RESEARCH ARTICLE

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Short-term Outcome of Patients with Acute Myocardial Infarction and SCAI-classified Cardiogenic Shock: An Egyptian Registry

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Abstract

Background and Aim: Acute myocardial infarction complicated by cardiogenic shock (AMI-CS) is associated with high morbidity and mortality. The Society for Cardiovascular Angiography and Interventions (SCAI) shock classification provides a structured approach to risk stratification. This study examines the predictors of in-hospital and 30-day mortality among AMI-CS patients using the SCAI staging classification.

Materials and Methods: A prospective cohort study was conducted on 150 patients admitted with AMI-CS at Department of Cardiology, Ain Shams University Hospitals from November 2023 to August 2024. Patients were categorized into SCAI stages (A to E) at presentation and reassessed 24 hours later. Demographic, clinical, biochemical, and hemodynamic parameters were collected.

Results: At presentation, 35.3% of patients were in stage A, 4% in stage B, 54.7% in stage C, 4.7% in stage D, and 1.3% in stage E. Overall in-hospital and 30-day mortalities were 28.7% and 37.3%, respectively. Higher SCAI stages correlated with increased mortality ($P < 0.001$). Independent predictors of mortality included a Sequential Organ Failure Assessment score greater than 5 [odds ratio (OR) =8.17, $P < 0.001$], an APACHE score greater than 7 (OR =3.71, $P = 0.008$), and a serum creatinine level greater than 1.53 mg/dL (OR =5.37, $P = 0.005$). The SCAI score at 24 hours demonstrated superior predictive accuracy for in-hospital mortality (area under the curve =0.889).

Conclusions: SCAI staging is a valuable prognostic tool for MI patients with CS. Reassessment at 24 hours enhances mortality prediction, emphasizing the importance of dynamic risk stratification.

Keywords: Myocardial infarction, cardiogenic shock, SCAI classification, mortality prediction, risk stratification

INTRODUCTION

Acute myocardial infarction (AMI) results from the partial or complete interruption of blood flow to a region of the myocardium. It remains a leading cause of morbidity and mortality worldwide, affecting over three million people annually.[1] It is diagnosed by a rise and/or fall in cardiac troponins above the 99th percentile, accompanied by at least one of the following: ischemic symptoms, segmental wall motion abnormalities on echocardiography, new left bundle branch block (LBBB) or pathological Q waves on Electrocardiogram (ECG), or the presence of an intracoronary thrombus detected by coronary angiography or at autopsy.[2] Multiple modifiable and non-modifiable risk factors play a role in AMI.[3]

Cardiogenic shock (CS) is a leading cause of morbidity and mortality among patients with myocardial infarction, characterized by critical end-organ hypoperfusion and hypoxia resulting from primary cardiac dysfunction.[4] It is defined by hemodynamic criteria, including systolic blood pressure (SBP)

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<90 mmHg for more than 30 minutes (without hypovolemia or requirement for vasopressors), a reduced cardiac index (less than 1.8 L/min/m² without IV support or less than 2.2 L/min/m² with IV support), and elevated left ventricular filling pressures (pulmonary capillary wedge pressure >18 mmHg).[5]

Clinical signs of organ hypoperfusion include cold extremities, reduced urine output, and altered mental status. Serum lactate is used to assess microcirculatory impairment. CS remains the most severe complication of AMI, contributing to nearly 50% of associated deaths.[6] To improve risk stratification, the Society for Cardiovascular Angiography and Interventions (SCAI) has proposed a five-stage classification, based on physical examination, biochemical markers, and hemodynamic parameters: stage A "at risk", stage B "beginning", stage C "classic", stage D "deteriorating", and stage E "extremis".[7]

Cardiogenic Shock Risk Scores

In the cardiac care unit (CCU), various risk scores are employed to assess the severity of CS and predict patient outcomes, including mortality and morbidity. These scores include:

1. The SCAI staging system (SCAI) classifies CS into five stages, from at-risk (stage A) to end-stage (stage E), to guide treatment and assess severity.
2. Sequential organ failure assessment (SOFA) score measures the extent of organ dysfunction by evaluating the respiratory, coagulation, hepatic, cardiovascular, renal, and neurological systems. Higher scores indicate more severe organ failure and a greater risk.
3. Acute physiology and chronic health evaluation II (APACHE II) score: Estimates mortality risk based on acute physiological variables, age, and chronic health conditions, incorporating factors such as temperature, blood pressure, and heart rate.
4. Simplified acute physiology score II predicts mortality risk based on acute physiological measurements and chronic health status, including heart rate, blood pressure, and laboratory results.
5. The CS prognosis score is specifically designed for CS and evaluates prognosis and severity by integrating clinical variables to guide treatment decisions.

Aim of The Work

This study aims to determine the predictors of in-hospital and 30-day outcomes in patients with AMI with CS (AMI-CS), based on the SCAI Shock classification.

METHODS

Design and Population

This prospective cohort study (a prospective observational study without intervention and therefore not eligible for registration in a clinical trial registry) included 150 AMI-CS patients, categorized according to the SCAI classification (applied prospectively at presentation: A to E), in the Cardiology Department of Department of Cardiology, Ain Shams University Hospitals from November 2023 to August 2024. The study protocol was approved by the Scientific and Ethical Committee of Ain Shams University Faculty of Medicine (approval number: FMASU MS161/2024, date: 03.03.2024). All participants provided informed consent; privacy and confidentiality were ensured.

