The Use of Intra-aortic Balloon Pump in a Real-World Setting: A Comparison between Survivors and Nonsurvivors from Acute Coronary Syndrome Treated with IABP. The Jakarta Acute Coronary Syndrome Registry

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Abstract

Real-world data on acute coronary syndrome (ACS) patients who received intra-aortic balloon pump (IABP) support are limited. The objective of this study was to evaluate the characteristics of ACS patients who received IABP support from a real-world ACS registry. Patients with ACS ($N = 121$) who received IABP support were enrolled. Characteristics of survivors and nonsurvivors were compared at 30 days. Mortality rate of patients with ACS who received IABP was 47%. The survivors ($N = 64$) had less often cardiogenic shock ($p < 0.001$), more often IABP usage as back-up for a revascularization procedure ($p = 0.002$), less often resuscitation ($p = 0.043$), and less mechanical ventilator support ($p < 0.001$) than nonsurvivors. The nonsurvivors had a significantly higher leukocyte count ($p = 0.033$), a higher serum creatinine level ($p < 0.001$), a higher blood sugar on admission ($p = 0.001$), higher creatine kinase MB levels ($p = 0.002$), and a higher serum uric acid level ($p < 0.001$), but significantly lower left and right ventricular function ($p = 0.014$ and $p = 0.003$, respectively) than survivors. At 30 days, non-ST elevation (STE)-ACS patients had lower mortality rate than ST segment elevation myocardial infarction patients (log-rank test, $p < 0.001$), and non-STE-ACS patients who had not suffered from cardiogenic shock showed the lowest mortality rate (log-rank test, $p < 0.001$). By multivariate analysis, a heart rate ≥ 100 beats per minute before IABP insertion was the strongest predictor of 30-day mortality (hazard ratio = 5.69; 95% confidence interval, 1.49 to 21.78; $p = 0.011$). In ACS patients presenting with either cardiogenic shock, resuscitated, or patients who needed mechanical ventilation suffered from high mortality, despite the use of IABP. IABP appears to be safe and tended to be favorable in noncardiogenic shock ACS patients, particularly non-STE-ACS. A heart rate of ≥ 100 beats per minute prior to IABP insertion was the strongest predictor of 30-day mortality.

Keywords

- IABP
- ACS
- survivor
- nonsurvivor

The intra-aortic balloon pump (IABP) was introduced nearly five decades ago,¹ as a simple but effective device to increase coronary perfusion, reduce afterload, and reduce myocardial work.^{2,3} Since then, IABP has become the most widely used form of mechanical circulatory support for patients with acute myocardial infarction (AMI) complicated by cardiogenic shock.

The American College of Cardiology and American Heart Association,⁴ and the American College of Cardiology Foundation/the American Heart Association/the Society for Cardiovascular Angiography and Interventions (ACCF/AHA/SCAI)⁵ practice guidelines strongly recommend supportive treatment with an IABP in patients with cardiogenic shock (class I), while the European Society of Cardiology (ESC) guidelines on ST segment elevation myocardial infarction (STEMI)⁶ now has given IABP a class IIb recommendation. However, the real utilization rate of IABP in STEMI patients complicated by cardiogenic shock is still low (20–39%),^{7,8} and the results from the recently published IABP-SHOCK II trial⁹ showed that the use of IABP in AMI patients complicated by cardiogenic shock does not improve survival as compared with medical therapy.

Those findings now strongly challenge the guidelines recommendations, thus more data are needed to establish the role of IABP in the daily management of acute coronary syndrome (ACS) patients. Furthermore, the body of evidence supporting IABP in the real-world ACS patients remains limited. Therefore, we studied the outcome of patients who received IABP treatment based on physician clinical judgment in our series of ACS patients from a real-world local registry, outside the setting of a randomized controlled trial, including patients with and without cardiogenic shock in whom an IABP was inserted.

Patients and Methods

This study was an observational retrospective analysis. Data were collected from the Jakarta Acute Coronary Syndrome registry in Intensive Cardiovascular Care Unit/ICVCU of National Cardiovascular Center Harapan Kita, Jakarta, Indonesia, from the period of August 2008 to July 2012, involving 121 consecutive patients with ACS in whom an IABP was used.

