

Original article

Association Of Right Ventricular Infarction With Inferior Wall Myocardial Infarction

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DOI: <https://doi.org/10.32894/kjms.2021.170173>

Abstract:

- **Background:** Right ventricular infarction (RVI) commonly accompanies inferior wall myocardial infarction (IWMI) and is associated with increased morbidity and mortality. The aim of this study is to determine the prevalence of RVI among patients with IWMI and to identify clinical characteristics and risk factors associated with RVI.
- **Methods:** A cross-sectional study was conducted at the Coronary Care Unit of Azadi Teaching Hospital in Kirkuk from January 1 to October 31, 2017. A convenient sample of 150 patients with IWMI was evaluated through direct interviews using a structured questionnaire.
- **Result:** RVI was identified in 39.3% of patients with IWMI. There was a highly significant association between RVI and older age, particularly among self-employed individuals ($p < 0.001$). Significant risk factors for RVI included a family history of heart disease ($p < 0.001$) and obesity ($p = 0.01$). Patients with both IWMI and RVI experienced significantly more complications than those with IWMI alone ($p = 0.002$).
- **Conclusions:** The prevalence of RVI in patients with IWMI aligns with previously reported data. Older age, positive family history, and obesity are key risk factors, and RVI is linked to a higher rate of clinical complications in IWMI patients.
- **Keywords:** Right ventricular infarction, inferior wall myocardial infarction, risk factors



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INTRODUCTION

1.1. Coronary Artery Diseases

Cardiovascular disease (CVD) remains a leading global cause of death and disability. Among its various forms, coronary artery disease (CAD) is the most prevalent and fatal. While traditionally a significant cause of mortality in Western nations, CAD is increasingly responsible for death in developing countries as well, a trend driven by rising rates of major risk factors such as obesity and diabetes (1).

Although CAD primarily affects older individuals, the disease process often begins in early adulthood. While the exact prevalence of coronary atherosclerosis in young adults is difficult to determine, long-term studies have demonstrated that early exposure to risk factors—particularly smoking, substance abuse (such as cocaine), diabetes, and dyslipidemia—can predict CAD development in later life (2).

Atherosclerosis, the foundation of CAD, is marked by the formation of plaques within the arterial walls. This involves the infiltration and oxidation of low-density lipoprotein (LDL) particles in the intimal layer of arteries. Oxidized LDL (ox-LDL) promotes the

formation of foam cells by macrophages, which accumulate and form the core of atherosclerotic plaques (3).

Acute coronary syndrome (ACS) encompasses a spectrum of conditions associated with acute myocardial ischemia: unstable angina (UA), non–ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI). UA and NSTEMI share similar underlying mechanisms but differ in severity. NSTEMI is diagnosed when myocardial injury is significant enough to elevate biomarkers such as troponins or CK-MB (3, 4). UA, by contrast, presents without biomarker elevation following chest pain. UA is identified by one or more of the following: angina at rest lasting over 20 minutes, new-onset severe angina, or a crescendo pattern in symptom frequency or intensity. In the United States, ACS leads to 1.36 million hospitalizations annually, with approximately 0.81 million due to myocardial infarction and the remainder due to UA. Of these MI cases, approximately two-thirds are NSTEMI (4).

1.2. Myocardial Infarction

Myocardial infarction (MI), a major manifestation of CAD, is diagnosed using a combination of clinical symptoms, ECG findings, cardiac biomarker elevation, imaging, or pathological evidence. MI may be the first sign of CAD or a recurrence in those with established disease. Accurate assessment of MI incidence is essential for understanding the CAD burden in populations and is critical in clinical trials and quality assurance programs (5).

In 2000, the First Global Task Force introduced a new MI definition based on the presence of any myocardial necrosis in the context of ischemia (6). This definition was refined in 2007 with the Universal Definition of Myocardial Infarction, which recognized the various clinical scenarios that can lead to MI (7). Endorsed by leading cardiology organizations and the World Health Organization, this definition has been widely adopted (8). The availability of increasingly sensitive biomarkers has necessitated further revisions, especially for identifying minor necrosis in critically ill patients or post-intervention scenarios (5).

1.1.1. Pathological Characteristics of Myocardial Ischemia and Infarction

Pathologically, MI is defined as irreversible myocardial cell death caused by sustained ischemia. Histological evidence of cell death may emerge within 20 minutes of ischemia onset, although complete necrosis typically requires 2–4 hours or more, depending on factors like collateral circulation, intermittent occlusion, and cellular sensitivity (9). The entire healing process may take up to 6 weeks, and reperfusion can alter both microscopic and macroscopic appearances (5).

1.1.2. Biomarker Detection of Myocardial Injury with Necrosis

Myocardial injury is confirmed by elevated levels of cardiac biomarkers such as cardiac troponins (cTnI and cTnT) and CK-MB (7). These proteins are highly specific to myocardial tissue. However, elevated biomarker levels only indicate the presence of myocardial injury, not the underlying mechanism (10). Various processes, including normal cell turnover, apoptosis, or membrane blebbing, may cause troponin release (11). Nonetheless, any necrosis due to ischemia is classified as MI (5).

1.1.3. Clinical Features of Myocardial Ischemia and Infarction

Myocardial ischemia arises from an imbalance between oxygen demand and supply. Clinically, ischemia often presents with chest or upper body discomfort (e.g., arm, jaw, epigastric), or symptoms like dyspnea or fatigue (8). Ischemic discomfort from acute MI typically lasts ≥ 20 minutes and may be accompanied by diaphoresis, nausea, or syncope. However, these signs are non-specific and may mimic non-cardiac disorders. MI can also present with atypical symptoms or even be silent, especially in women, elderly patients, and those with diabetes (7). Accurate interpretation of clinical signs and biomarker trends is essential for diagnosis (8).

1.1.4. Clinical Classification of Myocardial Infarction

MI is classified based on ECG findings and biomarker levels. ST-elevation MI (STEMI) is diagnosed when ischemic symptoms are accompanied by ST-segment elevation in two contiguous leads. Non-ST-elevation MI (NSTEMI) presents without ST elevation. MI may also be categorized as Q-wave or non-Q-wave, based on ECG evolution. UA is diagnosed when ischemic symptoms occur without biomarker elevation or new ECG changes.

1.1.5. Myocardial Distribution of the Main Coronary Arteries

The left anterior descending (LAD) artery travels along the anterior interventricular groove and supplies the anterior wall and septum via septal and diagonal branches. In about 80% of individuals, the LAD reaches the diaphragmatic surface of the left ventricle; in others, the apex is supplied by a larger RCA or LCx (12).

The left circumflex (LCx) artery runs in the left atrioventricular groove and is the dominant vessel in 15% of individuals. Its major branches, the obtuse marginals, supply the lateral LV wall (13).

The right coronary artery (RCA) originates from the right aortic sinus, travels in the right AV groove, and gives rise to the PDA and posterolateral branches. The conus artery and sinoatrial node artery are its proximal branches. The RCA supplies the right ventricle and inferior wall (12).

