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Predictors of heart failure at 3-month follow-up in patients undergoing pharmacoinvasive percutaneous coronary intervention for anterior wall STEMI: A cross-sectional study.

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Abstract

Background
Anterior wall ST-elevation myocardial infarction (STEMI) often leads to left ventricular (LV) dysfunction and clinical heart failure despite timely reperfusion. The pharmacoinvasive percutaneous coronary intervention (PCI) strategy, involving thrombolysis followed by planned PCI, is frequently used in regions where immediate primary PCI is not feasible. Identifying early predictors of heart failure after such interventions can improve risk stratification and follow-up strategies.

Objectives
To identify the baseline angiographic and clinical indicators of heart failure at the 3-month mark in patients who received PCI and thrombolysis for anterior wall STEMI.

Methods
480 individuals who received PCI and thrombolysis for anterior wall STEMI within 24 hours were included in this research. Age, gender, Killip class, cardiovascular risk factors, time to PCI (3–10, 10–17, and 17–24 hours), Baseline data included left ventricular ejection fraction (LVEF) at discharge and TIMI flow prior to and following PCI. At three months, heart failure was defined as echocardiographic LV dysfunction (LVEF <40%) and the presence of NYHA class II–IV symptoms. Independent predictors were found using multivariate logistic regression.

Results
Mean age was 57.3 ± 11.2 years; 82% were male. Sixty-eight patients (14.2%) developed HF at 3 months. HF was more common among those with Killip class ≥II (29.5% vs. 9.8%, p<0.001), delayed PCI (>17 hours) (20% vs. 10.3%, p=0.01), and lower baseline LVEF (40.1% vs. 46.7%, p<0.001). Independent predictors were baseline LVEF <40% (aOR 2.78, 95% CI 1.63–4.72), Killip class ≥II (aOR 2.15, 95% CI 1.24–3.71), and PCI >17 hours (aOR 1.96, 95% CI 1.08–3.57).

Conclusion
Low baseline LVEF, elevated Killip class, and PCI delay independently predict HF at 3 months following pharmacoinvasive PCI for anterior STEMI.

Recommendation
Early risk identification, minimizing PCI delays, and aggressive heart failure prevention strategies

Keywords: *STEMI, pharmacoinvasive PCI, heart failure, Killip class, left ventricular dysfunction.*
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Introduction
Heart failure (HF) remains one of the most frequent and serious complications following acute ST-segment elevation myocardial infarction (STEMI). Even with modern reperfusion strategies and guideline-directed medical therapy, the burden of post-infarction HF is substantial, leading to excess mortality, recurrent admissions, and reduced functional capacity [1,2]. The risk is especially pronounced in anterior wall infarctions supplied by the left anterior descending (LAD) artery, where extensive myocardial involvement predisposes to left ventricular (LV)

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dysfunction, adverse remodeling, and eventual symptomatic HF despite apparently successful reperfusion [3,4].

The mechanisms driving HF after myocardial infarction are multifactorial. Myocardial necrosis, ischemia–reperfusion injury, and microvascular obstruction combine with maladaptive neurohormonal activation to promote structural and functional deterioration of the LV [5]. These changes, which include chamber dilation, increased wall stress, and declining systolic performance, can begin within hours and progress for months if not addressed [6,7]. Clinically, post-MI HF may present as anything from mild exertional dyspnea to severe HF with reduced ejection fraction (HFrEF), with prognosis consistently worse in patients suffering anterior infarctions compared with inferior or lateral events [8].

Timely restoration of coronary blood flow is fundamental to STEMI management, as it reduces infarct size and mitigates adverse ventricular remodeling. PPCI, or primary percutaneous coronary intervention, is the recommended reperfusion technique, providing superior vessel patency and lower rates of reinfarction and stroke compared with fibrinolysis [9,10]. However, in many LMICs, barriers to immediate PPCI limit its availability. In such circumstances, a pharmacoinvasive strategy—initial fibrinolysis followed within 3–24 hours by angiography and PCI—has become widely adopted. Large randomized trials, including TRANSFER-AMI and STREAM, have shown this approach to deliver outcomes comparable to PPCI, while greatly expanding access to reperfusion in resource-constrained settings [11–13].

