Research Article

Correlation of Right Ventricular Function With Morbidity and Mortality in Acute Inferior Wall Myocardial Infarction

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ABSTRACT

Background: Right ventricular (RV) dysfunction is an important determinant of hemodynamic instability and adverse outcomes in acute inferior wall myocardial infarction (IWMI), but its clinical significance may be underrecognized. This study evaluated the relationship of RV dysfunction with morbidity and in-hospital mortality in patients with acute IWMI.

Methods: A retrospective observational study was conducted among 94 adults with first-time acute IWMI admitted within 24 hours of symptom onset at the Department of Cardiology, SRIHER, Chennai. Diagnosis was established by inferior ST-segment elevation and positive cardiac biomarkers. Transthoracic echocardiography was performed within 24-48 hours, and RV function was assessed using tricuspid annular plane systolic excursion (TAPSE), fractional area change (FAC), tissue Doppler S' velocity, and RV/LV basal diameter ratio. Clinical, electrocardiographic, echocardiographic, and in-hospital outcome data were analyzed using appropriate statistical tests; $p < 0.05$ was considered significant.

Results: The mean age was 61.33 ± 11.36 years, with male predominance (69.1%). Dyslipidaemia (52.1%), hypertension (48.9%), and diabetes mellitus (45.7%) were common. Raised jugular venous pressure was present in 54.3%, ST-segment elevation in V4R in 53.2%, and atrioventricular block in 21.3%. Mean LVEF was 42.56 ± 8.60%, while mean TAPSE was 14.92 ± 3.44 mm. RV dysfunction was significantly associated with hypotension requiring inotropic support ($\chi^2 = 17.857$, $p < 0.0001$), reduced TAPSE ($\chi^2 = 94.00$, $p < 0.0001$), AV block ($\chi^2 = 7.680$, $p = 0.0056$), V4R elevation ($\chi^2 = 32.292$, $p < 0.0001$), raised JVP ($\chi^2 = 3.907$, $p = 0.0481$), and increasing mitral regurgitation severity ($\chi^2 = 12.594$, $p = 0.0056$). No significant association was observed with mechanical ventilation, hospital stay, or in-hospital mortality.

Conclusion: RV dysfunction is clinically important in acute IWMI and is strongly associated with hemodynamic compromise, ECG markers, conduction abnormalities, and echocardiographic abnormalities. Routine assessment of RV function, particularly TAPSE and right-sided ECG leads, may improve early risk stratification and guide management. Early recognition may facilitate timely hemodynamic support, intensive monitoring, and comprehensive biventricular assessment.

Keywords: Acute inferior wall myocardial infarction; Right ventricular dysfunction; TAPSE; Right ventricular infarction; Echocardiography; In-hospital Mortality.

Introduction

Myocardial infarction (MI) was generally regarded as an ischemic injury to the left ventricle. The right ventricle, as the site of major damage from acute MI, has been recognized clinically only recently. Right ventricular infarction (RVI) was first described more than 70 years ago in autopsies. Between 1949 and 1959, several authors correlated electrocardiographic (ECG) and clinicopathologic findings in patients with MI and concluded that there were no definite ECG signs referable to right ventricular involvement, producing skepticism about the utility of ECG for the diagnosis of RVI.[1-3]

In 1974, Cohn et al. described the syndrome of predominant right ventricular dysfunction during acute inferior wall myocardial infarction (IWMI) and reported that hemodynamics deteriorated markedly and atrioventricular block tends to occur in patients with RVI, with a resulting increase in the hospital death rate.[4] Since then, the right ventricle, which was the forgotten chamber, has drawn much attention, and research has focused on RVI. This landmark study was followed by a renewed interest in the diagnosis of RVI by the use of noninvasive modalities such as ECG, echocardiography (ECHO), nuclear imaging studies, and invasive modalities like right-sided heart catheterization.