CONSORT Flow Diagram

Eligibility Criteria

The study included patients with AMI who were classified according to the SCAI shock staging system.[2,8] Patients were diagnosed with AMI according to the Fourth Universal Definition of Myocardial Infarction, which requires a rise and/or fall of cardiac troponin levels with at least one value above the 99th percentile upper reference limit, together with at least one of the following: ischemic symptoms; new ischemic ECG changes (such as ST-segment elevation or depression, or new LBBB); imaging evidence of new loss of viable myocardium or of new regional wall motion abnormalities; or identification of intracoronary thrombus via angiography.[2] Exclusion criteria included patients who died before reaching the coronary care unit when sufficient clinical and lab data could not be collected to classify them according to the SCAI staging system.

All Patients Were Subjected to Various Assessments, Including

1-Detailed history taking including: Detailed history taking included demographics (age, gender), comorbid conditions, current medications, history of myocardial infarction and heart failure, previous percutaneous coronary intervention (PCI) or coronary artery bypass grafting, presence of chronic kidney disease (CKD) or prior dialysis, timing of chest pain onset, and occurrence of out-of-hospital cardiac arrest.

The Charlson comorbidity index was used to quantify comorbidities and predict mortality risk.[9]

2-Clinical assessments upon admission included a special emphasis on measuring arterial blood pressure [systolic, diastolic, and mean arterial pressure (MAP), calculated as diastolic blood pressure +1/3 pulse pressure], heart rate, respiratory rate, and oxygen saturation. Chest and cardiac auscultation were performed to assess pulmonary and cardiac

status. Additionally, the Glasgow coma scale (GCS) was used to evaluate the neurological function.[10]

3-Twelve-lead ECG: ECGs were recorded at baseline using a CM 300 A (Comen, China) machine. ST-segment elevation was defined according to the fourth universal definition of myocardial infarction as an ST-segment elevation at the J-point in two or more contiguous leads, with the following thresholds—≥1 mm (0.1 mV) in all leads except V2-V3; in leads V2-V3, elevation of ≥2 mm (0.2 mV) in men aged ≥40 years, ≥2.5 mm (0.25 mV) in men aged <40 years, or ≥1.5 mm (0.15 mV) in women, regardless of age, was considered significant. T wave abnormalities were also assessed according to standard guidelines. Based on these findings, patients were classified as ST-elevation myocardial infarction (STEMI) or non-STMEI (NSTEMI).[2]

4-Laboratory investigations: Random blood sugar at admission, complete blood count, kidney function tests (blood urea nitrogen, creatinine), alanine aminotransferase, HbA1c, creatine kinase total, creatine kinase myocardial band, troponins, arterial pH, bicarbonate, serum lactate, and glomerular filtration rate (GFR) calculated using the CKD epidemiology collaboration equation.

5-Coronary angiography and revascularization: This procedure was performed by experienced interventional cardiologists, and comprehensive procedural details were documented, including balloon predilatation, thrombus aspiration, intracoronary GPIIb/IIIa inhibitors, and the choice of angioplasty alone, stenting alone, or angioplasty followed by stenting.

6-Thrombolysis in myocardial infarction (TIMI) flow grade: Assessed pre- and post-PCI (for the culprit vessel), classifying coronary perfusion from grade 0 (no flow) to grade 3 (complete perfusion).[11]

7-Myocardial blush grade: Recorded pre- and post-PCI (for the culprit vessel), ranging from grade 0 (no blush or persistent staining) to grade 3 (normal blush).[12]

8-Revascularization strategy: Complete versus culprit-only revascularization, with full revascularization defined as an intervention on all significantly diseased vessels with stenosis more than 70%.

9-Additional interventions: The need for mechanical ventilation before or during PCI, the use of mechanical circulatory support (e.g., intra-aortic balloon pump), and the presence of single- or multivessel disease.

10-SCAI shock stages: Classified based on hemodynamic stability, presence of hypoperfusion, and need for inotropic or mechanical support. Class A (at risk) included hemodynamically stable patients who had a large AMI and no signs of hypoperfusion. Class B (beginning CS) included patients with relative hypotension or tachycardia but without hypoperfusion or the need for vasopressors. Class C (classic CS) consisted of patients with hypotension (SBP ≤90 mmHg or MAP ≤60 mmHg) and hypoperfusion requiring therapy. Class D (deteriorating CS) included patients whose condition worsened despite treatment, as indicated by increasing lactate levels or escalating vasoactive drug requirements. Class E (extremis) included patients with prolonged cardiac arrest requiring cardiopulmonary resuscitation or extracorporeal membrane oxygenation (ECMO). Additionally, an A-modifier was applied to patients presenting with cardiac arrest. Hypotension and tachycardia were assessed within the first hour of CCU admission.[13]

11-Severity of illness assessment: The in-hospital severity of illness was evaluated using APACHE II and SOFA scores, both predictive of mortality in intensive care unit settings and validated for CCU patients to estimate short- and long-term mortality risk. The APACHE II score was calculated by summing the acute physiology score (based on 12 variables), age points (ranging from 0 for individuals under 44 years to 6 for those 75 years or older), and chronic health points, resulting in a total score ranging from 0 to 71. Severe acute kidney injury was defined as a doubling of serum creatinine from baseline (the lowest known value), an increase in serum creatinine to greater than 4.0 mg/dL, or initiation of new dialysis; patients on prior dialysis were excluded.