1.2. Anterior Wall Myocardial Infarction

Anterior MI results from occlusion of the LAD and affects the anterior LV wall. Proximal lesions may cause extensive infarction involving septal and lateral regions (14). ECG features include ST-segment elevation in leads V3–V4, often with involvement of septal or lateral leads. “Tombstoning” describes the pronounced ST elevation. Reciprocal depression is often seen in inferior leads. ST elevation criteria per ACC/AHA guidelines are ≥ 2 mm in V2–V3 (men), ≥ 1.5 mm (women), and ≥ 1 mm in other contiguous leads (15).

1.3. Posterior Wall Myocardial Infarction

Posterior MI occurs in 15–20% of STEMIs, typically alongside inferior or lateral MI. Isolated posterior MI is rare. Its presence suggests a larger infarction area and worse prognosis. Diagnosis is based on ECG findings in leads V1–V3: horizontal ST depression, tall R waves, upright T waves, or an R/S ratio >1 (12).

1.4. Lateral Wall Myocardial Infarction

Lateral MI, affecting the lateral LV wall, is usually part of a broader infarct but may be isolated. It is due to occlusion of LAD or LCx branches. ECG findings include ST elevation in leads I, aVL, V5–V6, and reciprocal depression in inferior leads (12).

1.5. Inferior Wall Myocardial Infarction

IWMI is commonly due to RCA occlusion (80%) and less often due to LCx. ST elevation in lead III > lead II and depression in I and aVL favor RCA as the culprit (16, 17). ST elevation in V1 may suggest concurrent RVMI (12). Posterior involvement is indicated by ST depression in V1–V2 (12). ECG sensitivity improves with greater ST deviation and early recording (19, 20).

1.5.1. Right Ventricular Myocardial Infarction

RVMI often accompanies IWMI, occurring in 24–50% of cases depending on detection method (22). RV infarction is less common due to its lower oxygen demand and rich collateral blood flow (22, 23).

Diagnosis involves observing hypotension, JVD, and clear lungs on physical exam. ST elevation in lead III > II and right precordial leads, especially V4R, are diagnostic (27, 28, 29). Echocardiography, radionuclide imaging, and hemodynamic studies also aid diagnosis (25, 26, 22).

Angiography typically shows RCA occlusion proximal to the acute marginal branch. LCx may be involved in left-dominant circulation (22).

Complications include increased arrhythmias and conduction blocks. RVMI has a higher risk of AV block, bradycardia, and life-threatening arrhythmias compared to other infarct locations (22, 30).

Therapy focuses on maintaining RV preload with cautious volume loading (31, 32, 33). Hemodynamic monitoring is recommended (22). Early reperfusion improves clinical outcomes and RV recovery (34, 35). Intra-aortic balloon counterpulsation may support circulation in refractory cases (34).

Prognosis is poorer with RV involvement, with higher rates of complications and mortality compared to IWMI without RVMI (22, 30). Some studies report no long-term mortality difference, indicating that most adverse outcomes occur during hospitalization (36, 37).

This study aimed to determine the prevalence of right ventricular infarction among patients with inferior wall myocardial infarction and to identify clinical and diagnostic characteristics associated with right ventricular involvement.

PATIENT and METHOD

Study Design and Setting

This cross-sectional study was conducted in the Coronary Care Unit (CCU) of Azadi Teaching Hospital in Kirkuk, from January 1 to October 31, 2017.

Study Population

The study included all patients admitted to the CCU with a confirmed diagnosis of inferior wall myocardial infarction (MI).

Inclusion Criteria

- Patients diagnosed with inferior wall myocardial infarction.

Exclusion Criteria

- Patients with anterior wall myocardial infarction.
- Patients with double wall infarctions.

Sampling Method

A convenient sample of 150 patients with inferior wall MI was selected based on their willingness to participate and provide verbal consent.

Data Collection

Data were collected through direct interviews using a structured questionnaire developed by the investigator. The questionnaire included the following domains:

1. **Socio-demographic data:** Age, gender, and occupation.
2. **Clinical risk factors:** Smoking status, hypertension, diabetes mellitus, previous ischemic heart disease (IHD), family history of cardiac diseases, and obesity.

3. **Clinical examination findings:** General appearance, baseline pulse rate, blood pressure, and jugular venous pressure (JVP).
4. **Clinical outcome:** Recovery or complication status.
5. **Right ventricular infarction (RVI):** Number of cases diagnosed among the study population.

Diagnosis of inferior wall MI was made by a specialist in internal medicine based on clinical evaluation, ECG findings, and cardiac biomarkers. Electrocardiograms were performed using the GE MAC 1600 system, and echocardiographic assessments were conducted by a certified physician using a Philips CX50 machine. The diagnosis of RVI was made by the same specialist based on clinical examination and ECG findings.

Statistical Analysis

Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 22. Descriptive statistics were presented as means \pm standard deviation for continuous variables and as frequencies and percentages for categorical variables. Data normality was assessed using the Kolmogorov–Smirnov test. The Chi-square test was applied to compare categorical variables (Fisher’s exact test was used when expected counts were less than 5). The independent sample t-test was used to compare means between two groups. A p-value of ≤ 0.05 was considered statistically significant.

RESULTS

This study included a total of 150 patients diagnosed with inferior wall myocardial infarction (MI) and admitted to the Coronary Care Unit (CCU). The mean age of the participants was 60.4 ± 7.8 years. Age distribution revealed that 6.7% of patients were in the 40–49-year age group, 24% were between 50–59 years, the majority (56%) were aged 60–69 years, and 13.3% were aged 70 years or older.

Regarding gender distribution, males were predominant, with a male-to-female ratio of 3.5:1.

As for occupational status, 22% of the patients were housewives, 5.3% were public servants, 37.4% were self-employed, and 35.3% were retired.

These demographic and occupational findings are summarized in Table 1 and illustrated in Figures 1 and 2.

Table 1: Socio-demographic characteristics of inferior wall MI patients

Variable	No.	%
Age (mean \pm SD = 60.4 \pm 7.8 years)		
40–49 years	10	6.7
50–59 years	36	24.0
60–69 years	84	56.0
\geq 70 years	20	13.3
Total	150	100.0
Gender		
Male	117	78.0
Female	33	22.0
Total	150	100.0
Occupation		
Housewife	33	22.0
Public servant	8	5.3
Self employed	56	37.4
Retired	53	35.3
Total	150	100.0

