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Original paper

Impact of cardiac arrest in patients with cardiogenic shock due to ST-elevation myocardial infarction

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Abstract

Background: Cardiogenic shock (CS) frequently complicates ST-elevation myocardial infarction (STEMI) and may be associated with cardiac arrest occurring either as out-of-hospital (OHCA) or in-hospital cardiac arrest (IHCA).

Aim: To compare clinical characteristics, coronary anatomy, management and survival among patients with CS without cardiac arrest (STEMI-CS-no CA), CS with OHCA (STEMI-CS-OHCA) and CS with IHCA (STEMI-CS-IHCA).

Methods: We conducted a retrospective study including consecutive patients with CS and STEMI undergoing immediate coronary angiography and percutaneous coronary intervention (PCI) who were admitted to two tertiary university hospitals between 2016 and 2025.

Results: Among 345 patients, 150 (43.5%) had STEMI-CS-no CA, 120 (34.8%) STEMI-CS-OHCA, and 75 (21.7%) STEMI-CS-IHCA. STEMI-CS-IHCA patients were older, less frequently presented with an initial shockable rhythm (36.0% vs 61.0%, $p = 0.002$) and had shorter time to return of spontaneous circulation (10.0 vs 19.6 min, $p < 0.001$) compared to STEMI-CS-OHCA. They had also lower arterial pressure, left ventricular ejection fraction, estimated glomerular filtration rate and higher arterial lactate compared to STEMI-CS-no CA and STEMI-CS-OHCA. Coronary complexity increased progressively with SYNTAX score rising from 18.6 in STEMI-CS-no CA to 21.5 in STEMI-CS-OHCA and to 27.2 in STEMI-CS-IHCA ($p < 0.001$). At 1-year, all-cause mortality was 67.3% in STEMI-CS-no CA, 78.3% in STEMI-CS-OHCA ($p = 0.004$) and 82.7% in STEMI-CS-IHCA ($p < 0.001$) without significant difference between cardiac arrest subgroups ($p = 0.555$).

Conclusion: In STEMI-related CS, concomitant OHCA or IHCA is associated with distinct clinical profiles, coronary anatomy, intensity of treatment and markedly impaired long-term survival.

Keywords: Cardiogenic shock, STEMI, Cardiac arrest, PCI, Ischemia, Prognosis

Introduction

Cardiogenic shock (CS), which nowadays occurs in 5–15% of patients with ST-elevation myocardial infarction (STEMI), remains the leading cause of hospital death.[1,2] Concomitant cardiac arrest, which accompanies CS in 40–70%, may represent an additional high-risk feature since post resuscitation brain injury frequently results in permanent vegetative state or even brain death.[3] Recent pooled analysis from three randomised trials including IABP-SHOCK II, CULPRIT-SHOCK and ECLS-SHOCK, however, showed differences in baseline and treatment characteristics between CS patients with or without cardiac arrest but no excess in mortality.[4–6] When interpreting this finding, it is important to notice that patients entering randomised clinical trials are carefully selected and results may not reflect realistic clinical scenario. We therefore aimed to

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address demographic, clinical, hemodynamic and angiographic characteristics of CS due to STEMI as well as treatment and survival according to the presence of out-of-hospital cardiac arrest (OHCA) or in-hospital cardiac arrest (IHCA) in a consecutive all-comer cohort presenting to two large tertiary university hospitals.

Methods

This retrospective, observational study included consecutive patients with CS due to STEMI undergoing immediate coronary angiography and primary percutaneous coronary intervention (PCI) who were admitted to the Centre for Intensive Internal medicine, University Medical Centre Ljubljana (Slovenia) and 2nd Department of Internal medicine at General University Hospital Prague (Czech Republic). Both institutions have a high volume "24-7" interventional cardiology service and access to advanced mechanical circulatory support (MCS) ensuring consistent diagnostic and therapeutic standards across the study period. The study was approved by the Institutional Ethics Committees of both participating centres and conducted in accordance with the Declaration of Helsinki. Given the retrospective design and full anonymization of patient data, informed consent was waived.

