	1	
e)	V1	
f)	3:2	

b) Right Sided leads

Sl No.	Lead	Observation
a)	V1R	
b)	V2R	
c)	V3R	
d)	V4R	
e)	V5R	
f)	V6R	

I- 2D ECHO

Sl No.	Parameter	Result
a)	LVEF	
b)	RWMA	
c)	Right Ventricular Dysfunction	
d)	LV Dysfunction	

J- Investigations

Sl No.	Parameter	Result
e)	Haemoglobin	
f)	Total Counts	

g)	Differential counts	
h)	ANC	
i)	ALC	
j)	Platelet Counts	
k)	NLR	
l)	PLR	
m)	Creatinine	
n)	Sodium	
o)	Potassium	
p)	Bicarbonate	
q)	RBS	

K- Treatment given

Sl No.	Parameter	Yes/No
a)	Ionotropes	
b)	Fluid Resuscitation	
c)	Thromboylsis	

L- Rhythm:

a) On Admission :
b) 4 hours after admission :
c) 24 hours after admission :

M- Outcome

ANNEXUREIV - KEY TO MASTER CHART

General	
Yes	1
No	0
Gender	
Male	0
Female	1
BP	
< /= 100/60	1
100/60 - 120/90	2
> 120/90	3
Pulse	
<60	1
60-100	2
>100	3
Saturation	
</ = 90	1
>90	2
JVP	
Raised	1
Not raised	0
Diet	
Vegetarian	1
Mixed	2
ECG 12 Lead	
ST Elevation	1
No ST Elevation	0
ECG Right Sided Leads	
ST Elevation > 1mm	1
No ST Elevation	0
RV Dysfunction	
Present	1
Absent	0
TAPSE	
<15 mm	1
15-20 mm	2

Haemoglobin		
MALE:		
<13	Anaemic	1
13-17	Not anaemic	0
FEMALE:		
<12	Anaemic	1
12-15	Not anaemic	0
PLR		
MALE:	36-149	1
	>149	2
FEMALE:	43-172	1
	>172	2
NLR		
1-3	1	
3-6	2	
6-9	3	
>9	4	
Creatinine		
<1.5	1	
>1.5	2	
Sodium		
<135	1	
135-145	2	
>145	3	
Potassium		
<3.5	1	
3.5-5	2	
>5	3	
Bicarbonate		
<23	1	
23-30	2	
>30	3	
RBS		
<= 100	1	
100-200	2	
>=200	3	

KEY TO MASTER CHART

LVEF	**LV Dysfunction**	
51-70	Normal	1
41-50	Mild	2
31-40	Moderate	3
<30	Severe	4

Outcome	
PTCA	1
Death	2
Conservative Management	3
Rhythm	
N/A	0
NSR	1
STC	2
1AV	3
CHB	4
JR	5
SBC	6
TPM	7
AF	8
VT	9

