Section: General Medicine

Original Research Article

INCIDENCE OF LEFT VENTRICULAR DYSFUNCTION FOLLOWING ACUTE MYOCARDIAL INFARCTION: A CROSS-SECTIONAL ANALYSIS

Pavankumar Annappa Magadum[1], Rajashekhar[2], Mithun R[3], MS Manjesh[1]

[1]Junior Resident, Department of General Medicine, Adichunchanagiri Institute of Medical Sciences, Adichunchanagiri University, BG Nagara, 571448, Nagamangala, Mandya, Karnataka, India
[2]Professor, Department of General Medicine, Adichunchanagiri Institute of Medical Sciences, Adichunchanagiri University, BG Nagara, 571448, Nagamangala, Mandya, Karnataka, India
[3]Assistant Professor, Department of General Medicine, Adichunchanagiri Institute of Medical Sciences, Adichunchanagiri University, BG 571448, Nagara, Nagamangala, Mandya, Karnataka, India

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Corresponding Author:
Dr. Mithun R.,
Assistant Professor, Department of General Medicine, Adichunchanagiri Institute of Medical Sciences, Adichunchanagiri University, BG Nagara, Nagamangala, Mandya, Karnataka, India.
Email: mithunr0000@gmail.com

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ABSTRACT

Background: Acute myocardial infarction (AMI) remains an important contributor to cardiovascular morbidity and mortality. Left ventricular dysfunction (LVD) is one of the important complications seen in patients of AMI. Despite advancements in revascularization procedures and pharmacotherapy LVD is seen in many cases and significantly affects prognosis. This study aimed to determine the incidence and severity of LVD in patients with AMI and its correlation advancing age of patients presenting with AMI.

Materials and Methods: A hospital-based cross-sectional observational study was conducted at Adichunchungiri Institute of Medical Sciences, India. 250 consecutive patients with confirmed AMI were included in this study on the basis of a predefined inclusion and exclusion criteria. Detailed clinical evaluation, ECG, cardiac biomarkers, and 2D transthoracic echocardiography were performed within 72 hours of admission. LVD was categorized based on left ventricular ejection fraction (LVEF) as: mild (40–49%), moderate (30–39%), or severe (<30%). Statistical analysis was done using SPSS 23.0. P value less than 0.05 was taken as statistically significant.

Results: Out of 250 patients there was a significant male preponderance in AMI cases with a M:F ratio of 1:0.38. Mean age of studied cases was found to be 66.25 ± 10.9 years. STEMI accounted for 66% and NSTEMI 34% of cases. Anterior wall MI was the most common type (42%). Hypertension (58%), obesity (52%) and diabetes mellitus (44%) were the predominant risk factors. LVD was present in 46% of patients out of which mild, moderate and severe LVD was seen in 20%, 16% and 10% respectively. A significant age-related increase in LVD incidence was observed. Pearson's correlation revealed a moderate positive association between age and LVD ($r = 0.41$, $p = 0.001$).

Conclusion: Early echocardiographic screening for LVD in cases of MI is essential for timely identification and initiation of appropriate management to prevent adverse outcomes.

Keywords: Myocardial Infarction, Left Ventricular Dysfunction, Echocardiography, Risk Factors.

INTRODUCTION

Acute myocardial infarction (AMI) is a leading cause of morbidity and mortality in elderly patients. It is usually the consequence of a sudden reduction or complete cessation of coronary blood flow secondary to the rupture of an atherosclerotic plaque followed by thrombus formation.[1] This abrupt interruption in perfusion causes ischemia and necrosis of the myocardial tissue. The extent and location of myocardial necrosis largely determine the clinical outcome including the risk of mechanical and

electrical complications. Several well-established risk factors contribute to the development of myocardial infarction. These factors include modifiable factors such as smoking, hypertension, diabetes mellitus, dyslipidemia and obesity, as well as non-modifiable factors like age, sex, and genetic predisposition. Among these, diabetes and hypertension play a particularly insidious role by promoting endothelial dysfunction, chronic inflammation and accelerated atherogenesis. Despite advancements in preventive cardiology the global burden of AMI remains high, particularly in low- and middle-income countries.[2]

