Biological and Clinical Sciences Research Journal
eISSN: 2708-2261; pISSN: 2958-4728
www.bcsrj.com
DOI: https://doi.org/10.54112/bcsrj.v6i6.1809
*Biol. Clin. Sci. Res. J., Volume 6(6), **2025**: 1809*
Original Research Article

Early Metoprolol Administration before Primary Percutaneous Coronary Intervention (PCI) in ST-Segment Elevation Myocardial Infarction: Impact on Left Ventricular Ejection Fraction

Muhammad Amin, Tariq Nawaz*, Sadam Hussain, Syed Muhammad Nayab Ali, Faisal Saleh Hayat

Department of Cardiology, Lady Reading Hospital Peshawar, Pakistan
**Corresponding author`s email address:* drtariqnz@gmail.com

(Received, 14th May 2025, Accepted 18th June 2025, Published 30th June 2025)

Abstract: *Ischemic heart disease is the main cause of death globally, with STEMI being its most severe sign. Beta-blocker lower systemic blood pressure, heart rate, decreases ischemia damage when given before PPCI, particularly when metoprolol is used.* ***Objective:*** *the aim of this study was to find out the effect of early metoprolol administration before Primary Percutaneous Coronary Intervention (PCI) in STEMI.* ***Methods:*** *The current Randomized Controlled Trial was carried out at the Department of Cardiology, Lady Reading Hospital, Peshawar from 1st February 2025 to 31st April after taking permission from the ethical committee of the institute. A total of 60 participants were included and divided in to two groups and each group had 30 individuals. All collected data was entered into a computerized database, and SPSS version 25.0 was used for data analysis. To compare the effectiveness of early metoprolol administration before PCI in ST-segment elevation myocardial infarction on LVEF, an independent sample t-test was used to compare the mean LVEF values between the treatment group and the control group. A p-value of <0.05 were considered statistically significant.* ***Results:*** *Over all 60 individuals participated in the current study. The LVEF of metoprolol group was substantially higher than the control group at one week, three months, and 6 months following PCI (P <0.05). Before starting therapy, the two groups' LVEFs did not differ significantly (P >0.05). The intravenous metoprolol group's LVEF was considerably greater than the control group's at one week, three months, and six months following PCI. The Intravenous Metoprolol group's 6 months overall incidence of MACE was noticeably lower than the control groups.* ***Conclusion:*** *The current study concluded that in individuals with ST-Segment Elevation Myocardial Infarction early metoprolol administration before Primary Percutaneous Coronary Intervention (PCI) in ST-Segment Elevation Myocardial Infarction increase left ventricular ejection, can improve heart rate regulation, lessen myocardial damage, and enhance cardiac function and lower the risk of serious adverse cardiac events.*

Keywords: Metoprolol; Primary Percutaneous Coronary Intervention; ST-Segment Elevation Myocardial Infarction; Left Ventricular Ejection Fraction

[***How to Cite:*** Amin M, Nawaz T, Hussain S, Ali SMN, Hayat FS. Early metoprolol administration before primary percutaneous coronary intervention (PCI) in st-segment elevation myocardial infarction: impact on left ventricular ejection fraction. *Biol. Clin. Sci. Res. J.*, **2025;** 6(6): 20-24. doi: https://doi.org/10.54112/bcsrj.v6i6.1809

