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CLINICAL STUDY

Use of intraaortic balloon pump in cardiogenic shock patients

Peter MISUN[1], Marek SRAMKO[1], Josef KAUTZNER[1], Jiri KETTNER[1], Michal ZELIZKO[1], Jan MARES[1], Allan BOHM[2,3], Marta KOLLAROVA[2], Branislav BEZAK[2,3], Michal PAZDERNIK[1]

Institute for Clinical and Experimental Medicine (IKEM), Prague, Czech Republic.
michal.pazdernik@ikem.cz

ABSTRACT
BACKGROUND AND OBJECTIVES: The relevance of the use of intra-aortic balloon pump (IABP) in cardiogenic shock (CS) has been discussed over the past years. The aim of this study is to describe a single-centre 10-year experience with IABP and analyse the risk factors for 30-day mortality.
METHODS: The data for this single-centre, observational, retrospective study were drawn from records dated from January 2012 to May 2022 pertaining to patients presenting with CS, treated with IABP and hospitalised at the Department of Acute Cardiology, Institute for Clinical and Experimental Medicine, Prague.
RESULTS: Among the patients included in the study, 87% patients presented with newly developed heart failure. The leading cause of CS was acute myocardial infarction accounting for 86% of cases. Hospital mortality was recorded at 39% and the 30-day mortality reached 43%. Upon multi-variable analysis, only the vasoactive inotropic score on day 5 emerged as a statistically significant predictor for 30-day mortality (p=0.0055). Cox regression analysis revealed that the presence of mechanical complications was the only variable identified as yielding a statistically significant impact on the 30-day survival (Log-rank p=0.014, HR 2.19, 95% CI: 1.15–4.15). There was no statistically significant difference in the 30-day mortality across the SCAI classes.
CONCLUSION: The main cause of CS was a newly developed acute heart failure secondary to acute myocardial infarction. Despite the implementation of mechanical circulatory support, both in-hospital and 30-day mortality rates remained high. Increased vasoactive inotropic score and presence of mechanical complications were identified as significant predictors the 30-day survival *(Tab. 6, Fig. 1, Ref. 36)*. Text in PDF www.elis.sk
KEY WORDS**:** cardiogenic shock, IABP, risk factors, mortality, Czech Republic, AMICS.

Abbreviations: ACEi – angiotensin-converting enzyme inhibitors, ACS – acute coronary syndrome, AMI – acute myocardial infarction, AMICS – acute myocardial infarction with cardiogenic shock, ARB – angiotensin receptor blockers, ARNI – angiotensin receptor/neprilysin inhibitor, AVR – aortic valve replacement, BB – beta blockers, BMI – body mass index, CABG – coronary artery bypass grafting, CAD – coronary artery disease, CI – confidence interval, CKD – chronic kidney disease, COPD – chronic obstructive pulmonary disease, CRT-D – cardiac resynchronization therapy – defibrillator, CRT-P – cardiac resynchronization therapy – pacemaker, CS – cardiogenic shock, CT – computed tomography, HB – haemoglobin, HF – heart failure, HFpEF – heart failure with preserved ejection fraction, HFrEF – heart failure with reduced ejection fraction, CHF – chronic heart failure, IABP – intra-aortic balloon pump, ICD – implantable cardioverter defibrillator, ICU – intensive care unit, IKEM – Institute for Clinical and Experimental Medicine, IQR – interquartile range, LDH – lactate dehydrogenase, LVAD – left-ventricle assist device, MAP – mean arterial pressure, MCS – mechanical circulatory support, MI – myocardial infarction, MRA – mineralocorticoid receptor antagonist, MVR – mitral valve replacement, OR – odds ratio, PCI – percutaneous coronary intervention, PM – pacemaker, SCAI – society for cardiovascular angiography and interventions, SD – standard deviation, SGLT2 – sodium-glucose cotransporter-2, SIRS – systemic inflammatory response syndrome, TIMI – thrombolysis in myocardial infarction, VA-ECMO – veno-arterial extracorporeal membrane oxygenation, VSD – ventricular septal defect

Introduction

Cardiogenic shock (CS) is the leading cause of in-hospital mortality in patients with acute myocardial infarction (AMI) (1). Despite recent advancements in diagnostic and therapeutic approaches, the mortality rate remains high (2), reaching almost 50% (3).

