CLINICAL STUDY

Clinical Factors Associated with In-Hospital Mortality in Patients with Acute Myocardial Infarction Who Required Intra-Aortic Balloon Pumping

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Summary

Recent guidelines do not recommend the routine use of intra-aortic balloon pumping (IABP) for patients with cardiogenic shock. However, IABP support is still selected for acute myocardial infarction (AMI) in clinical practice because an Impella device did not show superiority over IABP and the mortality of AMI with cardiogenic shock is still high. This study aimed to find factors associated with in-hospital mortality in patients with AMI who required IABP support. Overall, 104 patients with AMI who required IABP support were included as the study population. Of 104 patients, in-hospital death was observed in 19 (18.3%). Multivariate stepwise logistic regression analysis was performed to investigate the determinants of in-hospital death. Shock, resuscitation, estimated glomerular filtration rate (eGFR), pre-systolic blood pressure of IABP insertion, multivessel disease, fluoroscopy time, initial lactic acid dehydrogenase levels, and timing of IABP support were included as independent variables. Shock (OR 25.27, 95% CI 3.26-196.11, \(P = 0.002\)) was significantly associated with in-hospital death after controlling other covariates, whereas eGFR (every 10 mL/minute/1.73 m² increase: OR 0.65, 95% CI 0.51-0.82, \(P < 0.001\)) and pre-percutaneous coronary intervention (pre-PCI) insertion of IABP (versus on-PCI insertion of IABP: OR 0.06, 95% CI 0.008-0.485, \(P = 0.008\)) were inversely associated with in-hospital death. In conclusion, shock was significantly associated with in-hospital death, whereas eGFR and pre-PCI insertion of IABP were inversely associated with in-hospital death in patients with AMI who received IABP support. Pre-PCI insertion of an IABP catheter might be associated with better survival in AMI patients who potentially require IABP support.

Key words: Percutaneous coronary intervention, Cardiogenic shock, In-hospital death

Although the mortality due to acute myocardial infarction (AMI) has decreased with the development of multidisciplinary therapy including primary percutaneous coronary intervention (PCI), the mortality due to AMI with cardiogenic shock (CS) is still high. Intra-aortic balloon pumping (IABP) has been used for AMI with CS for decades. However, recent guidelines do not recommend routine use of IABP for patients with CS because a randomized trial (IABP-SHOCK II trial) could not show the benefit of IABP for AMI with CS. While an Impella device (Abiomed, Danvers, MA, USA) was expected to improve the clinical outcomes of patients with CS, a recent study that compared the 30 day mortality for AMI with CS could not show the superiority over IABP. Furthermore, long-term follow-up data of the IABP-SHOCK II trial revealed that two-thirds of patients with CS died despite contemporary treatment with revascularization therapy.

Nevertheless, IABP is still selected for AMI in clinical practice because an Impella device did not show the superiority over IABP and the mortality of AMI with CS is still high. It should be important to investigate the determinants of death in patients with AMI who required IABP support, which would help to select the appropriate patients who receive a clinical benefit from IABP support. This study aimed to find factors associated with in-hospital mortality in patients with AMI who required IABP support.

Methods

Study patients: We reviewed all PCI records from January 2014 to December 2017. The inclusion criterion was PCI during the study period. The exclusion criteria were (1) PCI to the non-AMI lesions and (2) PCI without IABP support. Thus, patients with consecutive AMI who underwent PCI with IABP support were included in the study population. Then, those study patients were divided...
into the survivor group and the in-hospital death group according to death in the index admission. The patient characteristics, including medical history, laboratory data, procedure of PCI, and timing of IABP support, were collected from our medical records, and those characteristics were compared between the survivor group and the in-hospital death group. This study was approved by the Institutional Review Board, and written informed consent was waived because of the retrospective study design.

