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The Impact of Eptifibatide on Midterm Outcomes in Patients Undergoing Primary Percutaneous Coronary Intervention: A Retrospective Cohort Study

Running Title: Eptifibatide in Primary Percutaneous Coronary Intervention

Abbas Andishmand[1], Seyed Ali Banifatemeh[1], Hamid Alian[2]*,

[1] Department of Cardiology, School of Medicine Afshar Heart Center- Shahid Sadoughi University of Medical Sciences, Yazd, Iran.
[2] Non-Communicable Diseases Research Center, Rafsanjan University of Medical Sciences, Rafsanjan, Iran.

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***Corresponding author**
Department of Cardiology, School of Medicine Afshar Heart Center- Shahid Sadoughi University of Medical Sciences, Yazd, Iran

Tel: +98-9131569821

E-mail
Hamid66480@gmail.com

ORCID ID
0009-0007-3209-6245

Abstract

Background: Primary percutaneous coronary intervention (PCI) is the standard reperfusion strategy for ST-elevation myocardial infarction (STEMI). Eptifibatide, a glycoprotein IIb/IIIa inhibitor, is used during primary PCI to reduce ischemic complications. However, its clinical benefit remains uncertain. This study aimed to compare the rates of cardiac death, recurrent myocardial infarction (MI), or target vessel revascularization (TVR) at 12 months between patients who received eptifibatide and those who did not.

Methods: A retrospective cohort study was performed on 268 STEMI patients who underwent primary PCI. Patients were divided into two groups: those who received eptifibatide (n=134) and those who did not (n=134). The primary endpoint was a composite of cardiac death, recurrent myocardial infarction, and TVR at 12 months. Secondary endpoints included the individual components of the primary endpoint and in-hospital major bleeding.

Results: Patients who received eptifibatide had a lower rate of major adverse cardiovascular events (MACEs) at 12 months than those who did not (11.2% vs 26.9%, p=0.001). Eptifibatide use was associated with lower rates of death (3% vs 11.2%, p=0.008), while rates of TVR (3.7% vs 8.2%, p=0.098) and recurrent MI (4.5% vs 7.5%, p=0.220) did not differ significantly between groups. The incidence of in-hospital major bleeding was not statistically significant (10.6% vs 4.5%, p=0.156). Comparison between groups showed significantly lower odds of cardiac death and of the composite major adverse cardiovascular events in patients who received eptifibatide (OR: 0.24, 95% CI: 0.08-0.76, p=0.015; and OR: 0.34, 95% CI: 0.18-0.66, p=0.001, respectively).

Conclusion: In STEMI patients undergoing primary PCI, administration of eptifibatide was associated with lower rates of death and MACE at 12 months without an increase in in-hospital major bleeding. Use of eptifibatide during primary PCI may improve mid-term outcomes.

Keywords: Eptifibatide, Primary PCI, STEMI, Cardiac death, Bleeding, TVR, Recurrent MI, MACE

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Introduction

ST-elevation myocardial infarction (STEMI) is a life-threatening condition requiring immediate reperfusion. Primary percutaneous coronary intervention (PCI) is the gold standard for reperfusion when feasible (1). Nevertheless, even after prompt reperfusion, patients can experience ischemic complications from factors such as coronary thrombus, distal embolization, and reperfusion injury. To reduce these risks, adjunctive pharmacotherapies are often used during primary PCI to improve myocardial reperfusion (2).

Eptifibatide, a reversible glycoprotein IIb/IIIa inhibitor, effectively blocks platelet aggregation. Its benefit in improving ischemic outcomes during primary PCI for STEMI has been demonstrated in numerous randomized controlled trials and meta-analyses (3, 4, 5, and 6). Nevertheless, routine use of glycoprotein IIb/IIIa inhibitors remains controversial because of potential increased bleeding risk (7). Observational data from real-world settings can provide valuable insights to complement randomized evidence.

This study aimed to assess 12-month outcomes of death, recurrent myocardial infarction, or unplanned repeat revascularization in patients with STEMI who received eptifibatide during primary PCI, compared with those who did not.

Methods

This study used a retrospective cohort design to assess the effect of eptifibatide on clinical outcomes in patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI) at Afshar Hospital, Yazd, Iran, from 2014 to 2020.