Inclusion criteria: All ACS patients (STEMI and non-ST elevation [STE]-ACS) presenting with and without cardiogenic shock, in whom an IABP was inserted and initial early revascularization was planned. The indications for IABP insertion in the noncardiogenic shock group were recurrent acute pulmonary edema despite optimal medical treatment, as an adjunct to complex revascularization procedures, recurrent prolonged chest pain despite optimal medical treatment, and persistent refractory malignant arrhythmia.

Intra-aortic Balloon Pump Insertion Technique

The IABP was inserted according to the standard technique. After local anesthesia with 10 mL of 2% lidocaine, the femoral artery was cannulated using a 17-G needle (modified Seldinger technique) and a 0.024-inch J-shaped guidewire was inserted and advanced until the level of the aortic arch, followed by a 7.5F or 8F introducer sheath placement. The

balloon was advanced gently over the guidewire into the descending aorta and the tip of the catheter was positioned 1 to 2 cm distal to the origin of the left subclavian artery, followed by guidewire removal. The catheter was flushed with a heparin solution when the catheter is in the standard position. The access site choice for insertion (left or right femoral artery) was left at the operator's discretion. After insertion, 5,000 IU bolus heparin was given in all patients. Choice of revascularization (percutaneous coronary intervention [PCI] on culprit lesion only, multivessel PCI or coronary artery bypass graft) was at the operator's discretion. Balloon size determination was done using height adjusted protocol: body height of 152 to 162 cm (34 mL) and 162 to 183 cm (40 mL).

IABP Initiation and Weaning Protocol

The balloon catheter was connected to the console and purged with helium. The central lumen of the catheter was connected to a pressure transducer and the pressure wave form was monitored.

Correct timing of balloon inflation and deflation during the cardiac cycle is vital for an optimal counterpulsation effect of IABP, the support was initiated using 1:1 electrocardiographic triggering (R wave triggered). However, the ratio was selected depending on the hemodynamic status. Auto pilot mode was used in most of the patients. The IABP weaning was achieved by reduction of trigger ratio every 1 to 4 hours, from 1:1 to 1:2 to 1:3. If 1:3 ratio was tolerated for 4 hours, the device was turned to standby mode and removed.

Study Endpoints

The endpoint was all cause mortality rate at 30-day follow-up. The blood pressure and heart rate before IABP insertion, hemoglobin, leukocyte, blood glucose, creatinine, uric acid and cardiac enzymes, echocardiography variables (left ventricular ejection fraction [LVEF] and right ventricular function), history of cardiopulmonary resuscitation, and mechanical ventilation support were also assessed as well as in-hospital bleeding events, stroke, acute limb ischemia, and/or vascular access site complication. Neither the Maquet nor Arrow Company had any involvement in the study.

Definitions

Cardiogenic shock was defined as systolic blood pressure less than 90 mm Hg for more than 30 minutes or need for infusion of catecholamines/inotropics to maintain a systolic pressure above 90 mm Hg with signs of tissue hypoperfusion such as cold extremities, clammy skin and extremities, and/or oliguria with urine output of less than 30 mL per hour.¹⁰

LVEF was measured with two-dimensional echocardiography using the modified Simpson's rule,¹¹ and right ventricular function was measured by ultrasound using tricuspid annular plane systolic excursion (TAPSE).¹²

In-hospital bleeding criteria were defined according to the Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries (GUSTO) criteria¹³ for classifying the severity of bleeding complications and described as (1) Severe

or life-threatening bleeding: Intracranial bleeding or bleeding that causes substantial hemodynamic compromise requiring treatment; (2) Moderate bleeding: Bleeding which needs blood transfusion; (3) Minor bleeding: Other bleeding, neither requiring transfusion nor causing hemodynamic compromise.

Stroke was defined as new ischemic stroke event (rapidly developed clinical signs of focal or global disturbance of cerebral function lasting more than 24 hours) occurring during the observation period.¹⁴

Acute limb ischemia was defined as sudden decrease in limb perfusion which could threaten limb viability, classified as type I (viable), type IIa (marginally threatened), type IIb (immediately threatened), and type III (irreversible). Duplex ultrasonography of the lower limb was used to confirm the diagnosis.¹⁵

Vascular access site complication was defined as any puncture site-related bleeding (at the IABP insertion site) and/or hematoma ≥ 5 cm in diameter at the IABP insertion site.

