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Original Research

SCAI Cardiogenic Shock Classification for Predicting In-Hospital and Long-Term Mortality in Acute Heart Failure

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A B S T R A C T

Background: SCAI classification in cardiogenic shock is simple and suitable for rapid assessment. Its predictive behavior in patients with primary acute heart failure (AHF) is not fully known. We aimed to evaluate the ability of the SCAI classification to predict in-hospital and long-term mortality in AHF.

Methods: We conducted a single-center study and performed a retrospective analysis of prospectively collected data of consecutive patients admitted with AHF between 2015 and 2020. The primary end points were in-hospital and long-term mortality from all causes.

Results: In total, 856 patients were included. The unadjusted in-hospital mortality was as follows: A, 0.6%; B, 2.7%; C, 21.5%; D 54.3%; and E, 90.6% (log rank, $P <$ .0001), and long-term mortality was as follows: A, 24.9%; B, 24%; C, 49.6%; D, 62.9%; and E, 95.5% (log rank, $P <$ .0001). After multivariable adjustment, each SCAI SHOCK stage remained associated with increased mortality (all $P <$ .001 compared with stage A). With the exception of the long-term end point, there were no differences between stages A and B for adjusted mortality ($P =$ .1).

Conclusions: In a cohort of patients with AHF, SCAI cardiogenic shock classification was associated with in-hospital and long-term mortality. This finding supports the rationale of the classification in this setting.

Introduction

Cardiogenic shock (CS) is the most severe form of acute heart failure (AHF) and is associated with a high mortality.[1-3] Although there have been significant advances in reperfusion therapy for acute myocardial infarction (AMI), pharmacologic interventions, and temporary and durable percutaneous mechanical circulatory support and transplant, mortality among patients with AMI and non-AMI heart failure (HF) CS remains obstinately high, ranging from 25% to 50%.[4,5]

Additionally, patients with CS present to the hospital at different stages of the disease. Populations with CS encompass a broad spectrum of hemodynamic derangements ranging from isolated hypoperfusion that is easily reversed with initial therapies to refractory shock with multiorgan failure and hemodynamic collapse.[6] This impacts application of different treatment options and clinical outcomes. Unfortunately, staging of patients is difficult and nonreproducible in the clinical setting.

Recognizing the heterogeneity of patient populations with CS, recently, a multidisciplinary group of SCAI derived a classification schema for CS that is simple, clinically based, and suitable for rapid bedside assessment. The purpose of this classification of CS was to provide a simple schema that would allow clear communication regarding patient status and to allow clinical trials to appropriately differentiate patient subsets.[7] This classification schema was developed based on expert consensus opinion, and its ability to discriminate among levels of mortality risk in patients who are critically ill in different datasets is necessary to establish the utility of this proposed classification schema. Recently, SCAI SHOCK classification was assessed at the time of cardiac intensive care unit admission, and less than half of the patients had HF. This classification provided robust hospital mortality risk stratification.[8] This classification was also evaluated in patients presenting with CS or large AMI, with similar findings for 30-day mortality[9]; however, the application of this classification in patients with primary acute decompensated HF and its association with short-term and long-term mortality

Abbreviations: AHF, acute heart failure; AMI, acute myocardial infarction; CPR, cardiopulmonary resuscitation; CS, cardiogenic shock; LVEF, left ventricular ejection fraction.

Keywords: acute heart failure; cardiogenic shock; heart failure; mortality; prognosis.

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Table 1. Definition of SCAI Classification for CS, based on the consensus statement.

