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Original Article

Risk factors and clinical characteristics of in-hospital death in acute myocardial infarction with IABP support

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Abstract: Background: Despite the widespread use of the intra-aortic balloon pump (IABP) in acute myocardial infarction (AMI), there were few clinical trials regarding the deceased's feature. Therefore, we conducted a study to investigate the clinical characteristics and risk factor led to in-hospital deaths among AMI patients with IABP support. Objective: To investigate the clinical characteristics and risk factors of in-hospital death with IABP support in AMI patients. Methods: The clinical data of 572 consecutive IABP supported patients with AMI within 72 hours from symptom onset from July 2005 to July 2013 were retrospectively analyzed. The evolution of the risk factors of in-hospital death and clinical characteristics was compared in 81 non-survivors and the survivors. Results: Non-survivors had a more severe clinical profile at admission. Fewer patients were treated with emergency reperfusion therapy in the non-survivors group. Cardiogenic shock, Mechanical complications, ventricular tachycardia/fibrillation and MODS were much common in non-survivors (P<0.001). Multivariate logistic regression analysis showed advanced age (>65 years), prolonged time from symptom onset to first medical contact (FMC), Killip class III/IV, renal dysfunction(GFR <60 ml/min/1.73 m^2), and left ventricular ejection fraction (LVEF) <30% were risk factors associated with higher in-hospital mortality. Conclusions: IABP support may be more effective combined with revascularization for AMI patients whose hemodynamics is compromised. Patients accompanied with cardiogenic shock and other life-threatening complications are often uselesswith IABP support. Meanwhile, patient whose hemodynamics parameters have significant response to IABP may get benefits with IABP to improve in-hospital survival.

Keyword: Intra-aortic balloon pump, acute myocardial infarction, in-hospital mortality, risk factors

Introduction

With the widespread use of invasive treatment modalities such as early revascularization and intensive health care, there has been a profound fall in mortality over last decade [1]. Nonetheless, Sustained hypotension, cardiogenic shock (CS), or heart failure at the time of AMI accompanied with considerably increased mortalities ranging from 45% to 80% [2-4]. Patients suffered from AMI have characteristics that vary across a range of severity. Studies have indicated that the IABP can improve diastolic coronary perfusion and systemic blood flow, and reduce myocardial afterload and myocardial oxygen consumption [5-7]. These physiologic effects are known to lead to improve myocardial and organs recovery after AMI. The optimal timing of IABP insertion in management of AMI remains controversial. Despite IABP had emerged as the most widely used circulatory assist device worldwide, there were few clinical trials regarding deceased's feature with IABP support. Therefore, we analyzed the risk factors and clinical characteristics of in-hospital death among AMI patients with IABP support.

Materials and methods

Study population

The clinical data of all AMI patients in Cardiology Department of Beijing Anzhen Hospital were retrospectively reviewed. For further analysis, this retrospective study included 572 AMI patients who received IABP in hospital from July 2005 to July 2013. All patients' symptom onset to admission was within 72 hours. Criteria for acute myocardial infarction [8], including elevation of serum biomarker values (i.e., >1×99th percentile upper reference limit) and at least

one of the following: ① Symptoms of ischaemia. ② New ischaemic ECG changes. ③ Imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality. ④ Angiographic findings of intracoronary thrombi. Baseline characteristics including demographics, echocardiography, coronary angiography, hemodynamic parameters in duration of IABP support and complications were acquired from patient medical records. The evolution of clinical characteristics and relative factors of in-hospital death was analyzed in the 81 non-survivors and 491 survivors. 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.

Treat regimen

Indications for IABP support were AMI with CS, acute pulmonary edema, hemodynamic support during percutaneous coronary intervention (PCI), re-infarction, mechanical complication, or intractable ventricular arrhythmia after AMI. IABP insertion was performed by experienced cardiologists, with the indication and the timing of the insertion at the discretion of the physician. 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. And the position of the balloon tip was verified by a chest radiograph or a fluoroscopy in catheter laboratory after the insertion. The duration of the IABP support was determined by the physician depending on the patient's hemodynamic status or intolerable complications to continue IABP support.