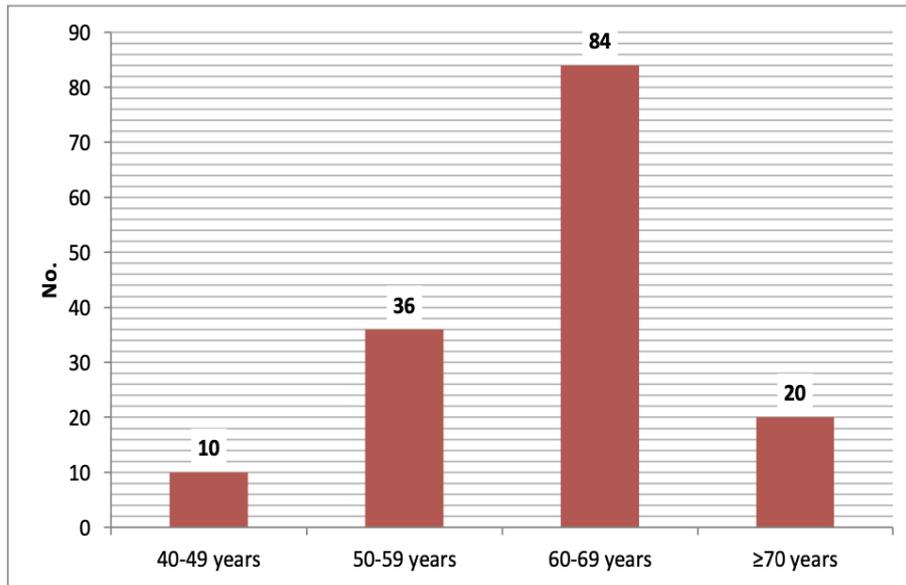


Figure 1: Age distribution.

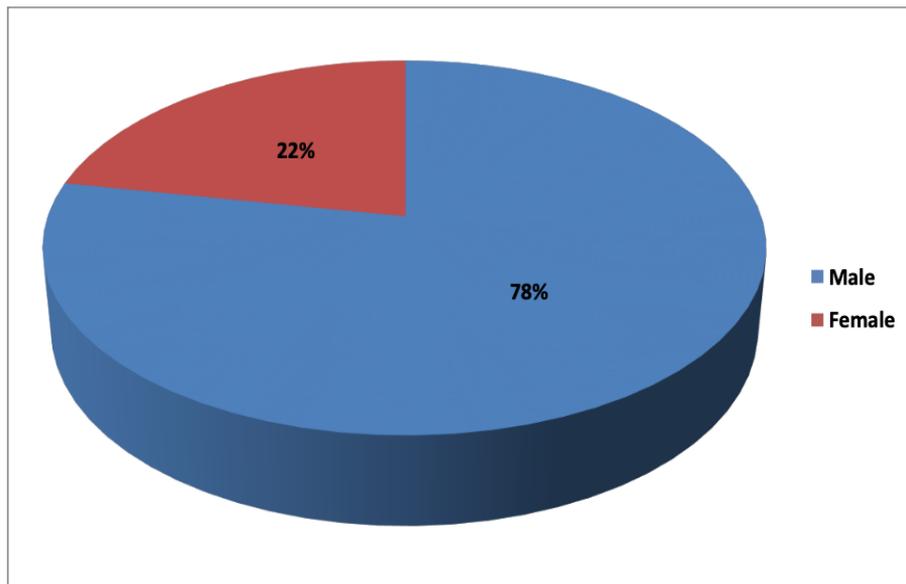


Figure 2: Gender distribution.

Table 2: Clinical risk factors of inferior wall MI patients.

Variable	No.	%
Smoking		
Yes	119	79.3
No	31	20.7
Total	150	100.0
HT		
Yes	55	36.7
No	95	63.3
Total	150	100.0
DM		
Yes	83	55.3
No	67	44.7
Total	150	100.0
Old IHD		
Yes	1	0.7
No	149	99.3
Total	150	100.0
Family history of heart diseases		
Positive	12	8.0
Negative	138	92.0
Total	150	100.0
Obesity		
Yes	4	2.7
No	146	97.3
Total	150	100.0

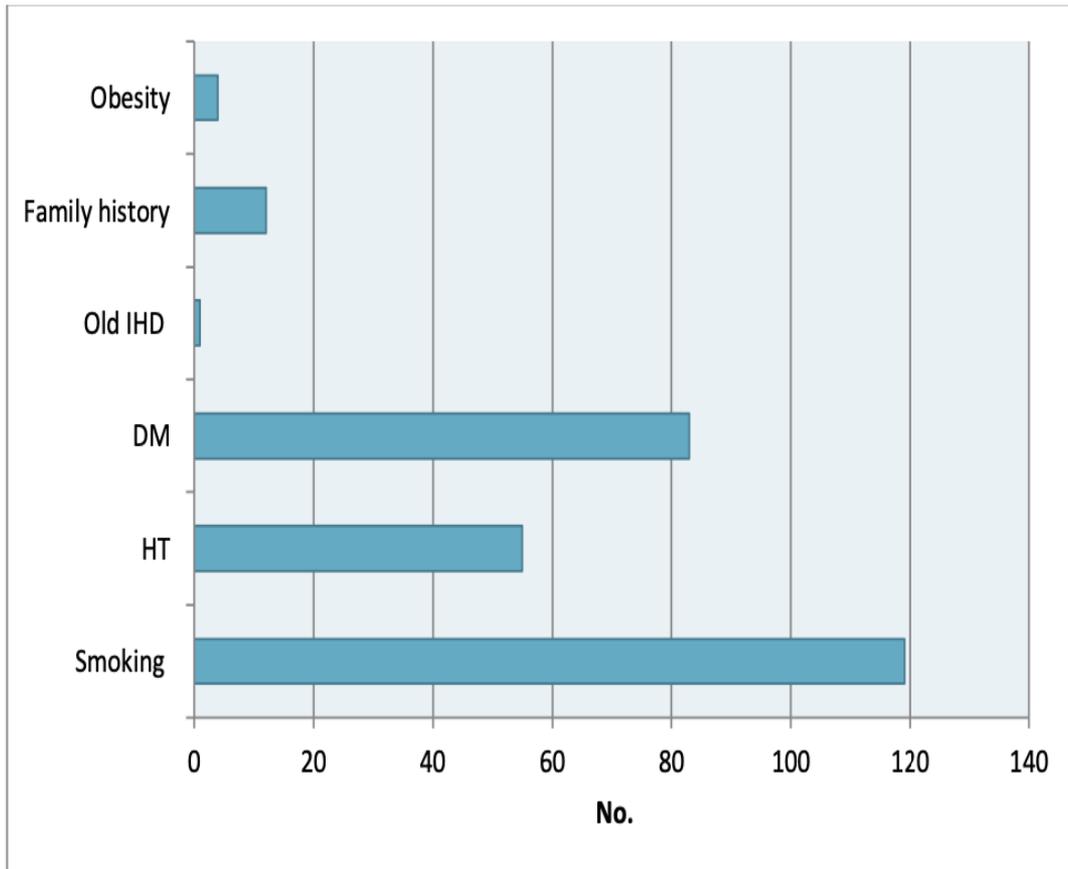


Figure 3: Risk factors of inferior wall MI patients.