Stage	Physical examination/bedside findings	Biochemical markers	Hemodynamics
A At risk	Normal JVP Lungs sound clear Warm and well perfused Normal mentation	Normal laboratory examination results	Normotensive (SBP ≥100 mm Hg)
B Beginning CS	Elevated JVP Rales in lung fields Warm and well perfused Normal mentation	Normal lactate Minimal renal function impairment	SBP <90 OR MAP <60 OR >30 mm Hg drop from baseline Pulse ≥100 beats/min If hemodynamics done • cardiac index ≥2.2 • PA sat ≥65%
C Classic CS	May Include Any of: Looks unwell Mottled, cold Volume overload Extensive rales Killip class 3 or 4 BiPap or mechanical ventilation Acute alteration in mental status	May Include Any of: Lactate ≥2 Creatinine doubling OR >50% drop in GFR Increased LFTs	May Include Any of: SBP <90 OR MAP <60 OR >30 mm Hg drop from baseline AND drugs/device used to maintain BP above these targets Hemodynamics • cardiac index <2.2 • PCWP >15
D Deteriorating/doom	Any of stage C	Any of Stage C AND: Deteriorating	Any of Stage C AND: Requiring multiple pressors OR addition of mechanical circulatory support devices to maintain perfusion
E Extremis	Near pulselessness Cardiac collapse Mechanical ventilation Defibrillator used	CPR (A-modifier) pH ≤7.2 Lactate ≥5	No SBP without resuscitation PEA or refractory VT/VF Hypotension despite maximal support

BP, blood pressure; CPR, cardiopulmonary resuscitation; CS, cardiogenic shock; GFR, glomerular filtration rate; JVP, jugular venous pressure; LFT, liver function test; MAP, mean arterial pressure; PA sat, pulmonary artery saturation; PCWP, pulmonary capillary wedge pressure; PEA, pulseless electrical activity; SBP, systolic blood pressure; VF, ventricular fibrillation; VT, ventricular tachycardia.

settings are unknown. The aim of this study was to evaluate the ability of the SCAI SHOCK staging classification to predict in-hospital and long-term mortality in patients with primary AHF.

Methods

Study design and population

We conducted a single-center cohort study and performed a retrospective analysis of prospectively collected data. Adult patients consecutively admitted to a hospital specialized in cardiovascular disease with a primary diagnosis of AHF (acute or acute on chronic) between January 2015 and January 2020 were included. The diagnosis was carried out independently by 2 cardiologists, specializing in HF, according to the data obtained from the interrogation, physical examination, and complementary studies. AHF secondary to AMI according to the fourth universal definition, severe sepsis, and pulmonary thromboembolism were excluded.

Definitions

The primary end points were in-hospital and long-term mortality from all causes. Long-term mortality of those patients discharged after hospitalization was assessed up to the last available outpatient assessment. Information on death was obtained from the patients' medical records.

Patients were divided according to the main clinical phenotype at admission to the hospital into the following 3 groups:

1. Patients with isolated signs of pulmonary congestion (pulmonary edema).
2. Patients with signs of mixed congestion (central and peripheral congestion).
3. Patients with low cardiac output signs.

History of coronary artery disease was defined using at least one of the following criteria: (1) presence of any epicardial coronary vessels with >75% stenosis tested on coronary angiography; (2) history of acute coronary syndrome; and (3) coronary revascularization (either percutaneous transluminal coronary angioplasty or coronary artery bypass grafting).[10]

Vasoactive was defined as the use of dobutamine, dopamine, norepinephrine, epinephrine, milrinone, isoproterenol, levosimendan, vasopressin, and/or phenylephrine.

Data collection and follow-up

Baseline demographic data, comorbid conditions, physical examination on admission (heart rate, systolic and diastolic blood pressure), type of clinical presentation, laboratory examination findings, previous medical treatment, implemented therapy, and outcome data were collected in a dedicated center database. The values of lactate, creatinine level, liver enzymes, and bilirubin were evaluated on admission. Baseline functional class prior to admission was inquired into as part of the history of all patients with HF.

SCAI classification

Patients were assigned to the SCAI classification for CS, based on the consensus statement (Table 1)[7]:

Stage A is "at risk" for CS: a patient who is not currently experiencing signs or symptoms of CS but is at risk of its development. These patients may include those with large AMI or prior infarction acute and/or acute on chronic HF symptoms.

Stage B is "beginning" shock: a patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.

