Long-term prognosis and a nomogram model for postoperative acute heart failure in patients with acute myocardial infarction

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Abstract

Background

Undefined adequate hydration may increase the risk of postoperative acute heart failure (AHF) while reducing the risk of contrast-induced acute kidney injury (CI-AKI) in patients with acute myocardial infarction (AMI). No relevant study exists regarding the association of postoperative AHF and long-term prognosis. This study is to evaluate the all-cause long-term mortality and establish a nomogram model for predicting postoperative AHF in this patient group.

Methods

In this prospective observational study, 1312 AMI patients undergoing coronary angiography (CAG) were included in the final analysis. Patients were assigned into a non-postoperative AHF-group (n=1235) or a postoperative AHF-group (n=77). The diagnosis of postoperative AHF was based on assessing symptom history, prior cardiovascular history, and potential cardiac and non-cardiac precipitants.

Results

The overall incidence of postoperative AHF was 77/1312 (5.9%). The incidence of all-cause long-term mortality was significantly higher in the postoperative AHF-group than in the non-postoperative AHF-group (50.6% vs. 17.0%, P<0.01). The median follow-up period was 7.0 years (interquartile range: 5.5 – 8.7). After adjusting for female, LVEF, eGFR, anemia, hypertension, diabetes mellitus, and PCI, postoperative AHF was the strongest predictor of all-cause long-term mortality (hazard ratio: 3.11; 95% CI: 1.83 – 5.30; P<0.01). A nomogram developed based on the four variables was with the AUC 0.83 on internal validation. Calibration curve showed that the predicted and actual probabilities of postoperative AHF were fitted well.

Conclusions

In patients with AMI undergoing CAG, postoperative AHF is the strongest predictor of all-cause long-term mortality. The nomogram showed an effective value of predicting postoperative AHF using preoperative predictions.

Background
Patients with acute myocardial infarction (AMI) have a higher risk of contrast-induced acute kidney injury (CI-AKI), which is associated with increased mortality and substantial increases in health care costs after the coronary angiography (CAG) or percutaneous coronary intervention (PCI) [1-6]. The periprocedural administration of adequate intravenous hydration has been the mainstay of preventing CI-AKI during CAG/PCI [7-9]. However, the most effective regimen of adequate hydration in AMI patients has not been determined [10, 11]. Undefined adequate hydration may lead to an increased risk of postoperative acute heart failure (AHF) in patients with impaired cardiac function [12, 13]. Previous studies showed that in ST-segment elevation myocardial infarction (STEMI) patients treated with primary PCI, AHF during hospitalization increased the risk of short-term mortality [14, 15]. However, relevant studies focusing on the association of postoperative AHF and all-cause long-term mortality in AMI patients are scarce. No clinical prediction model of postoperative AHF exists, either. Our study aimed to clarify this issue.

Methods
Subjects
This was a single-center, prospective observational study (PRECOMIN, ClinicalTrials.gov NCT01400295). From January 2010 to December 2013, we enrolled patients aged ≥ 18 years who continued to be hospitalized for 2 to 3 days after CAG. Exclusion criteria were set according to the International Society of Radiology guidelines [16] and previous studies [17, 18]. In total, 1312 patients with AMI undergoing CAG in our center were included in the final analysis. Follow-up information were obtained by clinical nurses through outpatient service revisit and telephone at 1, 6, 12, 24, and 36 months after CAG. The median follow-up period was 7.0 years (interquartile range: 5.5–8.7). The institutional ethical committees consented the study, and all subjects provided written informed consent.

Percutaneous coronary intervention
PCI was performed by experienced interventional cardiologists according to standard clinical practice using a standard technique. Patients were treated according to the standardization of clinical routines [7, 9]. According to the local clinical practices [17], the serum creatinine levels were measured at hospital admission and within 2 to 3 days after CAG/PCI. We evaluated estimated glomerular filtration
rate (eGFR) using the Modification of Diet in Renal Disease (MDRD) equation [19]. Patients undergoing elective PCI received continuous hydration therapy with normal saline at a rate of 0.5–1 ml/kg/h for at least 2–12 h before and 6–24 h after the procedure. Patients undergoing primary PCI received unlimited hydration therapy before the procedure.

