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Incidence, Echocardiographic Evolution, and Prognostic Impact of Ischemic Mitral Regurgitation Following Acute Myocardial Infarction: A Prospective Observational Study from South India

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ABSTRACT

Background: Ischemic mitral regurgitation (IMR) is a common secondary complication after acute myocardial infarction (AMI) that worsens prognosis through adverse left ventricular remodeling and heart failure progression. This study prospectively assessed the prevalence, severity, echocardiographic characteristics, and short-term clinical outcomes of IMR in patients with first AMI.

Materials and Methods: This prospective observational cohort study was conducted at Coimbatore Medical College Hospital, Coimbatore, Tamil Nadu, India, from August 2024 to October 2025. Fifty consecutive adults with first AMI (STEMI or NSTEMI) who underwent successful reperfusion were enrolled after informed consent. Transthoracic echocardiography was performed at baseline (24–48 hours), subacute (7–10 days), and chronic (4–6 weeks) phases to quantify mitral regurgitation severity (ASE guidelines), effective regurgitant orifice area (EROA), regurgitant volume, tenting parameters, and left ventricular ejection fraction (LVEF). Data were analyzed using chi-square/Fisher's exact tests, independent t-tests, repeated-measures ANOVA, and Kaplan-Meier estimates (SPSS v27.0; $p < 0.05$ significant).

Results: IMR was present in 56.0% at baseline (moderate-to-severe in 20.0%), declining to 48.0% (subacute) and 44.0% (chronic), with persistent moderate-to-severe IMR in ~16%. Inferior infarct location ($p = 0.018$; OR 6.00), female gender ($p = 0.032$), diabetes ($p = 0.048$), peak troponin I >15 ng/mL ($p = 0.011$), and LVEF <45% ($p = 0.024$) predicted moderate-to-severe IMR. Patients with moderate-to-severe IMR showed lower LVEF (38.60 ± 7.50% vs. 49.20 ± 8.10%; $p < 0.001$), greater tenting height/area, and higher regurgitant burden (all $p < 0.001$). The 6-month composite endpoint (heart failure hospitalization or cardiovascular death) occurred in 20.0%, with significantly worse event-free survival in the moderate-to-severe IMR group (log-rank $p = 0.008$).

Conclusion: Ischemic mitral regurgitation remains prevalent post-AMI in this reperfused South Indian cohort, with moderate-to-severe grades linked to inferior infarction, larger infarct size, diabetes, and reduced LVEF. Persistent moderate-to-severe IMR portends increased risk of heart failure and mortality at 6 months. Routine early echocardiography and risk-stratified management are warranted to mitigate adverse outcomes in resource-limited settings.

Keywords: *Mitral Valve Insufficiency, Myocardial Infarction, Echocardiography, Ventricular Remodeling, Percutaneous Coronary Intervention, Heart Failure.*

INTRODUCTION

Ischemic mitral regurgitation (IMR) represents a frequent and prognostically significant complication following acute myocardial infarction (AMI). It arises not from primary structural abnormalities of the mitral valve apparatus but as a secondary consequence of ischemic injury to the left ventricle (LV), leading to altered geometry, papillary muscle

dysfunction, and impaired leaflet coaptation [1]. Defined as mitral regurgitation occurring in the setting of coronary artery disease with structurally normal valve leaflets and chordae, IMR encompasses a spectrum from mild, transient insufficiency during acute ischemia to severe, persistent regurgitation that contributes to heart failure and excess mortality [2].

The prevalence of IMR after AMI varies depending on timing of assessment, reperfusion strategy, and echocardiographic criteria. In the modern era of primary percutaneous coronary intervention (PCI), moderate or severe IMR develops in approximately 10–20% of patients within days to weeks post-AMI, while mild IMR may be detected in up to 40–50% on early echocardiography [3].

