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Clinical Presentation, Classification, and Outcomes of Cardiogenic Shock in Children

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ABSTRACT

BACKGROUND Despite growing cardiogenic shock (CS) research in adults, the epidemiology, clinical features, and outcomes of children with CS are lacking.

OBJECTIVES This study sought to describe the epidemiology, clinical presentation, hospital course, risk factors, and outcomes of CS among children hospitalized for acute decompensated heart failure (ADHF).

METHODS We examined consecutive ADHF hospitalizations (<21 years of age) from a large single-center retrospective cohort. Patients with CS at presentation were analyzed and risk factors for CS and for the primary outcome of in-hospital mortality were identified. A modified Society for Cardiovascular Angiography and Interventions shock classification was created and patients were staged accordingly.

RESULTS A total of 803 hospitalizations for ADHF were identified in 591 unique patients (median age 7.6 years). CS occurred in 207 (26%) hospitalizations. ADHF hospitalizations with CS were characterized by worse systolic function ($P = 0.040$), higher B-type natriuretic peptide concentration ($P = 0.032$), and more frequent early severe renal ($P = 0.023$) and liver ($P < 0.001$) injury than those without CS. Children presenting in CS received mechanical ventilation (87% vs 26%) and mechanical circulatory support (45% vs 16%) more frequently (both $P < 0.001$). Analyzing only the most recent ADHF hospitalization, children with CS were at increased risk of in-hospital mortality compared with children without CS (28% vs 11%; OR: 1.91; 95% CI: 1.05-3.45; $P = 0.033$). Each higher CS stage was associated with greater inpatient mortality (OR: 2.40-8.90; all $P < 0.001$).

CONCLUSIONS CS occurs in 26% of pediatric hospitalizations for ADHF and is independently associated with hospital mortality. A modified Society for Cardiovascular Angiography and Interventions classification for CS severity showed robust association with increasing mortality. (J Am Coll Cardiol 2024;83:595-608) © 2024 by the American College of Cardiology Foundation.

Cardiogenic shock (CS) is a low cardiac output state resulting in tissue hypoxia and life-threatening end-organ hypoperfusion.[1] Recent studies in adult patients have shed new light on the epidemiology, risk stratification, and best practice for treatment of CS.[1-9] In an effort to more accurately predict mortality in patients with CS and provide harmonization of research across centers, the Society for Cardiovascular Angiography and Interventions (SCAI) proposed and validated a shock

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ABBREVIATIONS AND ACRONYMS

ADHF = acute decompensated heart failure

ALT = alanine transaminase

BNP = B-type natriuretic peptide

CPR = cardiopulmonary resuscitation

CS = cardiogenic shock

ECMO = extracorporeal membrane oxygenation

eGFR = estimated glomerular filtration rate

HF = heart failure

HRT = heart replacement therapy

MCS = mechanical circulatory support

NHS = native heart survival

SCAI = Society for Cardiovascular Angiography and Interventions

classification system for grading clinical severity of patients with or at risk for CS.[10,11] Similarly, contemporary reports of multidisciplinary shock teams recommend a standardized approach to diagnosis and treatment of CS in its early stages.[12-14] Such efforts raise the standard of care for patients hospitalized with CS and provide a means for reducing the stagnant mortality rates in adults.

However, while CS science has rapidly evolved in adult patients, there are no detailed studies describing the etiology, incidence, or risk of morbidity and mortality of CS in children. In a recent scientific statement from the American Heart Association, several research priorities in CS were highlighted, though none were focused on the pediatric population.[1] Hence, the primary aim of this study was to describe the epidemiology, clinical presentation, and outcomes of CS among children hospitalized for acute decompensated heart failure (ADHF). We also sought to create a modified SCAI shock classification suitable for use in children.

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METHOD

STUDY POPULATION. We retrospectively analyzed a cohort of consecutive patients <21 years of age who were hospitalized for ADHF at Texas Children's Hospital from January 1, 2004, to December 31, 2018. We defined ADHF as the gradual or rapid clinical deterioration with signs or symptoms of heart failure (HF), resulting in the need for hospitalization and urgent therapy.

Potential patients were identified from the daily patient census maintained by the inpatient HF service. A pediatric HF specialist reviewed each hospitalization record to determine whether the admission was primarily for the treatment of ADHF. Patients with alternative etiologies of decompensation or shock, including sepsis or hypovolemia, were excluded. Patients with critical obstructive outflow tract or aortic arch lesions (eg, critical aortic stenosis, coarctation of the aorta) were also excluded. Patients with other types of congenital heart disease and HF (eg, Fontan circulation with depressed systolic function, transposition of the great arteries with history of coronary insult and subsequent depressed systolic function) were included. The study was approved by the Institutional Review Board of the Baylor College of Medicine.

DATA AND DEFINITIONS. We collected data for vital signs, physical examination, demographics, clinical history, laboratory, echocardiography, radiography, procedures, treatments, and outcomes from the electronic medical record. Vital sign and laboratory data represent the first set recorded in the medical record at presentation to the hospital. Lactate concentrations are the highest value recorded in the first 24 hours of admission. Missing data were not imputed.

CS was defined as ADHF attributable to ventricular dysfunction plus ≥2 of the following within the first 24 hours of presentation: 1) lactate >2 mmol/L; 2) documentation of being cool to touch on physical examination; 3) systemic hypotension; or 4) cardiopulmonary resuscitation (CPR) within 24 hours of admission. Systemic hypotension was defined as a systolic blood pressure <5th percentile for age and height or <60 mm Hg for patients <1 year of age.[15,16] Heart replacement therapy (HRT) was defined as children who received a durable ventricular assist device or underwent cardiac transplantation during the hospitalization. Native heart survival (NHS) was defined as children who were discharged home without HRT. Variations of these definitions have been previously used in adult CS outcomes analysis.[17] Severe acute renal injury at presentation was defined as an estimated glomerular filtration rate (eGFR) <30 mL/min/1.73 m^2 at presentation. The eGFR was calculated using the revised bedside Schwartz equation.[18] Hepatic injury at presentation was defined as an alanine transaminase (ALT) concentration ≥100 U/L and/or prothrombin time ≥17 seconds at admission. Late deterioration was defined as increasing inotrope or vasopressor use, mechanical circulatory support (MCS) utilization, or CPR after 24 hours. A modified SCAI shock severity classification was created (Table 1).