Cardiac Biomarkers

Creatine kinase-MB (CK-MB) level in plasma was measured by immunoinhibition assay (Roche Hitachi 912, Mannheim, Germany) after arrival in the emergency department and 8–12 hours after admission. Serum troponin T concentration was measured with the cTnT assay from Roche Diagnostics (Mannheim, Germany) that has a 99th percentile of a healthy reference population of < 0.01 ng/mL.

Statistical Analysis

Continuous variables are presented as mean values $\pm$ standard deviation or median (minimum to maximum). Categorical variables are expressed as percentages or proportional. Normally distributed numerical variables were compared by *t* test, skewed distribution data by Mann–Whitney *U* test, and categorical variables by Pearson chi-square test or Fisher exact test.

Multivariate analysis was performed by Cox regression to identify the predictor(s) of 30-day mortality in all patients. Cumulative survival was assessed by Kaplan–Meier method. Differences between the two groups were analyzed using log-rank test. A *p* value of < 0.05 was regarded as a statistically significant. All computations were performed using a statistical package.

Results

Characteristics of Patients

The median age of the patients was 58 years ranging from 30 to 85 years, and most of them (91%) were male. STEMI was diagnosed in 51% and non-STE-ACS in 49% of patients. Baseline characteristic of all patients are seen in ►Table 1.

Fifty-three patients (44%) presented with cardiogenic shock and 56% were patients without cardiogenic shock. ►Table 2 shows the differences of clinical characteristics and indications for IABP insertion between survivors and nonsurvivors.

Table 1 Baseline characteristic of all patients

Variables	All patients (N = 121)
Demographic characteristics	
Age, years	58 (30–85)
Male gender	110 (91%)
Body surface area, m ²	1.75 (1.37–2.48)
Risk factors	
Hypertension	68 (56%)
Dyslipidemia	60 (49%)
DM	54 (45%)
Current smoking	81 (67%)
Cardiac history	
Previous MI	63 (52%)
Previous PCI	26 (21%)
Previous CABG	1 (0.8%)
Clinical presentation	
STEMI	62 (51%)
Non-STEMI	44 (36%)
UAP	15 (12%)
Cardiogenic shock	53 (44%)
Procedure during hospitalization	
Coronary angiography	108 (89%)
Primary PCI	17 (14%)
Fibrinolytic therapy	3 (2.5%)
30-day mortality	57 (47%)

Abbreviations: CABG, coronary artery bypass graft; DM, diabetes mellitus; MI, myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST elevation myocardial infarction; UAP, unstable angina pectoris.

Mechanical complications occurred in two patients, and both presented with cardiogenic shock. The survivors (*N* = 64) had a higher proportion of noncardiogenic shock (*p* = < 0.001) and IABP was more often used as an adjunct to complex revascularization procedures (*p* = 0.002). Survivors had both less history of resuscitation (*p* = 0.043) and mechanical ventilator support (*p* = < 0.001) compared with nonsurvivors.

Most of the patients had triple vessel coronary artery disease involvement (►Table 2), including the cardiogenic shock patients. The characteristics of the cardiogenic shock patients are depicted in ►Table 3.

Antiplatelet and Anticoagulation Regimens

The antiplatelet and anticoagulation regimens were similar between the survivors and nonsurvivors group. All patients received 160 mg salicylic acid and 300 mg loading dose of clopidogrel on admission. For patients who underwent PCI, a loading dose of 600 mg clopidogrel was given and intravenous unfractionated heparin was administered (100 or 50–60 IU/kg if GPIIb/IIIa was used). Enoxaparin, fondaparinux, and unfractionated heparin were given in 20 to 31% of patients (►Table 2).

