Research Square

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Physiological Determinants of In-Hospital Survival Following Intra-Aortic Balloon Pump–Supported Revascularisation in Acute Myocardial Infarction–Related Cardiogenic Shock and High-Risk PCI

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Abstract

Background

Acute myocardial infarction-related cardiogenic shock (ACS-CS) carries 40–60% mortality, and the intra-aortic balloon pump (IABP) remains widely used despite neutral IABP-SHOCK II results; its use in high-risk PCI (HR-PCI) also continues to expand despite limited evidence. We evaluated baseline predictors of in-hospital survival in both cohorts.

Methods

This retrospective study included 505 consecutive patients undergoing IABP-supported revascularisation (June 2019–June 2025), analysed as two cohorts: ACS-CS (n = 397) and HR-PCI (n = 108). Baseline variables were evaluated using multivariable logistic regression for independent predictors of survival. ROC analysis determined optimal pre-IABP lactate thresholds using the Youden index.

Results

In-hospital mortality was 41.1% (ACS-CS) and 32.4% (HR-PCI), rising with SCAI stage (22.4–68.3% and 11.8–76.9%, stages B–E). In ACS-CS, age, intubation, pacing, lactate (adjusted OR 0.89 per mmol/L increase), creatinine and LMCA predicted survival, while BiPAP showed an inverse association (OR 0.32). In HR-PCI, lactate (adjusted OR 0.71 per mmol/L increase), pacing and severe mitral regurgitation retained independent but exploratory associations, given limited events. Pre-IABP lactate showed modest discrimination in ACS-CS (cut-off > 4.0 mmol/L, AUC 0.697; survival 75.4% vs 44.3%) and good discrimination in HR-PCI (cut-off > 4.9 mmol/L, AUC 0.801; survival 85.7% vs 34.2%). Anatomical complexity and ejection fraction were not prognostic; IABP-related complications were infrequent (11.8%, 11.1%).

Conclusions

Baseline physiological status, not anatomical complexity, was the principal determinant of survival. Pre-IABP insertion lactate and SCAI shock stage were consistent risk markers in both cohorts, while renal dysfunction was predictive only in ACS-CS, supporting a selective, physiology-guided approach to risk stratification; prospective multicentre validation is warranted.

Key message

This study extends existing evidence by showing that physiological severity at presentation, rather than the indication for IABP or angiographic complexity alone, determines short-term outcome, providing a

physiology-guided framework for risk stratification.

INTRODUCTION

Acute myocardial infarction complicated by cardiogenic shock (AMI-CS) remains the most severe manifestation of acute coronary syndrome, with in-hospital mortality of 40–60% despite advances in PCI, pharmacological therapy, and critical care. [1–4] Progressive ventricular failure drives a self-perpetuating cycle of hypoperfusion and multiorgan dysfunction that is frequently fatal. Recognition of the heterogeneous nature of cardiogenic shock has led to the adoption of the Society for Cardiovascular Angiography and Interventions (SCAI) shock classification, which consistently demonstrates increasing mortality with progression from stages B to E and provides a standardised framework for risk stratification.[5,6]

Mechanical circulatory support is frequently used to stabilise patients with severe haemodynamic compromise while facilitating revascularisation. Among available devices, IABP remains the most widely used, owing to its simplicity, rapid insertion and favourable safety profile. [7,8] Through diastolic counter-pulsation, IABP augments coronary perfusion while reducing left ventricular afterload and myocardial oxygen demand, increasing cardiac output by approximately 0.5–1.0 L/min.[7,8] These effects provide the rationale for its continued use in AMI-CS and selected high-risk percutaneous coronary intervention (HR-PCI).

The role of IABP in AMI-CS, however, remains controversial. Intra-Aortic Balloon Pump in Cardiogenic Shock II (IABP-SHOCK II) demonstrated no mortality benefit with routine IABP use in patients undergoing early revascularisation, findings that persisted at 12-month and 6-year follow-up, leading to downgrading of routine IABP use in guidelines.[9,10] These trials, however, primarily evaluated routine deployment rather than patient selection: IABP was often inserted after reperfusion, in patients who had already progressed to advanced shock, when limited support may not reverse established collapse. [11–14]

In contrast, observational studies suggest IABP benefit may depend on timing and patient selection. The National Registry of Myocardial Infarction-2 (NRMI-2) demonstrated improved outcomes when IABP was combined with reperfusion therapy in cardiogenic shock.[15] Similarly, the Balloon Pump-Assisted Coronary Intervention Study (BCIS-1) reported improved long-term survival with elective high-risk PCI.[16] Ventricular unloading before reperfusion, lower shock severity, and preserved end-organ function may identify patients most likely to benefit.[12,13,17]

IABP also remains important during high-risk PCI involving severe LV dysfunction, unprotected left main disease, extensive multivessel disease, or complex anatomy, where transient deterioration may compromise procedural success.[16,18,19] This is especially relevant in India, given rising volumes of complex coronary intervention nationally.[20,21]

The persistent disparity between the physiological effects of IABP and the neutral results of routine-use trials highlights the need to identify characteristics that may help refine patient selection and timing of IABP use. We therefore evaluated a large contemporary cohort receiving IABP support for AMI-CS or high-risk PCI over six years, analysing both populations independently to identify baseline predictors of survival, evaluate the prognostic significance of SCAI shock stage, determine clinically relevant lactate thresholds, and characterise patient profiles associated with survival following IABP-supported revascularisation.

MATERIALS AND METHODS

This retrospective cohort study was conducted at a tertiary cardiac care centre in Western India and included all consecutive adult patients undergoing intra-aortic balloon pump (IABP) insertion between June 2019 and June 2025. The study was designed to evaluate in-hospital outcomes and identify baseline predictors of survival among patients receiving IABP support during contemporary coronary revascularization. Institutional Ethics Committee approval was obtained before commencement of the study, and the requirement for informed consent was waived because of the retrospective design. Reporting follows the STROBE guideline.

Patients were categorised by clinical indication for IABP support into two predefined cohorts. The first comprised patients with acute coronary syndrome complicated by cardiogenic shock (ACS-CS) who underwent IABP insertion before revascularisation, including primary PCI, rescue PCI following failed thrombolysis or delayed presentation with ongoing ischemia, and patients initially stabilised with IABP before deferred PCI. The second comprised patients undergoing high-risk PCI in whom IABP was inserted electively, prophylactically for anticipated haemodynamic compromise, or as intra-procedural/bailout support. Both cohorts were analysed independently throughout; patients who left against medical advice were excluded from the final outcome analysis.