12-Echocardiographic assessment: General electric S7 machine using an M4S matrix sector array probe having a frequency of 2.5 mega Hertz. Evaluation included assessment of left ventricular ejection fraction (LVEF) using the biplane Simpson's method from apical four- and two-chamber views; measurement of left ventricular dimensions and volumes; visual assessment of segmental wall motion abnormalities by an experienced cardiologist, scored according to a standardized 17-segment model with each segment classified as normal, hypokinetic, akinetic, or dyskinetic; and evaluation of the presence of mechanical complications.

Interobserver Variability

To ensure that the assessments in our study were consistent and reliable, two experienced cardiologists independently reviewed key measurements, including:

- SCAI shock stages at presentation and after 24 hours
- TIMI flow grade
- Myocardial blush grade
- LVEF by echocardiography
-

If they disagreed, they reviewed the case together to reach a final decision. We used simple percentage agreement and standard measures such as Cohen's kappa and the intraclass correlation coefficient to measure how well they agreed. Agreement was considered good if the score exceeded 0.7.

Sample Size

Using the power analysis and sample size 11 program for sample size calculation with a 95% confidence interval and a 10% margin of error, it was estimated that a sample size of 95 patients with AMI-CS was needed to detect an expected in-hospital mortality rate of 44.73%. Assuming a 10% dropout rate, a sample size of at least 105 patients with AMI-CS was required. In our study, we enrolled 150 patients, which constituted a larger cohort than the minimum estimated to assess mortality frequency. This larger sample size enhances the reliability of our regression analyses.

Handling Missing Data

Missing data were reviewed for all variables. Cases with missing key outcome data were excluded from analysis. For minor missing clinical or lab values (<5%), a complete-case analysis was performed without imputation.

Statistical Analysis

Data were collected, coded, reviewed, and entered into IBM SPSS version 20 for analysis. Categorical variables were summarized as frequencies and percentages, while continuous variables were reported as mean ± standard deviation for normally distributed data and as median with interquartile range for non-normally distributed data. A 95% confidence interval and a 5% margin of error were applied, with statistical significance defined as a *P*-value ≤0.05. Both univariate and multivariate logistic regression analyses were conducted to identify predictors of mortality. Receiver operating characteristic (ROC) curves were used to evaluate SCAI shock stages at presentation and at 24 hours after admission.

RESULTS

In this study, we aimed to evaluate the predictive value of the SCAI shock classification system in patients presenting to our center with AMI complicated by CS in an Egyptian population. Additionally, we sought to assess the role of other clinical parameters in predicting both in-hospital and 30-day mortality outcomes. Our analysis included 150 patients over a 6-month period who were classified according to the SCAI shock stages at presentation and were re-evaluated after 24 hours. We also examined several factors, including the incidence of out-of-hospital cardiac arrest (OHCA), the need for mechanical ventilation, and the impact of serum lactate levels. Of the 150 patients enrolled in our study, 127 underwent coronary intervention, either single- or multivessel, and 23 did not undergo intervention, either because they experienced cardiac arrest before reaching the cath-lab or for procedural reasons.

The study included 150 AMI-CS patients; males accounted for 72% and females for 28%. The mean age was 59±12 years, with a range of 26 to 83 years. The detailed characteristics of the study population are shown in Table 1.

Patients in SCAI stages D and E exhibited significantly worse clinical and biochemical parameters than those in stages A and B. The admission oxygen saturation, GCS, SBP, MAP, and lactate levels differed significantly ($P < 0.001$), indicating greater physiological deterioration. As shown in Table 1.

Advanced SCAI stages were associated with higher creatinine levels ($P = 0.018$), lower baseline GFR ($P < 0.001$), and elevated troponin levels ($P < 0.001$), indicating worsening organ perfusion and myocardial damage. Mechanical ventilation ($P < 0.001$) and OHCA ($P < 0.001$) were strongly associated with advanced SCAI stages, indicating critical illness. TIMI flow grade ($P < 0.001$) and myocardial blush grade ($P = 0.029$) showed significant differences, with lower TIMI flow and poorer myocardial perfusion in severe cases. Additionally, multivessel interventions ($P = 0.002$) were more frequent in SCAI stages D and E, reflecting the presence of complex coronary disease. These findings confirm that higher SCAI stages correlate with severe clinical deterioration, worse hemodynamics, and increased mortality risk, reinforcing the prognostic value of SCAI classification in patients with acute cardiac conditions as shown in Table 1.

SCAI staging showed a significant shift over 24 hours ($P < 0.001$). The proportion of patients in stage A increased from 35.3% to 43.3%, while stage C cases decreased from 54.7% to 30%. Notably, stage B cases increased from 4% to 16.7%, and stage E cases rose from 1.3% to 4.7%, indicating dynamic clinical progression as shown in Table 2.

Among patients who died during hospitalization, 55.8% had initially presented in SCAI stage C, 14.0% in stage D, and 4.7% in stage E. In contrast, among survivors, the majority (54.2%) were in stage C, with only 0.9% in stage D and none in stage E at presentation ($P < 0.001$). These percentages reflect the distribution of SCAI stages at presentation among deceased and surviving patients, not the mortality rate within each stage. Mortality rates per stage are presented separately for clarity as shown in Table 3 and Figure 1.