The pathophysiology of IMR is multifaceted and dynamic. Acute ischemia induces regional wall motion abnormalities, most commonly inferior or posterior akinesis/dyskinesis, which displaces the papillary muscles apically and laterally. This displacement tethers the mitral leaflets (predominantly the posterior leaflet), restricting their systolic motion (Carpentier type IIIb dysfunction) and preventing effective coaptation. Annular dilatation, particularly in the septolateral dimension, further exacerbates incomplete closure [4]. Reduced closing forces compound the problem: diminished LV contractility, loss of annular sphincteric contraction, papillary muscle dyssynchrony, and global LV dyssynchrony all decrease the force opposing tethering. In acute settings, transient ischemia may cause reversible papillary muscle dysfunction, but persistent stunning, infarction, or rupture (rare, <1%) can lead to flail leaflet and torrential regurgitation [5].

The vicious cycle of "MR begets MR" perpetuates progression: regurgitant volume overload promotes LV remodeling, eccentric dilatation, and further papillary muscle displacement, increasing regurgitant orifice area. This adverse remodeling is exacerbated by delayed or incomplete reperfusion, larger infarct size, and absence of viability in the infarct zone. Echocardiographic hallmarks include tenting height >10 mm, tenting area >2–3 cm^2, posterior leaflet restriction, and coaptation depth abnormalities. Dynamic changes in MR severity with loading conditions or exercise further complicate assessment and prognosis [6].

Clinically, IMR significantly worsens outcomes. Even mild-to-moderate IMR post-AMI independently predicts heart failure hospitalization, reduced functional capacity, and increased cardiovascular mortality. Moderate or severe IMR is associated with 2- to 4-fold higher risk of death or heart failure events at 1–5 years, even after successful revascularization. Mechanisms include pulmonary hypertension, right ventricular strain, atrial fibrillation, and accelerated LV remodeling leading to progressive systolic dysfunction. In acute severe IMR (e.g., papillary muscle rupture), cardiogenic shock and pulmonary edema ensue rapidly, with mortality exceeding 40–50% without urgent intervention [7].

Historically, management focused on conservative stabilization or emergency surgery for papillary muscle rupture. The advent of primary PCI has dramatically reduced mechanical complications such as complete rupture (now ~0.2–0.5%), but functional IMR persists as a major challenge. Guidelines recommend optimizing medical therapy (ACE inhibitors, beta-blockers, aldosterone antagonists) to attenuate remodeling, alongside guideline-directed heart failure management [8].

Surgical mitral valve repair (undersized annuloplasty ± subvalvular procedures) during concomitant coronary artery bypass grafting has been advocated for severe chronic IMR, though randomized trials (e.g., CTSN) have shown mixed results regarding durability and survival benefit over replacement or medical therapy alone. Percutaneous edge-to-edge repair (MitraClip) has emerged for high-surgical-risk patients with functional MR, with registries demonstrating feasibility in post-AMI acute severe IMR, albeit with limited long-term data [9].

Despite therapeutic advances, gaps remain in understanding the natural history, optimal timing of intervention, and predictors of progression in reperfused AMI patients. The prognostic impact of even mild IMR in the Indian context, influenced by high diabetes prevalence and variable reperfusion rates, warrants localized investigation. This study prospectively evaluates the incidence, severity, echocardiographic characteristics, and clinical correlates of IMR following AMI. By characterizing patterns of regurgitation, identifying predictors of moderate-to-severe disease, and assessing short-term outcomes, and highlight opportunities for timely intervention in a resource-constrained setting typical of many Indian teaching hospitals.

MATERIALS AND METHODS

Study Setting: This was a prospective observational cohort study designed to evaluate the occurrence, severity, and clinical implications of ischemic mitral regurgitation following acute myocardial ischemia. The study was conducted at Coimbatore Medical College Hospital, Coimbatore, Tamil Nadu, India. The investigation spanned 15 months, from August 2024 to October 2025, encompassing patient recruitment, inpatient evaluation, echocardiographic assessments, and follow-up.

Study Participants: Adult patients (aged ≥18 years) admitted with a first episode of acute myocardial ischemia (ST-elevation myocardial infarction [STEMI] or non-ST-elevation myocardial infarction [NSTEMI]) confirmed by standard criteria (symptoms, ECG changes, elevated cardiac biomarkers) were eligible for inclusion. Inclusion criteria required successful reperfusion (primary PCI or thrombolysis with TIMI 2–3 flow) and availability for serial echocardiography. Patients provided written informed consent prior to enrollment.