All patients with inferior wall myocardial infarction (MI) were conscious at presentation. The mean baseline pulse rate (PR) was 76.9 ± 15.5 beats per minute, with 8.7% exhibiting bradycardia and 12% presenting with tachycardia. The average blood pressure was $128/78.4 \pm 22.3/15.5$ mmHg; hypotension (BP $<100/50$ mmHg) was observed in 18% of patients, while 2.7% had hypertension (BP $>140/90$ mmHg). Elevated jugular venous pressure (JVP) was noted in 12% of cases. These clinical findings are detailed in Table 3 and illustrated in Figure 4.

Table 3: Examination results of inferior wall MI patients.

Variable	No.	%
General examination		
Conscious	150	100.0
Total	150	100.0
Baseline PR mean±SD (76.9±15.5 b/m)		
Slow	13	8.7
Normal	119	79.3
Rapid	18	12.0
Total	150	100.0
BP mean±SD (128/78.4±22.3/15.5 mmHg)		
Low	27	18.0
Normal	119	79.3
High	4	2.7
Total	150	100.0
JVP		
Normal	132	88.0
Increased	18	12.0
Total	150	100.0

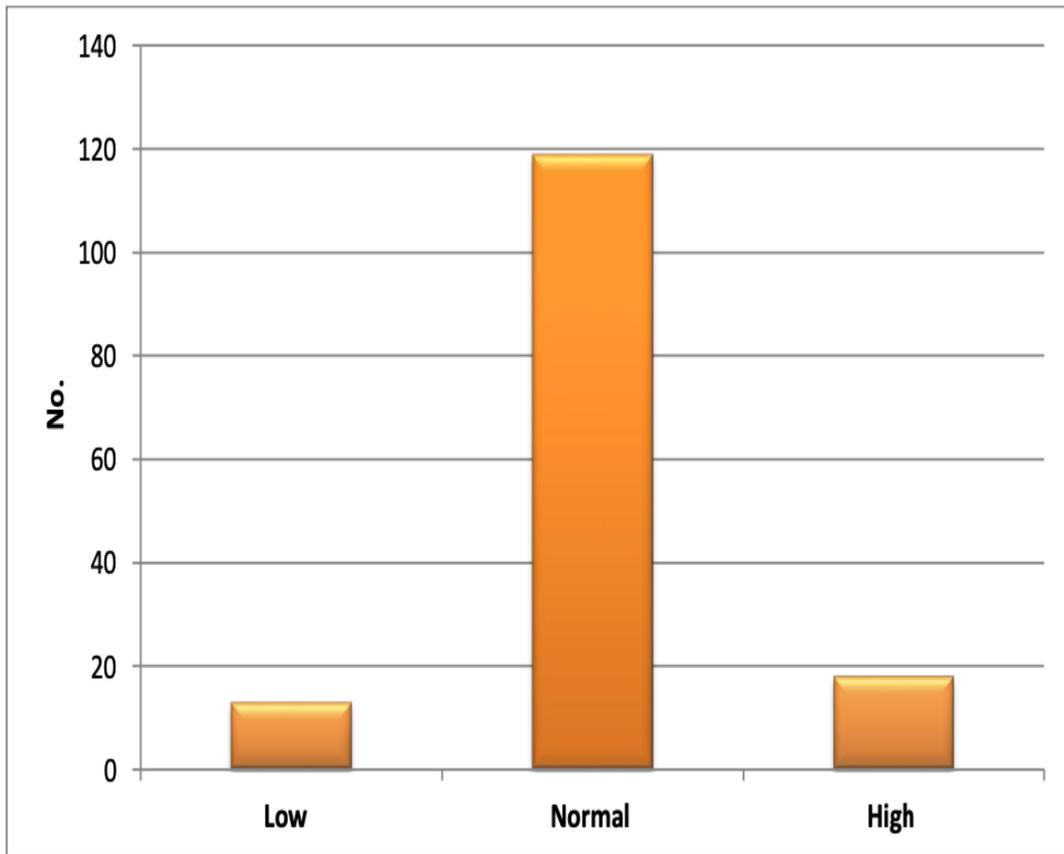


Figure 4: Baseline PR of inferior wall MI patients.

All patients with inferior wall myocardial infarction (MI) exhibited ST-segment elevation in leads II, III, and aVF on baseline electrocardiography (ECG). Additionally, 39.3% of patients showed ST elevation in the right precordial leads (V_{3R}, V_{4R}, V_{5R}, V_{6R}), indicating right ventricular involvement.

A positive troponin test was observed in 78.7% of patients, while the CK-MB test was positive in 14% of cases. Echocardiographic evaluation demonstrated inferior wall hypokinesia in 62% of patients. These diagnostic findings are summarized in Table 4 and illustrated in Figure 5.

Table 4: Investigation results of inferior wall MI patients.

Variable	No.	%
Baseline ECG: ST elevation in lead II, III, aVF		
Yes	150	100.0
Total	150	100.0
Baseline ECG: ST elevation in lead V3R, V4R, V5R, V6R		
Yes	59	39.3
No	91	60.7
Total	150	100.0
Troponin		
Positive	118	78.7
Negative	32	21.3
Total	150	100.0
CK-M		
Positive	21	14.0
Negative	129	86.0
Total	150	100.0
Echocardiography findings		
Normal	57	38.0
Inferior wall hypokinesia	93	62.0
Total	150	100.0

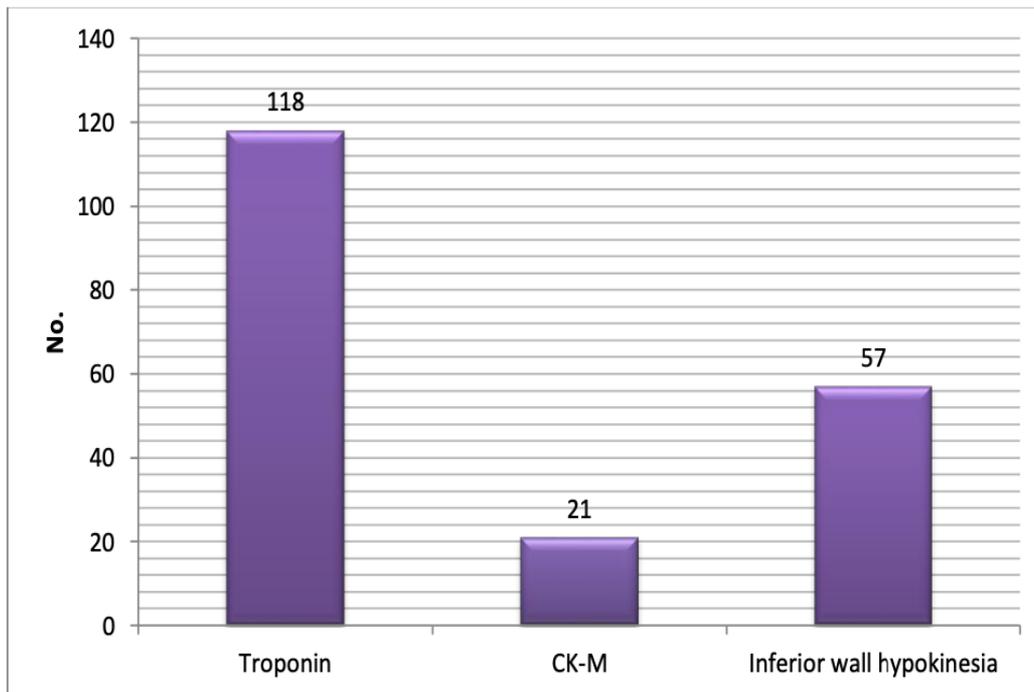


Figure 5: Investigation results.