Despite these advances, a proportion of patients treated with pharmacoinvasive PCI still develop HF, reflecting heterogeneity in baseline characteristics, infarct size, and revascularization outcomes. Prior studies in the PPCI era have identified several predictors of post-MI HF, including older age, female sex, diabetes, hypertension, baseline LV dysfunction, higher Killip class at admission, anterior infarction, multivessel coronary disease, reperfusion delay, and poor pre-PCI TIMI flow [14–17]. Circulating biomarkers such as NT-proBNP and high-sensitivity troponin have also been linked to increased risk [18]. However, evidence focusing specifically on predictors of HF following pharmacoinvasive management of anterior wall STEMI remains scarce [19]. This study was therefore designed to evaluate clinical, angiographic, and procedural predictors of HF at three months in this high-risk subgroup, to improve risk stratification and guide earlier, targeted interventions.

Materials and methods

Study design and setting

This was a cross-sectional study conducted over 12 months (January 2023 to December 2023) in the Department of Cardiology, Government Medical College, Thiruvananthapuram, Kerala, India.

Study population

Inclusion criteria were adults (≥18 years) with anterior STEMI, defined by chest pain for>20 minutes and new ST-segment elevation in ≥2 contiguous leads according to standard sex- and age-specific thresholds, who received fibrinolysis (tenecteplase or streptokinase) followed by planned PCI within 24 hours. Exclusion criteria included rescue PCI for failed thrombolysis, cardiogenic shock, prior MI or revascularization within 6 months, structural or congenital heart disease, cardiomyopathy, and inability or unwillingness to complete follow-up.

Study protocol

All patients received guideline-based initial therapy with aspirin, a P2Y12 inhibitor (ticagrelor or clopidogrel), and anticoagulation, followed by fibrinolysis and subsequent coronary angiography with PCI within 24 hours. PCI was performed via radial or femoral access using standard techniques. Infarct-related artery, lesion morphology, stent characteristics, and TIMI flow grades were recorded; procedural success was defined as TIMI 3 flow with <20% residual stenosis.

Data collection and outcomes

Baseline demographics, cardiovascular risk factors, clinical presentation (symptom-to-needle time, Killip class, hemodynamics), laboratory results, and echocardiographic findings were documented. Echocardiography was repeated at 3 months to assess LVEF, regional wall motion, and LV thrombus. The primary outcome was HF at 3 months, defined as NYHA class II–IV with LVEF <40% and/or HF hospitalization. Secondary outcomes included in-hospital major adverse cardiovascular events (MACE: death, reinfarction, arrhythmias, pulmonary edema) and 3-month all-cause mortality.

Ethical considerations:

Ethical clearance was obtained from the Institutional Ethics Committee, Government Medical College, Thiruvananthapuram (Ref No: GMC/IEC/2022/172; Approval Date: 15/12/2022). Written informed consent was obtained from all participants.

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Bias and quality control:

Selection bias was minimized by enrolling consecutive eligible patients. Observer bias was reduced by blinding echocardiographers to clinical and procedural data. Missing data were handled using standardized follow-up protocols.

Follow-up and statistical analysis

At one and three months, follow-up was done over the phone or through outpatient appointments. SPSS v26 was used for data analysis. ANOVA or t-test was used to compare continuous variables, which were expressed as mean ± SD; Fisher's exact test or chi-square was used to assess categorical data. To find independent predictors of 3-month HF, variables with $p<0.10$ on univariate analysis were added to multivariate logistic regression. Adjusted odds ratios (OR) with 95% CI were used to report the results. The threshold for statistical significance was $p<0.05$.

Results

Baseline characteristics

A total of 480 patients with STEMI in the anterior wall received fibrinolysis and PCI within 24 hours. The mean age was 57.3 ± 11.2 years, and 82% were male. Cardiovascular risk factors included smoking/tobacco use (50%), hypertension (48%), dyslipidemia (45%), diabetes (39%), and family history of premature CAD (14%). At 3 months, 68 patients (14.2%) developed HF, while 412 (85.8%) did not.

