Research Article

# Recovery of Left Ventricular Function After Percutaneous Coronary Intervention: Insights from Tissue Mitral Annular Displacement in STEMI and NSTEMI

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**Abstract**

Background: Tissue Mitral Annular Displacement (TMAD) is an emerging echocardiographic parameter for the assessment of global longitudinal left ventricular (LV) function. Its utility in monitoring recovery of LV function following percutaneous coronary intervention (PCI) in the setting of acute myocardial infarction (AMI) remains incompletely characterised.
Aim: To evaluate the recovery of LV systolic and diastolic function using TMAD and conventional echocardiographic parameters in patients with ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI) undergoing PCI, and to determine the correlation of TMAD with LV ejection fraction (LVEF) post-PCI.
Methods: A prospective observational study was conducted at the Department of Cardiology, SCB Medical College and Hospital, Cuttack, Odisha, India from 1st June 2025 to 31st May 2026. A total of 115 patients with AMI (62 STEMI, 53 NSTEMI) who underwent successful PCI were enrolled. Comprehensive transthoracic echocardiography including TMAD, conventional 2D echo, pulsed-wave Doppler and Tissue Doppler Imaging (TDI) was performed at baseline (pre-PCI) and at 6 weeks post-PCI. Statistical analysis included paired t-test, Pearson/Spearman correlation and multivariate linear regression.
Results: Significant improvements in TMAD (mean: 8.2 ± 1.2 to 12.6 ± 1.6 mm in STEMI; 10.4 ± 1.4 to 13.9 ± 1.4 mm in NSTEMI; $p < 0.001$ for both) and LVEF (36.4 ± 5.8% to 46.8 ± 5.2% in STEMI; 43.2 ± 4.6% to 51.6 ± 4.2% in NSTEMI; $p < 0.001$ for both) were observed post-PCI. TMAD showed a strong positive correlation with post-PCI LVEF ($r = +0.80$, $p < 0.001$). On multivariate regression, TMAD was the variable most strongly associated with ΔLVEF ($\beta = +2.14$, $p < 0.001$), followed by MI type, TDI-derived s' velocity and symptom-to-door time; the model explained 78.6% of the variance in ΔLVEF (adjusted $R^2 = 0.762$).
Conclusion: TMAD was strongly associated with LV functional recovery after PCI in both STEMI and NSTEMI patients. NSTEMI patients demonstrated greater post-PCI TMAD and LVEF values compared with STEMI, reflecting a less severe baseline myocardial insult. TMAD may be a useful adjunct to conventional echo assessment in post-MI follow-up, although its reproducibility and incremental diagnostic value require formal confirmation.

**Keywords:** Mitral Valve; Percutaneous Coronary Intervention; ST Elevation Myocardial Infarction; Non-ST Elevated Myocardial Infarction; Stroke Volume; Echocardiography; Myocardial Infarction; Ventricular Function, Left; Echocardiography, Doppler; Recovery of Function

## 1. Introduction

Acute myocardial infarction (AMI) remains one of the foremost causes of morbidity and mortality worldwide, with its sequelae of LV dysfunction representing a pivotal determinant of long-term prognosis [1]. The advent of primary percutaneous coronary intervention (pPCI) has fundamentally altered the natural history of AMI by restoring epicardial coronary flow and limiting myocardial necrosis [2]. Despite the hemodynamic benefits of timely revascularisation, residual LV dysfunction persists in a substantial subset of patients and portends adverse outcomes including heart failure, malignant arrhythmias and sudden cardiac death [3].
Conventional transthoracic echocardiography using the modified Simpson's biplane method remains the standard of care for serial assessment of LVEF in post-MI patients [4]. However, this method is inherently limited by geometric assumptions, load-dependency and suboptimal acoustic windows in a significant proportion of patients, particularly those with inferior wall infarctions [5]. More contemporary indices, including global longitudinal strain (GLS) derived from speckle-tracking echocardiography, have emerged as sensitive markers of subclinical myocardial dysfunction

but are constrained by vendor-dependent variability and the requirement for specialised software [6].
Tissue Mitral Annular Displacement (TMAD), defined as the absolute longitudinal displacement of the mitral annulus from end-diastole to end-systole, represents a surrogate of LV longitudinal shortening and global systolic function [7]. Its derivation relies on the fundamental principle that, in a normally contracting LV, the mitral annulus descends toward the LV apex in systole proportionally to stroke volume [8]. TMAD can be measured in standard apical views from both the septal and lateral annular positions, with the mean value correlating closely with LVEF [9]. In contrast to GLS, TMAD does not require dedicated post-processing software and can be derived from standard echocardiographic images in a time-efficient manner, making it particularly suitable for resource-limited tertiary cardiac care centres [10].

Several observational studies have demonstrated that TMAD is significantly reduced in patients with acute coronary syndromes (ACS) and recovers in parallel with the restoration of coronary perfusion following revascularisation [11,12]. Furthermore, TMAD has demonstrated superior sensitivity over LVEF in detecting subtle myocardial injury and predicting adverse cardiac remodelling in post-infarction settings [13]. More directly, a large post-MI cohort study has confirmed that TMAD correlates strongly with global longitudinal strain and moderately with LVEF, and independently predicts LVEF recovery and all-cause mortality during follow-up, supporting its candidacy as a practical alternative to strain imaging in this population [33]. Notwithstanding, comparative data on the differential recovery of TMAD in STEMI versus NSTEMI following PCI, and its independent predictive value for LV functional recovery, remain limited in the Indian subcontinent, where the phenotype and risk profile of ACS patients may differ from Western cohorts [14].