Definition
AMI was defined according to the universal definition of myocardial infarction [20, 21]. Postoperative AHF can be defined as a rapid change in signs and symptoms in patients with CHF or new-onset heart failure (HF) that requires urgent therapy after CAG/PCI. The diagnosis of postoperative AHF was according to a detailed history of symptoms, previous cardiovascular events, the evaluation of signs/symptoms of congestion and/or low perfusion by physical test with further confirmation by specific investigations such as ECG, chest X-ray, laboratory test (with cardiac biomarkers), and echocardiography [22, 23].

Statistical analysis
Comparisons between normally distributed continuous variables, expressed as the mean ± SD, were performed using t tests; non-normally distributed continuous variables, presented as the median and interquartile range, were analyzed using Wilcoxon rank-sum tests. The Pearson χ² or Fisher exact tests were used for categorical data. Cox proportional hazards regression analyses were performed and included the following potential factors: postoperative AHF, female, LVEF, eGFR, anemia, hypertension, diabetes mellitus (DM), and PCI. A follow-up analysis was performed using time-to-event data (for which patients were censored at the time of withdrawal from the study or at the last follow-up), with the event rates estimated by Kaplan-Meier methods and compared with the log-rank test. The significance of each variable was assessed by univariate logistic regression analysis to investigate the independent risk factors of postoperative AHF. All risk factors significantly associated with postoperative AHF were selected for following multivariate analysis. We developed a nomogram according to the results of multivariate logistic regression analysis. The nomogram is based on proportionally transforming each regression coefficient in multivariate logistic regression to a 0- to 100-point scale. The effect of the variable with the highest β coefficient (absolute value) is defined as
100 points. The points are added across independent variables to generate total points, which are transformed to predicted probabilities. The predictive performance of the nomogram was measured by the concordance index (C index) and calibration curve. Internal validation was performed via a bootstrap method with 1000 resamples. Data analyses were done in R software version 3.6.1 (http://www.r-project.org). A two-sided P < 0.05 was deemed significant.

Results
A total of 1312 eligible patients were included and divided into a non-postoperative AHF-group (n = 1235) and a postoperative AHF-group (n = 77). No decompensated HF occurred in all patients before CAG. Patients in the postoperative AHF group were more likely to be female, and have older age, anemia, DM, increased heart rate, increased calcium channel blocker (CCB) usage, diuretic usage compared to those in the non-postoperative AHF group. Patients in the postoperative AHF group also presented with worse cardiac function, decreased renal function, decreased ACEI/ARB usage, and beta blocker usage. The ratio of previous myocardial infarction (MI), hypertension, hyperlipidemia, preoperative systolic blood pressure (SBP), preoperative diastolic blood pressure (DBP), the ratio of preoperative low blood pressure (LBP), and lesion > 1 were not significantly different between the groups (Table 1).
### Table 1: Baseline characteristics of the patients.*