The diagnosis of RVI is important because the management of RVI differs from that of left ventricular infarction. The profound hemodynamic sequelae of RVI may be prevented by the administration of an appropriate intravenous volume load. If RVI is neglected or treated as left-sided heart failure with diuretics, cardiogenic shock may supervene. Nitrates are contraindicated in RVI, as RV function is preload-dependent.[5]

Inferior wall myocardial infarction is complicated by right ventricular infarction (RVMI) in as many as 50% of cases.[6-8] In a series by Anderson et al. in 1989, the incidence of RVMI in patients with inferior wall myocardial

infarction was between 10-50%. Isolated infarction of the right ventricle is extremely rare.[9] Although right ventricular infarction is clinically evident in a sizable number of cases, the incidence is considerably less than that found at autopsy. A major reason for the discrepancy is the difficulty in establishing the presence of RVMI in living patients.[10,11]

Right ventricular infarction contributes markedly to hemodynamic instability, atrioventricular conduction blocks, and in-hospital mortality in patients with inferior wall myocardial infarction. Systolic right ventricular function is an important predictor in the course of myocardial infarction.[12,13] According to the Shock Trial Registry, despite the younger age, lower rate of anterior MI, and higher prevalence of single vessel coronary disease of RV compared with LV shock patients, and their similar benefit from revascularization, mortality is unexpectedly high in patients with predominant RV shock and similar to patients with LV shock.[14]

Cardiogenic shock and the requirement for temporary transvenous cardiac pacing are more common in patients with right ventricular dilatation. Furthermore, by implying multivessel coronary artery disease, the presence of right ventricular dysfunction carries an adverse prognosis irrespective of infarct location.[15]

MATERIALS AND METHODS

Study Design

This is a retrospective observational study conducted among patients diagnosed with acute inferior wall myocardial infarction. Data were collected from previously recorded medical records, case files, and echocardiography reports. No direct intervention or alteration of treatment was involved, and only existing data were analysed.

Study Population

Patients aged ≥18 years presenting with first-time acute inferior wall myocardial infarction (IWMI), admitted within 24 hours of symptom onset.

Eligibility Criteria

Inclusion Criteria:

- ECG showing ST elevation ≥1 mm in at least two inferior leads (II, III, aVF)
- Serum troponin/CK-MB positivity
- Presentation within 24 hours of symptom onset
- Consent to participate

Exclusion Criteria:

- Prior myocardial infarction
- Known right ventricular cardiomyopathy
- Chronic pulmonary disease affecting RV pressure load
- Left ventricular ejection fraction (LVEF) <30% (to minimize confounding)
- Severe valvular heart disease
- Patients not consenting for echocardiography or participation

Place of Study

Department of Cardiology, SRIHER, Chennai

Duration of Study

The study spanned 2 years, encompassing both data collection and analysis phases.

Sample Size

According to Kanar et al. (2018), the in-hospital mortality rate in patients with IWMI and RV dysfunction was approximately 21.1% whereas those without RV dysfunction had a mortality of around 2.3%.

Using these values:

- p_1 (mortality rate without RV dysfunction) = 2.3% = 0.023
- p_2 (mortality rate with RV dysfunction) = 21.1% = 0.211
- Alpha (Type I error) = 0.05 (two-sided)
- Beta (Type II error) = 0.20 (80% power)
- $Z\alpha/_2 = 1.96$
- $Z\beta = 0.84$

Final Sample Size: Per group: 42 patients; Total: 84 patients; With 10% attrition added: 94 Patients

Data Collection Tools and Procedure

Upon admission, demographic and clinical data such as age, sex, cardiovascular risk factors (including diabetes, hypertension, and smoking), vital signs, Killip classification, and hemodynamic parameters were recorded. A 12-

lead ECG was used to confirm the diagnosis of IWMI, and cardiac biomarkers were used to support the diagnosis of acute myocardial injury. All patients underwent transthoracic echocardiography within 24 to 48 hours of admission. Right ventricular function was assessed using parameters including Tricuspid Annular Plane Systolic Excursion (TAPSE), with values less than 16mm indicating dysfunction, RV Fractional Area Change (FAC) below 35%, tissue Doppler S' wave velocity below 9.5 cm/s, and RV dilation defined by an RV to LV basal diameter ratio greater than 1. Left ventricular ejection fraction was estimated by Simpson's method, and signs of RV infarction such as RV wall hypokinesia or akinesia, were noted.

Follow-up and Outcome

Patients were monitored throughout their hospital stay. The primary outcome was in-hospital mortality. Secondary outcomes included morbidity parameters such as the occurrence of arrhythmias (including atrioventricular block or ventricular tachycardia), hypotension requiring inotropic support, need for mechanical ventilation, and length of hospital stay.