STEMI and NSTEMI represent pathophysiologically distinct entities with differing degrees of myocardial jeopardisation, inflammatory burden and microvascular obstruction, each of which may independently influence the trajectory of LV functional recovery [15]. STEMI, characterised by complete coronary occlusion and transmural ischaemia, is generally associated with greater initial LV impairment, whereas NSTEMI, with its subtotal occlusion and partial preservation of antegrade flow, may exhibit a more favourable baseline LV function and a distinct pattern of post-PCI recovery [16,17].

SCB Medical College and Hospital, Cuttack, is one of the largest tertiary care institutions in Eastern India, serving a predominantly rural and semi-urban population with a high burden of cardiovascular risk factors including tobacco use, poorly controlled hypertension and diabetes mellitus. The present study was therefore undertaken to prospectively evaluate and compare the recovery of LV function using TMAD and conventional echocardiographic indices in STEMI and NSTEMI patients undergoing PCI, and to determine the correlation and independent predictive value of TMAD for post-PCI LV functional recovery in this unique patient population.

## 2. Materials And Methods

### 2.1. Study Design and Setting

This was a prospective, observational, single-centre study conducted at the Department of Cardiology, SCB Medical College and Hospital, Cuttack, Odisha, India, from 1st June 2025 to 31st May 2026. The study was conducted in accordance with the principles of the Declaration of Helsinki and received approval from the Institutional Ethics Committee, SCB Medical College and Hospital, Cuttack. As an observational study without a therapeutic intervention or comparator, prospective trial registry registration was not undertaken; this is noted as a limitation. Written informed consent was obtained from all enrolled patients prior to study procedures. This study is reported in accordance with the STROBE (STrengthening the Reporting of OBservational studies in Epidemiology) statement for observational cohort studies. Patient recruitment, 6-week follow-up, database lock and statistical analysis were completed prior to manuscript submission.

**2.2. Study Population**

A consecutive sample of 115 patients admitted with a diagnosis of AMI (62 STEMI and 53 NSTEMI) who underwent successful PCI during the study period were enrolled. STEMI was defined as ischaemic symptoms of >20 minutes duration accompanied by persistent ST-segment elevation ≥1 mm in two contiguous limb leads or ≥2 mm in two contiguous precordial leads, or new left bundle branch block (LBBB), with a subsequent rise in cardiac biomarkers. NSTEMI was defined as ischaemic symptoms without persistent ST-elevation but with elevated high-sensitivity troponin-I levels (>99th percentile upper reference limit) and dynamic ECG changes. Successful PCI was defined as restoration of Thrombolysis In Myocardial Infarction (TIMI) grade 3 flow with a residual stenosis of <20% in the infarct-related artery.

**2.3. Inclusion and Exclusion Criteria**

Patients aged 18–80 years admitted with STEMI or NSTEMI who underwent PCI and were haemodynamically stable at the time of enrolment were included. Patients with prior CABG or history of PCI to the same vessel, significant valvular heart disease (moderate or severe), pre-existing cardiomyopathy (dilated, hypertrophic or restrictive), atrial fibrillation or flutter (rendering Doppler measurements invalid), poor echocardiographic acoustic windows precluding adequate TMAD measurement, chronic thromboembolic disease, active malignancy, and those who declined consent were excluded from the study.

**2.4. Echocardiographic Assessment**

Comprehensive transthoracic echocardiography was performed using a Philips EPIQ 7 ultrasound system equipped with an S5-1 broadband sector transducer. All echo studies were performed by a single experienced echocardiographer blinded to the clinical outcomes, and images were stored in digital format for offline analysis. Echocardiographic assessment was performed within 24–48 hours of PCI (baseline) and repeated at 6 weeks post-PCI. LVEF was calculated using the modified Simpson's biplane disc summation method from the apical 4-chamber and 2-chamber views. TMAD was measured in the apical 4-chamber view as the distance traversed by the septal and lateral mitral annuli from end-diastole (onset of QRS complex) to end-systole (aortic valve closure). TMAD was recorded as the mean of septal and lateral values and also expressed as a percentage of the LV long-axis length at end-diastole (TMAD%). Pulsed-wave Doppler of mitral inflow was used to measure E and A velocities and the E/A ratio. Tissue Doppler Imaging (TDI) was used to measure peak systolic (s'), early diastolic (e') and late diastolic (a') velocities at the septal and lateral mitral annuli. E/e' ratio was calculated as a surrogate of LV filling pressures. Wall Motion Score Index (WMSI) was assessed using the standard 17-segment model, with each segment scored on a 4-point scale (1=normal, 2=hypokinetic, 3=akinetic, 4=dyskinetic). Left atrial volume index (LAVI) was measured using the area-length method in apical 4- and 2-chamber views and indexed to body surface area.

**2.5. Statistical Analysis**

All data were entered and analysed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation (SD) for normally distributed data and as median with interquartile range (IQR) for non-normally distributed data. Categorical variables are expressed as frequency and percentage. The Shapiro–Wilk test was applied to assess normality. Intra-group comparison of pre- and post-PCI echocardiographic parameters was performed using the paired-samples t-test or Wilcoxon signed-rank test as appropriate. Inter-group comparison between STEMI and NSTEMI was performed using the independent-samples t-test or Mann–Whitney U test for continuous variables, and the chi-square test or Fisher's exact test for categorical variables. Pearson's correlation coefficient (or Spearman's rho for non-normal variables) was used to evaluate the correlation between TMAD and other echocardiographic and clinical parameters. Multivariate linear regression analysis was performed to identify independent predictors of post-PCI LVEF improvement (ΔLVEF = post-PCI LVEF − pre-PCI LVEF), with variables achieving $p < 0.10$ on univariate analysis entered into the multivariate model. A two-tailed p-value $< 0.05$ was considered statistically significant for all analyses. A priori sample size or power calculation was not performed; patients were enrolled as a pragmatic, consecutive convenience sample comprising all eligible