OUTCOMES. The primary outcome in our study was in-hospital death, defined as any-cause death during the index hospitalization. For this analysis as well as for the Kaplan-Meier in-hospital survival curves, we included only the most recent hospitalization for each unique patient (to avoid confounding by multiple admissions, as each patient can experience mortality only once). Secondary outcomes included HRT and NHS.

TABLE 1 Modified SCAI Classification for Severity of CS

CS Stage	Modified SCAI Definition
Stage A: "at risk"	Children with ADHF who are hemodynamically stable with normal perfusion but are at risk of developing CS
Stage B: "beginning"	Children with ADHF who are hypotensive OR: require treatment with vasoactive medications but have normal perfusion
Stage C: "classic"	Children with ADHF and CS who are hypotensive AND receive treatment with vasoactive medications OR: display features of hypoperfusion (ie, cool extremities or lactate >2 mmol/L)
Stage D: "deteriorating"	Children with ADHF and CS whose hemodynamic instability requires >2 vasoactive medications or mechanical circulatory support
Stage E: "extremis"	Children with ADHF and CS with overt circulatory collapse necessitating cardiopulmonary resuscitation

ADHF = acute decompensated heart failure; CS = cardiogenic shock; SCAI = Society for Cardiovascular Angiography and Interventions.

END-ORGAN INJURY IN CS COHORT. Among the subset of unique patients with CS, we examined the risk of in-hospital mortality based on severe acute renal and hepatic injury at the time of presentation. Such clinical phenotypes of end-organ injury have been described in adult CS.[19]

STATISTICAL ANALYSIS. For the primary aim of our study, we pursued a descriptive statistical analysis to characterize the case cohort that presented in CS. We used frequency and proportion to describe categorical variables and median (Q1-Q3) or mean ± SD to describe nonparametric or normally distributed continuous variables, respectively. We performed univariate analysis to describe clinical features associated with CS, using the chi-square or Fisher exact test (as statistically appropriate) for categorical variables and paired Student's *t*-test (if normal distribution) or nonparametric Mann-Whitney *U* test (if not normally distributed) for continuous variables. We then performed binary logistic regression to assess for factors independently associated with CS using 2 different models. For the conventional data-driven model construction, we included variables with univariate $P < 0.10$. For the theory-driven model, we included age, race/ethnicity (surrogate for socioeconomic determinants and potentially reduced access to timely health care), etiology of HF (myocarditis may be more likely to present in CS), eGFR at presentation (renal dysfunction predisposing to fluid overload may predispose to presentation in CS), and ejection fraction at presentation (worse systolic function predisposing to presentation in CS).

For the primary outcome of hospital mortality, we performed univariate analysis (as statistically appropriate, as described previously). We then performed binary logistic regression using 2 different models—a conventional data-driven model including variables with univariate $P < 0.10$ and a theory-driven model. For the latter, we included age, sex, etiology of HF, prior history of HF, and CS at presentation. A separate logistic regression analysis was performed to determine the association between severity of shock (per modified SCAI classification) with inpatient mortality, adjusting for age, sex, diagnosis, history of HF, and eGFR at admission.

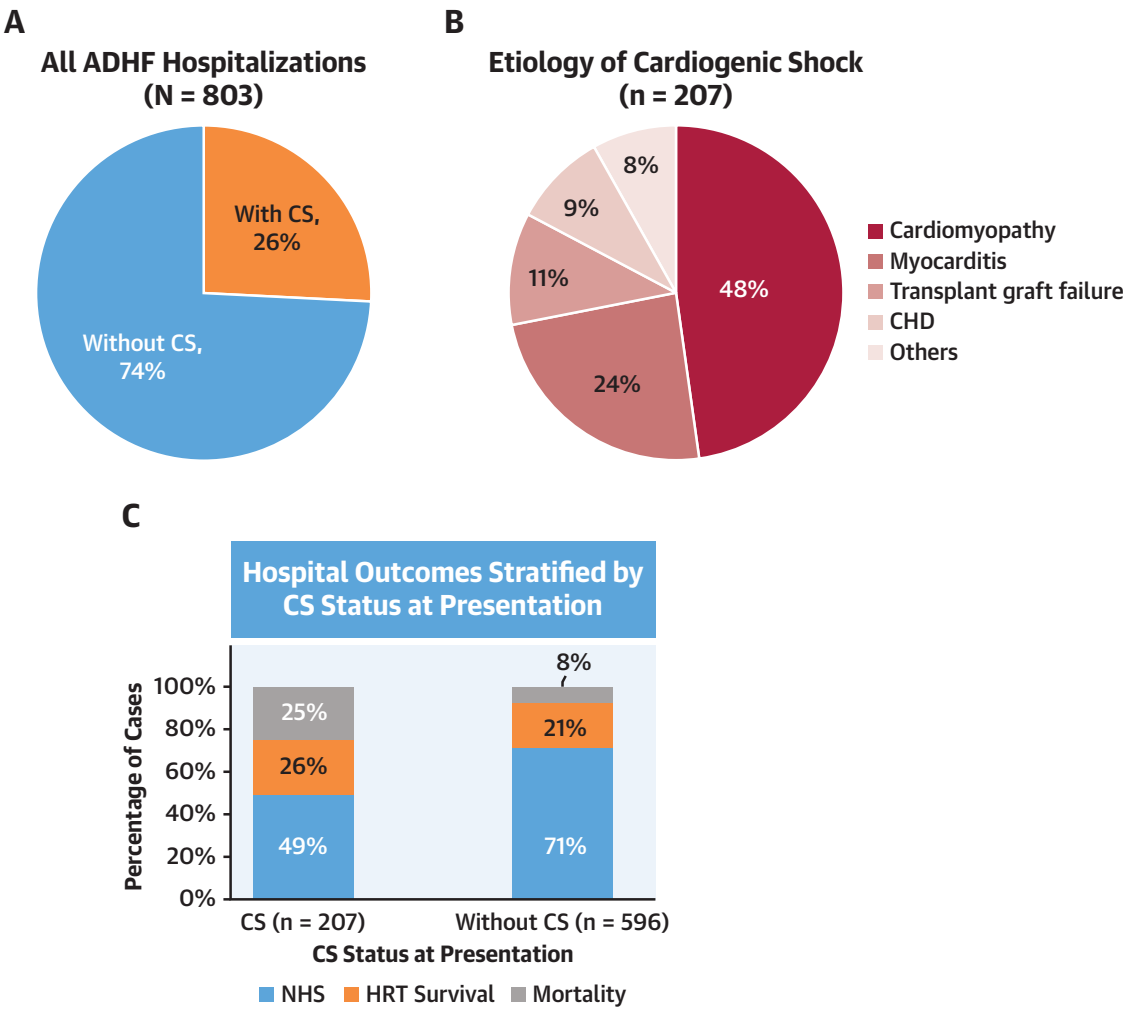
To examine the association of eGFR <30 mL/min/1.73 m^2 and hepatic injury with mortality in CS, unique patients were analyzed, using data from the most recent hospitalization in cases of multiple admissions. Logistic regression analysis was performed to determine associations of severe renal and liver injury with worse outcomes among patients with CS. A P value <0.05 was considered statistically significant.