Table 1: Characteristics of different SCAI stages at presentation						
Variable	**A (n=53)**	**B (n=6)**	**C (n=82)**	**D (n=7)**	**E (n=2)**	***P*-value**
Gender						
Female	14 (26.4%)	1 (16.7%)	23 (28%)	3 (42.9%)	1 (50%)	0.792
Male	39 (73.6%)	5 (83.3%)	59 (72%)	4 (57.1%)	1 (50%)	
Age, years	59.17±10.9	54.83±14.51	59.63±11.65	61.71±18.54	35.5±9.19	0.061
SO_2, %	94.75±3.18	96.33±0.82	92.82±4.12	84.14±5.05	82.5±3.54	<0.001*
GCS	14.68±1.4	15±0	14.27±1.39	8±2.31	4.5±2.12	<0.001*
Heart rate, bpm	90±16.7	93.33±17.51	94.48±26.1	109.29±46.41	85±63.64	0.371
SBP, mmHg	104.15±14.73	88.33±17.22	79.27±9.91	72.86±4.88	60±0	<0.001*
MAP, mmHg	91.79±13.87	75.83±16.56	68.29±9.27	63.57±3.78	52.5±3.54	<0.001*
Myocardial infarction type						
STEMI	44 (83%)	4 (66.7%)	66 (80.5%)	7 (100%)	2 (100%)	0.542
NSTEMI	9 (17%)	2 (33.3%)	16 (19.5%)	0 (0%)	0 (0%)	
CCI	3 (2-4)	2 (1-4)	4 (3-6)	4 (0-6)	2 (1-3)	0.020*
Prior PCI or CABG	14 (26.4%)	1 (16.7%)	26 (31.7%)	1 (14.3%)	0 (0%)	0.656
Heart failure	8 (15.1%)	0 (0%)	16 (19.5%)	1 (14.3%)	0 (0%)	0.699
Previous MI	12 (22.6%)	0 (0%)	22 (26.8%)	1 (14.3%)	0 (0%)	0.504
CKD	6 (11.3%)	1 (16.7%)	14 (17.1%)	2 (28.6%)	0 (0%)	0.706
Hypertension	18 (34%)	3 (50%)	41 (50%)	4 (57.1%)	1 (50%)	0.415
Diabetes	25 (47.2%)	1 (16.7%)	51 (62.2%)	4 (57.1%)	1 (50%)	0.158
Smoking	33 (62.3%)	5 (83.3%)	55 (67.1%)	3 (42.9%)	1 (50%)	0.574
SOFA score	3 (2-6)	3.5 (3-4)	6.5 (5-7)	10.5 (10-12)	12.5 (11-14)	<0.001*
APACHE score	3 (3-7)	2 (1-7)	7 (3-12)	32.5 (12-35)	51.5 (30-73)	<0.001*
Lactate (peak)	2.6±2.13	2.0±0.68	4.76±2.35	8.27±2.22	6.25±1.06	<0.001*
HCO_3	23.18±4.66	23.8±3.25	18.29±3.61	13.75±3.9	15.5±3.54	<0.001*
pH	7.49±0.07	7.42±0.03	7.32±0.1	7.22±0.1	7.17±0.1	<0.001*
Creatinine	1.08 (0.8-1.29)	1.06 (0.8-1.2)	1.17 (0.96-1.5)	1.84 (1.44-2.5)	1.45 (0.8-2.1)	0.018*
Troponin	50000 (3796-50000)	1422.5 (30-5980)	1228 (20.3-10680)	8869.5 (1598-30000)	27500 (5000-50000)	<0.001*
CK-MB	86 (37-170)	41 (20-69)	70.5 (39-164)	94 (67-278)	119.5 (53-186)	0.496
CK total	825 (330-1657)	177.5 (150-339)	645 (209-1788)	425 (333-950)	574.5 (149-1000)	0.267
Baseline GFR	78 (63-98)	92.5 (47-105)	66.5 (43-93)	35 (25-40)	75.5 (43-108)	<0.001*
Ventilator	4 (81.1%)	6 (100%)	53 (64.6%)	0 (0%)	0 (0%)	<0.001*
EF, %	40.3±9.01	44.0±12.6	38.0±9.65	35.7±5.35	37.5±3.54	0.344
OHCA	**No** 53 (100.0%)	6 (100.0%)	72 (87.8%)	1 (14.3%)	2 (100.0%)	<0.001*
	Yes 0 (0%)	0 (0%)	10 (12.2%)	6 (85.7%)	0 (0%)	
Coronary intervention						
No intervention	7 (13.2%)	2 (33.3%)	8 (9.8%)	5 (71.4%)	1 (50.0%)	0.002*
Single vessel	35 (66.0%)	4 (66.7%)	54 (65.9%)	2 (28.6%)	1 (50.0%)	
More than one vessel	11 (20.8%)	0 (0.0%)	20 (24.4%)	0 (0.0%)	0 (0.0%)	
No. of stents	1 (1-2)	1 (0-2)	1 (1-2)	2 (2-2)	1 (1-1)	0.731
TIMI flow						
Zero	6 (11.3%)	1 (16.7%)	4 (4.9%)	5 (71.4%)	1 (50.0%)	<0.001*
I	2 (3.8%)	0 (0.0%)	6 (7.3%)	0 (0.0%)	0 (0.0%)	
II	10 (18.9%)	1 (16.7%)	28 (34.1%)	1 (14.3%)	0 (0.0%)	
III	35 (66.0%)	4 (66.7%)	44 (53.7%)	1 (14.3%)	1 (50.0%)	