All patients were also treated with standard therapies for AMI, such as oxygen, morphine for pain relief, anti-platelet therapy, and anticoagulation with heparin. Pharmacological treatment also included administration of inotropic agents (e.g., dopamine and dobutamine) if peripheral hypoperfusion occurred (e.g., hypotension, decreased renal function), diuretics and fluids were given on the basis of the estimated filling pressures. Those who had appropriate indication for reperfusion therapy were treated with thrombolysis or PCI according to the clinical circumstances. Mechanical ventilation was applied if necessary. Laboratory tests [e.g., complete blood cell count (CBC), cardiac biomarkers, blood urea nitrogen (BUN) and serum creatinine (Scr)] were performed once the patients entered into emergency room or department of cardiology ward, other laboratory and auxiliary examination were completed within 24 hours in hospital.

Statistical analysis

The baseline characteristics of patients were presented as mean ± SD for continuous variables and compared by the Student t-test if the data were of normal distribution, otherwise presented as median (25th and 75th percentiles) and Wilcoxon signed-rank test was used. Categorical variables were presented as percentage and compared by Pearson χ^2 or Fisher' exact test, where appropriate. Multivariate logistic regression was used to identify independent clinical and laboratory risk factors at admission associated with in-hospital mortality. All baseline characteristic variables entered the regression if $P<0.05$. 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).

Definitions

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

Renal dysfunction was defined as an estimated glomerular filtration rate (eGFR) less than 60 mL/min/1.73 m^2, and the eGFR was calculated based on an admission laboratory analysis using the Modification of Diet in Renal Disease equation [9].

The IABP failure was defined as poor augmentation, inability to deploy or any IABP leak suggested by blood inside the catheter tubing or gas loss [10].

Thrombocytopenia was defined as at least 2 times platelet count of less than 40,000,000/mL during IABP support.

Table 1. Baseline characteristics in patients with AMI [n (%), M (QR), $\bar{x}\pm s$]

Variable	clinical characteristics of AMI patients with IABP insertion		*P*
	death in hospital (n=81)	survived (n=491)	
Age (years)	65.25±10.73	60.03±11.81	<0.001
Female (%)	26 (32.1)	105 (21.4)	0.033
Past history			
Smoking (%)	43 (53.1)	303 (61.8)	0.135
Hypertension (%)	55 (67.9)	244 (49.7)	0.002
Diabetes (%)	34 (42)	139 (28.3)	0.013
Pre-stroke (%)	10 (12.3)	41 (8.4)	0.245
Family-CAD history	0 (0)	6 (1.2)	0.601
CAD history			
Pre-MI	18 (22.2)	86 (17.6)	0.317
Pre-PCI	11 (13.6)	48 (9.8)	0.300
Pre-CABG	3 (3.7)	5 (1.0)	0.091
Presentation history			
time from symptom onset to FMC (hour)	7 (4, 14.5)	6 (3, 10)	0.05
HR (bpm)	91.59±25.86	83.86±19.46	0.011
SBP (mmHg)	99.46±29.54	107.58±24.19	0.002
DBP (mmHg)	61.81±17.83	67.48±15.17	0.004
Killip class III~IV	76 (93.8)	173 (35.2)	<0.001
Laboratory examination and auxiliary examination			
Hemoglobin (g/L)	134.80±21.14	139.16±20.48	0.131
leukocyte count ($\times10^9$/L)	11.6 (8.4, 15.7)	11.4 (9.1, 14.0)	0.613
Neutrophil (%)	77.97±13.73	78.40±14.84	0.526
Creatine kinase MB (U/L)	508 (154.0, 1966.5)	430.0 (108.8, 2290.5)	0.434
Cardiac troponin I (ng/ml)	9.19 (0.67, 39.22)	7.79 (0.47, 78.60)	0.886
BUN (mmol/L)	7.51 (5.85, 9.72)	6.64 (5.24, 8.64)	0.012
Serum creatinine (μmol/L)	103.00 (83.11, 135.30)	88.40 (72.7, 104.15)	<0.001
LDL (mmol/L)	2.71 (2.11, 3.47)	2.86 (2.29, 3.44)	0.351
LVEF (%)	40.97±12.96	48.14±10.77	<0.001

DBP: diastolic blood pressure; SBP: systolic blood pressure; BUN: blood urea nitrogen; LDL: low density lipoprotein; FMC: first medical contact; CAD: coronary artery disease; LVEF: left ventricular ejection fraction; *P value between groups.