Study Population and Matching

Patients diagnosed with STEMI by ECG and cardiac biomarkers were included. We categorized them into two groups: those receiving eptifibatide during PCI (eptifibatide group) and those not receiving it (control group). To mitigate baseline imbalances and enhance internal validity, stratified randomization and caliper matching on key demographic and clinical variables were implemented to achieve a 1:1 ratio between the groups.

Inclusion and Exclusion Criteria

Patients aged ≥18 years presenting with prolonged chest pain within 12 hours of symptom onset were eligible. Exclusion criteria included prior coronary revascularization (PCI or CABG), presentation >12 hours after symptom onset, cardiogenic shock requiring mechanical support, failed thrombolysis requiring rescue or facilitated PCI, failed PCI or stent implantation, significant comorbidities limiting life expectancy (<1 year), or death during initial hospitalization.

Percutaneous Coronary Intervention (PCI) Protocol

PCI procedures adhered to established guidelines for STEMI management. Upon identification and evaluation of patients with suggestive symptoms (chest pain, ECG changes), a thorough clinical assessment (medical history review, physical examination, and relevant lab tests) was conducted. Confirmation of STEMI prompted activation of the primary PCI team (interventional cardiologists, nurses, and support personnel). The cardiac catheterization laboratory was prepared, and pre-

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procedural medications (antiplatelet agents – aspirin 325mg, clopidogrel 600mg – and anticoagulants – unfractionated heparin 70 U/kg) were administered.

Coronary angiography was performed under local anesthesia via femoral access to identify the culprit lesions causing STEMI. Primary PCI was then carried out to restore blood flow in the affected artery.

In the eptifibatide group, the drug was given intravenously as a bolus (weight-based initial loading dose – two 180 mcg/kg doses) followed by a maintenance infusion for 12-18 hours. Dosing adjustments were made according to individual clinical status and comorbidities. Thrombus aspiration might have been used at the operator's discretion.

Post-PCI, patients were monitored in the cardiac care unit and received guideline-based medications (dual antiplatelet therapy, beta-blockers, statins, ACE inhibitors/ARBs). Vital signs, ECG, cardiac enzymes, and potential complications (bleeding, reperfusion injury) were close monitored.

Discharge Medications and Follow-up

Upon discharge, all patients received standardized medication regimens including aspirin (80 mg daily), clopidogrel (75 mg daily), and atorvastatin (40 mg daily). Additional medications (beta-blockers, ACE inhibitors/ARBs, diuretics) were prescribed based on individual needs and physician judgment. Pre-discharge echocardiography assessed left ventricular systolic function and potential complications.

A dedicated educational team (nutritionist, psychologist, and rehabilitation specialist) provided secondary prevention recommendations to patients and families, encouraging participation in cardiac rehabilitation programs and attendance follow-up appointments. Information on patient follow-up was obtained from the rehabilitation unit and follow-up clinic records at Afshar Hospital. Patients who missed appointments were contacted by phone to assess their health status and identify any cardiac events.

Data Collection and Outcomes

Medical records were reviewed to collect relevant data. The midterm outcome was a composite of all-cause death, recurrent myocardial infarction, or target vessel revascularization (TVR) within 12 months. Secondary outcomes included individual components of the primary outcome and major bleeding events (defined as bleeding requiring blood transfusion).

Statistical Analysis

Data analysis was performed using IBM SPSS Statistics 26 software (IBM Corp., Armonk, NY, USA). Quantitative data are presented as mean ± standard deviation (SD), while qualitative data are expressed as frequencies and percentages.

Two-tailed independent samples t-tests were used to compare quantitative variables between groups. For qualitative variables, Chi-square and Mann-Whitney U tests were employed to assess associations with group membership.

Logistic regression analyses were conducted to evaluate the association between treatment and clinical outcomes. This approach provided odds ratios (ORs) with 95% confidence intervals (CIs) for adverse events, allowing a comparison of event likelihood between treated and untreated groups.

Survival analysis was performed using Kaplan-Meier curves and the log-rank test to assess time-to-event

data, specifically overall survival, between groups. A p-value < 0.05 was considered statistically significant. All tests were two-sided, with $\alpha = 0.05$.