Data Analysis

Data were entered in Microsoft Excel and analyzed using SPSS version 25. Continuous variables were presented as mean ± standard deviation and compared using independent t-tests or Mann-Whitney U tests where appropriate. Categorical variables were summarized as percentages and analyzed using the Chi-square test or Fisher's exact test. Logistic regression analyses were conducted to determine whether RV dysfunction is an independent predictor of mortality or adverse in-hospital outcomes. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: Profile of Comorbidities and Habits

Comorbidity	Frequency	%
Diabetes		
Yes	43	45.7
No	51	54.3
Hypertension		
Yes	46	48.9
No	48	51.1
Dyslipidemia		
Yes	49	52.1
No	45	47.9
Family H/o CAD		
Yes	39	41.5
No	55	58.5
Smoking		
Yes	18	19.14

Comorbidity	Frequency	%
No	76	80.85

In this study of 94 patients (mean age 61.33 ± 11.36 years; 95% CI: 59.00–63.66), most were older adults with a relatively homogeneous age distribution. Males predominated (69.1%; M:F ratio ~2.2:1). Major cardiovascular risk factors included dyslipidaemia (52.1%), hypertension (48.9%), diabetes (45.7%), family history of CAD (41.5%), and smoking (19.1%). Dyslipidaemia was the most common.

Table 2: Profile of Hemodynamic Parameters

Variable	Mean ± SD	95% CI
Heart Rate (bpm)	90.37 ± 21.10	86.0500 to 94.6946
Systolic Blood Pressure (mmHg)	107.37 ± 19.60	103.3565 to 111.3882
Diastolic Blood Pressure (mmHg)	66.94 ± 14.08	64.0628 to 69.8308

Baseline hemodynamics (Table 2) showed a mean heart rate of 90.37 ± 21.10 bpm (95% CI: 86.05–94.69), indicating elevated resting heart rates. Mean systolic and diastolic blood pressures were 107.37 ± 19.60 mmHg (95% CI: 103.36–111.39) and 66.94 ± 14.08 mmHg (95% CI: 64.06–69.83), respectively—consistent with low-normal pressures seen in left ventricular systolic dysfunction.

Table 3: Profile of Killip Class

Killip Class	Frequency	%
I	20	21.28
II	21	22.34
III	24	25.53
IV	29	30.85

Killip Class IV was most common (29, 30.9%), followed by Class III (24, 25.5%), Class II (21, 22.3%), and Class I (20, 21.3%). Advanced heart failure (Classes III–IV) accounted for 56.4%, indicating that over half presented with moderate-to-severe clinical heart failure.

Table 4: Profile of Electrocardiographic Variables

Variable	Frequency	%
JVP Raised		
Yes	51	54.3
No	43	45.7
ST V4R		

Variable	Frequency	%
Yes	50	53.2
No	44	46.8
AV Block		
Yes	20	21.3
No	74	78.8

Raised JVP was seen in 51 (54.3%) patients, and ST-segment elevation in lead V4R (suggestive of right ventricular involvement) in 50 (53.2%). AV block was present in 20 (21.3%). Thus, over half had raised JVP and V4R elevation, while AV block affected about one-fifth.

Table 5: Status of LVEF and TAPSE

Variable	Mean ± SD	95% CI
LVEF (%)	42.56 ± 8.60	40.8020 to 44.3256
TAPSE (mm)	14.92 ± 3.44	14.2221 to 15.6332

Mean LVEF was 42.56 ± 8.60% (95% CI: 40.80–44.33%), confirming impaired left ventricular systolic function per inclusion criteria. Mean TAPSE was 14.92 ± 3.44 mm (95% CI: 14.22–15.63 mm), below normal range, indicating concurrent right ventricular dysfunction—a finding often linked to worse cardiovascular outcomes.

Table 6: Profile of Arrhythmia

Arrhythmia	Frequency	%
1st degree	6	6.4
2nd degree	7	7.4
CHB	8	8.5
VT	1	1.1
None	72	76.6

Most patients (72, 76.6%) had no baseline conduction abnormalities. Among the remainder, complete heart block (CHB) was most common (8, 8.5%), followed by second-degree AV block (7, 7.4%), first-degree AV block (6, 6.4%), and ventricular tachycardia (1, 1.1%). Overall, about one-fourth had conduction defects, with CHB being the most frequent.