**Definition:** In the present study, AMI was defined as persistent chest pain, elevation of cardiac enzyme (>99th percentile of troponin I value or elevated creatine kinase > twice the upper limit of normal value) and ST segment elevation or depression in electrocardiograms compatible with AMI. Dyslipidemia was defined as a total cholesterol level 220 mg/dL and over, a low-density lipoprotein cholesterol level 140 mg/dL and over, or treatment for dyslipidemia. Diabetes mellitus was defined as hemoglobin A1c level 6.5% and over or medical treatment with anti-hyperglycemia medicines.

We calculated the estimated glomerular filtration rate (eGFR) from the serum creatinine levels, age, weight, and gender using the following formula: eGFR = 194 × Cr−1.094 × age−0.287 (male) and eGFR = 194 × Cr−1.094 × age−0.287 × 0.739 (female). Shock was defined as either systolic blood pressure under 90 mmHg or status requiring vasopressor at emergency room. Diagnostic ST elevation was defined as new ST elevation at J point in at least two contiguous leads of 2 mm (0.2 mV). Multi-vessel disease was defined as more than one vessel having >75% stenosis. Regarding the timing of the insertion of IABP catheter, pre-PCI insertion was defined as the timing when an IABP catheter was inserted before PCI. On-PCI insertion was defined as the timing when an IABP catheter was inserted during PCI.

**Statistical analysis:** Data were expressed as mean ± SD or percentage. Categorical variables were presented as numbers (percentage) and were compared using the chi-square test. The Shapiro-Wilk test was performed to determine if the continuous variables were normally distributed. Normally distributed continuous variables were compared between the groups using an unpaired Student’s t-test. Otherwise, continuous variables were using a Mann-Whitney U test. Multivariate logistic regression analysis using backward elimination stepwise methods was applied to investigate the determinants of in-hospital death. In-hospital death was adopted as a dependent variable. Independent variables were selected from variables that had marginal difference (P < 0.10) between the survival group and the in-hospital death group. If several variables were closely related to each other, only one variable was entered in the model.

**Results**

Overall, 2723 patients received PCI during the study period. Among them, 1776 PCI to non-AMI lesions were excluded. Furthermore, 843 PCI to AMI lesions without IABP support were excluded. Finally, 104 patients were included as the final study population and were divided into the survivor group (n = 85) and the in-hospital death group (n = 19) (Figure).

Table I shows the comparison of patient characteristics between the two groups. The prevalence of shock was significantly higher in the in-hospital death group (84.2%) than the survivor group (38.8%) (P < 0.001). Cardiopulmonary resuscitation was more frequently performed in the in-hospital death group (47.4%) than the survivor group (21.2%) (P = 0.004). eGFR was significantly lower in the in-hospital death group (26.4 ± 8.4 mL/minute/1.73 m²) than the survivor group (60.2 ± 5.2 mL/minute/1.73 m²) (P < 0.001).

Table II shows the comparison of lesion characteristics between the two groups. The prevalence of multi-vessel disease was significantly higher in the in-hospital death group (94.7%) than the survivor group (75.3%) (P = 0.049). Table III shows the comparison of procedural characteristics between the two groups. Systolic blood pressure before the insertion of IABP catheter was significantly lower in the in-hospital death group (96 ± 13 mmHg) than the survivor group (114 ± 6 mmHg) (P = 0.003). Pre-PCI insertion of IABP tended to be lower in the in-hospital death group (63.1%) than the survivor group (82.3%) (P = 0.066). The indication for IABP support between pre-PCI insertion (n = 82) and on-PCI insertion (n = 22) was also compared, as shown in Table IV. The prevalence of shock as the indication for IABP support was similar between pre-PCI insertion of IABP (42.7%) and on-PCI insertion of IABP (45.5%).

