Original Article

Efficacy of Intra-aortic Balloon Pump before versus after Primary Percutaneous Coronary Intervention in Patients with Cardiogenic Shock from ST-elevation Myocardial Infarction

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Abstract

Background: Previous studies showed that patients with cardiogenic shock (CS) from ST-elevation acute myocardial infarction (STEMI) supported by intra-aortic balloon pump (IABP) before primary percutaneous coronary intervention (PCI) decreased the risk of in-hospital mortality than patients who received IABP after PCI. However, little evidence is available on the optimal order of IABP insertion and primary PCI. The aim of this study was to investigate the impact of the sequence of IABP support and PCI and its association with major adverse cardiac and cerebrovascular events (MACCEs).

Methods: Data were obtained from 218 consecutive patients with CS due to STEMI in Beijing Anzhen Hospital between 2008 and 2014, who were treated with IABP and PCI. The patients were divided into two groups: Group A in whom IABP received before PCI (n = 106) and Group B in whom IABP received after PCI (n = 112). We evaluated the myocardial perfusion using myocardial blush grade and resolution of ST-segment elevation. The primary endpoint was 12-month risk of MACCE.

Results: Most baseline characteristics were similar in patients between the two groups. However, patients received IABP before PCI were associated with a delay of door-to-balloon time (DBT) and higher troponin I level ($P < 0.05$). However, myocardial perfusion was significantly improved in patients treated with IABP before PCI ($P < 0.05$). Overall, IABP support before PCI was not associated with significantly lower risk of MACCE ($P > 0.05$). In addition, risk of all-cause mortality, bleeding, and acute kidney injury (AKI) was similar between two groups ($P > 0.05$). Multivariate analysis showed that DBT (odds ratio [*OR*] 2.5, 95% confidence interval [*CI*] 1.1–4.8, $P = 0.04$), IABP support after PCI (*OR* 5.7, 95% *CI* 2.7–8.4, $P = 0.01$), and AKI (*OR* 7.4, 95% *CI* 4.9–10.8, $P = 0.01$) were the independent predictors of mortality at 12-month follow-up.

Conclusions: Early IABP insertion before primary PCI is associated with improved myocardial perfusion although DBT increases. IABP support before PCI does not confer a 12-month clinical benefit when used for STEMI with CS.

Key words: Acute Myocardial Infarction; Cardiogenic Shock; Intra-aortic Balloon Counterpulsation; Mortality; Percutaneous Coronary Intervention

Introduction

Acute myocardial infarction (AMI) complicated by cardiogenic shock (CS) has a mortality of more than 50%.[1] Despite the use of early revascularization therapy, CS is the major cause of death in patients admitted with AMI.[2] Evidence for intra-aortic balloon pump (IABP) use in patients with AMI complicated by CS is largely based on pathophysiological considerations and nonrandomized, small studies of patients treated with thrombolytic therapy.[3,4] Although most recent large randomized trials, meta-analyses, and guidelines showed that IABP was not associated with survival benefit in patients with AMI complicated by CS,[5,6] IABP used in those studies was initiated before primary percutaneous coronary intervention (PCI). IABP therapy before PCI was associated with a delay of door-to-balloon

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time (DBT), which would increase the risk of myocardial injury and mortality.[7] Therefore, IABP support after primary PCI may have a better clinical survival benefit.

In this study, we sought to investigate the impact of sequence of IABP support and PCI and its association with major adverse cardiac and cerebrovascular events (MACCEs) and mortality in patients with ST-elevation acute myocardial infarction (STEMI) complicated by CS.

Methods

Study population

We retrospectively analyzed the data of 218 patients with CS complicating STEMI, who were treated with IABP and primary PCI at Beijing Anzhen Hospital from January 2008 to December 2014. Three patients were excluded owing to incomplete data. This was a retrospective study, so we did not have Ethics Committee approval or informed consent.

Patient management

In accordance with the STEMI guidelines, primary PCI was the standard treatment of STEMI. Immediately after the diagnosis of STEMI, patients received aspirin and clopidogrel (300–600 mg) loading dose before primary PCI. Use of periprocedural glycoprotein IIb/IIIa antagonists and thrombus aspiration devices was left to physician's discretion. After PCI, all patients took aspirin (100 mg/d) indefinitely. Meanwhile, clopidogrel (75 mg/d) was administered for at least 3 months when treated with bare metal stents and at least 12 months when treated with drug-eluting stents. Moreover, inotropic drugs used in our center were catecholamines (dobutamine, dopamine, and/or norepinephrine) and phosphodiesterase inhibitors (enoximone).