Cardiogenic shock was defined according to the Shock Academic Research Consortium (SHARC) criteria as persistent systolic blood pressure < 90 mmHg for ≥ 30 minutes, or the requirement for vasopressor, inotropic or mechanical circulatory support to maintain systolic blood pressure ≥ 90 mmHg, in the presence of objective evidence of tissue hypoperfusion.[3–5] Shock severity was retrospectively classified according to the SCAI shock staging system using available clinical, haemodynamic, laboratory and imaging data at presentation.[5,6]

Baseline demographic characteristics, cardiovascular risk factors, previous cardiovascular history, clinical presentation, haemodynamic variables, rhythm disturbances, cardiopulmonary resuscitation, temporary pacing, ventilatory support, laboratory investigations (including serum lactate and renal function), echocardiographic findings, coronary angiographic characteristics, and timing of IABP insertion and revascularisation were retrieved from patient medical records. Duration of IABP support, hospitalisation, in-hospital adverse events and device-related complications were also recorded. Only variables available before or at the time of IABP insertion were included in prognostic analyses. The

primary endpoint was all-cause in-hospital mortality; successful discharge was the principal secondary outcome.

Data Analysis

Statistical analyses were performed in IBM SPSS Statistics 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation, and categorical variables as frequencies and percentages. Baseline variables significant at $p < 0.05$ on univariate analysis, or of established clinical relevance, were subsequently entered into multivariable logistic regression models to identify independent predictors of in-hospital survival within each cohort. ROC curve analysis was performed to evaluate the prognostic performance of pre-IABP insertion serum lactate and to determine the optimal mortality threshold using the Youden index. Mortality was additionally analysed according to individual SCAI stage and to the derived lactate threshold. A two-sided p value < 0.05 was considered statistically significant.

RESULTS

Overall Study Population

A total of 511 consecutive patients underwent IABP insertion during the study period. Following exclusion of six patients who left against medical advice, the final study population comprised 505 patients. Of these, 397 (78.6%) underwent IABP-supported revascularisation for ACS-CS, with IABP inserted before PCI in every patient. The remaining 108 (21.4%) underwent IABP-supported high-risk PCI, with IABP deployed electively or as intra-procedural/bailout support. The two cohorts were analysed independently throughout (Table 1), given their differing presentation and indication for IABP.

Table 1
Baseline demographic, clinical, haemodynamic, laboratory, angiographic and in-hospital outcome characteristics of the ACS-CS and HR-PCI cohorts

Variables	**Total N = 505(%)**	**Acute ACS group N = 397(%)**	**High Risk PCI group N = 108(%)**
Age (Years)	58.13 ± 11.98 range (23–92)	57.73 ± 12.40 Range (23–92)	59.62 ± 10.20 Range (27–85)
Male	377(74.66%)	306(77.08%)	71(65.7%)
Female	128(25.35%)	91(22.92%)	37(34.3%)
Hypertension (mm Hg)	192(38.02%)	140(32.26%)	52(48.1%)
Type 2 Diabetes mellitus	174(34.46%)	126(31.74%)	48(44.4%)
Previous ACS	78(15.45%)	51(12.85%)	27(25%)
Previous CABG	15(2.98%)	5(1.26%)	10(9.3%)
Previous PTCA	50(9.91%)	38(9.57%)	12(11.1%)
Initial Rhythm Disturbance	**62(12.28)**	**58(14.6%)**	**4(3.7%)**
AF	1(0.2%)	1(0.25%)	0
CHB	51(10.1%)	47(11.84%)	4(3.7%)
Junctional Rhythm	3(0.59%)	3(0.76%)	0
Mobitz type II	7(1.39%)	7(1.39%)	0
Initial Presentation			
VT/DC Cardioversion	140(27.72%)	116(29.22%)	24(22.2%)
CPR	92(18.22%)	77(19.40%)	15(13.9%)
Intubation	186(36.83%)	151(38.04%)	35(38.04%)
BiPAP	115(22.77%)	98(24.69%)	17(15.7%)
Failed Thrombolysis	44(8.71%)	43(10.83%)	1(0.9%)

ACS=Acute coronary syndrome; CABG-Coronary artery bypass grafting; PTCA= Percutaneous transluminal coronary angioplasty; VT/DC= Ventricular Tachycardia / Direct Current; CPR= Cardiopulmonary resuscitation; BiPAP= Bilevel Positive Airway Pressure, RV=Right ventricular; LVEF= left ventricular ejection fraction; MR=Mitral regurgitation; TPI= Temporary Pacemaker Implantation; SCAI= Society for Cardiovascular Angiography and Interventions; LAD= Left Anterior Descending artery; LCX= Left circumflex artery; RCA= Right Coronary Artery; LMCA= Left Main Coronary Artery; SVD= Single vessel disease; DVD=Double vessel disease; TVD=Tripple vessel disease; ISR = In-stent restenosis; POBA= Plain Old Balloon Angioplasty; IABP= Intra-aortic balloon pump

Variables	Total N = 505(%)	Acute ACS group N = 397(%)	High Risk PCI group N = 108(%)
RV Dysfunction	21(4.16%)	18(4.53%)	3(2.8%)
Window period (Hrs)	8.16 ± 7.58	8.16 ± 7.58	24(22.2%)
LVEF	31.47 ± 7.93	31.32 ± 7.23	31.99 ± 10.14
Normal	13(2.57%)	4(1.01%)	9(8.3%)
Mild	34(6.73%)	26(6.55%)	8(7.4%)
Moderate	114(22.57%)	92(23.2%)	22(20.4%)
Severe	344(68.12%)	275(69.3%)	69(63.9%)
MR			
Trivial	121(23.96%)		
Mild	243(48.12%)	194(48.9%)	49(45.4%)
Moderate	105(20.79%)	72(18.1%)	33(30.6%)
Severe	35(6.93%)	25(6.3%)	10(9.3%)
TPI	122(24.16%)	104(26.2%)	18(16.7%)
SCAI Stage			
B	92(18.22%)	58(14.6%)	34(31.5%)
C	200(39.6%)	163(41.1%)	37 (34.3%)
D	137(27.13%)	113(28.5%)	24(22.2%)
E	76(15.05%)	63(15.9%)	13(12%)
Culprit vessel			
LAD	359(71.09%)	281(70.8%)	78(72.2%)
LCX	99(19.6%)	64(16.1%)	35(32.4%)
RCA	75(14.85%)	58(14.6%)	17(15.7%)
LMCA	32(6.34%)	18(4.5%)	14(13%)