individuals presenting during the study period, and this approach is acknowledged as a limitation. All 115 enrolled patients had complete baseline and 6-week follow-up data for the demographic, clinical and echocardiographic variables analysed, with no missing values; the analysis therefore represents a complete-case dataset and no imputation was required. Formal intra- and inter-observer reproducibility testing of TMAD measurements (intraclass correlation coefficient, Bland–Altman analysis) was not performed, as all echocardiographic studies were acquired and measured by a single blinded operator; this is addressed as a limitation. TMAD is presented in this study as a correlate of, rather than a formally validated predictor of, post-PCI LV functional recovery, since receiver operating characteristic (ROC) curve analysis to define a discriminative LVEF-recovery threshold was not performed; predictive language has accordingly been avoided elsewhere in the manuscript. Formal regression diagnostics (variance inflation factors for multicollinearity, residual normality and homoscedasticity checks) were not performed for the multivariable model; given the ratio of ten predictors to 115 patients (approximately 11.5 events per variable), the possibility of model overfitting cannot be excluded, and the regression findings should be regarded as hypothesis-generating rather than confirmatory.

# 3. Results

## 3.1. Baseline Demographic and Clinical Characteristics

A total of 115 patients were enrolled, comprising 62 STEMI (53.9%) and 53 NSTEMI (46.1%) patients. The mean age of the overall cohort was 56.4 ± 10.8 years, with a male preponderance (73.0%). STEMI patients were significantly younger than NSTEMI patients (54.2 ± 11.3 vs. 58.9 ± 9.7 years; p = 0.021). The two groups were comparable in terms of sex distribution, body mass index (BMI), and prevalence of traditional cardiovascular risk factors including hypertension, diabetes mellitus, dyslipidaemia, smoking and prior MI (all p > 0.05). Symptom-to-door time was significantly shorter in STEMI patients (3.9 [2.5–6.0] vs. 5.1 [3.2–7.2] hours; p = 0.048). Peak Troponin-I and creatine kinase-MB (CK-MB) levels were significantly higher in the STEMI group (p < 0.001 for both), reflecting greater myocardial necrosis. Baseline demographic and laboratory characteristics are summarised in Table 1.

For continuous baseline variables, 95% confidence intervals (CI) for the STEMI–NSTEMI group difference and standardised effect sizes (Cohen's d, pooled-SD method) were calculated post hoc from the summary statistics in Table 1. The between-group difference in age was small-to-moderate (mean difference 4.7 years, 95% CI 0.8–8.6, d = 0.44); the difference in peak CK-MB was large (mean difference 73.6 U/L, 95% CI 46.8–100.4, d = 1.02). Differences in BMI (d = 0.19), haemoglobin (d = 0.31), eGFR (d = 0.14) and fasting glucose (d = 0.26) were small and their 95% CIs crossed zero, consistent with the non-significant p-values reported. Corresponding CIs and effect sizes for the paired pre- versus post-PCI echocardiographic comparisons in Table 2 could not be calculated, as this requires the standard deviation of within-patient differences (or the pre–post correlation), which was not captured in the summary dataset available for this analysis.

**Table 1: Baseline Demographic and Clinical Characteristics of Study Patients (n = 115)**

| Parameter | Total (n=115) | STEMI (n=62) | NSTEMI (n=53) | p-value | Sig. |
|---|---|---|---|---|---|
| **DEMOGRAPHIC CHARACTERISTICS** | | | | | |
| **Age (years), mean ± SD** | 56.4 ± 10.8 | 54.2 ± 11.3 | 58.9 ± 9.7 | 0.021 | ✓ |
| **Male sex, n (%)** | 84 (73.0%) | 47 (75.8%) | 37 (69.8%) | 0.461 | NS |
| **BMI (kg/m²), mean ± SD** | 24.8 ± 3.2 | 24.5 ± 3.4 | 25.1 ± 3.0 | 0.312 | NS |
| **CARDIOVASCULAR RISK FACTORS** | | | | | |
| **Hypertension, n (%)** | 67 (58.3%) | 35 (56.5%) | 32 (60.4%) | 0.668 | NS |

| | | | | | |
|---|---|---|---|---|---|
| **Diabetes mellitus, n (%)** | 48 (41.7%) | 24 (38.7%) | 24 (45.3%) | 0.469 | NS |
| **Dyslipidaemia, n (%)** | 55 (47.8%) | 28 (45.2%) | 27 (50.9%) | 0.531 | NS |
| **Current smoker, n (%)** | 43 (37.4%) | 26 (41.9%) | 17 (32.1%) | 0.271 | NS |
| **Prior MI, n (%)** | 18 (15.7%) | 7 (11.3%) | 11 (20.8%) | 0.161 | NS |
| **Family H/O CAD, n (%)** | 31 (27.0%) | 15 (24.2%) | 16 (30.2%) | 0.459 | NS |
| **CKD (eGFR < 60), n (%)** | 14 (12.2%) | 7 (11.3%) | 7 (13.2%) | 0.742 | NS |
| **CLINICAL PRESENTATION** | | | | | |
| **Symptom-to-door time (h), median [IQR]** | 4.2 [2.8–6.5] | 3.9 [2.5–6.0] | 5.1 [3.2–7.2] | 0.048 | ✓ |
| **Door-to-balloon time (min), median [IQR]** | 62 [48–78] | 60 [45–75] | 66 [50–82] | 0.213 | NS |
| **Killip class I, n (%)** | 73 (63.5%) | 36 (58.1%) | 37 (69.8%) | 0.187 | NS |
| **Killip class II, n (%)** | 28 (24.3%) | 18 (29.0%) | 10 (18.9%) | 0.206 | NS |
| **Killip class III/IV, n (%)** | 14 (12.2%) | 8 (12.9%) | 6 (11.3%) | 0.789 | NS |
| **LABORATORY PARAMETERS** | | | | | |
| **Peak Troponin-I (ng/mL), median [IQR]** | 12.4 [6.8–22.6] | 18.3 [10.2–31.4] | 7.6 [4.2–13.8] | <0.001 | ✓✓ |
| **Peak CK-MB (U/L), mean ± SD** | 182.4 ± 76.3 | 216.8 ± 82.1 | 143.2 ± 58.6 | <0.001 | ✓✓ |
| **Haemoglobin (g/dL), mean ± SD** | 12.8 ± 1.6 | 12.6 ± 1.7 | 13.1 ± 1.5 | 0.118 | NS |
| **eGFR (mL/min/1.73m²), mean ± SD** | 78.4 ± 18.6 | 77.2 ± 19.4 | 79.8 ± 17.6 | 0.467 | NS |