Table 1. Baseline characteristics of the study population

Variable	HF at 3 Months (n=68)	No HF (n=412)	p-value
Age (years, mean ± SD)	60.5 ± 10.9	56.7 ± 11.3	0.01
Male sex, n (%)	56 (82.4)	338 (82.0)	0.94
Hypertension, n (%)	35 (51.5)	196 (47.6)	0.56
Diabetes mellitus, n (%)	29 (42.6)	159 (38.6)	0.53
Dyslipidemia, n (%)	34 (50.0)	182 (44.2)	0.37
Smoking, n (%)	30 (44.1)	210 (51.0)	0.29
Family history, n (%)	12 (17.6)	55 (13.3)	0.36
Killip class ≥ II, n (%)	20 (29.5)	40 (9.8)	<0.001
Baseline LVEF (%)	40.1 ± 6.8	46.7 ± 7.4	<0.001

Clinical predictors of HF

In addition to being considerably older, patients who developed HF were more likely to have poorer baseline LVEF and Killip class ≥II.

- Killip ≥II was related to a fourfold increased HF risk (aOR 2.15, 95% CI 1.24–3.71).
- Baseline LVEF <40% was the strongest predictor (aOR 2.78, 95% CI 1.63–4.72).

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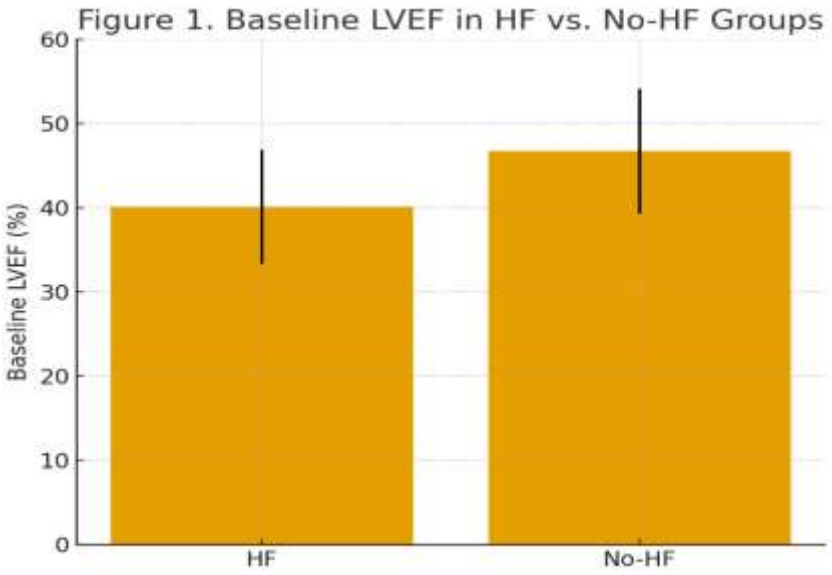

Figure 1. Baseline LVEF in HF vs. No-HF groups

Procedural characteristics

Delays in PCI strongly influenced outcomes. Patients treated >17 hours after fibrinolysis had higher HF rates (20% vs. 10.3%, p=0.01). This timing remained an independent predictor (aOR 1.96, 95% CI 1.08–3.57). In contrast, angiographic success rates were similar between groups, with no significant differences in pre-PCI TIMI flow, final TIMI 3 flow, or multivessel disease.

Table 2. Procedural and angiographic findings

Variable	HF (n=68)	No HF (n=412)	p-value
PCI 3–10 h, n (%)	25 (36.8)	185 (44.9)	0.21
PCI 10–17 h, n (%)	29 (42.6)	111 (26.9)	0.01
PCI 17–24 h, n (%)	14 (20.6)	42 (10.2)	0.01
Pre-PCI TIMI 0–1, n (%)	55 (80.9)	318 (77.2)	0.51
Post-PCI TIMI 3, n (%)	62 (91.2)	389 (94.4)	0.33
Multivessel disease, n (%)	23 (33.8)	102 (24.8)	0.12

Adverse outcomes

Patients who later developed HF experienced significantly more in-hospital complications and higher 3-month mortality.