Table 2 Clinical characteristics between survivors and nonsurvivors

	Survivors (N = 64)	Nonsurvivors (N = 57)	p value
Demographic characteristics			
Age, years	55 (30–85)	61 (36–75)	NS
Male gender	58 (91%)	52 (91%)	NS
Body surface area, m ²	1.75 (1.38–2.32)	1.75 (1.37–2.48)	NS
Risk factors			
Hypertension	35 (55%)	33 (58%)	NS
Dyslipidemia	35 (55%)	25 (44%)	NS
DM	26 (41%)	28 (49%)	NS
Current smoking	41 (64%)	40 (70%)	NS
Cardiac history			
Previous MI	35 (55%)	28 (49%)	NS
Clinical presentation			
STEMI	24 (37%)	38 (67%)	0.001
Cardiogenic shock	17 (26%)	36 (63%)	< 0.001
Location of MI			
Anterior	20 (31%)	26 (46%)	NS
Indication for IABP treatment			
Cardiogenic shock	17 (26%)	36 (63%)	< 0.001
Recurrent pulmonary edema	5 (8%)	3 (5%)	NS
Prolonged chest pain	7 (11%)	3 (5%)	NS
Refractory malignant arrhythmia	4 (6%)	3 (5%)	NS
Adjunct to complex revascularization	31 (48%)	12 (21%)	0.002
Mechanical complication			
Interventricular septal rupture	0 (0)	2 (3%)	NS
Blood pressure, mm Hg			
Systolic	113 (70–191)	114 (70–160)	NS
Diastolic	73 (43–113)	70 (37–100)	NS
Heart rate on admission, beats per minute	93 (51–146)	98 (50–168)	NS
Laboratory parameter			
Hemoglobin, g/dL	13 ± 2.1	14 ± 2.0	NS
Leukocyte count/μL	10,565 (3,900–62,380)	12,500 (6,190–25,200)	0.033
Creatinine level, mg/dL	1.10 (0.5–11)	1.6 (0.8–10)	<0.001
Calculate CCT	69 (5.9–178)	42 (4.8–133)	<0.001
Random blood glucose, mg/dL	144 (82–554)	194 (72–488)	0.001
Initial CK-MB, U/L	30 (2–628)	47 (3–1,965)	NS
CK-MB after 8–12 h, U/L	42 (9–724)	136 (7–1,388)	0.002
Initial troponin T, ng/mL	0.5 (0.01–16)	1.1 (0.03–25)	NS
Uric acid, mg/dL	6.1 (2.6–11)	8.1 (3.5–15.4)	< 0.001
Echocardiography parameters			
LVEF, %	37 (14–70)	30 (15–76)	0.014
TAPSE, cm	1.9 (1.1–2.6)	1.6 (0.6–2.7)	0.003
Cardiopulmonary resuscitation	6 (9%)	13 (23%)	0.043
Mechanical ventilator support	15 (23%)	39 (68%)	< 0.001

Table 2 (Continued)

	Survivors (N = 64)	Nonsurvivors (N = 57)	p value
Coronary angiography	62 (97%)	46 (81%)	0.004
Revascularization			
PCI	28 (44%)	29 (51%)	NS
CABG	27 (42%)	7 (12%)	< 0.001
Manual thrombectomy	9/62 (14%)	7/46 (15%)	NS
Number of diseased vessel			
1	8/62 (13%)	2/46 (4%)	NS
2	7/62 (11%)	11/46 (24%)	NS
3	47/62 (76%)	33/46 (72%)	NS
Culprit vessel			
LAD	59/62 (95%)	45/46 (98%)	NS
LCX	48/62 (77%)	39/46 (85%)	NS
RCA	49/62 (79%)	36/46 (78%)	NS
LM	25/62 (40%)	16/46 (35%)	NS
Anticoagulation			
Unfractionated heparin	16 (25%)	10 (17%)	NS
Enoxaparin	22 (34%)	16 (28%)	NS
Fondaparinux	15 (23%)	10 (17%)	NS

Abbreviations: CABG, coronary artery bypass graft; CCT, calculated clearance test; CK-MB, creatine kinase MB isoform; DM, diabetes mellitus; IABP, intra-aortic balloon pump; LAD, left anterior descending artery; LCX, left circumflex artery; LM, left main artery; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NS, not significant; PCI, percutaneous coronary intervention; RCA, right coronary artery; STEMI, ST elevation myocardial infarction; TAPSE, tricuspid annular plane systolic excursion.

Table 3 Characteristics of cardiogenic shock patients treated with IABP

	Cardiogenic shock patients (N = 53)
Age, years	56 (30–72)
Risk factors	
Hypertension	29 (57%)
Dyslipidemia	19 (36%)
DM	24 (45%)
Current smoking	36 (68%)
STEMI	38 (72%)
Heart rate, beats per minute	100 (50–168)
Creatinine level, mg/dL	1.50 (0.70–10)
LVEF, %	30 (15–76)
Mechanical complication	2 (4%)
Triple vessel coronary artery disease	33/44 (75%)

Abbreviations: DM, diabetes mellitus; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction; STEMI, ST elevation myocardial infarction.