Access-site complications were defined as a vascular complication at access site resulting in hematoma, false aneurysm, or femoral artery occlusion requiring surgical or percutaneous intervention at the IABP inserted site.

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

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

Major adverse clinical events (MACE) including cardiogenic shock, mechanical complications, cerebral hemorrhage, pulmonary embolism, Ventricular tachycardia or fibrillation and multiple organ dysfunction syndromes.

Results

Demographics

A total of 572 IABP-supported patients with AMI hospitalized between July 2005 and July 2013 were included. 491 patients survived in hospital, whereas 81 patients did not. Baseline char-

Table 2. Characteristics of angiography and treatments in-hospital [n (%)]

Variable	Non-survivors (n=81)	Survivors (n=491)	*P
Emergency coronary angiography	60 (81.1)	425 (87.4)	0.143
Culprit vessels			
LM	7 (8.6)	9 (1.8)	0.010
LAD	38 (46.9)	229 (46.6)	0.963
RCA	27 (33.3)	191 (38.9)	0.339
Multivessels	20 (24.7)	72 (14.7)	0.023
Infarction region			
Anterior	55 (67.9)	271 (55.2)	0.032
Inferior/Right ventricular	28 (34.6)	216 (44.0)	0.112
NSTEMI	7 (8.6)	29 (5.9)	0.348
Coronary arteries with >70% stenosis			
1	17 (21.0)	186 (37.9)	0.002
2	35 (43.2)	163 (33.2)	
3	10 (12.3)	23 (4.7)	
Emergency reperfusion therapy			
Primary PCI	41 (50.6)	376 (76.6)	<0.001
Thrombolysis	9 (11.1)	39 (8.0)	0.344
Rescue PCI	6 (7.4)	28 (5.7)	0.548
No reperfusion therapy	31 (38.3)	73 (14.9)	<0.001
Duration of IABP support (days)	1 (1.4)	1 (1.2)	0.638
Pacing	10 (12.3)	54 (11.0)	0.721
CRRT	7 (9.0)	7 (1.4)	<0.001
Medications in-hospital			
Aspirin	78 (96.3)	489 (99.6)	0.088
Clopidogrel	79 (97.5)	489 (99.6)	0.098
ACEI/ARB	22 (27.2)	377 (76.8)	<0.001
β-blocker	23 (28.4)	392 (79.8)	<0.001
Calcium channel blocker	3 (3.7)	32 (6.5)	0.466
Diuretics	39 (48.1)	203 (41.3)	0.251
Long acting nitrates	43 (53.1)	288 (58.7)	0.347
Inotropic agents	77 (95.1)	280 (57.0)	<0.001
Length of hospital stays (days)	2 (1, 6.5)	9 (7, 13)	<0.001

LAD: left anterior descending coronary artery, LCX: left circumflex coronary artery, RCA: right coronary artery, CRRT: continuous renal replacement therapy, ACEI: angiotensin converting enzyme inhibitor, ARB: angiotensin receptor 1 blocker, NSTEMI: Non-st-segment elevation myocardial infarction. *P value between groups.

acteristics of all treated patients were depicted in **Table 1**. Clinical presentation at admission of the non-survivors was more severe. They exhibited greater clinical and hemodynamic evidence of impairment of cardiac function at admission to the hospital. As shown in **Table 1**, the non-survivors had an advanced age (65.25±10.73 vs. 60.03±11.81 years, P<0.001) and higher proportion of female gender [26 (32.1%) vs. 105 (21.4%), P=0.033], significantly higher heart rate (91.59±25.86 vs. 83.86± 19.46 bpm, P=0.011), lower systolic blood presures (99.46±29.54 vs. 107.58± 24.19 mmHg, P=0.002) and diastolic blood pressures (61.81±17.83 vs. 67.48± 15.17 mmHg, P=0.004), more prevalence of hypertension [55 (67.9%) vs. 244 (49.7%), P=0.002] and diabetes mellitus [34 (42%) vs. 139 (28.3%), P=0.013], prolonged time from symptom onset to FMC [7 (4, 14.5) vs. 6 (3, 10) hours, P=0.05], and Killip III~ IV [76 (93.8%) vs. 173 (35.2%), P<0.001]. The non-survivors had higher BUN [7.51 (5.85, 9.72) vs. 6.64 (5.24, 8.64) mmol/L, P= 0.012] and Scr [103.00 (83.11, 135.30) vs. 88.40 (72.7, 104.15) umol/L, P< 0.001] and a more often impaired LVEF (40.97±12.96 vs. 48.14±10.77%, P<0.001) compared with the survivors.