Introduction

A major risk to individuals with coronary artery disease is ST-segment elevation myocardial infarction (STEMI). According to a 2016 World Health Organization report, ischemic heart disease is the leading cause of death globally, with STEMI being its most severe sign. (1) To enhance outcomes and lower death rates, immediate intervention needs to be taken and recent developments in medical therapy have improved the management of STEMI. (2) Despite these advancements, congestive heart failure, cardiac arrhythmia, and sudden death remain among the recurring cardiac problems that STEMI survivors are more likely to have. (3, 4) Employing primary percutaneous coronary intervention (PPCI) to enable early reperfusion within 120 minutes after diagnosis is the current standard for treating STEMI. (5) It has been demonstrated that beta-blockers, a family of medications that lower systemic blood pressure, heart rate, myocardial contractility, and neutrophil function, decrease ischemia damage when given before PPCI, particularly when metoprolol is used. (6) It is believed that this benefit results from the drug's capacity to reduce reperfusion injury. According to current management recommendations for STEMI, if the patient has a systolic blood pressure of more than 120 mmHg, no contraindications, and no evidence of acute cardiac failure, early intravenous beta-blockade should be administered upon presentation before PPCI. The METOCARD-CNIC Trial led to this recommendation since it demonstrated the benefits of metoprolol in cardioprotection following an acute myocardial infarction (MI). (7) According to previous studies metoprolol significantly reduces the size of myocardial infarcts while maintaining left ventricular (LV) function. (8, 9) In one study, when metoprolol was administered intravenously during out-of-hospital assistance and transfer to the primary angioplasty center, the infarct size was smaller (23.4 g) and the left ventricular ejection fraction (LVEF) was higher (48.1 ± 8.4) than in controls (34.0 g and 43.1 ± 10.2, respectively), yet the safety risks were not increased. (8) In a different research, IV-metoprolol was linked to reduced rates of severely depressed LVEF <35%, lower rates of patients needing ICD installation, lower rates of heart failure hospitalization, and greater LVEF (48.7±9.9 vs. 45.0± 11.7% in control individuals) . (9) The main objective of this research was to examine how pre-PCI metoprolol treatment affects LVEF in our population's STEMI patients. Although a number of studies have been carried out in other groups, none have been carried out in our community as of yet, and it is crucial to ascertain if the findings of these research apply to our population. Furthermore, the findings of earlier research have been ambiguous; some have shown that pre-PCI metoprolol treatment improves LVEF, while others have found no discernible impact. Thus, the purpose of this study is to fulfil the information gap by examining how pre-PCI metoprolol administration affects LVEF in our population. This might have significant consequences for the treatment of STEMI patients in our locality.

Methodology

The current Randomized Controlled Trial was carried out at the Department of Cardiology, Lady Reading Hospital, Peshawar from 1st February 2025 to 31st April after taking permission from the ethical committee of the institute. Individuals of both gender and of different age groups (18-70 years) with ST-segment elevation myocardial infarction (STEMI) undergoing PCI within 12 hours of symptom onset were

included while individuals with Acute heart failure, Non ST-elevation MI (NSTEMI), Systolic blood pressure persistently <120 mm Hg, PR interval >240 milliseconds, Type II to III atrioventricular block (AV Block) and heart rate persistently <60 beats/min were excluded. The sample size was determined using the WHO software for sample size calculation, with a power of 80% and a level of significance of 5%. Consecutive sampling (non-probability) was used for data collection. A total of 60 participants were included in this study. The participants were divided in to two groups and each group had 30 individuals. Patients in the treatment group received intravenous metoprolol up to 3 5- mg boluses of metoprolol tartrate before PCI while individuals in the control group received standard treatment as per guidelines, including long-term oral treatment with ß-blockers (first dose after PCI) in all participants with no side effect.

Patients presented with signs and symptoms suggestive of myocardial infarction to emergency department of cardiology unit LRH and meeting the inclusion criteria were enrolled in this study. Informed consent was obtained from the next of kin who were willing to participate in the study. Detailed medical history and physical examination were performed. ECG were performed to confirm the diagnosis of STEMI as per operational definition and cardiac biomarkers were sent. Baseline demographic data, including age, gender, address, and co-morbidities (hypertension, diabetes mellitus, smoking status, and dyslipidemia), were recorded for each patient. LVEF were measured at baseline (within 24 hours), 3rd post-PCI, and at discharge using echocardiography. The LVEF were classified as normal, mild LV dysfunction, moderate LV dysfunction, or severe LV dysfunction. Collected data were entered into a pre-designed proforma and analyzed using appropriate statistical methods. The data collected were kept confidential, and measures were taken to ensure patient safety.All collected data were entered into a computerized database, and statistical analysis were performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize patient demographics, comorbidities, and LVEF measurements. Continuous variables such as age, LVEF, time of PCI from symptoms onset were expressed as means + standard deviations (SD), whereas categorical variables such as gender, comorbidities were expressed as frequencies and percentages. To compare the effectiveness of early metoprolol administration before PCI in ST-segment elevation myocardial infarction on LVEF, an independent sample t-test was used to compare the mean LVEF values between the treatment group and the control group at each time point (baseline, 3rd post-PCI, and discharge). The p value less than 0.05 were considered statistically significant. To analyze the association between LVEF and categorical variables (such as co-morbidities), a chi-squared test were used. The value of p less than 0.05 was considered statistically significant. The study results were presented in tables and figures to facilitate data interpretation and reporting.