Exclusion criteria included: prior myocardial infarction, known pre-existing valvular heart disease (rheumatic, degenerative, or congenital), prosthetic valves, severe aortic valve disease, atrial fibrillation at presentation, poor echocardiographic windows precluding accurate mitral regurgitation assessment, refusal of consent, or expected survival <30 days due to non-cardiac comorbidities.

Sample Size and Sampling Technique: The sample size was calculated as 50 participants, based on an anticipated prevalence of moderate-to-severe ischemic mitral regurgitation of 15–20% post-AMI (from regional and international data), with 80% power, alpha 0.05, and allowance for 10% attrition. Consecutive eligible patients admitted during the study period were enrolled using convenience sampling until the target was achieved.

Study Tools: Transthoracic echocardiography (TTE) was performed using a commercially available system (GE Vivid E9 or equivalent) with phased-array transducers (M5S or 4V). Standard views (parasternal long-axis, apical four-chamber, two-chamber, three-chamber) were obtained. Mitral regurgitation was quantified according to American Society of Echocardiography guidelines: color flow jet area, vena contracta width, proximal isovelumic surface area (PISA) method for effective regurgitant orifice area (EROA) and regurgitant volume, and supportive parameters (pulmonary vein flow, continuous-wave Doppler density). Severity was graded as none/trace, mild, moderate, or severe. LV ejection fraction (biplane Simpson's method), wall motion score index, tenting height, tenting area, and coaptation depth were measured. Additional tools included 12-lead ECG, cardiac biomarkers (troponin, CK-MB), coronary angiography reports, and clinical proforma for demographics, risk factors, Killip class, and complications.

Study Procedure: Patients admitted with acute myocardial ischemia underwent initial stabilization, reperfusion (primary PCI preferred), and guideline-directed medical therapy. Baseline echocardiography was performed within 24–48 hours of admission (after hemodynamic stabilization) to document early IMR. Follow-up TTE was repeated at 7–10 days (subacute phase) and 4–6 weeks post-discharge (chronic phase) to assess evolution of regurgitation severity, LV remodeling, and systolic function. Clinical follow-up occurred at 30 days and 3–6 months for adverse events (heart failure hospitalization, reinfarction, death). All echocardiograms were interpreted by two experienced cardiologists blinded to clinical details, with discrepancies resolved by consensus.

Ethical Issues: The study protocol was approved by the Institutional Ethics Committee of Coimbatore Medical College Hospital. Written informed consent was obtained from all participants in Tamil or English, detailing study purpose, procedures, risks (minimal, related to echocardiography), benefits, confidentiality, and voluntary participation. No incentives were provided. Data were anonymized and stored securely. Participants could withdraw at any time without affecting standard care. Adverse events were reported per institutional guidelines.

Statistical Analysis: Data were analyzed using SPSS version 27.0. Continuous variables (e.g., EROA, tenting height, LVEF) were expressed as mean ± standard deviation (normal distribution) or median (interquartile range) if skewed. Categorical variables (e.g., MR severity grade, infarct location) were summarized as frequencies and percentages. Between-group comparisons (e.g., moderate/severe vs. mild/none IMR) used independent t-tests or Mann-Whitney U tests for continuous data and chi-square or Fisher's exact tests for categorical data. Changes in MR severity and LV parameters over time were assessed with repeated-measures ANOVA or Friedman test as appropriate. Predictors of moderate-to-severe IMR were explored using logistic regression (univariate and multivariate). A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Table 1 summarizes the demographic, risk factor, and infarct-related characteristics of the 50 patients with acute myocardial ischemia. The cohort had a mean age of 58.40 ± 11.20 years, with a male predominance (68.0%) and high prevalence of diabetes (44.0%) and hypertension (56.0%), consistent with typical Indian post-AMI populations. Inferior infarcts were the most common location (48.0%), followed by anterior (40.0%), and primary PCI was the dominant reperfusion strategy (84.0%). Baseline LVEF averaged 46.80 ± 9.40%, reflecting moderate LV systolic impairment in this reperfused cohort.

Table 1: Demographic and Baseline Clinical Characteristics of Study Participants (N = 50).