With the introduction of various mechanical circulatory support (MCS) devices (4). the management of CS has undergone significant advancements in optimising cardiac function and improving patient outcomes. However, this progress has not yet yielded a substantial impact on patient survival.

[1]Department of Cardiology, Institute for Clinical and Experimental Medicine (IKEM), Prague, Czech Republic, [2]Premedix Academy, Bratislava, Slovakia, and [3]Faculty of Medicine, Comenius University in Bratislava, Bratislava, Slovakia

Address for correspondence: Michal PAZDERNIK, MD, Assoc Prof, PhD, FESC, Institute for Clinical and Experimental Medicine (IKEM), 140 21 Prague, Czech Republic.
Phone: +420 602 258544

Over the past decades, the utility and relevance of the intra-aortic balloon pump (IABP) in the setting of acute coronary syndrome (ACS) have faced scrutiny, particularly after the publication of the IABP SHOCK II trial by Thiele et al in 2012 (5). While current evidence and the guidelines do not recommend the use of IABP in the setting of post-MI CS, recent studies have shown potential benefits of IABP in some of high-risk subsets of patients in scenarios such as anterior STEMI or persistent ischemia after pPCI (6–9). Moreover, despite evolving recommendations, IABP remains widely adopted worldwide owing to its cost-effectiveness, low complication rates, and ease of use (10).

Further research that would help identify subgroups of patients who could benefit from IABP in the setting of ACS or CS is therefore warranted.

The objective of this study was to analyse the population of patients suffering from CS treated with IABP and to identify potential factors impacting 30-day mortality.

Tab. 1. Patient's demographics and medical history.

	Mean±SD/N (%)
Age (years)	67.2±10.37
Male sex	77 (77%)
Transfer from another hospital	42 (42%)
BMI	28.77±4.66
Treated diabetes	30 (30%)
Treated hypertension	66 (66%)
Treated dyslipidaemia	32 (32%)
Current smoker	24 (24%)
CAD history	17 (17%)
Previous MI	13 (13%)
Prior coronary revascularisation	13 (13%)
• PCI	7 (54%)
• CABG	2 (15,3%)
• PCI+CABG	4 (30,7%)
Atrial fibrillation	12 (12%)
CKD	17 (17%)
• No dialysis	15 (88,2%)
• Dialysis	2 (11,8%)
COPD	5 (5%)
Stroke	7 (7%)
Intervention for peripheral artery disease	8 (8%)
Previous dg. of HF	13 (13%)
• HFrEF	12 (92%)
• HFpEF	1 (8%)
>2 hospitalisations for HF	10 (10%)
Previous treatment with BB	35 (35%)
Previous treatment with ACEi, ARB or ARNI	43 (43%)
Previous treatment with MRA	12 (12%)
Previous treatment with SGLT-2 inhibitors	1 (1%)
Implantable cardiac device	13 (13%)

ACEi – angiotensin-converting enzyme inhibitors, ARB – angiotensin receptor blockers, ARNI – angiotensin receptor/neprilysin inhibitor, BB – beta blockers, BMI – body mass index, CABG – coronary artery bypass grafting, CAD – coronary artery disease, CKD – chronic kidney disease, COPD – chronic obstructive pulmonary disease, HF – heart failure, HFrEF – heart failure with reduced ejection fraction, HFpEF – heart failure with preserved ejection fraction, MI – myocardial infarction, MRA – mineralocorticoid receptor antagonist, PCI – percutaneous coronary intervention, SGLT2 – Sodium-glucose cotransporter-2

Methods

The main objective of this study was to analyse the demographics and hospitalisation data of patients presenting with CS treated with IABP. The secondary objective was to identify potential risk and protective factors associated with 30-day mortality.

Study design and data collection

This study was designed as a retrospective, observational, single-centre study.