Stage C is "classic" shock: a patient with hypoperfusion that requires an initial set of interventions (inotropes, pressor, mechanical support, or extracorporeal membrane oxygenation) beyond volume resuscitation to restore perfusion.

Stage D is "deteriorating": a patient who has failed to stabilize despite intense initial efforts and further escalation is required. Classification at this stage requires that the patient has had some degree of appropriate treatment/medical stabilization. In addition, at least 1 hour has elapsed but the patient has not responded with resolution of hypotension or end-organ hypoperfusion.

Stage E is "extremis": a patient with circulatory collapse, frequently in refractory cardiac arrest with ongoing cardiopulmonary resuscitation or are being supported by multiple simultaneous acute interventions, including extracorporeal membrane oxygenation-facilitated cardiopulmonary resuscitation.

Statistical analysis

Categorical variables are reported as numbers and percentages, and the Pearson χ^2 test was used to compare groups. Ordinal data and continuous variables inconsistent with normal distribution are expressed as median and IQR and were compared with Kruskal–Wallis or an analysis of variance test according to the distribution of the variables. Survival probability was estimated using the Kaplan–Meier method and compared across SCAI stages using a log-rank test. Cox proportional hazards models were used to determine the association between SCAI stages and mortality after adjusting for age, male sex, left ventricular ejection fraction (LVEF), use of vasoactive medication, mechanical circulatory assist devices, mechanical ventilation, percutaneous coronary intervention, and cardiac surgery. Two-tailed *P* values <.05 were considered statistically significant. Statistical analysis was performed using the SPSS statistics, Version 23.0 (IBM Corporation).

Ethical considerations

The study was approved by the institutional research and ethics board and was registered on the PRIISA.BA platform of Buenos Aires city Ministry of Health. The study was a retrospective investigation with deidentified data, and according to national regulations, request for informed consent was excepted. At the time of hospitalization, patients signed consent forms for the transfer of personal data for scientific purposes. The study was conducted in accordance with national and international standards for the protection of research subjects, such as the Declaration of Helsinki, Resolution of the Ministry of National Health 1480/2011 Ciudad de Buenos Aires law 3301, ANMAT resolution 6677/10, and amendments 4008 and 4009.

Results

In total, 856 patients with primary AHF were identified and consecutively included in the analysis.

Baseline characteristics

The mean age of the overall cohort was 74.7 years (± 13 years), and 63.7% of the patients were men.

The most frequent clinical profile was systemic congestion in 78% of the patients, followed by a low cardiac output in 12% and isolated acute pulmonary edema in 10%. The main causes of decompensation identified were diet nonadherence (11.6%) and infection (12.3%). Coronary artery disease accounted for 35.6% of the HF etiology, and 37.6% of patients had a previous hospitalization for AHF. The median LVEF was 42 (IQR, 29-58), and reduced LVEF was <40% accounted for 45.7% of the cases.

The median hospital stay was 5 days (IQR, 2-6). Additional baseline characteristics are summarized in Table 2.

Application of the SCAI classification

The proportion of patients with SCAI SHOCK stages A through E were 39.8%, 39.4%, 14.1%, 4.1%, and 2.6%, respectively. Baseline demographics, comorbid conditions, laboratory values, and critical care therapies varied significantly across the SCAI SHOCK stages (Table 3). There was a stepwise increase in organ failure with each higher SCAI SHOCK stage in these patients.

Table 2. Baseline table of the overall cohort.

	Overall cohort (N = 856)
Age, y	74.7 ± 13
Male sex	545 (63.7)
Arterial hypertension	253 (29.6)
Dyslipidemia	530 (61.9)
Diabetes	253 (29.6)
Smoking (current or prior)	417 (48.7)
Chronic kidney disease	186 (21.7)
Peripheral vascular disease	115 (13.4)
Atrial fibrillation	124 (14.5)
Chronic obstructive disease	111 (13)
Prior myocardial infarction	207 (24.2)
Etiology heart failure	
Coronary artery disease	305 (35.6)
Valvular heart disease	235 (27.5)
Idiopathic	61 (7.1)
Hypertrophy	25 (2.9)
Infiltrative	19 (2.2)
Other (myocarditis, Chagas, cardiotoxicity, hypertensive)	211 (24.7)
LVEF <40%	391 (45.7)
Prior heart failure hospitalization	322 (37.6)
Prior NYHA functional class	
I	245 (28.6)
II	263 (30.7)
III	137 (16)
IV	14 (1.6)
Not available	197 (23)

Values are expressed as mean ± SD or n (%).
LVEF, left ventricular ejection fraction; NYHA, New York Heart Association.

