

**ORIGINAL RESEARCH**

# Timing of revascularization in NSTEMI: impact on clinical outcomes and functional recovery

<sup>1</sup>Dr. Jignesh Patel, <sup>2</sup>Dr. Karthik Natarajan, <sup>3</sup>Dr. Pratik Raval

<sup>1</sup>Consultant Cardiologist, Bharat Multispeciality Hospital and Research Institute, India

<sup>2,3</sup>Associate Professor, Department of Cardiology, U N Mehta Institute of Cardiology and Research Centre, India

**Corresponding Author**

Dr. Jignesh Patel

Consultant Cardiologist, Multispeciality Hospital and Research Institute, India

**Email:** [drkhudabaksh50@gmail.com](mailto:drkhudabaksh50@gmail.com)

Received: 09 February, 2025

Accepted: 26 February, 2025

Published: 16 March, 2025

**ABSTRACT**

**Background:** The optimal timing for percutaneous coronary intervention (PCI) in non-ST-segment elevation myocardial infarction (NSTEMI) remains controversial, particularly when intervention is delayed beyond 24 hours. The present study explores the clinical outcomes and functional recovery—focusing on left ventricular function—associated with different intervals of delayed PCI. **Methods:** In this prospective study, 120 NSTEMI patients who underwent PCI more than 24 hours after the index event were evaluated. Participants were grouped according to the time from symptom onset to revascularization (24–48 hours, 48–72 hours, and >72 hours). Baseline clinical data, risk profiles, and echocardiographic parameters were recorded. The primary outcomes included improvements in ejection fraction (EF) and global longitudinal strain (GLS). Mortality and major adverse cardiac events (MACE) were tracked for up to 6 weeks post-PCI. **Results:** Out of 120 patients, 56.67% underwent PCI between 24 and 48 hours, 25% between 48 and 72 hours, and the remaining beyond 72 hours. Patients revascularized at 24–48 hours demonstrated the greatest improvement in EF (88.24%) and GLS (94.12%). In contrast, those treated beyond 72 hours exhibited significantly lower, yet still notable, improvements. The rate of in-hospital complications was low across all groups, with an overall mortality of 4.17%. Risk factors such as diabetes, hypertension, and smoking status did not differentially affect the benefit of earlier versus later PCI in this cohort. **Conclusion:** Delaying PCI beyond 24 hours does not preclude significant clinical and functional improvement in NSTEMI patients. An intervention window of 24–48 hours appears optimal for maximizing LV functional recovery, although salvageable myocardium and clinical stability may allow benefits even after 72 hours. Further prospective trials are needed to definitively recommend the best timing strategy for these patients.

**Keywords:** Timing of PCI, NSTEMI, Delayed Revascularization, Ejection Fraction, Myocardial Recovery

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

**INTRODUCTION**

The management of non-ST-segment elevation myocardial infarction (NSTEMI) has evolved significantly over the past two decades, particularly with advances in diagnostic and interventional cardiology [1]. While early invasive strategies—often within 24 hours of presentation—are generally recommended in high-risk groups, real-world constraints and clinical variability can lead to delayed intervention [2]. Factors such as late hospital arrival, the availability of cardiac catheterization laboratories, and initial misclassification of patient risk can result in PCI occurring beyond the first 24 hours [3].

There remains a gap in understanding the impact of such delays on both clinical outcomes (e.g., mortality, recurrent ischemia) and functional recovery. Several clinical trials have investigated the concept of an

“early invasive strategy” versus a “delayed invasive strategy,” but these typically define “delayed” as 24–72 hours [4]. What happens when revascularization is pushed even further, to beyond 72 or 96 hours? Some observational data suggest that meaningful myocardial salvage can still occur if there is viable myocardium, while others argue that the benefits might wane considerably with longer delays [5].

Ejection fraction (EF) is an established prognostic indicator following myocardial infarction, serving as a key determinant of heart failure risk and long-term survival [6]. Global longitudinal strain (GLS) can detect subtle changes in myocardial systolic function earlier than EF and may serve as a sensitive endpoint in evaluating timing strategies [7]. Although theoretically, each hour of delay might translate into a proportion of myocardium at risk, the clinical reality

is more nuanced. Some myocardium may remain “stunned” or hibernating, waiting for perfusion to be restored, even days after the initial ischemic insult [7]. In this study, we aimed to evaluate the clinical outcomes and LV functional recovery (via EF and GLS) across specific delayed PCI intervals in NSTEMI patients. We postulated that there would be a graded benefit favoring shorter delays but hypothesized that even patients who underwent PCI after 72 hours might still demonstrate meaningful recovery. By analyzing a single-center cohort of NSTEMI patients treated with delayed PCI, this study seeks to elucidate the timing threshold beyond which benefits become marginal, and offer practical guidance for clinicians managing late-presenting or complex NSTEMI cases [3].