Multivariate logistic regression analysis using backward elimination stepwise methods was performed to investigate the determinants of in-hospital death (Table V). Since using catecholamine, veno-arterial extracorporeal membrane oxygenation (V-A ECMO), access site, and shock were closely related to each other, we only adopted shock as the variable. Since creatinine levels were closely related to eGFR, we also included eGFR in the model.
Table I. Comparison of Patient Characteristics between the Survivor Group and In-Hospital Death Group

|                                | All (n = 104) | Survivor group (n = 85) | In-hospital death group (n = 19) | P value |
|--------------------------------|---------------|-------------------------|---------------------------------|---------|
| Age, years                     | 70 ± 2        | 70 ± 2                  | 73 ± 5                          | 0.372   |
| Male sex, n (%)                | 84 (80.8)     | 69 (81.2)               | 15 (78.9)                       | 0.824   |
| Body mass index, kg/m²         | 23.5 ± 0.6    | 23.0 ± 1.4              | 23.6 ± 3.6                      | 0.456   |
| Hypertension, n (%)            | 73 (70.2)     | 60 (70.6)               | 13 (68.4)                       | 0.852   |
| Dyslipidemia, n (%)            | 53 (50.1)     | 46 (54.1)               | 7 (36.8)                        | 0.173   |
| Diabetes mellitus, n (%)       | 47 (45.2)     | 37 (43.5)               | 10 (52.6)                       | 0.471   |
| Current smoker, n (%)          | 23 (22.1)     | 18 (21.2)               | 5 (26.3)                        | 0.76    |
| Previous old myocardial infarction, n (%) | 14 (13.5) | 10 (11.8) | 4 (21.1) | 0.281 |
| Previous PCI, n (%)            | 15 (14.4)     | 12 (14.1)               | 3 (15.8)                        | 1.000   |
| Previous CABG, n (%)           | 4 (3.8)       | 2 (2.4)                 | 2 (10.5)                        | 0.152   |
| Hemodialysis, n (%)            | 7 (6.7)       | 4 (4.7)                 | 3 (15.8)                        | 0.112   |
| Shock, n (%)                   | 49 (47.1)     | 33 (38.8)               | 16 (84.2)                       | <0.001  |
| Using catecholamine, n (%)     | 24 (25.0)     | 16 (18.8)               | 10 (52.6)                       | 0.006   |
| V-A ECMO, n (%)                | 17 (16.5)     | 8 (9.4)                 | 9 (47.4)                        | <0.001  |
| Cardiopulmonary resuscitation, n (%) | 27 (26.0) | 18 (21.2) | 9 (47.4) | 0.039 |

Laboratory data

|                               | All (n = 104) | Survivor group (n = 85) | In-hospital death group (n = 19) | P value |
|--------------------------------|---------------|-------------------------|---------------------------------|---------|
| Hemoglobin, g/dL               | 13.2 ± 0.5    | 13.2 ± 0.5              | 13.0 ± 1.5                      | 0.718   |
| Albumin, g/dL                  | 3.7 ± 0.1 (n = 103) | 3.7 ± 0.1 | 3.6 ± 0.2 (n = 18) | 0.446   |
| C-reactive protein, mg/dL      | 3.0 ± 1.0 (n = 102) | 2.7 ± 1.0 (n = 83) | 4.2 ± 3.5                       | 0.962   |
| eGFR, mL/minute/1.73 m²        | 54.0 ± 5.4    | 60.2 ± 5.2              | 26.4 ± 8.4                      | <0.001  |
| Total bilirubin, mg/day        | 0.7 ± 0.1 (n = 103) | 0.7 ± 0.2 (n = 84) | 0.7 ± 0.1                       | 0.397   |
| Lactic acid dehydrogenase, U/L | 460 ± 69      | 431 ± 66                | 590 ± 251                       | 0.045   |
| Creatinine, mg/dL              | 1.8 ± 0.4     | 1.4 ± 0.4               | 3.4 ± 1.5                       | <0.001  |
| White blood cell, 10⁶/L         | 11.4 ± 0.7    | 11.3 ± 0.7              | 11.8 ± 1.8                      | 0.834   |
| BNP, pg/mL                     | 519 ± 140 (n = 101) | 505 ± 158 (n = 84) | 588 ± 318 (n = 17)              | 0.27    |

Data are expressed as the mean ± SD or number (percentage). PCI indicates percutaneous coronary intervention; CABG, coronary artery bypass graft; V-A ECMO, veno-arterial extracorporeal membrane oxygenation; eGFR, estimate glomerular filtration rate; and BNP, brain natriuretic peptide.