In-hospital and long-term mortality

The mean follow-up was 16.8 months. There was a stepwise increase in unadjusted mortality with each higher SCAI SHOCK stage in the population. The unadjusted in-hospital mortality in each stage was as following: A, 0.6%; B, 2.7%; C, 21.5%; D 54.3%; and E, 90.6% (log rank, $P < .0001$), and long-term cumulative mortality was as following: A, 24.9%; B, 24%; C, 49.6%; D, 62.9%; and E, 95.5% (log rank, $P < .0001$) (Central Illustration). If we analyzed only those patients who were discharged at the index hospitalization (n = 780), the mortality during follow-up was as following: A, 24%; B, 22%; C, 36%; D, 44%; and E, 50%.

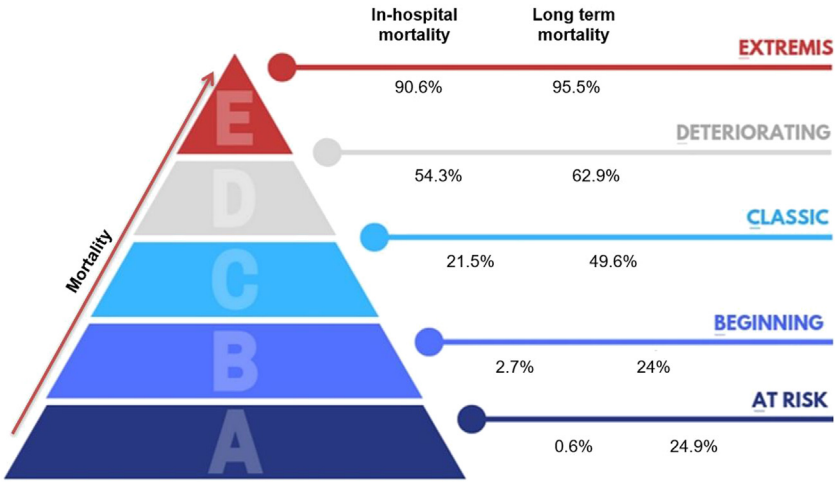
After adjustment for multiple relevant confounders, each SCAI class was significantly associated with the primary end point. All SCAI SHOCK stages remained associated with increased in-hospital mortality (all $P < .001$ compared with stage A). Compared with SCAI SHOCK stage A, adjusted hazard ratio values in SCAI SHOCK stages B through E were 5.2, 31, 107, and 185, respectively (Figure 1).

After application of the SCAI SHOCK classification, SCAI classes C, D, and E were significantly associated with a higher adjusted long-term mortality (all $P < .001$ compared with stage A). There were no statistical differences between stages A and B for adjusted mortality ($P = .1$) (Figure 2).

Discussion

In the present cohort analysis that consecutively included patients with a diagnosis of AHF, we evaluated the ability of the SCAI SHOCK staging classification to predict in-hospital and long-term mortality in patients with primary AHF. The findings suggest that the staging classification was associated with the primary outcome. There was a stepwise increase in unadjusted in-hospital and long-term mortality with each higher SCAI SHOCK stage in these patients.