A favorable outcome was observed in 72.7% of patients with inferior wall myocardial infarction (MI). Complications included cardiogenic shock in 9.2% of patients, atrial fibrillation in 4%, bradycardia in 4%, heart failure in 2.7%, ventricular fibrillation in 2.7%, and hypotension in 2.7%. Additionally, 2% of patients experienced a combination of bradycardia and hypotension. Right ventricular infarction was identified in 39.3% of patients. These clinical outcomes are summarized in Table 5 and illustrated in Figure 6.

Table 5: Outcome of inferior wall MI patients.

Variable	No.	%
Outcome		
Normal	109	72.7
AF	6	4.0
Heart failure	4	2.7
Bradycardia	6	4.0
VF	4	2.7
Hypotension	4	2.7
Bradycardia and hypotension	3	2.0
Cardiogenic shock	14	9.2
Total	150	100.0
Right ventricular infarction (RVI)		
Present	59	39.3
Absent	91	60.7
Total	150	100.0

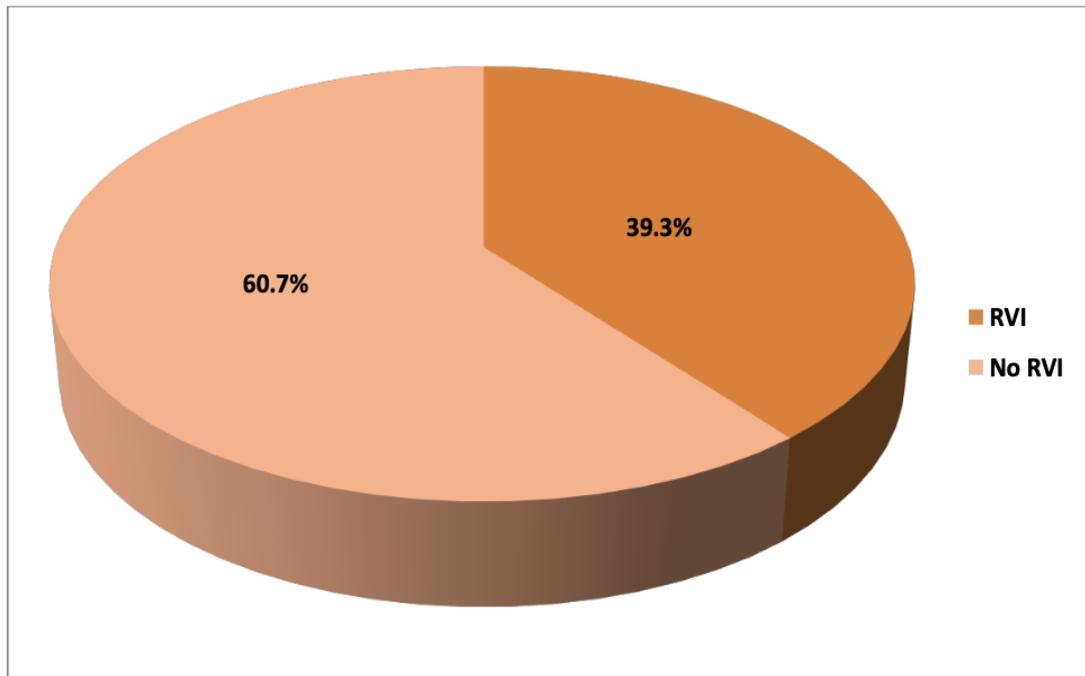


Figure 6: Prevalence of RVI among inferior wall MI patients.

A highly significant association was found between older age and the presence of right ventricular infarction (RVI) among patients with inferior wall myocardial infarction (MI) ($p < 0.001$). No statistically significant difference was observed between patients with and without RVI in terms of gender. However, a strong association was noted between self-employment status and the presence of RVI ($p < 0.001$). These findings are detailed in Table 6 and illustrated in Figure 7.

Table 6: Distribution of socio-demographic characteristics of MI patients

according to RVI.

Variable	RVI		No RVI		χ^2	P
	No.	%	No.	%		
Age					33.5	< 0.001*
40–49 years	10	16.9	44	48.4		
50–59 years	25	42.4	41	45.0		
60–69 years	13	22.0	6	6.6		
≥70 years	11	18.6	0	-		
Gender					0.6	0.4**
Male	44	74.6	73	80.2		
Female	15	25.4	18	19.8		
Occupation					25.8 ^f	< 0.001*
Housewife	15	25.4	18	19.8		
Public servant	8	13.6	0	-		
Self employed	27	45.8	29	31.9		
Retired	9	15.3	44	48.4		

*Significant, **Not significant, ^fFishers exact test.

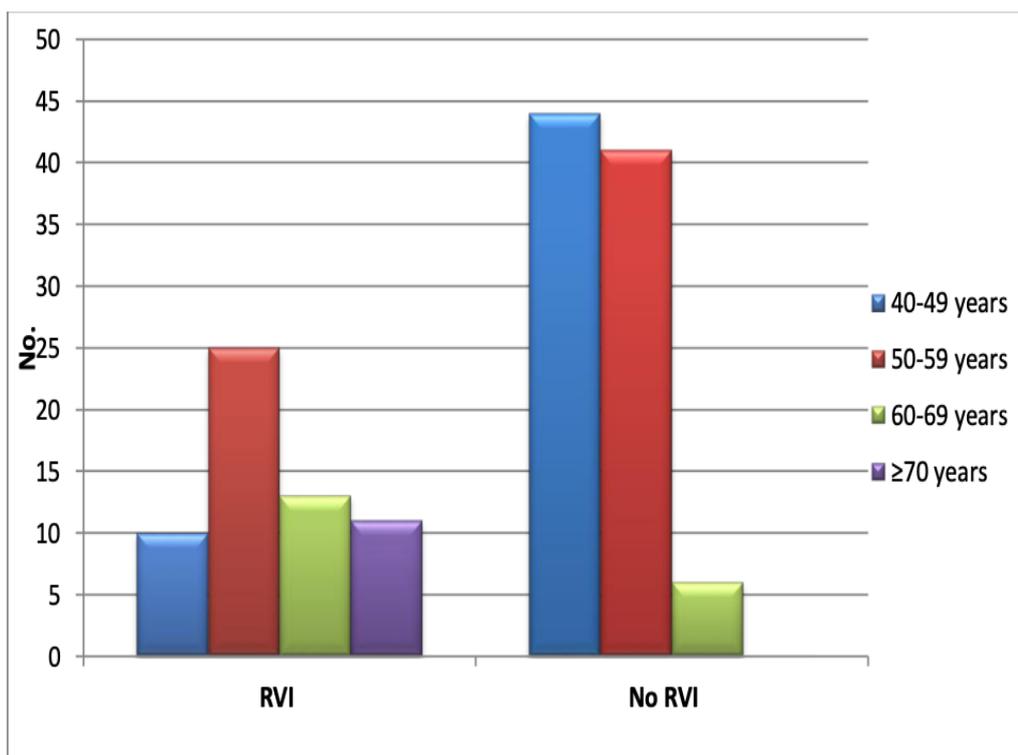


Figure 7: Distribution of age according to RVI.

