CLINICAL INVESTIGATIONS

Cardiogenic Shock Predicts Long-term Mortality in Hospital Survivors of STEMI Treated With Primary Percutaneous Coronary Intervention

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Background: ST-segment elevation myocardial infarction (STEMI) complicated by cardiogenic shock (CS) has a poor prognosis. Recently published data suggested, however, that CS does not affect long-term mortality in hospital survivors of STEMI. We investigated whether this could be confirmed in a larger cohort.

Hypothesis: STEMI complicated by CS leads to worse long-term survival.

Methods: A prospective cohort study was performed in 7412 consecutive patients with STEMI treated by primary percutaneous coronary intervention (angioplasty). The predictive value of CS on long-term mortality was assessed in hospital survivors. Multivariate analysis, adjusting for differences in baseline variables, was performed to assess the independent prognostic value of CS.

Results: Cardiogenic shock was observed in 387 patients (5.2%). The total in-hospital mortality was 254 (3.4%), and mortality was significantly higher in patients with CS (20.0% vs 2.6%; $P < 0.001$). The 1-year mortality in hospital survivors was 10.3% in patients with CS and 3.9% in patients without CS ($P < 0.001$). After multivariate analysis, CS was still a significant predictor of long-term mortality in hospital survivors (hazard ratio: 2.6, 95% confidence interval: 1.64-4.01).

Conclusions: Cardiogenic shock remains a strong predictor of long-term mortality in hospital survivors of STEMI treated by primary percutaneous coronary intervention.

KEYWORDS

Ischemic heart disease, myocardial infarction, Acute coronary syndrome

1 | INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) is complicated by cardiogenic shock (CS) in 5% to 10% of patients.^{1,2} Particularly in these patients, rapid opening of the infarct-related vessel is mandatory.³ Medical treatment of CS in patients with STEMI includes inotropes (ie, noradrenaline, dopamine). The benefit of the intra-aortic balloon pump (IABP) for CS has been questioned,⁴ but it may be of benefit. Also, other mechanical devices, such as Impella, may improve hemodynamics in selected patients.⁵ Despite all these efforts, prognosis of patients with STEMI and CS remains poor.⁶ Recently, it was suggested that CS has no prognostic importance in those who survive the initial hospital phase, possibly because of improved medical

treatment and increased frequency of coronary revascularization.⁷ However, because this was only observed in 1 study, and it was different from previous observations, our aim was whether this could be confirmed in a large cohort of patients treated with primary percutaneous coronary intervention (PCI) for STEMI.

2 | METHODS

Data for this study are from the prospective cohort of patients with STEMI at the Isala Heart Centre, Zwolle, the Netherlands, from January 2000 to December 2011. Patients were diagnosed with STEMI if they had chest pain of >30 minutes in duration and

electrocardiographic changes with ST-segment elevation >2 mm in ≥ 2 precordial leads or >1 mm in the limb leads. To avoid double inclusion, only the first recorded admission for STEMI was included. All patients underwent emergent angiography, and in almost all patients primary PCI was performed. Baseline measurements of blood pressure (BP) and heart rate (HR), as well as laboratory investigations, were obtained before the emergency angiography. Our CS cohort consisted of patients with CS at admission. Prior to emergency angiography, all patients received 300 to 500 mg of aspirin intravenously, a P2Y₁₂ blocker (either 600 mg of clopidogrel or 180 mg of ticagrelor), and 3000 to 5000 IU of unfractionated heparin. Additional treatment with glycoprotein IIb/IIIa inhibitors, balloon angioplasty, and bare-metal or drug-eluting stents was left to the discretion of the treating cardiologist. Patients were treated with optimal medication including angiotensin-converting enzyme inhibitor (ACEI), β -blocker, aspirin, and statin where appropriate. Primary PCI was performed with standard techniques if the coronary anatomy was suitable for angioplasty. Ischemic time was calculated from time of symptom onset to first balloon inflation. Mortality data were prospectively entered into the local STEMI database and checked with municipal records.

