BMC Cardiovascular Disorders

RESEARCH

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Safety and efficacy of post-procedure anticoagulation in ST-elevation myocardial infarction complicated by cardiogenic shock undergoing primary percutaneous coronary intervention

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Abstract

Introduction Cardiogenic shock (CS) is a lethal complication of ST-elevation myocardial infarction (STEMI). The impact of post-procedure anticoagulants (PPAC) in STEMI-CS patients undergoing primary percutaneous coronary intervention (PPCI) remains unknown.

Method In the Improving Care for Cardiovascular Disease in China-Acute Coronary Syndrome registry (2014–2019), STEMI patients with CS on admission undergoing PPCI were stratified into two groups based on the use of PPAC or not. The primary outcome was all-cause mortality during hospitalization. Other outcomes including major bleeding were also investigated.

Results Of 36,873 patients with STEMI, 855 eligible patients with CS undergoing PPCI were included in our study, among which 614 patients were treated by PPAC and 241 were not. Adjusted by multi-variable Cox regression, PPAC was associated with a lower risk of in-hospital all-cause mortality (14.9% vs. 30.3%; adjusted HR: 0.60; 95% CI: 0.37 to 0.97; $p=0.037$), while a non-significant difference in major bleeding (4.6% vs. 7.0%; adjusted HR: 1.05; 95% CI: 0.36 to 3.05; $p=0.925$) was observed between PPAC and non-PPAC. Consistent results were observed in the sensitivity analyses adjusted by propensity score matching and inverse probability of treatment weighting.

Conclusion Our study suggested the use of PPAC in STEMI-CS patients undergoing PPCI was associated with a lower risk of in-hospital all-cause mortality without increasing the risk of major bleeding.

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Trial registration ClinicalTrials.gov, NCT02306616. Registered 29 November 2014.

Keywords Cardiogenic shock, Primary percutaneous coronary intervention, Anticoagulation, ST-elevation myocardial infarction

Introduction

Cardiogenic shock (CS) is complicated in nearly 5% of patients with ST-elevated myocardial infarction (STEMI) and is the leading cause of in-hospital mortality in these patients [1, 2]. Treatment is limited to immediate revascularization, especially primary percutaneous coronary intervention (PPCI), to restore blood flow to the culprit coronary artery and improve clinical prognosis in patients with CS secondary to STEMI (STEMI-CS) [3, 4]. However, mortality in STEMI-CS after PPCI remains high, with hospital mortality between 30% and 50% [5].

Anticoagulant therapy during PCI is mandatory for STEMI [1]. However, data regarding the safety and efficacy of post-procedure anticoagulants (PPAC) after PPCI is limited [6, 7]. The latest guideline from the European Society of Cardiology suggested that continuation of anticoagulant is not recommended except in specific clinical scenarios like left ventricular thrombus formation or atrial fibrillation requiring anticoagulation [1]. However, this recommendation was mainly based on expert consensus rather than adequate supporting evidence. Recent findings from a multicenter, double-blinded randomized clinical trial (RCT) suggested that the use of enoxaparin after primary PCI might be associated with a lower risk of a composite event of death, myocardial infarction, stroke, stent thrombosis, or urgent revascularization at 30 days [8]. However, the effect of PPAC is unknown in STEMI-CS patients, as these patients were excluded from all relevant studies.

The CCC-ACS project (Improving Care for Cardiovascular Disease in China–Acute Coronary Syndrome) is a nationwide, multicenter registry for acute coronary syndrome in China. Data from this project was used to examine the impact of PPAC on in-hospital outcomes in STEMI-CS patients with PPCI.

Method

Study population

The CCC-ACS project is a joint program between the American Heart Association and the Chinese Society of Cardiology, to enhance the quality of care provided for acute coronary syndrome (ACS) patients. Data was collected from 150 tertiary and 42 secondary hospitals in China between November 2014 and July 2019. Details regarding the study design and rationale have been published previously [9]. This project was approved by the institutional review board of Beijing Anzhen Hospital and registered at ClinicalTrial.gov (unique identifier: NCT02306616).

Among a total of 113,650 patients enrolled in the CCC-ACS registry, those with STEMI-CS on admission and confirmed by in-patient diagnosis were included. Patients were excluded from analysis if they did not undergo PPCI, had indications for anticoagulation (including atrial fibrillation, left ventricular thrombus, or heart valve surgery), or had a bleeding complication including platelet count $< 50 \times 10^9$/L on admission.

Definitions and study variables

An electronic data capture platform was used to collect information on patients from medical records by abstractors. Patient demographics, medical history, procedural details, and anticoagulants were obtained. All collected variables were based on standardized definitions. STEMI was defined by the Chinese Society of Cardiology guidelines for the diagnosis and extracted from patients' medical records [10]. CS was defined as the status of patients on admission confirmed by in-patient diagnosis with systolic blood pressure < 90 mmHg for > 30 min or the need for supportive management to maintain systolic blood pressure > 90 mmHg, an arterial lactate level ≥ 3 mmol per liter, and clinical signs of perfusion impairment with at least one of the following criteria: altered mental status, cold extremities or decreased urine output. PPCI was defined as the performance of emergency PCI on the infarct-related artery in STEMI-CS patients using a balloon, stent, or other approved devices after the onset of the symptom. Patients included in the final analysis were divided into two groups according to the use of PPAC or not. PPAC was defined as patients receiving anticoagulants including unfractionated heparin, low-molecular-weight heparin, or fondaparinux within 24 h after PCI. Those who did not receive any anticoagulant after PCI were defined as "non-PPAC".