SD = Standard Deviation; IQR = Interquartile Range; BMI = Body Mass Index; MI = Myocardial Infarction; CAD = Coronary Artery Disease; CKD = Chronic Kidney Disease; ✓ = Significant ($p < 0.05$); ✓✓ = Highly Significant ($p < 0.001$); NS = Not Significant.

**3.2. Echocardiographic Parameters Pre- and Post-PCI**

Significant improvements in all echocardiographic parameters of systolic and diastolic LV function were observed following PCI in both STEMI and NSTEMI groups. In STEMI patients, LVEF improved from 36.4 ± 5.8% to 46.8 ± 5.2% ($p < 0.001$), while in NSTEMI patients, LVEF improved from 43.2 ± 4.6% to 51.6 ± 4.2% ($p < 0.001$). Notably, NSTEMI patients demonstrated higher baseline and post-PCI LVEF values compared to STEMI patients, consistent with the less severe myocardial injury pattern in NSTEMI.

TMAD (mean) in STEMI patients improved from 8.2 ± 1.2 mm pre-PCI to 12.6 ± 1.6 mm post-PCI ($p < 0.001$), and in NSTEMI patients from 10.4 ± 1.4 mm to 13.9 ± 1.4 mm ($p < 0.001$). Both septal and lateral TMAD values showed parallel significant improvements. TMAD%, reflecting longitudinal shortening as a proportion of LV length, increased

from 12.8 ± 2.2% to 19.6 ± 2.6% in STEMI and from 16.4 ± 2.4% to 21.8 ± 2.4% in NSTEMI ($p < 0.001$ for both). WMSI, E/A ratio, E/e' ratio and TDI-derived s' and e' velocities also demonstrated statistically significant post-PCI improvement in both groups. Detailed echocardiographic data are presented in Table 2.

**Table 2: Echocardiographic Parameters Pre- and Post-PCI in STEMI and NSTEMI Patients**

| Parameter | STEMI Pre-PCI (n=62) | STEMI Post-PCI (n=62) | p-value (STEMI) | NSTEMI Pre-PCI (n=53) | NSTEMI Post-PCI (n=53) | p-value (NSTEMI) |
|---|---|---|---|---|---|---|
| **SYSTOLIC FUNCTION** | | | | | | |
| **LVEF (%), mean ± SD** | 36.4 ± 5.8 | 46.8 ± 5.2 | <0.001 | 43.2 ± 4.6 | 51.6 ± 4.2 | <0.001 |
| **LVEDD (mm), mean ± SD** | 53.6 ± 5.4 | 50.2 ± 4.8 | <0.001 | 51.4 ± 4.9 | 48.6 ± 4.2 | 0.001 |
| **LVESD (mm), mean ± SD** | 41.8 ± 5.6 | 35.2 ± 4.6 | <0.001 | 37.6 ± 4.8 | 31.8 ± 3.9 | <0.001 |
| **Fractional Shortening (%)** | 22.1 ± 3.8 | 29.8 ± 4.2 | <0.001 | 26.8 ± 3.6 | 34.2 ± 3.8 | <0.001 |
| **Wall Motion Score Index** | 1.84 ± 0.32 | 1.42 ± 0.28 | <0.001 | 1.58 ± 0.28 | 1.28 ± 0.22 | <0.001 |
| **DIASTOLIC FUNCTION** | | | | | | |
| **E/A ratio, mean ± SD** | 0.74 ± 0.18 | 0.92 ± 0.16 | <0.001 | 0.82 ± 0.14 | 1.02 ± 0.15 | <0.001 |
| **E/e' ratio (septal), mean ± SD** | 14.8 ± 3.2 | 11.6 ± 2.6 | <0.001 | 12.8 ± 2.4 | 10.2 ± 2.1 | <0.001 |
| **Deceleration time (ms), mean ± SD** | 162.4 ± 24.6 | 186.2 ± 22.4 | <0.001 | 172.6 ± 20.8 | 196.4 ± 19.8 | <0.001 |
| **LA volume index (mL/m²)** | 34.8 ± 6.4 | 31.2 ± 5.8 | 0.002 | 32.4 ± 5.6 | 29.6 ± 5.2 | 0.004 |
| **TMAD MEASUREMENTS** | | | | | | |
| **TMAD – Septal (mm), mean ± SD** | 7.2 ± 1.4 | 11.4 ± 1.8 | <0.001 | 9.6 ± 1.6 | 12.8 ± 1.6 | <0.001 |