ACS=Acute coronary syndrome; CABG-Coronary artery bypass grafting; PTCA= Percutaneous transluminal coronary angioplasty; VT/DC= Ventricular Tachycardia / Direct Current; CPR= Cardiopulmonary resuscitation; BiPAP= Bilevel Positive Airway Pressure, RV=Right ventricular; LVEF= left ventricular ejection fraction; MR=Mitral regurgitation; TPI= Temporary Pacemaker Implantation; SCAI= Society for Cardiovascular Angiography and Interventions; LAD= Left Anterior Descending artery; LCX= Left circumflex artery; RCA= Right Coronary Artery; LMCA= Left Main Coronary Artery; SVD= Single vessel disease; DVD=Double vessel disease; TVD=Tripple vessel disease; ISR = In-stent restenosis; POBA= Plain Old Balloon Angioplasty; IABP= Intra-aortic balloon pump

Variables	Total N = 505(%)	Acute ACS group N = 397(%)	High Risk PCI group N = 108(%)
Type of vessel			
SVD	162(32.08%)	143(36%)	19(17.6%)
DVD	178(35.25%)	138(34.8%)	40(37%)
TVD	165(32.67%)	116(29.2%)	49(45.4%)
Lesion Characteristics			
Acute Stent Thrombosis	16(3.17%)	16(4%)	-
Ostial	181(35.84%)	143(36%)	38(35.2%)
Bifurcation	27(5.35%)	13(3.3%)	14(13%)
Calcified	24(4.75%)	6(1.5%)	18(16.7%)
ISR	6(1.19%)	3(0.8%)	3(2.8%)
Intervention			
Only stenting	451(89.31%)	351(88.4%)	100(92.6%)
Only POBA	38(7.52%)	34(8.6%)	4(3.7%)
Stent with POBA	10(1.98%)	6(1.5%)	4(3.7%)
POBA + CABG	6(1.19%)	6(1.5%)	-
In-hospital complications			
Acute kidney injury	166(32.87%)	129(32.5%)	37(34.3%)
Sepsis	35(6.93%)	24(6%)	11(10.2%)
Mods	45(8.91%)	36(9.1%)	9(8.3%)
Pulmonary oedema	259(51.29%)	212(53.4%)	47(43.5%)
Diabetic ketoacidosis	11(2.18%)	7(1.8%)	4(3.7%)
Pneumonia	14(2.77%)	11(2.8%)	3(2.8%)

ACS=Acute coronary syndrome; CABG-Coronary artery bypass grafting; PTCA= Percutaneous transluminal coronary angioplasty; VT/DC= Ventricular Tachycardia / Direct Current; CPR= Cardiopulmonary resuscitation; BiPAP= Bilevel Positive Airway Pressure, RV=Right ventricular; LVEF= left ventricular ejection fraction; MR=Mitral regurgitation; TPI= Temporary Pacemaker Implantation; SCAI= Society for Cardiovascular Angiography and Interventions; LAD= Left Anterior Descending artery; LCX= Left circumflex artery; RCA= Right Coronary Artery; LMCA= Left Main Coronary Artery; SVD= Single vessel disease; DVD=Double vessel disease; TVD=Tripple vessel disease; ISR = In-stent restenosis; POBA= Plain Old Balloon Angioplasty; IABP= Intra-aortic balloon pump

Variables	Total N = 505(%)	Acute ACS group N = 397(%)	High Risk PCI group N = 108(%)
Others	11(2.18%)	7(1.8%)	4(3.7%)
Dialysis	68(13.47%)	56(14.1%)	12(11.1%)
Days on IABP	2.65 ± 2.15 (1–20 range)	2.61 ± 2.01 (1–13 range)	2.78 ± 2.61 (1–20 range)
Hospital stay	6.52 ± 4.63 (1–27 range)	5.76 ± 4.15 (1–27 range)	9.33 ± 5.19 (1–25 range)
In-hospital outcome			
Death	307(60.79%)	163(41.1%)	35(32.4%)
Discharge	198(39.21%)	234(58.9%)	73(67.6%)

ACS=Acute coronary syndrome; CABG-Coronary artery bypass grafting; PTCA= Percutaneous transluminal coronary angioplasty; VT/DC= Ventricular Tachycardia / Direct Current; CPR= Cardiopulmonary resuscitation; BiPAP= Bilevel Positive Airway Pressure, RV=Right ventricular; LVEF= left ventricular ejection fraction; MR=Mitral regurgitation; TPI= Temporary Pacemaker Implantation; SCAI= Society for Cardiovascular Angiography and Interventions; LAD= Left Anterior Descending artery; LCX= Left circumflex artery; RCA= Right Coronary Artery; LMCA= Left Main Coronary Artery; SVD= Single vessel disease; DVD=Double vessel disease; TVD=Tripple vessel disease; ISR = In-stent restenosis; POBA= Plain Old Balloon Angioplasty; IABP= Intra-aortic balloon pump

Acute Coronary Syndrome–Cardiogenic Shock Cohort

The ACS-CS cohort consisted of 397 patients who underwent IABP insertion before revascularisation, including primary, rescue and delayed PCI after initial haemodynamic stabilisation. Mean age was 57.73 ± 12.40 years (range 23–92), with a marked male predominance (306 patients; 77.1%). Hypertension and diabetes mellitus were present in 35.3% and 31.7% of patients, respectively, while previous acute coronary syndrome, prior PCI and previous coronary artery bypass grafting were documented in 12.9%, 9.6% and 1.3%, respectively. Rescue PCI following failed thrombolysis accounted for 10.8% of cases.

The cohort demonstrated profound haemodynamic compromise at presentation. Rhythm disturbances other than ventricular tachyarrhythmia occurred in 14.6%, predominantly complete heart block (11.8%). VT/DC cardioversion was required in 29.2%, and 19.4% required CPR before revascularisation. Mechanical ventilatory support was frequent, with 38.0% intubated and 24.7% receiving BiPAP. Mean symptom-to-revascularisation interval was 8.16 ± 7.58 hours.

Echocardiography demonstrated marked impairment of left ventricular systolic function, with mean LVEF of 31.32 ± 7.23%. Severe LV dysfunction was present in 69.3%, right ventricular dysfunction in 4.5%, and moderate-to-severe mitral regurgitation in 24.4%. Temporary transvenous pacing was required in 26.2%.

Retrospective SCAI classification showed 14.6%, 41.1%, 28.5% and 15.9% of patients as stages B, C, D and E, respectively, indicating that 84.5% presented with established or advanced shock (SCAI stages C–E).

Coronary angiography demonstrated that the left anterior descending artery (LAD) was the most frequently involved vessel (70.8%), followed by the left circumflex (LCX-16.1%), right coronary (RCA-14.6%) and left main coronary arteries (LMCA- 4.5%). Multivessel disease was common, with 34.8% having double-vessel (DVD) and 29.2% triple-vessel disease (TVD). Coronary stenting was the predominant revascularisation strategy, successfully performed in 88.4% of patients.