Results

The study enrolled 268 patients, who were evenly divided into two groups. One group received eptifibatide (n=134), while the other group did not (n=134). The two groups were well-matched concerning their baseline characteristics **Table 1.**

Table 1. Baseline Characteristics of Patients with STEMI: Eptifibatide treated vs untreated Groups

Variable	Treated group (N=134) (n(%)/(mean±SD))	Untreated group (N=134) (n(%)/(mean±SD))	P-value
Age(yr)	59.0±11.7	58.0±11.0	0.452
sex **Male** **Female**	 104(77.6) 30(22.4)	 96(71.6) 38(28.4)	 0.163
CAD risfactor **HTN** **DM** **HLP** **Current tobacco Smoker**	 55(47.0) 33(28.2) 39(33.3) 34(29.1)	 53(45.7) 43(37.1) 37(31.9) 36(31.0)	 0.472 0.228 0.463 0.426
History of IHD	16(13.7)	10(8.6)	0.186
Comorbidity	1(2)	1(2)	0.748
BMI (kg/m^2)	26.7±3.9	27.0±4.6	0.620
Drug history **ASA** **Clopidogrel** **BB** **CCB** **Statin** **ACEI** **ARB** **Diuretic**	 24(20.5) 2(1.7) 10(8.5) 4(3.4) 20(17.1) 4(3.4) 18(15.5) 2(1.7)	 23(19.8) 3(1.7) 11(9.5) 7(6) 22(19) 9(7.8) 18(15.4) 3(2.6)	 0.513 0.686 0.492 0.265 0.420 0.123 0.561 0.496
Lab tests **Hb** **Cr**	 14.4(1.88) 1.18(0.25)	 14.8(1.74) 1.2(0.22)	 0.072 0.496
BS **Platelet count** **HDL-c** **LDL-c** **Uric acid**	179.6(84.7) 217.4(52.8) 32.5(8.1) 120.2(33.4) 4.8(1.8)	172.5(86.9) 217.8(52.4) 32.9(8.5) 121.6(35.8) 4.6(1.3)	0.497 0.950 0.693 0.741 0.298
MI territory **Anterior** **Non anterior**	 79(59.0) 55(41.0)	 70(52.6) 63(47.4)	 0.180
Door to balloon time (min)	53.7(28.6)	54.1(24.2)	0.946
Angiography result **One vessel** **Two vessels** **Three vessels** **LM disease**	 47(35.1) 39(29.1) 48(35.8) 4(3.4)	 59(44.0) 42(31.3) 33(24.6) 5(4.3)	 0.118
Culprit vessel **LAD** **LCX** **RCA** **LM** **Other branches**	 78(59.5) 13(13.0) 38(29.0) 1(0.8) 4(2.9)	 66(50.4) 19(14.5) 45(34.4) 0(0.0) 5(3.7)	 0.335
Syntax score **Residual syntax score**	17.4(8.3) 5.5(8.2)	15.5(8.2) 4.7(7.8)	0.181 0.592
LVEF(%)	41.9±7.8	41.7±7.0	0.844
Stent size (mm) **Length** **Diameter**	 26.3±11.5 3.0±0.6	 24.6±8.8 3.0±0.4	0.196 0.557
Post-procedure TIMI flow grade 3	116(86.8)	118(88.4)	0.553
In hospital Stay(day)	4.9±2.3	3.6±1.9	0.0001

STEMI: ST Elevation Myocardial Infarction, CAD: Coronary Artery Disease, HTN: Hypertension, DM: Diabetes Mellitus, HLP: Hyperlipidemia, PCI: Percutaneous Coronary Intervention, IHD: Ischemic Heart Disease, ASA: Aspirin, BB: Beta-Blocker, CCB: Calcium Channel Blocker, ACEI: Angiotensin-

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Converting Enzyme Inhibitor, ARB: Angiotensin II Receptor Blocker, Hb: Hemoglobin, Cr: Creatinine, BS: Blood Sugar, LAD: Left Anterior Descending Artery, LCX: Left Circumflex Artery, RCA: Right Coronary Artery, LM: Left Main Coronary Artery, LVEF: Left Ventricular Ejection Fraction.