Table II. Comparison of Lesion Characteristics between the Survivor Group and In-Hospital Death Group

|                                | All (n = 104) | Survivor group (n = 85) | In-hospital death group (n = 19) | P value |
|--------------------------------|---------------|-------------------------|---------------------------------|---------|
| STEMI, n (%)                   | 75 (72.1)     | 63 (74.1)               | 12 (63.1)                       | 0.335   |
| De novo lesion, n (%)          | 94 (90.4)     | 76 (89.4)               | 18 (94.7)                       | 0.685   |
| Infarct-related artery, n (%)  |               |                         |                                 | 0.392   |
| Left main                      | 27 (26.0)     | 23 (27.1)               | 4 (21.1)                        |         |
| Left anterior descending artery| 40 (38.4)     | 35 (41.2)               | 5 (26.3)                        |         |
| Left circumflex artery         | 15 (14.4)     | 11 (12.9)               | 4 (21.1)                        |         |
| Right coronary artery          | 20 (19.2)     | 14 (16.5)               | 6 (31.6)                        |         |
| Saphenous vein graft           | 2 (1.9)       | 2 (2.4)                 | 0 (0)                           |         |
| CTO in non-infarct-related artery, n (%) | 28 (26.2) | 22 (25.9) | 6 (31.6) | 0.613 |
| Number of vessel disease, n (%)|               |                         |                                 | 0.132   |
| 1                              | 21 (20.2)     | 21 (24.7)               | 1 (5.3)                         |         |
| 2                              | 33 (31.7)     | 27 (31.7)               | 6 (31.6)                        |         |
| 3                              | 49 (47.1)     | 37 (35.6)               | 12 (63.1)                       |         |
| Multi-vessel disease, n (%)    | 82 (78.8)     | 64 (75.3)               | 18 (94.7)                       | 0.049   |
| Initial TIMI grade flow, n (%) |               |                         |                                 | 0.809   |
| 0                              | 44 (42.3)     | 37 (35.6)               | 7 (36.8)                        |         |
| 1                              | 12 (11.5)     | 9 (10.6)                | 3 (15.8)                        |         |
| 2                              | 9 (8.7)       | 8 (9.4)                 | 1 (5.3)                         |         |
| 3                              | 31 (29.8)     | 31 (36.5)               | 8 (42.1)                        |         |
| Final TIMI grade flow, n (%)   |               |                         |                                 | 0.471   |
| 0                              | 1 (7.1)       | 1 (1.2)                 | 0 (0)                           |         |
| 1                              | 1 (7.1)       | 1 (1.2)                 | 0 (0)                           |         |
| 2                              | 8 (7.8)       | 5 (5.9)                 | 3 (15.8)                        |         |
| 3                              | 94 (90.4)     | 78 (91.8)               | 16 (84.2)                       |         |

Data are expressed as the mean ± SD or number (percentage). STEMI indicates ST elevation myocardial infarction; CTO, chronic total occlusion; and TIMI, thrombolysis in myocardial infarction.
related to eGFR, we only adopted eGFR as the variable. Moreover, we did not include total stent length as the variable because total stent length should be missing value in patients who did not receive stent implantation. The initial model included eight variables such as shock, resuscitation, eGFR, pre-systolic blood pressure of IABP insertion, multi-vessel disease, fluoroscopy time, initial Lactic acid dehydrogenase (LDH), and timing of IABP support as independent variables, and then four variables were eliminated from the final model, which included shock, eGFR, multi-vessel disease, and timing of IABP support. Shock (OR 25.27, 95% CI 3.26-196.11, P = 0.002) was significantly associated with in-hospital death after controlling other covariates, whereas eGFR (every 10 mL/ minute/1.73 m² increase: OR 0.65, 95% CI 0.51-0.82, P = 0.001) and pre-PCI insertion (versus on-PCI: OR 0.06, 95% CI 0.008-0.485, P = 0.008) were inversely associated with in-hospital death after controlling other covariates.