Study Endpoints

At 30 days follow-up, the overall mortality of the study population was 47%. Compared with survivors, the nonsurvivors had a significantly higher leukocyte counts ($p = 0.033$), higher serum creatinine levels ($p < 0.001$), higher blood sugar on admission ($p = 0.001$), higher creatine kinase MB (CK-MB) levels ($p = 0.002$), and higher serum uric acid levels ($p < 0.001$).

Echocardiography showed a significant lower LVEF ($p = 0.014$) and TAPSE ($p = 0.003$) in the nonsurvivors compared with survivors (► **Table 2**).

There was no difference in in-hospital bleeding and stroke rate, but four cases of acute limb ischemia were found in the nonsurvivors (► **Table 4**). After multivariate analysis, a heart rate ≥ 100 beats per minute before IABP insertion demonstrated to be the strongest predictor of 30-day mortality (hazard ratio = 5.69; 95% confidence interval, 1.49–21.78; $p = 0.011$) (► **Table 5**).

Survival Functions

At 30 days, the non-STE-ACS patients had lower mortality rate than STEMI patients (log-rank test, $p < 0.001$) (► **Fig. 1A**). Patients with cardiogenic shock, either STEMI or non-STE-ACS, had a significantly higher mortality rate than noncardiogenic STEMI or non-STE-ACS patients (log-rank test, $p < 0.001$). Patients with the lowest mortality were non-STE-ACS patients without

Table 4 Safety endpoints of the study

	Survivors (N = 64)	Nonsurvivors (N = 57)	p value
In-hospital bleeding ^a			
Severe or life threatening	1 (1.5%)	5 (9%)	NS
Moderate	13 (20%)	7 (12%)	NS
Minor	6 (9%)	10 (17%)	NS
Stroke	1 (1.5%)	1 (2%)	NS
Acute limb ischemia	0 (0)	4 (7%)	0.046
Vascular access site complication	2 (3%)	3 (5%)	NS

Abbreviation: NS, not significant.

^aIn-hospital bleeding was according to GUSTO criteria.

cardiogenic shock (►Fig. 1B). Finally, patients with a heart rate $\geq$ 100 beats per minute prior to IABP insertion had a higher mortality rate than the patients with a heart rate $<$ 100 beats per minute (log-rank test, $p < 0.001$). (►Fig. 1C).

Discussion

In the real world, IABP is used not only for patients presenting with cardiogenic shock complicating an AMI but also in other variety of ACS conditions such as refractory ventricular failure, cardiac support for high-risk general surgery patients, refractory unstable angina,¹⁶ refractory malignant arrhythmia despite optimal medical treatment,¹⁷ adjunct to revascularization,^{18,19} and/or bridging to a heart transplant.²⁰

The important findings from this study are despite the lack of support for IABP treatment in cardiogenic shock patients, we observed a favorable safety profile and possibly a good performance of IABP treatment in conditions such as (1) noncardiogenic shock ACS; (2) IABP as an adjunct to revascularization procedures; (3) patients without resuscitation; and (4) patients without mechanical ventilator support. As expected, we also found that non-STE-ACS patients had better 30 days survival compared with STEMI patients.

IABP in Cardiogenic Shock Patients

The incidence of cardiogenic shock complicating an AMI is around 7.2%^{21,22} and is associated with high mortality (50–80%).²³ More recently, some randomized studies and

Table 5 Multivariate predictors of 30-day mortality of ACS patients treated with IABP