Table 1: Continued

Variable	A (n=53)	B (n=6)	C (n=82)	D (n=7)	E (n=2)	*P*-value
Myocardial blush						
Grade 0	5 (9.6%)	1 (16.7%)	2 (2.5%)	0 (0.0%)	0 (0.0%)	
Grade I	2 (3.8%)	0 (0.0%)	5 (6.3%)	1 (50.0%)	0 (0.0%)	0.029*
Grade II	10 (19.2%)	1 (16.7%)	36 (45.0%)	0 (0.0%)	0 (0.0%)	
Grade III	35 (67.3%)	4 (66.7%)	37 (46.3%)	1 (50.0%)	1 (100.0%)	

Data were presented as Mean ± standard deviation, median (interquartile range), n (%), *: Statistically significant *P*-value as $P < 0.05$, CCI: Charlson comorbidity index, SO_2: Oxygen saturation, GCS: Glasgow coma scale, SBP: Systolic blood pressure, MAP: Mean arterial pressure, STEMI: ST-elevation myocardial infarction, NSTEMI: Non-ST-elevation myocardial infarction, PCI: Percutaneous coronary intervention, CABG: Coronary artery bypass grafting, MI: Myocardial Infarction, CKD: Chronic kidney disease, SOFA: Sequential organ failure assessment, APACHE: Acute physiology and chronic health evaluation, HCO_3: Bicarbonate, PH: Potential of Hydrogen (blood pH), CK-MB: Creatine kinase myocardial band, GFR: Glomerular filtration rate, EF: Ejection fraction, OHCA: Out-of-hospital cardiac arrest, TIMI: Thrombolysis in myocardial infarction, HCO_3: Bicarbonate

The table shows a clear clinical deterioration across SCAI shock stages A to E, with significant declines in oxygen saturation, blood pressure, GCS, and pH, and significant increases in lactate, severity scores (SOFA, APACHE), troponin, and creatinine (all $P < 0.05$). Advanced stages were associated with worse perfusion (lower TIMI flow and reduced myocardial blush), more frequent OHCA, and fewer coronary interventions. Demographics, comorbidities, and MI type did not differ significantly

Table 2: SCAI staging at presentation and after 24 hours among the studied patients

		At presentation	After 24 hours	*P*-value
SCAI staging	**A**	53 (35.3%)	65 (43.3%)	<0.001*
	B	6 (4%)	25 (16.7%)	
	C	82 (54.7%)	45 (30%)	
	D	7 (4.7%)	8 (5.3%)	
	E	2 (1.3%)	7 (4.7%)	

*: Statistically significant *P*-value as $P < 0.05$, SCAI: Society for cardiovascular angiography and interventions

Table 2 shows significant changes in SCAI staging over 24 hours ($P < 0.001$), with an overall shift toward less severe stages. The proportion of patients in stage A increased from 35.3% to 43.3%; in stage B, it rose from 4% to 16.7%; and in stage C, it decreased from 54.7% to 30%

Table 3: Relation of in-hospital and 30-day mortality with SCAI staging at presentation and after 24 hours among the studied patients

		In hospital mortality		*P*-value
		Alive (n=107)	**Died (n=43)**	
SCAI staging at presentation	**A**	42 (39.3%)	11 (25.6%)	<0.001*
	B	6 (5.6%)	0 (0%)	
	C	58 (54.2%)	24 (55.8%)	
	D	1 (0.9%)	6 (14%)	
	E	0 (0%)	2 (4.7%)	
24-hour SCAI staging	**A**	62 (57.9%)	3 (7%)	<0.001*
	B	24 (22.4%)	1 (2.3%)	
	C	21 (19.6%)	24 (55.8%)	
	D	0 (0%)	8 (18.6%)	
	E	0 (0%)	7 (16.3%)	
		30 days mortality		
		Alive (n=94)	**Died (n=56)**	
SCAI staging at presentation	**A**	39 (41.5%)	14 (25%)	0.006
	B	5 (5.3%)	1 (1.8%)	
	C	49 (52.1%)	33 (58.9%)	
	D	1 (1.1%)	6 (10.7%)	
	E	0 (0%)	2 (3.6%)	

Table 3: Continued

		In hospital mortality		*P*-value
		Alive (n=107)	Died (n=43)	
24-hour SCAI staging at hours	A	57 (60.6%)	8 (14.3%)	<0.001*
	B	22 (23.4%)	3 (5.4%)	
	C	15 (16%)	30 (53.6%)	
	D	0 (0%)	8 (14.3%)	
	E	0 (0%)	7 (12.5%)	

*: Statistically significant *P*-value as $P < 0.05$, SCAI: Society for cardiovascular angiography and interventions, n: Number

Table 3 shows that higher SCAI stages are strongly associated with increased in-hospital and 30-day mortality rates. At presentation, most deaths occurred in stages C-E, with significantly fewer in A and B ($P < 0.001$). After 24 hours, mortality was highest in stages C-E, while nearly all survivors were in stages A or B

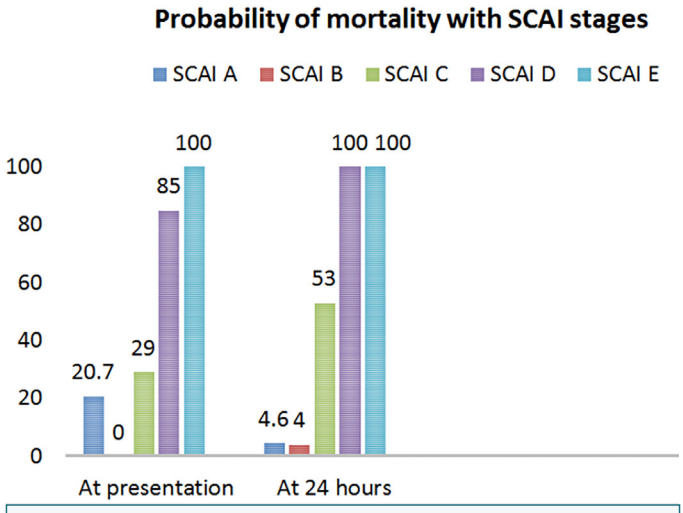

Figure 1: Probability of mortality with advancing SCAI stages at presentation and at 24 hours