According to clinical considerations, the interventional cardiologists decided the timing of IABP insertion. In all patients, IABP was inserted in the catheterization laboratory and Arrow 8 French catheters (Arrow Corp., Reading, PA, USA) were used. According to the timing of initiation of IABP therapy, all patients were divided into two groups such as (1) Group A (n = 106): IABP support before PCI; (2) Group B (n = 112): IABP support after PCI.

Definitions

Diagnosis of STEMI in symptomatic patients was based on the electrocardiogram (ECG) criteria. The established criteria of myocardial infarction define STEMI as new ST-elevation at the J point in at least 2 contiguous leads of ≥2 mm (0.20 mV) in men or ≥1.5 mm (0.15 mV) in women in leads V2–V3 and/or of ≥1 mm (0.10 mV) in other contiguous chest leads or the limb leads. CS was defined by the attending operator as systolic blood pressure (SBP) persistently <90 mmHg or vasopressors required to maintain SBP >90 mmHg due to cardiac insufficiency with evidence of end-organ hypoperfusion (e.g., oliguria or cold/diaphoretic extremities or altered mental status), not responsive to fluid resuscitation.[8] DBT was defined as the interval between the time admitted to our hospital and first balloon dilatation of the culprit artery.

Myocardial reperfusion was evaluated by the myocardial blush grade (MBG) and resolution of ST-segment elevation (STR).[9,10] On the basis of the maximal densitometric degree of contrast opacification, myocardial perfusion was scored as MBG 0/1 (no or minimal myocardial contrast opacification), MBG 2 (moderate contrast opacification but less than in either an ipsilateral or contralateral noninfarct artery), and MBG 3 (normal myocardial blush or contrast opacification, comparable with the other coronary arteries). ECGs obtained pre-PCI and at 60-min post-PCI were analyzed. STR was evaluated using standardized techniques and divided into complete (>70%), partial (30–70%), or none (<30%).

According to the Kidney Disease Improving Global Outcomes (KDIGO) Work Group criteria,[11] acute kidney injury (AKI) is defined by either an increase of serum creatinine (sCr) or an episode of oliguria: increase of sCr 3 mg/L within 48 h, or increase of sCr by 1.5-fold above baseline, known or assumed to have occurred within 7 days.

Endpoints

Primary endpoint was the risk of MACCEs, which included cardiac mortality, myocardial reinfarction, revascularization, definite stent thrombosis, and stroke. Secondary endpoints were changes of cardiac biomarkers, myocardial perfusion, risk of AKI, and bleeding.

Data collection and follow-up

All 218 patients were followed-up for 12 months. Baseline characteristics including demographics, clinical presentation, procedural and postprocedural characteristics, and follow-up data were retrospectively reviewed and recorded in a dedicated database in our hospital. All data were checked for completeness and consistencies. In addition, hemodynamic data and specific IABP therapy-related data were collected by review of the electronic medical records.

Statistical analysis

All statistical analysis was performed with SPSS 23.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation (SD). Independent continuous variables were compared with two-tailed Student's t-tests. Categorical variables were expressed as frequency or ratio and compared with the Pearson Chi-squared statistic. Stepwise multivariate logistic regressions were used to analyze the predictors of MACCE. Kaplan-Meier survival curves were used to evaluate the cumulative all-cause mortality. A $P < 0.05$ was considered statistically significant.

Results

Baseline characteristics of patients

Two-hundred eighteen patients were recruited in this study. The characteristics of two groups are illustrated in Table 1. There were no significant differences between two groups in