The clinical presentation of acute myocardial infarction may vary widely from classic to silent infarctions. The most commonly reported symptom is retrosternal chest pain often described as a pressure-like sensation which typically radiates to the left arm, neck or jaw. Associated symptoms include diaphoresis, dyspnoea, nausea and a sense of impending doom. In some cases, particularly with inferior wall infarctions patients may present with abdominal discomfort, vomiting, or bradycardia. Silent myocardial infarctions are more frequent in patients with autonomic neuropathy and may go undetected until complications ensue. Electrocardiographic changes such as ST-segment elevation, new-onset left bundle branch block or pathological Q waves are also important diagnostic clues. Cardiac biomarkers like troponins confirms myocardial necrosis.[3]

Acute myocardial infarction is often complicated by a spectrum of immediate and early complications that can significantly influence prognosis. These include arrhythmias (ventricular tachycardia, fibrillation, heart block), cardiogenic shock, pericarditis and papillary muscle dysfunction leading to acute mitral regurgitation. Among these, left ventricular dysfunction (LVD) stands out as one of the most significant complications in cases of AMI. Extensive Myocardial damage resulting in impaired contractility is the basic pathology behind LVF in cases of MI. This loss of systolic function may be regional or global depending on the infarct size and location. In severe cases LVF can lead to reduced cardiac output, hypotension, and development of heart failure.[4]

Left ventricular dysfunction in cases of AMI can manifest as either systolic dysfunction (reduced ejection fraction) or diastolic dysfunction (impaired ventricular filling). Echocardiography remains the cornerstone for diagnosing and quantifying LVD and helps in guiding further management.[5] LVF may activate renin-angiotensin-aldosterone system (RAAS) and sympathetic nervous system and activation of these systems further exacerbates myocardial remodeling and progression to chronic heart failure. LVF may present with exertional dyspnoea, orthopnoea and fatigue. Importantly, patients with post-infarction LVD have a significantly higher risk of recurrent ischemic events, hospital readmissions and mortality.[5]

The management of LVD following AMI is usually aimed at preventing further myocardial injury, supporting cardiac function and mitigating adverse remodeling. Early revascularization through percutaneous coronary intervention (PCI) is known to control damage to myocardium.[7] Pharmacological therapy including use of beta-blockers, angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) and sodium-glucose cotransporter-2 (SGLT2) inhibitors. All of these pharmacological agents have demonstrated benefit in reducing mortality and improving left ventricular function. Diuretics are used for symptom control in volume-overloaded patients. Selected patients with severe LVD may benefit from cardiac resynchronization therapy (CRT) or implantable cardioverter-defibrillators (ICDs) to prevent sudden cardiac death.[8]

While the pathophysiology, clinical features, and broad management strategies of AMI and its complications are well-documented, the true incidence and early detection of LVD following AMI remain underexplored. This study intends to fill these knowledge gaps by providing a contemporary, cross-sectional analysis of the incidence of left ventricular dysfunction following acute myocardial infarction.

MATERIALS AND METHODS

This was a hospital-based, cross-sectional observational study conducted in the Department of Internal Medicine, Adichunchungiri Institute of Medical Sciences, India. The study was carried out after obtaining approval from the Institutional Ethics Committee (IEC). The study adhered to the ethical principles laid out in the Declaration of Helsinki. Written informed consent was obtained from all participating patients.A total of 250 consecutive patients diagnosed with acute myocardial infarction (AMI), irrespective of age and gender, and admitted to the intensive care unit (ICU) were included in this study. The sample size was determined keeping a power of 80% (1-β error) and a 95% confidence interval (1-α error). Inclusion of 250 patients exceeded the minimum required sample size for adequate statistical validity.