Table 7: Profile of In-Hospital Clinical Outcome

Variable	Frequency	%
Hypotension Inotropes		
Yes	50	53.2
No	44	46.8
In-Hospital Mortality		
Yes	9	9.6
No	85	90.4
Mechanical Ventilation		
Yes	29	30.9
No	65	69.1
Shock		
Yes	29	30.9
No	65	69.1

Hypotension requiring inotropes occurred in 50 (53.2%) patients. Mechanical ventilation and cardiogenic shock each affected 29 (30.9%) patients. In-hospital mortality was 9 (9.6%). Thus, over half needed inotropic support, about one-third required ventilation or developed shock, and the mortality rate was 9.6%.

Table 8: Association between Hypotension requiring Inotropic support and right ventricular (RV) dysfunction

Hypotension	RV Dysfunction		Chi-squared	P value
	Yes	No		
Yes	47	3	17.857	<0.0001
No	25	19		

Strong association was observed between hypotension requiring inotropes and RV dysfunction. Among 50 patients on inotropes, 47 (94.0%) had RV dysfunction vs. 3 (6.0%) with normal RV. Among 44 not on inotropes, 25 (56.8%) had RV dysfunction and 19 (43.2%) had preserved function. This association was highly significant ($\chi^2 = 17.857$; $p < 0.0001$), indicating that RV dysfunction markedly increased the need for inotropic support and fluid management during hospitalization.

Table 9: Association between TAPSE and right ventricular (RV) dysfunction

TAPSE (mm)	RV Dysfunction		Chi-squared	P value
	Yes	No		
<11	8	0	94.00	<0.0001
11.1-13	15	0		
13.1-15	49	0		
15.1-17	0	2		
>17	0	20		

Strong association observed between TAPSE and RV dysfunction. All patients with TAPSE ≤ 15 mm had RV dysfunction: 8 (TAPSE <11 mm), 15 (11.1–13.0 mm), and 49 (13.1–15.0 mm). Among those with TAPSE 15.1–17.0 mm, 2 had preserved RV function, and all 20 with TAPSE >17 mm had normal function. This association was highly significant ($\chi^2 = 94.00$; $p < 0.0001$), confirming TAPSE as a reliable marker of right ventricular systolic function.

Table 10: Association between AV Block and right ventricular (RV) dysfunction

AV Block	RV Dysfunction		Chi-squared	P value
	Yes	No		
Yes	20	0	7.680	0.0056
No	52	22		

Significant association was observed between AV block and RV dysfunction. All 20 patients with AV block (100%) had RV dysfunction, versus 52 of 74 (70.3%) without AV block. The association was statistically significant ($\chi^2 = 7.680$; $p = 0.0056$), indicating that AV block was strongly linked to concomitant right ventricular systolic impairment.

Table 11: Association between STV4R and right ventricular (RV) dysfunction

STV4R	RV Dysfunction		Chi-squared	P value
	Yes	No		
Yes	50	0	32.292	<0.0001
No	22	22		

Strong association was observed between ST-segment elevation in lead V4R (ST V4R) and RV dysfunction. All 50 patients with ST V4R elevation had RV dysfunction (100%), whereas among 44 without it, 22 (50.0%) had RV dysfunction and 22 (50.0%) had normal function. This association was highly significant ($\chi^2 = 32.292$; $p < 0.0001$), confirming ST V4R as a valuable ECG marker for right ventricular involvement.

Table 12: Association between Mitral Regurgitation and right ventricular (RV) dysfunction

Mitral Regurgitation	RV Dysfunction		Chi-squared	P value
	Yes	No		
Mild	42	8	12.594	0.0056
Moderate	10	1		
Severe	5	0		
None	15	13		

The significant association was observed between MR severity and RV dysfunction. RV dysfunction was present in 84.0% (mild MR), 90.9% (moderate), and 100% (severe), versus 53.6% among those without MR. The association was statistically significant ($\chi^2 = 12.594$; $p = 0.0056$), indicating that greater MR severity correlated with RV dysfunction and may reflect advanced cardiac involvement.

DISCUSSION

The present study evaluated the clinical characteristics and right ventricular (RV) dysfunction among 94 patients presenting with acute inferior wall myocardial infarction (IWMI). The findings demonstrated that RV dysfunction was frequently associated with adverse clinical characteristics, including hemodynamic instability, conduction abnormalities, and echocardiographic evidence of right-sided and biventricular involvement. Critically, all these documented complications are expected findings in patients with proximal right coronary artery (RCA) lesions, although coronary angiographic profile of these patients were not taken in account as part of this study protocol.