In-hospital outcomes

The primary outcome was all-cause mortality during hospitalization. Other outcomes were major bleeding, major adverse cardiovascular event (MACE), and net adverse cardiovascular event (NACE). Major bleeding was defined as Bleeding Academic Research Consortium (BARC) type 3 or 5 bleeding during hospitalization [11]. MACE was defined as a composite outcome of all-cause mortality, cardiac arrest, reinfarction or in-stent thrombosis, or stroke during hospitalization. NACE was defined as a composite of all-cause mortality, cardiac arrest, reinfarction or in-stent thrombosis, stroke, or major bleeding during hospitalization. In-stent

Table 1 Baseline information of study population

v	Before PSM			After PSM		
Baseline characteristic	**PPAC (*n* = 614)**	**Non-PPAC (*n* = 241)**	***P* value**	**PPAC (*n* = 229)**	**Non-PPAC (*n* = 229)**	***P* value**
Age, mean ± SD, years	64.5 ± 11.9	65.1 ± 12.2	0.536	65.9 (12.3)	64.9 (12.1)	0.395
Male sex, n (%)	445 (72.5)	176 (73.0)	0.870	161 (70.3)	166 (72.4)	0.605
Body mass index, kg/m^2	23.8 ± 3.5	23.7 ± 4.0	0.483	23.7 (3.5)	23.7 (4.0)	0.875
eGFR, mL/min/1.73m^2, n (%)			0.157			0.550
eGFR < 30	90 (14.7)	48 (20.0)		36 (15.7)	44 (19.2)	
eGFR 30–60	388 (63.2)	140 (58.1)		146 (63.8)	136 (59.4)	
eGFR > 60	136 (22.2)	53 (22.0)		47 (20.5)	49 (21.4)	
LVEF	50.7 ± 0.5	48.2 ± 1.0	0.024	48.8 ± 0.8	47.9 ± 0.9	0.423
Medical history, n (%)						
Myocardial infarction	25 (4.1)	9 (3.7)	0.820	6 (0.3)	8 (0.3)	0.587
Prior PCI	30 (4.9)	12 (5.0)	0.955	10 (4.3)	11 (4.8)	0.823
Diabetes	141 (23.0)	59 (24.5)	0.637	62 (27.1)	56 (24.5)	0.521
Hypertension	288 (46.9)	90 (37.3)	0.011	80 (34.9)	88 (38.4)	0.438
Heart failure	7 (1.1)	3 (1.2)	0.898	3 (1.3)	3 (1.3)	1.000
Ischemic stroke	64 (10.4)	13 (5.4)	0.021	10 (4.4)	13 (5.0)	0.521
Procedural characteristics, n (%)						
Prior-PCI fibrinolysis	21 (3.4)	8 (3.3)	0.815	13 (5.7)	8 (3.5)	0.264
PCI < 12 h from Onset	508 (82.7)	200 (83.0)	0.930	190 (83.0)	185 (80.8)	0.143
PCI ≥ 12 h from Onset	106 (17.3)	41 (17.0)	0.930	39 (17.0)	44 (19.2)	0.143
Radial Access	475 (77.4)	183 (75.9)	0.410	176 (76.9)	168 (73.4)	0.569
IABP	54 (8.8)	27 (11.2)	0.457	29 (12.6)	24 (10.4)	0.465
Angiographic Characteristics, n (%)						
Culprit artery			0.040			0.592
LM	41 (6.7)	27 (11.2)		26 (11.4)	20 (8.7)	
LAD	284 (46.3)	113 (46.9)		98 (48.0)	109 (48.9)	
RCA	241 (39.3)	77 (32.0)		84 (36.7)	76 (70.0)	
LCx	36 (5.7)	14 (6.5)		15 (6.6)	14 (6.1)	
Not identified	13 (2.1)	10 (4.1)		6 (2.6)	10 (4.3)	
Multivessel Disease	195 (31.8)	92 (38.2)	0.074	94 (41.0)	84 (36.7)	0.338
DAPT status in the first 24 h of medical contact, n (%)						
Non-DAPT	15 (2.4)	32 (13.3)	< 0.001	219 (95.6)	216 (94.3)	0.521
DAPT	599 (97.6)	209 (86.7)		10 (4.4)	13 (5.7)	
DAPT using ticagrelor as $P2Y_{12}$ inhibitor	301 (49.0)	106 (44.0)	0.907	113 (49.3)	113 (49.3)	1.000
Anticoagulation Therapy following PCI, n (%)						
Unfractionated Heparin	45 (7.3)	-		7 (3.1)	-	
LMWH	550 (89.6)	-		204 (89.1)	-	
Other Agents	19 (3.1)	-		18 (7.8)	-	

SD = standard deviation; PCI = percutaneous coronary intervention; PSM = propensity-score matching; PPAC = post-procedural anticoagulant; eGFR = estimated glomerular filtration rate; LVEF = left ventricular ejection fraction; IABP = intra-aortic balloon pump; LM = Left main coronary artery; LAD = left anterior descending artery; LCx = left circumflex coronary artery; RCA = right coronary artery; DAPT = dual antiplatelet therapy; LMWH = low-molecular-weight heparin

thrombosis was defined as an acute or subacute occlusion of the stent after PCI [12].