## MATERIALS AND METHODS

### Study Design

A prospective observational study was conducted between April 2021 and December 2022. Written informed consent was obtained from all participants.

### Patient Selection and Inclusion/Exclusion Criteria

- **Inclusion Criteria:**

- Adults (>18 years) with NSTEMI presenting  $\geq 24$  hours after symptom onset.
- Scheduled for PCI on the infarct-related artery beyond 24 hours from index presentation.

- **Exclusion Criteria:**

- STEMI presentations or patients requiring emergency reperfusion.
- Ventilator-dependent or in Killip Class IV at presentation.

### Data Collection

Baseline demographic and clinical variables were recorded, including age, gender, comorbidities

(hypertension, diabetes), and smoking status. Vital signs (BP, heart rate) and risk scores such as GRACE and TIMI were documented. Echocardiographic measurements of EF and GLS were obtained prior to PCI and repeated post-procedure during follow-up (4–6 weeks).

### Grouping by PCI Timing

Patients were stratified into three main groups based on time from index event to PCI:

1. 24–48 hours
2. 48–72 hours
3. 72 hours

### Procedural Details

PCI was performed per institutional protocol using drug-eluting stents. Adjunctive therapies (antiplatelet agents, anticoagulants, statins, beta-blockers) were utilized as appropriate.

### Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS (v21). Continuous variables are presented as mean  $\pm$  SD or median (IQR). Between-group comparisons were made using ANOVA or Kruskal–Wallis tests for continuous variables, and chi-square or Fisher’s exact tests for categorical variables. A p-value  $< 0.05$  was considered statistically significant.

## RESULTS

### Baseline Characteristics

Among the 120 patients, the distribution of risk factors was similar across all timing groups. Diabetes was observed in 49.17%, hypertension in 45%, and active smoking in 52.5%. The majority of patients (56.67%) underwent PCI between 24 and 48 hours, 25% between 48 and 72 hours, and 18.33% beyond 72 hours (Table 1).

**Table 1. Distribution by Timing of Revascularization**

| Time Interval | n  | %      |
|---------------|----|--------|
| 24–48 hours   | 68 | 56.67% |
| 48–72 hours   | 30 | 25.00% |
| >72 hours     | 22 | 18.33% |

(Note: The total N=120. Percentages may not sum to exactly 100% due to rounding.)

### Clinical Outcomes

Overall mortality was 4.17% (5 deaths). Cardiogenic shock at presentation was documented in 18.33% (22/120) of patients. However, the occurrence of cardiogenic shock did not differ significantly between the 24–48 hour group and the >72 hour group ( $p=0.18$ ). The majority of patients (80.83%) were hemodynamically stable (SBP  $\geq 100$  mmHg) at presentation.

**Table 2. Adverse Events and Mortality**

| Outcome                  | 24–48 hrs (n=68) | 48–72 hrs (n=30) | >72 hrs (n=22) | p-value |
|--------------------------|------------------|------------------|----------------|---------|
| Death                    | 2                | 2                | 1              | 0.68    |
| Cardiogenic Shock        | 12               | 5                | 5              | 0.54    |
| Readmission (LVF/Angina) | 10               | 3                | 4              | 0.32    |

**EF and GLS Recovery**

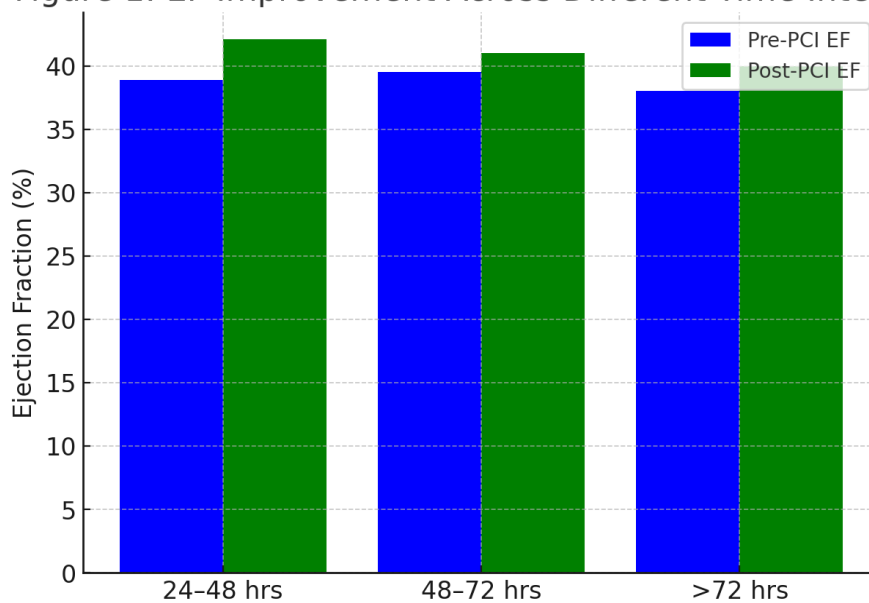
Pre-PCI EF across the groups was comparable, averaging around 38–40%. Post-PCI EF improved significantly ( $p < 0.05$ ) in all intervals, with the greatest change in the 24–48 hour group (mean improvement ~3.2%). GLS also showed a similar pattern: borderline and low GLS categories experienced improvement rates >90% (Table 3).