A detailed clinical history was obtained including age, gender, occupation and socioeconomic status. Cardiovascular risk factors such as hypertension, diabetes mellitus, dyslipidemia, smoking, alcohol intake, obesity and family history of coronary artery disease. Duration and nature of chest pain, presence of dyspnea, palpitations and syncope were also documented. Physical examination including general and systemic examination was done. Particular attention was given to note the signs of volume overload such as presence of third or fourth heart sounds, murmurs, gallops or pedal edema. All patients underwent a comprehensive panel of investigations upon admission. A 12-lead electrocardiogram (ECG) was performed to identify

ischemic changes with serial ECGs obtained during the hospital stay as clinically indicated.

Diagnosis of acute myocardial infarction (AMI) was established based on a combination of clinical presentation, characteristic electrocardiographic (ECG) findings and elevated cardiac biomarkers (Troponin I or T). ST-elevation myocardial infarction (STEMI) was defined by the presence of new ST-segment elevation at the J-point in two or more anatomically contiguous leads with the following thresholds: ≥2.0 mm in men or ≥1.5 mm in women in leads V2–V3, and or ≥1.0 mm in other. Non-ST-elevation myocardial infarction (NSTEMI) was diagnosed in patients with ischemic symptoms and elevated troponin levels in the absence of ST-segment elevation, but with supportive ECG changes such as horizontal or downsloping ST-segment depression ≥0.5 mm, and/or T wave inversion ≥1.0 mm in two or more contiguous leads.9

Cardiac biomarkers (serum troponin I and T levels) were measured to confirm myocardial injury. Routine laboratory tests included complete blood count, renal and hepatic function tests and serum electrolytes were done in all cases. Fasting and postprandial blood glucose levels, lipid profile, and urine analysis was done to evaluate comorbidities and systemic status. Two-dimensional transthoracic echocardiography was conducted within 48 to 72 hours of admission using the modified Simpson's method to assess left ventricular ejection fraction (LVEF). The other findings such as any wall motion abnormalities, pericardial effusion, or valvular lesions were also documented.

Left ventricular function was assessed using 2D transthoracic echocardiography (2D-ECHO) in accordance with American Society of Echocardiography (ASE) guidelines.10 The left ventricular ejection fraction (LVEF) was calculated using the modified Simpson's biplane method. Based on LVEF values, patients were classified into categories of left ventricular dysfunction as follows [Table 1].

Table 1: Severity of Left Ventricular Dysfunction on the basis of Ejection Fraction.

Severity of LVD	LVEF (%)	Interpretation
Normal	≥ 50%	No left ventricular dysfunction
Mild LVD	40–49%	Borderline systolic dysfunction
Moderate LVD	30–39%	Moderate systolic dysfunction
Severe LVD	< 30%	Severe systolic dysfunction

The data regarding age and gender distribution, risk factors, presenting complaints, type and topography of MI and severity of left ventricular dysfunction was analysed. Microsoft excel was used to presenting the data. For statistical analysis SSPS 23.0 software was used. For statistical purposes P value less than 0.05 was taken as significant.

Inclusion Criteria

1. Patients of any age or gender admitted with a confirmed diagnosis of myocardial infarction.
2. Diagnosis of AMI established based on clinical presentation, characteristic electrocardiographic changes, and elevated cardiac biomarkers (Troponin I or T).
3. Patients who provided written informed consent.

Exclusion Criteria

1. Patients with previously diagnosed structural heart disease, including congenital heart disease, rheumatic valvular disease, or idiopathic cardiomyopathy.
2. Refusal to give informed consent.
3. Incomplete clinical, echocardiographic, or laboratory data.

RESULTS

The analysis of the gender distribution of the studied cases showed that males were the most commonly affected, accounting for 180 patients (72.0%), while females constituted 70 patients (28.0%). There was a significant male preponderance in AMI cases with a M:F ratio of 1:0.38 [Figure 1].