Variables	HR	95% CI	p value
Age ($>$ 65 years)	0.85	0.36–2.03	0.722
Male gender	1.15	0.29–4.48	0.844
Risk factors			
Diabetes mellitus	3.19	1.01–10.08	0.048
Hypertension	3.11	1.31–7.42	0.01
Dyslipidemia	0.47	0.16–1.36	0.163
Current smoking	1.79	0.63–5.04	0.272
Prior myocardial infarction	1.35	0.52–3.56	0.537
Anterior wall infarction	1.12	0.46–2.71	0.807
Killip class IV	1.1	0.49–2.48	0.813
Systolic pressure $<$ 90 mm Hg	0.58	0.23–1.45	0.245
Heart rate $\geq$ 100x/minute	5.69	1.49–21.78	0.011
Creatinine level $>$ 1.5 mg/dL	1.31	0.67–2.55	0.427
Leukocyte count $>$ 11,000/ μ L	0.97	0.41–2.36	0.963
Blood glucose $\geq$ 200 mg/dL	0.87	0.37–2.04	0.751
LVEF $<$ 35%	1.7	0.83–3.48	0.144
Triple vessel diseased	0.64	0.29–1.39	0.257

Abbreviations: CI, confidence interval; HR, Hazard ratio; LVEF, left ventricular ejection fraction.

Note: The variables were tested for presence or absence unless stated otherwise.

Fig. 1 Kaplan–Meier survival curve for all cause of death at 30 days among patients with: STEMI and non-STE-ACS (A); cardiogenic shock STEMI/non-STE-ACS and noncardiogenic shock STEMI/non-STE-ACS (B); and in patients with higher and lower heart rate prior to IABP insertion (C). ACS, acute coronary syndrome; IABP, intra-aortic balloon pump; STE, ST segment elevation; STEMI, ST segment elevation myocardial infarction.

registries reported a mortality of 42 to 48%,^{24,25} despite emerging innovative treatment modalities. The difference in mortality is due to the difference in cardiogenic shock criteria used in each of the trials. This high mortality rate has lead most IABP trials to enroll patients with cardiogenic shock.

The mortality rate of the cardiogenic shock patients in our study was higher than the mortality rate in the IABP group of IABP SHOCK II trial⁹ (63 vs. 39.7%). Several explanations may be plausible to explain the differences. Compared with the IABP group in the IABP SHOCK II trial, our study population (the cardiogenic shock group) had a higher risk profile—higher proportion of diabetes mellitus (45 vs. 35%), higher median heart rate (100 vs. 92 beats per minute), higher median creatinine level (1.5 vs. 1.3 mg/dL), more patients with triple vessel disease (75 vs. 52%), and lower LVEF (30 vs. 35%).

It has been proven that diabetes mellitus,²⁶ heart rate,²⁷ creatinine level,²⁸ triple vessel disease,²⁹ and low LVEF³⁰ are associated with high mortality rates in patients with ACS. Moreover, patients with mechanical complications were included in our study. On the contrary, the IABP SHOCK II trial has more patients with a history of resuscitation compared with our study (45 vs. 16%).

The survival curves showed that patients who might benefit the most from IABP treatment are possibly the non-STE-ACS patients, particularly noncardiogenic shock non-STE-ACS patients, who had the highest cumulative survival compared with noncardiogenic shock STEMI, cardiogenic shock non-STE, and cardiogenic shock STEMI patients. These findings deserve further investigation by means of a multicenter randomized trial to establish the true efficacy and safety in such populations.

Survivor versus Nonsurvivor Patients

We observed that nonsurvivor patients more often presented with cardiogenic shock with lower LVEF and lower TAPSE compared with the survivors. The higher CK-MB levels after 8–12 hours found in the nonsurvivors and its relation to the lower ventricular functions need to be investigated further, although CK-MB level is associated with extensiveness of a myocardial infarction. Moreover, higher leukocyte count, creatinine, and uric acid levels in the nonsurvivors might explain the higher risk profile of this group, and this merits further investigation as well. However, the safety profile between the two groups was not different, except for the four patients with acute limb ischemia found in the nonsurvivors group, which may reflect their more serious baseline disease.

Predictor of 30-Day Mortality

After multivariate analysis, we found that a heart rate ≥ 100 beats per minute before IABP insertion was a strong predictor of 30-day mortality. Morrow et al also showed the importance of heart rate as a variable in predicting 30-day mortality in STEMI patients.²⁷ The potential pathophysiological mechanism through which higher heart rate adversely affects mortality remains unclear, but the significance of

sympathetic blockade and reduced infarct size may explain the benefit of lowering heart rate.³¹ Thus, the result of present study could alert us to manage patients with higher heart rate on admission (≥ 100 beats per minute) more aggressively.