Statistical analysis was performed with IBM SPSS Statistics version 25.0 and R version 4.3.1 (R Foundation for Statistical Computing).

RESULTS

PATIENT CHARACTERISTICS AND ASSOCIATIONS WITH CS. During the study period, 803 ADHF hospitalizations for 591 unique patients met inclusion criteria. CS occurring within 24 hours of presentation was identified in 26% (n = 207) of ADHF hospitalizations (Central Illustration). Table 2 displays demographics and baseline characteristics of children admitted in ADHF with and without CS. The median age of the cohort was 7.6 years (Q1-Q3: 1.1-14.7 years). The most common etiology of HF overall was cardiomyopathy (52%). The median central venous pressure was 16 mm Hg (Q1-Q3: 13-19 mm Hg) for those with data available (n = 92). Patients presenting

CENTRAL ILLUSTRATION **Etiology and In-Hospital Outcomes of Admissions for Acute Decompensated Heart Failure and Cardiogenic Shock**



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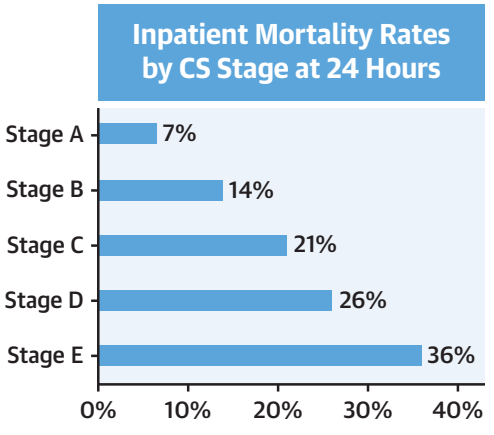
CS Stage	Study Definition
Stage A "At risk"	No hypotension, no vasoactive agents, no other criteria for CS
Stage B "Beginning"	Hypotension OR vasoactive agents without other criteria for CS
Stage C "Classic"	Hypotension AND vasoactive agents OR other criteria for CS
Stage D "Deteriorating"	MCS or >2 vasoactive agents
Stage E "Extremis"	Circulatory collapse with CPR



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(A) Pie chart depicting the distribution of children with acute decompensated heart failure (ADHF) who presented in cardiogenic shock (CS). (B) Pie chart depicting the distribution of the etiology of CS. (C) Bar graphs illustrating outcomes of hospitalization including in-hospital mortality, heart replacement therapy (HRT) with survival, and native heart survival (NHS). (D) Modified Society for Cardiovascular Angiography and Interventions stage definitions and hospital mortality stratified by Society for Cardiovascular Angiography and Interventions stage at 24 hours. CHD = congenital heart disease; CPR = cardiopulmonary resuscitation.

in CS were more likely to have a diagnosis of myocarditis than patients without CS. Systolic function on echocardiogram was worse, and B-type natriuretic peptide (BNP) concentrations were greater in children with CS. There was greater evidence of hepatic and renal end-organ injury in the CS cohort compared with patients without CS. The median length of stay for CS hospitalizations was significantly longer than non-CS hospitalizations (median 29 days vs 17 days; $P < 0.001$). Admission laboratory and resource utilization comparisons are displayed in Table 3. Multivariable analyses of baseline clinical characteristics associated with CS at presentation are shown in Supplemental Tables 1 and 2.

RESOURCE UTILIZATION IN CS AND ASSOCIATIONS WITH MCS. The overwhelming majority of hospitalizations that met criteria for CS were first admitted to the intensive care unit (95%). Patients with CS were more likely to receive treatment with mechanical ventilation, vasoactive agents, and renal replacement therapy compared with those without CS (Table 3). Early utilization of MCS was higher in CS admissions with 25% of the CS cohort receiving MCS within 24 hours of admission (Figure 1).

The case cohort receiving MCS during their admission for ADHF (n = 190) is described in Supplemental Tables 3 and 4. Admissions for ADHF with CS are characterized based on initiation of MCS in Tables 4 and 5. Those receiving MCS were older in age and more likely to have either myocarditis or transplant graft failure as the underlying etiology of cardiac dysfunction. They also had higher BNP and worse systolic function on echocardiogram. Those receiving MCS also had higher resource utilization (Table 4), with over one-third undergoing renal replacement therapy, and a 2 times longer hospital length of stay, compared with the non-MCS group. Among hospitalizations of ADHF with CS who received MCS during the admission, 26% died (compared with 24% of those who did not receive MCS; $P = 0.724$), while nearly 52% were discharged without a heart transplant (compared with 67% of those who did not receive MCS; $P = 0.006$).

MODIFIED CS SHOCK CLASSIFICATION STAGES. The distribution of SCAI shock stages in all ADHF hospitalizations at presentation, at 24 hours, and the maximum/highest shock severity during admission is shown in Figure 2. Early escalation of shock stage within 24 hours of admission was observed in 16% (n = 128 of 803). Among those who escalated early to stages D or E, 72% (n = 89 of 123) presented in stage C initially. CPR was performed in 9% of (n = 71 of 803) patients within 24 hours of presentation, and of them, 32 patients arrested in the emergency department very shortly after arrival. Late deterioration after 24 hours occurred in 18% (n = 144 of 803) of patients, including 15% (n = 119 of 803) who were in stages A or B at 24 hours. Among those in stage A or B who developed late deterioration, the maximum stage progression was C in 2 hospitalizations, D in 74 hospitalizations, and E in 43 hospitalizations. Median time to late deterioration was 14 days (Q1-Q3: 7-31 days).