SCAI: Society for Cardiovascular Angiography and Interventions

Univariate logistic regression identified several significant predictors of in-hospital mortality, including SOFA score >5 ($P < 0.001$), APACHE score >7 ($P < 0.001$), GCS ≤13 [odds ratio (OR) =10.023, $P < 0.001$), and creatinine >1.53 mg/dL ($P < 0.001$)]. In the multivariate analysis, SOFA score >5 ($P < 0.001$), APACHE score >7 ($P = 0.008$), and creatinine >1.53 mg/dL ($P = 0.005$) remained independent predictors of mortality, indicating their strong prognostic value. Ventilator use showed a trend towards significance (OR =3.083, $P = 0.053$), while other factors lost significance after adjustment as shown in Table 4.

Our data also showed no statistically significant differences in in-hospital or 30-day mortality between genders as shown in Table 5.

Our data also showed no statistically significant difference between single-vessel and complete revascularisation with respect to in-hospital and 30-day mortality Table 6.

The SCAI staging demonstrates an improvement in predictive accuracy over time for both in-hospital and 30-day mortality. At presentation, a cut-off score of >2 yields an area under the curve (AUC) of 0.631 for 30-day mortality and 0.637 for in-hospital mortality, with sensitivities of 73.21% and 74.42% and specificities of 46.81% and 44.86% for 30-day and in-hospital mortality, respectively. While initial predictive ability is moderate, at 24 hours the AUC improves significantly to 0.843 for 30-day mortality and to 0.889 for in-hospital mortality. Sensitivity increases to 80.36% and 90.7%, while specificity increases to 84.04% and 80.37%, respectively. Correspondingly, positive predictive values increased from 45.1% to 75.0% (30-day) and from 35.2% to 65.0% (in-hospital), while negative predictive values increased from 74.6% to 87.8% (30-day) and from 81.4% to 95.6% (in-hospital), highlighting the SCAI score at 24 hours as a more reliable predictor of mortality Figure 2 (A-B) and Table 7.

DISCUSSION

The SCAI shock classification categorizes acute CS into stages A (at risk) through E (extremis). While primarily designed for acute presentations, the SCAI classification also aids in identifying patients at risk of developing CS, particularly those with AMI.[14] Our study aims to validate the SCAI classification for predicting in-hospital and 30-day mortality among AMI patients with CS, while examining demographic and procedural factors that influence short-term outcomes.

Our findings indicate that 35.3% of patients were classified as SCAI stage A at presentation, increasing to 43.3% after 24 hours. Only three deaths occurred in this group, suggesting a relatively favourable prognosis consistent with Baran et al.'s[15] findings. In contrast, patients in stage C or worse exhibited significantly increased mortality, particularly with worsening SCAI stage over 24 hours, further supporting the classification's predictive value for CS severity and outcomes.

Our study found that 35.3% of patients were in stage A, 4% in stage B, 54.7% in stage C, 4.7% in stage D, and 1.3% in stage E at presentation; this differs from Baran et al.[15] who reported no stage A patients and a higher proportion in advanced stages (D and E). These discrepancies may reflect variations in patient demographics, classification criteria, or disease severity across different study populations.

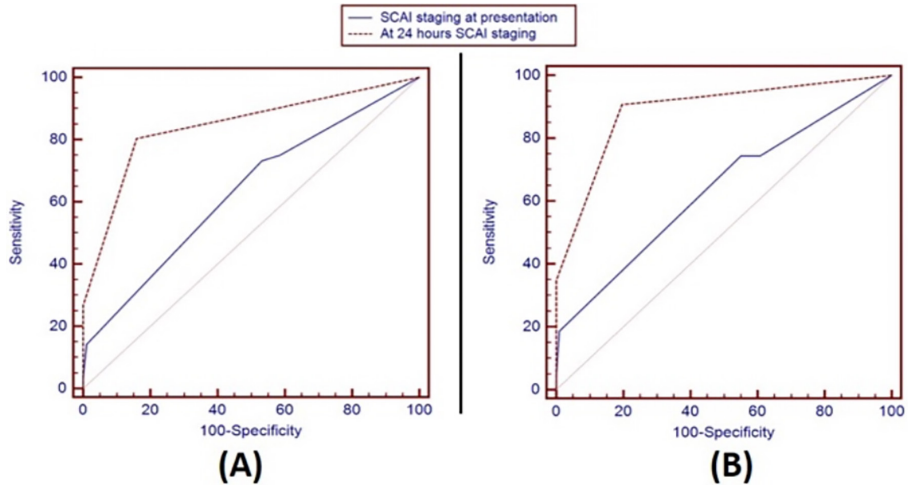

Figure 2: ROC curves assessing SCAI staging at presentation and 24 hours for predicting (A) in-hospital, (B) and 30-day mortality among the studied patients

SCAI: Society for Cardiovascular Angiography and Interventions

Table 4: Univariate and multivariate logistic regression analysis to assess factors associated with the occurrence of in-hospital mortality among the studied patients