The data were drawn from records dated from January 2012 to May 2022. Inclusion criteria encompassed the use of IABP support in CS and hospitalisation at the cardiologic ICU in the Institute for Clinical and Experimental Medicine (IKEM) between January 2012 and May 2022. Diagnosis of CS was based at the discretion of the attending physician, with CS stages classified according to the SCAI Shock Classification Expert Consensus (11).

Exclusion criteria comprised the transfer of patients after insertion of IABP at another centre, ongoing CPR on admission or the presence of other causes of shock such as sepsis, anaphylaxis, or hypovolaemia.

The data was obtained from the IKEM hospital information system. A total of 206 cases were reviewed, of whom 100 patients meeting all of the inclusion and none of the exclusion criteria were included in the analysis.

All procedures were performed in compliance with the principles outlined in the Declaration of Helsinki.

Baseline data

Baseline data included demographics, clinical characteristics, medical history, duration of symptoms, treatment, use of MCS devices and their duration, causes of CS, complications during the hospital stay and in-hospital and 30-day mortality rates.

Statistical analysis

Descriptive statistics are presented as means with standard deviations, medians with interquartile ranges, or as counts and percentages.

Predictors of 30-day mortality were analysed using the logistic regression model. First, a separate model was fitted for each variable. A subset of statistically significant predictors from the separate models were included into a single multivariate logistic regression model. To optimise the number of variables included in the model and to avoid overfitting, only one variable was chosen in case of similar variables *(e.g., vasoactive inotropic scores on days 5 and 7, mean and diastolic arterial blood pressure values).*

Kaplan–Meier curve analysis with a log-rank test and the Cox proportional hazard model were performed to analyse the effect of individual variables on the 30-day survival.

Results from the logistic regression and survival analyses are presented as odds ratios or hazard ratios along with 95% confidence intervals.

The standard alpha level of 5% was used to assess statistical significance ($p<0.05$ and 95% confidence intervals for OR or HR not containing the value 1). The ps and the confidence intervals

were not corrected for multiple testing, and therefore must be interpreted with caution, primarily in an exploratory fashion or in combination with other research results, i.e., not as conclusive evidence on their own.

The statistical analysis was performed using R version 4.2.2.

Results

Patient demographics and medical history:

The study cohort included 100 adult patients. Their mean age was 67.2±10.37 years and 77% of the subjects were male. The summary of their demographic characteristics and medical history data is presented in Table 1.

Patient characteristics at the time of admission:

The majority of patients (87%) presented with newly developed heart failure, with AMI being the leading cause of CS, accounting for 86% of cases.

Mechanical complications of acute myocardial infarction were observed in 36 cases, distributed as follows: ventricular septal defect (26 cases), rupture of the free wall of the left ventricle (2 cases), rupture of the papillary muscles of the mitral valve with consecutive acute mitral valve regurgitation (8 cases).

According to the CS stage, 27% of cases were categorised as class B, 60% as class C, and 13% as class D of SCAI classification.

Upon admission, the mean arterial pressure (MAP) was 82.66 mmHg (±17.9), and the mean arterial lactate was 4.299 mmol/L.

Tab. 2. Patient characteristics at the time of admission.