Angiographic, procedural characteristics

Left main coronary artery (LM) as culprit vessel [7 (8.6%) vs. 9 (1.8%), P=0.01] and multivessels disease (P=0.002) were frequently observed in non-survivors. And these patients were significantly less likely to be treated with angiotensin converting enzyme inhibitor (ACEI) or angiotensin receptor 1 blocker (ARB) [22 (27.2%) vs. 377 (76.8%), P<0.001] and β-blocker [23 (28.4%) vs. 392 (79.8%), P<0.001], while more likely to get inotropic agents [77 (95.1%) vs. 280 (57.0%), P<0.001] during hospitalization. Fewer patients were treated with emergency reperfusion therapy in the non-survivors group (P<0.001). Continuous renal replacement therapy (CRRT) [7 (9.0%) vs.

Table 3. Pre-IABP and post-IABP hemodynamics in survivors and non-survivors

	Non-survivors (n=81)	Survivors (n=491)	*P*
SBP			
Pre	80 (61, 89)	85 (80, 95)	<0.001*
Post	96 (74, 118.5)	111 (104, 120)	<0.001*
Change	11 (0, 31)	23 (11, 36)	<0.001*
	<0.001†	<0.001†	
Mean	99 (75, 115.8)	112 (106.6, 120)	<0.001*
DBP			
Pre	48 (40, 60)	56 (50, 61)	<0.001*
Post	58 (40, 70)	67 (60, 74)	<0.001*
Change	6 (-3, 19)	10 (0, 20)	0.058*
	0.002†	<0.001†	
Mean	60 (40, 68.87)	67 (61, 72.5)	<0.001*
HR			
Pre	102 (80, 120)	80 (70, 95)	<0.001*
Post	92 (77.5, 107)	80 (71, 88.25)	<0.001*
Change	-7 (-22, 1.5)	-2 (-12, 8)	<0.001*
	0.001†	0.007†	
Mean	87.5 (77.4, 106)	79 (71, 88)	<0.001*

SBP: systolic blood pressure. DBP: diastolic blood pressure. HR: Heart rate. **P* value between groups. †*P* value within group.

7 (1.4%), P<0.001] was used more often in the non-survivors group (**Table 2**).

Utilization of IABP

Notably, significant differences in hemodynamics parameters between the two groups were observed. The non-survivors exhibited a lower systolic blood pressure/diastolic blood pressure (P<0.001) and a higher heart rate (P<0.001) before their IABP insertion. Both survivors and non-survivors trended toward higher systolic blood pressure/diastolic blood pressure and lower heart rates after their IABP insertion (P<0.001). This trend was still significant on analysis of mean hemodynamics parameters during IABP support (P<0.001). However, significant differences in hemodynamic response to IABP between the 2 groups could be observed (P<0.001), changes in systolic blood pressure after IABP insertion were much significant in survivors group (P<0.001). However, greater changes in heart rate after IABP insertion could be found in non-survivors (P<0.001) (**Table 3**).

The most frequently observed complications were thrombocytopenia and access-site complication. There was a trend towards a higher incidence of complications in the non-survivors group (P<0.05), including thrombocytopenia [6 (7.4%) vs. 3 (0.6), P<0.001], access-site complication [7 (8.6%) vs. 12 (2.4%), P=0.004] and IABP failure [2 (2.5%) vs. 0 (0), P=0.02]. And more non-survivors prematurely withdrew IABP due to the intolerable complications to continue IABP support [4 (4.9%) vs. 7 (1.4%), P=0.033]. No episodes of aortic dissection attributed to IABP counterpulsation in either group were noted. The incidences of systemic embolization or critical limb ischaemia were similar between the groups (P>0.05). The non-survivors were generally associated with a much higher rate of in-hospital Major adverse clinical events. **Table 4** showed that cardiogenic shock, Mechanical complications, ventricular tachycardia/ventricular fibrillation and MODS were much common in non-survivors (P<0.001).