Statistical analysis

Continues variables were presented as the mean ± standard deviation or median with the 25th and 75th percentile and were compared between groups using 2-sample t-tests or rank sum test. Categorical variables were described as counts and percentages and compared using Pearson's chi-squared test. Kaplan-Meier curves were plotted for PPAC or non-PPAC groups. The hazard ratios (HR) and 95% confidence intervals (CI) were calculated with Cox regression. Considering that differences in baseline characteristics might potentially confound outcomes, we estimated the impact of PPAC on outcomes through multivariable-adjusted Cox regression models that included variables with a p-value < 0.05 in univariate Cox regression or with potential clinical significance, encompassing age, sex, body mass index (BMI), previous disease history (diabetes, hypertension,

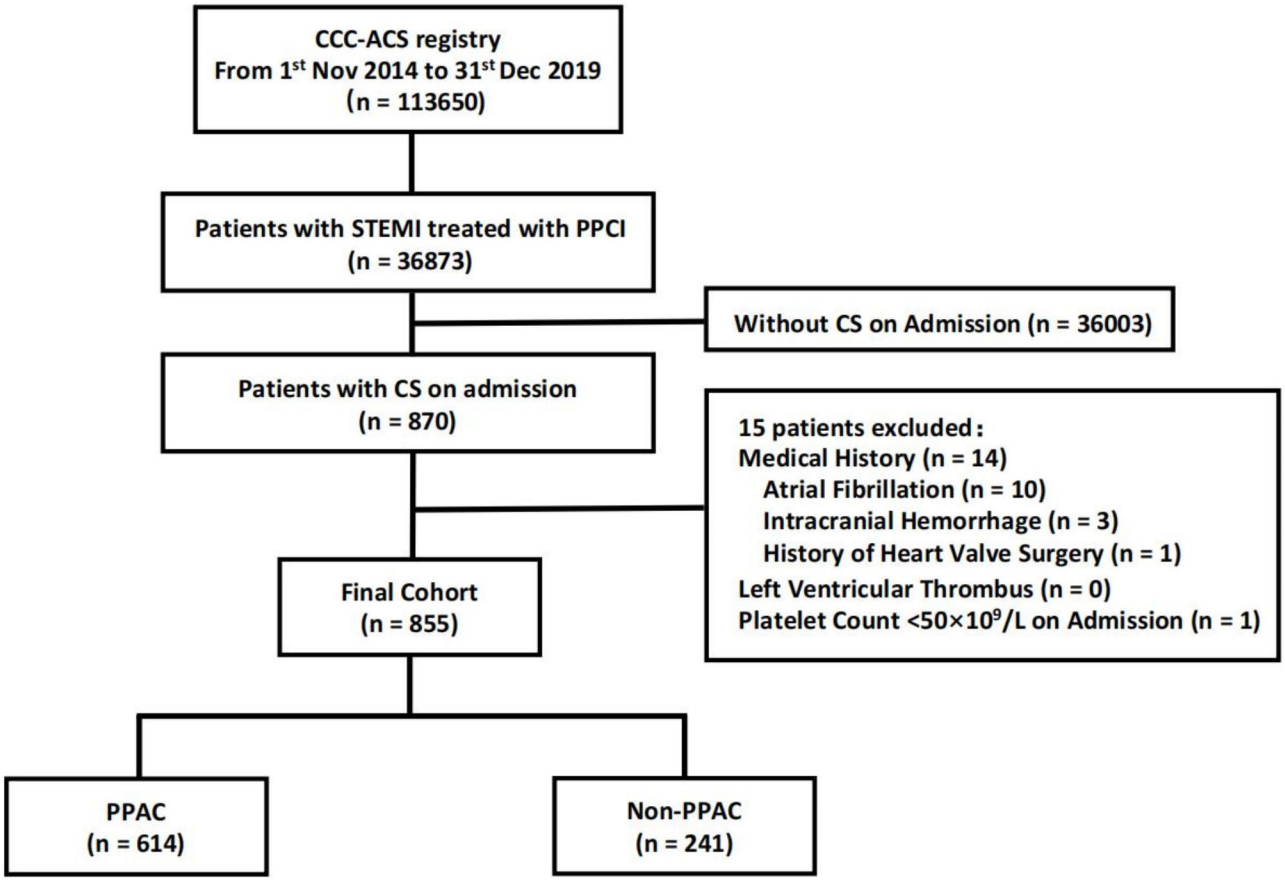

Fig. 1 Study flow chart
The study population was derived from the nationwide, multicenter, prospective CCC-ACS (Improving Care for Cardiovascular Disease in China–Acute Coronary Syndrome) registry. STEMI = ST-segment Elevation Myocardial Infarction; CS = Cardiogenic shock; PPCI = primary percutaneous coronary intervention; PPAC = post-procedural anticoagulant