| Characteristic                        | Non-postoperative AHF (N = 1235) | Postoperative AHF (N = 77) | P Value |
|---------------------------------------|----------------------------------|-----------------------------|---------|
| **Demographic**                       |                                  |                             |         |
| Age — yr                              | 61.2 ± 12.0                      | 68.8 ± 12.5                 | < 0.01  |
| Age > 75 year — no.(%)                | 172 (13.9)                       | 25 (32.5)                   | < 0.01  |
| Female — no.(%)                       | 188 (15.2)                       | 23 (29.9)                   | < 0.01  |
| LVEF — %                              | 54.4 ± 10.6                      | 45.0 ± 12.0                 | < 0.01  |
| LVEF < 40% — no.(%)                   | 110 (8.9)                        | 24 (31.2)                   | < 0.01  |
| Killip class > 1 — no. (%)            | 259 (21.0)                       | 47 (61.0)                   | < 0.01  |
| **Medical history**                   |                                  |                             |         |
| Previous MI — no. (%)                 | 51 (4.1)                         | 2 (2.6)                     | 0.77    |
| Hypertension — no.(%)                 | 600 (48.6)                       | 51 (66.2)                   | < 0.01  |
| Hyperlipidemia — no. (%)              | 193 (15.6)                       | 10 (13.0)                   | 0.53    |
| Anemia — no.(%)                       | 398 (32.2)                       | 35 (45.5)                   | 0.02    |
| Diabetes mellitus — no. (%)           | 242 (19.6)                       | 26 (33.8)                   | < 0.01  |
| Smoking — no.(%)                      | 607 (49.1)                       | 22 (28.6)                   | < 0.01  |
| **Laboratory test**                   |                                  |                             |         |
| eGFR, ml/min/1.73 m²                  | 81.4 ± 26.1                      | 62.1 ± 29.5                 | < 0.01  |
| eGFR < 60 ml/min/1.73 m²              | 223 (18.1)                       | 40 (51.9)                   | < 0.01  |
| **Procedure**                         |                                  |                             |         |
| PCI — no.(%)                          | 809 (65.5)                       | 31 (40.3)                   | 0.50    |
| Lesion > 1 — no.(%)                   | 653 (52.9)                       | 27 (35.1)                   | 0.49    |
| Preoperative SBP, mmHg                | 122.0 ± 19.9                     | 121.5 ± 24.8                | 0.86    |
| Preoperative DBP, mmHg                | 73.5 ± 11.8                      | 74.3 ± 13.9                 | 0.62    |
| Preoperative LBP — no. (%)            | 125 (10.1)                       | 11 (14.3)                   | 0.23    |
| Preoperative HR, bpm                  | 77 ± 15                          | 88 ± 20                     | < 0.01  |
| **Medication**                        |                                  |                             |         |
| ACEI/ARB — no.(%)                     | 1106 (89.6)                      | 62 (80.5)                   | 0.01    |
| Beta-blockers — no.(%)                | 1025 (83.0)                      | 51 (66.2)                   | < 0.01  |
| CCB — no.(%)                          | 101 (8.2)                        | 10 (13.0)                   | 0.14    |
| Diuretic — no.(%)                     | 345 (27.9)                       | 61 (79.2)                   | < 0.01  |

* Plus-minus values are mean ± SD. AHF: acute heart failure. IQR: interquartile range. LVEF: left ventricular ejection fraction. MI: myocardial infarction. eGFR: estimated glomerular filtration rate. PCI: percutaneous coronary intervention. SBP: systolic blood pressure. DBP: diastolic blood pressure. LBP: low blood pressure. HR: heart rate. ACEI/ARB: angiotensin-converting enzyme inhibitor/angiotensin receptor blocker.

### Table 2: Univariate and multivariate logistic regression analysis of postoperative AHF

| Variable                | Univariate OR (95% CI) | P value | Multivariate OR (95% CI) | β | P value |
|-------------------------|------------------------|---------|--------------------------|---|---------|
| Age                     | 1.06 (1.04–1.08)       | < 0.01  |                         |   |         |
| Age > 75 year — yes     | 3.05 (1.87–4.97)       | < 0.01  | 2.01 (1.05–3.83)         | 0.70 | 0.03    |
| Female — yes            | 2.37 (1.42–3.96)       | < 0.01  | 1.44 (0.74–2.80)         | 0.37 | 0.28    |
| LVEF < 40% — yes        | 0.93 (0.91–0.95)       | < 0.01  |                         |   |         |
| Killip class > 1 — yes  | 4.56 (2.69–7.72)       | < 0.01  | 3.08 (1.59–5.97)         | 1.12 | < 0.01  |
| Hypertension — yes      | 7.17 (4.17–12.32)      | < 0.01  |                         |   |         |
| Anemia — yes            | 2.07 (1.28–3.37)       | < 0.01  | 1.52 (0.83–2.80)         | 0.42 | 0.17    |
| Diabetes mellitus — yes | 1.77 (1.11–2.83)       | 0.02    | 1.27 (0.70–2.29)         | 0.24 | 0.43    |
| Smoking — yes           | 2.09 (1.28–3.42)       | < 0.01  | 1.26 (0.67–2.39)         | 0.23 | 0.48    |
| eGFR < 60 — yes         | 0.41 (0.25–0.69)       | < 0.01  |                         |   |         |
| Pre-PCI HR — yes        | 4.99 (3.11–8.00)       | < 0.01  | 2.41 (1.30–4.47)         | 0.88 | < 0.01  |
| ACEI/ARB — yes          | 1.04 (1.03–1.06)       | < 0.01  | 1.03 (1.01–1.04)         | 0.03 | < 0.01  |
| Beta-blockers — yes     | 0.48 (0.26–0.87)       | 0.01    |                         |   |         |
| Diuretic — yes          | 9.84 (5.59–17.29)      | < 0.01  |                         |   |         |