Multivariate binary logistic regression was summarized in **Table 5**. Risk factors associated with in-hospital mortality were advanced age (age > 65 years) (OR: 2.224, CI: 1.106-4.475, P=0.025), prolonged time from symptom onset to FMC (OR: 1.021, CI: 1.005-1.038, P=0.012), diminished LVEF (EF<30%) (OR: 3.043, CI: 1.117-8.288, P=0.029), Killip class III~IV (OR: 11.167, CI: 3.895-32.019, P<0.001) and renal dysfunction (eGFR <60 ml/min/1.73 m^2) (OR: 2.497, CI: 1.325-4.705, P=0.008).

Discussion

IABP is used as the first-line treatment in AMI, with the goal of providing temporary mechanical support and allowing time for myocardial recovery. However, few randomized evidence has suggested the survival benefit of IABP treatment in AMI complicated by cardiogenic shock. And IABP is often incapable of overcoming hemodynamic compromise in severe refractory cardiogenic shock [11]. There remains an ongoing debate on the use of IABP in high-risk AMI patients who develop hemodynamic instability. It is suggested that timing of initiation of IABP therapy could be of great importance. Our study aimed to provide an insight into the clinical characteristics and risk factors of in-hospital death with IABP support in AMI patients.

Analyzing the data of in-hospital major adverse clinical events, we conclude that patients who

Table 4. In-hospital balloon-related complications adverse clinical events

	Non-survivors (n=81)	Survivors (n=491)	*P*
The complications			
IABP failure	2 (2.5)	0 (0)	0.02
Aortic dissection (%)	0 (0)	0 (0)	——
Systemic embolization (%)	0 (0)	2 (0.4)	0.565
Critical limb ischemia (%)	1 (1.2)	0 (0)	0.142
Thrombocytopenia (%)	6 (7.4)	3 (0.6)	<0.001
Access-site complication (%)	7 (8.6)	12 (2.4)	0.004
Withdraw IABP due to the complications	4 (4.9)	7 (1.4)	0.033
Major adverse clinical events			
Cardiogenic shock	68 (85.0)	84 (17.5)	<0.001
Mechanical complications	17 (21.0)	3 (0.6)	<0.001
Cerebral hemorrhage	1 (1.2)	0 (0)	0.142
Pulmonary embolism	1 (1.2)	3 (0.6)	0.463
VT/VF	34 (42)	96 (19.6)	<0.001
MODS	6 (7.4)	3 (0.6)	<0.001

VT/VF: Ventricular tachycardia/ventricular fibrillation. MODS: multiple organ dysfunction syndromes.

died in hospital were more frequently accompanied with CS and mechanical complications. Most of these patients have poor end-organ function which gets amplified during sustained hypoperfusion [12]. In addition, Patients with CS often develop into deleterious consequences of systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndromes (MODS) that ensues [13]. Previous studies have shown the occurrence of CS, mechanical complications, and ventricular arrhythmia often carries a grave prognosis [14-19]. Consequently, these life-threatening complications may still lead to the poor prognosis though intensive and multifaceted therapy including IABP is used.

It is noteworthy that significant differences in vital signs including blood pressure and heart rate response to IABP between the 2 groups. In the non-survivors, there was lesser increase of systolic blood pressure after IABP insertion. Considering the irreplaceable role of systolic blood pressure influencing the perfusion of coronary artery [20-23], this mild augmentation systolic blood pressure is correlated with poor in-hospital prognosis. Although heart rates decrease to some extent in both 2 groups during IABP support, the magnitude of heart rate in non-survivors is still compromised comparing to survivors. Although temporal improvements of hemodynamic parameters such as blood pressure and heart rate are observed, IABP cannot improve the circulatory collapse status for patients whose cardiac function is already severely damaged. Patients whose hemodynamics parameters have no significant response to IABP may derive less benefit from IABP support in terms of survival.

