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Original Article

In-hospital outcome of acute ST-segment elevation myocardial infarction in patients with IABP support according to different TIMI risk scores

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Abstract: Objective: To investigate the in-hospital mortality rate of STEMI patients treated with IABP according to different TIMI risk scores (TRS). Methods: The clinical data of 897 consecutive ST-segment elevation myocardial infarction (STEMI) patients, without mechanical complications, from July 2005 to July 2013, were retrospectively analysed. The in-hospital outcomes were compared for the 293 patients with intra-aortic balloon pumps (IABPs) versus those without, using the thrombolysis in myocardial infarction risk score (TRS) for risk stratification. Results: According to the ROC curve results for TRS, patients were divided into three risk stratifications. Patients with IABP support had a lower in-hospital mortality (4.3% versus 12.2%, $P = 0.011$) for TRSs of 4-8. However, there was no significant difference between the two groups in other risk stratifications. Univariate logistic regression analysis indicated a significant association between IABP and in-hospital mortality in patients with TRSs of 4-8 (OR: 0.326, 95% CI: 0.136-0.786, $P = 0.013$). After propensity stratification analysis, there was still a significant difference in the odds for mortality (OR: 0.357, 95% CI: 0.143-0.889, $P = 0.027$). Conclusions: IABP support may be more effective in reducing in-hospital mortality for STEMI patients whose hemodynamics is compromised with a TRS of 4-8.

Keyword: Intra-aortic balloon pump, ST-segment elevation myocardial infarction, TIMI risk score, in-hospital mortality, propensity stratification analysis

Introduction

With the widespread use of invasive treatment modalities such as early revascularisation and intensive health care, there has been a profound reduction in mortality due to ST-segment elevation myocardial infarction (STEMI) in recent decades [1, 2]. Nonetheless, sustained hypotension, cardiogenic shock (CS) or heart failure at the time of STEMI is associated with considerably increased mortalities, ranging from 45% to 80% [3, 4]. Patients suffering from STEMI have characteristics that vary across a range of severities. Studies have indicated that the intra-aortic balloon pump (IABP) can improve diastolic coronary perfusion and end-organ perfusion and reduce myocardial afterload and myocardial oxygen consumption [5, 6]. These physiological effects are known to lead to improving myocardial and organ recovery after acute myocardial infarction (AMI). The optimal timing of IABP insertion in management of STEMI remains controversial.

However, there remains an ongoing debate about the use of IABPs in high-risk AMI patients who develop hemodynamic instability. The identification of appropriate patients for IABP support and the timing of insertion are pivotal to optimising the use of this intensive therapy and improving outcomes after STEMI. The thrombolysis in myocardial infarction (TIMI) risk score (TRS) for STEMI is a simple arithmetic score that can predict short-term mortality based on clinical data at admission [7, 8]. Accordingly, we investigate the in-hospital outcomes of patients with STEMI treated with IABP counterpulsation according to different TIMI risk scores.

Materials and methods

The clinical data of all STEMI patients in the Cardiology Department of Anzhen Hospital

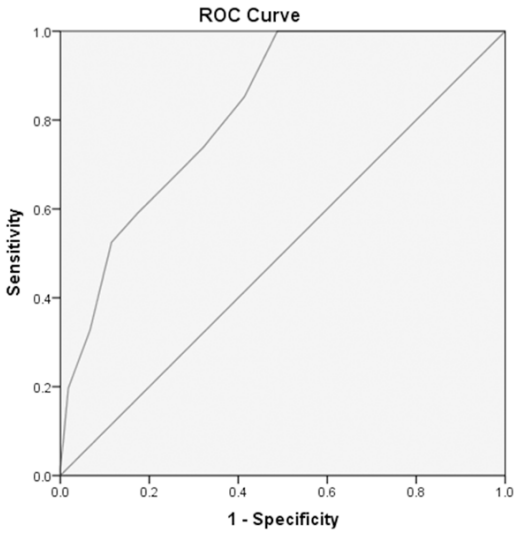

Figure 1. ROC analysis of TIMI Risk Scores of 0-14 for STEMI.

were retrospectively reviewed. For further analysis, this retrospective study included 897 STEMI patients without mechanical complications from July 2005 to July 2013. The duration between symptom onset and admission to hospital for all patients selected was within 72 hours and all patients survived the first 24 hours after admission. Patients with mechanical complications of STEMI (e.g., ventricular septal defects or papillary muscle rupture), significant aortic regurgitation, severe cerebral damage, resuscitation 130 min before admission or severe peripheral vascular disease were excluded. Patients with mechanical complications were excluded because IABP support should be indicated in these patients [9]. The definition of acute STEMI [10] includes symptoms of ischaemia in combination with dynamic ischaemic electrocardiographic changes consistent with STEMI (ST elevation in contiguous leads of the electrocardiogram or associated with new left bundle branch block morphology) and elevation of serum cardiac biomarker values, including creatine kinase-MB and troponin I.