Cardiogenic shock was defined according to European society of cardiology (ESC) and American heart Association (AHA) guidelines as systolic arterial pressure <90 mm Hg for >30 min or need for vasopressor support or (MCS to achieve that, associated with signs of end-organ hypoperfusion including altered mental status, oliguria and arterial lactate >2 mmol/L.[7,8] Patients were divided into three groups including patients with CS and no cardiac arrest (STEMI-CS-no CA), patients with CS and OHCA (STEMI-CS-OHCA) and patients with CS and IHCA (STEMI-CS-IHCA). Patients with non-cardiac causes of cardiac arrest and patients who did not undergo immediate coronary angiography were excluded.

Demographic and clinical data were retrospectively obtained from institutional electronic health records and certified hospital registries, which prospectively document all cases of CS due to STEMI. Data consistency was verified by cross-referencing emergency, catheterization laboratory, and intensive care databases. Baseline vital signs and laboratory values for STEMI-CS-no CA and STEMI-CS-OHCA were obtained in the catheterization laboratory and for STEMI-CS-IHCA immediately following intensive care admission after coronary angiography and PCI. Consciousness after reestablishment of spontaneous circulation (ROSC) was defined as patient ability to follow verbal commands during the initial neurological assessment in the intensive care unit. Vital status and date of death were ascertained through linkage with the national health insurance registry using each patient's unique health insurance card number. Neurological outcome at 1 year in surviving patients was assessed using Cerebral Performance Category (CPC), which was considered as favourable if CPC 1–2 was documented. When registry data were unavailable or incomplete, survival status was further verified by structured telephone follow-up conducted with the patient or relatives.

Coronary angiograms were analysed offline by a single experienced interventional cardiologist who was blinded to the group assignment. Features of infarct related artery and extend of non-culprit coronary artery disease including $\geq$50% unprotected left main stenosis, number of epicardial vessels with $\geq$50% stenosis, presence of chronic total occlusion and bifurcation involvement were investigated. Complexity of coronary artery disease before and after PCI was quantified by "Synergy between Percutaneous Coronary Intervention with Taxus and Cardiac Surgery" (SYNTAX) score.[9] TIMI flow in culprit artery was assessed before and after primary PCI.

Intensive care management was based on institutional protocols aligned with international recommendations and included inotropic and vasopressor support, mechanical ventilation and MCS with intra-aortic balloon pump (IABP), Impella and veno-arterial extracorporeal membrane oxygenation (VA-ECMO). VA-ECMO was used as mechanical circulatory support for post-resuscitation CS after ROSC. Patients with refractory cardiac arrest treated with extracorporeal cardiopulmonary resuscitation (E-CPR) were not included in the study.

The primary endpoint was all-cause mortality at one year. Secondary endpoints included hemodynamic, metabolic, angiographic parameters, intensive care treatment as well as 30-day mortality and neurological outcome estimated by Cerebral performance scale (CPC) at 1 year. Distribution of continuous variables was evaluated by Shapiro–Wilk and Kolmogorov–Smirnov tests. Normally distributed variables were compared by one-way analysis of variance (ANOVA) followed by Bonferroni-adjusted post-hoc tests for pairwise comparisons. Abnormally distributed variables were compared by Kruskal–Wallis test with Dunn's correction for multiple comparisons. Categorical variables were compared using the χ^2 test or Fisher's exact test as appropriate. Survival was assessed by the Kaplan–Meier method, with group differences evaluated by global and pairwise log-rank tests. Multivariable Cox proportional hazards models were constructed using a hierarchical approach:

- Model 1 included study group (STEMI-CS-no CA, STEMI-CS-OHCA, STEMI-CS-IHCA) and baseline demographic and clinical variables;
- Model 2 additionally included arterial lactate on admission to account for metabolic severity and systemic hypoperfusion;
- Model 3 additionally included baseline SYNTAX score and infarct-related artery TIMI flow to account for coronary anatomical complexity and ischemic burden.

Variables reflecting post-admission management or procedural results were not included to avoid over adjustment. A two-tailed $p < 0.05$ was considered statistically significant. All statistical analyses were performed using SPSS software (version 30.0, IBM Corp., Armonk, NY).