ischemic stroke, myocardial infarction, dyslipidemia, heart failure, prior PCI), estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73m2, Left Ventricular Ejection Fraction (LVEF) ≤ 40%, dual antiplatelet status, and angiographic characteristics including left main artery or left anterior descending artery as a culprit vessel, and multivessel disease. Propensity score calculations were the following variables: age, sex, BMI, previous disease history (diabetes, hypertension, ischemic stroke, myocardial infarction, dyslipidemia, heart failure, prior PCI), eGFR < 60 mL/min/1.73m^2, LVEF ≤ 40%, dual antiplatelet status, and angiographic characteristics including left main artery or left anterior descending artery as a culprit vessel, and multivessel disease. A caliper of 0.05 for the propensity-score matching (PSM) was used. Univariate Cox analysis was used to evaluate the treatment effects with the adjustment via PSM or the inverse probability of treatment weighting (IPTW) using the propensity score mentioned above. Subgroup analyses were implemented to support the credibility of the results.

Results

Baseline characteristics

From November 1, 2014, to December 31, 2019, of 36,873 patients with STEMI undergoing PPCI enrolled in the CCC-ACS registry, 855 eligible patients with STEMI-CS on admission were included in the final analysis as shown in Fig. 1.

The baseline characteristics of participants divided into two groups are shown in Table 1. Patients with PPAC were more likely to have a medical history of hypertension and ischemic stroke, had a higher LVEF, and a higher rate of dual antiplatelet therapy, but were less likely to have left main disease. The incidence of different mechanical complications in our population is presented in Supplementary Table S2. After adjustment using PSM methods, the baseline characteristics were well balanced in Table 1.

Primary and other outcomes

The overall all-cause mortality of included patients was 16.3% during hospitalization. After the adjustment of

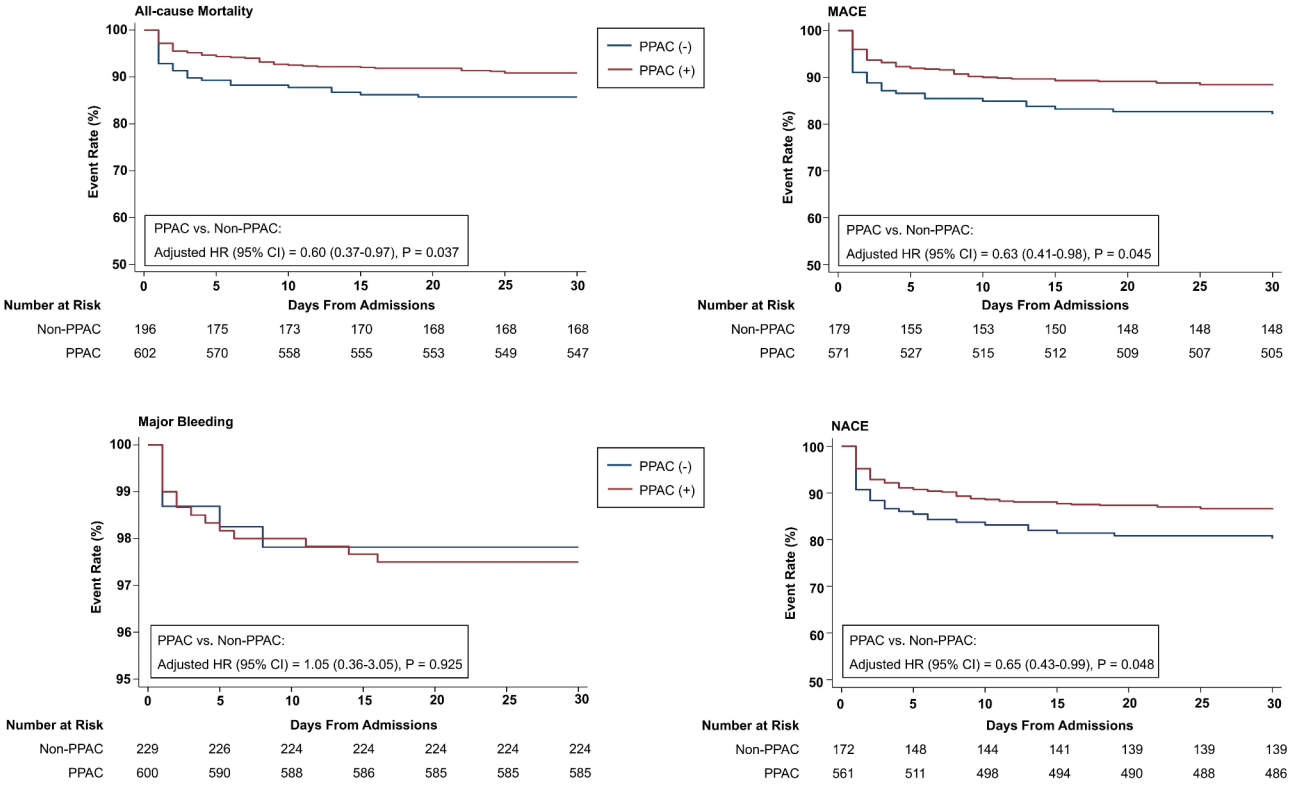

Fig. 2 Cumulative Kaplan-Meier curves of all-cause mortality, major adverse cardiovascular event (MACE), net adverse cardiovascular event (NACE), and major bleeding
HR = hazard ratio; CI = confidence interval; PPAC = post-procedural anticoagulant