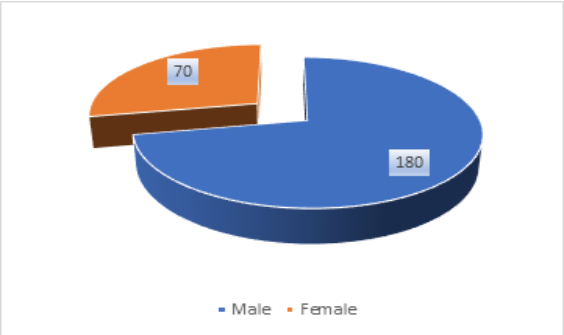

Figure 1: Gender Distribution of studied cases.

The analysis of the gender-wise age distribution among the studied cases showed that the mean age of female patients was higher at 68.3 years (±11.3), compared to male patients whose mean age was 64.2 years (±10.5). The overall mean age of the study population was 66.25 years (±10.9). The difference in mean age between males and females was found to be statistically significant ($p < 0.05$) [Table 2].

Table 2 : Gender-wise comparison of mean age in studied cases.

Gender	Number of Cases (n)	Mean Age (years)	Standard Deviation (SD)
Male	180	64.2	±10.5
Female	70	68.3	±11.3
Total	250	66.25	±10.9
P = 0.0071 (Significant)			

The analysis of the presenting symptoms among the studied cases showed that retrosternal chest pain, a typical symptom, was the most common presentation, observed in 190 patients (76.0%). This was followed by shortness of breath in 120 patients (48.0%), profuse sweating in 85 patients (34.0%), and radiation of pain to the left arm, jaw, or back in 70 patients (28.0%). Other symptoms included palpitations in 50 patients (20.0%), epigastric pain or burning (atypical) in 30 patients (12.0%), giddiness or syncope (atypical) in 15 patients (6.0%), and extreme fatigue or weakness (atypical) in 10 patients (4.0%) [Figure 2].

The analysis of the type of myocardial infarction among the studied cases showed that ST-Elevation Myocardial Infarction (STEMI) was the most common, observed in 165 patients (66.0%). This was followed by Non-ST-Elevation Myocardial Infarction (NSTEMI), which was seen in 85 patients (34.0%) [Table 3].

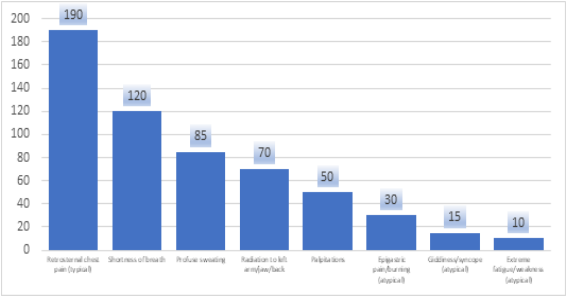

Figure 2: Presenting Complaints in studied cases.

Table 3: STEMI vs Non-STEMI in studied cases.

Type of Myocardial Infarction	Number of Cases	Percentage (%)
ST-Elevation Myocardial Infarction (STEMI)	165	66.0%
Non-ST-Elevation Myocardial Infarction (NSTEMI)	85	34.0%
Total Patients	250	100.0%

The analysis of the topographic type of myocardial infarction among the studied cases showed that anterior wall myocardial infarction was the most common (42.0%). This was followed by inferior wall MI in 85 patients (34.0%), posterior wall MI in 20 patients (8.0%), and both lateral wall MI and multi-territory/extensive MI in 15 patients each (6.0%). Septal MI was the least common, seen in 10 patients (4.0%) [Table 4].

Table 4: Topographic Type of Myocardial infarction in studied cases.