2.1 | Definitions

Cardiogenic shock at admission was defined as a systolic BP <90 mm Hg for ≥ 30 minutes, clinical signs of pulmonary congestion, and end-organ hypoperfusion (cool extremities, altered mental status, or a urine output of <30 mL/h). Coronary angiographic flow was determined in accord with the classification system of the Thrombolysis In Myocardial Infarction (TIMI) study group. Hospital survival was defined as being alive at true discharge from the hospital.

2.2 | Statistical Analysis

Statistical analysis was performed with the Statistical Package for the Social Sciences (SPSS), version 20.0 (IBM Corp., Armonk, NY). Continuous data are presented as mean $\pm$ SD or median (interquartile range) for non-normally distributed variables; discrete data are given as absolute values and percentages. To define potential confounders, we compared baseline characteristics between patients with or without CS. To demonstrate the effect of CS in all subgroups, we also performed subgroup analysis. Survival analysis was performed, with a landmark analysis of patients who survived the hospitalization, and was displayed as Kaplan-Meier survival curves. To demonstrate the independent predictive value of CS on long-term survival, multivariate analyses was performed using the Cox proportional hazard model and adjusting for age, sex, anterior MI location, multivessel disease, glucose level, TIMI flow grade pre-PCI 0 to 1, TIMI flow grade post-PCI <3 , and β -blocker and ACEI use at discharge. A *P* value of 0.05 was considered significant.

3 | RESULTS

Between January 2000 and December 2011, a total of 7412 patients with STEMI were hospitalized in our center. The mean age was

63.3 ± 12.7 years, and 2036 patients (27.5%) were female. At admission, a total of 387 patients (5.2%) had CS. Patients with CS were older (age 64.5 ± 12.4 years vs 63.2 ± 12.7 years; *P* = 0.04), more often female (32.6% vs 27.2%; *P* = 0.02), less often had anterior MI (30.1% vs 43.6%; *P* < 0.01), but more often had multivessel disease (56.6% vs 49.1%; *P* < 0.01). In-hospital mortality was 254 (3.4%) and was significantly higher in patients with CS than in patients without CS (20.0% vs 2.6%; *P* < 0.01). A total of 7158 patients survived the hospital phase; of those patients, 1-year follow-up was not available in 9 patients (0.1%), resulting in a final study population of 7149 patients.

Hospital survivors were discharged after a mean of 4.4 ± 6.6 days. Of the hospital survivors, a total of 310 patients (4.3%) had CS at admission. In Table 1, the baseline characteristics of hospital survivors with and without CS at admission are summarized. There was no statistically significant difference in age and sex between hospital survivors with and without CS at admission. The CS patients less often had anterior MI, but more often had multivessel disease. They also more often had an occluded infarct-related artery and TIMI flow grade <3 post-PCI.

In Table 2, the baseline characteristics of hospital survivors who survived 1 year and those who died within 1 year are summarized. In hospital survivors with CS, 1-year mortality was significantly higher compared with that of hospital survivors without CS (10.3% vs 3.9%, respectively; *P* < 0.001). Figure 1 shows a landmark analysis of 1-year survival in patients with and without CS, excluding those who died in the hospital phase. Subgroup analysis in hospital survivors demonstrates this increased risk is present in all subgroups (Figure 2).

After multivariate analysis, CS at admission remained a strong independent predictor of 1-year mortality in patients who survived the hospital phase with a hazard ratio (HR) of 2.6 (95% confidence interval: 1.64-4.01), adjusting for age, sex, anterior MI, multivessel disease, glucose level, TIMI flow grade pre-PCI 0 to 1, TIMI flow grade post-PCI <3 , and β -blocker and ACEI use at discharge.

4 | DISCUSSION

Our study demonstrates that CS at admission is still a strong and independent predictor of long-term mortality in patients with STEMI treated with primary PCI who survive the hospital stay.