multi-variable Cox regression, PPAC was associated with a lower risk of all-cause mortality (14.9% vs. 30.3%; adjusted HR: 0.60; 95% CI: 0.37 to 0.97; $p = 0.037$), while the non-significant difference in major bleeding (4.6% vs. 7.0%; adjusted HR: 1.05; 95% CI: 0.36–3.05; $p = 0.925$) was observed between PPAC and non-PPAC as shown in Fig. 2; Table 2. PPAC was associated with lower risks of MACE (17.9% vs. 39%; HR: 0.63; 95% CI: 0.41 to 0.98; $p = 0.045$) and NACE (21.0% vs. 42.7%; HR: 0.65; 95% CI: 0.43 to 0.99; $p = 0.048$).

Sensitivity analyses using PSM and IPTW adjustment consistently showed that PPAC was associated with lower risks of in-hospital all-cause mortality, MACE, and NACE, without significantly increasing the risk of bleeding as shown in Table 2. The balance between groups after PSM or IPTW adjustment was evaluated by standardized mean differences as shown in Fig. 3 and Table s1 in the Supplemental Material. Results showed a successful balance between groups.

Subgroup analysis

The prognostic impact of PPAC on all-cause mortality among the various subgroups is shown in Fig. 4. The results showed that except for sex, hypertension, and types of anticoagulants, there were no significant interactions with baseline variables. STEMI-CS patients undergoing PPCI who were male, lacked a history of hypertension, and received low-molecular-weight heparin as PPAC agents seemed to benefit more from PPAC (p for interaction < 0.05).

Discussion

Using data from a nationwide, multicenter, prospective registry, we accessed the effect of PPAC on in-hospital outcomes in STEMI-CS patients with PPCI. The main finding was that: without significantly increasing the major bleeding risk, PPAC was associated with a lower risk of mortality, MACE, and NACE during hospitalization in STEMI-CS patients treated with PPCI.

The latest European Society of Cardiology guidelines did not support the routine use of PPAC except for specific indications for anticoagulation therapy, whereas guidelines for heart associations from America and Asian countries including China made no mention of the preference for the anticoagulants after PCI [1, 13]. In real-world practice, the use of PPAC for patients with STEMI remains geographically different. A post hoc analysis of two RCTs showed that 16.6% of patients received PPAC in the USA and 49.8% in Europe countries [7]. Our results showed that anticoagulants were used in 71.8% of STEMI-CS patients undergoing PPCI. Controversy