There are multiple characteristics of the analyzed population that are important to mention. A possible reason that may explain why the patients in classes D and E were younger could be because we are a center



Central Illustration. In-hospital and long-term mortality according to SCAI SHOCK stage.

with an advanced HF service, with circulatory mechanical devices and heart transplantation, with multiple referrals of young patients without noncardiac comorbid conditions as candidates for these advanced therapies. Almost half of the patients had a reduced ejection fraction, 37% of the patients had previous AHF hospitalizations, and the majority of the cohort was previously symptomatic (New York Heart Association > II). Patients with severely impaired LVEF, New York Heart Association III to IV, and previous hospitalizations were found more frequently in classes C, D, and E.

Analysis in this specific population with no myocardial infarction, CS is relevant because patients with decompensation in the context of chronic HF may present with different symptomatology and may also have different hemodynamic profiles in that they may have developed adaptations to allow them to tolerate lower cardiac output and blood pressure.[11] Indeed, because of compensatory mechanisms and adaptations, patients with chronic HF may display a lower SCAI SHOCK stage than those without such adaptive mechanisms or may provide a falsely reassuring clinical picture despite high-risk hemodynamics.[12]

Based on underlying differences in pathophysiology and time course, AMI and AHF shock have different hemodynamic profiles and clinical outcomes.[13]

In the Cardiogenic Shock Working Group registry, patients in the AMI cohort had higher LVEF and lower mean pulmonary pressures, and hospital mortality was significantly higher in patients with AMI.[14] These differences were recently evaluated in another large cohort of patients with CS. Those with HF were younger, had fewer cardiac arrests, less vasopressor use, lower cardiac output, and higher pulmonary capillary wedge pressure than patients with AMI.[15] The HF-CS cohort was younger, had fewer cardiac arrests, less vasopressor utilization, lower cardiac power output, and higher pulmonary capillary wedge pressure than patients with AMI-CS. Patients with HF-CS received less temporary mechanical circulatory support and experienced lower rates of in-hospital mortality (23.9% vs 39.3%, $P < .001$). After discharge, patients with HF-CS had a lower 1-year mortality (42.6%) than patients with AMI-CS (52.9%, $P = .03$).

In the present study, the SCAI classification successfully identified hospitalized patients with AHF in different risk strata. To understand the predicted ability of this classification in a more homogenous population with a primary diagnosis of AHF, we decided to exclude patients with AMI, severe sepsis, and pulmonary embolism, because in these cases, HF is secondary to these diseases.

Since the publication of the SCAI SHOCK stage classification in 2019,[7] several groups have produced observational validation studies

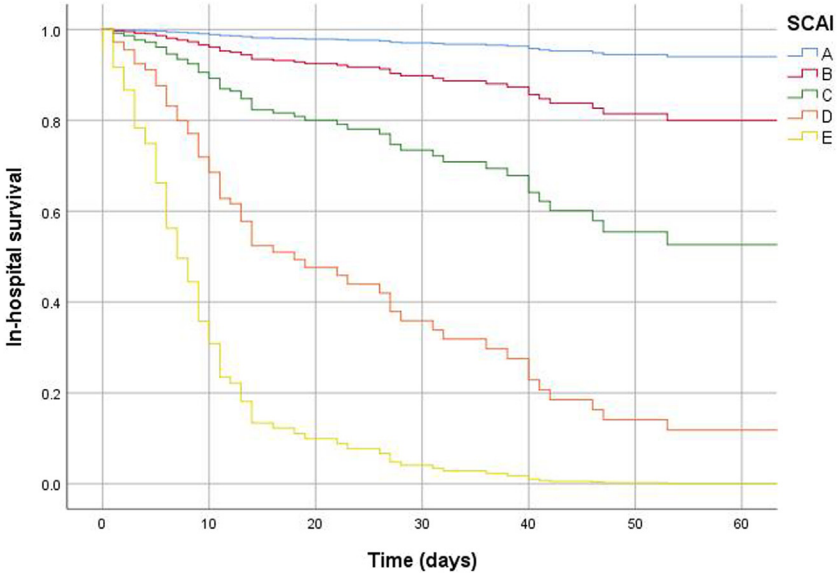


Figure 1. In-hospital adjusted survival per SCAI class.

Table 3. Baseline characteristics and therapies of patients according to SCAI SHOCK stage.