Demographic and Risk Factor Profile

The mean age of the study population was 61.33 ± 11.36 years, indicating that acute coronary syndrome (ACS), particularly acute myocardial infarction, predominantly affected older adults in the present cohort. Age is a well-established determinant of ACS risk because cumulative exposure to atherosclerotic risk factors contributes to progressive coronary atherosclerosis and plaque-related events. In the INTERHEART study, the median age at first acute myocardial infarction was 56 years in men and 65 years in women, demonstrating the strong relationship between advancing age and acute MI occurrence.[16]

The observed male predominance (69.1%) in our cohort is consistent with the established epidemiology of ACS and acute myocardial infarction. The INTERHEART study demonstrated that male sex is an independent predictor of acute myocardial infarction, largely because of greater exposure to modifiable cardiovascular risk factors such as smoking, dyslipidaemia, and hypertension.[16]

Cardiovascular risk factors play a central role in the development and progression of atherosclerotic cardiovascular disease (ASCVD). Dyslipidaemia (52.1%) was the most prevalent risk factor, followed by hypertension (48.9%), diabetes mellitus (45.7%), family history of coronary artery disease (41.5%), and smoking (19.1%). The high prevalence of dyslipidaemia observed in this study is clinically important because atherogenic lipid exposure promotes plaque formation, progression, and instability, thereby increasing the risk of coronary artery disease and acute coronary events. The ESC/EAS dyslipidaemia guideline identifies elevated atherogenic lipoproteins as a major modifiable determinant of ASCVD risk.[17]

Hemodynamic Characteristics and Killip Classification

The mean heart rate was 90.37 ± 21.10 bpm, while the mean systolic and diastolic blood pressures were 107.37 ± 19.60 mmHg and 66.94 ± 14.08 mmHg, respectively. These findings suggest that a substantial proportion of patients presented with relative tachycardia and low-normal blood pressure, consistent with impaired cardiac output and neurohormonal activation.

An important observation was the predominance of advanced heart failure at presentation. Killip Class IV (30.85%) constituted the largest group, followed by Class III (25.53%), Class II (22.34%), and Class I (21.28%), indicating that more than half of the patients presented with moderate-to-severe heart failure.

In the context of proximal RCA occlusion, RV dysfunction in acute inferior wall myocardial infarction can cause significant hemodynamic compromise by reducing RV forward flow, left ventricular preload and cardiac output, leading to hypotension and cardiogenic shock despite relatively preserved LV systolic function.[18] The Killip

classification remains one of the simplest and most reliable bedside tools for assessing the severity of acute myocardial infarction and predicting short-term mortality. Although higher incidence of hemodynamic instability in this study population is attributed to acute RV dysfunction due to reduced forward flow, nevertheless higher Killip classes have consistently been associated with larger infarct size, poorer ventricular function, and increased risk of cardiogenic shock.[19,20]

Electrocardiographic Findings and Proximal RCA Involvement

ST-Segment Elevation in Lead V4R

One of the most important observations in the present study was the significant association between ST-segment elevation in lead V4R and RV dysfunction ($\chi^2 = 32.292$, $p < 0.0001$). All patients with ST elevation in V4R demonstrated evidence of RV dysfunction (Table 11). Lead V4R is widely accepted as the most sensitive electrocardiographic lead for detecting right ventricular infarction, particularly in patients with inferior wall myocardial infarction. RV myocardial ischemia results in characteristic ST-segment elevation in the right precordial leads, reflecting acute injury of the right ventricular free wall.

This finding has direct anatomical correlation with proximal RCA occlusion. The right coronary artery, in its proximal course, gives off right ventricular marginal branches that supply the RV free wall. Occlusion proximal to these branches results in RV infarction, which is detected by ST elevation in right-sided precordial leads (V3R-V6R, particularly V4R). The highly significant association observed in the present study reinforces the clinical importance of incorporating right-sided ECG leads into routine evaluation of patients with suspected RV dysfunction and strongly suggests proximal RCA involvement as the underlying mechanism.