TABLE 2 Demographics and Clinical Characteristics of ADHF Hospitalizations With and Without CS

	With CS (n = 207)	Without CS (n = 596)	*P* Value
Age, y	4.0 (0.4-11.7)	8.7 (1.6-15.2)	<0.001
Male	100 (48)	330 (55)	0.079
Race/ethnicity[a]			
Asian	8 (4)	17 (3)	0.577
Hispanic	53 (26)	149 (25)	
Non-Hispanic Black	49 (24)	171 (29)	
Non-Hispanic White	96 (46)	258 (43)	
Era			
2004-2010	74 (36)	219 (37)	0.798
2011-2018	133 (64)	377 (63)	
Prior history of HF	55 (27)	349 (59)	<0.001
Etiology of HF			
Cardiomyopathy	99 (48)	321 (54)	<0.001
Myocarditis	50 (24)	42 (7)	
Transplant graft failure	22 (11)	87 (15)	
Congenital heart disease	19 (9)	107 (18)	
Others	17 (8)	39 (7)	
Clinical history			
Fatigue or decreased activity	133 (64)	349 (59)	0.110
Dyspnea or increased WOB	152 (73)	383 (64)	0.011
Nausea or vomiting	98 (47)	239 (40)	0.066
Loss of appetite or decreased oral intake	99 (48)	228 (38)	0.016
Abdominal pain[b]	50/103 (49)	153/396 (26)	0.068
Syncope	20 (10)	24 (4)	0.002
Chest pain[b]	27/103 (26)	86/396 (22)	0.344
Physical examination findings			
Tachypnea	114 (55)	301 (51)	0.045
Tachycardia	125 (60)	235 (39)	<0.001
Cool to touch	129 (62)	72 (12)	N/A
Hypotension	89 (43)	52 (9)	N/A
Retractions	63 (30)	70 (12)	<0.001
Rales	40 (19)	100 (17)	0.369
Gallop	104 (50)	279 (47)	0.312
Hepatomegaly	115 (56)	294 (49)	0.107
Peripheral edema	27 (13)	173 (29)	<0.001

Values are median (Q1-Q3), n (%), or n/N (%). [a]One patient of "Other" race/ethnicity in each of the "With CS" and "Without CS" cohorts. [b]Among children ≥3 years of age.

HF = heart failure; N/A = not applicable; WOB = work of breathing; other abbreviations as in Table 1.

TABLE 3 Admission Laboratory and Resource Utilization for ADHF Hospitalizations With and Without CS

	With CS (n = 207)	Without CS (n = 596)	*P* Value
Radiographic findings			
Cardiomegaly	164 (79)	501 (84)	0.156
Pulmonary congestion	111 (54)	258 (43)	0.010
Pleural effusion	48 (23)	166 (28)	0.177
Echocardiogram			
EF (n = 535), %	22 (16-30)	26 (19-38)	<0.001
SF (n = 426), %	13 (9-18)	16 (10-22)	0.005
LVEDD *z* score (n = 540)	2.9 (1.2-6.3)	3.6 (0.2-6.2)	0.589
Qualitative systolic function			
Preserved	1/199 (1)	57/553 (10)	<0.001
Mildly depressed	6/199 (3)	31/553 (6)	
Moderately depressed	26/199 (13)	99/553 (18)	
Severely depressed	166/199 (83)	366/553 (66)	
Laboratory			
Lactate (n = 404), mmol/L	4.6 (2.5-9.3)	1.5 (1.1-2.0)	N/A
BNP (n = 737), pg/mL	3,304 (1,293-5,000)	1,648 (752-3,270)	<0.001
Sodium (n = 790), mmol/L	138 (134-141)	137 (135-139)	0.006
Hemoglobin (n = 739), g/dL	12.1 (10.4-13.7)	12.5 (11.0-14.3)	0.005
ALT (n = 614), U/L	60 (32-197)	34 (24-58)	<0.001
AST (n = 604), U/L	94 (51-302)	47 (32-83)	<0.001
Total bilirubin (n = 565), mg/dL	0.6 (0.3-1.4)	0.6 (0.3-1.0)	0.193
PT (n = 357), s	19.9 (17.0-24.1)	16 (14.9-18.0)	<0.001
INR (n = 356)	1.7 (1.4-2.3)	1.3 (1.2-1.5)	<0.001
eGFR (n = 788), mL/min/1.73 m^2	63 (43-83)	82 (65-103)	<0.001
Resource utilization			
Admit to intensive care unit	197 (95)	430 (72)	<0.001
Vasoactive agent			
1 agent	38 (18)	218 (37)	<0.001
≥2 agents	160 (77)	165 (28)	
Mechanical ventilation	180 (87)	155 (26)	<0.001
Ventricular tachycardia	81 (39)	144 (24)	<0.001
Cardiopulmonary resuscitation	77 (37)	48 (8)	N/A
Renal replacement therapy	35 (17)	29 (5)	<0.001
MCS during hospitalization	93 (45)	97 (16)	<0.001
Heart transplantation during hospitalization	33 (16)	81 (14)	0.404
Hospital LOS, d	29 (17-72)	17 (9-46)	<0.001
Death during hospitalization	51 (25)	46 (8)	<0.001

Values are n (%), median (Q1-Q3), or n/n (%).