Results

Among 345 consecutive patients admitted between 2016 and 2025 to both institutions, there were 150 STEMI-CS-no CA (43.5%), 120 STEMI-CS-OHCA (34.8%) and 75 STEMI-CS-IHCA (21.7%) patients (Table 1). STEMI-CS-IHCA patients were significantly older compared to STEMI-CS-no CA and STEMI-CS-OHCA and had more often dyslipidaemia. There was no other statistically significant difference in baseline characteristics between the groups.

OHCA occurred predominantly at home (42.5%) or in ambulance (39.2%) and IHCA in the cath lab (33.3%) or following intensive care unit admission (66.7%) (Table 2). OHCA was bystander-witnessed in 45.8%, emergency medical personal witnessed in 33.4% and unwitnessed in 20.8% while each IHCA was witnessed by hospital personal. Initial shockable rhythm was less often documented in STEMI-CS-IHCA patients (36% vs 61%; $p = 0.002$) who had nevertheless shorter delay to ROSC (10.0 vs 19.6 min; $p < 0.001$).

Table 1 – Patient characteristics.

	STEMI-CS-no CA (*n* = 150)	STEMI-CS-OHCA (*n* = 120)	STEMI-CS-IHCA (*n* = 75)	*p*
Age, years	65 ± 9	66 ± 10	69.0 ± 9	0.008
Man	119 (79.3%)	89 (74.2%)	58 (77.3%)	0.603
BMI (kg/m²)	26.6 ± 4.1	26.8 ± 3.9	27.5 ± 3.4	0.267
Diabetes	47 (31.3%)	32 (26.7%)	21 (28.0%)	0.687
Hypertension	88 (58.7%)	66 (55.0%)	47 (62.7%)	0.567
Dyslipidemia	84 (56.0%)	57 (47.5%)	49 (65.3%)	0.049
Current smoker	30 (20.0%)	31 (25.8%)	17 (22.7%)	0.523
PAD	9 (6.0%)	11 (9.2%)	7 (9.3%)	0.541
Prior stroke/TIA	14 (9.3%)	9 (7.5%)	7 (9.3%)	0.752
Prior MI/PCI/CABG	30 (20.0%)	30 (25.0%)	16 (21.3%)	0.607

STEMI = ST elevation myocardial infarction; CS = Cardiogenic shock; CA = Cardiac arrest; OHCA = Out-of-hospital cardiac arrest; IHCA = In-hospital cardiac arrest; BMI = Body mass index; eGRF = Estimated glomerular filtration; PAD = Peripheral artery disease; TIA = Transitory ischemic attack; MI = Myocardial infarction; PCI = Percutaneous coronary intervention; CABG = Coronary artery bypass grafting.

Consciousness after ROSC was regained in about one third of cardiac arrest patients without significant difference between the groups. Systolic arterial pressure in STEMI-CS-IHCA (76 mmHg) was significantly lower than in STEMI-CS-no CA (90 mmHg; $p < 0.001$) and STEMI-CS-OHCA (83 mmHg; $p = 0.020$). This is true also for left ventricular ejection fraction and estimated glomerular filtration rate. Arterial lactate progressively increased from STEMI-CS-no CA (3.5 mmol/L) to STEMI-CS-OHCA (5.4 mmol/L) and STEMI-CS-IHCA 6.8 mmol/L; $p < 0.001$). Vasopressors (80.0%) and mechanical ventilation (72.0%) were more frequently used in STEMI-CS-IHCA ($p < 0.010$) while IABP (41.7%; $p = 0.032$) and VA ECMO (25.0%; $p < 0.001$) in STEMI-CS-OHCA patients (Table 2). Use of Impella varied from 18.7% to 26.7% without significant difference between the groups ($p = 0.297$).

SYNTAX score progressively increased from STEMI-CS-no CA (18.6 ± 7.7) to STEMI-CS-OHCA (21.5 ± 7.2) and STEMI-CS-IHCA (27.2 ± 10.1) ($p < 0.001$) with number of vessels with >50% stenosis following the same pattern (Table 3). Chronic total occlusion was most often present in STEMI-CS-OHCA (33.3%) while bifurcation lesions in STEMI-CS-IHCA. Distribution of infarct related artery varied significantly between the groups with unprotected left main (20.0%) and left anterior descending artery (61.3%) being most prevalent in CS-IHCA ($p < 0.001$). Acute occlusion of infarct artery (TIMI 0–1) was documented in 13.3%, 63.3% and 29.3% ($p < 0.001$) in STEMI-CS-no CA, STEMI-CS-OHCA and STEMI-CS-IHCA groups, respectively. Primary PCI resulted in patent infarct related artery with TIMI 2–3 flow in 95.8% in STEMI-CS-OHCA while the result was less favourable in STEMI-CS-no CA (88.0%) and STEMI-CS-IHCA (85.3%) ($p = 0.006$). Residual SYNTAX score followed the preprocedural pattern.