The result of a meta-analysis³² and the IABP SHOCK II trial⁹ showed no benefit of IABP in AMI patients with cardiogenic shock, which seems to be reflected in our study. Thus, the ACCF/AHA/SCAI and the AHA guidelines on IABP treatment in cardiogenic shock patients may be challenged. Importantly, it should be noted that in the real-world practice, the use of IABP is not restricted to cardiogenic shock patients complicating an AMI. Randomized studies are needed to investigate the efficacy of IABP treatment in noncardiogenic shock ACS patients.

Limitation

This study was designed as an observational retrospective analysis, with its inherent limitations.

Conclusion

In ACS patients presenting with cardiogenic shock, resuscitated or with need for mechanical ventilation, the short-term mortality remains high, despite the use of IABP. IABP appears to be safe and possibly favorable in noncardiogenic shock ACS patients particularly in patients presenting with non-STE-ACS. A heart rate of ≥ 100 beats per minute before IABP insertion was the strongest predictor of 30-day mortality.

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References

- 1 Kantrowitz A, Tjonneland S, Freed PS, Phillips SJ, Butner AN, Sherman JL Jr. Initial clinical experience with intraaortic balloon pumping in cardiogenic shock. *JAMA* 1968;203(2):113–118

- 2 Weber KT, Janicki JS. Intraaortic balloon counterpulsation. A review of physiological principles, clinical results, and device safety. *Ann Thorac Surg* 1974;17(6):602-636
- 3 Scheidt S, Wilner G, Mueller H, et al. Intra-aortic balloon counterpulsation in cardiogenic shock. Report of a co-operative clinical trial. *N Engl J Med* 1973;288(19):979-984
- 4 Antman EM, Anbe DT, Armstrong PW, et al; American College of Cardiology; American Heart Association Task Force on Practice Guidelines; Canadian Cardiovascular Society. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction). *Circulation* 2004;110(9):e82-e292
- 5 Levine GN, Bates ER, Blankenship JC, et al; ACCF; AHA; SCAI. 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions. *Catheter Cardiovasc Interv* 2012;79(3):453-495
- 6 Steg PG, James SK, Atar D, et al; Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC). ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *Eur Heart J* 2012;33(20):2569-2619
- 7 Babaev A, Frederick PD, Pasta DJ, Every N, Sichrovsky T, Hochman JS; NRMI Investigators. Trends in management and outcomes of patients with acute myocardial infarction complicated by cardiogenic shock. *JAMA* 2005;294(4):448-454
- 8 Goldberg RJ, Samad NA, Yarzebski J, Gurwitz J, Bigelow C, Gore JM. Temporal trends in cardiogenic shock complicating acute myocardial infarction. *N Engl J Med* 1999;340(15):1162-1168
- 9 Thiele H, Zeymer U, Neumann FJ, et al; IABP-SHOCK II Trial Investigators. Intraaortic balloon support for myocardial infarction with cardiogenic shock. *N Engl J Med* 2012;367(14):1287-1296
- 10 Forrester JS, Diamond G, Chatterjee K, Swan HJ. Medical therapy of acute myocardial infarction by application of hemodynamic subsets (second of two parts). *N Engl J Med* 1976;295(25):1404-1413
- 11 Lang RM, Bierig M, Devereux RB, et al; Chamber Quantification Writing Group; American Society of Echocardiography's Guidelines and Standards Committee; European Association of Echocardiography. Recommendations for chamber quantification. *J Am Soc Echocardiogr* 2005;18(12):1440-1463
- 12 Bleeker GB, Steendijk P, Holman ER, et al. Assessing right ventricular function: the role of echocardiography and complementary technologies. *Heart* 2006;92(Suppl 1):i19-i26
- 13 The GUSTO Investigators. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. The GUSTO investigators. *N Engl J Med* 1993;329(10):673-682
- 14 MONICA criteria: WHO Monica Project: MONICA manual. Part IV: Event registration. Available at: <http://www.ktl.fi/publications/monica/manual/part4/iv-2.htm#s1-1>