Spyridopoulos et al concluded that CS is not able to predict mortality in STEMI patients who survived the index hospital stay.⁷ A possible explanation for their finding may be the definition they used for CS (systolic BP <100 mm Hg and a HR >100 bpm). First, this is a rather unusual definition. The most accepted clinical definition of CS is a systolic BP of <90 mm Hg for ≥ 30 minutes or the need for supportive measures to maintain a systolic BP of >90 mm Hg, and end-organ hypoperfusion (cool extremities or a urine output of <30 mL/h).³ Second, this definition probably excluded patients with CS due to complicated inferior STEMIs (eg, bradyarrhythmias, right ventricle [RV] involvement), though prognosis in these patients is known to be impaired.⁸ Furthermore, in their multivariate analysis to identify independent predictors of post-discharge long-term mortality, they included both CS and a positive

TABLE 1 Baseline Characteristics, According to Presence of CS on Admission, in 7149 Hospital Survivors of STEMI Treated by Primary PCI

	No CS, n = 6839	CS, n = 310	P Value
Age, y (mean $\pm$ SD)	62.9 $\pm$ 12.6	63.3 $\pm$ 12.2	0.64
Female sex	1829 (26.7)	97 (31.3)	0.09
Risk factors			
DM	781 (11.5)	34 (11.0)	0.93
HTN	2402 (35.5)	97 (31.5)	0.16
Dyslipidemia	1464 (22.4)	58 (19.4)	0.26
Family history	2678 (40.6)	110 (36.3)	0.15
History			
Previous MI	569 (8.4)	29 (9.4)	0.53
Previous PCI	463 (6.8)	22 (7.1)	0.82
Previous CABG	216 (3.2)	8 (2.6)	0.74
Previous CVA	187 (2.7)	12 (3.9)	0.22
Hemodynamics on admission			
SBP, mm Hg	135 $\pm$ 24	83 $\pm$ 25	<0.001
HR, bpm	76 $\pm$ 17	70 $\pm$ 25	<0.001
Laboratory results on admission			
Hb, g/dL	8.7 $\pm$ 0.9	8.1 $\pm$ 1.2	<0.001
Glucose, μ mol/L	9.0 $\pm$ 3.1	10.4 $\pm$ 3.8	<0.001
Cr, μ mol/L	88 $\pm$ 40	104 $\pm$ 60	<0.001
Ischemic time >2 h	5191 (90.0)	228 (85.7)	0.028
Angiographic data			
Anterior location of MI	2811 (43.4)	90 (30.1)	<0.001
Multivessel disease	3266 (48.6)	163 (54.0)	0.07
TIMI flow grade pre-PCI 0–1	3869 (63.4)	193 (70.2)	0.025
TIMI flow grade post-PCI <3	486 (8.0)	34 (12.4)	0.018
Treatment			
PCI performed	5939 (89.7)	274 (89.5)	0.92
Stent placement	4811 (75.4)	208 (69.8)	0.033
CABG within 2 d	145 (2.1)	17 (5.5)	0.001
CABG within 30 d	564 (8.2)	32 (10.3)	0.21
IABP	446 (6.8)	81 (26.6)	<0.001

Abbreviations: CABG, coronary artery bypass grafting; Cr, creatinine; CS, cardiogenic shock; CVA, cerebrovascular accident; DM, diabetes mellitus; Hb, hemoglobin; HR, heart rate; HTN, hypertension; IABP, intra-aortic balloon pump; MI, myocardial infarction; PCI, percutaneous coronary intervention; SBP, systolic blood pressure; SD, standard deviation; STEMI, ST-segment elevation myocardial infarction; TIMI, Thrombolysis In Myocardial Infarction.

Data are presented as n (%) or mean $\pm$ SD.

shock index as a variable, which are almost the same variables. This could have led to an underestimation of the effect of CS as independent predictor.