- **In-hospital MACE:** 22.1% vs. 10.2% (p=0.004)
- **Reinfarction:** 5.9% vs. 1.7% (p=0.04)
- **3-month mortality:** 8.8% vs. 2.9% (p=0.02)
- **Serious arrhythmias (VT/VF):** 10.3% vs. 3.6% (p=0.03)

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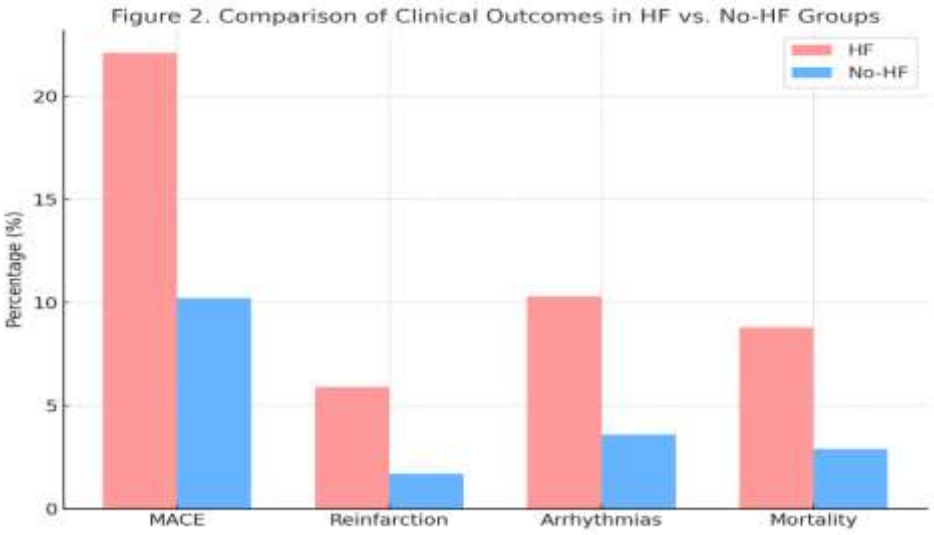

Figure 2. Comparison of clinical outcomes in HF vs. No-HF groups

Predictors of HF

On univariate analysis, Killip class ≥II, baseline LVEF <40%, and PCI >17 hours were associated with HF. Multivariate logistic regression confirmed these as independent predictors.

Table 3. Predictors of HF at 3 months

Predictor	Adjusted OR (95% CI)	p-value
LVEF <40%	2.78 (1.63–4.72)	<0.001
Killip class ≥ II	2.15 (1.24–3.71)	0.005
PCI >17 hours	1.96 (1.08–3.57)	0.026

The final model demonstrated good calibration (Hosmer–Lemeshow p=0.64) and strong discrimination (AUC=0.79).

Discussion

In this prospective study of 480 anterior wall STEMI patients treated with a pharmacoinvasive PCI strategy, the incidence of HF at 3 months was 14.2%. This is comparable to rates reported in previous STEMI cohorts managed with either primary PCI or pharmacoinvasive approaches, where post-MI HF rates range between 10–20% [2,14]. The risk is understandably higher in anterior wall infarctions due to the larger myocardial territory supplied by the LAD artery and its propensity for extensive necrosis and remodeling [3].

The strongest predictor of HF in our study was baseline LVEF <40%, which independently increased HF risk nearly threefold. This finding is consistent with studies demonstrating that initial LV dysfunction reflects the extent of myocardial injury and is a robust predictor of adverse outcomes, including HF and mortality [4,5]. Early echocardiographic assessment thus plays an essential role in identifying high-risk patients for intensified follow-up and optimized neurohormonal blockade. Similar findings were observed in the GISSI-3 and TRACE trials, where impaired LV function was closely linked with subsequent HF and long-term mortality. [20,21]

Patients presenting with Killip class ≥II were at double the risk of HF development at 3 months, even after adjusting for baseline LVEF and other confounders. This underscores the significance of initial hemodynamic compromise as a marker of larger infarct size and increased filling pressures.[22] Several registries, including the GRACE and FAST-MI programs, have consistently reported higher rates of HF, cardiogenic shock, and mortality in patients with elevated Killip class on admission [13,23]. Our findings confirm the predictive value of this simple bedside assessment in the pharmacoinvasive era.