Topographic Type of MI	Number of Cases	Percentage (%)
Anterior wall MI	105	42.0%
Inferior wall MI	85	34.0%
Posterior wall MI	20	8.0%
Lateral wall MI	15	6.0%
Septal MI	10	4.0%
Multi-territory/Extensive MI	15	6.0%
Total Patients	250	100.0%

The analysis of the risk factors among the studied cases showed that hypertension was the most prevalent, present in 145 patients (58.0%), followed by obesity (BMI ≥25 kg/m²) in 130 patients (52.0%) and diabetes mellitus in 110 patients (44.0%). Other commonly observed risk factors included dyslipidemia in 95 patients (38.0%), current or past smoking in 90 patients (36.0%), and a sedentary lifestyle in 85 patients (34.0%). Additionally, 65 patients (26.0%) had a family history of coronary artery disease, 55 patients (22.0%) reported alcohol consumption, and stress or type A personality traits were noted in 40 patients (16.0%) [Figure 3].

The analysis of the severity of left ventricular dysfunction (LVD) among the studied cases showed that the majority had no LVD, with 135 patients (54.0%) having a left ventricular ejection fraction (LVEF) of ≥50%. Mild LVD (LVEF 40–49%) was observed in 50 patients (20.0%), followed by moderate LVD (LVEF 30–39%) in 40 patients (16.0%), while severe LVD (LVEF <30%) was present in 25 patients (10.0%) [Table 5].

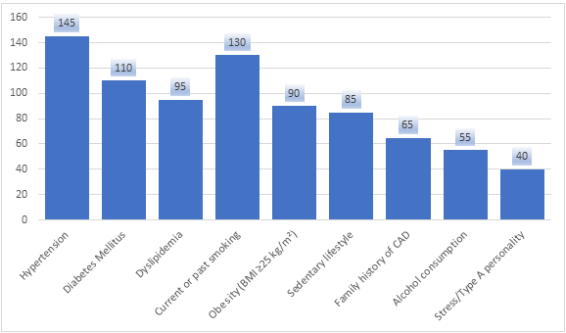

Figure 4: Analysis of Risk factors for MI in studied cases.

Table 5: Analysis of severity of LVD on the basis of LVEF in studied cases.

Severity of LVD	Number of Cases	Percentage (%)
No LVD (LVEF ≥ 50%)	135	54.0%
Mild LVD (LVEF 40–49%)	50	20.0%
Moderate LVD (LVEF 30–39%)	40	16.0%
Severe LVD (LVEF < 30%)	25	10.0%
Total Patients	250	100.0%

The analysis of left ventricular dysfunction (LVD) across different age groups among the studied cases showed an increasing trend with advancing age. The highest proportion of LVD was observed in patients aged over 70 years, with 21 out of 35 patients affected (60.0%), followed closely by the 65–70 age group, where 28 out of 50 patients had LVD (56.0%). In the 60–64 age group, 33 out of 65 patients were affected (50.8%), while 23 out of 60 patients in the 50–59 age group had LVD (38.3%). The lowest incidence was in the 40–49 age group, with 10 out of 40 patients affected (25.0%). Overall, 115 out of 250 patients (46.0%) in the study population had LVD [Table 6].

Table 6: Age wise Distribution of LVD in studied cases.

Age Group (years)	Number of Patients with LVD	Total Patients in Age Group	Percentage with LVD (%)
40-49	10	40	25.0%
50–59	23	60	38.3%
60–64	33	65	50.8%
65-70	28	50	56.0%
> 70	21	35	60.0%
Total	115	250	46.0%

The analysis of the correlation between age and the presence of left ventricular dysfunction (LVD) showed a moderate positive correlation with a Pearson's r value of +0.41. The association was statistically significant with a p-value of 0.001 ($p < 0.05$), indicating that increasing age is significantly associated with a higher likelihood of developing LVD [Table 7].

Table 7: Correlation between age of patients and presence of LVD.