Table 1: Characteristics of all patients enrolled in this study

Variables	IABP before PCI (n = 106)	IABP after PCI (n = 112)	P
Baseline characteristics			
Age (years)	63.1 ± 11.3	65.2 ± 11.2	0.58
Male	68 (64.2)	71 (63.4)	0.86
BMI (kg/m^2)	27.7 ± 0.3	28.3 ± 0.4	0.53
Cardiovascular risk factors			
Current smoking	47 (44.3)	53 (47.3)	0.76
Hypertension	39 (36.8)	43 (38.4)	0.81
Diabetes mellitus	32 (30.2)	34 (29.1)	0.87
Hypercholesterolemia	49 (46.2)	52 (46.4)	0.97
Prior MI	11 (10.4)	16 (14.3)	0.51
Prior PCI	5 (4.7)	7 (6.2)	0.13
Prior stroke	3 (2.8)	3 (2.6)	0.91
Hemodynamics			
HR (beats/min)	90.2 ± 1.3	92.3 ± 2.4	0.32
SBP (mmHg)	76.5 ± 16.7	75.7 ± 17.3	0.51
DBP (mmHg)	53.2 ± 14.3	53.7 ± 15.2	0.88
MBP (mmHg)	68.3 ± 15.5	69.1 ± 16.4	0.49
Cardiac biomarkers			
Troponin I (μg/L)	13.4 ± 5.7	11.3 ± 4.9	0.12
BNP (pg/ml)	312.6 ± 109.5	320.4 ± 98.3	0.23

Data are presented as n (%) or mean ± SD. IABP: Intra-aortic balloon pump; PCI: Percutaneous coronary intervention; BMI: Body mass index; MI: Myocardial infarction; HR: Heart rate; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MBP: Mean blood pressure; BNP: B-type natriuretic peptide; SD: Standard deviation.

age, body mass index, rate of smoking, diabetes, hypertension, hypercholesterolemia, blood pressure, and cardiac biomarkers. In addition, most procedural characteristics were similar between the two groups. However, DBT was significantly longer in patients received IABP before PCI ($P < 0.05$).

Cardiac biomarkers and myocardial perfusion

The procedural and postprocedural characteristics of two groups are illustrated in Table 2. After primary PCI, the peak troponin I level was higher in Group A ($P < 0.05$). However, B-type natriuretic peptide levels were similar between two groups. However, IABP therapy before PCI was associated with better myocardial perfusion, characterized by lower rate of MBG 0/1 and none STR (STR < 30%) ($P < 0.05$).

Major adverse cardiac and cerebrovascular events

MACCE occurred in 157 patients with AMI and CS (72.0%). There were no significant differences between the two groups in the risk of MACCE (70.7% vs. 73.2%, $P = 0.53$). Moreover, there were no significant differences in cardiac mortality, myocardial reinfarction, clinical-driven revascularization, definite stent thrombosis, and stroke between two groups. The outcomes are illustrated and compared in Table 3.

Acute kidney injury and bleeding

According to the KDIGO criteria, 40 patients had AKI (18.3%). In addition, Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries (GUSTO) bleeding events occurred in 50 patients (22.9%). There were no significant differences between two groups in AKI and GUSTO bleeding. Furthermore, risk of life-threatening or severe bleeding was also similar between two groups. The outcomes are illustrated and compared in Table 3.

Twelve-month all-cause mortality

Sixty-four patients died during follow-up (29.4%). Mean follow-up duration was 12.5 months and ranged from 1.0 to 14.0 months. Kaplan-Meier survival curves did not show significant difference in mortality between two groups ($P = 0.92$) [Figure 1]. After adjustment in multivariable analysis, DBT (odds ratio [*OR*] 2.5, 95% confidence interval [*CI*] 1.1–4.8, $P = 0.04$), IABP support after PCI (*OR* 5.7, 95% *CI* 2.7–8.4, $P = 0.01$), and AKI (*OR* 7.4, 95% *CI* 4.9–10.8, $P = 0.01$) were the independent predictors of mortality at 12 months.

Discussion

Our study demonstrated that IABP support before primary PCI was associated with longer DBT. However, myocardial perfusion was significantly improved in patients treated with IABP before PCI. In addition, risk of mortality and MACCE was not significantly different between two groups. After 12-month follow-up, DBT, IABP support after PCI, and AKI were the independent predictors of mortality. More studies are needed to confirm our findings.