During the index hospitalisation (Table 1), acute kidney injury developed in 32.5% of patients, pulmonary oedema in 53.4%, multiorgan dysfunction syndrome in 9.1%, sepsis in 6%, and dialysis was required in 14.1%, reflecting disease severity rather than IABP-attributable complications. Mean duration of IABP support was 2.61 ± 2.01 days, and mean hospital stay was 5.76 ± 4.15 days. Overall, 163 patients (41.1%) died during the index hospitalisation and 234 (58.9%) survived to discharge.

Stage-specific analysis demonstrated a progressive increase in mortality with advancing SCAI stage: 22.4% (13/58) in stage B, 30.7% (50/163) in stage C, 50.4% (57/113) in stage D, and 68.3% (43/63) in stage E. This stage specific in-hospital outcome was mentioned in Fig. 1.

IABP-related complications occurred in 47 patients (11.8%): thrombocytopenia in 26 (6.5%), femoral haematoma/bleeding in 11(2.8%), acute limb ischaemia in 6(1.5%), pseudoaneurysm in 3 (0.8%), and balloon rupture in 1 (0.3%) mentioned in Table 2.

Table 2
IABP device-related vascular and haematological complications

Variable	**Total**	**ACS-CS**	**HR-PCI**
IABP-related complications	59(11.68%)	47 (11.8%)	12 (11.1%)
Acute limb ischemia	7(1.39%)	6 (1.5%)	1 (0.9%)
Femoral haematoma/bleeding	14(2.77%)	11 (2.8%)	3 (2.7%)
Thrombocytopenia	33(6.53%)	26 (6.5%)	7 (6.4%)
Balloon rupture	1(0.20%)	1 (0.3%)	0
Femoral pseudoaneurysm	4(0.79%)	3 (0.8%)	1 (0.9%)
IABP= Intra-aortic balloon pump			

Prognostic analyses were restricted to variables available before or at IABP insertion. On univariate logistic regression, younger age (OR 0.97, 95% CI 0.96–0.99; p = 0.002), initial BiPAP use (OR 5.96, 95% CI 3.24–10.96; p < 0.001) were associated with higher odds of survival. Conversely, initial VT/DC shock

(OR 0.49, 95% CI 0.32–0.76; p = 0.001), CPR before intervention (OR 0.34, 95% CI 0.20–0.57; p < 0.001), initial intubation (OR 0.16, 95% CI 0.10–0.25; p < 0.001), temporary pacemaker insertion (TPI) (OR 0.32, 95% CI 0.20–0.51; p < 0.001), serum lactate (OR 0.83 per mmol/L increase, 95% CI 0.78–0.88; p < 0.001), serum creatinine (OR 0.81, 95% CI 0.68–0.96; p = 0.016) and LMCA involvement (OR 0.34, 95% CI 0.18–0.63; p < 0.001) were associated with lower odds of survival.

On multivariable analysis, age, initial intubation, initial BiPAP use, TPI, serum lactate, serum creatinine and LMCA involvement remained independently associated with in-hospital survival (Table 3). Age (OR 0.97, 95% CI 0.95–0.99; p = 0.005), serum lactate (OR 0.89 per mmol/L increase, 95% CI 0.83–0.95; p = 0.001) and serum creatinine (OR 0.85, 95% CI 0.75–0.96; p = 0.010) independently predicted lower survival, while initial intubation (OR 3.24, 95% CI 1.76–5.96; p < 0.001), TPI (OR 2.09, 95% CI 1.19–3.67; p = 0.010) and LMCA involvement (OR 3.75, 95% CI 1.79–7.83; p = 0.001) retained independent significance. Initial BiPAP use was associated with higher odds of survival on univariate analysis (OR 5.96, 95% CI 3.24–10.96; p < 0.001) but showed an inverse association after multivariable adjustment (adjusted OR 0.32, 95% CI 0.15–0.66; p = 0.002), suggesting substantial confounding by baseline clinical severity rather than a true adverse effect of non-invasive ventilation. CPR and initial VT/DC shock likewise lost significance after adjustment in the multivariable model.

Table 3
Predictors of in-hospital survival for ACS-CS cohort

Univariate Logistic Regression for ACS-CS cohort

Variable	B	S.E.	Wald	P-value (Sig.)	Exp(B)	95% CI Lower	95% CI Upper
Age	-0.03	0.01	9.44	0.001	0.97	0.96	0.99
Previous ACS	-0.28	0.30	0.87	0.35	0.76	0.42	1.36
Initial VT/DC before	-0.72	0.22	10.23	0.001	0.49	0.32	0.76
Initial CPR before	-1.08	0.26	16.99	< 0.001	0.34	0.20	0.57
initial intubation	-1.84	0.23	65.28	< 0.001	0.16	0.10	0.25
Initial BiPAP	1.79	0.31	32.95	< 0.001	5.96	3.24	10.96
TPI	-1.14	0.24	23.37	< 0.001	0.32	0.20	0.51
Lactate	-0.19	0.03	37.11	< 0.001	0.83	0.78	0.88
Hemoglobin	0.00	0.02	0.00	0.98	1.00	0.97	1.03
Serum Creatinine	-0.21	0.09	5.78	0.02	0.81	0.68	0.96
Acute stent thrombosis	-0.38	0.51	0.55	0.46	0.69	0.25	1.87
LMCA	-1.08	0.32	11.68	< 0.001	0.34	0.18	0.63
TVD	-0.22	0.22	0.96	0.33	0.80	0.52	1.24

Multivariate Logistic Regression for ACS-CS cohort

Variable	B	S.E.	Wald	P-value (Sig.)	Exp(B)	95% C.I. for EXP(B)	
						Lower	Upper
Age	-0.03	0.01	7.99	0.01	0.97	0.95	0.99
Initial intubation	1.17	0.31	14.23	0.00	3.24	1.76	5.96
Initial CPR	-0.13	0.39	0.11	0.74	0.88	0.41	1.87
Initial VT/DC	0.29	0.32	0.79	0.37	1.33	0.71	2.50
Initial BiPAP	-1.16	0.37	9.54	0.002	0.32	0.15	0.66
TPI	0.74	0.29	6.60	0.01	2.09	1.19	3.67
Lactate	-0.12	0.04	11.81	0.001	0.89	0.83	0.95
Serum Creatinine	-0.16	0.06	6.67	0.01	0.85	0.75	0.96
LMCA	1.32	0.38	12.35	0.001	3.75	1.79	7.83
Constant	1.57	0.87	3.24	0.07	4.79		

ACS=Acute coronary syndrome; VT/DC= Ventricular Tachycardia / Direct Current; CPR= Cardiopulmonary resuscitation; BiPAP= Bilevel Positive Airway Pressure; TPI= Temporary Pacemaker Implantation; LMCA= Left Main Coronary Artery; TVD=Tripple vessel disease; p value <0.05 shows statistically significant