Sl No.	IP Number	Age	Gender	Presentation					Vitals				Personal History			Comorbidities		Systemic Examination	ECG (12 Lead)							ECG (Right Sided leads)						2D ECHO				Investigations								Treatment Given				Rhythm			Outcome
				Chest Pain	Palpitations	Dyspnoea	Diaphoresis	Syncope	BP	Pulse	Saturation	JVP	Smoking	Alcohol Consumption	Diet	Hypertension	Diabetes Mellitus	Basal Crepitations	2	3	AVF	1	V1	V2	3:2	V1R	V2R	V3R	V4R	V5R	V6R	LVEF	RV Dysfunction	TAPSE	LV Dysfunction	Haemoglobin	NLR	PLR	Creatinine	Sodium	Potassium	Bicarbonate	RBS	Ionotropes	Fluid Resuscitation	Thromboylsis	Temporary PC	On Admission	4 Hours After Admission	24 hours after Admission	
1	973709	52	0	1	0	0	0	0	2	2	2	1	0	0	2	0	1	0	1	1	1	1	1	0	1	0	0	0	1	1	1	2	1	1	2	0	3	2	1	1	2	1	3	0	0	1	0	1	1	1	1
2	975451	72	0	1	1	0	1	0	2	1	2	1	1	0	2	1	1	1	1	1	1	1	1	1	0	1	1	1	1	1	2	1	1	2	1	1	1	2	2	2	1	3	0	0	1	1	4	7	1	1
3	967773	77	0	1	0	0	1	0	3	2	2	0	1	0	1	1	1	1	1	1	1	1	1	1	0	0	1	1	1	1	3	1	1	3	1	3	2	1	2	3	1	3	0	0	1	0	1	6	1	1
4	953376	54	0	1	0	0	0	0	2	2	2	0	0	1	1	1	0	0	1	1	1	1	1	1	1	1	1	1	1	1	3	1	1	3	0	3	2	1	2	2	1	2	0	0	1	0	1	1	1	3
5	965740	65	0	1	1	0	1	0	2	2	2	1	1	0	2	0	0	1	1	1	1	1	0	0	1	0	0	0	1	1	1	3	1	1	3	0	1	1	1	2	2	2	2	0	1	1	0	3	3	3	1
6	970999	72	1	1	0	1	1	0	2	2	2	1	0	0	1	1	0	0	1	1	1	1	0	1	1	0	0	1	1	1	0	2	1	1	2	0	3	2	1	1	2	1	2	0	1	1	0	3	3	1	3
7	1009934	79	1	1	1	1	0	0	1	1	1	1	0	0	2	1	1	1	1	1	1	1	1	0	0	0	0	1	1	1	1	3	1	1	3	0	2	1	2	1	3	1	3	1	1	0	0	6	6	0	2
8	967569	57	0	1	0	1	1	0	2	2	2	1	0	0	2	0	0	0	1	1	1	1	1	1	1	0	0	0	1	1	1	2	1	1	2	0	2	1	1	1	1	1	2	0	1	1	0	3	3	1	3
9	969198	52	0	1	1	0	1	0	3	2	2	1	1	1	2	0	0	1	1	1	1	1	1	1	1	0	0	1	1	1	1	2	1	1	2	0	2	1	1	2	2	1	2	0	0	1	0	3	6	3	1
10	509012	29	0	1	0	0	1	0	1	2	2	1	0	1	1	0	0	0	1	1	1	1	1	1	1	0	0	0	1	1	1	2	1	1	2	0	2	1	1	1	2	1	2	1	1	1	0	1	1	1	1
11	962763	67	0	1	1	0	1	1	1	1	1	1	0	1	2	0	0	1	1	1	1	0	1	1	0	0	1	1	1	1	1	3	1	1	3	1	3	1	2	2	2	1	2	1	1	0	0	2	2	0	2
12	964055	55	0	1	0	0	1	0	1	1	2	1	0	0	2	0	0	0	1	1	1	1	0	1	1	0	0	0	1	0	0	2	1	1	2	1	2	1	1	1	2	1	2	1	1	1	0	1	3	3	1
13	973409	45	0	1	0	1	1	0	1	2	2	1	0	0	2	0	0	1	1	1	1	1	0	1	1	0	0	0	1	1	1	2	0	1	2	0	3	1	1	2	2	1	2	1	1	1	0	1	1	1	3
14	967557	55	0	1	0	1	1	0	1	2	2	1	1	0	2	0	0	1	1	1	1	1	0	1	1	0	0	1	1	0	0	2	1	2	2	1	1	1	1	1	2	2	1	1	1	0	0	1	1	1	3
15	953811	40	1	1	0	0	1	0	2	2	2	0	0	0	2	0	0	0	1	1	1	1	1	1	1	0	0	1	1	1	1	3	1	1	3	1	2	2	1	1	2	1	2	0	0	1	0	3	6	3	3
16	960125	64	0	1	0	1	1	1	2	2	1	0	0	1	2	1	1	1	1	1	1	1	1	1	0	0	0	1	1	0	2	0	1	2	1	4	2	2	1	3	1	3	1	1	0	0	3	3	8	2
17	975487	38	0	1	0	0	0	0	3	3	2	0	0	1	1	0	0	0	1	1	1	0	0	1	1	0	0	0	1	0	0	2	0	1	2	1	2	1	1	2	2	1	2	0	0	1	0	2	2	1	3