On admission		Mean±SD/N (%)	Median/(Q1;Q3)
Type of the current episode of HF	Newly developed	87 (87%)	
	Acute decompensation	13 (13%)	
Cause of current episode of CS	AMICS	86 (86%)	
	Valvular heart disease	3 (3%)	
	Dilated cardiomyopathy	3 (3%)	
	Chronic ischaemic cardiomyopathy	4 (4%)	
	Other	4 (4%)	
In patients with AMICS – duration of MI symptoms	0–6 hours	19 (23%)	
	6–24 hours	23 (28%)	
	>24 hours	41 (49%)	
Ventricular septal defect		26 (26%)	
Rupture of the free wall of the left ventricle		2 (2%)	
Papillary muscle necrosis – acute mitral regurgitation		8 (8%)	
Obvious trigger of acute decompensation of CHF	Infection	1 (8%)	
	Ischaemia	3 (23%)	
	No	7 (54%)	
	Other	2 (15%)	
Prior resuscitation		16 16%	
Use of vasopressors	Noradrenaline	27 (27%)	
	Vasopressin	3 (3%)	
Inotropes on admission	Dobutamine	7 (7%)	
	Milrinone	1 (1%)	
	Levosimendan	1 (%)	
SCAI class	B	27 (27%)	
	C	60 (60%)	
	D	13 (13%)	
Non-invasive ventilation		5 (5%)	
Mechanical ventilation		23 (23%)	
MAP		82.66±17.91	83.0/(70;94)
Creatinine		161.5±96.74	124.55/(100.7;187)
Heart rate (bpm)		107.9±80.30	100/(89;117)
pH		7.306±0.17	7.36/(7.22;7.42)
Arterial lactate (mmol/l)		4.299±4.26	2.75/
Serum glucose (mmol/l)		12.88±5.36	11.40/7.4
Left ventricular ejection fraction (%)		33.97±11.60	35/15

HF – heart failure, IQR – interquartile range, AMICS – acute myocardial infarction with cardiogenic shock, MI – myocardial infarction, CHF – chronic heart failure, MAP – mean arterial pressure, SCAI – Society for Cardiovascular Angiography and Interventions, SD – standard deviation, BMI – body mass index, CAD – coronary artery disease, PCI – percutaneous coronary intervention, CABG – coronary artery bypass grafting, atrial fibrillation, CKD – chronic kidney disease, COPD – chronic obstructive pulmonary disease, HF – heart failure, HFrEF – heart failure with reduced ejection fraction, HFpEF – heart failure with preserved ejection fraction, BB – beta-blockers, ACEi – angiotensin-converting enzyme inhibitors, ARB – angiotensin receptor blockers, ARNI – angiotensin receptor-neprilysin inhibitors, MRA – mineralocorticoid receptor antagonists, SGLT-2 inhibitors – sodium-glucose cotransporter-2 inhibitors, PM – pacemaker, ICD – implantable cardioverter defibrillator, CRT-P – cardiac resynchronization therapy – pacemaker, CRT-D – cardiac resynchronization therapy – defibrillator

Tab. 3. Summary of treatment modality

Treatment		
Patients presenting with AMICS – Treatment	CABG	16 (18.8%)
	Conservative Treatment	25 (29.4%)
	PCI	38 (44.7%)
	PCI+CABG	6 (7.1%)
AMICS culprit lesion treated with PCI – Flow	PCI with TIMI flow 0	3 (6.8%)
	PCI with TIMI flow 1	9 (20.5%)
	PCI with TIMI flow 2	11 (25%)
	PCI with TIMI flow 3	21 (47.7%)
AMICS treated with CABG – Number of grafts	1	5 (22.7%)
	2	11 (50%)
	3	3 (13.6%)
	4	3 (13.6%)
AMICS treated with CABG – Type of surgery	CABG + valvular surgery (MVR/AVR)	10 (45.5%)
	CABG	12 (54.5%)
Non-coronary non-surgical intervention		10 (10%)
Surgical intervention for mechanical complication of AMI	VSD surgery	19 (19%)
	MVR	7 (7%)
	Suture of the free wall of LV	1 (1%)
Impella		3 (3%)
Type of Impella device	5.0	1 (33%)
	CP	2 (66%)
VA-ECMO		17 (17%)

AMICS – acute myocardial infarction with cardiogenic shock, CABG – coronary artery bypass grafting, PCI – percutaneous coronary intervention, MVR – mitral valve replacement, AVR – aortic valve replacement, VSD – ventricular septal defect, TIMI – thrombolysis in myocardial infarction, IABP – intra-aortic balloon pump, VA-ECMO – veno-arterial extracorporeal membrane oxygenation

Tab. 4. Summary of the clinical course.