Abbreviations: AHF: acute heart failure; OR: odds ratio; CI: confidence interval; LVEF: left ventricular ejection fraction; eGFR: estimated glomerular filtration rate; PCI: percutaneous coronary intervention; HR: heart failure; ACEI/ARB: angiotensin-converting enzyme inhibitor/angiotensin receptor blocker.
The median follow-up period was 7.0 years (interquartile range: 5.5–8.7), and data were available for all subjects who survived to discharge. In total, 39 of the 77 (50.6%) patients died in long-term follow-up in the postoperative AHF group, while 210 of the 1235 (17.0%) patients died in long-term follow-up in the non-postoperative AHF group. Log-rank survival analyses revealed that patients in the postoperative AHF group showed worse survival rates than patients in the non-postoperative AHF group (log-rank P < 0.001; Fig. 1).

Potential predictive factors for all-cause long-term mortality including postoperative AHF, female, LVEF, eGFR, anemia, hypertension, diabetes mellitus, and PCI were evaluated using multivariable cox regression analysis. Postoperative AHF was the strongest predictor of all-cause long-term mortality (HR: 3.11, 95% CI: 1.83–5.30, P < 0.01; Fig. 2). LVEF and eGFR were also associated with all-cause long-term mortality (C-index = 0.86).

Based on the result of the multivariate analysis, age > 75 years (2.01 [1.05–3.83]), eGFR < 60 ml/min/1.73m² (2.41 [1.30–4.47]), LVEF < 40% on admission (3.08 [1.59–5.97]) and Killip class > 1 on admission (4.36 [2.38–7.99]) were independently associated with postoperative AHF. These variables were selected to create a nomogram for predicting postoperative AHF (Fig. 3). The final model was internally validated using the bootstrap validation method and show a certain prediction efficiency in the risk of postoperative AHF. The unadjusted C index was 0.83. After a bootstrap-correction, the C index showed no significant change. The calibration curve graphically presented highly consistence on the occurrence of postoperative AHF between the risk estimation by the nomogram and clinical diagnosis of postoperative AHF (Fig. 4).