	SCAI SHOCK Stage					*P*
	A	B	C	D	E	
Age, y	77.7 ± 11	74.5 ± 13.5	71.9 ± 14	64.3 ± 14.3	61. ± 18.4	<.001
Male sex	210 (61.6)	207 (61.4)	89 (73.6)	26 (74.3)	13 (59.1)	.78
Arterial hypertension	91 (26.7)	105 (31.2)	39 (32.2)	10 (28.6)	8 (36.4)	.61
Diabetes	91 (26.7)	105 (31.2)	39 (32.2)	10 (28.6)	8 (36.4)	.61
Dyslipidemia	215 (63)	211 (62.6)	77 (63.6)	18 (51.4)	9 (40.9)	.60
LVEF <40%	126 (37)	139 (41.2)	81 (66.9)	30 (85.7)	15 (68.2)	<.001
Prior NYHA functional class						<.001
Not available	82 (24)	83 (24.7)	19 (15.7)	9 (25.7)	4 (18.2)	
I	92 (27)	107 (31.8)	38 (31.4)	5 (14.3)	3 (13.6)	
II	116 (34)	104 (30.9)	28 (23.1)	10 (28.6)	5 (22.7)	
III	47 (13.8)	41 (12.2)	32 (26.4)	8 (22.9)	9 (40.9)	
IV	4 (1.2)	2 (0.6)	4 (3.3)	3 (8.6)	1 (4.5)	
Prior HF hospitalization	122 (35.8)	102 (30.3)	70 (57.9)	15 (42.9)	13 (59.1)	<.001
Vasoactives	0 (0)	0 (0)	78 (64.5)	31 (88.6)	17 (77.3)	<.001
IABP	0 (0)	0 (0)	1 (0.8)	20 (57.1)	11 (50)	<.001
VA ECMO	0 (0)	0 (0)	0 (0)	4 (11.4)	4 (18.2)	<.001
Renal replacement therapy	1 (0.3)	5 (1.5)	3 (2.5)	6 (17.1)	2 (9.1)	<.001
Mechanical ventilatory assistance	1 (0.3)	6 (1.8)	14 (11.6)	29 (82.9)	13 (59.1)	<.001
Lactate, mmol/L	1.5 (1.2-2.1)	1.9 (1.4-2.7)	3.3 (2.4-4.7)	6.6 (4.6-9.8)	15.3 (10.4-18)	<.001
Aspartate-aminotransferase, U/L	28 (18-30)	35 (25-49)	48 (32-97)	90 (55-642)	520 (134-3414)	<.001
Alanine aminotransferase, U/L	18 (14-25)	35 (21-51)	46 (26-97)	72 (39-318)	328 (98-1830)	<.001
Creatinine, mg/dL	1.4 (1.1-1.8)	1.47 (1.2-1.9)	1.88 (1.5-2.4)	2.33 (1.6-3)	2.48 (1.8-3.1)	<.001
Bilirubin, mg/dL	1 (0.7-1.5)	1 (0.7-1.6)	1.98 (1.2-3)	2.65 (1.3-4)	3.98 (2-6)	<.001

Values are expressed as mean ± SD, n (%), or median (Q1-Q3).
HF, heart failure; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; VA ECMO, veno-arterial extracorporeal membrane oxygenation.

that uniformly demonstrate an association between the SCAI SHOCK stage and mortality risk in a variety of populations.[16] Ensuing validation studies documented its ease and rapidity of use as well as its ability to meaningfully discriminate patient risk across the spectrum of CS, including various phenotypes, presentations, and health care settings.[8,9,14,17-21] Finally, the simplicity and speed of its use was also highlighted, which is a fundamental characteristic for this type of tool.