Clinical course	Mean±SD/N (%)	Median/IQR
Total duration of Impella support (days)	9.33±4.04	
Total duration of IABP support (Days)	6.364±4.69	5/6
Total duration of VA-ECMO support (Days)	8.824±6.52	10/8
Noradrenaline used	91 (91%)	
Vasopressin used	42 (42%)	
Number of days on vasopressors	12.84±22.2	7/14
Dobutamine used	50 (50%)	
Milrinone used	58 (58%)	
Levosimendan used	55 (55%)	
Number of days on inotropes	8.476±7.29	7/8
Arterial lactate day 1 (mmol/l)	3.546±3.49	2.4/1.5
Arterial lactate day 3 (mmol/l)	3.272±9.57	1.45/0.925
Arterial lactate day 5 (mmol/l)	2.122±2.94	1.40/0.9
Vasoactive-Inotropic Score on day 1	40.3±92.55	10/40
Vasoactive-Inotropic Score on day 3	98.91±49.16	5.5/30
Vasoactive-Inotropic Score on day 5	30.98±64.14	5/23
Renal replacement therapy	46 (46%)	
Therapeutic hypothermia	1 (1%)	
Sepsis (SIRS criteria and ≥2 positive blood cultures)	30 (30%)	
Pulmonary oedema	38 (38%)	
Invasive CO output measurement	28 (28%)	
Duration of non-invasive ventilation (days)	0.1538±0.54	
Duration of invasive ventilation (days)	11.55±15.0	

IQR – Interquartile range, IABP – intra-aortic balloon pump, VA-ECMO – veno-arterial extracorporeal membrane oxygenation, SIRS – systemic inflammatory response syndrome

The summary of patient characteristics at the time of admission is presented in Table 2.

Treatment

Most of the patients presenting with AMICS (86%) were treated with PCI (44.7%), 18.8% underwent coronary artery bypass grafting (CABG) and 7.1% were treated with PCI combined with CABG. Twenty-five patients (29.4%) were managed conservatively (Tab. 3).

Clinical course

Mean duration of IABP support was 6.36 (±4.69) days. Regarding vasopressors, noradrenaline was used in 91%, while vasopressin was utilised in 42% of cases. The mean duration of vasopressor support was 12.84 (±22.2) days. Regarding inotropes, the use of dobutamine, milrinone and levosimendan was almost equally frequent (50%, 58%, 55%). Renal replacement therapy was required in 46% of patients. Sepsis developed in 30% of patients, and pulmonary oedema in 38% of cases. Overall data pertaining to the clinical course is presented in Table 4.

Tab. 5. Complications during hospital stay.

Left ventricular thrombus formation	3 (3%)
Hypoxic brain damage on CT	6 (6%)
Intracerebral bleeding on CT	1 (1%)
Ischemic stroke on CT	2 (2%)
Haemorrhagic stroke on CT	0 (0%)
Moderate bleeding (transfusion of red blood cells without hemodynamic impairment)	19 (19%)
Life-threatening or severe bleeding (haemodynamic impairment with intervention or intracranial haemorrhage)	18 (18%)
Any surgical intervention due to bleeding	20 (20%)
Haemolysis (LDH ≥1000 µkat/l and/or free Hb >100 mg/l and/or haptoglobin <0,3 g/l in 2 consecutive blood samples within 24 hours)	11 (11%)
Peripheral ischaemia requiring an intervention	10 (10%)

CT – computed tomography, LDH – lactate dehydrogenase, Hb – haemoglobin, AMI – acute myocardial infarction

Complications during hospital stay

The most frequent complication was bleeding which required surgical intervention in 20 patients (20%). Ten patients (10%) developed peripheral ischemia requiring intervention.

The summary of complications during the hospital stay is presented in Table 5.

Summary of hospitalization

The median duration of ICU stay was 13 days (IQR=17). Death during the hospital stay occurred in 39% of patients and 30-day mortality was recorded at 43%. The median interval between admission and death was 10.5 days (IQR=14). Durable left ventricular assist device (LVAD) was implanted in 14% of patients and 3% underwent heart transplantation.

Predictors of 30-day mortality

The 30-day mortality rate in our cohort was recorded at 43.4% (43/99, 95% CI: 34.1% to 53.3%; notably with 1 case of unavailable status) (Tab. 6).