There were no statistically significant differences in the prevalence of smoking or a history of ischemic heart disease (IHD) between inferior wall myocardial infarction (MI) patients with and without right ventricular infarction (RVI). However, hypertension and diabetes mellitus were significantly associated with the absence of RVI ($p < 0.001$ for both). Notably, a family history of heart disease and the presence of obesity emerged as significant risk factors for RVI among patients with inferior wall MI ($p < 0.001$ and $p = 0.01$, respectively). These findings are summarized in Table 7 and illustrated in Figure 8.

Table 7: Distribution of clinical risk factors of MI patients according to RVI.

Variable	RVI		No RVI		χ^2	P
	No.	%	No.	%		
Smoking					0.1	0.7**
Yes	46	78.0	73	80.2		
No	13	22.0	18	19.8		
HT					29.4	< 0.001*
Yes	6	10.2	49	53.8		
No	53	89.8	42	46.2		
DM					18.1	< 0.001*
Yes	20	33.9	63	69.2		
No	39	66.1	28	30.8		
Old IHD					1.5 ^f	0.2**
Yes	1	1.7	0	-		
No	58	98.3	91	100.0		
Family history of heart diseases					20.1 ^f	< 0.001*
Positive	12	20.3	0	-		
Negative	47	79.7	91	100.0		
Obesity					6.3 ^f	0.01*
Yes	4	6.8	0	-		
No	55	93.2	91	100.0		

*Significant, **Not significant, ^fFishers exact test.

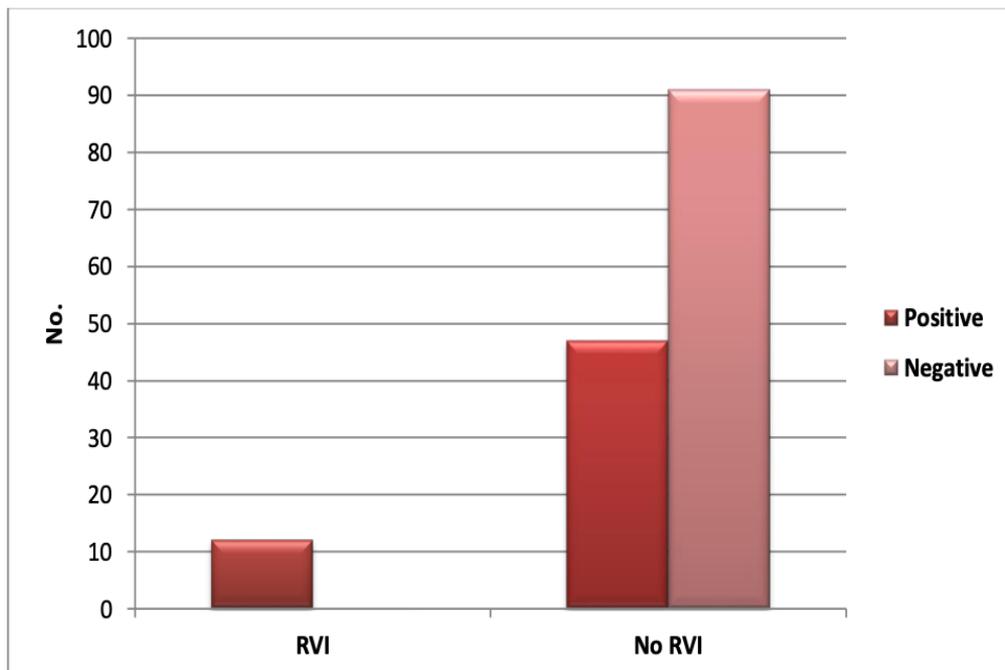


Figure 8: Distribution of family history of heart diseases according to RVI.

A significant association was observed between bradycardia (slow pulse rate) and the presence of right ventricular infarction (RVI) in patients with inferior wall myocardial infarction (MI) ($p = 0.003$). Additionally, low blood pressure was significantly associated with RVI in these patients ($p < 0.001$). A highly significant relationship was also noted between elevated jugular venous pressure (JVP) and the presence of RVI ($p < 0.001$). These findings are summarized in Table 8 and illustrated in Figure 9.

Table 8: Distribution of examination results of MI patients according to RVI.

Variable	RVI		No RVI		χ^2	P
	No.	%	No.	%		
Baseline PR					11.5	0.003*
Slow	10	16.9	3	3.3		
Normal	46	78.0	73	80.2		
Rapid	3	5.1	15	16.5		
BP					2.8	< 0.001*
Low	22	37.3	5	5.5		
Normal	37	62.7	77	84.6		
High	0	-	9	9.9		
JVP					22.7 ^f	< 0.001*
Normal	41	69.5	91	100.0		
Increased	18	30.5	0	-		