Results

Over all 60 individuals participated in the current study classified in to control group and treatment group equally. None of the baseline features differed significantly between both groups (P value greater than 0.05). The demographic features of the both group are represented in table 1.

It was found that there was no noteworthy variance in LAD among the individuals in both groups before the therapy (Value of p greater than 0.05), and that the metoprolol group's LVEF was substantially greater than the control group's at one week, three months, and 6 months following PCI (P <0.05). Before starting therapy, the two groups' LVEFs did not differ significantly (P >0.05). The intravenous metoprolol group's LVEF was considerably greater than the control group's at one week, three months, and six months following PCI (P <0.05) as presented in table 2.

Starting the therapy the heart rate was not different significantly in both groups (value of P less than 0.05). At one week and one month following PCI, the intravenous metoprolol group's heart rate was considerably lower than the control groups (the value of P less than 0.05). No important change in pulse in both groups at three and 6 months following PCI (value of P less than 0.05). Blood pressure levels in both of the groups before and following therapy did not differ significantly as presented in table 3.

Each individual was followed up for 6 months. In the Intravenous Metoprolol group, the overall frequency of heart mortality, failure of heart readmission, & recurring cardiac issues was 2.6%, compared to 20.percent in the placebo group. According to Kaplan-Meier analysis, the Intravenous Metoprolol group's 6 months overall incidence of MACE was noticeably lower than the control groups (table 4). On Days 2 and 3 following PCI, the Intravenous Metoprolol group's B-type natriuretic peptide was considerably lower than the control group's (P <0.05). On Day 5 following PCI, the Intravenous Metoprolol group's high-sensitivity cardiac troponin I was considerably lower than the control group's (P <0.05).The two groups' baseline and 6 months follow-up levels of β-blocker, and dual antiplatelet medicines did not differ significantly (P >0.05). (Table 5)

After 6 months follow-up, the two groups' liver and kidney functions did not differ significantly (P >0.05). **(Table 6)**

Table 1. Demographic features of the study participants

	All patients = 60	
	Intravenous Metoprolol	**Control group**
Age in years	58.7±12.7	58.2±10.8
Sex		
Male	24(80%)	24(80%)
Female	6(20%)	6(20%)
Smoker %		
Ex-smoker (0–10 y before	3(10%)	3(10%)
Current smoker	27(90%)	27(90%)
Dyslipidemia	12(40%)	14(46.6%)
Co-morbidities		
hypertension	26(86.6%)	25(83.3%)
diabetes mellitus	15(50%)	20(66.6%)

Table 2. Comparing the two groups' echocardiography-derived parameters

Parameters	**Baseline changes**					
	Metoprolol group	Control group	Value of P	Metoprolol group	Control group	P value
LVEF %						
Baseline level	53.04 ±6.44	51.07 ±9.04	0.622			

Seven days	58.00 ±7.87	51.70 ±10.79	0.042	5.30 ±5.96	0.35 ±5.36	0.010
3 months	57.57 ±9.99	51.97 ±10.30	0.023	4.39 ±8.86	0.70 ±8.26	0.107
6 months	59.32 ±8.11	51.94 ±11.75	0.006	6.87 ±8.14	1.35 ±10.35	0.022