Demographics and Clinical Presentation:

The mean age in the treated group was 59.0 ± 11.7 years, compared to 58.0 ± 11.0 years in the untreated group (p = 0.452). The majority of patients were male, accounting for 77.6% in the treated group and 71.6% in the untreated group (p = 0.163). There were no significant differences in the prevalence of hypertension (47.0% vs. 45.7%, p = 0.472), diabetes mellitus (28.2% vs. 37.1%, p = 0.228), or hyperlipidemia (33.3% vs. 31.9%, p = 0.463) between the groups. The proportion of current tobacco smokers was similar between the treated (29.1%) and untreated (31.0%) groups (p = 0.426).

Medical History and Laboratory Findings

Previous history of ischemic heart disease (IHD) was present in 13.7% of the treated group and 8.6% of the untreated group (p = 0.186). There were no significant differences in the use of medications such as aspirin, clopidogrel, beta-blockers, calcium channel blockers, statins, ACE inhibitors, ARBs, and diuretics between the two groups (all p > 0.05).

Laboratory values, including hemoglobin, creatinine, blood sugar, platelet count, HDL, LDL, and uric acid levels, showed no significant differences between the groups (all p > 0.05).

Angiographic and Procedural Characteristics

The location of myocardial infarction (MI) was predominantly anterior in both groups (59.0% in the treated group vs. 52.6% in the untreated group, p = 0.180). Door-to-balloon times were comparable between the treated (53.7 ± 28.6 minutes) and untreated (54.1 ± 24.2 minutes) groups (p = 0.946).

Angiographic results showed that the proportions of one, two, and three-vessel disease were similar between the two groups (p = 0.118). The culprit vessel was most frequently the left anterior descending artery (LAD) in both groups (59.5% in the treated group vs. 50.4% in the untreated group, p = 0.335).

The mean Syntax score, indicating the complexity of coronary artery disease, was 17.4 ± 8.3 in the treated group and 15.5 ± 8.2 in the untreated group (p = 0.181). The residual Syntax score post-PCI was also comparable (5.5 ± 8.2 vs. 4.7 ± 7.8, p = 0.592). Left ventricular ejection fraction (LVEF) was similar between the two groups (41.9 ± 7.8% in the treated group vs. 41.7 ± 7.0% in the untreated group, p = 0.844).

The average stent length was 26.3 ± 11.5 mm in the treated group and 24.6 ± 8.8 mm in the untreated group (p = 0.196), with a similar stent diameter (3.0 ± 0.6 mm vs. 3.0 ± 0.4 mm, p = 0.557). Post-procedure TIMI flow grade 3 was achieved in 86.8% of the treated group and 88.4% of the untreated group (p = 0.553). The in-hospital stay was significantly longer in the treated group (4.9 ± 2.3 days) than in the untreated group (3.6 ± 1.9 days, p = 0.0001).

Clinical Outcomes

Mortality and Major Adverse Cardiovascular Events (MACE):

MAC is a composite endpoint frequently used in cardiovascular research; classical 3-point MACE is

defined as a composite of cardiac death, recurrent MI, and TVR.

The incidence of cardiac death was significantly lower in the treated group compared to the untreated group (3.0% vs. 11.2%, p = 0.008). Although the rates of recurrent myocardial infarction (MI) (4.5% vs. 7.5%, p = 0.220) and target vessel revascularization (TVR) (3.7% vs. 8.2%, p = 0.098) were lower in the treated group, these differences were not statistically significant.

The composite measure of major adverse cardiovascular events (MACE), which includes cardiac death, recurrent MI, and TVR, was observed in 11.2% of the treated group compared to 26.9% of the untreated group and was statistically significant (p = 0.001) **Table 2.**

Table 2. Comparison of 12-month outcomes of patients who received Eptifibatide and those who did not.