Baseline characteristics including demographics, echocardiography, coronary angiography and hemodynamic parameters during IABP support and complications were acquired from patient medical records. Written informed consent was received from participants prior to inclusion in the study, which was undertaken in accordance with ethical regulations imposed by the Chinese legislation.

The evolution of in-hospital outcomes was compared in the 293 patients with IABP versus those without IABP. TRS was used for risk stratification and calculated according to the score criterion [11]. The necessary data at admission used to calculate the TRS were available for all patients. In addition, to reflect the hemodynamic status before IABP insertion, systolic blood pressure (SBP), heart rate and heart function (Killip classification) before IABP treatment started were used to calculate the TRS of patients with IABP support. The prognostic accuracy of TRS was assessed using receiver operating characteristic (ROC) curves. The Youden index was used to determine the critical cut-off point on the ROC curves.

IABP insertion was performed by experienced cardiologists, with the timing of the insertion at the discretion of the physician. The indications for IABP support were STEMI with CS, hemodynamic support in catheterization laboratory, reinfarction, and intractable ventricular arrhythmia. An 8-French IABP catheter (30 or 40 ml, Arrow Corp, USA) was placed percutaneously, via the femoral artery, using the Seldinger technique. The tip of the balloon was placed 2-3 cm distal to the junction with the left subclavian artery. The position of the balloon tip was verified by a chest radiograph or a fluoroscopy in the catheter laboratory after insertion. The duration of the IABP support was determined by the physician, depending on the patient's hemodynamic status or intolerable complications to continued IABP support.

Definitions

CS [12] was defined according to clinical and hemodynamic criteria, including a SBP of <90 mmHg for ≥30 min or supportive measures such as inotropic agents or IABP required to maintain a SBP of ≥90 mmHg, evidence of end-organ hypoperfusion (e.g., persistent oliguria with a urine output of <30 mL/hour, cool and diaphoretic extremities, changes in mental status).

Thrombocytopenia was defined as at least two platelet counts of less than 40,000,000/mL during IABP support.

Access-site complications [13] were defined as a vascular complication at the access site resulting in hematoma, false aneurysm, or femoral artery occlusion requiring surgical or per-

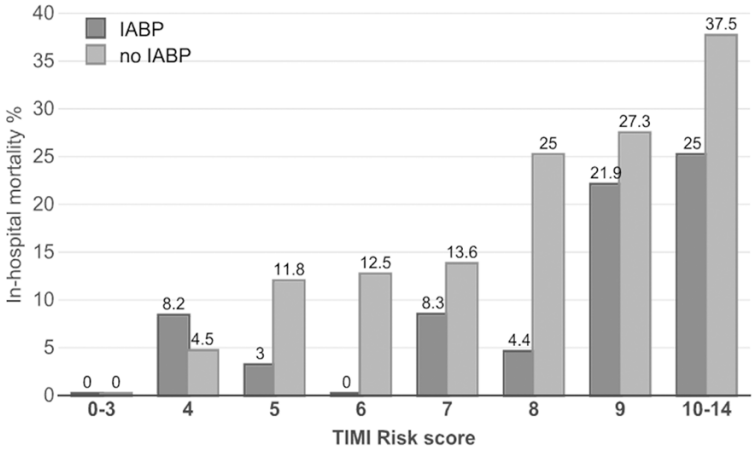

Figure 2. In-hospital mortality with TIMI risk score for STEMI.

Table 1. In-hospital mortality (IABP versus no IABP) for different TIMI risk scores

TIMI risk score	In-hospital mortality	*P*	1-β	OR	95% CI
4-8	4.3 vs 12.2%	0.013	0.651	0.326	0.136-0.786
4-9	7.9 vs 13.1%	0.097	0.645	0.571	0.295-1.107
9-14	23.9 vs 31.6	0.475	0.732	0.679	0.234-1.965
10-14	25 vs 37.5%	0.452	0.742	0.556	0.120-2.573

TIMI: Thrombolysis in Myocardial Infarction; OR: Oodds ratio; 1-β (power) is the probability of rejecting the null hypothesis when it is false.