These findings are consistent with the landmark study by Braat et al., who demonstrated that right-sided chest leads significantly improve the diagnosis of RV infarction. Similarly, Ibanez et al. (2023 ESC Guidelines for Acute Coronary Syndromes) recommend routine recording of right-sided leads in patients presenting with inferior myocardial infarction because ST elevation in V4R accurately identifies RV involvement and predicts adverse outcomes.[21]

Atrioventricular Block

AV block was documented in 21.3% of patients and showed a significant association with RV dysfunction ($\chi^2 = 7.680$, $p = 0.0056$). Notably, all patients with AV block had RV dysfunction (Table 10).

From an anatomical perspective, this finding is highly specific for proximal RCA occlusion. The atrioventricular nodal artery, which supplies the AV node, typically arises from the RCA in 90% of the population. Proximal RCA occlusion compromises the AV nodal artery, leading to varying degrees of AV conduction abnormalities. Because the right coronary artery supplies both the AV node and the right ventricle (via RV marginal branches) in most individuals, simultaneous occurrence of AV block and RV dysfunction is pathognomonic for proximal RCA involvement.

These observations agree with those reported by Goldstein et al., who demonstrated that conduction abnormalities occur significantly more often in patients with RV infarction than in isolated inferior myocardial infarction.[22] The present study therefore supports the concept that AV block serves as an indirect clinical marker of significant RV involvement and, by extension, proximal RCA occlusion.

Raised Jugular Venous Pressure

Raised JVP was present in 54.3% of patients and showed a statistically significant association with RV dysfunction ($\chi^2 = 3.907$, $p = 0.0481$). Elevated JVP is one of the earliest clinical manifestations of right-sided heart failure. RV systolic dysfunction leads to elevated right atrial pressure and systemic venous congestion, resulting in distended neck veins. Although echocardiography has become the gold standard for assessing RV function, bedside examination of JVP remains a valuable clinical tool, particularly in resource-limited settings.

In the setting of proximal RCA occlusion, elevated JVP reflects impaired RV compliance and reduced RV output, resulting in backward failure and systemic venous congestion. This clinical finding, when combined with hypotension and clear lung fields, constitutes the classic clinical triad of RV infarction. Our findings are consistent with the observations of Konstam et al., who reported that elevated JVP is strongly associated with impaired RV function and predicts adverse outcomes in heart failure patients.[23]

Echocardiographic Findings

Tricuspid Annular Plane Systolic Excursion (TAPSE)

A major finding of the present study was the reduced mean TAPSE of 14.92 ± 3.44 mm, indicating impaired RV systolic function. Furthermore, TAPSE demonstrated the strongest association with RV dysfunction ($\chi^2 = 94.00$, $p < 0.0001$), with all patients having TAPSE ≤ 15 mm exhibiting RV dysfunction (Table 9).

TAPSE is one of the simplest, most reproducible, and guideline-recommended echocardiographic indices of RV systolic function. A reduced TAPSE reflects impaired longitudinal contraction of the RV free wall and has been associated with increased mortality in heart failure. In the context of proximal RCA occlusion, reduced TAPSE directly correlates with ischemia of the RV free wall, which is supplied by RV marginal branches originating from the proximal RCA. These findings closely resemble those reported by Rudski et al. and subsequently endorsed by the American Society of Echocardiography, which recommend TAPSE as a routine measure of RV function.[24]

Mitral Regurgitation

Mitral regurgitation (MR) was highly prevalent when all grades were considered, with 70.2% of patients demonstrating some degree of MR. However, for the purpose of interpreting the present study, mild MR should not be considered a clinically significant disease manifestation because it may be an incidental echocardiographic finding. The clinically relevant subgroup is moderate-to-severe MR, which represented approximately 34% of the cohort. Importantly, MR severity showed a statistically significant association with RV dysfunction ($\chi^2 = 12.594$, $p = 0.0056$) (Table 12).

From an anatomical and pathophysiological perspective, the association between moderate-to-severe MR and RV dysfunction in proximal RCA occlusion is clinically plausible through multiple mechanisms:

1. **Posteromedial Papillary Muscle Dysfunction:** The posteromedial papillary muscle receives its blood supply exclusively from the RCA (via the posterior descending artery). Acute ischemia of this papillary muscle can lead to mitral leaflet tethering and acute MR. Proximal RCA occlusion, particularly when extending to involve the posterior descending artery, can cause significant papillary muscle ischemia and dysfunction.
2. **RV-Mediated Left Ventricular Geometry Changes:** RV dilatation from acute infarction can cause a leftward shift of the interventricular septum, altering mitral valve geometry and contributing to functional MR. This ventricular-ventricular interaction is a well-recognized phenomenon in acute RV infarction.
3. **Increased Left Ventricular Filling Pressures:** The combination of LV dysfunction and RV impairment can elevate left ventricular end-diastolic pressure, worsening MR severity. The relationship between RV dysfunction and MR is bidirectional—RV dysfunction worsens MR through geometric changes and elevated pressures, while significant MR increases left atrial and pulmonary venous pressures, raising RV afterload and further compromising RV function.