Characteristic	Value
Age (years), M ± SD	58.40 ± 11.20
Gender, n (%) Male	34 (68.0)
Gender, n (%) Female	16 (32.0)
Diabetes mellitus, n (%)	22 (44.0)
Hypertension, n (%)	28 (56.0)
Current smoker, n (%)	18 (36.0)
Prior coronary artery disease, n (%)	8 (16.0)
Infarct type, n (%) STEMI	38 (76.0)
Infarct type, n (%) NSTEMI	12 (24.0)

Infarct location, n (%) Anterior	20 (40.0)
Infarct location, n (%) Inferior	24 (48.0)
Infarct location, n (%) Lateral/Other	6 (12.0)
Reperfusion strategy, n (%) Primary PCI	42 (84.0)
Reperfusion strategy, n (%) Thrombolysis	8 (16.0)
Peak troponin I (ng/mL), median (IQR)	12.50 (4.80–28.60)
LVEF at baseline (%), M ± SD	46.80 ± 9.40

Note. Continuous variables presented as mean ± SD or median (IQR); categorical variables as frequency (percentage). STEMI = ST-elevation myocardial infarction; PCI = percutaneous coronary intervention; LVEF = left ventricular ejection fraction.

Table 2 illustrates the temporal prevalence and severity distribution of ischemic mitral regurgitation across three echocardiographic assessments. At baseline (24–48 hours post-AMI), IMR was present in 56.0% of patients, with moderate-to-severe grades in 20.0%. Prevalence decreased slightly in subacute (48.0%) and chronic (44.0%) phases, with persistent moderate-to-severe IMR in ~16%.

Table 2: Prevalence and Severity of Ischemic Mitral Regurgitation at Baseline and Follow-up (N = 50).

Time Point / MR Severity	None/Trace n (%)	Mild n (%)	Moderate n (%)	Severe n (%)	Total with MR n (%)
Baseline (24–48 h post-AMI)	22 (44.0)	18 (36.0)	8 (16.0)	2 (4.0)	28 (56.0)
Subacute (7–10 days)	26 (52.0)	16 (32.0)	6 (12.0)	2 (4.0)	24 (48.0)
Chronic (4–6 weeks)	28 (56.0)	14 (28.0)	6 (12.0)	2 (4.0)	22 (44.0)

Note. MR = mitral regurgitation; percentages sum to 100% per time point. Moderate-to-severe MR prevalence declined modestly over time.

Table 3 compares key echocardiographic parameters between patients with none/mild versus moderate/severe ischemic mitral regurgitation at baseline. Moderate/severe IMR was associated with significantly lower LVEF (38.60 ± 7.50% vs. 49.20 ± 8.10%; $p < 0.001$), larger LV end-systolic volume index, greater tenting height and area, increased effective regurgitant orifice area and regurgitant volume, and higher wall motion score index (all $p < 0.01$).

Table 3: Echocardiographic Parameters by Ischemic Mitral Regurgitation Severity at Baseline (N = 50).

Parameter	None/Mild MR (n = 40) M ± SD	Moderate/Severe MR (n = 10) M ± SD	p-value (independent t-test)
LVEF (%)	49.20 ± 8.10	38.60 ± 7.50	<0.001
LV end-systolic volume index (mL/m²)	32.40 ± 9.80	48.70 ± 12.30	<0.001
Tenting height (mm)	8.20 ± 2.40	12.80 ± 3.10	<0.001
Tenting area (cm²)	1.80 ± 0.70	3.40 ± 1.10	<0.001
Effective regurgitant orifice area (cm²)	0.09 ± 0.05	0.28 ± 0.09	<0.001
Regurgitant volume (mL/beat)	12.50 ± 6.80	38.40 ± 14.20	<0.001
Wall motion score index	1.42 ± 0.28	1.78 ± 0.31	0.002

Note. MR = mitral regurgitation; LVEF = left ventricular ejection fraction. All parameters significantly worse in moderate/severe group.

Table 4 examines factors associated with moderate-to-severe ischemic mitral regurgitation at baseline using chi-square/Fisher's exact tests. Inferior infarct location ($p = 0.018$; OR 6.00), female gender ($p = 0.032$), diabetes mellitus ($p = 0.048$), peak troponin I >15 ng/mL ($p = 0.011$), and LVEF <45% ($p = 0.024$) were significantly linked to higher risk.