Table 2. Patients in cardiogenic shock for different TIMI risk scores [n/N (%)]

TIMI scores	IABP	No IABP	*P*
0-3	0/23 (0)	0/405 (0)	-
4-8	33/161 (20.5)	5/180 (2.8)	<0.001
9-14	40/109 (36.7)	3/19 (15.8)	0.113

TIMI: Thrombolysis in Myocardial Infarction; n: Number of patients who was in cardiogenic shock; N: Total number of patients in group.

cutaneous intervention at the IABP inserted site.

Systemic embolisation included thrombotic embolisation to any vascular territory, with the exception of the pulmonary arteries and their tributaries.

Critical limb ischaemia was defined as a loss of pulse, intractable rest pain, abnormal limb temperature or pallor requiring surgical intervention on the balloon inserted limb.

An IABP failure was defined as poor augmentation, an inability to deploy or any IABP leak suggested by blood inside the catheter tubing or gas loss.

Statistical analysis

Continuous variables were presented as means with standard deviations and compared by the Student's t-test if the data were of normal distribution, otherwise they were presented as a median (inter-quartile range) and the Mann-Whitney U test was used. Categorical variables were presented as percentages and compared using chi-square analysis or Fisher's exact test, where appropriate. The diagnostic accuracy of TRS was assessed using ROC curves. The Youden index was used to determine the critical cut-off point on the ROC curves. Odds ratios of in-hospital mortality for the IABP versus the no IABP group were derived from univariate logistic analyses, and in attempting to minimise the influence of confounding and bias, from propensity stratification analyses. The propensity score stratification analyses were performed using age, gender, SBP and heart rate at admission and multi-vessel disease. The discriminative ability of the derived propensity scores was assessed using the area under the curve (AUC) of the ROC (>0.7 good discriminative ability). For the propensity score stratification analyses, strata were created based on quarters of the score where the two groups could be compared. All statistical tests were two-tailed, and *P* values were statistically significant at <0.05. All statistical analyses were carried out using the SPSS statistical software V.20.0 (SPSS Inc., Chicago, Illinois, USA).

Results

The analysis included 897 patients with STEMI either supported or not by an IABP. The in-hospital mortalities of patients with IABP support versus those without IABP support were 11.3% versus 4.6% (*P*<0.01). An ROC curve was used

Table 3. Baseline characteristics in patients with TIMI risk score 4-8 [n (%), M (QR), X±s]

	IABP (*n* = 161)	No IABP (*n* = 180)	*P*
Male	134 (83.2)	113 (62.8)	<0.01
Age (years)	57.6±11.2	63.0±12.6	<0.01
Age >65 years	41 (25.5)	90 (50)	<0.01
Cardiovascular disease Risk factors			
Hypertension	74 (46.2)	107 (59.4)	0.015
Diabetes	42 (26.2)	57 (32.0)	0.136
Angina	59 (36.9)	13 (7.2)	<0.01
Family-CAD history	10 (6.2)	0 (0)	<0.01
Smoking	111 (68.9)	98 (54.4)	<0.01
Past history			
Pre-stroke	7 (4.3)	20 (11.1)	0.026
Peripheral vascular disease	3 (1.9)	1 (0.6)	0.347
Pre-PCI	20 (12.4)	19 (10.6)	0589
Pre-CABG	1 (0.6)	3 (1.7)	0.625
Pre-MI	23 (14.3)	18 (10.0)	0.224
Presentation history			
SBP/mmHg	108.8±24.2	115.1±24.9	0.018
SBP<100 mmHg	73 (45.3)	71 (39.4)	0.271
DBP/mmHg	68.1±15.8	71.6±17.1	0.049
HR/bpm	85.2±19.8	76.7±17.5	<0.01
HR>100 bpm	38 (23.6)	20 (11.1)	<0.01
Body weight <67 kg	11 (6.8)	21 (11.7)	0.126
Hemoglobin <110 g/L	9 (5.6)	19 (10.6)	0.095
Platelet/10^9/L	199 (164, 243)	202 (160, 240)	0.510
Serum creatinine/μmol/L	87.2 (74.1, 103.6)	82 (99.0, 67.3)	0.058
EF<35%	25 (15.5)	15 (8.3)	0.044
Killip class II-IV	103 (64.0)	90 (50.0)	<0.01
Time from symptom onset to reperfusion >4 hours	94 (58.4)	57 (31.7)	<0.01
Length of stay (day)	10 (6, 13)	9 (7, 13)	0.384