Event	Treated group (n=134) (n, %)	Untreated group (n=134) (n, %)	P value
Cardiac Death	4(3)	15(11.2)	0.008
Recurrent MI	6(4.5)	10(7.5)	0.220
TVR	5(3.7)	11(8.2)	0.098
Composite (MACE)	15(11.2)	36(26.9)	0.001
Major bleeding	7(10.6)	3(4.5)	0.156

TVR: target vessel revascularization, MACE: major adverse cardiac events

Logistic Regression Analysis of Clinical Outcomes

The analysis showed a significant reduction in the odds of cardiac death and MACE with eptifibatide treatment, while other outcomes did not reach statistical significance **Table 3.**

Table 3. Odds Ratios of Major Cardiac Events in Patients Treated with Eptifibatide Versus Untreated Patients

Event	B coefficient	SE	Odds ratio	95% CI	P value
Cardiac Death	-1.410	**0.577**	0.24	0.08-0.76	0.015
Recurrent MI	-0.543	0.532	0.58	0.21-1.6	0.307
TVR	-0.836	0.554	0.43	0.15-1.3	0.131
Composite (MACE)	-1.070	0.336	0.34	0.18-0.66	0.001
Major bleeding	0.929	0.713	2.5	0.63-10.3	0.193

TVR: target vessel revascularization, MACE: major adverse cardiac events

Bleeding Complications

The incidence of in-hospital major bleeding was higher in the treated group (10.7%) than in the untreated group (4.5%), though the difference did not reach statistical significance (p = 0.156) (Fig 1).

Logistic Regression Analysis of Clinical Outcomes:

The analysis showed a significant reduction in the odds of cardiac death and MACE with eptifibatide treatment, while other outcomes did not reach statistical significance. (Table 3).

Survival Analysis:

Kaplan-Meier survival analysis revealed a significant improvement in overall survival for patients in the eptifibatide-treated group compared to the untreated group (log-rank test, p = 0.009) **Figure 1.**

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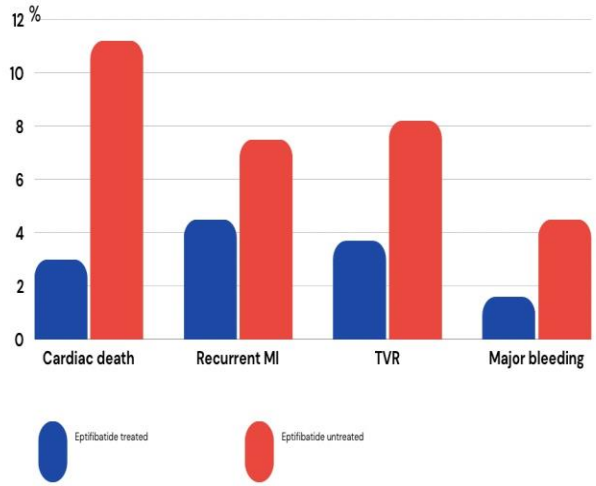

Figure 1. Comparison of major adverse cardiac events (MACE) and major bleeding between patients treated with eptifibatide (blue group) and those who did not (red group). TVR: target vessel revascularization

Survival Analysis

Kaplan-Meier survival analysis revealed a significant improvement in overall survival for patients in the eptifibatide-treated group compared to the untreated group (log-rank test, p = 0.009) Figure 2.

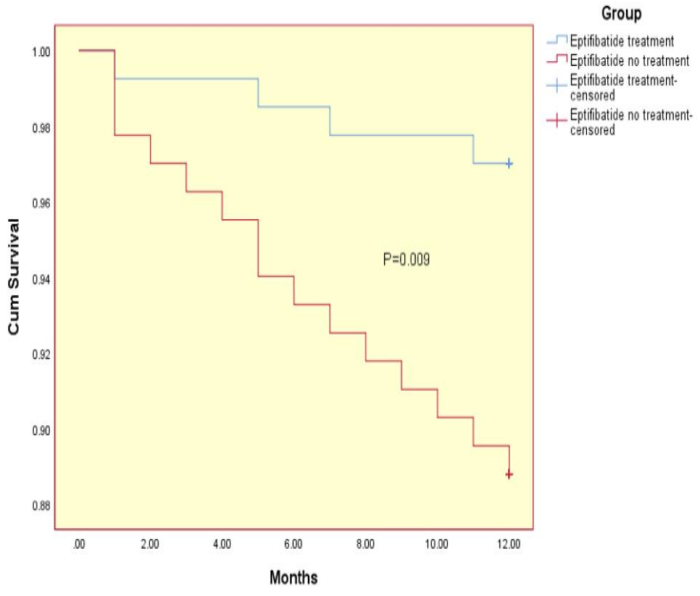

Figure 2. Kaplan-Meier survival curve showing the probability of 12-month survival for two groups: those who received eptifibatide (blue) and those who did not (red).