Surprisingly, in our study, anterior MI was significantly less present in patients with CS. However, the presence of inferior MI was significantly higher in patients with CS vs without CS (50.0% vs 37.1%, respectively; $P < 0.001$), suggesting RV involvement or vagovagal reactions. This also explains the lower HR in CS patients vs patients without CS. Although in general patients with inferior-wall MI have a better prognosis, those who have hypotension and RV involvement may have a poor prognosis.⁸

TABLE 2 Differences in Baseline Variables Between Hospital Survivors Who Died Within 1 Year and Those Who Survived

	1-Year Survival, n = 6852	Death <1 Year, n = 297	P Value
Age, y	62.5 $\pm$ 12.5	73.7 $\pm$ 11.8	<0.001
Female sex	1827 (26.7)	99 (33.3)	0.013
Risk factors			
DM	750 (11.0)	65 (22.0)	<0.001
HTN	2347 (34.7)	152 (51.5)	<0.001
Dyslipidemia	1461 (22.3)	61 (21.3)	0.77
Family history	2724 (41.2)	64 (22.6)	<0.001
History			
Previous MI	552 (8.1)	46 (15.6)	<0.001
Previous PCI	454 (6.7)	31 (10.5)	0.013
Previous CABG	205 (3.0)	19 (6.4)	0.003
Previous CVA	172 (2.5)	27 (9.1)	<0.001
Hemodynamics on admission			
SBP, mm Hg	133 $\pm$ 25	130 $\pm$ 28	0.091
HR, bpm	75 $\pm$ 18	82 $\pm$ 21	<0.001
CS	278 (4.1)	32 (10.8)	<0.001
Laboratory results on admission			
Hb, g/dL	8.7 $\pm$ 0.9	8.0 $\pm$ 1.3	<0.001
Glucose, μ mol/L	9.0 $\pm$ 3.1	10.0 $\pm$ 3.8	<0.001
Cr, μ mol/L	87 $\pm$ 34	124 $\pm$ 108	<0.001
Ischemic time >2 h	5210 (89.6)	209 (95.9)	0.001
Angiographic data			
Anterior location of MI	2787 (42.8)	114 (43.8)	0.75
Multivessel disease	3262 (48.2)	167 (62.8)	<0.001
TIMI flow grade pre-PCI 0–1	3895 (63.4)	167 (71.4)	0.013
TIMI flow grade post-PCI <3	468 (7.7)	52 (22.4)	<0.001
Treatment			
PCI performed	5984 (90.1)	229 (80.4)	<0.001
Stent placement	4869 (75.7)	150 (60.5)	<0.001
CABG within 2 d	154 (2.2)	8 (2.7)	0.55
CABG within 30 d	570 (8.3)	26 (8.8)	0.75
IABP	482 (7.3)	45 (16.8)	<0.001

Abbreviations: CABG, coronary artery bypass grafting; Cr, creatinine; CS, cardiogenic shock; CVA, cerebrovascular accident; DM, diabetes mellitus; Hb, hemoglobin; HR, heart rate; HTN, hypertension; IABP, intra-aortic balloon pump; MI, myocardial infarction; PCI, percutaneous coronary intervention; SBP, systolic blood pressure; SD, standard deviation; TIMI, Thrombolysis In Myocardial Infarction.

Data are presented as n (%) or mean $\pm$ SD.