18	968016	53	0	1	0	0	1	0	1	2	2	1	1	0	2	1	0	0	1	1	1	1	1	1	1	0	0	0	1	1	1	2	1	1	2	0	2	1	2	2	2	1	2	1	1	1	0	1	1	1	1
19	969191	59	0	1	1	0	1	0	1	2	1	1	1	1	2	1	0	1	1	1	1	1	1	0	1	0	0	0	1	1	0	2	0	1	2	0	4	2	1	2	2	1	1	1	1	1	0	1	1	1	3
20	975434	77	0	1	0	0	0	0	1	2	2	1	0	0	2	1	1	1	1	1	1	1	1	0	1	0	0	0	1	0	0	2	0	2	2	1	2	2	1	1	2	1	2	1	1	0	0	1	1	1	3
21	964655	64	1	1	0	0	1	0	1	1	2	1	0	0	1	1	1	1	0	1	1	0	1	1	0	0	0	1	1	1	1	3	1	1	3	0	2	1	1	1	3	1	3	0	1	1	0	5	6,3	6,3	3
22	947658	68	1	1	1	1	1	0	1	2	2	1	0	0	2	0	0	1	1	1	1	0	1	1	1	0	0	0	1	0	0	2	1	1	2	0	1	1	1	2	2	1	2	1	1	0	0	1	1	1	3
23	964210	49	1	0	0	0	1	0	3	2	2	1	0	0	2	1	1	1	1	1	1	1	0	1	0	0	0	0	1	1	1	2	1	1	2	1	3	1	1	1	3	1	3	0	0	1	0	1	1	1	3
24	997125	73	1	1	0	1	0	0	3	3	2	0	0	0	1	1	0	0	0	1	1	1	1	0	1	0	0	0	1	1	0	2	0	1	1	1	2	2	2	1	0	0	1	0	2	2	1	1	2	1	1
25	1014699	67	0	1	0	1	0	0	2	3	2	1	0	0	2	1	1	1	1	1	1	0	0	1	1	1	1	1	1	0	0	3	1	1	3	1	3	2	2	1	3	1	3	0	0	1	0	1	1	1	3
26	1014926	32	0	1	0	1	0	0	1	2	2	1	0	0	1	0	1	0	1	1	1	1	1	1	1	0	0	1	1	1	1	2	1	1	2	0	2	1	1	2	2	1	2	1	1	0	0	1	1	1	3
27	959608	72	1	1	0	1	1	1	1	1	2	1	0	0	1	0	0	0	1	1	1	1	1	1	1	0	0	1	1	1	1	3	1	1	3	1	2	1	2	1	2	1	3	0	1	0	0	3	1	1	3
28	959423	85	0	1	0	0	1	0	3	2	1	1	1	0	2	1	0	1	1	1	1	1	1	1	1	0	0	0	1	1	1	3	1	1	3	0	3	2	1	1	2	1	2	0	0	0	0	1	2	1	3
29	1014907	73	0	1	0	1	1	0	2	2	2	0	1	1	2	0	0	1	1	1	1	1	0	0	0	0	0	1	1	1	1	2	0	0	2	1	4	2	1	2	3	1	1	0	0	0	0	1	1	1	3
30	960019	62	0	1	1	1	1	0	1	2	2	0	0	0	2	1	0	0	1	1	1	1	1	1	1	0	0	1	1	1	1	2	1	1	2	0	3	2	1	2	2	1	2	1	1	0	0	1	1	1	3
31	1014772	58	1	0	0	0	1	1	1	1	1	1	0	0	2	1	0	0	1	1	1	1	1	0	1	0	1	1	1	1	1	2	1	1	2	1	3	2	2	2	2	1	2	1	1	1	0	5	6	1	1
32	970434	65	1	1	0	1	1	1	1	1	2	1	0	0	2	0	0	0	1	1	1	1	1	1	1	0	0	1	1	1	1	3	1	1	3	1	3	2	1	2	2	1	3	1	1	0	0	6	6	6	2
33	954334	68	1	1	0	1	1	0	1	3	2	0	0	0	2	0	1	0	1	1	1	1	1	1	1	0	1	1	1	1	1	3	1	1	3	0	3	2	1	2	3	1	3	1	1	0	0	2	2	1	3
34	1014623	48	0	1	0	0	0	0	3	1	2	1	0	1	2	0	1	0	1	1	1	1	1	0	0	0	0	0	1	1	0	2	0	1	2	1	1	1	1	2	2	1	3	0	0	1	0	6	6	1	1
35	972081	75	0	1	1	1	1	1	2	3	1	1	0	1	2	1	1	1	1	1	1	0	0	1	0	0	1	1	1	1	1	4	1	1	4	1	4	1	1	1	3	1	3	0	0	0	0	2	4	0	2
36	1014325	75	0	1	0	1	1	1	1	3	2	1	0	1	2	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	3	1	1	3	0	2	1	2	1	3	1	2	1	1	0	0	2	2	1	3
37	967366	64	1	1	0	1	1	0	1	3	1	1	0	0	2	1	1	0	1	1	1	1	1	1	1	0	1	1	1	1	1	4	1	1	4	0	2	2	1	2	2	1	2	1	1	0	0	2	9	6	3
38	972934	66	1	1	0	1	1	0	2	2	2	0	0	0	2	1	1	0	1	1	1	1	1	0	0	0	1	1	0	0	2	0	0	2	0	2	1	2	1	2	1	3	0	1	0	0	1	1	1	1	1
39	1014227	52	0	1	0	0	0	0	1	2	2	0	0	1	2	0	0	0	1	1	1	1	0	0	0	0	0	1	1	0	0	2	0	0	2	1	1	1	1	2	2	2	1	1	1	1	0	1	6	1	1
40	1013756	75	1	1	0	1	1	0	1	2	2	1	0	0	2	0	0		1	1	1	1	1	1	1	0	1	1	1	1	1	2	1	1	2	0	4	2	2	1	2	1	2	1	1	1	1	0	8	6	0	2