Discussion

The introduction of potent oral antiplatelet agents such as ticagrelor and prasugrel has revolutionized treatment for STEMI patients. These newer medications offer improved efficacy and convenience compared to injectable options like eptifibatide, leading to a significant decline in their use.

Our current investigation focuses on a period preceding the widespread adoption of these powerful oral medications. Back then, interventional cardiologists frequently relied on eptifibatide to ensure adequate platelet inhibition during PCI, despite ongoing debates regarding its long-term benefits. This study aimed to evaluate the mid-term efficacy and safety of eptifibatide within this specific context.

Our findings demonstrated that patients who received intracoronary eptifibatide followed by an intravenous infusion after primary PCI experienced a significant reduction in major adverse cardiac events (MACE) at 12-month follow-up compared with the control group. Notably, mortality rates were significantly lower in the eptifibatide group, suggesting a potential long-term survival benefit. This finding contrasts with the study by Jalalian et al., which found no significant difference in clinical outcomes between patients receiving intravenous eptifibatide and those receiving heparin alone during primary PCI (9).

While the eptifibatide group exhibited lower rates of target vessel revascularization and reinfarction, these differences were not statistically significant. This suggests that eptifibatide may targets and reduces the

occurrence of fatal events, with less impact on non-fatal ones.

On the other hand, the optimal protocol for eptifibatide administration remains a topic of ongoing debate. Our institution employed a regimen consisting of a bolus injection followed by a minimum 12-hour infusion. Several studies have investigated the efficacy of different protocols. Fischer et al. demonstrated that bolus-only administration was noninferior to bolus-plus-infusion in reducing infarct size (10). Also, a meta-analysis revealed no significant difference in long-term MACE between intravenous and intracoronary eptifibatide administration. However, intracoronary administration yielded superior results for MACE occurrence and TIMI III flow recovery up to 3 months post-infarction (11). A systematic review reported that bolus-only administration significantly reduced heart failure risk within one month compared to bolus-plus-infusion (12). Nevertheless, Sanati et al. did not observe a difference in MACE at 1 month across three protocols (13). Hasan et al. suggested that intracoronary administration might be more effective and safer in the short and medium-term (14). Our study observed more frequent major bleeding events in the eptifibatide group; however, the difference was not statistically significant and did not translate to increased mortality. These findings align with the observations reported by Mousavi et al. (15). Conversely, Kassaian et al. reported a higher bleeding risk associated with eptifibatide infusion compared to bolus-only administration after PCI (16).

The current guidelines advocate for a limited role of GP IIb/IIIa inhibitors alongside potent oral antiplatelet medications for patients with ACS. Their administration is typically reserved for specific scenarios, such as the no-reflow phenomenon, acute ischemic events, or situations where oral antiplatelet therapy is contraindicated (17).

Conclusion

Given limited access to newer oral medications, our study suggests that eptifibatide may offer a mid-term mortality benefit for patients with STEMI undergoing primary PCI. However, the risk of bleeding remains a concern, and the optimal administration protocol necessitates further investigation. In the present era, GP IIb/IIIa inhibitors play a more restricted role in managing patients with ACS, with newer oral medications taking center stage.

Study limitations

Our study possesses inherent limitations associated with its retrospective cohort design. Reliance on existing medical records raises concerns about incomplete or inaccurate data. Additionally, the absence of randomization and control over treatment allocation introduces selection bias and potential confounding variables. This limits our ability to definitively establish a causal relationship between eptifibatide use and patient outcomes.

Furthermore, the single-center design limits the generalizability of the findings. Variations in patient populations, treatment protocols, and clinical practices across different institutions can affect the study's external validity.

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Ethical considerations:

The study protocol was approved by the Ethics Committee of Azad Medical School of Yazd. Informed consent was obtained from all individual participants included in the study. Throughout the study, ethical principles and regulations were rigorously followed in accordance with the Declaration of Helsinki.