| | | | | | | |
|---|---|---|---|---|---|---|
| **TMAD – Lateral (mm), mean ± SD** | 9.2 ± 1.8 | 13.8 ± 2.0 | <0.001 | 11.2 ± 1.8 | 15.0 ± 1.8 | <0.001 |
| **TMAD – Mean (mm), mean ± SD** | 8.2 ± 1.2 | 12.6 ± 1.6 | <0.001 | 10.4 ± 1.4 | 13.9 ± 1.4 | <0.001 |
| **TMAD% (% of LV length)** | 12.8 ± 2.2 | 19.6 ± 2.6 | <0.001 | 16.4 ± 2.4 | 21.8 ± 2.4 | <0.001 |
| **TISSUE DOPPLER IMAGING** | | | | | | |
| **s' septal (cm/s), mean ± SD** | 5.2 ± 1.2 | 7.6 ± 1.4 | <0.001 | 6.4 ± 1.2 | 8.4 ± 1.2 | <0.001 |
| **s' lateral (cm/s), mean ± SD** | 6.4 ± 1.4 | 9.2 ± 1.6 | <0.001 | 7.8 ± 1.4 | 10.4 ± 1.4 | <0.001 |
| **e' septal (cm/s), mean ± SD** | 5.8 ± 1.2 | 7.8 ± 1.3 | <0.001 | 6.6 ± 1.1 | 8.6 ± 1.2 | <0.001 |

SD = Standard Deviation; LVEF = Left Ventricular Ejection Fraction; LVEDD = LV End-Diastolic Diameter; LVESD = LV End-Systolic Diameter; TMAD = Tissue Mitral Annular Displacement; LA = Left Atrium; TDI = Tissue Doppler Imaging. All p-values by paired t-test or Wilcoxon signed-rank test as appropriate.

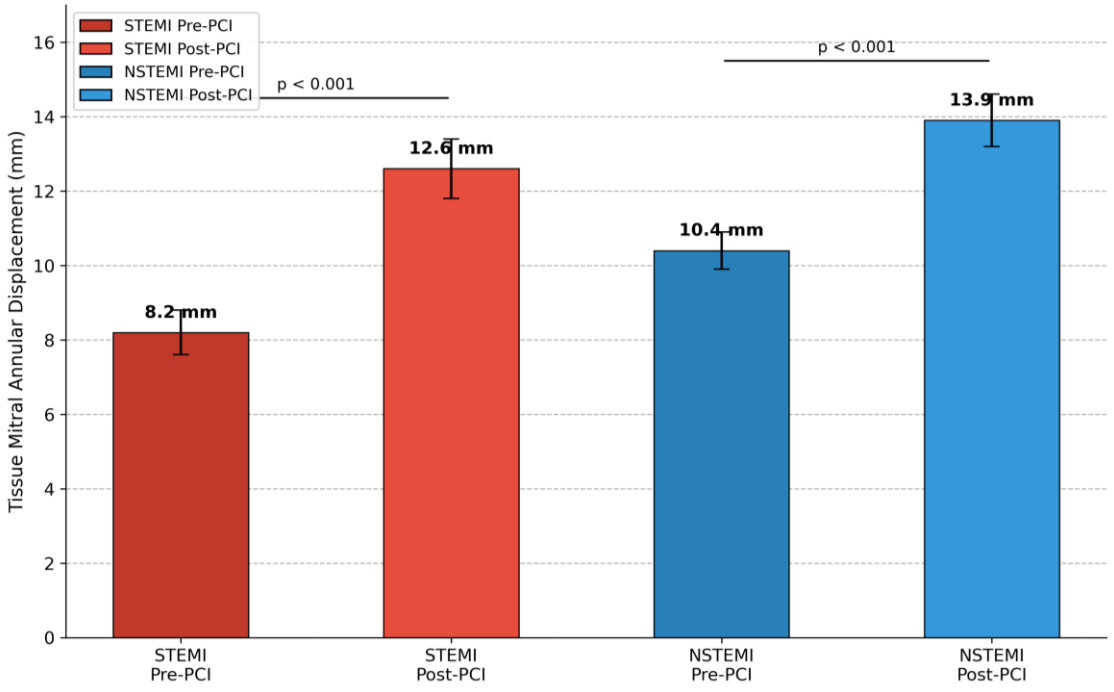


**Figure 1: Tissue Mitral Annular Displacement (TMAD) Values Pre- and Post-PCI in STEMI and NSTEMI Patients**

STEMI, n = 62; NSTEMI, n = 53. Error bars represent ± 1 standard deviation. Pre- vs. post-PCI comparisons within each group were made using the paired-samples t-test; statistical significance brackets indicate p < 0.001 for paired comparisons within each group.