At 30 days, cumulative mortality was 41.3% of STEMI-CS-no CA, 65.8% in STEMI-CS-OHCA and 72.0% in STEMI-CS-IHCA ($p < 0.001$) (Fig. 1). There was no significant difference between STEMI-CS-OHCA and STEMI-CS-IHCA ($p = 0.539$). At 1-year, cumulative mortality further increased to 67.3% in STEMI-CS-no CA, to 78.3% in STEMI-CS-OHCA and to 82.7% in STEMI-CS-IHCA patients (Fig. 2). Mortality was significantly lower in STEMI-CS-no CA compared to both, STEMI-CS-OHCA ($p = 0.004$) and STEMI-CS-IHCA ($p < 0.001$) whereas there were no significant differences between STEMI-CS-OHCA and CS–IHCA patients ($p = 0.555$). Median survival time was substantially longer in STEMI-CS-no CA (52 days, 95% CI 23.8–80.2 days) than in STEMI-CS-OHCA (11 days, 95% CI 5.6–16.4 days) and STEMI-CS-IHCA (10 days, 95% CI 5.3–14.7 days) confirming that excess mortality associated with cardiac arrest was predominantly confined to the early phase of treatment. In surviving patients CPC 1–2 at 1-year was significantly higher in STEMI-CS-no CA (89.9%) compared to STEMI-CS-OHCA (57.7%) or STEMI-CS-IHCA (69.2%) ($p = 0.005$).

Across all models, CS complicated by cardiac arrest was consistently associated with increased 1-year mortality compared to CS without cardiac arrest. Adjustment for metabolic severity (Model 2) and coronary anatomical complexity (Model 3) attenuated but did not abolish the association between cardiac arrest type and long-term mortality (Table 4). No significant difference in mortality was observed between STEMI-CS-OHCA and STEMI-CS-IHCA patients in any of the multivariable models.

Discussion

Our study confirmed that significant proportion of CS patients with STEMI suffers concomitant OHCA (35.0%) or IHCA (22.0%) resulting in more than 60.0% of comatose survivors who do not regain consciousness immediately after ROSC. STEMI-CS-no CA, STEMI-CS-OHCA and STEMI-CS-IHCA patients differ significantly in terms of initial presentation including age, arterial pressure, left

Table 2 – Admission features and treatment.

	STEMI-CS-no CA (*n* = 150)	STEMI-CS-OHCA (*n* = 120)	STEMI-CS-IHCA (*n* = 75)	*p*
Site of cardiac arrest				NA
Home	NA	51(42.5%)	NA	
Public	NA	22 (18.3%)	NA	
Ambulance	NA	47 (39.2%)	NA	
Cath lab	NA	NA	25 (33.3%)	
ICU	NA	NA	50 (66.7%)	
Witnessed, *n* (%)				<0.001
Lay bystander	NA	55 (45.8%)	0	
EMT/HP	NA	40 (33.4%)	75 (100%)	
Unwitnessed	NA	25 (20.8%)	0	
Bystander CPR, *n* (%)	NA	97 (80.8%)	NA	NA
Initial shockable rhythm	NA	73 (61.0%)	27 (36.0%)	0.002
Cardiac arrest to ROSC, minutes	NA	19.6 ± 8.3	10.0 ± 5.0	<0.001
Consciousness after ROSC	NA	45 (38%)	26 (35%)	0.689
SAP, mmHg	90 ± 14	83 ± 17	76 ± 10	<0.001
HR, bmp	97 ± 21	94 ± 20	92 ± 18	0.202
LVEF, %	40 ± 8	37 ± 9	31 ± 9	<0.001
eGFR, mL/min/1.73 m^2	74.9 ± 11.6	75.1 ± 12.2	60.8 ± 16.0	0.007
pH	7.31 ± 0.52	7.12 ± 0.12	7.10 ± 0.70	<0.001
Arterial lactate, mmol/L	3.5 ± 1.5	5.4 ± 1.9	6.8 ± 3.0	<0.001
Intensive care treatment				
Inotropes	102 (68.0%)	94 (78.3%)	59 (78.7%)	0.090
Vasopressors	74 (49.3%)	90 (75.0%)	60 (80.0%)	<0.001
Mechanical ventilation	61 (40.7%)	74 (61.7%)	54 (72.0%)	<0.001
IABP	40 (26.7%)	50 (41.7%)	27 (36.0%)	0.032
VA ECMO	8 (5.3%)	30 (25.0%)	10 (13.3%)	<0.001
Impella	28 (18.7%)	30 (25.0%)	20 (26.7%)	0.297
CPC 1–2 at 1-year	44 (89.8%)	15 (57.7%)	9 (69.2%)	0.005