Table :4 Predictors of in-hospital survival for HR-PCI cohort

Univariate Logistic Regression for High-risk PCI

Variable	B	S.E.	Wald	P value (Sig.)	Exp(B)	95% CI Lower	95% CI Upper
Initial VT/DC before	-1.20	0.48	6.31	0.01	0.30	0.12	0.77
Initial CPR before	-2.07	0.63	10.77	0.001	0.13	0.04	0.44
Initial intubation	-1.64	0.45	13.41	<0.001	0.19	0.08	0.47
Initial BiPAP	1.45	0.78	3.43	0.06	4.27	0.92	19.83
LVEF	0.02	0.02	1.23	0.27	1.02	0.98	1.07
Severe LVD	-0.70	0.45	2.38	0.12	0.50	0.20	1.21
Severe MR	-1.76	0.73	5.91	0.02	0.17	0.04	0.71
TPI	-2.86	0.68	17.65	<0.001	0.06	0.02	0.22
Lactate	-0.36	0.08	19.95	<0.001	0.70	0.60	0.82
Hemoglobin	0.17	0.09	3.77	0.05	1.19	1.00	1.42
Serum Creatinine	0.07	0.13	0.28	0.59	1.07	0.83	1.38
TVD	-0.36	0.41	0.76	0.38	0.70	0.31	1.57
Bifurcation	2.00	1.06	3.55	0.06	7.37	0.92	58.79

Multivariate Logistic Regression for HR-PCI cohort

Variable	B	S.E.	Wald	P value (Sig.)	Exp(B)	95% C.I.for EXP(B)	
						Lower	Upper
Initial VT/DC	-0.13	0.95	0.02	0.89	0.88	0.14	5.62
Initial CPR	1.48	1.17	1.59	0.21	4.38	0.44	43.70
Initial Intubation	-0.93	0.88	1.10	0.29	0.40	0.07	2.24
TPI	2.99	0.99	9.15	0.002	19.82	2.86	137.24
Lactate	-0.35	0.10	11.43	0.001	0.71	0.58	0.87
Severe MR	2.55	0.87	8.66	0.003	12.82	2.34	70.13
Constant	-2.82	1.38	4.14	0.04	0.06		

VT/DC= Ventricular Tachycardia / Direct Current; CPR= Cardiopulmonary resuscitation; BiPAP= Bilevel Positive Airway Pressure; TPI= Temporary Pacemaker Implantation; MR=Mitral regurgitation; LMCA= Left Main Coronary Artery; TVD=Tripple vessel disease; p value <0.05 shows statistically significant

Table 5
ROC Analysis in ACS and HR-PCI cohort

ACS-CS	N = 505	AUC	Cut-off	Sensitivity	Specificity	95% CI lower-upper	P value
	397	**0.697**	**> 4**	**71.8**	**60.3**	**0.65 to 0.74**	**< 0.0001***
HR-PCI	108	0.801	> 4.9	71.4	82.2	0.71 to 0.87	< 0.0001*

ACS-CS=Acute coronary syndrome- Cardiogenic Shock; HR-PCI= High risk Percutaneous transluminal coronary angioplasty; *p value < 0.05 shows statistically significant

ROC analysis identified an optimal pre-IABP insertion serum lactate threshold of 4.0 mmol/L (Fig. 2a). Of 397 patients, 187 (47.1%) had lactate < 4.0 mmol/L and 210 (52.9%) had lactate ≥ 4.0 mmol/L. Patients below the threshold had markedly better outcomes (75.4% survival) than those at or above it (44.3% survival) mentioned in Fig. 3. Overall, baseline SCAI shock stage and pre-IABP insertion serum lactate were the strongest determinants of survival in this cohort.

High-Risk Percutaneous Coronary Intervention Cohort

The high-risk PCI cohort comprised 108 patients (21.4%) undergoing IABP-supported complex coronary intervention, including elective prophylactic insertion, insertion for anticipated haemodynamic compromise, and bailout insertion following intraprocedural deterioration. All patients shared a common indication requiring mechanical support and were analysed as a single cohort.

Mean age was comparable to the ACS-CS cohort (59.62 ± 10.20 years, range 27–85), with 71 patients (65.7%) male. Hypertension and type 2 diabetes were present in 48.1% and 44.4%, respectively, and previous ACS, prior PCI and previous CABG were documented in 25.0%, 11.1% and 9.3%, respectively.

Although haemodynamic compromise was generally less severe than in ACS-CS, a substantial proportion required advanced support before IABP insertion: VT/DC cardioversion in 22.2%, CPR in 13.9%, intubation in 32.4%, and BiPAP in 15.7%. Temporary pacing was necessary in 16.7%, while RV dysfunction was uncommon (2.8%). Mean LVEF was 31.99 ± 10.14%; severe left ventricular dysfunction (LVD) was present in 63.9%, moderate in 20.4%, and mild in 7.4%. Moderate-to-severe mitral regurgitation was present in 39.9%.

Retrospective SCAI classification showed 31.5%, 34.3%, 22.2% and 12.0% of patients as stages B, C, D and E, respectively; approximately two-thirds (65.8%) were stages B or C, indicating IABP was instituted before progression to advanced circulatory collapse in most patients. In-hospital outcome according to SCAI stages was mentioned in Fig. 1.

Cardiac angiography indicated extensive, complex coronary artery disease, with LAD involvement at 72.2%, LCX at 32.4%, RCA at 15.7%, and LMCA at 13.0%. Triple-vessel disease was present in 45.4%, double-vessel in 37.0%, and single-vessel in 17.6%. Complex lesion morphology was common (ostial 35.2%, bifurcation 13.0%, heavily calcified 16.7%). Coronary stenting was successfully performed in 92.6% of patients.

During the index hospitalisation (Table 1), acute kidney injury developed in 34.3% of patients, pulmonary oedema in 43.5%, multiorgan dysfunction syndrome in 8.3%, sepsis in 10.2%, and dialysis was required in 11.1%. Mean duration of IABP support was 2.78 ± 2.61 days, and mean hospital stay was 9.33 ± 5.19 days. Overall, 35 patients (32.4%) died during hospitalisation and 73 (67.6%) survived to discharge. Stage-specific mortality increased progressively: 11.8% (4/34) in stage B, 18.9% (7/37) in stage C, 58.3% (14/24) in stage D, and 76.9% (10/13) in stage E.

IABP-related complications occurred in 12 patients (11.1%) (Table 2): thrombocytopenia in 7 (6.4%), femoral haematoma/bleeding in 3 (2.7%), acute limb ischaemia in 1 (0.9%), and pseudoaneurysm in 1 (0.9%); no balloon rupture was observed.