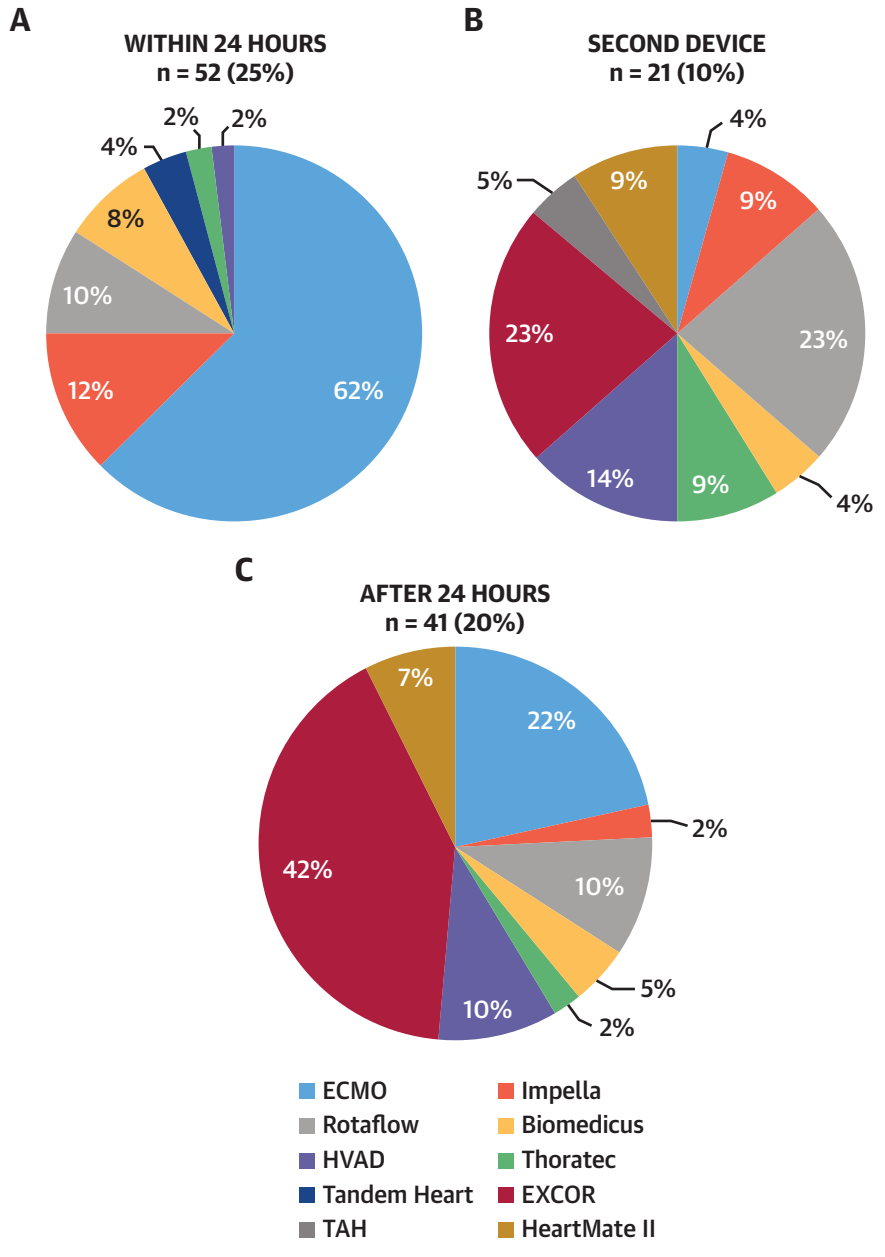
ALT = alanine transaminase; AST = aspartate transaminase; BNP = B-type natriuretic peptide; EF = ejection fraction; eGFR = estimated glomerular filtration rate; INR = international normalized ratio; LOS = length of stay; LVEDD = left ventricular end-diastolic dimension; MCS = mechanical circulatory support; N/A = not applicable; PT = prothrombin time; SF = shortening fraction; other abbreviations as in Table 1.

HOSPITALIZATION OUTCOMES AND ASSOCIATION OF CS WITH HOSPITAL MORTALITY. Figure 3 demonstrates in-hospital outcomes for all ADHF hospitalizations with and without CS. The in-hospital case fatality rate for the entire cohort was 12% (n = 97). A total of 203 (25%) hospitalizations resulted in HRT (durable MCS [17%] and/or heart transplantation [14%]), while in 525 (65%) hospitalizations, the patients were discharged home with NHS. Cases admitted with CS were more likely to receive HRT than cases without CS (32% vs 23%; unadjusted OR: 1.57; 95% CI: 1.11-2.22; $P = 0.011$) and more likely to receive temporary MCS (32% vs 3%; unadjusted OR: 18.13; 95% CI: 10.05-32.71; $P < 0.001$). Hospitalizations with CS were also less likely to result in NHS (49% vs 71%; unadjusted OR: 0.51; 95% CI: 0.40-0.64; $P < 0.001$). CS was present in 53% of the cases who died and in 22% of cases that survived. In other words, case fatality rate of ADHF admissions with CS at presentation was 25%, compared with 8% in those without CS at presentation (unadjusted OR: 3.91; 95% CI: 2.53-6.05; $P < 0.001$) (Table 3). Remarkably, 69 (74%) of 93 patient hospitalizations with CS that received MCS survived to hospital discharge, very similar to the survival rate for cases without CS that received MCS (n = 78 of 97 [80%]).

When testing for the association of clinical variables with inpatient mortality, we restricted our analysis to unique individuals only, using data from their most recent hospitalization (n = 591), shown in Supplemental Table 5. Among this cohort, the incidence of CS was 31% (n = 181 of 591), and the in-hospital mortality for the entire cohort was 16% (n = 96 of 591). Individual patients presenting with CS had a significantly higher mortality compared with those without CS (28% vs 11%; unadjusted OR: 3.18; 95% CI: 2.03-4.98; $P < 0.001$). The predictive values of the individual components of the CS definition are also shown in Supplemental Table 5. On multivariable regression analysis, CS at presentation remained independently associated with hospital mortality in both the data-driven and the theory-driven models (Table 6, Supplemental Table 6). Inpatient 90-day survival estimates for individuals with and without CS are presented in Figure 4 (with censoring at discharge; HR: 2.53; 95% CI: 1.67-3.85).

As the shock stage progressively increased, inpatient mortality significantly increased at all time points of assessment of CS severity (all $P < 0.001$) (Figure 5, Central Illustration, Supplemental Figure 1). When examining CS severity at 24 hours, compared with shock stage A, the adjusted ORs for inpatient mortality in shock stages B through E were 2.30 (95% CI: 1.10-4.80), 4.40 (95% CI: 1.80-10.70), 5.80 (95% CI: 2.20-15.50), and 9.50 (95% CI: 3.90-22.90), respectively ($P < 0.001$). Mortality rates were high among patients with early escalation of shock stage (n = 37 of 113 [33%]) and late deterioration (n = 42 of 122 [34%]). Hospital mortality was greater among patients who experienced late deterioration

FIGURE 1 MCS Characteristics in Children Hospitalized With CS



Utilization of mechanical circulatory support (MCS) by initial type, at 24 hours, and throughout the hospitalization for children with cardiogenic shock (CS). (A) One-quarter of children with CS received MCS within 24 hours of admission. Extracorporeal membrane oxygenation (ECMO) was the most common type of support during the first 24 hours (62%). (B) Second MCS devices used to transition to a more durable form of support at any point in the hospitalization. EXCOR (23%) and Rotaflow (23%) were the most commonly deployed devices when a second form of support was used. (C) MCS devices utilized beyond the first 24 hours of hospital admission. EXCOR was utilized most commonly as the initial form of support beyond the first 24 hours of hospitalization (42%). HVAD = Medtronic HeartWare HVAD ventricular assist device; TAH = SynCardia Total Artificial Heart.