STEMI = ST elevation myocardial infarction; CS = Cardiogenic shock; CA = Cardiac arrest; OHCA = Out-of-hospital cardiac arrest; IHCA = In-hospital cardiac arrest; ICU = Intensive care unit; CPR = Cardiopulmonary resuscitation; EMT = Prehospital emergency medical team; HP = Witnessed by hospital personal; ROSC = Reestablishment of spontaneous circulation; SAP = Systolic arterial pressure; HR = heart rate; LVEF = Left ventricular ejection fraction; eGFR = Estimated glomerular filtration rate. IABP = Intraaortic balloon pump; VA ECMO = Veno- arterial extracorporeal membrane oxygenation; CPC = Cerebral performance category calculated among 1-year survivors.

Table 3 – Angiographic features.

	STEMI-CS-no CA (*n* = 150)	STEMI-CS-OHCA (*n* = 120)	STEMI-CS-IHCA (*n* = 75)	*p*
Initial SYNTAX score	18.6 ± 7.7	21.5 ± 7.2	27.2 ± 10.1	<0.001
Vessels with ≥50% stenosis	1.7 ± 0.9	2.0 ± 0.8	2.6 ± 0.9	<0.001
≥1 CTO	4 (2.7%)	40 (33.3%)	9 (12.0%)	<0.001
Bifurcation	46 (30.7%)	33 (27.5%)	48 (64.0%)	<0.001
IRA with TIMI 0-1 (%)	20 (13.3%)	76 (63.3%)	22 (29.3%)	<0.001
IRA distribution				<0.001
LM	4 (2.7%)	11 (9.2%)	15 (20.0%)	
LAD	55 (36.7%)	65 (54.2%)	46 (61.3%)	
LCX	35 (23.3%)	20 (16.7%)	4 (5.3%)	
RCA	56 (37.3%)	24 (20.0%)	10 (13.3%)	
Final TIMI 2–3 after primary PCI	132 (88.0%)	115 (95.8%)	65 (85.3%)	0.006
Residual SYNTAX score	6.7 ± 3.7	8.7 ± 5.4	12.3 ± 5.8	<0.001

STEMI = ST elevation myocardial infarction; CS = Cardiogenic shock; CA = Cardiac arrest; OHCA = Out-of-hospital cardiac arrest; IHCA = In-hospital cardiac arrest; CTO = Chronic total occlusion; IRA = Infarct related artery; LM = Left main; LAD = Left anterior descending artery; LCX = Left circumflex artery; RCA = Right coronary artery; PCI = Percutaneous coronary intervention.

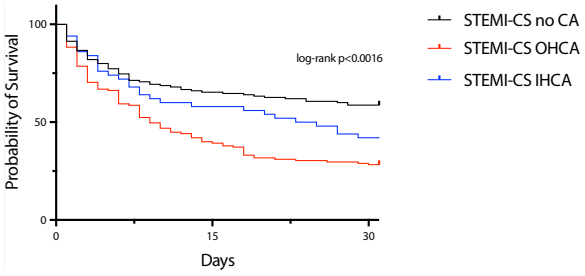

Fig. 1 – Kaplan-Meier estimate of 30-day survival in consecutive patients with CS and no cardiac arrest (STEMI-CS-no CA), patients with CS and OHCA (STEMI-CS-OHCA) and patients with CS and IHCA (STEMI-CS-IHCA).