Unlike the patients included in our study, the vast majority of published validation studies included patients with heterogeneous pathologies admitted to the coronary intensive care unit, CS, AMI, or out-of-hospital cardiac arrest. Additionally, they compared the primary end point with a maximum observation time of 30 days.[8,9,17,20] In the group of patients with primary AHF, further follow-up is necessary because the period immediately after discharge from hospitalization carries a particularly high risk of poor clinical outcomes and is known as the "vulnerable phase" of the disease.[22,23] Although morbidity and mortality still occur during hospitalization, substantially, the majority of events occur after patients with HF have been discharged from the hospital.[24]

In the present study and those previously published, stratification of mortality risk in the validation studies remained consistent, underscoring the strength of the SCAI classification scheme (Figure 3).[9,14,16–21,25] The aforementioned adaptive mechanisms in patients with chronic HF may possibly explain the lower in-hospital mortality found in stages B and C than other cohorts of patients with pathologies of acute presentation. Unlike the patients evaluated in other cohorts with acute pathologies,

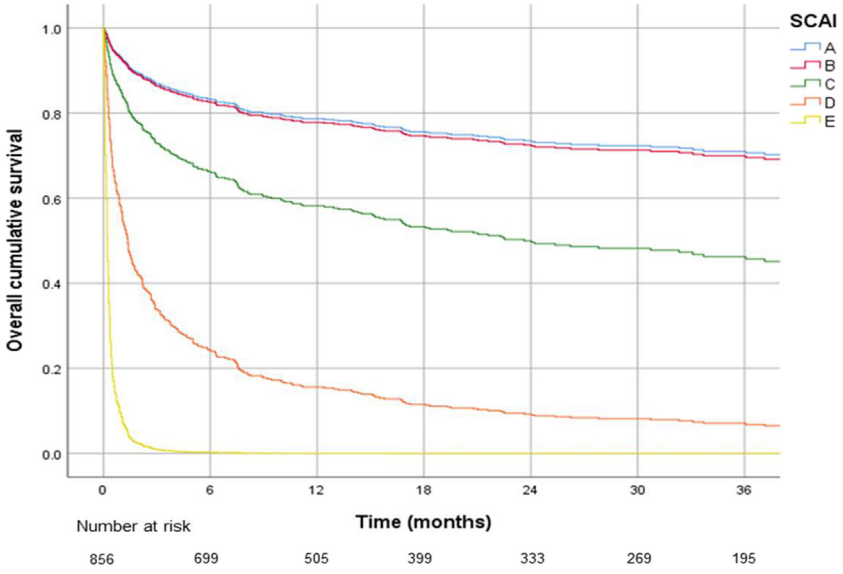


Figure 2. Long-term cumulative adjusted survival per SCAI SHOCKclass.

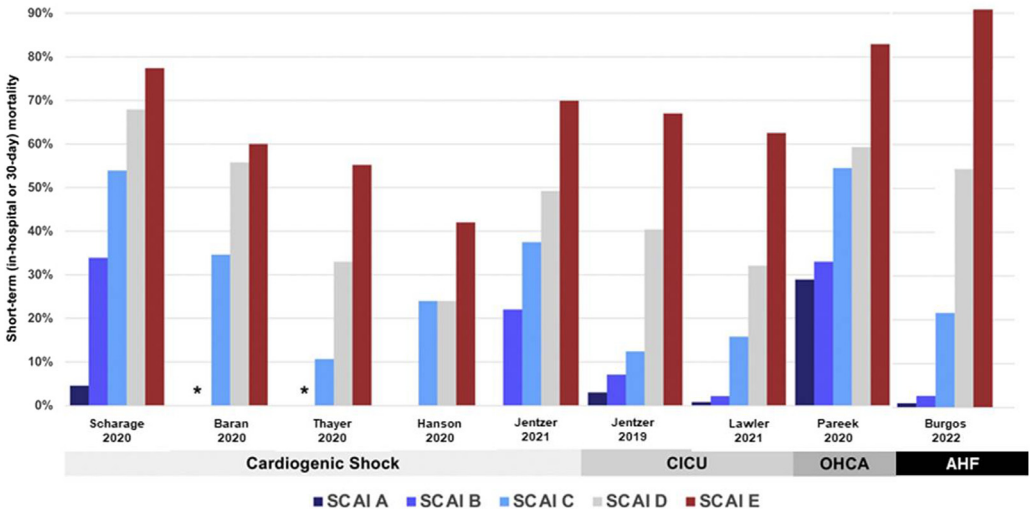


Figure 3. Short-term mortality as a function of SCAI SHOCK stages in each study. AHF, acute heart failure; CICU, cardiac intensive care unit; OHCA, out-of-hospital cardiac arrest.
Modified from Naidu SS, et al.[16]