TABLE 4 Comparison of Characteristics of MCS and Non-MCS Cohorts Among ADHF Hospitalizations With CS at Presentation

	MCS (n = 93)	No MCS (n = 114)	*P* Value
Age, y	6.4 (0.7-15.0)	2.0 (0.4-10.4)	0.011
Male	38 (41)	62 (54)	0.053
Etiology of HF			0.001
Cardiomyopathy	35 (38)	64 (56)	
Myocarditis	27 (29)	23 (20)	
Congenital heart disease	5 (5)	14 (12)	
Transplant graft failure	17 (18)	5 (4)	
Others	9 (10)	8 (7)	
Physical examination findings			
Tachycardia	62 (67)	63 (55)	0.133
Gallop	51 (55)	53 (46)	0.187
Dyspnea	64 (69)	88 (77)	0.176
Retractions	19 (20)	44 (39)	0.006
Clinical history			
Abdominal pain	25 (27)	28 (25)	0.131
Chest pain	38 (41)	38 (33)	0.135
Chest x-ray film with pleural effusion	26 (28)	22 (19)	0.134
BNP at admission, pg/mL	2,445 (1,172-4,460)	3,739 (1,434-7,590)	0.033
Sodium, mmol/L	137 (133-140)	139 (136-142)	0.009
Qualitative systolic function at admission			0.007
Preserved	1 (1)	0 (0)	
Mildly depressed	0 (0)	6 (5)	
Moderately depressed	8 (9)	18 (16)	
Severely depressed	81 (90)	85 (75)	
Shortening fraction on echo (n = 112)	11 (9-18)	14 (9-20)	0.121
LVEDD *z* score on echo (n = 149)	2.2 (0.3-6.1)	3.6 (1.7-6.8)	0.108

Values are median (Q1-Q3) or n (%). Factors with $P < 0.20$ shown in table. Other factors not significant on univariate analysis were race/ethnicity ($P = 0.759$); era ($P = 0.344$); prior h/o HF ($P = 0.589$); history/symptoms of fatigue ($P = 0.772$), syncope ($P = 0.350$), diaphoresis ($P = 0.439$), nausea ($P = 0.258$), or loss of appetite ($P = 0.703$); examination findings of tachypnea ($P = 0.541$), hepatomegaly ($P = 0.932$), rales ($P = 0.794$), jugular venous distension ($P = 0.699$), peripheral edema ($P = 0.691$); chest x-ray film findings of cardiomegaly ($P = 0.776$) or pulmonary congestion/edema ($P = 0.711$); laboratory findings of eGFR <30 mL/min/1.73 m^2 ($P = 0.539$), hemoglobin at admission ($P = 0.842$), ALT ($P = 0.385$), AST ($P = 0.904$), PT ($P = 0.395$), or hepatic injury ($P = 0.794$).

Abbreviations as in Tables 2 and 3.

TABLE 5 Resource Utilization and Outcomes of MCS and Non-MCS Cohorts Among ADHF Hospitalizations With CS at Presentation

Morbidity/Outcome	MCS (n = 93)	No MCS (n = 114)	*P* Value
Initial admission to intensive care unit	91 (98)	106 (93)	0.297
Mechanical ventilation	88 (95)	92 (81)	0.003
Vasoactive agent requirement			<0.001
None	0 (0)	9 (8)	
1 agent	7 (8)	31 (27)	
2 agents	86 (92)	74 (65)	
Renal replacement therapy	32 (34)	3 (3)	<0.001
Cardiopulmonary resuscitation	43 (46)	34 (30)	0.015
Ventricular tachycardia	47 (51)	34 (30)	0.002
Total inpatient time, d	47 (22-109)	23 (12-47)	<0.001
Time to death or discharge, d	42 (22-105)	22 (12-46)	<0.001
Heart transplantation during admission	22 (24)	11 (10)	0.006
Death in hospital	24 (26)	27 (24)	0.724

Values are n (%) or median (Q1-Q3).

Abbreviations as in Tables 2 and 3.

compared with patients without late deterioration (n = 42 of 122 [34%] vs n = 54 of 469 [12%]; OR: 4.04; 95% CI: 2.50-6.40; $P < 0.001$).

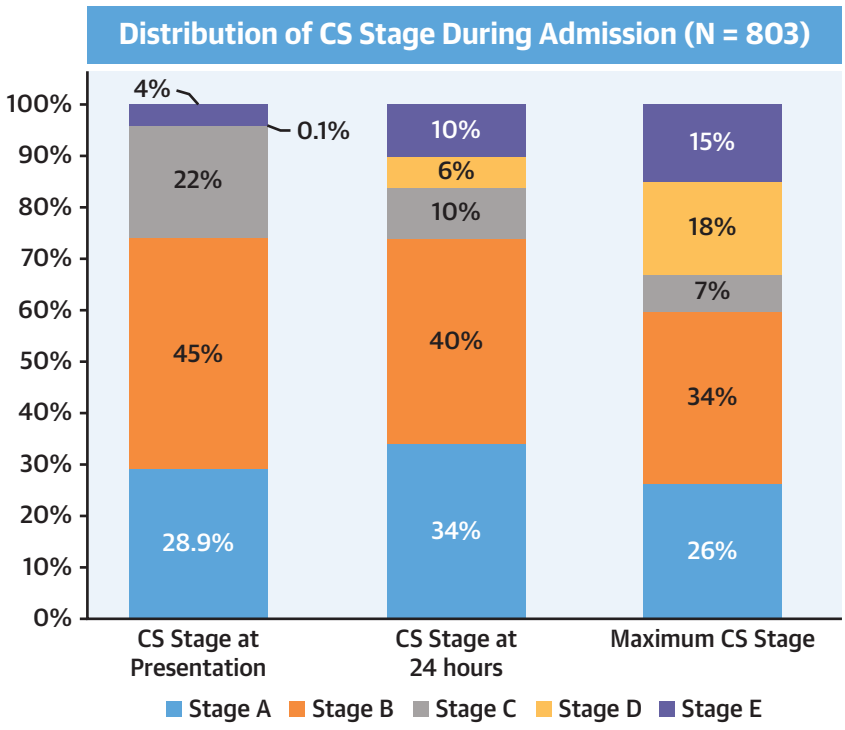
END-ORGAN INJURY IN CS SUBSET. Among 198 individual patients presenting in CS at their most recent admission, an eGFR could be calculated in 192 (97%). Median eGFR was 62 mL/min/1.73 m^2 (Q1-Q3: 43-83 mL/min/1.73 m^2). A total of 18 (9%) patients with CS and available eGFR data had eGFR <30 mL/min/1.73 m^2 at admission. Among these, in-hospital mortality rates were 56% (n = 10 of 18) vs 21% (n = 36 of 174) for those with and without severe renal injury, respectively (univariate $P = 0.002$). After adjusting for age, severe renal injury at admission was associated with in-hospital mortality (OR: 4.97; 95% CI: 1.80-13.75; $P = 0.002$) but was not associated with CPR (OR: 1.27; 95% CI: 0.46-3.49; $P = 0.644$). An ALT and/or prothrombin time value was measured at admission in 181 (91%) patients presenting in CS. Median ALT was 60 U/L (Q1-Q3: 32-195 U/L) and median prothrombin time was 20.0 seconds (Q1-Q3: 17.0-24.5 seconds).