On univariate logistic regression, higher haemoglobin showed a borderline positive association with survival (OR 1.19, 95% CI 1.00–1.42; p = 0.052). Initial VT/DC shock (OR 0.30, 95% CI 0.12–0.77; p = 0.012), CPR before intervention (OR 0.13, 95% CI 0.04–0.44; p = 0.001), initial intubation (OR 0.19, 95% CI 0.08–0.47; p < 0.001), severe mitral regurgitation (OR 0.17, 95% CI 0.04–0.71; p = 0.015), TPI (OR 0.06, 95% CI 0.02–0.22; p < 0.001) and serum lactate (OR 0.70 per mmol/L increase, 95% CI 0.60–0.82; p < 0.001) were associated with lower odds of survival.

On multivariable analysis, TPI, serum lactate and severe mitral regurgitation remained independent predictors of survival (Table 4). Serum lactate remained independently associated with lower survival (OR 0.71 per mmol/L increase, 95% CI 0.58–0.87; p = 0.001), while TPI (OR 19.82, 95% CI 2.86–137.24; p = 0.002) and severe mitral regurgitation (OR 12.82, 95% CI 2.34–70.13; p = 0.003) showed positive independent associations with survival, most plausibly reflecting confounding by baseline severity rather than a true protective effect. Given the extremely wide confidence intervals and the low events-per-variable ratio in this cohort, these two associations should be regarded as exploratory and hypothesis-generating, requiring external validation, rather than firm prognostic predictors. CPR, initial intubation and initial VT/DC shock did not retain significance after adjustment.

ROC analysis identified a pre-IABP insertion serum lactate threshold of 4.9 mmol/L as optimal for this cohort (Fig. 2b). Of 108 patients, 70 (64.8%) had lactate < 4.9 mmol/L and 38 (35.2%) had lactate ≥ 4.9 mmol/L; survival was 85.7% below the threshold versus 34.2% at or above it (Fig. 3). Collectively, these findings indicate that physiological status before IABP insertion, rather than angiographic complexity alone, is the principal determinant of outcome following IABP-supported high-risk PCI.

DISCUSSION

The present study provides contemporary real-world evidence on determinants of in-hospital survival among patients receiving IABP support for two distinct indications: ACS-CS and high-risk PCI. Rather than evaluating the efficacy of IABP itself, this study sought to identify baseline characteristics associated with favourable outcomes, analysing both cohorts independently given their differing pathophysiology and therapeutic objective.

The principal finding is that baseline physiological status, rather than the mechanical support device itself, is the dominant determinant of short-term survival. Across both cohorts, increasing SCAI shock stage and elevated pre-IABP lactate were associated with progressively worse outcomes, while baseline renal dysfunction independently predicted survival in the ACS-CS cohort on multivariable analysis. Conventional cardiovascular risk factors, previous coronary disease and angiographic disease burden showed limited prognostic value in either cohort. This reinforces the concept that, once circulatory collapse is established, the severity of systemic hypoperfusion and end-organ dysfunction outweighs chronic cardiovascular comorbidity in determining outcome, consistent with contemporary cardiogenic shock registries and consensus statements.[1–5]

Despite representing an exceptionally high-risk population, outcomes were encouraging: 58.9% of the ACS-CS cohort survived to discharge despite 84.5% presenting with SCAI stage C–E shock and 69.3% with severe LV dysfunction, and 67.6% of the high-risk PCI cohort survived despite advanced ventricular dysfunction and extensive multivessel disease. Although the absence of a control group precludes conclusions regarding IABP efficacy, these findings indicate that acceptable outcomes remain achievable with contemporary revascularisation and selective mechanical support.[3,4,17]

The role of IABP continues to be debated following IABP-SHOCK II, which showed no mortality benefit with routine use, findings that persisted at long-term follow-up and led guidelines to downgrade routine use.[9,10] These trials, however, evaluated routine deployment rather than patient selection: IABP was often inserted after reperfusion in patients already in advanced shock, when modest support may not reverse established failure.[11–14] The question is therefore not whether IABP should be used routinely, but which patients are most likely to benefit and when; our findings support this by showing that objective physiological markers provide meaningful risk stratification among patients selected for IABP therapy.[1,5,9]

Pre-IABP insertion serum lactate was a particularly robust prognostic marker, with ROC-derived thresholds of 4.0 mmol/L (ACS-CS) and 4.9 mmol/L (high-risk PCI), consistent with previous work identifying lactate as an integrated marker of tissue hypoperfusion and adverse prognosis.[22–24] This study additionally provides practical, cohort-specific thresholds for bedside risk stratification. The higher threshold in the high-risk PCI cohort may reflect differences in baseline clinical phenotype and timing of mechanical support between the two cohorts, although this hypothesis cannot be established from the present observational data.[12–14,16] Because this cohort included both elective/prophylactic and bailout indications for IABP, the markedly better survival below the 4.9 mmol/L threshold (85.7% versus 34.2%) is consistent with the hypothesis that outcomes may be more favourable when support is instituted

electively, before hypoperfusion drives lactate upward, rather than as bailout support after haemodynamic compromise has already set in. This study did not formally compare outcomes by indication for insertion, so this hypothesis requires direct prospective evaluation of elective versus bailout timing.

A further observation is the limited prognostic value of coronary anatomical complexity once IABP-supported revascularisation is undertaken. Although the high-risk PCI cohort was selected primarily for multivessel disease, left main involvement, severe LV dysfunction or complex anatomy, these characteristics did not independently predict outcome; similarly, in ACS-CS, apart from LMCA involvement, neither culprit vessel nor overall disease burden was consistently prognostic. Coronary anatomy therefore appears to identify patients selected for mechanical support, while systemic haemodynamic compromise determines short-term prognosis.[2,5,12,22,23] Baseline LV systolic function showed a similarly limited contribution: despite a mean LVEF of approximately 31% in both populations, LVEF was not independently associated with survival, whereas markers of systemic hypoperfusion and end-organ dysfunction retained significance. [2,5,22,24]

LMCA involvement was the only angiographic variable to retain statistical significance after multivariable adjustment in the ACS-CS cohort; however, its direction of association reversed from the unadjusted analysis (univariate OR 0.34 vs adjusted OR 3.75), indicating substantial confounding and precluding interpretation as a protective effect. A similar reversal in direction was observed for initial BiPAP use, initial intubation and TPI in ACS-CS, and for TPI and severe mitral regurgitation in high-risk PCI, most likely reflecting confounding by baseline clinical severity rather than a genuine protective or adverse effect. The adjusted model provides the more reliable estimate of independent association in each case, although the very wide confidence intervals around the high-risk PCI estimates mean the TPI and severe mitral regurgitation findings in that cohort should be regarded as exploratory associations requiring external validation rather than firm prognostic predictors.