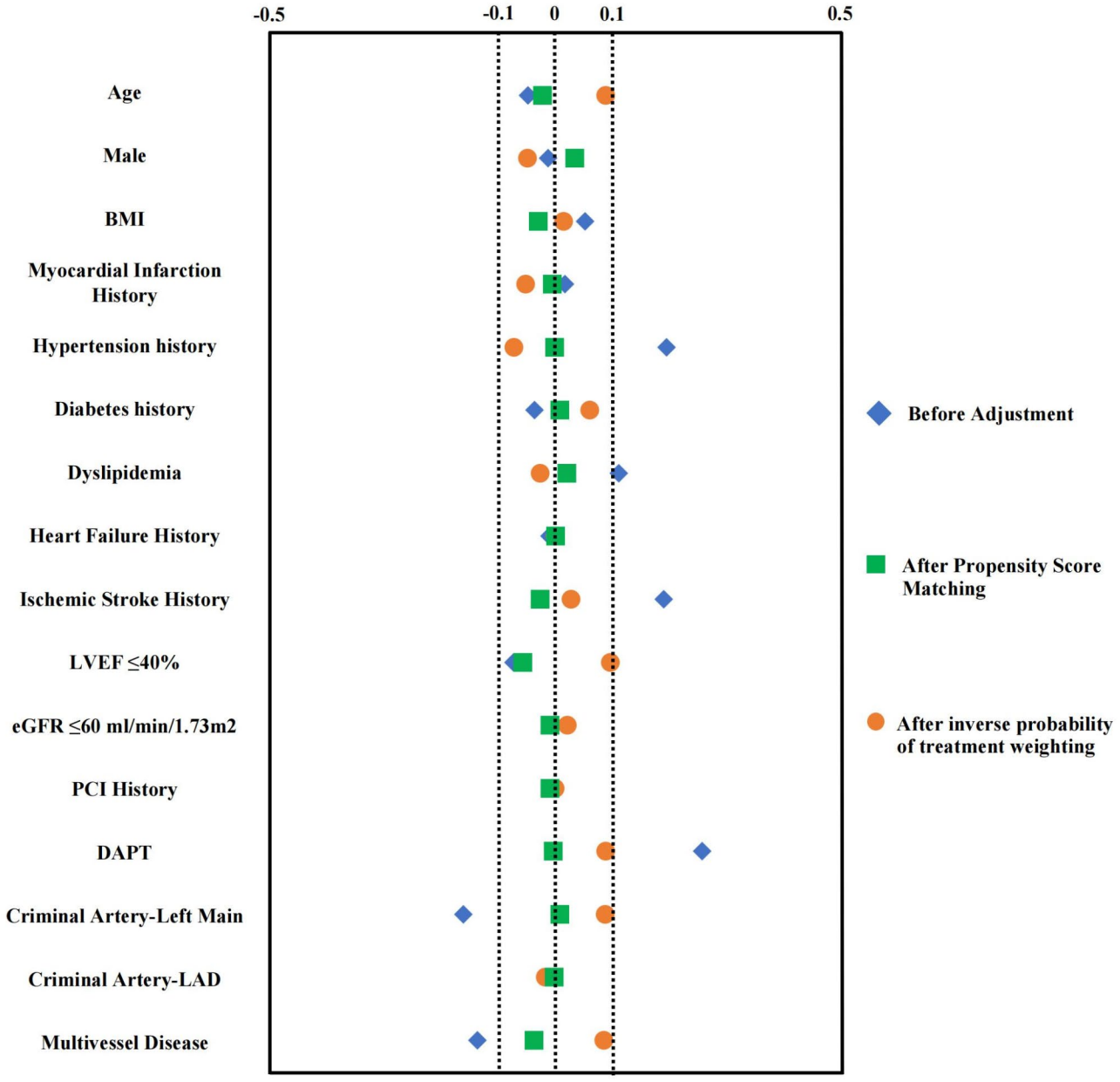

Fig. 3 Standardized mean differences for adjusted variables before and after adjustment of propensity score matching and inverse probability of treatment weighting in patients with STEMI-CS undergoing PPCI

The X-axis represents the standardized mean differences value, and the Y-axis represents baseline adjusted variables. STEMI = ST-segment Elevation Myocardial Infarction; CS = Cardiogenic shock; PPCI = primary percutaneous coronary intervention; BMI = body mass index; LVEF = left ventricular ejection fraction; PCI = primary percutaneous coronary intervention; eGFR = estimated glomerular filtration rate; DAPT = dual antiplatelet therapy; LAD = left anterior descending artery

remains relating to the effect of PPAC on the prognosis of STEMI patients.

The effect of PPAC on STEMI patients remains unclear, especially for those with CS. A post hoc analysis showed that a reduction of adverse ischemic events in the PPAC group after PCI was not observed [6]. A similar conclusion was drawn from a pooled analysis of two RCTs that revealed no statistically significant difference in 30-day mortality between patients with or without PPAC following PCI [7]. The Comparison of Anticoagulation Prolongation vs. no Anticoagulation in ST-segment Elevation Myocardial Infarction (STEMI) Patients After PPCI (RIGHT) trial is by far the only RCT in this field [8]. Prespecified subgroup analysis demonstrated a reduction in the death or ischemic events with enoxaparin compared with placebo. Of note, all those trials excluded patients