	Univariate				Multivariate			
	P-value	OR	95% CI for OR		*P*-value	OR	95% CI for OR	
			Lower	Upper			Lower	Upper
SCAI staging at presentation	0.003*	1.671	1.186	2.355				
SO2 ≤93	<0.001*	4.590	2.261	9.319				
GCS ≤13	<0.001*	10.023	3.943	25.480				
SBP ≤80	0.003*	2.965	1.461	6.017				
MAP ≤65	0.001*	3.421	1.653	7.081				
Charlson comorbidity index >3	<0.001*	4.421	2.127	9.191				
CKD	0.001*	4.971	1.896	13.034				
SOFA score >5	<0.001*	21.737	8.858	53.344	<0.001*	8.174	2.656	25.159
APACHE >7	<0.001*	11.687	5.012	27.254	0.008	3.712	1.405	9.803
Hb ≤ 12	0.001*	3.138	1.563	6.302				
Lactate (peak) >3.8	<0.001*	4.177	2.057	8.485				
HCO_3 ≤17.9	<0.001*	5.123	2.357	11.135				
PH ≤7.34	<0.001*	3.755	1.839	7.668				
Creatinine >1.53	<0.001*	8.933	3.496	22.825	0.005*	5.375	1.670	17.299
Baseline GFR ≤47	<0.001*	7.825	3.441	17.794				
Ventilator	<0.001*	8.831	4.044	19.285	0.053	3.083	0.984	9.657
Myocardial blush	0.091	0.700	0.464	1.058				
TIMI flow	0.001*	0.562	0.397	0.796				
OHCA	<0.001*	15.333	3.334	70.522				

*: Statistically significant *P*-value as $P < 0.05$, SCAI: Society for cardiovascular angiography and interventions, SO_2: Oxygen saturation, OR: Odds ratio, GCS: Glasgow coma scale, SBP: Systolic blood pressure, MAP: Mean arterial pressure, CKD: Chronic kidney disease, SOFA: Sequential organ failure assessment, APACHE: Acute physiology and chronic health evaluation, Hb: Hemoglobin, HCO_3: Bicarbonate, PH: Potential of hydrogen (blood pH), GFR: Glomerular filtration rate, TIMI: Thrombolysis in myocardial infarction, OHCA: Out-of-hospital cardiac arrest Table 4 identifies key predictors of in-hospital mortality from univariate and multivariate logistic regression analyses.

Univariate analysis identified significant associations with multiple factors, including low SpO_2, GCS ≤13, SBP ≤80, MAP ≤65, elevated SOFA and APACHE scores, elevated lactate and creatinine, low HCO_3 and pH, CKD, and OHCA (all $P < 0.05$). In multivariate analysis, the strongest independent predictors were SOFA score >5 (OR =8.17, $P < 0.001$), APACHE score >7 (OR =3.71, $P = 0.008$), and creatinine >1.53 (OR =5.38, $P = 0.005$). Ventilator use approached statistical significance ($P = 0.053$)

Table 5: Relation between gender and in hospital and 30 days mortality

		In hospital mortality		Test value	*P*-value	Sig.
		Alive	Died			
		No=107	No=43			
Gender	Female	29 (27.1%)	13 (30.2%)	0.149*	0.699	NS
	Male	78 (72.9%)	30 (69.8%)			
		30 days mortality		Test value	*P*-value	Sig.
		Alive	Died			
		No=94	No=56			
Gender	Female	26 (27.7%)	16 (28.6%)	0.014*	0.904	NS
	Male	68 (72.3%)	40 (71.4%)			

*: ,NS: Not significant, Sig: Significant

Table 6: Relation of single and multivessel intervention to both in hospital and 30-day mortality

Procedure	In hospital mortality	Test value	*P*-value	Sig.		
	Alive	Died				
	No= 107	No= 43				
Single vs multivessel intervention	No	13 (12.1%)	10 (23.3%)	2.915*	0.233	NS
	Single	71 (66.4%)	25 (58.1%)			
	Multi	23 (21.5%)	8 (18.6%)			
Procedure	30 days mortality	Test value	*P*-value	Sig.		
	Alive	Died				
	No= 94	No= 56				
Single vs multivessel intervention	No	12 (12.8%)	11 (19.6%)	2.020*	0.364	NS
	Single	64 (68.1%)	32 (57.1%)			
	Multi	18 (19.1%)	13 (23.2%)			

NS: Not significant, Sig.: Significant

Table 7: Receiver operating characteristics curve to assess SCAI at presentation and at 30 day (table above) and at 24 hours (table below) to detect mortality among the studied patients

Variable	Cut-off point	AUC	Sensitivity	Specificity	+PV	-PV
At presentation	>2	0.631	73.21	46.81	45.1	74.6
At 24 hours	>2	0.843	80.36	84.04	75.0	87.8
Variable	Cut-off point	AUC	Sensitivity	Specificity	+PV	-PV
At presentation	>2	0.637	74.42	44.86	35.2	81.4
At 24 hours	>2	0.889	90.7	80.37	65.0	95.6

AUC: Area under the curve

In our study, the mean patient age was 59.05±12.06 years (range: 26-83 years), closely aligning with the mean age of 58.1±16.2 years reported by Baran et al.[15] Additionally, our cohort was predominantly male (72%), consistent with previous studies highlighting a male predominance in CS populations.