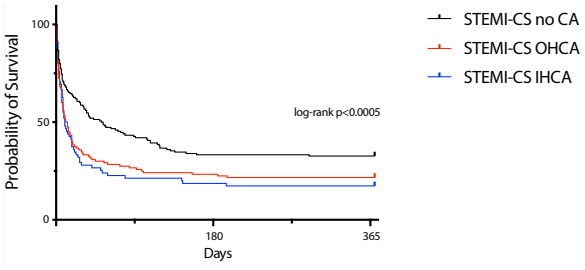

Fig. 2 – Kaplan-Meier estimate of 1-year survival in consecutive patients with CS and no cardiac arrest (STEMI-CS-no CA), patients with CS and OHCA (STEMI-CS-OHCA) and patients with CS and IHCA (STEMI-CS-IHCA).

ventricular ejection fraction, glomerular filtration rate, pH and arterial lactate. Among these groups, STEMI-CS-IHCA patients seem to be more hemodynamically and metabolically compromised. This severity of deterioration may have been even underestimated because two thirds of patients suffered IHCA in intensive care after initial assessment. More severe hemodynamic and metabolic deterioration is reflected also in intensity of intensive care treatment including vasopressors, mechanical ventilation and especially MCS which should be, however, not used routinely but only in carefully selected patients with good neurological prognosis.[10,11]

Using SYNTAX score we further documented progressive complexity of coronary artery disease from STEMI-CS-no CA to STEMI-CS-OHCA and STEMI-CS-IHCA with significant unprotected left main stenosis and bifurcation lesions being more prevalent in STEMI-CS-IHCA and chronic total occlusion in STEMI-CS-OHCA. We documented also significant differences in infarct related artery distribution and incidence of acute occlusion with TIMI 0–1 flow. Unprotected left main and left anterior descending artery, which are as infarct related arteries typically associated with larger amount of myocardium at ischemic risk in case of acute occlusion, were found more often in patients with concomitant cardiac arrest. This may indicate that increased complexity of coronary disease and larger amount of myocardium at ischemic risk together with myocardial scaring related to chronic total occlusion, may predispose CS patients to concomitant cardiac arrest.

Importantly, observed demographic, clinical and angiographic differences between different groups of CS patients resulted in significant differences in 30-day and 1-year mortality, which was lower in patients with STEMI-CS-no CA compared with those with CS complicated by OHCA or IHCA. This in contrast with pooled analysis of three randomised trials including IABP-SHOCK II, CULPRIT-

Table 4 – Multivariable Cox regression for 1-year all-cause mortality.

Variable	Model 1 HR (95% CI)	Model 2 HR (95% CI)	Model 3 HR (95% CI)
OHCA vs no CA	1.47 (1.11–1.96)	1.58 (1.03–2.42)	1.46 (1.07–2.00)
IHCA vs no CA	1.72 (1.20–2.48)	1.80 (1.19–2.72)	1.85 (1.25–2.74)
Age (per year)	1.00 (0.98–1.01)	1.00 (0.98–1.01)	1.00 (0.98–1.01)
Man	0.73 (0.55–0.97)	0.73 (0.55–0.97)	0.75 (0.56–1.00)
Diabetes	1.17 (0.90–1.53)	1.18 (0.90–1.54)	1.17 (0.89–1.53)
Dyslipidemia	1.21 (0.94–1.56)	1.20 (0.93–1.55)	1.20 (0.93–1.54)
Hypertension	1.00 (0.78–1.28)	1.00 (0.78–1.28)	0.98 (0.76–1.26)
eGFR	1.00 (0.99–1.01)	1.00 (0.99–1.01)	1.00 (0.99–1.01)
Lactate, mmol/L	–	0.98 (0.91–1.07)	–
Baseline SYNTAX score	–	–	0.99 (0.98–1.01)
IRA TIMI 0–1	–	–	1.08 (0.81–1.43)

OHCA = Out-of-hospital cardiac arrest; CA = Cardiac arrest; IHCA = In-hospital cardiac arrest; eGRF = Estimated glomerular filtration; IRA = Infarct related artery.