Discussion

For all we know, this is the first study exploring the association of postoperative AHF and all-cause long-term mortality in AMI patients undergoing CAG/PCI. Our results show that postoperative AHF is the strongest predictor of all-cause long-term mortality. Furthermore, LVEF and eGFR were also independent predictors of all-cause long-term mortality in this patient group. In the National Registry of Myocardial Infarction (NRMI) -2 and – 3, the incidence of HF on admission was 20.4% [24]. In the Global Registry of Acute Coronary Events (GRACE), 15.6% of the patients presented with signs of HF
on admission [25]. In general, the incidence of HF during hospitalization is significantly lower than in earlier studies (40%-50%) [26]. In general, the incidence of HF after AMI is on the decline. However, in our study of AMI patients undergoing CAG, we were first to report the incidence of postoperative AHF (5.9%) but not HF during hospitalization. The incidence of postoperative AHF, which was not present before CAG, was probably related to the undetermined adequate hydration during CAG/PCI in our center. Although it has been confirmed that PCI is beneficial to reducing myocardial infarction size and preventing cardiac dysfunction [27, 28], the proportion of primary PCI in our center is low, which represents the overall treatment status of AMI in middle-income areas in China. According to the baseline characteristic in our study, the early revascularization of culprit vessels may alleviate myocardial injury and reduce the risk of postoperative AHF. Patients in the postoperative AHF group were more likely to have older age, anemia, DM, increased heart rate, increased CCB usage, and diuretic usage compared to those in the non-postoperative AHF group. Patients in the postoperative AHF group also presented with worse cardiac function, decreased renal function and decreased ACEI/ARB and beta blocker usage. These data confirm that AMI patients were less likely to receive clear benefit from primary PCI if they had the following characteristics: occurrence of postoperative AHF, impaired cardiac function, renal insufficiency, and multivessel disease. An earlier study found that although the extensive application of primary PCI in AMI patients may reduce the risk of HF during hospitalization when HF develops, the short-term prognosis (1 and 6 months after discharge) remains poor [15]. Another recent study confirmed that HF during hospitalization was associated with in-hospital mortality in STEMI patients undergoing primary PCI [14]. Our study focused on exploring the association of postoperative AHF and all-cause long-term mortality in AMI patients undergoing CAG/PCI. Based on those preoperative predictions in the nomogram, our predictive model for postoperative AHF might serve as a guide for clinicians in the early identification of patients at high risk of postoperative AHF, leading to prompt intervention. The results of our study facilitate the further exploration of potential clinical intervention targets for preventing postoperative AHF in AMI patients undergoing CAG/PCI.

Limitation
There are some limitations to this study. First, this was a prospective, observational study that was implemented in a single center, and the sampling bias and generalizability of the study results to other regions are potential concerns. Our center, however, is the most representative cardiovascular center in southern China. It includes a relatively large number of patients undergoing CAG/PCI, and our results comprise the complete assessment of current practice patterns in China. The cohort in our study is representative of the patient population commonly referred to centers in low-income and middle-income countries for CAG/PCI in the real world. Second, patients with cardiogenic shock were not included in our final analysis although this patient group has the most severe HF. The impact of a different PCI strategy in STEMI patients with cardiogenic shock has been discussed in several previous studies. Finally, patients undergoing elective PCI received a continuous hydration therapy of isotonic saline at a speed of 0.5–1 mL/kg/h for at least 2–12 hours before and continuing 6–24 hours after the procedure, while patients undergoing primary PCI received unlimited hydration therapy before the procedure. We did not, however, compare the difference in hydration volume and velocity between these two groups. First-line drugs, such as ACEI/ARB, beta blockers, and diuretics, were not used adequately. These confounding factors might have influenced the occurrence of AHF and all-cause long-term mortality. However, we did adjust for the important clinical variables in a Cox regression model including female, LVEF, eGFR, anemia, hypertension, diabetes mellitus, and PCI. The C-index of the model shows moderate accuracy.

Conclusions
In patients with AMI undergoing CAG, postoperative AHF is the strongest predictor of all-cause long-term mortality. The nomogram showed an effective value of predicting postoperative AHF prior to CAG. Using the model, the risk of each individual AMI patient developing postoperative AHF can be estimated, which is beneficial for clinicians in making early clinical decisions.

Abbreviations
ACEI/ARB: angiotension-converting enzyme inhibitor/angiotension receptor blocker; AHF: acute heart failure; CCB: calcium channel blockers; CI: confidence interval; DBP: diastolic blood pressure; eGFR: estimated glomerular filtration rate; HR: heart rate; IQR: interquartile range; LBP: low blood
Declarations

**Ethics approval and consent to participate**

Ethical approval for PRECOMIN was received from the ethics committees of the Guangdong Provincial People's Hospital and was conducted in accordance with the Helsinki Declaration and its later amendments. All the patients included gave written informed consent.

**Consent for publication**

Not applicable.

**Availability of data and materials**

Not applicable.