Data Availability Statement

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

Authors' contribution: Abbas Andishmand contributed to the conceptualization, design, and data analysis. Hamid Alian contributed to the investigation and drafted the manuscript. Both authors reviewed and edited the manuscript.

Conflict of interest: The authors declare that there are no conflicts of interest.

References

1. Li K, Zhang B, Zheng B, Zhang Y, Huo Y. Reperfusion Strategy of ST-Elevation Myocardial Infarction: A Meta-Analysis of Primary Percutaneous Coronary Intervention and Pharmaco-Invasive Therapy. Front Cardiovasc Med. 2022 Mar 17;9:813325. doi: 10.3389/fcvm.2022.813325. PMID: 35369319; PMCID: PMC8970601.
2. Levine GN, Bates ER, Blankenship JC, Bailey SR, Bittl JA, Cercek B, Chambers CE, Ellis SG, Guyton RA, Hollenberg SM, Khot UN, Lange RA, Mauri L, Mehran R, Moussa ID, Mukherjee D, Ting HH, O'Gara PT, Kushner FG, Ascheim DD, Brindis RG, Casey DE Jr, Chung MK, de Lemos JA, Diercks DB, Fang JC, Franklin BA, Granger CB, Krumholz HM, Linderbaum JA, Morrow DA, Newby LK, Ornato JP, Ou N, Radford MJ, Tamis-Holland JE, Tommaso CL, Tracy CM, Woo YJ, Zhao DX. 2015 ACC/AHA/SCAI Focused Update on Primary Percutaneous Coronary Intervention for Patients With ST-Elevation Myocardial Infarction: An Update of the 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention and the 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction. J Am Coll Cardiol. 2016 Mar 15;67(10):1235-1250. doi: 10.1016/j.jacc.2015.10.005. Epub 2015 Oct 21. Erratum in: J Am Coll Cardiol. 2016 Mar 29;67(12):1506. PMID: 26498666.
3. Montalescot G, Andersen HR, Antoniucci D, Betriu A, de Boer MJ, Grip L, Neumann FJ, Rothman MT. Recommendations on percutaneous coronary intervention for the reperfusion of acute ST-elevation myocardial infarction. Heart. 2004 Jun;90(6):e37. PMID: 15145901; PMCID: PMC1768296.
4. Shah I, Khan SO, Malhotra S, Fischell T. Eptifibatide: The evidence for its role in the management of acute coronary syndromes. Core Evid. 2010 Jun 15;4:49-65. doi: 10.2147/ce.s6008. Erratum in: Core Evid. 2014;9:49. PMID: 20694065; PMCID: PMC2899786.
5. Tonin G, Klein J. Eptifibatide, an Older Therapeutic Peptide with New Indications: From Clinical Pharmacology to Everyday Clinical Practice. Int J Mol Sci. 2023 Mar 13;24(6):5446. doi: 10.3390/ijms24065446. PMID: 36982519; PMCID: PMC10049647.
6. Zeymer U. The role of eptifibatide in patients undergoing percutaneous coronary intervention. Expert Opin Pharmacother. 2007 Jun;8(8):1147-54. doi: 10.1517/14656566.8.8.1147. PMID: 17516878.
7. Hanna EB, Rao SV, Manoukian SV, Saucedo JF. The evolving role of glycoprotein IIb/IIIa inhibitors in the setting of percutaneous coronary intervention strategies to minimize bleeding risk and optimize outcomes. JACC Cardiovasc Interv. 2010 Dec;3(12):1209-19. doi: 10.1016/j.jcin.2010.09.015. PMID: 21232714.
8. Tao L, Ren S, Zhang L, Liu W, Zhao Y, Chen C, Mao X, Chen Z, Gu X. A Review of the Role of the Antiplatelet Drug Ticagrelor in the Management of Acute Coronary Syndrome, Acute Thrombotic Disease, and Other Diseases. Med Sci Monit. 2022 May 16;28:e935664. doi: 10.12659/MSM.935664. PMID: 35570383; PMCID: PMC9121774.
9. Jalalian R, Golshani S, Farsavian H, Vatani M, Farsavian AA. Efficacy of intravenous eptifibatide in primary percutaneous coronary intervention patients. J Med Life. 2021 May-Jun;14(3):376-382. doi: 10.25122/jml-2021-0035. PMID: 34377204; PMCID: PMC8321601