Table 4: Association of Clinical and Infarct-Related Factors with Moderate-to-Severe Ischemic Mitral Regurgitation at Baseline (N = 50).

Factor	Moderate/Severe MR (n = 10) n (%)	None/Mild MR (n = 40) n (%)	p-value (chi-square/ Fisher's exact)	Odds Ratio (95% CI)
Inferior infarct location (vs. anterior)	8 (80.0)	16 (40.0)	0.018	6.00 (1.12–32.10)
Female gender	6 (60.0)	10 (25.0)	0.032	4.50 (1.10–18.42)
Diabetes mellitus	7 (70.0)	15 (37.5)	0.048	3.97 (0.92–17.14)
Peak troponin I >15 ng/mL	8 (80.0)	14 (35.0)	0.011	7.43 (1.38–39.98)
LVEF <45%	7 (70.0)	12 (30.0)	0.024	5.44 (1.25–23.68)

Primary PCI (vs. thrombolysis)	7 (70.0)	35 (87.5)	0.192	0.40 (0.09–1.78)

Table 5 reports Kaplan-Meier estimates for freedom from the composite endpoint of heart failure hospitalization or cardiovascular death over 6 months post-AMI. The cumulative event-free proportion declined to 78.0% at 6 months (20.0% event rate). Patients with moderate-to-severe IMR exhibited significantly worse event-free survival (log-rank p = 0.008).

Table 5: Kaplan-Meier Estimates of Freedom from Heart Failure Hospitalization or Cardiovascular Death at 6 Months (N = 50).

Time Point (Months Post-AMI)	At Risk (n)	Events (n)	Cumulative Proportion Event-Free	Standard Error	95% CI Lower	95% CI Upper
1	50	3	0.940	0.034	0.873	1.000
3	47	4	0.860	0.049	0.764	0.956
6	43	3	0.780	0.059	0.664	0.896

Note. Total events: 10 (20.0%; 7 heart failure hospitalizations, 3 cardiovascular deaths). Moderate/severe IMR group had significantly lower event-free survival (log-rank p = 0.008). Median time-to-event not reached.

DISCUSSION

The present prospective observational study conducted at Coimbatore Medical College Hospital provides valuable contemporary data on the incidence, echocardiographic evolution, and prognostic implications of ischemic mitral regurgitation (IMR) in a reperfused South Indian cohort of 50 patients with first acute myocardial ischemia. The baseline prevalence of any IMR was 56.0%, with moderate-to-severe grades observed in 20.0% within 24–48 hours post-AMI. The slight decline in prevalence over time—to 48.0% at 7–10 days and 44.0% at 4–6 weeks, with persistent moderate-to-severe IMR in approximately 16%—reflects partial spontaneous improvement in many patients due to resolution of myocardial stunning, recovery of regional contractility, and favorable early remodeling following timely reperfusion [10].

Inferior infarct location emerged as the strongest clinical predictor of moderate-to-severe IMR at baseline (80.0% vs. 40.0% in the none/mild group; p = 0.018; OR 6.00), confirming the well-established anatomical vulnerability of the posteromedial papillary muscle, which is typically supplied by a single dominant vessel (right coronary or circumflex artery). This finding aligns with similar studies demonstrating 2- to 6-fold higher risk of significant functional MR after inferior or inferoposterior infarction compared with anterior infarction [11].

Female gender (p = 0.032; OR 4.50), diabetes mellitus (p = 0.048; OR 3.97), larger infarct size (peak troponin I >15 ng/mL; p = 0.011; OR 7.43), and reduced baseline LVEF <45% (p = 0.024; OR 5.44) were also significantly associated with more severe IMR. These associations highlight the synergistic impact of metabolic comorbidity, greater myocardial necrosis, and impaired global systolic function in promoting leaflet tethering and annular dilatation [12].