TIMI: Thrombolysis in Myocardial Infarction; DBP: Diastolic blood pressure; SBP: Systolic blood pressure; LDL: Low density lipoprotein; CAD: Coronary artery disease; EF: Left ventricular ejection fraction.

to assess the sensitivity and specificity of TRSs for predicting in-hospital mortality (**Figure 1**). The AUC was 0.820 (P = 0.022). The best TRS cut-off point for predicting in-hospital mortality was 3.5 based on the ROC curve and the sensitivity and specificity were 100.0% and 51.2%, respectively. The in-hospital mortalities for STEMI with different TRSs are shown in **Figure 2**. With the threshold predicted by the ROC curve, 428 patients with TRS ≤3 survived in hospital. Furthermore, the data for patients with TRS ≥3 and ≥4 were analysed using the ROC curve. The best TRS cut-off point for these two groups was 8.5 with AUCs of 0.706 (P = 0.035) and 0.632 (P = 0.044), respectively. According to the ROC curve results for TRS, patients were divided into three risk stratifications (i.e., 0-3 scores, 4-8 scores and 9-14 scores) (**Table 1**).

Univariate logistic regression analysis showed that IABP was significantly associated with lower in-hospital mortality in patients with TRSs between 4 and 8 (OR: 0.326, 95% CI: 0.136-0.786, P = 0.013). However, there was no significant association between IABP and in-hospital mortality in patients with TRSs of 4-9, 9-14 and 10-14 (P>0.05). In addition to univariate logistic regression analysis, chi-square analysis showed that patients with IABP support had a lower in-hospital mortality (4.3% versus 12.2%, P = 0.011) for TRSs of 4-8. However, there was no significant difference between the two groups with TRSs above 9 (23.9% versus 31.6%, P = 0.566).

Table 4. Characteristics of angiography and revascularization treatments in TIMI risk score 4-8

Variable	IABP (*n* = 294)	No IABP (*n* = 604)	*P*
Infarction region			
Anterior	94 (58.4)	113 (62.8)	0.407
Inferior/Right ventricular	77 (47.8)	70 (38.9)	0.096
Multivessel disease	93 (57.8)	72 (40.0)	<0.01
Reperfusion			
Thrombolysis	16 (10.0)	13 (7.2)	0.360
Primary PCI	131 (81.4)	115 (63.9)	<0.01
ECABG	0	0	-

TIMI: Thrombolysis in Myocardial Infarction; IABP: Intra-aortic balloon pump; ECABG: Emergency coronary artery bypass grafting.

Table 5. Propensity score stratification analysis IABP vs. no IABP in TIMI risk score 4-8

Strata	Propensity stratifications	IABP n/N	No IABP n/N	*P*	*OR*	95% CI
1	1.92-2.29	0/21	1/47	0.998	<0.01	-
2	2.29-2.50	0/37	1/37	0.998	<0.01	-
3	2.50-2.73	1/34	8/39	0.049	0.117	0.014-0.994
4	2.73-3.39	6/58	7/18	0.020	0.212	0.057-0.780
Overall	1.92-3.39	7/150	17/141	0.027	0.357	0.143-0.889

TIMI: Thrombolysis in Myocardial Infarction; n: Number of patients who died; N: Total number of patients in group; OR: Odds ratio.

Table 6. Pre-IABP and post-IABP hemodynamics in different TIMI scores

	4-8 (n = 161)	9-14 (n = 109)	**P*
SBP (mmHg)			
Pre	89.8±22.9	82.7±14.8	<0.01
Post	111.2±15.6	107.4±17.7	0.066
	<0.01†	<0.01†	
DBP (mmHg)			
Pre	56.4±13.2	52.2±12.0	0.01
Post	66.9±11.5	62.2±12.4	<0.01
	<0.01†	<0.01†	
HR (Bpm)			
Pre	83.4±24.6	96.5±28.1	<0.01
Post	82.5±22.9	87.9±19.3	0.042
	0.738†	<0.01†	

SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HR: Heart rate; **P* value between groups; †*P* value within group; 1 mmHg = 0.133 kPa.

Table 2 indicated that more patients were in cardiogenic shock with IABP support (20.5% versus 2.8%, *P*<0.001) for TRSs of 4-8. On the other hand, there was no significant difference between the two groups with TRSs above 9 (36.7% versus 15.8%, *P* = 0.113).