10. Fischer F, Buxy S, Kurz DJ, Eberli FR, Senn O, Zbinden R, Held U, Meyer MR. Efficacy and Safety of Abbreviated Eptifibatide Treatment in Patients With ST-Segment Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention. Am J Cardiol. 2021 Jan 15;139:15-21. doi 10.1016/j.amjcard.2020.09.054. Epub 2020 Oct 13. PMID: 33065082.
11. Hahn J, Jeon J, Geum MJ, Lee HW, Shin J, Chung WY, Yu YM, Ah YM. Intracoronary versus intravenous glycoprotein IIb/IIIa inhibitors during primary percutaneous coronary intervention in patients with STEMI: a systematic review and meta-analysis. Thromb J. 2023 Jul 14;21(1):76. doi: 10.1186/s12959-023-00519-x. PMID: 37452333; PMCID: PMC10347711.
12. Elbadawi A, Elgendy IY, Megaly M, Ha LD, Mahmoud K, Alotaki E, Ogunbayo GO, Baig B, Abuzaid AS, Saad M, Depta JP. Meta-Analysis of Randomized Trials of Intracoronary Versus Intravenous Glycoprotein IIb/IIIa Inhibitors in Patients With ST-Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention. Am J Cardiol. 2017 Oct 1;120(7):1055-1061. doi: 10.1016/j.amjcard.2017.06.040. Epub 2017 Jul 21. PMID: 28826897.
13. Sanati HR, Zahedmehr A, Firouzi A, Farrashi M, Amin K, Peighambari MM, Shakerian F, Kiani R. Intracoronary versus Intravenous eptifibatide during percutaneous coronary intervention for acute ST-segment elevation myocardial infarction; a randomized controlled trial. Cardiovasc
14. Hassan W, Al-Sergani H, Al Buraiki J, Dunn B, Al Turki F, Akhras N, Elshaer F, Nawaz M, Kharabsheh S, ElKum N. Immediate and intermediate results of intracoronary stand-alone bolus administration of eptifibatide during coronary intervention (ICE) study. Am Heart J. 2007 Aug;154(2):345-51. doi: 10.1016/j.ahj.2007.04.020. PMID: 17643587.
15. Mousavi M, Sehati F, Tayebi A, Nayeri AD, Movahed MR. A Double-Blinded Randomized Controlled Trial Comparing Eptifibatide Bolus Only Versus Bolus Plus Infusion In Patients Undergoing Primary Percutaneous Coronary Intervention For ST-Elevation Myocardial Infarction. Cardiovasc Revasc Med. 2023 Jun;51:1-7. doi 10.1016/j.carrev.2023.01.023. Epub 2023 Jan 31. PMID: 36737382.
16. Kassaian SE, Fathi Y, Lotfi-Tokaldany M, Salarifar M, Alidoosti M, Haji-Zeinali AM, Aghajani H, Amirzadegan A, Nozari Y, Mortazavi SH, Jalali A, Saroukhani S. Comparison of 1-year Major Adverse Cardiac Events in Patients Undergoing Primary Percutaneous Coronary Intervention Receiving Intracoronary Bolus Only Versus Intracoronary Bolus Plus Infusion of Glycoprotein IIb/IIIa Inhibitors. Crit Pathw Cardiol. 2016 Sep;15(3):89-94. doi: 10.1097/HPC.0000000000000079. PMID: 27465002.
17. Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, Claeys MJ, Dan GA, Dweck MR, Galbraith M, Gilard M, Hinterbuchner L, Jankowska EA, Jüni P, Kimura T, Kunadian V, Leosdottir M, Lorusso R, Pedretti RFE, Rigopoulos AG, Rubini Gimenez M, Thiele H, Vranckx P, Wassmann S, Wenger NK, Ibanez B; ESC Scientific Document Group. 2023 ESC Guidelines for the management of acute coronary syndromes. Eur Heart J. 2023 Oct 12;44(38):3720-3826. doi: 10.1093/eurheartj/ehad191. PMID: 37622654..

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