Table 3. Blood pressure and heart rate comparison between the 2 different groups

Parameters	**Changes from baseline**					
	Metoprolol group	**Control group**	**Value of P**	**Metoprolol group**	**Control group**	**P value**
Heart rate (bpm)						
Baseline level	91.00 ±8.81	88.95 ±9.65	0.213			
seven days	74.20 ±4.54	77.95 ±5.87	0.010	- 17.80 ±8.23	-11.93 ±5.78	0.001
3 months	66..38 ±4.06	67.83 ±4.40	0.142	-25.63 ±6.61	-22.23 ±8.99	0.025
6 months	66.50 ±3.34	67.83 ±3.18	0.657	-25.50 ±7.12	21.93 ±8.98	0.022
Systolic /diastolic blood pressure						
Baseline level	135.23 ±22.05/ 77.65 ±12.56	128.43±19.5/ 75.20±9.00	0.14			0.404
7 days	130.23 ±12.98 / 76.00 ±7.85	124.83±13.18/ 73.03±6.56	0.74	-1.65 ±5.56	-1.63±2.93	o.5999
3 months	130.50±11.63 /75.55±5.55	126.45±9.54/ 74.23±4.90	0.69	2.10 ±7.83	-2.10±4.92	0.444
6 months	65.50±3.34 / 75.71 ±8.71	127.35±17.43/ 74.77 ±10.22	0.722	-1.67 ±15.78	-167±12.96	0.78

Table 4. Comparison of the two populations' respective major adverse cardiac events incidences

Features	**Metoprolol group**	**Control group**	**P value**
Cardiogenic death	0(0.00)	0(0.00)	1.000
Heart failure readmission	0(0.00)	5(16.6%)	0.018
Reinfarction	2(3)	2(3%)	1.000
major adverse cardiac events	2(3%)	6(20%)	0.034

Table 5. Evaluation of the two groups' liver and renal functions

Features	**Metoprolol group**	**Control group**	**P value**
Uric acid median mmol/L)]	378	358 .00	0.47
Nitrogen urea median mmol/L)	5.2100	7.12	0.162
Creatinine median μmol/L)	81.00	82.12	0.50
AST mmol/L)]	21	21	0.89
ALT mmol/L)]	19	21	0.38

Discussion

Following an acute myocardial infarction, long-term beta-blocker medication increases survival. (10) According to present guidelines for ST-segment elevation myocardial infarction (STEMI), individuals without contraindications should begin taking beta-blocker medications during the first 24 hours following myocardial infarction. (11)It is unclear, therefore, if administering an intravenous beta-blocker early in a continuing myocardial infarction offers any further benefits. (12) The prereperfusion era provides the majority of the data on the advantages of starting an intravenous beta-blocker early in myocardial infarction, with not much data available to individuals having primary angioplasty. (13) the current study was carried out to determine the Impact on Left Ventricular Ejection of Early Metoprolol Administration before PPCI STEMI. According to our data, metoprolol administered intravenously during EMS support as well as transfer to the PPCI centre is safe and reduces the size of infarcts while increasing left ventricular ejection fraction when compared to individuals who did not receive metoprolol prior to percutaneous coronary intervention. In STEMI survivors, the two primary indicators of long-term morbidity and death are infarct size and left ventricular ejection percent. (14) Echocardiography was used in this investigation to assess heart function. At various time intervals, it was discovered that the Metoprolol group group's LVEF was noticeably greater than that of the control group. In their evaluation of cardiac remodeling using cardiac magnetic resonance, Gerbaud et al (15) discovered that metoprolol combined to standard medication therapy might considerably increase LVEF three months following PCI. Metoprolol may increase LVEF among individuals with cardiogenic shock aggravating STEMI, according to research by Barilla et a. (16) At three months following PCI, Xu et al (17) discovered left ventricle end diastolic volume (LVEDV), left ventricle peak systolic volume (LVESV), while left ventricular end EF were significantly higher in the metoprolol group than in the control group.; however, at six months following PCI, no significant changes were seen. Thus, they came to the conclusion that Metoprolol's impact on cardiac remodeling was short-lived. At three months following PCI, Xu et al (17) discovered that the metoprolol group's The left ventricular end diastolic volume (LVEDV), left ventricular end systolic volumes (LVESV), & left ventricular end ejection fraction (LVEF) were all significantly higher than those of the control group for six months after PCI.Thus, they came to the conclusion that metoprolols impact on cardiac remodeling was short-lived. Consequently, additional analysis was done on the variations in heart rate. The Metoprolol group's heart rate variations at one week, one month, three months, and one year following PCI were noticeably greater than those of the control group. Rezq et al (18) reported that using Metoprolol in conjunction with bisoprolol was linked to improved resting heart rate