Previous studies showed that IABP therapy before PCI was associated with survival benefit in patients with CS and AMI compared to postponing the treatment after PCI. IABP was introduced in 1968 for hemodynamic support of patients undergoing revascularization with coronary artery bypass surgery[12] and is still the most used method of left ventricular (LV) unloading and hemodynamic support in the catheterization laboratory. Concomitant acute circulatory support and LV unloading have been supported to provide superior infarct salvage and, therefore, improved long-term outcome over reperfusion alone. The landmark SHOCK trial, in which >86% of participants received IABP, demonstrated a mid- to long-term survival benefit for early revascularization versus medical therapy for AMI complicating CS.[6] *Post hoc* analysis of the parallel SHOCK trial registry also confirmed the benefit of IABP for reducing in-hospital mortality.[13,14] Moreover, Abdel-Wahab *et al*.[15] retrospectively analyzed 48 patients with CS complicating AMI, and found that patients treated by IABP before primary PCI had a significantly lower risk of MACCE ($P = 0.004$) and in-hospital mortality ($P = 0.007$). One recently published meta-analysis found that IABP was associated with reduced mortality in high-risk coronary artery bypass grafting patients Risk ratio (*RR*) 0.40, 95% *CI* 0.25–0.67).[16]

However, the Euro Heart Survey PCI Registry revealed that IABP was only used in 24.8% of patients with CS and AMI, and there was no hint of a survival beneficial effect of IABP therapy.[17] The IABP-SHOCK II trial showed that IABP combined with PCI therapy was not associated with 30-day and 12-month all-cause mortality compared to PCI

Table 2: Procedural and postprocedural characteristics of all patients in this study

Variables	IABP before PCI (n = 106)	IABP after PCI (n = 112)	P
Culprit vessel			
LM	11 (10.4)	14 (12.5)	0.82
LAD	40 (37.7)	41 (36.6)	0.73
LC	12 (11.3)	17 (15.2)	0.57
RCA	43 (40.6)	40 (35.7)	0.51
Multi-vessel disease	26 (24.5)	33 (29.5)	0.44
Door-to-Balloon time (min)	77.8 ± 53.4	63.7 ± 32.1	0.04
Symptom onset-to-balloon time (h)	7.5 ± 5.1	7.9 ± 6.3	0.13
Complete revascularization	35 (33.0)	39 (34.8)	0.85
Post-PCI TIMI 3 flow			
Glycoprotein IIb/IIIa inhibitor use	78 (73.6)	79 (70.5)	0.64
Need for high-dose vasopressor	27 (25.5)	31 (27.7)	0.58
Need for mechanical ventilation	65 (61.3)	73 (65.2)	0.61
Cardiac biomarkers 24h Post-PCI			
Troponin I (μg/L)	41.4 ± 32.7	31.6 ± 30.5	0.03
BNP (pg/ml)	397.5 ± 272.1	407.8 ± 327.6	0.36
Myocardial Perfusion			
MBG 0/1	24 (22.6)	38 (33.9)	0.03
MBG 2	39 (36.7)	33 (29.5)	0.17
MBG 3	43 (40.6)	41 (36.6)	0.28
STR <30%	21 (19.8)	33 (29.5)	0.03
STR >50%	38 (35.8)	33 (29.5)	0.08
STR >70%	47 (44.3)	46 (41.1)	0.55

Data are presented as n (%) or mean ± SD. IABP: Intra-aortic balloon pump; PCI: Percutaneous coronary intervention; LM: Left main; LAD: Left anterior descending; LC: Left circumflex; RCA: Right coronary artery; BNP: B-type natriuretic peptide; STR: Resolution of ST-segment elevation; TIMI: Thrombolysis in myocardial infarction; SD: Standard deviation.

Table 3: Clinical Outcomes of all patients at month 12 in this study

Variables	IABP before PCI (n = 106)	IABP after PCI (n = 112)	P
MACCE	75 (70.7)	82 (73.2)	0.53
All-cause mortality	46 (43.4)	49 (43.7)	0.97
Cardiac mortality	43 (40.6)	43 (38.4)	0.79
Reinfarction	9 (8.5)	11 (9.8)	0.64
Revascularization	17 (16.4)	20 (17.6)	0.61
Definite stent thrombosis	4 (3.8)	6 (5.4)	0.49
Stroke	2 (1.9)	2 (1.8)	1.00
Acute kidney injury	19 (17.9)	21 (18.7)	0.62
GUSTO Bleeding	23 (21.7)	27 (24.1)	0.45
Life-threatening or severe bleeding	2 (1.9)	2 (1.8)	1.00

Data are presented as n (%). IABP: Intra-aortic balloon pump; PCI: Percutaneous coronary intervention; MACCE: Major adverse cardiac and cerebrovascular event; GUSTO: Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries.