### Discussion

A retrospective analysis was conducted including 104 patients with AMI who required IABP support to find clinical factors associated with in-hospital death. Multi-

Table III. Comparison of Procedure Characteristics between the Survivor Group and In-Hospital Death Group

|                          | All (n = 104) | Survivor group (n = 85) | In-hospital death group (n = 19) | P value |
|--------------------------|--------------|------------------------|---------------------------------|---------|
| Access site, n (%)       |              |                        |                                 |         |
| Radial                   | 24 (23.1)    | 2 (25.9)               | 2 (10.5)                        | 0.041   |
| Femoral                  | 77 (74.0)    | 62 (72.9)              | 15 (78.9)                       |         |
| Brachial                 | 3 (2.9)      | 1 (1.2)                | 2 (10.5)                        |         |
| Contrast volume, mL      | 145 ± 10 (n = 103) | 142 ± 11 (n = 84) | 158 ± 28 (n = 19) | 0.228   |
| Fluoroscopy time, minutes| 34 ± 4       | 32 ± 4                 | 42 ± 11                         | 0.043   |
| LMT-LAD crossover stent, (%) | 41 (39.4)    | 36 (42.4)              | 5 (26.3)                        | 0.196   |
| Aspiration, (%)          | 31 (29.8)    | 26 (30.6)              | 5 (26.3)                        | 0.713   |
| Rotablator, n (%)        | 8 (7.8)      | 8 (9.4)                | 0 (0)                           | 0.222   |
| Total stent length (mm)  | 31 ± 4 (n = 84) | 29 ± 4 (n = 69) | 41 ± 12 (n = 15) | 0.023   |
| Average stent diameter, mm | 2.9 ± 0.1 (n = 84) | 2.9 ± 0.1 (n = 69) | 2.9 ± 0.2 (n = 15) | 0.885   |
| Sheath Fr, n (%)         |              |                        |                                 | 0.373   |
| 6 Fr                     | 35 (33.7)    | 31 (36.5)              | 4 / (21.1)                      |         |
| 7 Fr                     | 68 (65.4)    | 53 (62.4)              | 15 (78.9)                       |         |
| 8 Fr                     | 1 (7.1)      | 1 (1.2)                | 0 (0)                           |         |
| Final procedures         |              |                        |                                 | 0.354   |
| Drug-eluting stent, n (%)| 82 (78.8)    | 68 (80.0)              | 14 (73.7)                       |         |
| Bare-metal stent, n (%)  | 2 (1.9)      | 1 (1.2)                | 1 (5.3)                         |         |
| POBA, n (%)              | 15 (14.4)    | 13 (15.3)              | 2 (10.5)                        |         |
| POBA + CABG, n (%)       | 5 (4.8)      | 3 (3.5)                | 2 (10.5)                        |         |
| Timing of the insertion of IABP, n (%) |          |                        |                                 | 0.066   |
| Pre-PCI insertion        | 82 (78.8)    | 70 (82.3)              | 12 (63.1)                       |         |
| On-PCI insertion         | 22 (21.1)    | 15 (17.6)              | 7 (36.8)                        |         |
| Indication for IABP support, n (%) |         |                        |                                 | 0.350   |
| Shock                    | 45 (43.3)    | 33 (33.8)              | 12 (63.2)                       |         |
| PCI to the left main trunk | 24 (23.1)    | 22 (25.9)              | 2 (10.5)                        |         |
| Multi-vessel disease     | 24 (23.1)    | 20 (23.5)              | 4 (16.7)                        |         |
| Slow flow/high risk for slow flow | 10 (9.6) | 9 (10.6) | 1 (5.3) | |
| Bridge to CABG           | 1 (1.0)      | 1 (1.2)                | 0 (0)                           |         |
| Temporary Pacemaker, n (%) | 14 (13.5)    | 9 (10.6)               | 5 (26.3)                        | 0.128   |
| Systolic blood pressure before IABP, mmHg | 114 ± 6 (n = 100) | 118 ± 6 (n = 81) | 96 ± 13 | 0.003   |
| Diastolic blood pressure before IABP, mmHg | 71 ± 4 (n = 99) | 72 ± 4 (n = 80) | 65 ± 9 | 0.155   |