**Competing interests**

The Authors declare that they have no competing interests

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**Authors’ contributions**

All authors contributed to conception and design, critically revised the manuscript, gave final approval, and agreed to be accountable for all aspects of work ensuring integrity and accuracy (ZG, GS, FS, LL, YH, BL, SC, YL, JC). ZG contributed to data acquisition, analysis, and the first draft of the manuscript.

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References

1. Chong E, Poh KK, Liang S, Soon CY, Tan HC. Comparison of risks and clinical predictors of contrast-induced nephropathy in patients undergoing emergency versus nonemergency percutaneous coronary interventions. J Interv Cardiol. 2010;23:451-9.

2. Kume K, Yasuoka Y, Adachi H, Noda Y, Hattori S, Araki R, et al. Impact of contrast-induced acute kidney injury on outcomes in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. Cardiovasc Revasc Med. 2013;14:253-7.

3. Marenzi G, Lauri G, Assanelli E, Campodonico J, De Metrio M, Marana I, et al. Contrast-induced nephropathy in patients undergoing primary angioplasty for acute myocardial infarction. J Am Coll Cardiol. 2004;44:1780-5.

4. Narula A, Mehran R, Weisz G, Dangas GD, Yu J, Genereux P, et al. Contrast-induced acute kidney injury after primary percutaneous coronary intervention: results from the HORIZONS-AMI substudy. Eur Heart J. 2014;35:1533-40.

5. Pyxaras SA, Sinagra G, Mangiacapra F, Perkan A, Di Serafino L, Vitrella G, et al. Contrast-induced nephropathy in patients undergoing primary percutaneous coronary intervention without acute left ventricular ejection fraction impairment. Am J Cardiol. 2013;111:684-8.
6. Sadeghi HM, Stone GW, Grines CL, Mehran R, Dixon SR, Lansky AJ, et al. Impact of renal insufficiency in patients undergoing primary angioplasty for acute myocardial infarction. Circulation. 2003;108:2769-75.

7. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the task force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J. 2018;39:119-77.

8. Neumann FJ, Sousa-Uva M, Ahlsson A, Alfonso F, Banning AP, Benedetto U, et al. 2018 ESC/EACTS guidelines on myocardial revascularization. Eur Heart J. 2019;40:87-165.

9. Roffi M, Patrono C, Collet JP, Mueller C, Valgimigli M, Andreotti F, et al. 2015 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: task force for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J. 2016;37:267-315.

10. Mehran R, Dangas GD, Weisbord SD. Contrast-associated acute kidney injury. N Engl J Med. 2019;380:2146-55.

11. O’Gara PT, Kushner FG, Ascheim DD, Casey DE, Jr., Chung MK, De Lemos JA, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American college of cardiology foundation/American heart association task force on practice guidelines. J Am Coll Cardiol. 2013;61:e78-140.

12. Chong E, Poh KK, Liang S, Tan HC. Risk factors and clinical outcomes for contrast-induced nephropathy after percutaneous coronary intervention in patients with normal serum creatinine. Ann Acad Med Singapore. 2010;39:374-80.

13. Briguori C, Signoriello G. Acute kidney injury: intravenous hydration for the prevention of CIAKI.
4. Auffret V, Leurent G, Gilard M, Hacot JP, Filippi E, Delaunay R, et al. Incidence, timing, predictors and impact of acute heart failure complicating ST-segment elevation myocardial infarction in patients treated by primary percutaneous coronary intervention. Int J Cardiol. 2016;221:433-42.

5. Santoro GM, Carrabba N, Migliorini A, Parodi G, Valenti R. Acute heart failure in patients with acute myocardial infarction treated with primary percutaneous coronary intervention. Eur J Heart Fail. 2008;10:780-5.

6. Stacul F, Van Der Molen AJ, Reimer P, Webb JA, Thomsen HS, Morcos SK, et al. Contrast induced nephropathy: updated ESUR contrast media safety committee guidelines. Eur Radiol. 2011;21:2527-41.

7. Liu Y, Chen JY, Tan N, Zhou YL, Yu DQ, Chen ZJ, et al. Safe limits of contrast vary with hydration volume for prevention of contrast-induced nephropathy after coronary angiography among patients with a relatively low risk of contrast-induced nephropathy. Circ Cardiovasc Interv. 2015;8:e001859.