Delayed PCI (>17 hours) was independently associated with a nearly twofold higher risk of HF compared to earlier PCI

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(3–10 hours). Timely revascularization reduces infarct size, limits adverse remodeling, and improves long-term LV function [9]. In the STREAM trial, earlier PCI following fibrinolysis resulted in outcomes comparable to primary PCI, whereas procedural delays were associated with increased adverse events [11]. Our results emphasize that even in pharmacoinvasive strategies, reducing transfer and procedural delays is crucial for optimal myocardial salvage and prevention of HF.

Traditional risk factors such as hypertension, diabetes, dyslipidemia, and smoking were not independent predictors of HF at 3 months. While these factors contribute to the pathogenesis of STEMI and long-term atherosclerotic progression, they appear to have less influence on short-term post-MI remodeling when compared with acute hemodynamic status and infarct size [4,8]. These findings are in line with prior studies showing that early post-MI HF is more closely linked to acute injury characteristics rather than chronic comorbidities [24].

Even though more than 90% of patients in both groups attained ultimate TIMI 3 flow, it did not independently predict HF development. This highlights that while epicardial patency is crucial, microvascular dysfunction, reperfusion injury, and infarct size may still drive remodeling and HF despite angiographic success [25]. Studies have shown that microvascular obstruction assessed by cardiac MRI correlates more closely with adverse remodeling than angiographic parameters alone [6].

Patients who developed HF had significantly higher in-hospital MACE and higher 3-month mortality (8.8% vs. 2.9%). This aligns with multiple studies confirming that HF following MI is linked to worse short- and long-term survival [18]. Early identification of high-risk patients provides an opportunity for aggressive optimization of guideline-directed medical therapy, structured rehabilitation, and closer follow-up.

The three independent predictors identified—baseline LVEF <40%, Killip class ≥II, and PCI delay >17 hours—are clinically relevant and easily measurable. Incorporating these into clinical decision-making can enhance early risk stratification. For example, patients with both impaired LVEF and delayed PCI could be prioritized for early initiation of RAAS inhibitors, beta-blockers, and possibly device therapy evaluation if dysfunction persists.

Generalizability

The study findings are most applicable to similar tertiary-care centers in resource-limited settings where pharmacoinvasive strategies are common. Although single-center in nature, the large sample size and uniform treatment protocol enhance external validity.

Conclusion

This study demonstrates that lower baseline LVEF, higher Killip class, and delayed PCI are independent predictors of HF at 3 months following anterior wall STEMI managed with a pharmacoinvasive PCI approach. These easily identifiable parameters should be integrated into risk stratification protocols to guide follow-up intensity and early therapeutic interventions aimed at reducing the burden of post-MI HF.

Limitations

The study's single-center design may have limited its generalizability. Biomarkers of neurohormonal activation (e.g., NT-proBNP) and advanced imaging (e.g., cardiac MRI) were not included but could provide additional insights into HF pathophysiology. Follow-up was limited to 3 months; long-term remodeling and HF outcomes were not assessed. However, the strength of the study lies in its focus on a homogeneous pharmacoinvasive STEMI population, an increasingly relevant strategy in regions without universal primary PCI access.

Abbreviation full form

STEMI ST-Elevation Myocardial Infarction
PCI Percutaneous Coronary Intervention
LVEF Left Ventricular Ejection Fraction
HF Heart Failure
LV Left Ventricle
NYHA New York Heart Association
MACE Major Adverse Cardiovascular Events
LAD Left Anterior Descending artery
OR Odds Ratio
CI Confidence Interval

Acknowledgement

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Data availability

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

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Conflict of interest
The authors declare no conflict of interest related to this study.

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Author contribution
Ramesh R. S. Conceptualization, Data Collection, Statistical Analysis, Manuscript Drafting
Praveen Velappan Study Supervision, Interpretation, Critical Review
Bijesh S. Methodology Design, Data Verification, Editing, and Final Approval

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