Table 3 showed the baseline characteristics of the patients with TRSs of 4-8. **Table 4** showed the characteristics of angiography and revascularisation treatments for TRSs of 4-8. Although these patients were classified into the same risk stratification by TRS, their characteristics significantly differed in a number of baseline, angiography and revascularisation treatments. Those in the IABP group were younger and more likely to be male. They more often presented with lower blood pressure, a higher heart rate and a longer time from symptom onset to reperfusion. They were more likely to have multivessel disease. To minimise the influence of bias and confounding, the final propensity score model to predict the likelihood of the use of IABP therapy for patients with TRSs of 4-8 included five independent predictors, age, gender, SBP, heart rate at admission and multivessel disease. The discriminatory capacity of the propensity score was determined by the AUC of the ROC curve. The estimated propensity stratification analysis for IABP versus no IABP had an AUC of 0.714 (95% CI: 0.657-0.771), indicating adequate discrimination. The odd ratio of mortality after stratification by quarters of the propensity score is detailed in **Table 5**. The positive validity for odds ratio of mortality for IABP therapy versus no IABP therapy was still significant with adjustment by the propensity stratification (OR: 0.357, 95% CI: 0.143-0.889, *P* = 0.027).

Notably, significant differences in hemodynamic parameters between groups with TRSs of 4-8 and 9-14 were observed. The patients in the group with a TRS of 9-14 had a lower SBP (*P*<0.01) and DBP (*P* = 0.01) and a higher heart rate (*P*<0.01) before their IABP insertion. Both groups tended toward higher SBP and DBP after their IABP insertion (*P*<0.01). However, significant differences in the hemodynamic response to IABP therapy between the 2 groups

Table 7. In-hospital balloon-related complications in different TIMI risk scores [n (%)]

The complications	0-3	4-8	9~	*P*
Aortic dissection	0	0	0	-
Systemic embolization	0	1 (0.6)	0	0.664
Critical limb ischemia	0	0	0	-
Thrombocytopenia	0	0	7 (6.4)	<0.01
Gastrointestinal hemorrhage	0	7 (4.4)	10 (9.2)	0.073
Access-site complication	0	5 (3.1)	7 (6.4)	0.161
IABP failure	0	0	1 (0.9)	0.380
Withdraw IABP due to the complications	0	2 (1.2)	6 (5.5)	0.067

TIMI: Thrombolysis in Myocardial Infarction.

could be observed. S/DBP after IABP insertion was significantly higher in the group with TRSs of 4-8 (P<0.01). However, a higher heart rate after IABP insertion was found in the group with TRSs of 9-14 (P = 0.042) (**Table 6**).

The most frequently observed complications were thrombocytopenia, access-site complications and gastrointestinal haemorrhaging. There was a trend towards a higher incidence of complications in patients with a TRS above 9 (P<0.05). No episodes of aortic dissection or critical limb ischaemia attributed to IABP counterpulsation were noted among the groups (**Table 7**).

Discussion

IABP support is used as the first-line treatment in AMI, with the goal of providing temporary mechanical support and allowing time for myocardial recovery. However, this therapy is often incapable of overcoming hemodynamic compromise in severe refractory CS and data on the usefulness of IABPs in STEMI are conflicting [14, 15].

The published data of IABP-SHOCK II trial [16] failed to show an in-hospital mortality benefit. The study resulted in a downgrading of the use of IABP in post-infarction CS patients by certain professional organisations, such as the European Society of Cardiology (ESC) and the American Heart Association [17, 18]. The Counterpulsation to Reduce Infarct Size Pre-PCI-Acute Myocardial Infarction (CRISP AMI) trial [19] did not demonstrate any significant reduction of the infarct size in patients treated with IABP. In line with the ESC guidelines, the routine application of IABP for STEMI patients without CS is not recommended [17]. There remains an ongoing debate about the use of IABP in high-risk STEMI patients who develop hemodynamic instability, suggesting that the timing of initiation of IABP therapy could be of great importance [20]. Our study aimed to investigate the efficiency of IABP for STEMI patients, according to different severities classified by TRS.