- 15 Hirsch AT, Haskal ZJ, Hertzner NR, et al; American Association for Vascular Surgery; Society for Vascular Surgery; Society for Cardiovascular Angiography and Interventions; Society for Vascular Medicine and Biology; Society of Interventional Radiology; ACC/AHA Task Force on Practice Guidelines Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease; American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; Vascular Disease Foundation. ACC/AHA 2005 Practice guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic). *Circulation* 2006;113(11):e463-e654
- 16 Ferguson JJ III, Cohen M, Freedman RJ Jr, et al. The current practice of intra-aortic balloon counterpulsation: results from the Benchmark Registry. *J Am Coll Cardiol* 2001;38(5):1456-1462
- 17 Fotopoulos GD, Mason MJ, Walker S, et al. Stabilization of medically refractory ventricular arrhythmia by intra-aortic balloon counterpulsation. *Heart* 1999;82(1):96-100
- 18 Perera D, Stables R, Thomas M, et al; BCIS-1 Investigators. Elective intra-aortic balloon counterpulsation during high-risk percutaneous coronary intervention: a randomized controlled trial. *JAMA* 2010;304(8):867-874
- 19 Rubino AS, Onorati F, Santarpino G, et al. Early intra-aortic balloon pumping following perioperative myocardial injury improves hospital and mid-term prognosis. *Interact Cardiovasc Thorac Surg* 2009;8(3):310-315
- 20 Cochran RP, Starkey TD, Panos AL, Kunzelman KS. Ambulatory intraaortic balloon pump use as bridge to heart transplant. *Ann Thorac Surg* 2002;74(3):746-751, discussion 751-752
- 21 The GISSI Investigators. Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI). *Lancet* 1986;1(8478):397-402
- 22 Van de Werf F; The International Study Group. In-hospital mortality and clinical course of 20,891 patients with suspected acute myocardial infarction randomised between alteplase and streptokinase with or without heparin. *Lancet* 1990;336(8707):71-75
- 23 Hochman JS, Boland J, Sleeper LA, et al; SHOCK Registry Investigators. Current spectrum of cardiogenic shock and effect of early revascularization on mortality. Results of an International Registry. *Circulation* 1995;91(3):873-881
- 24 Goldberg RJ, Spencer FA, Gore JM, Lessard D, Yarzebski J. Thirty-year trends (1975 to 2005) in the magnitude of, management of, and hospital death rates associated with cardiogenic shock in patients with acute myocardial infarction: a population-based perspective. *Circulation* 2009;119(9):1211-1219
- 25 Alexander JH, Reynolds HR, Stebbins AL, et al; TRIUMPH Investigators. Effect of tilarginine acetate in patients with acute myocardial infarction and cardiogenic shock: the TRIUMPH randomized controlled trial. *JAMA* 2007;297(15):1657-1666
- 26 Gandhi GY, Roger VL, Bailey KR, Palumbo PJ, Ransom JE, Leibson CL. Temporal trends in prevalence of diabetes mellitus in a population-based cohort of incident myocardial infarction and impact of diabetes on survival. *Mayo Clin Proc* 2006;81(8):1034-1040
- 27 Morrow DA, Antman EM, Charlesworth A, et al. TIMI risk score for ST-elevation myocardial infarction: A convenient, bedside, clinical score for risk assessment at presentation: An intravenous nPA for treatment of infarcting myocardium early II trial substudy. *Circulation* 2000;102(17):2031-2037
- 28 Granger CB, Goldberg RJ, Dabbous O, et al; Global Registry of Acute Coronary Events Investigators. Predictors of hospital mortality in the global registry of acute coronary events. *Arch Intern Med* 2003;163(19):2345-2353
- 29 Sorajja P, Gersh BJ, Cox DA, et al. Impact of multivessel disease on reperfusion success and clinical outcomes in patients undergoing primary percutaneous coronary intervention for acute myocardial infarction. *Eur Heart J* 2007;28(14):1709-1716
- 30 Taylor GJ, Humphries JO, Mellits ED, et al. Predictors of clinical course, coronary anatomy and left ventricular function after recovery from acute myocardial infarction. *Circulation* 1980;62(5):960-970
- 31 Singh BN. Increased heart rate as a risk factor for cardiovascular disease. *Eur Heart J* 2003;24:G3-G9
- 32 Sjauw KD, Engström AE, Vis MM, et al. A systematic review and meta-analysis of intra-aortic balloon pump therapy in ST-elevation myocardial infarction: should we change the guidelines? *Eur Heart J* 2009;30(4):459-468