SCAI shock stages were reassessed after 24 hours: 64 patients remained in the same stage, while others shifted significantly. Notably, 29 patients who were initially in stage C improved to stage A, whereas four patients in stage D progressed to stage E. The significant association between changes in SCAI staging and mortality (P = 0.006) reinforces its prognostic value and is consistent with the findings of Baran et al.[15]

Our findings support those of Baran et al.[15] confirming that changes in SCAI staging within the first 24 hours are significantly associated with mortality. This reinforces the 24-hour reassessment as a reliable prognostic tool, emphasizing its critical role in predicting patient outcomes.

In our study, in-hospital mortality was 28.7%, while 30-day mortality reached 37.3%, corresponding to a 62.7% 30-day survival rate. This is higher than the 30% mortality reported by Hanson et al.[16] which may reflect differences in patient characteristics, limited use of mechanical circulatory support, and their exclusion of SCAI stages A and B. Conversely, Ryabov et al.[17] reported a 54% mortality rate, likely influenced by their inclusion of only three patients in SCAI stages A and B. Notably, mortality among patients with SCAI stages C-E in our study was 35%, highlighting the severity of illness in these patients.

In our study, 82% of patients had STEMI; 55.3% presented with anterior STEMI, while 18% had NSTEMI. This contrasts with Pham et al.[18] in which 70.5% of patients had STEMI. Notably, 22 patients initially classified as SCAI stage A in our study deteriorated within 24 hours, whereas Pham et al.[18] reported no stage progression among stage A patients during the same period, highlighting potential differences in patient characteristics and disease progression.

Our study demonstrated that patients with SCAI stage C or worse who improved within the first 24 hours had better short-term and 30-day survival than those initially in stages A or B who deteriorated after 24 hours. This finding aligns with Baran et al.[15] and Morici et al.[19] and underscores the prognostic significance of early hemodynamic improvement in CS.

In our study, 32% of patients required mechanical ventilation, and the in-hospital mortality rate was 79.1%, highlighting a poor prognosis. Similarly, Morici et al.[19] reported a 30.8% ventilation rate in a larger cohort of 237 patients, emphasizing the critical nature of this intervention.

Serum lactate emerged as a strong independent predictor of mortality, especially in patients with advanced SCAI stages. Elevated lactate reflects tissue hypoperfusion and impaired lactate clearance, making it a practical and dynamic tool for risk stratification. Our findings mirror those of Jentzer et al.[20] reinforcing lactate's utility not only as a biomarker of shock severity but also as a guide for therapeutic escalation and response assessment.

Our study demonstrated that OHCA was associated with significantly worse. The outcomes showed a low 30-day survival rate. This aligns with Sarma et al.[21] who emphasize the poor prognosis and high mortality risk among OHCA patients. These findings highlight the critical need for early intervention and advanced supportive measures to improve survival in this high-risk group. In our study, revascularisation was performed only in patients with a good neurological prognosis after OHCA.

Angiographic outcomes also had a strong impact on mortality. Achieving both optimal TIMI flow grade III and myocardial blush grade III was significantly associated with improved survival. This affirms prior studies by Overtchouk et al.[22] and Mehta et al.[23] which demonstrate the importance of timely and effective reperfusion. These findings advocate for a procedural goal beyond simple vessel patency: ensuring full microvascular reperfusion may be critical in improving outcomes.

Multivessel coronary interventions were significantly more frequent in patients classified as SCAI stages D and E, reflecting the increased complexity and severity of their coronary artery disease. This finding underscores the progression of CS and its association with extensive myocardial ischemia requiring aggressive revascularization strategies. However, performing multivessel interventions in critically ill patients presents challenges, as it may increase procedural risk. These observations warrant further investigation into optimizing revascularization approaches for patients with advanced CS.

The lack of availability of mechanical circulatory support in Egypt resulted in the low number of patients in our study who received such support: only 2% (one ECMO and one intra-aortic balloon pump) were placed on mechanical circulatory support (MCS), in contrast to Koester et al.'s[24] report of US trends among patients presenting with acute coronary syndrome complicated by CS, in which 44.4% were placed on MCS devices. The in-hospital mortality rate in their study was 42.9%.[24]

Study Limitations

Despite our findings, several limitations should be considered. The generalizability of the results may be restricted because the study was conducted at a single centre and had a limited sample size. Additionally, the lack of long-term follow-up and the unavailability of mechanical circulatory support devices could have influenced patient outcomes. Furthermore, delayed presentations may have impacted disease severity and treatment efficacy.

CONCLUSIONS

The SCAI shock classification effectively predicts in-hospital and 30-day mortality in patients with AMI and CS. Higher SCAI stages correlate with worse hemodynamics, more severe organ dysfunction, and increased mortality risk. Reassessment at 24 hours enhances prognostic accuracy, identifying patients at risk of deterioration. Early hemodynamic improvement is associated with better outcomes, emphasizing the need for timely intervention.

Ethics

Ethics Committee Approval: The study protocol was approved by the Scientific and Ethical Committee of Ain Shams University Faculty of Medicine (approval number: FMASU MS161/2024, date: 03.03.2024).

Informed Consent: All participants provided informed consent; privacy and confidentiality were ensured.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Y.A., W.E., Concept: Y.A., W.E., Design: Y.A., W.E., M.S.A., Data Collection or Processing: Y.A., M.E.I., M.S.A., Analysis or Interpretation: M.E.I., W.E., M.S.A., Literature Search: M.E.I., W.E., M.S.A., Writing: Y.A., M.E.I., M.S.A.

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