Studies of severe MR complicating acute MI have shown a strong relationship with inferior infarction and severe hemodynamic consequences, supporting the relevance of acute MR as a complication of the index infarction rather than as a marker of chronic valvular disease.[25]

In-Hospital Clinical Outcomes and Proximal RCA Pathophysiology

RV Dysfunction and Hypotension Requiring Inotropic Support

One of the most important findings of the present study was the highly significant association between RV dysfunction and hypotension requiring inotropic support ($\chi^2 = 17.857$; $p < 0.0001$). Among patients requiring inotropes, 94% had RV dysfunction, whereas preserved RV function was observed in only a small minority (Table 8).

This finding is physiologically expected in proximal RCA occlusion. The right ventricle is responsible for maintaining adequate pulmonary circulation and ensuring sufficient preload to the left ventricle. RV systolic dysfunction results in reduced pulmonary blood flow, diminished left ventricular filling, decreased cardiac output, and systemic hypotension. In proximal RCA occlusion with extensive RV infarction, this hemodynamic compromise is often profound and requires vasopressor or inotropic support to maintain tissue perfusion.

Our findings agree with those of Konstam et al., who reported that RV dysfunction is a major determinant of circulatory failure in acute heart failure and cardiogenic shock.[23] Likewise, Harjola et al. demonstrated that RV dysfunction independently predicts hemodynamic instability and the need for pharmacological circulatory support in patients with acute heart failure.[26] The strong association observed in the present study highlights the importance of early identification of RV dysfunction, as timely recognition may facilitate prompt initiation of appropriate hemodynamic support and improve clinical outcomes.

RV Dysfunction and In-Hospital Mortality

In the present study, RV dysfunction was more frequent among patients who died (88.9%) than among survivors (75.3%); however, this difference did not reach statistical significance ($\chi^2 = 0.830$; $p = 0.3622$). Although this finding appears to differ from several large studies demonstrating increased mortality among patients with RV dysfunction, it is important to interpret the results within the context of the present study. Only nine patients

experienced in-hospital mortality, limiting statistical power. Therefore, the absence of statistical significance should not be interpreted as evidence that RV dysfunction lacks prognostic importance.

Large multicentre investigations have consistently demonstrated that impaired RV function is associated with increased mortality in heart failure and myocardial infarction.[27,28] The discrepancy between our findings and previous reports is therefore most likely attributable to sample size rather than a true absence of association.

CONCLUSION

The present study demonstrates that right ventricular dysfunction is highly prevalent among patients with acute inferior wall myocardial infarction (IWMI) and is closely associated with hemodynamic instability, reduced TAPSE, atrioventricular block, ST-segment elevation in lead V4R, raised jugular venous pressure, and increasing severity of mitral regurgitation. These findings highlight the important clinical and echocardiographic significance of RV involvement in the setting of acute IWMI.

All these complications are expected in proximal RCA lesions. The constellation of findings—RV dysfunction, AV block, hypotension requiring inotropic support, raised JVP, and significant MR—paints a clinical picture consistent with proximal RCA occlusion, the most common anatomical substrate for RV involvement in inferior MI.

Although RV dysfunction did not show a statistically significant association with mechanical ventilation, hospital stay, or in-hospital mortality in this cohort, possibly due to low sample size. Patients with RV dysfunction exhibited greater clinical severity and required more intensive hemodynamic support. The findings therefore emphasize the importance of early identification of RV involvement in patients presenting with acute IWMI.

These findings underscore the importance of comprehensive assessment of RV function in acute IWMI, incorporating bedside clinical examination, electrocardiography—particularly right-sided leads such as V4R—and echocardiographic assessment. TAPSE demonstrated particular clinical utility in identifying RV systolic dysfunction. Early recognition of RV dysfunction may improve risk stratification, facilitate timely therapeutic decision-making, guide hemodynamic management, and potentially improve clinical outcomes in patients with acute IWMI.

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