In the separate model analysis, the statistically significant risk factors for 30-day mortality included the history of diabetes mellitus (p=0.0180), history of CAD (p<0.001), previous MI (p=0.0054), PCI prior to hospitalisation (p=0.0358), creatinine level on admission (p=0.0462), arterial lactate level on admission (p=0.0487) and vasoactive inotropic scores on days 5 (p=0.0098) and 7 (p=0.0137). Protective factors were dBP on admission (p=0.0127), mean arterial pressure on admission (p=0.0268), nicotinism (p=0.0488), duration of IABP (p=0.0254) and implantation of a durable LVAD during the hospitalization (p=0.0165).

The multi-variable analysis identified the vasoactive inotropic score on day 5 as the only statistically significant predictor of mortality (p=0.0055).

Regarding the 30-day mortality across SCAI classes, there was no statistically significant difference between SCAI groups C and B (OR 0.96, CI 0.38–2.46, p=0.92). Patients presenting with SCAI D stage of CS showed a numerically higher 30-day mortality rate compared to patients in stages B or C without reaching statistical significance (OR 3.27, CI 0.84–14.69, p=0.10).

We observed a trend of increased 30-day mortality in patients experiencing mechanical complications of acute myocardial infarction in the logistic regression model (OR 1.97, 95% CI: 0.79–4.97).

Upon Cox regression analysis, out of all analysed variables, only the presence of mechanical complications yielded a statistically significant impact on the 30-day survival (Log-rank p=0.014, HR2.19, 95% CI: 1.15–4.15) (Fig. 1).

Tab. 6. Predictors of 30-day mortality: statistically significant predictors from separate logistic regression models and a combined multi-variable logistic regression model.

Predictor	**Separate model**		**Combined model**	
Risk factors	**OR (95% CI)**	**p**	**OR (95% CI)**	**p**
History of treated diabetes	2.9 (1.2 to 7.4)	0.018	2.1 (0.4 to 11.4)	0.4
History of coronary artery disease	14.5 (3.7 to 96.03)	<0.001	not included	
Previous myocardial infarction	9.3 (2.3 to 62.5)	0.005	4.4 (0.2 to 94.8)	0.35
PCI prior to the hospitalisation (compared to no revascularisation)	10.1 (1.6 to 195.8)	0.036	18.2 (0.6 to 541.5)	0.09
Creatinine level on admission (per 100 µmol/l increase)	1.6 (1.03 to 2.6)	0.046	1.8 (0.8 to 4.0)	0.17
Arterial lactate level on admission (per 10 mmol/l increase)	3.2 (1.1 to 12.1)	0.048	0.0 (0.0 to 1.3)	0.065
Vasoactive inotropic score on day 5 (per 10 score points)	1.2 (1.1 to 1.4)	0.001	1.5 (1.1 to 1.9)	**0.006**
Vasoactive inotropic score on day 7 (per 10 score points)	1.3 (1.1 to 1.5)	0.014	not included	
Protective factors				
Diastolic blood pressure on admission (per 10 mmHg increase)	0.70 (0.52 to 0.92)	0.013	not included	
Mean arterial pressure on admission (per 10 mmHg increase)	0.76 (0.59 to 0.96)	0.027	0.66 (0.40 to 1.10)	0.11
Current smoking	0.35 (0.12 to 0.95)	0.049	4.10 (0.41 to 65.0)	0.26
Total duration of IABP support (per 1 day of duration)	0.89 (0.80 to 0.98)	0.025	not included	
Implantation of a durable LVAD during the hospitalisation	0.08 (0.00 to 0.42)	0.017	not included	

OR – odds ratio, CI – confidence interval, PCI – percutaneous coronary intervention, IABP – intra-aortic balloon pump, LVAD – left-ventricle assist device