**Significant, **Not significant, ^FFishers exact test.*

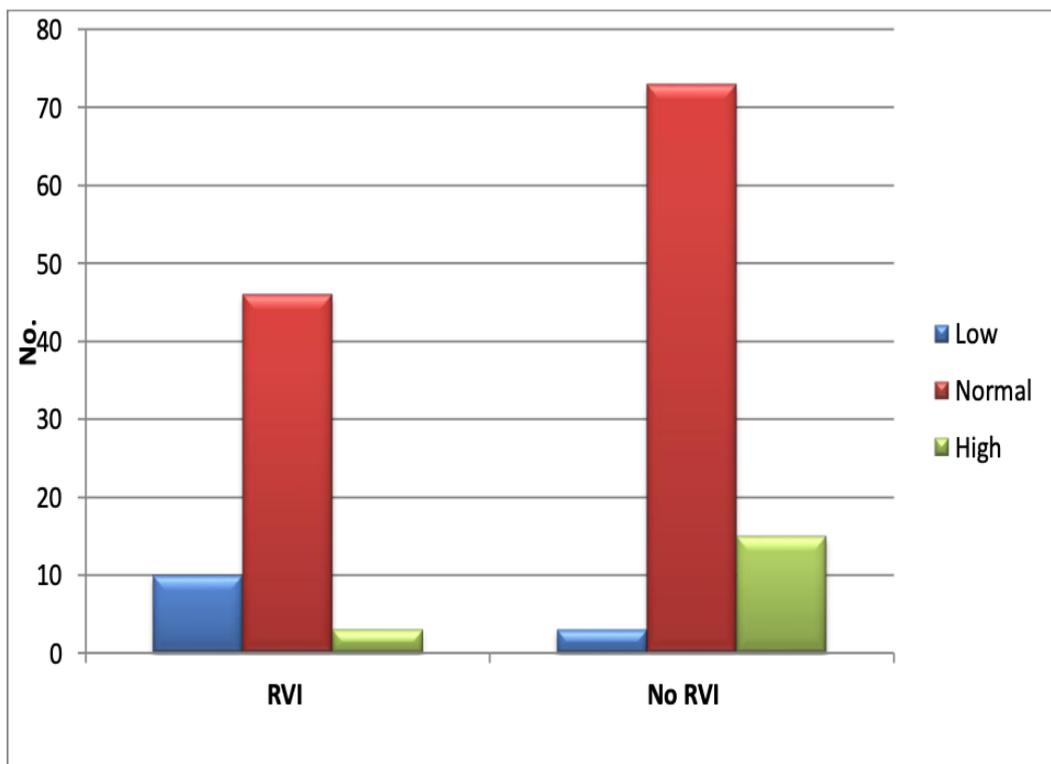


Figure 9: Distribution of baseline PR according to RVI.

ST-segment elevation in leads V3R, V4R, V5R, and V6 on baseline ECG was significantly more common among patients with right ventricular infarction (RVI) ($p < 0.001$). Conversely, a positive troponin test was significantly associated with inferior wall myocardial infarction (MI) patients without RVI ($p < 0.001$). In contrast, CK-MB positivity was significantly more frequent in patients with RVI ($p = 0.001$). Echocardiographic evidence of inferior wall hypokinesia was significantly higher among MI patients without RVI ($p = 0.02$). These findings are summarized in Table 9.

Table 9: Distribution of investigation results of MI patients according to RVI.

Variable	RVI		No RVI		χ^2	P
	No.	%	No.	%		
Baseline ECG: ST elevation in lead V3R, V4R, V5R, V6					150	< 0.001*
Yes	59	100.0	0	-		
No	0	-	91	100.0		
Troponin					62.7	< 0.001*
Positive	27	45.8	91	100.0		
Negative	32	54.2	0	-		
CK-M					10.5	0.001*
Positive	15	25.4	6	6.6		
Negative	44	74.6	85	93.4		
Echocardiography findings					5.1	0.02*
Normal	29	49.2	28	30.8		
Inferior wall hypokinesia	30	50.8	63	69.2		

*Significant.

Patients with inferior wall myocardial infarction (MI) who had right ventricular infarction (RVI) experienced significantly more complicated outcomes compared to those without RVI ($p = 0.002$). These findings are summarized in Table 10.

Table 10: Distribution of outcome of MI patients according to RVI.

Outcome	RVI	RVI	No RVI		χ^2	P
	No.	%	No.	%		
Normal	33	55.9	76	83.5	22.7 ^f	0.002*
AF	0	-	6	6.6		
Heart failure	3	5.1	1	1.1		
Bradycardia	3	5.1	3	3.3		
VF	0	-	4	4.4		
Hypotension	3	5.1	1	1.1		
Bradycardia and hypotension	3	5.1	0	-		
Cardiogenic shock	14	23.7	0	-		

*Significant, ^FFishers exact test.

As presented in Table 11 and Figure 10, a significant association was found between higher mean age and the presence of right ventricular infarction (RVI) ($p < 0.001$). A highly significant relationship was also observed between slower baseline pulse rate and RVI ($p < 0.001$). Furthermore, the mean systolic and diastolic blood pressures were significantly lower in MI patients with RVI compared to those without RVI ($p < 0.001$ and $p = 0.001$, respectively).

Table 11: Distribution of patients characteristics means according to RVI.

Variable	RVI Mean±SD	No RVI Mean±SD	t-test	P
Age	62.7±5.2	56.8±9.6	2.2	<0.001*
Baseline PR	68.7±15.1	82.2±13.3	8.1	<0.001*
Systolic BP	118.5±19.4	134.1±22.1	15.7	<0.001*
Diastolic BP	73.4±14.2	81.6±15.5	15.7	0.001*

**Significant.*

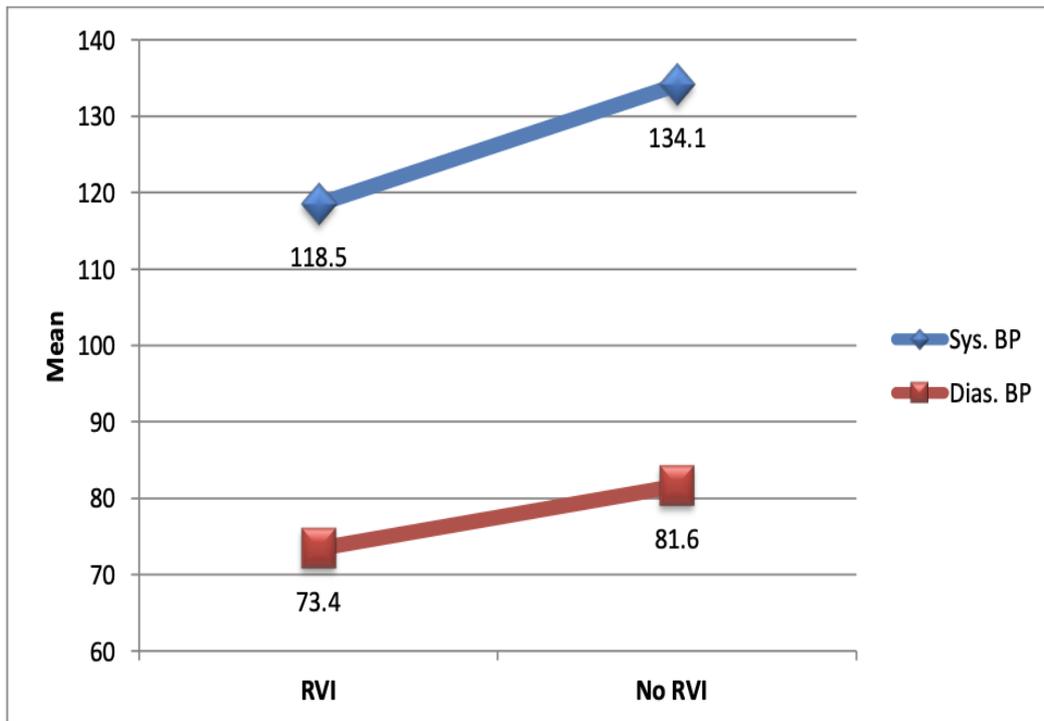


Figure 10: Distribution of BP according to RVI.

DISCUSSION

Myocardial infarction (MI) remains a major public health concern, with its prevalence in developing countries now approaching that seen in developed nations. The majority of MI-related deaths result from underlying pathophysiological changes and complications. Prompt recognition of MI type and immediate, effective management are essential to reduce morbidity and mortality (38).