Gender	
Male	0
Female	1
General	
Yes	1
No	0

BP	
< /= 100/60	1
100/60 - 120/90	2
> 120/90	3
Pulse	
<60	1
60-100	2
>100	3

Saturation	
</ = 90	1
>90	2

JVP	
Raised	1
Not raised	0

Diet	
Vegetarian	1
Mixed	2

ECG 12 Lead	
ST Elevation	1
No ST Elevation	0

ECG Risht Sided Leads	
ST Elevation > 1mr	1
No ST Elevation	0

Outcome	
PTCA	1
Death	2
Conservative Man	3

Rhythm	
N/A	0
NSR	1

Haemoglobin			
Male	<13	Anaemic	1
	13-17	Not anaemic	0
Female	<12	Anaemic	1
	12-15	Not anaemic	0

NLR	
1-3	1
3-6	2
6-9	3
>9	4

PLR		
Male	36-149	1
	>149	2
Female	43-172	1
	>172	2

Creatinine	
<1.5	1
>1.5	2

Sodium	
<135	1
135-145	2
>145	3

Potassium	
<3.5	1
3.5-5	2
>5	3

Bicarbonate	
<23	1
23-30	2
>30	3

RBS	
<= 100	1
100-200	2
>=200	3

STC	2
1AV	3
CHB	4
JR	5
SBC	6
TPM	7
AF	8
VT	9

LVEF		**LVDysfn**	
51-70	1	Normal	
41-50	2	Mild	2
31-40	3	Moderate	3
<30	4	Severe	3

RV Dysfunction	
Present	1
Absent	0

TAPSE	
<15 mm	1
15-20 mm	2