SHOCK and ECLS-SHOCK, which did not distinguish between OHCA and IHCA, and found similar 1-year mortality compared to CS without cardiac arrest (hazard ratio 1.05; 95% confidence interval 0.92–1.20). The most likely explanation for this discrepancy is that these randomised CS studies excluded patients with prolonged cardiac arrest and cardiopulmonary resuscitation who have low likelihood of good neurological outcome and long-term survival.[4–6,12] Our results are in accordance with contemporary multicentre analyses in which cardiac arrest remained an independent predictor of long-term mortality of CS also after adjusting for angiographic and hemodynamic variables.[13–15] Moreover, the need for stratification by cardiac arrest type and baseline shock severity for future studies evaluating MCS is further underscored by recent post hoc analysis of ECLS-SHOCK randomised trial.[16]

The hierarchical modelling strategy adopted in the present study allowed us to explore whether the excess long-term mortality associated with cardiac arrest in CS was explained by baseline patient characteristics, metabolic derangement or coronary anatomical complexity. While adjustment for metabolic severity and coronary anatomy attenuated the hazard ratios, the association between cardiac arrest type and 1-year mortality remained significant across all models. This finding suggests that cardiac arrest identifies a distinct high-risk CS subgroup that is not fully captured by individual markers of systemic hypoperfusion or coronary disease burden. Importantly, metabolic and anatomical variables were intentionally incorporated in separate models to minimise collinearity and avoid over adjustment, as these factors are intrinsically linked to the clinical presentation of CS complicated by cardiac arrest.

Limitations

Our results should be interpreted in view of several limitations. Despite enrolment of consecutive unselected patient population admitted to two high volume tertiary centres and standardised data collection, our study was retrospective and observational. Interpretation of coronary angiography was not performed by a core lab but by a single interventional cardiologist blinded to group assignment. Moreover, grouping patients according to the presence and setting of cardiac arrest may be considered somewhat artificial since onset of cardiac arrest can be a time-dependent surrogate of ischemic burden. Site of cardiac arrest in terms of OHCA and IHCA may therefore be influenced by patient-related delay, prehospital transport time and in-hospital logistics. Ideally, patients should be stratified according to the total time from symptom onset to cardiac arrest. Unfortunately, time variables such as symptom onset to first medical contact, hospital arrival and reperfusion were not a part of the institutional registries. Due to the retrospective design of our study and non-uniform reporting, we were also not able to further classify IHCA onset before, during or immediately after the cath lab procedure. A very useful SCAI shock state on admission is not described because it was not systematically used during the study period. Furthermore, we cannot provide major in-hospital complications and causes of death, which were also not captured in the institutional registries. Although we could document survival and CPC after 1 year, more detailed neurological assessment along with hemodynamic status could not be obtained.

Conclusion

Our study reflects consecutive, unselected CS patients with STEMI including both, OHCA and IHCA, thus providing a realistic representation of the contemporary population. While STEMI-CS-no CA represents the mechanically “reperfusable” shock, STEMI-CS-OHCA may be viewed as “reperfusable” but neurologically fragile form, and STEMI-CS-IHCA as structurally advanced and metabolically exhausted form combined by neurological fragility. The integration of OHCA-IHCA and coronary assessment provide a new framework for interpreting CS not as a monolithic syndrome but as a family of related pathophysiological states. Future randomised studies in CS should therefore respect these differences to properly design study protocols.

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CRediT authorship contribution statement

Danilo Franco: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Jan Belohlavek:** Writing – review & editing, Supervision. **Daniel Rob:** Formal analysis, Data curation. **Tomas Kovarnik:** Data curation, Investigation, Methodology. **Tomaz Goslar:** Methodology, Data curation. **Misa Fister:** Data curation. **Peter Radsel:** Validation, Data curation. **Raffaele Izzo:** Validation, Supervision, Data curation. **Giuseppe Di Gioia:** Formal analysis, Data curation. **Giovanni Esposito:** Validation, Supervision, Methodology, Investigation, Conceptualization. **Marko Noc:** Writing – review & editing, Writing – original draft, Methodology, Investigation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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