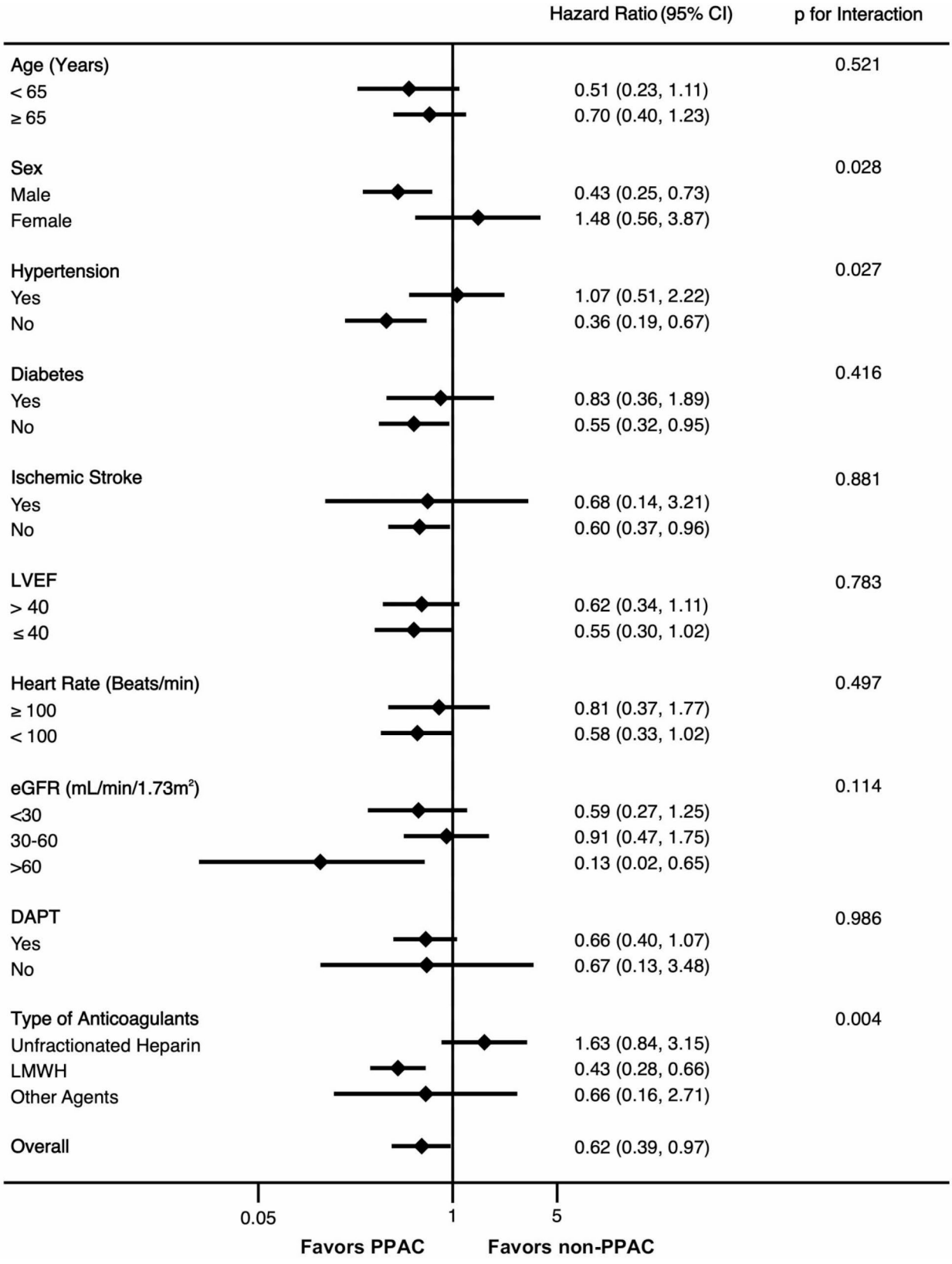

Fig. 4 Subgroup analysis for all-cause mortality during hospitalization

LVEF = left ventricular ejection fraction; eGFR = estimated glomerular filtration rate; DAPT = dual antiplatelet therapy; LMWH = low-molecular-weight heparin; PPAC = post-procedural anticoagulant

Table 2 Comparison of in-hospital clinical outcomes according to treatment strategy

			Unadjusted		Multivariable-adjusted		Propensity-Score Matched		IPTW-Adjusted	
	PPAC (*n* = 614)	Non-PPAC (*n* = 241)	HR (95% CI)	*P* value	HR (95% CI)	*P* value	HR (95% CI)	*P* value	HR (95% CI)	*P* value
All-cause mortality	67 (14.9)	73 (30.3)	0.62 (0.39–0.97)	0.038	0.60 (0.37–0.97)	0.037	0.57 (0.33–0.99)	0.046	0.55 (0.31–0.95)	0.034
MACE	110 (17.9)	94 (39.0)	0.63 (0.42–0.96)	0.033	0.63 (0.41–0.98)	0.045	0.56 (0.32–0.97)	0.039	0.57 (0.32–0.94)	0.028
Major bleeding	28 (4.6)	17 (7.0)	1.14 (0.42–3.15)	0.793	1.05 (0.36–3.05)	0.925	1.95 (0.49–7.84)	0.341	1.86 (0.45–7.48)	0.296
NACE	129 (21.0)	103 (42.7)	0.66 (0.44–0.99)	0.045	0.65 (0.43–0.99)	0.048	0.61 (0.37–0.99)	0.048	0.59 (0.36–0.96)	0.032

Multivariable Cox analysis adjusted by age, male sex, diabetes history, hypertension history, ischemic stroke history, heart failure history, dyslipidemia history, previous history of percutaneous coronary intervention, eGFR, LVEF, dual antiplatelet status, angiographic characteristics including left main artery or left anterior descending artery as a culprit vessel, and multivessel disease. Propensity score calculations were the following variables: age, male sex, diabetes history, hypertension history, ischemic stroke history, heart failure history, dyslipidemia history, previous history of percutaneous coronary intervention, eGFR, LVEF, dual antiplatelet status and angiographic characteristics including left main artery or left anterior descending artery as a culprit vessel, and multivessel disease. Univariate Cox analysis was used in an inverse probability of treatment weighting sample using the above propensity score. Univariate Cox analysis was used to evaluate the treatment effects with the adjustment via propensity score matching or the inverse probability of treatment weighting using the propensity score mentioned above. MACE = major adverse cardiovascular event; NACE = net adverse cardiovascular event. HR = hazard ratio; CI = confidence interval; PPAC = post-procedural anticoagulant; IPTW = inverse probability of treatment weighting

with CS, therefore, the impact of PPAC after PCI on the prognosis of STEMI-CS remained unknown. In our study, PPAC after PPCI was associated with a lower mortality risk in patients with STEMI-CS. This effect remained consistent across most subgroups. Limited by an observational nature, our data might be influenced by potential confounders and should be interpreted carefully. Further clinical trials will provide a solid answer to this question.