**Table 3. Changes in EF and GLS by Timing Group**

| Timing Group | EF Pre (%)  | EF Post (%) | GLS Improvement (%) |
|--------------|-------------|-------------|---------------------|
| 24–48 hrs    | 38.9 ± 9.1  | 42.1 ± 8.5  | 94.12               |
| 48–72 hrs    | 39.5 ± 10.2 | 41.0 ± 9.6  | 66.67               |
| >72 hrs      | 38.0 ± 9.5  | 40.0 ± 8.4  | 65.00               |

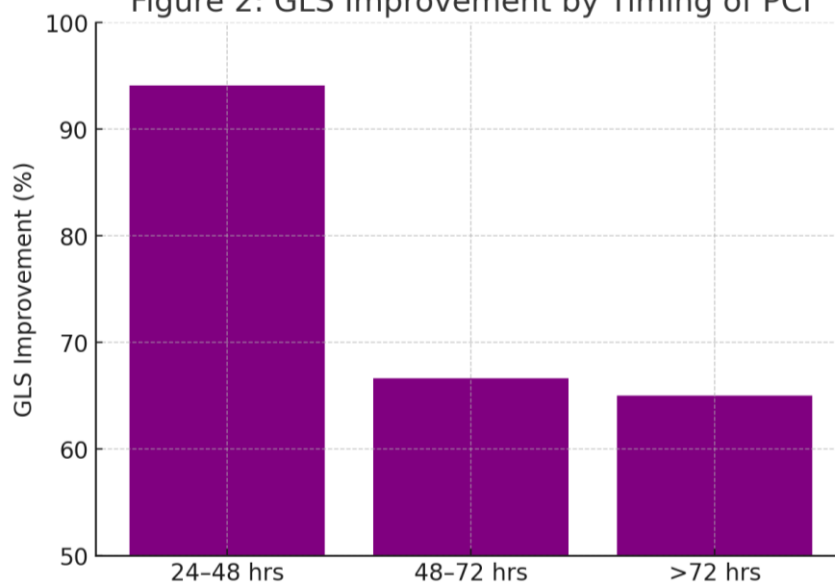
(Improvement rates in GLS refer to proportion of patients whose GLS normalized or improved by at least one category.)

**Figure 1: EF Improvement Across Different Time Intervals**



**Figure 1. EF Improvement Across Different Time Intervals**  
(Bar chart showing EF improvement for each timing group.)

**Figure 2: GLS Improvement by Timing of PCI**



**Figure 2. GLS Improvement by Timing of PCI**

(Bar chart or line graph illustrating the proportion of patients showing GLS improvement in each timing group.)

## OTHER OBSERVATIONS

- **Risk Factor Influence:** Diabetes, hypertension, and dyslipidemia did not significantly affect the magnitude of EF/GLS improvement across the three time intervals ( $p>0.05$ ).
- **Hospital Stay and Readmission:** Average length of hospital stay was slightly higher for the >72 hour group, though not statistically significant. Readmissions within 6 weeks were mostly for LV failure or angina.

## DISCUSSION

Our study confirms that, although early intervention (within 24 hours) is ideal for NSTEMI, a delay up to 48 or even 72 hours does not eliminate the potential for meaningful clinical and functional improvements [8]. Patients revascularized between 24 and 48 hours demonstrated the highest rate of EF and GLS improvement, underscoring the advantages of intervening sooner rather than later [9]. However, the observation that patients treated beyond 72 hours still had notable improvements (though less pronounced) suggests that a portion of the myocardium remains salvageable beyond the classical early window, possibly due to hibernation or residual collateral circulation [10].