Strengths

This study represents one of the larger contemporary single-centre analyses evaluating IABP across two major indications. Prognostic modelling was restricted to baseline pre-procedural variables, identifying genuine predictors rather than post-procedural complications, and the derived lactate thresholds enhance clinical applicability.

Limitations

Several limitations should be considered. The retrospective, single-centre design limits generalisability, and the absence of a control group precludes conclusions about comparative efficacy. Retrospective SCAI staging may have introduced classification bias. The smaller high-risk PCI cohort (108 patients, 35 events) yielded fewer than 10 events per variable in the six-variable model, below conventional

thresholds for stable estimates, likely explaining the very wide confidence intervals for temporary pacing and severe mitral regurgitation. Model calibration (e.g. Hosmer–Lemeshow) and multicollinearity (e.g. variance inflation factors) were not formally assessed and should be incorporated in future validation. Finally, some angiographic variables were not mutually exclusive (see Table 1 footnote).

CONCLUSION

The present study suggests that the success of IABP-supported revascularisation depends less on routine device use and more on appropriate patient selection based on the severity of physiological compromise. Pre-IABP insertion serum lactate and SCAI shock stage provided consistent markers of risk across both cohorts, while baseline renal dysfunction independently predicted survival in the ACS-CS cohort. These findings support further evaluation of a physiology-guided approach to risk stratification among patients selected for IABP-supported revascularisation. Prospective multicentre studies with formal calibration and adequately powered multivariable models are warranted to validate these findings, refine physiology-based selection algorithms, and determine whether earlier, targeted IABP use can improve outcomes in carefully selected patients.

DECLARATIONS

Author Contribution

KS, RC and SS contributed in conceptualization, writing, analysis and interpretation of the manuscript. KP, AJ and AB contributed with data acquisition, content development, data collection and analysis. All authors review and approved the manuscript.

Data Availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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Figures