Echocardiographic parameters provided mechanistic insight into these clinical associations. Patients with moderate-to-severe IMR exhibited markedly worse LV remodeling indices (lower LVEF 38.60 ± 7.50% vs. 49.20 ± 8.10%; larger LV end-systolic volume index 48.70 ± 12.30 vs. 32.40 ± 9.80 mL/m²; both p < 0.001), greater leaflet tethering (tenting height 12.80 ± 3.10 vs. 8.20 ± 2.40 mm; tenting area 3.40 ± 1.10 vs. 1.80 ± 0.70 cm²; both p < 0.001), and higher regurgitant burden (EROA 0.28 ± 0.09 vs. 0.09 ± 0.05 cm²; regurgitant volume 38.40 ± 14.20 vs. 12.50 ± 6.80 mL/beat; both p < 0.001). The elevated wall motion score index (1.78 ± 0.31 vs. 1.42 ± 0.28; p = 0.002) further highlights the role of extensive regional dysfunction in driving papillary muscle displacement and dyssynchrony [13].

The prognostic significance of IMR was clearly demonstrated in the 6-month follow-up period. The composite endpoint of heart failure hospitalization or cardiovascular death occurred in 20.0% of the cohort, with Kaplan-Meier estimates showing event-free survival declining to 78.0% at 6 months. Patients with moderate-to-severe IMR at baseline had significantly worse event-free survival (log-rank p = 0.008), consistent with extensive literature demonstrating that even moderate functional MR post-AMI independently predicts adverse remodeling, heart failure progression, and mortality. Mechanisms likely include chronic volume overload, pulmonary venous hypertension, right ventricular strain, and accelerated eccentric LV dilatation, all of which compound the initial ischemic insult [14].

The predominance of primary PCI (84.0%) in this cohort reflects adherence to contemporary reperfusion guidelines and likely contributed to the relatively low incidence of severe persistent IMR and mechanical complications (no papillary muscle ruptures observed). Nonetheless, the persistence of moderate-to-severe IMR in ~16% at 4–6 weeks highlights an ongoing therapeutic challenge in reperfused patients, particularly those with inferior infarction, diabetes, and larger infarct size [15].

Current guidelines emphasize guideline-directed medical therapy to limit adverse remodeling; however, the optimal role and timing of mitral valve intervention remain controversial. Randomized trials have shown that routine mitral annuloplasty

at the time of coronary bypass does not consistently improve survival or reverse remodeling in moderate IMR, while percutaneous edge-to-edge repair has shown promise in high-risk functional MR cohorts. In resource-constrained settings such as many Indian teaching hospitals, where advanced structural interventions are not universally available, early identification of high-risk features (inferior location, tenting height >10 mm, EROA >0.2 cm², LVEF <45%) could guide intensified medical optimization, close surveillance, and selective referral for advanced therapies [16].

The study has certain limitations. The single-center design and modest sample size (n = 50) may limit generalizability, although the demographic and infarct characteristics are representative of South Indian AMI populations. Serial echocardiography was limited to three time points, precluding capture of longer-term remodeling trajectories or dynamic changes during exercise. Quantitative assessment relied on standard parameters without three-dimensional or stress echocardiography, which could have provided additional mechanistic insight. The observational nature precludes causal inference regarding management strategies.

Despite these constraints, the study offers important localized evidence on the burden and prognostic impact of IMR following reperfused AMI in an Indian tertiary care setting. The high prevalence of early IMR, its association with inferior infarction, diabetes, and larger infarct size, and the clear link between moderate-to-severe regurgitation and adverse 6-month outcomes reinforce the need for routine early post-AMI echocardiography and risk-stratified follow-up. Strengthening guideline-directed medical therapy, optimizing diabetes control, and advocating for broader access to advanced imaging and percutaneous mitral interventions could mitigate the substantial heart failure burden attributable to ischemic mitral regurgitation in similar high-risk populations.

CONCLUSION

This prospective study confirms that ischemic mitral regurgitation remains a frequent and prognostically important complication after acute myocardial ischemia, even in the era of timely reperfusion. Moderate-to-severe IMR identifies a vulnerable subgroup at heightened risk of adverse remodeling and clinical events, driven by greater leaflet tethering, LV dilatation, and infarct-related factors. These findings support systematic echocardiographic screening, aggressive remodeling prevention, and consideration of targeted interventions to improve long-term outcomes in resource-limited settings.

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