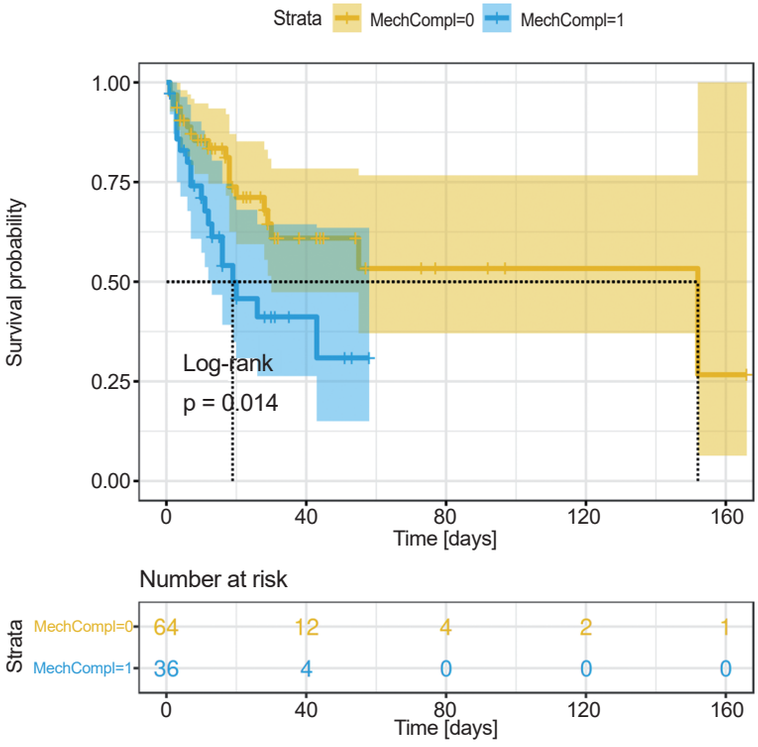

Fig. 1. Survival probability based on the presence or absence of mechanical complication.

Discussion

We conducted a retrospective observational study of our 10-year experience with IABP in CS patients. We observed that the main cause of CS implicated in the use of IABP was newly developed acute heart failure due to acute myocardial infarction which is in accordance with other published studies (12). This acute loss of ventricular function is caused by regional cardiac myocyte necrosis or stunning as a result of impaired perfusion due to coronary occlusion (1).

In our cohort, almost 50% of the patients exhibited symptom duration longer than 24 hours. The duration of symptoms is an acknowledged risk factor for both mortality and development of cardiogenic shock (13). A report from the SHOCK Trial registry indicated that the median time lapse from MI symptoms onset to the development of CS was 6.2 hours and that in most patients (74.1%), CS developed within the first 24 hours (14). A shorter symptom duration, compared to our cohort, is also reported in other contemporary registries such as the AMICS Dutch Cohort published in 2023 (15). On the other hand, our cohort represents only a subpopulation of patients treated by IABP, not the entire population of AMICS patients hospitalised at our centre. Other observed basic demographics such as the average age around 65 years or significantly higher proportion of male patients are comparable to those from other AMICS registries.

Noradrenaline was the most commonly used vasoactive agent on admission as well as the most commonly administered vasopressor during hospitalisation, with more than 90% of patients receiving it. This strategy is consistent with both the European and American recommendations on medical therapy in CS, where norepinephrine is suggested as the first-choice vasopressor (16). The mean duration of vasopressor support was 12.84 (±22.2) days. Out of all analysed predictors, only the vasoactive inotropic score on day 5 was a statistically significant predictor for 30-day mortality in the multi-variable analysis. Vasoactive inotropic score is calculated as a weighted sum of all administered inotropes and vasopressors reflecting the total pharmacological support of the cardiovascular system (17, 18). A higher vasoactive drug score has been associated with increased mortality. Particularly in cases with a high vasoactive score early after admission. This scoring system provides incremental prognostic information about CS patients beyond the SCAI Shock Classification. Interestingly, in a study by Ki Hong et al, the predictive properties of this score for mortality were significantly more accurate among AMICS patients treated with medications alone compared to those treated with MCS such as IABP or ECMO (19). Other variables that were significant in the separate model analysis but did not reach statistical significance in the multivariable model, such as previous mean arterial pressure, arterial lactate and creatinine level on admission or history of previous MI, were recognized also as significant predictors in other studies (20, 21). We assume that the reason for these variables not reaching statistical significance in the multi-variable model stems from the relatively small sample size of our cohort which represents one of the major limitations of our study.