Recent trials have revealed mixed findings about the benefit of an “early invasive strategy” within 24 hours, as opposed to a more delayed approach. Nonetheless, those trials often define “early” as 12–24 hours and “delayed” as 48–72 hours [11]. In real-world practice, logistical constraints can push interventions beyond 72 hours [12]. Our data offer reassurance that while prolonged delay is not optimal, it does not necessarily condemn patients to poorer outcomes if they remain hemodynamically stable and do not exhibit ongoing ischemia [13]. This has significant implications for centers with limited catheterization lab availability and for patients who arrive late.

An interesting finding is the independence of risk factors like diabetes, hypertension, and smoking from the observed benefit of revascularization in each time group. This implies that the crucial factor might be the inherent viability of myocardial segments rather than the presence or absence of conventional risk factors. Mortality was relatively low (4.17%), suggesting that delayed PCI can be performed safely with proper monitoring. However, the proportion of patients presenting with cardiogenic shock underscores the importance of vigilant clinical assessment. Delayed intervention in an unstable patient is generally contraindicated, so appropriate risk stratification remains paramount [14].

In conclusion, our study highlights that while earlier revascularization (within 24–48 hours) is preferable, clinically stable patients can still derive substantial benefits even with interventions beyond 72 hours. Timely identification of persistent ischemia, thorough viability assessment, and good patient selection

criteria are vital for maximizing the success of delayed PCI strategies in NSTEMI populations.

## CONCLUSION

This study demonstrates that meaningful improvements in left ventricular function and favorable clinical outcomes can be achieved even when PCI for NSTEMI is delayed beyond 24 hours. Revascularization within 48 hours is associated with the most pronounced EF and GLS gains, while intervention up to or beyond 72 hours still offers measurable benefits for selected patients. These findings highlight the importance of balancing clinical urgency and logistical constraints in real-world settings, suggesting that a nuanced approach to timing can optimize patient outcomes without entirely forfeiting the potential for myocardial recovery.

## REFERENCES

1. **B De Bruyne et al.** J Am Coll Cardiol 67:1170, 2016.
2. **Levine GN et al.** 2016 ACC/AHA guideline focused update on duration of dual antiplatelet therapy in patients with coronary artery disease: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines: An Update of the 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention, 2011 ACCF/AHA Guideline for Coronary Artery Bypass Graft Surgery, 2012 ACC/AHA/ACP/AATS/PCNA/SCAI/STS Guideline for the Diagnosis and Management of Patients for the Management of ST-Elevation Myocardial Infarction, 2014 AHA/ACC Guideline for the Management of Patients with Non-ST-Elevation Acute Coronary Syndromes, and 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery. *Circulation* 134:e123, 2016.
3. **Mozaffarian D et al.** Heart disease and stroke statistics—2016 update: A report from the American Heart Association. *Circulation* 133:e38, 2016.
4. **Virchow RLK.** *Sechszehnte Vorlesung. Die Cellularpathologie.* Berlin, A. Hirschwald, 1858, pp. 308–329.
5. **Weisse AB.** The elusive clot: The controversy over coronary thrombosis in myocardial infarction. *J Hist Med Allied Sci* 61:66–78, 2006.
6. **Constantinides P.** Plaque fissures in human coronary thrombosis. *J Atheroscler Res* 6:1–17, 1966.
7. **Chapman I.** Morphogenesis of occluding coronary artery thrombosis. *Arch Pathol* 80:256–261, 1965.
8. **Friedman M, Van den Bovenkamp GJ.** The pathogenesis of a coronary thrombus. *Am J Pathol* 48:19–44, 1966.
9. **Van der Wal AC, Becker AE, van der Loos CM, Das PK.** Site of intimal rupture or erosion of thrombosed coronary atherosclerotic plaques is characterized by an inflammatory process irrespective of the dominant plaque morphology. *Circulation* 89:36–44, 1994.
10. **Mody P et al.** Antianginal agents for the management of stable ischemic heart disease: A review. *Cardiol Rev* 24:177–185, 2016.
11. **Omland T, White HD.** State of the art: Blood biomarkers for risk stratification in patients with stable ischemic heart disease. *Clin Chem* 63:165–176, 2017.

12. **LJ Shaw et al.** J Am Coll Cardiol 54:1561–1574, 2009.
13. **Hollander JE et al.** State-of-the-art evaluation of emergency department patients presenting with potential acute coronary syndromes. *Circulation* 134:547–564, 2016.
14. **Montecucco F et al.** Pathophysiology of ST-segment elevation myocardial infarction: Novel mechanisms and treatments. *Eur Heart J* 37:1268–1280, 2016.