such as AMI, without previous cardiovascular disease and without adaptive mechanisms, these patients frequently had previous hospitalizations for AHF, and in those whose functional class could be evaluated, >60% were symptomatic prior to the index hospitalization. This also highlights the importance of evaluating the classification in this group of patients exclusively.

For the SCAI classification, in addition to providing mortality risk stratification, its greatest value is linked to the standardization of shock severity assessment to improve clinical communication and decision making. All patients with CS should be rapidly transferred to a tertiary care center that has a 24/7 service of cardiac catheterization and a dedicated intensive care unit with availability of short-term mechanical circulatory support.[26] In low and middle income countries, most centers are not equipped with all modalities for CS care, and, therefore, some patients should be transferred to a primary shock center, also called a "hub," which has the capacity and technology to care for all patients.[2,27] In a large real-world study, the "hub-and-spoke" triage system and treatment of CS at transfer hubs was associated with significantly lower mortality.[28] SCAI classification, if incorporated as a standard of care in the evaluation of patients with decompensated HF, could help identify those patients who should be transferred early to specialized CS centers. This assessment must be systematized when the patient is admitted on duty and applied with each new evaluation in order to request a transfer before hemodynamic collapse, and, in this way, avoid executing a high-risk transfer with the patient in advanced shock. Additionally, it is important to note that this classification can allow us to identify the most severe patients in whom the possibility of survival is lower, such as stage E – close to 95%, in which the referral to these centers of patients with high-risk, unrecoverable CS may be futile.

In countries like Argentina with great heterogeneity in the distribution of resources and extensive territories, the standardized application of this score could be especially useful for managing transfers within the framework of a structured health network. To implement it, the leadership of national and regional organizations will be strengthened to lead the implementation of CS care systems.

As limitations, we can mention it is a retrospective analysis with the biases inherent to this type of design. However, the analysis of a prospectively loaded database, avoiding missing data, and with a hard end point of interest such as the mortality that was assessed in all patients, could reduce the existence of potential biases. Another important limitation to mention is that natriuretic peptides were not used for the group classification, given the limitation of their use in our country because of lack of medical coverage. Finally, the study was conducted in a highly complex cardiovascular center; therefore, the sample may not be representative of other centers.

To the best of our knowledge, the ability of the SCAI SHOCK staging classification to predict in-hospital and long-term mortality in patients with primary AHF was not previously evaluated. The SCAI classification of shock is a consensus-based classification that was designed to be a pragmatic and practical tool that could be applied in clinical practice in this selected population.

Conclusions

We provide real-world validation of the SCAI SHOCK staging scheme as an approach to identify patients with AHF at risk of in-hospital and long-term mortality. In this large clinical cohort of patients with primary AHF exclusively, the SCAI SHOCK staging classification was associated with in-hospital and long-term mortality. This finding supports the rationale of the classification in this setting. Further prospective studies are needed to validate these findings.

Declaration of competing interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding sources

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethics statement

This study was approved by the institutional research and ethics board and was registered on the PRIISA.BA platform of Buenos Aires city Ministry of Health. The study was a retrospective investigation with deidentifed data, and according to national regulations, request for informed consent was excepted. At the time of hospitalization, patients signed consent for the transfer of personal data for scientific purposes. The study was carried out in accordance with national and international standards for the protection of research subjects such as the Declaration of Helsinki, Resolution of the Ministry of National Health 1480/2011

Ciudad de Buenos Aires law 3301, ANMAT resolution 6677/10, and amendments 4008 and 4009.

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