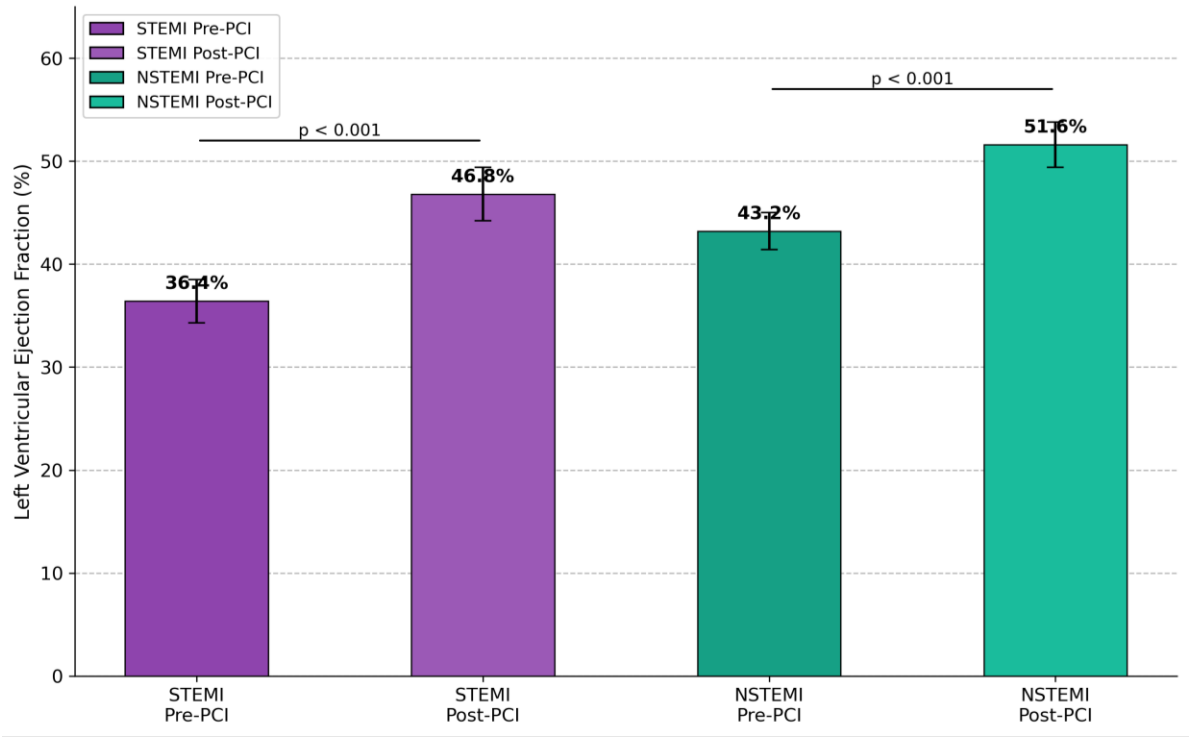


**Figure 2: Left Ventricular Ejection Fraction (LVEF) Recovery Pre- and Post-PCI in STEMI and NSTEMI Patients**

STEMI, n = 62; NSTEMI, n = 53. Error bars represent ± 1 standard deviation. Pre- vs. post-PCI comparisons within each group were made using the paired-samples t-test; statistical significance brackets indicate p < 0.001 for paired comparisons within each group.

Correlation of TMAD with Echocardiographic Parameters and Multivariate Regression

TMAD (mean) demonstrated a strong positive correlation with post-PCI LVEF (r = +0.80, p < 0.001) and with the magnitude of LVEF change (ΔLVEF; r = +0.74, p < 0.001). Strong correlations were also observed with TDI-derived s' lateral (r = +0.79) and s' septal velocities (r = +0.77), while significant negative correlations were noted with WMSI (r = −0.70), E/e' ratio (r = −0.56), LV end-systolic diameter (r = −0.66) and peak troponin-I levels (r = −0.50). The correlation patterns were consistent between both STEMI and NSTEMI subgroups, with slightly stronger coefficients observed in the STEMI group.

On multivariate linear regression analysis, after adjusting for age, sex, BMI, renal function and Killip class, TMAD (mean) pre-PCI (β = +1.82, 95% CI: 1.31–2.33, p < 0.001) and the change in TMAD post-PCI (β = +2.14, 95% CI: 1.53–2.75, p < 0.001) emerged as the strongest independent predictors of post-PCI LVEF improvement. MI type (STEMI vs. NSTEMI; β = −4.62, p < 0.001), TDI s' septal velocity post-PCI (β = +1.46, p = 0.001), peak troponin-I (β = −1.68, p = 0.002), and symptom-to-door time (β = −0.64, p = 0.008) were additional independent predictors. The multivariate model accounted for 78.6% of the variance in ΔLVEF ($R^2$ = 0.786, adjusted $R^2$ = 0.762, F = 34.2, p < 0.001). These data are summarised in Table 3.

**Table 3: Correlation of TMAD with LVEF and Multivariate Regression Analysis for Predictors of LV Recovery Post-PCI**

| **Section A: Pearson/Spearman Correlation of TMAD (Mean) with Selected Echo Parameters** | | | | | |
|---|---|---|---|---|---|
| **Parameter** | **STEMI r** | **STEMI p** | **NSTEMI r** | **NSTEMI p** | **Overall r** |
| LVEF (%) post-PCI | +0.82 | <0.001 | +0.78 | <0.001 | +0.80 |
| LVEF Δ change (post–pre) | +0.76 | <0.001 | +0.72 | <0.001 | +0.74 |
| Wall Motion Score Index | −0.71 | <0.001 | −0.68 | <0.001 | −0.70 |
| E/A ratio post-PCI | +0.64 | <0.001 | +0.61 | <0.001 | +0.63 |
| E/e' ratio post-PCI | −0.58 | <0.001 | −0.54 | 0.001 | −0.56 |
| s' septal TDI post-PCI | +0.79 | <0.001 | +0.74 | <0.001 | +0.77 |
| s' lateral TDI post-PCI | +0.81 | <0.001 | +0.76 | <0.001 | +0.79 |
| Peak Troponin-I | −0.52 | 0.003 | −0.48 | 0.004 | −0.50 |
| LV End-Systolic Diameter | −0.68 | <0.001 | −0.64 | <0.001 | −0.66 |
| Symptom-to-door time | −0.44 | 0.012 | −0.38 | 0.028 | −0.41 |
| **Section B: Multivariate Linear Regression – Independent Predictors of ΔLVEF (% improvement) Post-PCI** | | | | | |
| Predictor Variable | β coeff. | Std. Error | 95% CI | t-stat | p-value |
| TMAD Mean (mm) pre-PCI | +1.82 | 0.26 | 1.31 – 2.33 | 7.02 | <0.001 |
| TMAD Δ change post–pre (mm) | +2.14 | 0.31 | 1.53 – 2.75 | 6.90 | <0.001 |
| Type of MI (STEMI=1) | −4.62 | 0.98 | −6.56 – −2.68 | −4.71 | <0.001 |
| s' septal post-PCI (cm/s) | +1.46 | 0.38 | 0.71 – 2.21 | 3.84 | 0.001 |
| Peak Troponin-I (log ng/mL) | −1.68 | 0.48 | −2.63 – −0.73 | −3.50 | 0.002 |
| Symptom-to-door time (h) | −0.64 | 0.22 | −1.07 – −0.21 | −2.91 | 0.008 |
| Age (years) | −0.22 | 0.09 | −0.40 – −0.04 | −2.44 | 0.021 |
| Diabetes mellitus (yes=1) | −1.84 | 0.88 | −3.58 – −0.10 | −2.09 | 0.042 |
| Wall Motion Score Index | −3.26 | 1.12 | −5.48 – −1.04 | −2.91 | 0.008 |
| E/A ratio pre-PCI | +2.18 | 0.86 | 0.48 – 3.88 | 2.53 | 0.016 |
| $R^2$ = 0.786 | Adj $R^2$ = 0.762 | F = 34.2 | df = 10,104 | p < 0.001 | |