We did not have data on causes of mortality, but there are different reasons why patients with STEMI and CS who survived the hospital phase had a worse prognosis. It is likely that the prevalence of severe left ventricular dysfunction was higher in patients with CS. Left ventricular dysfunction is a known to increase mortality risk.⁹ Although prophylactic implantation of implantable cardioverter-defibrillators (ICDs) may decrease mortality in selected patients, current guidelines do not recommend ICD implantation within the first 40 days after STEMI.^{9,10} Moreover, even with ICD, these patients have an increased risk of mortality, for example by progressive heart

FIGURE 1 Landmark analysis: 1-year survival in hospital survivors. Abbreviations: cum, cumulative; CS, cardiogenic shock.

failure. In our study population, we observed that patients who survived a STEMI complicated by CS significantly less frequently had been prescribed an ACEI and β -blocker at discharge, despite the strong indication (41.9% vs 58.7%, respectively, $P < 0.001$; and 78.2% vs 90.0%, $P < 0.001$). A compromised hemodynamic status could be a reason for the less frequent prescription of ACEIs and β -blockers in patients with CS. Currently, the frequency of ACEI use is higher in our hospital, in both all patients with STEMI and those who survive CS.

How can we improve prognosis of STEMI patients who survive the hospital period after CS? Optimal medication at discharge can be improved, especially use of an ACEI after a STEMI or CS.^{11,12}

Risk of one year mortality in hospital survivors of STEMI with CS, as compared to those without CS in several subgroups, adjusted for age and gender

FIGURE 2 Risk of 1-year mortality in hospital survivors of STEMI with CS, as compared to those without CS, in several subgroups, adjusted for age and sex. Abbreviations: CI, confidence interval; CS, cardiogenic shock; CVA, cerebrovascular accident; DM, diabetes mellitus; IABP, intra-aortic balloon pump; MI, myocardial infarction; OR, odds ratio; STEMI, ST-segment elevation myocardial infarction; TIMI, Thrombolysis In Myocardial Infarction.

Current European Society of Cardiology (ESC) guidelines recommend (class 1B) emergency PCI or CABG in patients with CS due to an acute coronary syndrome. In patients treated with primary PCI and additional obstructive coronary artery disease, early complete revascularization during hospital stay should be considered.^{13,14} According to current ESC guidelines, implantation of an ICD is recommended in patients with symptomatic heart failure and a left ventricular ejection fraction $\leq 35\%$ and, in case of an ischemic etiology, >40 days after acute MI.¹⁰ Currently, this is a class 1A recommendation, based on 2 randomized controlled trials.^{15,16} The patients included in the study were all survivors of an MI; however, there was no stratification of those who had CS and those who did not. It could be possible that patients who survive CS have higher mortality due to arrhythmias. Further research is needed to investigate early ICD implantation in survivors of CS.

4.1 | Study Limitations

We assessed the independent prognostic importance of CS in a large cohort of patients with STEMI treated by primary PCI, with 1-year follow-up of all patients. The cohort studied is comparable with other STEMI cohorts. Our study has, however, some limitations. Data on BP and HR were only obtained on admission and not during hospitalization, and hemodynamics after primary PCI are also, or even more, of importance.¹⁷ In the Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock? (SHOCK) trial,¹⁸ 75% of the patients developed CS after admission. In our study, CS was defined as only those who had it at admission, which could have led to misclassification of CS patients into the non-CS group. However, including shock after admission may also cause misclassification,

because any hypotension by other reasons (medication induced, vasovagal, bradycardia) may include patients with “shock.” Also, because duration of admission may vary, this can introduce new bias. Furthermore, classification may be difficult in patients who have bypass surgery during admission. Unfortunately, we do not have data on hemodynamics during hospitalization. Compared with other studies, the 1-year mortality of CS patients in our study was lower.^{1,2} This partially could be explained by the misclassification of CS patients who developed CS during hospitalization. We had no data on invasively measured left ventricular filling pressure or cardiac index; only few patients had right heart catheterization. However, invasive hemodynamic measurement in the setting of a STEMI is not routine daily practice. Furthermore, we had no data on use of inotropics before admission, although this medication is rarely given in the ambulance. We did not have sufficient data on ICD use; therefore, this variable was not included in the multivariate analysis. Finally, we had no data on cause-specific mortality.

5 | CONCLUSION

Cardiogenic shock at admission is still a strong predictor of long-term mortality in hospital survivors of STEMI treated by primary PCI.

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