Data are expressed as the mean ± SD or number (percentage). LMT-LAD indicates left main trunk-left arterial descending artery; POBA, plan old balloon angioplasty; CABG, coronary artery bypass graft; and IABP, intra-aortic balloon pumping.

Table IV. The Comparison of Indication for IABP Support between Pre-PCI Insertion and On-PCI Insertion

|                          | Pre-PCI insertion (n = 82) | On-PCI insertion (n = 22) | P value |
|--------------------------|---------------------------|---------------------------|---------|
| Indication for IABP support, n (%) |              |                        | 0.001   |
| Shock                    | 35 (42.7)                 | 10 (45.5)                 |         |
| PCI to the left main trunk | 22 (26.8)                | 2 (9.1)                   |         |
| Multi-vessel disease     | 21 (25.6)                 | 3 (13.6)                  |         |
| Slow flow/high risk for slow flow | 3 (3.7) | 7 (31.8) |     |
| Bridge to CABG           | 1 (1.2)                   | 0                         |         |
Since impaired renal function was associated with in-hospital death, whereas eGFR and pre-PCI insertion of an IABP catheter were inversely associated with in-hospital death. Our results may underscore the limited utility of IABP in AMI patients with shock or impaired renal function. Our results may also suggest the importance of pre-PCI insertion of IABP to maximize the efficacy of IABP support.

First, we should discuss the strong association between shock and in-hospital death in the present study. We included AMI patients with CS as well as without CS. It is not surprising that in-hospital death was more frequently observed in patients with CS than without CS. Our results may confirm the finding of the IABP-SHOCK II trial, which could not show the benefit of IABP for AMI with CS. However, we should mention that two-thirds of patients with CS survived with IABP support and one-third of patients with CS died with IABP support in the present study. IABP support might be helpful for some patients with AMI and CS. Although the routine use of IABP for patients with CS was downgraded in clinical guidelines, it would be still important to find appropriate patients who would receive the benefit from IABP support among patients with AMI and CS.

The significant association between eGFR and in-hospital death in the present study should be discussed. Impaired renal function has been recognized as the determinant of in-hospital death in patients with AMI. Since impaired renal function was associated with multiple risk factors, such as hypertension, diabetes mellitus, and smoking, patients with AMI and impaired renal function might have advanced atherosclerosis, which was not evaluated in the present study. Furthermore, low eGFR levels in the “in-hospital death group” might be a result of malperfusion following CS. Thus, acute renal failure caused by malperfusion might be associated with in-hospital death. We might have difficulty to control fluid volume overload in patients with AMI and impaired renal function. Furthermore, we should discuss the possibility that IABP support might worsen the renal function. There are few studies to investigate the association between IABP support and renal function in patients with AMI. Muniraju et al. conducted a prospective study to determine the degree of possible renal injury when IABP was used for patients undergoing off-pump coronary artery bypass surgery and reported that the use of IABP was not associated with renal injury. Therefore, patients with AMI patients and impaired renal function might suffer from their advanced atherosclerosis, acute renal failure, or fluid volume overload.