management and a decreased likelihood of hospitalization for heart failure or unstable angina.

BNP has a significant correlation with the development of heart failure in individuals with AMI and is a valid predictor of the outcome of myocardial infarction. (19) According to this study, the Metoprolol group's BNP following PCI was less than that of the control groups, and on the second and third days after PCI, the variance was statistically significant. This suggests that Metoprolol may help prevent early heart failure following myocardial infarction, most likely by slowing the heart rate more quickly and improving cardiac function. Following proper management, elevated troponin levels might lessen heart ischemia, reduction troponin issue, and enhance patients' long-term diagnosis. Metoprolol has been demonstrated in studies to increase coronary artery reserve, which lowers the area of myocardial infarction and improves myocardial blood flow. This impact is separate from its effect on lowering heart rate. (20)

Conclusion

The current study concluded that in individuals with ST-Segment Elevation Myocardial Infarction, early metoprolol administration before Primary Percutaneous Coronary Intervention (PCI) in ST-Segment Elevation Myocardial Infarction increase left ventricular ejection, can improve heart rate regulation, lessen myocardial damage, and enhance cardiac function and lower the risk of serious adverse cardiac events.

Declarations

Data Availability statement

All data generated or analysed during the study are included in the manuscript.

Ethics approval and consent to participate

Approved by the department concerned. (IRBEC-24)

Consent for publication

Approved

Funding

Not applicable

Conflict of interest

The authors declared the absence of a conflict of interest.

Author Contribution

MA (PGR)
Manuscript drafting, Study Design,
TN (Assistant Professor)
Review of Literature, Data entry, Data analysis, and drafting articles.
SH (PGR)
Conception of Study, Development of Research Methodology Design,
SMNA (PGR)
Study Design, manuscript review, critical input.
FSH (PGR)
Manuscript drafting, Study Design,

All authors reviewed the results and approved the final version of the manuscript. They are also accountable for the integrity of the study.

References

1. Jalbani FA, Shaikh S, Fatima S. Frequency and determinants of timely arrival among patients of acute myocardial infarction at a public sector tertiary care hospital in Karachi. Pakistan Journal of Medical Sciences. 2020;36(5):914.2. Mongkhon P, Dilokthornsakul P, Tepwang K, Tapanya K, Sopitprasan C, Chaliawsin P, et al. The effects of fibrinolytic before referring STEMI patients: A systematic review and meta-analysis. IJC Heart & Vasculature. 2017;15:9-14.

3. Puymirat E, Simon T, Cayla G, Cottin Y, Elbaz M, Coste P, et al. Acute myocardial infarction: changes in patient characteristics, management, and 6-month outcomes over a period of 20 years in the FAST-MI program (French Registry of Acute ST-Elevation or Non-ST-Elevation Myocardial Infarction) 1995 to 2015. Circulation. 2017;136(20):1908-19.

4. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). European heart journal. 2018;39(2):119-77.

5. Motawea KR, Gaber H, Singh RB, Swed S, Elshenawy S, Talat NE, et al. Effect of early metoprolol before PCI in ST-segment elevation myocardial infarction on infarct size and left ventricular ejection fraction. A systematic review and meta-analysis of clinical trials. Clinical Cardiology. 2022;45(10):1011-28.

6. García-Prieto J, Villena-Gutiérrez R, Gómez M, Bernardo E, Pun-García A, García-Lunar I, et al. Neutrophil stunning by metoprolol reduces infarct size. Nature communications. 2017;8(1):14780.