8. Liu Y, Li H, Chen S, Chen J, Tan N, Zhou Y, et al. Excessively high hydration volume may not be associated with decreased risk of contrast-induced acute kidney injury after percutaneous coronary intervention in patients with renal insufficiency. J Am Heart Assoc. 2016;5:e003171.

9. Aguiar-Souto P, Ferrante G, Del Furia F, Barlis P, Khurana R, Di Mario C. Frequency and predictors of contrast-induced nephropathy after angioplasty for chronic total occlusions. Int J Cardiol. 2010;139:68-74.

10. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). Eur Heart J. 2019;40:237-69.

11. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, et al. Third universal definition of myocardial infarction. J Am Coll Cardiol. 2012;60:1581-98.
2. McMurray JJ, Adamopoulos S, Anker SD, Auricchio A, Bohm M, Dickstein K, et al. ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: the task force for the diagnosis and treatment of acute and chronic heart failure 2012 of the European society of cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC. Eur Heart J. 2012;33:1787-847.

3. Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JGF, Coats AJS, et al. 2016 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure: the task force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) developed with the special contribution of the Heart Failure Association (HFA) of the ESC. Eur Heart J. 2016;37:2129-200.

4. Spencer FA, Meyer TE, Gore JM, Goldberg RJ. Heterogeneity in the management and outcomes of patients with acute myocardial infarction complicated by heart failure: the national registry of myocardial infarction. Circulation. 2002;105:2605-10.

5. Steg PG, Dabbous OH, Feldman LJ, Cohen-Solal A, Aumont MC, Lopez-Sendon J, et al. Determinants and prognostic impact of heart failure complicating acute coronary syndromes: observations from the Global Registry of Acute Coronary Events (GRACE). Circulation. 2004;109:494-9.

6. Emanuelsson H, Karlson BW, Herlitz J. Characteristics and prognosis of patients with acute myocardial infarction in relation to occurrence of congestive heart failure. Eur Heart J. 1994;15:761-8.

7. Ndrepepa G, Mehilli J, Schwaiger M, Schuhlen H, Nekolla S, Martinoff S, et al. Prognostic value of myocardial salvage achieved by reperfusion therapy in patients with acute myocardial infarction. J Nucl Med. 2004;45:725-9.

8. Sciagra R, Bolognese L, Rovai D, Sestini S, Santoro GM, Cerisano G, et al. Detecting myocardial salvage after primary PTCA: early myocardial contrast echocardiography versus delayed
Figures

Kaplan–Meier estimates of long-term mortality according to postoperative AHF

|                | Non postoperative AHF | Postoperative AHF |
|----------------|-----------------------|-------------------|
| Number at risk | 1192                  | 76                |
|                | 1085                  | 43                |
|                | 975                   | 36                |
|                | 501                   | 12                |
|                | 0                     | 0                 |

Log rank p<0.001

Percent survival

Follow-up years

Non postoperative AHF
Postoperative AHF
Predictive factors for long-term mortality in multivariable Cox regression analysis

| Variable                | Hazard ratio for death | HR   | 95% CI          | P value |
|-------------------------|------------------------|------|-----------------|---------|
| Postoperative AHF       | 3.11                   | 1.83-5.3 | <0.01          |
| Female                  | 1.27                   | 0.83-1.93 | 0.27          |
| LVEF                    | 0.98                   | 0.97-0.99 | <0.01          |
| eGFR                    | 0.97                   | 0.96-0.99 | <0.01          |
| Anemia                  | 0.99                   | 0.7-1.4  | 0.96           |
| Hypertension            | 1.24                   | 0.89-1.72 | 0.21          |
| Diabetes mellitus       | 1.25                   | 0.87-1.79 | 0.23          |
| PCI                     | 0.81                   | 0.52-1.27 | 0.36          |
Figure 3

Nomogram to estimate the risk of postoperative AHF
The calibration curve for predicting postoperative AHF in the primary cohort