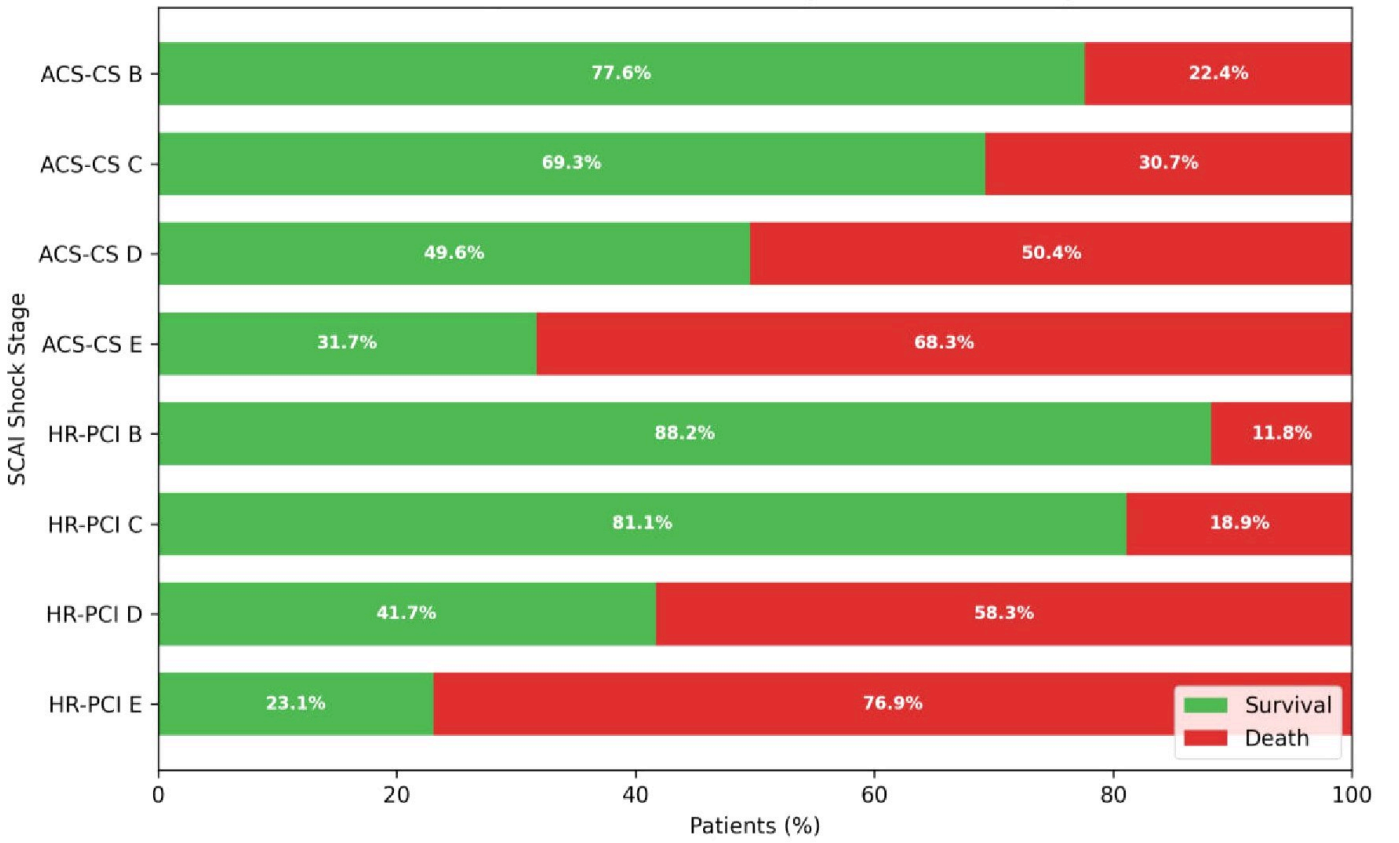


Figure 1

In-hospital Outcome According to SCAI Shock Stage

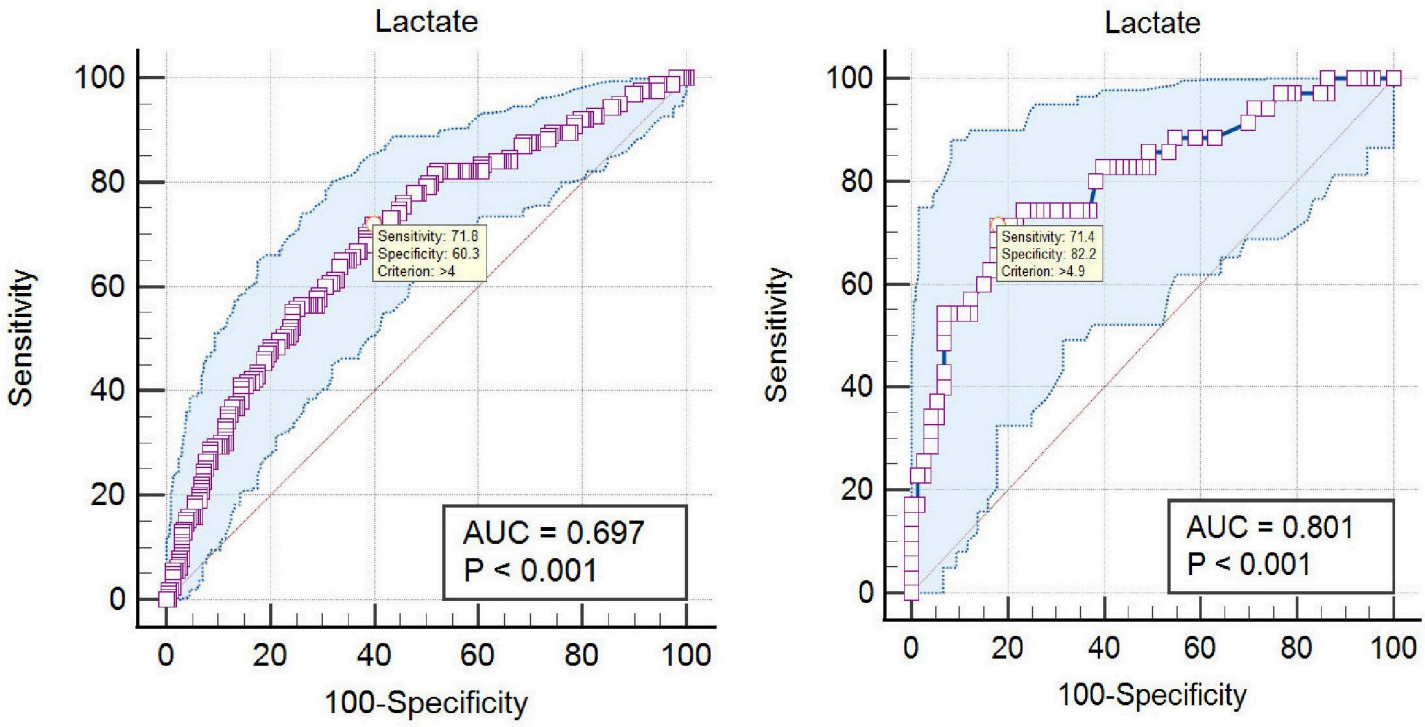


Figure 2

ROC curve for pre-IABP insertion serum lactate for predicting in-hospital mortality

2a. ROC curve for ACS-CS cohort

2b. ROC for High-Risk PCI cohort

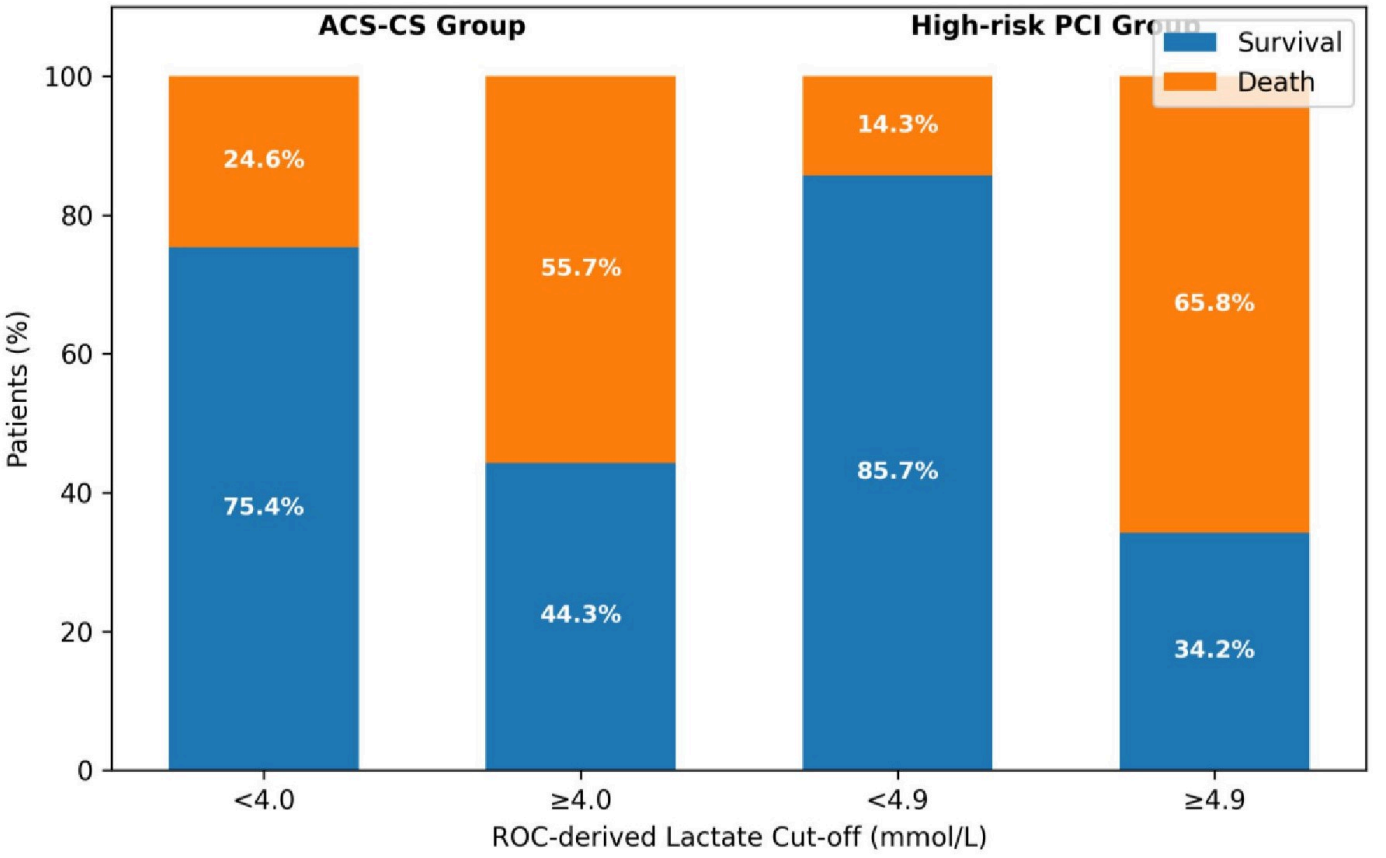


Figure 3

In-hospital Outcome According to ROC- derived Lactate Cut-off