alone.[18] Combining 2155 patients undergoing high-risk PCI procedure from 12 randomized trials, Wan *et al*.[16] confirmed that IABP did not significantly decrease short-term

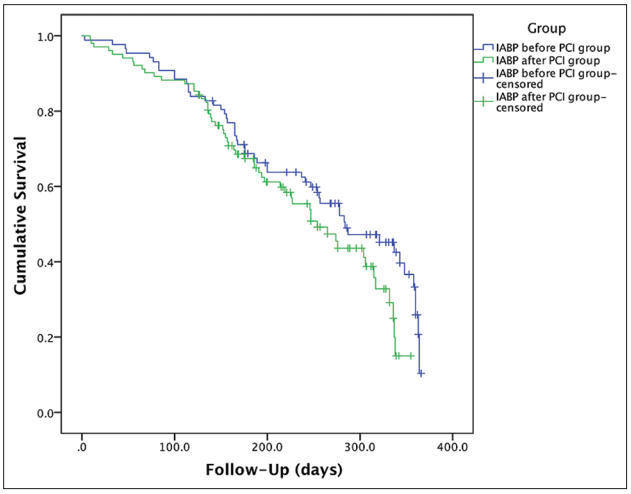

Figure 1: Kaplan-Meier curve for all-cause mortality up to 12 months (P = 0.92).

mortality (*RR* 0.66, 95% *CI* 0.42–1.01) or long-term mortality (*RR* 0.79, 95% *CI* 0.47–1.35). In consistent with these studies, we found that risk of 12-month mortality and MACCE was similar between the patients treated with IABP before and after primary PCI. As such, our study was underpowered to detect a difference in mortality and more studies are needed to confirm our findings.

Delay in mechanical reperfusion therapy during STEMI is associated with greater injury to the microcirculation. In the HORIZONS-AMI trial of 2056 patients with STEMI, Prasad *et al*. examined the effect of symptom onset-to-balloon time and DBT on myocardial reperfusion during primary PCI. They found that absent microvascular perfusion after PCI was significantly more common in patients with longer DBT and MBG 0/1 and STR <30% identified patients with increased 3-year mortality.[7] Our results were consistent with findings that IABP therapy was associated with improved myocardial perfusion. The predominant benefit of IABP on high-risk patients with severe coronary stenosis might relate to a reduction in oxygen demand through LV systolic unloading over and above that stimulated by diastolic augmentation of coronary blood flow.[19] We found that myocardial perfusion, which was assessed by ECG and angiography, was markedly improved in patients treated with IABP before PCI versus after PCI. Meanwhile, these beneficial effects of IABP are offset by the increased reperfusion delay associated with the time needed for IABP insertion. A previous randomized study in STEMI patients without CS reported an additional delay of approximately 10 min in patients who received an IABP before primary PCI compared to those who did not receive an IABP before PCI.[20] Although this delay of approximately 10 min seems small, it has been well shown that increased time to reperfusion markedly increases the extent of irreversible myocardial damage, especially in the 1st h after symptom onset.[21]

The international guidelines endorsed the use of IABP in treating CS postmyocardial infarction with Class 1

recommendation despite the lack of adequately powered randomized trials and the recent meta-analysis data that showed limited efficacy of IABP use.[7] Giving the accumulated body of evidence from observational studies, it seems appropriate to criticize the recommendation from 2008 guidelines on the timing of IABP treatment. New guidelines from 2015 have downgraded the recommendation to Class IIb, but it does not mention when IABP treatment should start.[22] The previous guidelines specifically recommended that IABP should be commenced before start of angiography and PCI. As the documentation for the timing of IABP treatment in relation to primary PCI is weak and ambiguous, more studies are needed to address the long-term effect of IABP sequence on survival.

This study is not a randomized clinical trial but an observational retrospective cohort study. Despite using multivariable analysis to adjust for possible confounders that may be correlated to study outcomes, we cannot exclude the possibility of residual confounding. However, there were no significant differences in baseline characteristics between the two groups so that the outcomes could be more easily compared.

In conclusion, our study showed that early IABP insertion before primary PCI is associated with a delay of DBT. However, myocardial perfusion is markedly improved with IABP therapy before PCI. Overall, IABP support before PCI does not confer a 12-month clinical benefit when used for STEMI with CS. More studies are needed to confirm our findings.

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Conflicts of interest

There are no conflicts of interest.

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