Underlying mechanism for benefits of anticoagulant in patients with STEMI-CS

Underlying pathophysiological mechanism that relates STEMI-CS to poor prognosis include microcirculatory dysfunction, systemic hypoperfusion, inflammation, and multi-organ failure [14]. Coronary microcirculatory dysfunction is probably one of the most relevant factors in the development of multi-organ failure and is associated with a poor prognosis in STEMI-CS patients [15]. The presence of thrombotic aggregation in microcirculation plays an important role in the no-reflow phenomenon, highlighting the potential benefit of antithrombotic agents including anticoagulants in these settings [16, 17]. In addition, changes in blood flow to tissues and dysfunction of organs, such as acute hepatic and kidney injury, might cause unpredictable pharmacokinetic and pharmacodynamic changes, leading to uncertain effects on STEMI-CS patients [18]. Patients in shock status may experience reduced effectiveness of oral antithrombotic drugs due to reduced blood flow and movement in the digestive system, delayed stomach emptying, or reduced absorption. Therefore, parenteral antithrombotic agents may be particularly relevant in critically ill patients [14].

Limitation

First, the main limitation of our study concerns about observational nature and the existence of potential unmeasured confounders. We attempted to minimize the bias from different baseline variables with sensitivity analysis by different adjustments. Second, during the enrollment of our registry, the Society for Cardiovascular Angiography and Interventions shock stage was not available and had not been adapted to differentiate CS patients with different stages [19]. However, in our study, more than 60% of the patients achieved improvement in blood pressure after initial therapy and were classified as stage C according to the Society for Cardiovascular Angiography and Intervention classification, which suggests a relatively lower severity of CS in this cohort. The 30-day mortality rate of 16.3% and the 9.5% usage rate of intra-aortic balloon pump align with reported outcomes for low to moderate-risk CS patients. Further studies are warranted to investigate the comparative impact of PPAC in more severe AMI-CS patients [20–24]. Third,

the CCC-ACS registry did not collect data on the usage of mechanical circulatory support, intubation, and exact dosage, duration, or specific indications for PPAC in treating STEMI-CS. To reduce the potential bias, we excluded patients with well-accepted indications including atrial fibrillation or left ventricular thrombus. Further studies are warranted to explore the optimal doses as well as duration to maximize the benefits of anticoagulants in STEMI-CS patients.

Conclusions

Results from our study support that the use of PPAC after PPCI was associated with a lower risk of in-hospital all-cause mortality without significantly increasing the major bleeding risk in patients with STEMI-CS. Large clinical trials are warranted to further testify these results.

Abbreviations

ACS	Acute coronary syndrome
BARC	Bleeding Academic Research Consortium
BMI	Body mass index
CCC-ACS	The Improving Care for Cardiovascular Disease in China-Acute Coronary Syndrome Project
CI	Confidence intervals
CS	Cardiogenic shock
eGFR	Estimated glomerular filtration rate
HR	Hazard ratios
IPTW	Inverse probability of treatment weighting
LVEF	Left ventricular ejection fraction
MACE	Major adverse cardiovascular events
NACE	Net adverse cardiovascular events
PPAC	Post-procedure anticoagulants
PPCI	Primary percutaneous coronary intervention
PSM	Propensity-score matched
RCT	Randomized clinical trials
RIGHT	Comparison of Anticoagulation Prolongation vs. no Anticoagulation in STEMI Patients After Primary PCI
STEMI	ST-segment elevation myocardial infarction

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-025-04639-2.

Supplementary Material 1

Acknowledgements

Not applicable.

Author contributions

C. Z., M. Z., and Z. Z. wrote the first draft of the manuscript; M. Z. and Z. Z. performed the statistical analysis; E. L., Y. Z., W. L., K. Z., Y. L., C. Y. wrote sections of the manuscript and contributed to manuscript revision; D. Z. performed the material preparation, data collection and takes responsibility for the data, X. Z. and H. G. conceived and designed the study; All authors read and approved the submitted version.

Funding

This work was financially supported by the grant from the National Key R&D Program of China (2022YFC3600201), Alar City of the First Division of the Corps Science and Technology Program Projects (2022YL16), Corps Science and Technology Program Projects (2023CB017-01), and Capital's Funds for Health Improvement and Research (CFH 2022-1-2062).

Data availability

Availability of data and materials: The data underlying this article cannot be publicly disclosed due to intellectual property rights and are available upon reasonable request to the corresponding author.

Declarations

Ethics approval and consent to participate

The study was approved by the Institutional Review Board of Beijing Anzhen Hospital, and was conducted according to the guidelines outlined in the Declaration of Helsinki. Informed consent was secured from all individual participants involved in the study.

Competing interests

The authors declare no competing interests.

Clinical trial number

ClinicalTrials.gov, NCT02306616. Registered 29 November 2014.

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Received: 18 November 2024 / Accepted: 7 March 2025
Published online: 28 March 2025

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