Among patients with CS and available ALT/prothrombin time data, 129 (71%) met the definition of hepatic injury at admission. Hospital mortality rates were 26% (n = 34 of 129) vs 13% (n = 7 of 52) for those with and without hepatic injury, respectively (univariate $P = 0.061$). When adjusting for age, hepatic injury at admission was associated with CPR (OR: 4.15; 95% CI: 1.80-9.55; $P = 0.001$) but not with mortality (OR: 2.31; 95% CI: 0.95-5.60; $P = 0.065$).

DISCUSSION

This analysis of a large cohort of children hospitalized with all-cause ADHF is, to our knowledge, the first detailed description of the epidemiology, clinical presentation, and outcomes of CS in children. It is also the first attempt to classify CS severity into progressive clinical stages. This study yields several important findings. The prevalence of CS is high, occurring in about 1 in 4 pediatric admissions for ADHF. The in-hospital mortality rate for children with CS is also high (28%), similar to reports in adults and much greater than what is reported in other forms of pediatric shock (eg, 4%-11% in septic shock).[20] CS is a strong and independent predictor of in-hospital mortality, with affected children having a nearly 2-fold mortality rate compared with unaffected children. The most common etiology of CS in children is cardiomyopathy, with congenital heart disease representing <10% of the cohort. Invasive therapies are used frequently, and comorbid conditions develop more commonly in patients with CS

FIGURE 2 Distribution of CS Stages During ADHF Admissions



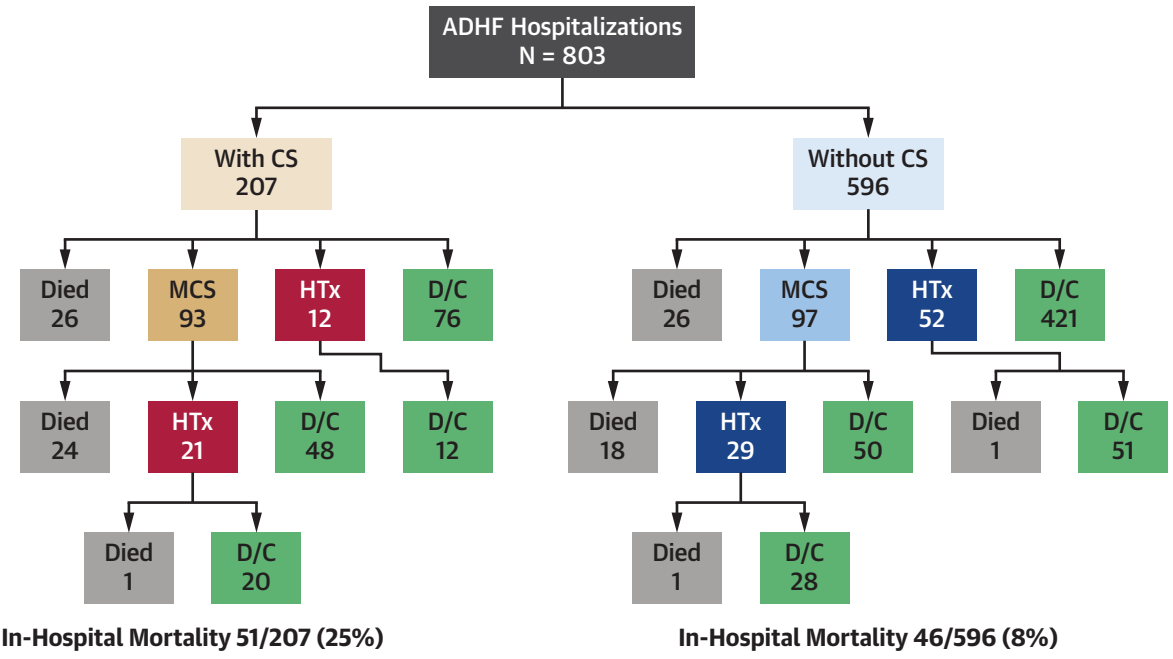
Bar graph depicting proportions of modified Society for Cardiovascular Angiography and Interventions stages A, B, C, D, and E as classified at presentation and at 24 hours, and the highest cardiogenic shock (CS) severity attained during the hospitalization. Early escalation of shock stage within 24 hours of admission was observed in 16%, while late deterioration after 24 hours occurred in 18%. ADHF = acute decompensated heart failure.

compared with those without CS. Hospital mortality increases with worsening shock severity stage. Early end-organ injury among CS patients portends a worse prognosis.

Previous study designs and data sources have thus far prohibited a reliable detailed empirical analysis of pediatric CS. Accordingly, the methodology of this study at a high-volume single center affords more granular data collection and greater confidence that these hospitalizations are primarily for ADHF, rather than noncardiac admissions for children with a prior diagnosis of HF. A recent report of 35 children hospitalized with the multi-inflammatory syndrome associated with COVID-19 infection showed that up to one-third presented in CS.[21] Likewise, Chan et al[22] reviewed a cohort of neonates with acute decompensated shock and found that CS accounted for 23% of cases over a 5-year period. Reports of acute HF in critically ill children more broadly have also been published, though limited by the reliance of large administrative databases on billing codes and restricted to a particular etiology of HF or a primarily descriptive report.[23-25]

CLINICAL FEATURES. We found that hospitalization for pediatric ADHF with CS is characterized by a diagnosis of cardiomyopathy or myocarditis, early and more frequent multisystem organ injury, greater use of advanced cardiac therapies, and higher mortality rates compared with ADHF without CS. Children with CS are >3 times as likely to have a diagnosis of acute myocarditis as the etiology of their HF compared with patients presenting without CS. Predictably, ADHF hospitalization presenting with CS were associated with more severely depressed left ventricular systolic function on echocardiography and higher concentrations of serum BNP. These observed differences across a cohort of children with ADHF signal that the markers of hypoperfusion used in our definition of CS, including physical examination, hypotension, and serum lactate, are key to identifying a shock state early in admission and may enable timely escalation of treatment.