7. Ibanez B, Macaya C, Sánchez-Brunete V, Pizarro G, Fernández-Friera L, Mateos A, et al. Effect of early metoprolol on infarct size in ST-segment–elevation myocardial infarction patients undergoing primary percutaneous coronary intervention: the effect of metoprolol in cardioprotection during an acute myocardial infarction (METOCARD-CNIC) trial. Circulation. 2013;128(14):1495-503.

8. Mateos A, García-Lunar I, García-Ruiz JM, Pizarro G, Fernández-Jiménez R, Huertas P, et al. Efficacy and safety of out-of-hospital intravenous metoprolol administration in anterior ST-segment elevation acute myocardial infarction: insights from the METOCARD-CNIC trial. Annals of Emergency Medicine. 2015;65(3):318-24.

9. Pizarro G, Fernández-Friera L, Fuster V, Fernández-Jiménez R, García-Ruiz JM, García-Álvarez A, et al. Long-term benefit of early pre-reperfusion metoprolol administration in patients with acute myocardial infarction: results from the METOCARD-CNIC trial (Effect of Metoprolol in Cardioprotection During an Acute Myocardial Infarction). Journal of the American College of Cardiology. 2014;63(22):2356-62.

10. Group MTR. Metoprolol in acute myocardial infarction (MIAMI). A randomised placebo-controlled international trial. European Heart Journal. 1985;6(3):199-226.

11. Members ATF, Steg PG, James SK, Atar D, Badano LP, Lundqvist CB, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC). European heart journal. 2012;33(20):2569-619.

12. Hearse D, Yellon D, Downey J. Can beta blockers limit myocardial infarct size? European heart journal. 1986;7(11):925-30.

13. Ibanez B, Prat-González S, Speidl WS, Vilahur G, Pinero A, Cimmino G, et al. Early metoprolol administration before coronary reperfusion results in increased myocardial salvage: analysis of ischemic myocardium at risk using cardiac magnetic resonance. Circulation. 2007;115(23):2909-16.

14. Larose E, Rodés-Cabau J, Pibarot P, Rinfret S, Proulx G, Nguyen CM, et al. Predicting late myocardial recovery and outcomes in the early hours of ST-segment elevation myocardial infarction: traditional measures compared with microvascular obstruction, salvaged myocardium, and necrosis characteristics by cardiovascular magnetic resonance. Journal of the American College of Cardiology. 2010;55(22):2459-69.

15. Gerbaud E, Montaudon M, Chasseriaud W, Gilbert S, Cochet H, Pucheu Y, et al. Effect of ivabradine on left ventricular remodelling after reperfused myocardial infarction: a pilot study. Archives of Cardiovascular Diseases. 2014;107(1):33-41.
16. Barillà F, Pannarale G, Torromeo C, Paravati V, Acconcia MC, Tanzilli G, et al. Ivabradine in patients with ST-elevation myocardial infarction complicated by cardiogenic shock: a preliminary randomized prospective study. Clinical drug investigation. 2016;36:849-56.
17. Xu Y, Zhang W, Zhong X, Yan S, Chen H, Guo R, et al. Effect of early use of ivabradine on left ventricular remodeling after primary percutaneous coronary intervention in patients with acute ST-segment elevation myocardial infarction: A pilot test. Annals of Noninvasive Electrocardiology. 2021;26(2):e12816.
18. Rezq A, Saad M, Al Mahmoudy A, El Nozahi M. Value of Ivabradine in patients with anterior ST-Elevation myocardial infarction: the VIVA-STEMI study. Cardiology and Cardiovascular Medicine. 2020;4(6):630-9.
19. Oleynikov V, Dushina E, Golubeva A, Barmenkova JA. Early predictors of heart failure progression in patients after myocardial infarction. Kardiologiia. 2020;60(11):84–93.
20. Heusch G, Skyschally A, Gres P, van Caster P, Schilawa D, Schulz R. Improvement of regional myocardial blood flow and function and reduction of infarct size with ivabradine: protection beyond heart rate reduction. European heart journal. 2008;29(18):2265-75.