β = Unstandardised regression coefficient; CI = Confidence Interval; TMAD = Tissue Mitral Annular Displacement; Δ = change (post–pre PCI); r = Pearson correlation coefficient; Multivariate model adjusted for age, sex, BMI, renal function, and Killip class.

## 4. Discussion

The present study provides prospective, serial echocardiographic evidence that TMAD is a sensitive, clinically applicable marker of LV functional recovery following PCI in patients with both STEMI and NSTEMI, in an Eastern Indian tertiary care setting. The principal findings are threefold: (1) significant improvements in TMAD, LVEF and all complementary echocardiographic parameters occurred in both STEMI and NSTEMI patients post-PCI; (2) NSTEMI patients demonstrated higher baseline and post-PCI TMAD and LVEF values, consistent with a less severe myocardial insult; and (3) TMAD emerged as the strongest independent predictor of post-PCI LVEF improvement on multivariate regression analysis.

The robust improvement in LVEF observed in our STEMI cohort (from 36.4% to 46.8%) is broadly consistent with other reports of substantial post-PCI functional recovery. In a prospective STEMI cohort undergoing PCI, Attar et al. found that 63.3% of patients achieved a clinically meaningful (≥5%) LVEF improvement by 6-month follow-up, with the magnitude of recovery tracking closely with early changes in longitudinal strain [18]. This aligns with the broader principle, shown using strain-based viability assessment, that the extent of viable myocardium at baseline is a key determinant of subsequent functional recovery after AMI [19].

The differential baseline and post-PCI LVEF between STEMI and NSTEMI patients observed in our study is consistent with the pattern reported by Zahid et al., who found that annular displacement indices were relatively better preserved in NSTEMI than in STEMI, and that reduced annular motion independently predicted mortality in NSTEMI patients [20]. This difference is mechanistically attributable to the greater degree of transmural ischaemia, plaque rupture with complete occlusion and microvascular obstruction characterising STEMI, in contrast to the typically subtotal occlusion, collateral circulation and partial flow preservation seen in NSTEMI [21].

The strong positive correlation between TMAD and post-PCI LVEF ($r = +0.80$) observed in our study is in keeping with earlier investigations validating TMAD as a surrogate of LVEF. Suzuki et al. demonstrated a curvilinear but strong relationship between speckle-tracking-derived mitral annular displacement and LVEF across a range of cardiac diagnoses, including ischaemic heart disease [22]. Gjesdal et al. similarly showed that annular-displacement-based indices and global longitudinal strain are closely related, largely interchangeable descriptors of longitudinal LV function in chronic ischaemic heart disease [23], supporting the validity of TMAD as an LVEF surrogate in the post-infarction setting.

The significant improvement in TMAD% (from 12.8% to 19.6% in STEMI; 16.4% to 21.8% in NSTEMI) paralleled improvements in LV longitudinal shortening captured by TDI-derived s' velocities, which also improved significantly post-PCI. This finding supports the concept that longitudinal fibre recruitment, which is captured by both TMAD and TDI, is an early and sensitive indicator of myocardial viability and functional recovery following reperfusion [24]. Using cardiac MRI, Mayr et al. showed that a related longitudinal-function index, septal mitral annular plane systolic excursion, independently predicted major adverse cardiovascular events after STEMI and outperformed LVEF for this purpose, reinforcing the prognostic value of annular longitudinal motion beyond volumetric LVEF alone [25].

The finding that WMSI significantly improved post-PCI in both groups (1.84 to 1.42 in STEMI; 1.58 to 1.28 in NSTEMI) is consistent with the literature on myocardial stunning and hibernation. Panza et al., in a large trial of patients with ischaemic cardiomyopathy, showed that the extent of viable myocardium — of which WMSI is a bedside surrogate — was associated with the potential for functional recovery after revascularisation, although viability status alone did not reliably predict which individual patients would benefit most [26]. Importantly, in our multivariate model, WMSI independently predicted ΔLVEF ($\beta = -3.26$, $p = 0.008$), reinforcing the clinical utility of this straightforward, widely available index.

The negative independent association of symptom-to-door time with ΔLVEF (β = −0.64, p = 0.008) underscores the well-established time-dependency of myocardial salvage in AMI. The time-to-treatment relationship with LV functional outcomes has been extensively documented; Brodie et al. showed that longer door-to-balloon times were associated with higher late cardiac mortality, particularly in high-risk patients and those presenting early after symptom onset, underscoring that even modest treatment delays can measurably affect the amount of myocardium salvaged [27]. Our data from a predominantly rural referral population, where symptom-to-door times are inherently prolonged by geographic and logistical barriers, highlight the ongoing urgency of early MI recognition campaigns and the establishment of spoke-hub primary PCI networks in Odisha [28].