In our cohort, there was also no statistically significant difference in survival across the patient groups categorized by SCAI shock stage. These results are in contradiction with previously published evidence (22–25). We believe that similar to the results of the multi-variable analysis, these contradictory results arise from the small sample size. For example, the 2019 study by Jentzer et al published in JACC included more than 10,000 retrospectively analysed patients.

Upon Cox regression analysis, only the presence of mechanical complications yielded a significant impact on the 30-day survival. Mechanical complications are relatively rare, affecting less than 1% of patients suffering from AMI. On one hand, the low incidence of these complications is fortunate, particularly as they are burdened with very high mortality. On the other hand, producing evidence-based recommendations for this subpopulation of patients is a challenging undertaking. Currently there is a significant variability in the management of these patients and multiple gaps in evidence, such as for the role of point-of-care echocardiography and other imaging modalities, as well as for the use and timing of MCS or surgical intervention. A recent scientific statement from the American Heart Association recommends that this specific subpopulation of patients could be studied in a fashion similar to

rare diseases, i.e. by utilizing study designs requiring only small patient cohorts necessary for an adequately powered randomized controlled trial (26).

In our cohort, hospital mortality was recorded at 39% and the 30-day mortality reached 43%. These results are in line with other reported survival data for CS patients and remain alarmingly high (15, 27). Despite the initially high expectations, MCS has not brought about a significant reduction in the mortality of CS patients. The role of IABP in AMICS was questioned particularly after the publication of the IABP SHOCK II trial by Thiele et al in 2012 (5). Even ECMO, offering arguably the most extensive cardiovascular support with a temporarily complete replacement of the function of both heart and lungs, has repeatedly failed to significantly improve survival (28–30). The long-awaited results of the ECLS-SHOCK trial by Thiele et al, in which 417 patients with AMICS were randomised to either standard treatment with medications or ECMO, revealed that ECMO did not exhibit any mortality reduction in a 30-day follow-up. Mortality in both arms reached almost 50%, underscoring the extreme risk associated with the development of cardiogenic shock despite significant advances in diagnosis and treatment in recent decades. Furthermore, no mortality decrease was demonstrated in the analysis of individual subgroups, even when comparing ST with non-ST IM, or the infarction of the anterior wall with that of other locations (31).

While the current evidence and the latest guidelines do not recommend the use of IABP in the setting of post-MI CS, recent studies have shown a potentially favourable effect of IABP in some high-risk subsets of patients, such as cases with anterior STEMI or persistent ischaemia after pPCI (6–9). Identification of such high risk groups may lead to improved outcomes (32) and possibly to a new era of the IABP, which has recently been significantly receding. Utilisation of modern data-driven technologies such as machine learning and artificial intelligence may be a key factor in this process (33, 34). A major advance in the management of these patients could also lie in the implementation of precision medicine aiming to identify high risk patients even prior to the development of CS, allowing for pre-emptive measures and CS prevention such as the STOPSHOCK initiative (35, 36).

Conclusion

This study summarises our 10-year experience with the use of IABP support in CS patients. The newly developed acute heart failure due to acute myocardial infarction was identified as the main cause of CS. Both in-hospital and 30-day mortality were high despite the use of MCS. Increased vasoactive inotropic score and presence of mechanical complications had a significant impact on the 30-day survival. Further research focusing on the identification of specific high-risk subgroups that would benefit from this treatment is warranted.

Limitations

This study has several limitations. Primarily, as a single-centre study, it refers to a specific population, allowing for potential confounders by local practice, narrowing the applicability of results to other patient populations. Secondly, by default of it being an observational study, it cannot prove causation, only correlations. Moreover, the ps and the confidence intervals were not corrected for multiple testing, and therefore must be interpreted with caution, mainly in an exploratory fashion or in combination with other research results. Thirdly, the sample size was relatively small. Lastly, not all variables with potential impact on survival were collected.

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