It is noteworthy that the distribution of patients supported by IABP therapy in TRS is unbalanced. Among the 428 patients whose TRSs were ≤3, only 23 (5.4%) patients used IABP while 405 (94.6%) patients did not. No patient died in this risk stratification. Although most of the patients were not supported by IABP, the prognosis of these patients was expected to be good. However, among the 128 patients whose TRSs were ≥9, 109 patients (85.2%) received IABP therapy. The mortality of patients with IABP support versus those without was 23.9% versus 31.6% in this risk stratification (P>0.05). In general, patients who received IABP therapy had more frequent high-risk features and a higher TRS (**Figure 2**). This is representative of the real world situation, with cardiologists preferentially giving IABP support to patients with a poorer hemodynamic status. It is difficult to deny patients aggressive treatment with IABP, even if their prognosis is extremely bleak. However, IABP is not significantly associated with in-hospital mortality in patients with TRSs of 9-14 (OR: 0.679, 95% CI: 0.234-1.965, P = 0.475). Most of these patients may have had more progressive end-organ dysfunction and impaired heart function. Consequently, these critically ill patients may still have a poor prognosis despite the use of intensive and multifaceted therapy, including IABP support [21].

TRSs may be useful in selecting patients for IABP support. Based on our findings, the selection of patients for IABP insertion before circulatory collapse can improve the in-hospital survival rate. The mortality of patients with and without IABP support was 4.3% versus 12.2% for TRSs of 4-8 (P = 0.011). Univariate logistic regression showed that IABP therapy was associated with lower in-hospital mortality in patients with TRSs of 4-8 (OR: 0.326, 95% CI:

0.136-0.786, $P = 0.013$). The association between IABP and in-hospital mortality was still significant, with adjustment by the propensity stratification model for patients with TRSs of 4-8 (**Table 5**).

The results showed a much higher revascularisation rate in patients with IABP support than in the no IABP group. Consistent with the results of previous studies [22, 23], this work shows that the widespread use of primary PCI as reperfusion therapy may reduce mortality following STEMI. Previous studies have shown that the short-term outcome for STEMI patients supported with IABP is influenced by the reperfusion method. In recent years, many clinical trials have shown that reperfusion therapy [24-27], especially primary PCI, is superior to conservative treatment for improving left ventricular function, reducing the in-hospital mortality of patients with STEMI. This suggests that IABP may be a more effective therapy modality when it is associated with effective revascularisation. Without an effective reperfusion therapy to reduce the amount of myocardium at risk of irreversible damage, short-term salvage strategies such as IABP may not affect mortality. Therefore, it is of extreme importance for patients with STEMI to receive timely and effective reperfusion therapy, which includes either thrombolysis or PCI.

It is noteworthy that there were significant differences in vital signs, including blood pressure and heart rate response, between IABP patients with TRSs of 4-8 and those with TRSs of ≥9. In the group with TRSs ≥9, there was less of an increase in diastolic blood pressure after IABP insertion. Considering the role of diastolic blood pressure influencing the perfusion of the coronary artery, this mild augmentation diastolic blood pressure may be correlated with poor in-hospital prognosis [28]. Although heart rates decreased to some extent in the group with TRSs ≥9, during IABP support the magnitude of the heart rate in this group was still compromised compared to patients with TRSs of 4-8. Although temporal improvements in hemodynamic parameters such as blood pressure and heart rate were observed, IABP cannot improve the circulatory collapse status for patients whose cardiac function is already severely damaged. Meanwhile, previous studies have shown that patients whose hemodynamic parameters responded significantly to IABP may receive benefits from IABP therapy, improving their short-term survival [29].

Our study has several limitations. First, this was a retrospective study, with the shortcomings common to this approach. Second, data regarding angiographic results of primary PCI were not available for all patients. Third, the results might be attributable to some imbalances between the groups. According to the data presented in **Table 3**, a treatment selection bias is likely to be present in in patients with TRSs of 4-8, younger patients with multivessel diseases and depressed EF receiving more frequently IABP and PCI. Despite the refined results of the risk stratification according to TRS and propensity stratification analysis, it remains important to note that the study could still have been subject to residual confounding and bias. We did not have adequate patients to bring more clinical characteristics into the propensity score model as predictors of the likelihood of the use of IABP therapy. In addition, there remained an imbalance in the distribution of patient support by IABP in TRS, especially among those patients with TRS scores of ≤3 and ≥9. If more patients in these two risk stratifications were involved in our study, a more convincing result may have been achieved.

IABP may reduce in-hospital mortality among STEMI patients with TRSs of 4-8. Patients with TRSs ≥9 may still have a poor prognosis, despite the use of intensive and multifaceted therapy, including IABP. Patients with TRSs of 4-8 may have more significant responses to IABP than patients with TRSs ≥9.

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Disclosure of conflict of interest

None.

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