In the current study, the prevalence of right ventricular infarction (RVI) among patients with inferior wall MI was 39.3%. This finding is comparable to the 40% prevalence reported by Ravikeerthy et al. in India (39). However, our results are lower than those reported in studies from Iran (75.8%) (40) and Pakistan (48.5%) (41), but higher than the prevalence reported by Azhar et al. in Saudi Arabia (30%) (42). These variations may be attributed to differences in sample size, study design, diagnostic criteria, healthcare quality, and the severity of MI across populations.

In Iraq, Sallman (43) found that patients with inferior MI complicated by RVI had significantly higher complication and mortality rates. Similarly, a meta-analysis by

Hamon et al. (44) in France confirmed that RVI is frequently associated with serious complications such as ventricular arrhythmias, cardiogenic shock, atrioventricular (AV) block, and mechanical issues. Reported prevalence rates of RVI among inferior MI patients range from 10% to 50% (45).

Isolated RVI, however, is rare, with an estimated prevalence of 3–5%. This rarity is likely due to the right ventricle's lower susceptibility to ischemia, which stems from its thin wall, lower muscle mass, and dual perfusion during both systole and diastole (46). Studies employing different diagnostic approaches have reported RVI prevalence as high as 50% (47). The anatomical explanation for the frequent coexistence of RVI with inferior MI lies in the shared blood supply from the right coronary artery to the inferior wall of the left ventricle and the posterior wall of the right ventricle (48). Additional factors limiting isolated RVI include a well-developed intercoronary collateral network and the “throttling” effect of left ventricular contraction (49).

Hemodynamic instability in inferior MI may be exacerbated by the presence of RVI and left ventricular dysfunction (50). Despite the high mortality associated with RVI, it remains underrecognized. Reasons for this include underappreciation of RVI's

hemodynamic effects, the rapid and severe deterioration in some patients, and the masking of RVI symptoms by concurrent left ventricular dysfunction (51, 52).

The use of right-sided precordial ECG leads is essential in detecting RVI. ST-segment elevation of ≥ 1.0 mm in lead V4R has demonstrated 100% sensitivity, 87% specificity, and 92% predictive accuracy in identifying RVI (53). However, a key limitation is that such elevation may be absent in up to 50% of RVI cases within 12 hours of symptom onset. Furthermore, the presence of ST elevation in V4R is often correlated with reduced right ventricular ejection fraction (54).

Our study found a highly significant association between advanced age and the presence of RVI in inferior MI patients ($p < 0.001$). This is consistent with findings from Bueno et al. in Spain (55), who reported an age-related increase in both the prevalence and complications of RVI. In contrast, Kukla et al. in Poland found no significant age difference between inferior MI patients with and without RVI (56), a discrepancy that may stem from differences in sample sizes or study designs.

No significant gender-based differences were observed in our study, contradicting the findings of Obradovic et al. in Serbia, who reported a higher RVI risk among female patients (57).

Self-employed patients were found to have a significantly higher risk of developing RVI ($p < 0.001$). This finding is in line with Ginghina et al. in Romania (58), who highlighted physical labor, psychological stress, and low socioeconomic status as key RVI risk factors.

Smoking was not significantly associated with RVI in our study, which aligns with Zornoff et al. (59). However, Alemu et al. (60) reported that smoking significantly increases the risk of inferior MI.

Neither hypertension nor diabetes mellitus showed a significant association with RVI in our cohort, contrasting with findings from Akram et al. in Iran (61) and Roifman et al. in Canada (62), who identified both conditions as major RVI risk factors. This difference may be due to the high prevalence of these comorbidities among the overall patient population in our study.

A significant association was found between family history of heart disease and the presence of RVI in inferior MI patients ($p < 0.001$), consistent with findings by Namana et al. in the United States (63). Ravikeerthy et al. also found a positive family history to be a risk factor for isolated RVI (39).

Obesity was another significant risk factor for RVI in our study ($p = 0.01$). This is in agreement with Lavie et al. (64), who found that obesity contributes significantly to RVI risk and that weight reduction can lower this risk by more than 50%.

Clinically, RVI was characterized by a triad of bradycardia, hypotension, and elevated jugular venous pressure—findings that mirror those reported by Ondrus et al. in the Czech Republic and Khalid et al. in Pakistan (65, 66). Niaki et al. (40) also described similar symptoms, including chest pain, altered consciousness, and hypotension.

ECG findings in our RVI patients included significant ST-segment elevation in leads V3R, V4R, V5R, and V6R ($p < 0.001$), supporting results from Jeremiah (67), who described these changes as pathognomonic for RVI in inferior MI.

Biochemically, elevated CK-MB levels were significantly associated with RVI ($p = 0.001$), as supported by Shemirani et al. (68), who advocated CK-MB testing as a key

diagnostic tool for isolated RVI. In contrast, troponin levels and echocardiographic findings were more often positive in inferior MI patients without RVI in our study.

Nonetheless, the European Society of Cardiology recommends the use of both troponins and echocardiography in diagnosing RVI and related complications (69).

Our results showed that complications were significantly more common in patients with RVI, particularly cardiogenic shock ($p = 0.002$). This finding is consistent with studies by Inohara et al. in Japan (70) and Bari et al. (71), both of which documented elevated morbidity and mortality rates in inferior MI patients with RVI.

CONCLUSION

This study found that the prevalence of right ventricular infarction (RVI) among patients with inferior myocardial infarction (MI) falls within the expected range reported in previous literature. RVI was notably more frequent in older patients, suggesting age as a significant contributing factor. Among the key risk factors associated with RVI were a positive family history of heart disease and obesity, both of which were significantly more common in affected individuals. Clinically, patients with RVI presented with a characteristic triad of slow pulse rate, hypotension, and elevated jugular venous pressure. Electrocardiography, particularly using right-sided precordial leads, proved to be an effective diagnostic tool. Furthermore, the study highlighted a high rate of complications in patients with RVI, with cardiogenic shock emerging as the most frequently observed and serious adverse outcome. These findings underscore the importance of early recognition and targeted management of RVI in the context of inferior MI to improve clinical outcomes.

RECOMMENDATIONS

It is essential to provide targeted instruction to junior doctors working in coronary care units and emergency departments on the prevalence, clinical features, and diagnostic importance of right ventricular infarction (RVI) in the context of inferior myocardial infarction (MI). Enhancing their awareness will promote early recognition and appropriate management of this condition. Junior doctors should also be trained to consider RVI as a potential cause of hypotension in MI patients, as its management—particularly with intravenous fluid resuscitation—differs from other causes of hypotension. Additionally, improving the infrastructure and resources of coronary care units is crucial to reduce the risk of complications associated with RVI. Finally, further multi-center research should be encouraged and supported to provide more comprehensive data on the prevalence, diagnosis, and outcomes of RVI in inferior MI patients.

Ethical Clearance:

- Institutional approval was obtained from the administration of Azadi Teaching Hospital.
- Verbal informed consent was obtained from all patients.
- Patients were managed ethically and respectfully by the investigator and supervisor.

Financial support and sponsorship:

Nil.

Conflicts of interest:

There are no conflicts of interest.

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