Variable 1	Variable 2	Pearson's r	p-value	Interpretation
Age	Presence of LVD	+0.41	0.001	Moderate positive correlation

DISCUSSION

In the present cross-sectional study analysis of risk factors among patients with acute myocardial infarction (AMI) showed that the common risk factors were hypertension (58.0%), obesity (52.0%), and diabetes mellitus (44.0%). These comorbidities are known contributors to both the development of coronary artery disease as well as progression to post-infarction complications such as left ventricular dysfunction (LVD). Bell et al analyzing the Zibaeenezhad et al reported that major adverse cardiac events (MACE) AMI in short, mid and long-term intervals were common in individuals with diabetes and hypertension.[11] This is likely to be due to chronic endothelial injury, myocardial fibrosis, and impaired microvascular perfusion. Similarly Velagaleti et al in the Framingham Heart Study linked insulin resistance and metabolic syndrome with adverse left ventricular remodeling even in the absence of prior infarction.[12] The clustering of these risk factors in our cohort may partly explain the high incidence of LVD (46.0%).

Chest pain was the most common presenting complaint in this study (76.0%). The other common presenting complaints included dyspnea (48.0%) and diaphoresis (34.0%). ST-elevation myocardial infarction (STEMI) was seen in 66.0% of all cases. Anterior wall infarction was the predominant type (42.0%) followed by Inferior wall MI which was seen in 34.0% cases. These clinical and electrocardiographic patterns are often associated with larger infarct size and higher risk of complications. Kim DH et al reported that anterior STEMI particularly involving the left anterior descending artery was a strong predictor of post-infarction systolic dysfunction due to the extensive myocardial territory at risk.[13] Supporting this Sjogren H et al showed that patients with anterior STEMI exhibited higher rates of adverse remodeling and persistent LVD despite early reperfusion.[14]

Echocardiographic evaluation of patients with MI in this study revealed that 46.0% of patients had some or the other degree of LVD. This high incidence of LVD following MI underscores the importance of early echocardiographic screening following AMI. In line with our methodology Lin X et al also endorsed trans-thoracic 2D echocardiography as a reliable modality for detecting wall motion abnormalities and quantifying ejection fraction post-MI.[15] Solomon et al found that early echocardiographic assessment (within 72 hours of AMI) can effectively predict long-term outcomes and facilitate initiation of therapies such as ACE inhibitors and beta-blockers in cases of LVD after MI.[16]

The contribution of infarct characteristics to the development of LVD was an important factor for consideration in cases of LVD after MI. Anterior wall infarction and multi-territory infarcts are reported to be frequently associated with moderate to severe LVD. Jenča D reported that infarct size and anterior location were significant predictors of post-MI systolic dysfunction and adverse outcomes.[17] Similarly, White et al demonstrated that anterior wall infarctions had the lowest recovery of left ventricular ejection fraction and the highest mortality among patients of MI.[18]

Age was another significant determinant of LVD in our study. We found a significant positive correlation between advancing age and the incidence of LVD ($r = +0.41$; $p = 0.001$). Patients above the age of 65 years were found to have a notably higher burden of systolic dysfunction. This age-associated vulnerability is consistent with the findings of Shih H

et al who in a review study showed that aging independently correlates with declining LVEF and elderly patients are more likely to develop heart failure as compared to young patients following MI.[19] Rask-Madsen C et al conducted a retrospective study to evaluate outcome in patients after acute myocardial infarction.[20] The authors found that in-hospital mortality increased with age. It was 11% in patients <50 years and 43% in those ≥80 years. Heart failure affected two-thirds of patients ≥80 and significantly increased post-discharge mortality. These findings align with the findings of our study where we found a significant positive correlation between age of patient and incidence of LVF in cases of MI.

CONCLUSION

Left ventricular dysfunction is a common complication following AMI affecting nearly half of the patients particularly older individuals. Early echocardiographic screening for LVD in cases of MI is essential for timely identification and initiation of appropriate management to prevent adverse outcomes.

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