Peak Troponin-I independently predicted ΔLVEF negatively in our multivariate analysis (β = −1.68, p = 0.002), consistent with its established role as a surrogate of infarct size and a determinant of post-MI remodelling. Agewall et al. reviewed the determinants and interpretation of troponin elevation in coronary versus non-coronary disease, underscoring that the magnitude of troponin release in ACS reflects the extent of myocardial necrosis and, by extension, the amount of myocardium at risk for impaired recovery [29]. The prognostic implication is that patients with higher troponin burden warrant more intensive echo surveillance and earlier initiation of guideline-directed medical therapy to mitigate adverse remodelling.

The independent negative impact of diabetes mellitus on ΔLVEF (β = −1.84, p = 0.042) in our multivariate model is noteworthy, given the high prevalence of type 2 diabetes in our cohort (41.7%). Diabetic myocardiopathy, characterised by microvascular dysfunction, impaired calcium handling and increased myocardial fibrosis, may impair the capacity for functional recovery even after successful revascularisation [30]. These findings are consistent with a recent cardiac MRI study by Gao et al., which found that diabetes had an additive deleterious effect on LV strain after acute MI and that glycaemic control (HbA1c) was independently associated with the degree of myocardial deformation impairment [31], suggesting that glycaemic management may be a relevant therapeutic target for optimising post-PCI LV outcomes.

Our findings are consistent with, and add to, a growing recent literature on annular-displacement-based indices of longitudinal LV function. A 2024 peer-reviewed review of mitral annular plane systolic excursion (MAPSE, a closely related longitudinal-displacement index) across cardiovascular conditions concluded that such measures are a dependable and easy-to-use alternative to LVEF or global longitudinal strain for the assessment of LV systolic function, are readily obtainable even with suboptimal acoustic windows, and carry meaningful diagnostic and prognostic value across a range of clinical settings, while noting that no major imaging guideline currently mandates their routine measurement [32]. Our data, generated in an Eastern Indian STEMI/NSTEMI population undergoing PCI, extend this literature to the specific context of serial post-PCI recovery.

Strengths of this study include its prospective design, use of a standardised echocardiographic protocol by a single blinded operator, and a geographically distinct Indian cohort underrepresented in the existing literature. Several limitations should be considered when interpreting these findings. First, this was a single-centre study without a pre-specified a priori sample size calculation; the enrolled cohort of 115 patients should therefore be regarded as a pragmatic convenience sample, and the ratio of predictors to events in the multivariate model warrants cautious interpretation of the regression estimates.

Second, we did not perform a formal intra- or inter-observer reproducibility analysis (e.g., intraclass correlation coefficient or Bland–Altman analysis) for TMAD measurements in this cohort; given that a single operator performed all studies, some observer-dependent bias cannot be excluded, and reproducibility data should be reported in future work. Third, we did not perform ROC/AUC analysis to define a discriminative TMAD threshold for predicting LVEF recovery; the correlation and regression coefficients reported here describe association, not diagnostic discrimination, and should not be interpreted as validating TMAD as a binary predictive test.

Fourth, formal regression diagnostics (variance inflation factors for multicollinearity, residual normality and homoscedasticity checks) were not reported; given the number of predictors relative to the sample size, the possibility of model overfitting cannot be excluded, and the regression estimates should be regarded as hypothesis-generating rather than confirmatory. Fifth, this study lacks cardiac MRI for infarct size and microvascular obstruction quantification, long-term clinical follow-up (mortality, heart failure hospitalisation or major adverse cardiovascular events) beyond 6 weeks, and independent external validation.

Selection bias inherent to a single tertiary-centre consecutive sample, and the possibility of residual confounding despite multivariate adjustment, should also be considered. Pre-specified sensitivity analyses (e.g., restricting the regression model to STEMI or NSTEMI patients alone, or excluding patients with diabetes mellitus) were not performed in the present cohort and are recommended in future work to confirm the robustness of the TMAD–ΔLVEF association across clinical subgroups. Future multi-centre, adequately powered studies with pre-specified sample size calculations, formal reproducibility assessment, ROC-based threshold validation, cardiac MRI co-validation, and longer clinical follow-up are warranted to confirm and extend these findings.

## 5. Conclusion

The present study shows that TMAD was strongly and independently associated with the magnitude of LV functional recovery following PCI in both STEMI and NSTEMI patients. Both groups exhibited significant post-PCI improvements in TMAD, LVEF and complementary echocardiographic indices, with NSTEMI patients showing superior baseline and post-PCI values reflecting a less severe myocardial insult. On multivariate analysis, TMAD showed the strongest association with the magnitude of LVEF recovery post-PCI among the variables tested. These observational findings support the routine incorporation of TMAD into post-PCI echocardiographic assessment protocols, particularly in resource-constrained tertiary cardiac care settings where speckle-tracking echocardiography is not universally available.

## 6. Declarations

**Ethical Approval:** The study was approved by the Institutional Ethics Committee, SCB Medical College and Hospital, Cuttack, Odisha, India.

**Informed Consent:** Written informed consent was obtained from all participants.

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**Conflict of Interest:** The authors declare no conflict of interest.

**Data Availability:** The datasets generated and/or analysed during the current study are not publicly available due to institutional data-sharing restrictions but are available from the corresponding author upon reasonable request, subject to institutional and ethics committee approval.

**Author Contributions:** BD and PKN conceptualised and designed the study. SS performed the echocardiographic assessments and data collection. NKM and SKM supervised the study and critically revised the manuscript. All authors read and approved the final manuscript.

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