The appropriate timing of IABP support should be discussed. Pre-PCI insertion of an IABP catheter was inversely associated with in-hospital death. A possible explanation is that IABP support was useful for prevention of severe status such as refractory shock but not for the treatment of such severe status. Therefore, if operators inserted an IABP catheter before PCI, IABP support might work well to prevent refractory shock during PCI, which resulted in the patients’ survival. If operators inserted an IABP catheter during PCI, IABP support could not change severe status, which might result in in-hospital death. Another explanation is that the indication of IABP was different between pre-PCI insertion and on-PCI insertion, whereas the prevalence of shock as the indication for IABP support was similar between pre-PCI insertion and on-PCI insertion. In our study, shock state included a wide range of blood pressure levels such as systolic blood pressure of 85 mmHg (can be called as stable shock) or systolic blood pressure of <50 mmHg (can be called as severe shock). Even the prevalence of shock was similar between the two groups, the severity of shock might be different between the two groups. In pre-PCI insertion, operators might insert an IABP catheter for the prevention of severe condition during PCI as well as the treatment for CS. In on-PCI insertion, operators might insert an IABP catheter to treat refractory shock during PCI or to treat no-reflow phenomenon following primary PCI. If the reason for IABP was different between the two timings, the in-hospital outcomes should be different. Abdel-Wahab et al. also reported the better outcomes of IABP insertion before PCI as compared to IABP insertion after PCI in AMI with CS, although the classification of timing of IABP insertion (before PCI or after PCI) was different from our classification of timing of IABP insertion (pre-PCI or on-PCI).

Study limitations: The present study has following limitations. Because this study was a retrospective single-center study, there is a risk of patient selection bias. Especially, as the prevalence of cardiopulmonary resuscitation and V-A ECMO was significantly greater in the in-hospital death group than the survivor group, the severity of AMI itself should be different between the two groups. Since the study population was relatively small, the statistical analysis has an inherent risk of beta error. Furthermore, although shock state had a wide range of severity, it was difficult to compare or analyze the severity of shock state between the two groups because of small study sample. The decision and timing of IABP support was left at

### Table V. Multivariate Stepwise Logistic Regression Analysis to Find Associations with In-Hospital Death

| Dependent variable: In-hospital death | Odds ratio | 95% confidence interval | P value |
|--------------------------------------|------------|------------------------|---------|
| Shock (versus without shock)          | 25.27      | 3.26-196.11            | 0.002   |
| eGFR (10 mL/minute/1.73 m² increase) | 0.65       | 0.51-0.82              | <0.001  |
| Multi-vessel disease (versus single vessel disease) | 11.39 | 0.57-22.78 | 0.112 |
| Pre-PCI insertion of IABP (versus On-PCI insertion) | 0.06 | 0.008-0.485 | 0.008 |

Backward elimination stepwise methods. Initial model included shock, resuscitation, eGFR, pre-systolic blood pressure of IABP insertion, multi-vessel disease, fluoroscopy time, initial LDH and timing of IABP support as independent variables.
the discretion of our interventional cardiologists, which should yield a selection bias, especially for patients with AMI without shock. Unlike PCI to the stable lesions, it is not uncommon to have unexpected complications or hemodynamic collapse during PCI to the culprit of AMI. Therefore, we tended to prophylactically insert IABP for patients with AMI without shock. Moreover, since our study included only patients with IABP, we might have missed some patients who required IABP support but actually did not receive IABP support during the study period. Thus, our retrospective study could not provide a clear answer to what types of patients potentially require IABP support. Furthermore, we could not use an Impella device during the study period. The available percutaneous mechanical support devices were only IABP and V-A ECMO.

Conclusions

In patients with AMI who received IABP support, shock was significantly associated with in-hospital death, whereas eGFR and pre-PCI insertion of an IABP catheter were inversely associated with in-hospital death. In patients with AMI and CS, the utility of IABP may be limited. Pre-PCI insertion of an IABP catheter might be associated with better survival in patients with AMI who potentially require IABP support. Further studies are warranted to investigate the timing of IABP insertion for patients with AMI.

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Disclosure

Conflicts of interest: Dr. Sakakura has received speaking honoraria from Abbott Vascular, Boston Scientific, Getinge, Medtronic Cardiovascular, Terumo, OrbusNeich, Japan Lifeline, and NIPRO. He has served as a proctor for Rotablator for Boston Scientific and has served as a consultant for Abbott Vascular and Boston Scientific. Prof. Fujita served as a consultant for Mehergen Group Holdings, Inc.

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