In our study, advanced age, prolonged time from symptom onset to FMC, diminished LVEF, Killip class III~IV and renal dysfunction (eGFR <60 ml/min/1.73 m^2) were considered to be independent risk factors associated with in-hospital mortality. Identification and attention to these risk factors might provide a mean of reducing in-hospital mortality. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction suggest that risk stratifications can provide an opportunity to integrate various patient characteristics and estimate a patient's prognosis [24]. Therefore, a better understanding of the prognostic effects of these risk factors associated with in-hospital death in AMI supported by IABP would be helpful for clinical decision-making and adequate timing of IABP insertion. In-hospital outcome may be improved by early application of IABP prior to the emergence of end-organ failure. In addition, the multivariate binary logistic regression didn't show admission SBP or HR was associated with increased risk of short-term deaths. This is presumably because some patients might be administered medication treatment that influenced the SBP and HR such

Table 5. Risk factors of in-hospital mortality

Multivariate logistic regression	Odds ratios	B	95% CI	*P*
Age >65 years	2.224	0.799	1.106-4.475	0.025
time from symptom onset to FMC	1.021	0.021	1.005-1.038	0.012
Killip class III-IV	11.167	2.413	3.895-32.019	<0.001
Renal dysfunction	2.497	0.915	1.325-4.705	0.008
LVEF <30%	3.043	1.113	1.117-8.288	0.029

FMC: first medical contact; LVEF: left ventricular ejection fraction.

as inotropic or vasoactive agents before admission to the medical center. Hemodynamic parameters were obtained at hospital admission may have not accurately reflected the declining status of the patient prior to balloon insertion.

The most frequent complications in our study were access-site complication and thrombocytopenia. Although the incidence of complications associated with IABP is similar to other studies [10, 25], it is still higher in the non-survivors than the survivors. Though most of the deaths were related to the primary disease process, there are some of deaths attributed to premature withdrawing of balloon due to intolerable complications to proceed with counterpulsation. We could presume that premature discontinuation of IABP's support may cause hemodynamics deterioration.

Emergency coronary angiography was performed in over 80% of the patients. However, the results showed a much lower percent of revascularization rate in the non-survivors than in the survivors. Consistently with the results of previous studies [26, 27], present study showed that the widespread use of primary PCI as reperfusion therapy may reduce the mortality following AMI. Previous studies have shown the short term outcome in AMI patients supporting with IABP is influenced by reperfusion method [28, 29]. In recent years, many clinical trials [30-33] have shown that reperfusion therapy, especially primary PCI, was superior to conservative treatment for improving left ventricular function, reducing the in-hospital mortality for patients with AMI. This suggests that IABP may be a more effective therapy modality when it is associated with effective revascularization. Without an effective reperfusion therapy to reduce the amount of myocardium at risk of irreversible damage, short term salvage strategies like IABP may not affect mortality. Therefore, it is of extreme significance for patients with AMI to receive timely and effective reperfusion therapy, which includes either thrombolysis or PCI. Also, the higher proportion of conservative treatment with medication alone in non-survivors group may also reflect a fact, that some of the sickest patients may have died before they could receive emergency revascularization therapy.

Limitation

The present study has some limitations. First, it is a retrospective observational analysis, the cohort included unselected, consecutive patients with a diagnosis of AMI supported by IABP, and ST-elevation myocardial infarction was not discriminated from non-ST-elevation myocardial infarction. The results might be attributed to some imbalances between the groups. Second, this is not a random and controlled experiment which might bring some bias. Third, data regarding clinical characteristics and results of emergency coronary angiography were not available for all study patients. Fourth, we did not have adequate data regarding underlying diseases in the patients and non-fatal major bleeding complications during the hospitalization.

Conclusion

IABP support may be more effective combined with revascularization for AMI patients whose hemodynamics is compromised. Patients accompanied with cardiogenic shock and other life-threatening complications may still have a poor prognosis though intensive and multifaceted therapy including IABP is used. Meanwhile, patient whose hemodynamics parameters have significant response to IABP may get benefits with IABP to improve in-hospital survival.

Disclosure of conflict of interest

None.

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