FIGURE 3 Outcomes of ADHF Hospitalizations With and Without CS



Flowchart depicting the outcomes of all hospital admissions for children in acute decompensated heart failure with and without CS. Approximately one-third of admissions with CS underwent heart replacement therapy, while 49% were discharged home with native heart survival. One-fourth of CS admissions experienced in-hospital mortality. D/C = discharge; HTx = heart transplantation; other abbreviations as in Figures 1 and 2.

RESOURCE UTILIZATION AND OUTCOMES. While hospitalized, children with CS experience high acuity and have high rates of extracorporeal membrane oxygenation, CPR, ventricular tachycardia, and treatment with multiple vasoactive medications. Remarkably, CPR is needed in more than 1 in 3 CS hospitalizations. End-organ injury is common, manifesting as hepatic insufficiency, respiratory failure, and renal failure necessitating renal replacement therapy. Not surprisingly, hospital length of stay is prolonged at a median of nearly 1 month. Although the very high hospital mortality rate of 28% is comparable to rates reported for CS in adults, it markedly exceeds most other common pediatric conditions and illnesses. To place these findings into perspective, this observed mortality rate is nearly 3 times that of STAT-5 (Society of Thoracic Surgeons-European Association for Cardio-Thoracic Surgery category 5) congenital heart surgery at our center and 5 times the mortality rate for pediatric septic shock, highlighting urgent unmet needs for improving outcomes.[26,27]

TABLE 6 Multivariable Analysis (Theory-Based Model) for Variables Associated With In-Hospital Mortality Among Most Recent ADHF Hospitalizations (N = 591)

	Univariate *P* Value	Multivariable *P* Value	OR (95% CI)
Age	0.325	0.466	0.99 (0.95-1.02)
Male	0.063	0.080	0.67 (0.42-1.05)
Etiology of HF	0.072	0.072	
Cardiomyopathy		Ref.	Ref.
Myocarditis		0.478	0.79 (0.41-1.52)
Congenital heart disease		0.960	0.98 (0.49-1.98)
Transplant graft failure		0.076	1.83 (0.94-3.58)
Others		0.053	0.30 (0.09-1.02)
Prior history of HF	0.472	0.588	1.16 (0.68-1.96)
Shock at presentation	<0.001	<0.001	3.37 (2.10-5.40)

Abbreviations as in Tables 1 and 2.

INPATIENT MORTALITY AND RISK STRATIFICATION. The overall in-hospital mortality rate in our CS cohort was high (28%) but was similar to adults with CS not complicated by myocardial infarction (31%).[5] The SCAI shock classification system of stages A to E categorizes CS from "at risk" to "extremis" based on clinical classification at the time of admission.[10] Validation studies have shown that the SCAI staging provides a robust scheme for predicting risk of both in-hospital mortality and postdischarge mortality in

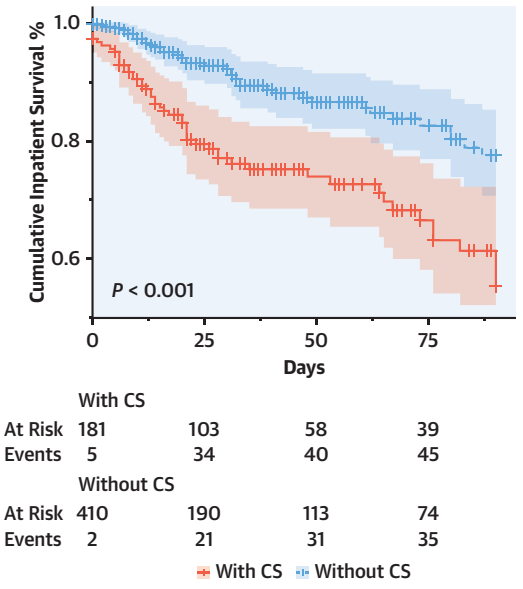
hospital survivors.[4,11] As in adults, we found higher inpatient mortality rates in children presenting in more advanced stages of CS. We also found that late worsening in shock severity was associated with an increase in mortality, a finding that has been corroborated in studies in adults.[11,28] Reclassification of shock severity after 24 hours of admission has also been studied and proposed to better prognosticate hospital outcomes, which may be an avenue for future work in pediatric CS as well.[29]

END-ORGAN INJURY. We found that severe renal injury (eGFR <30 mL/min/1.73 m^2) and hepatic injury at presentation were common and associated with in-hospital mortality in the ADHF cohort overall. Among the CS group, early end-organ injury heralded adverse outcomes, with severe renal injury being associated with death and liver injury with CPR during hospitalization. Worsening renal function is a well-known predictor of poor outcomes in both adults and children with ADHF.[30,31] Hepatic insufficiency, however, has never been described or tested in children with ADHF or CS. Manifesting as elevated transaminase levels and/or coagulopathy, acute hepatic injury was associated with a nearly 3-fold increased risk of CPR and a trend towards an increase in-hospital mortality compared with patients without hepatic injury. This is similar to what has been described in adult CS patients, with the highest in-hospital mortality rates occurring in those with lactic acidosis, kidney dysfunction, and transaminitis (often termed hemometabolic shock).[19,32]

FUTURE WORK. In contrast to the progress in CS in adult medicine, advances in pediatric CS have been lacking, even though rates of HF-related hospitalizations are increasing in children and currently represent 6% to 24% of pediatric cardiac intensive care unit admissions.[25,29,33] We believe that our sobering findings should serve as a clarion call for attention and study from the pediatric cardiology and critical care communities as well as guidance from professional societies such as the American Heart Association and the Society for Critical Care Medicine. A pediatric CS task force akin to the successful Society for Critical Care Medicine Surviving Sepsis Campaign,[20] in which evidence-based recommendations are created for clinicians caring for children with CS, may help advance the care of these patients.

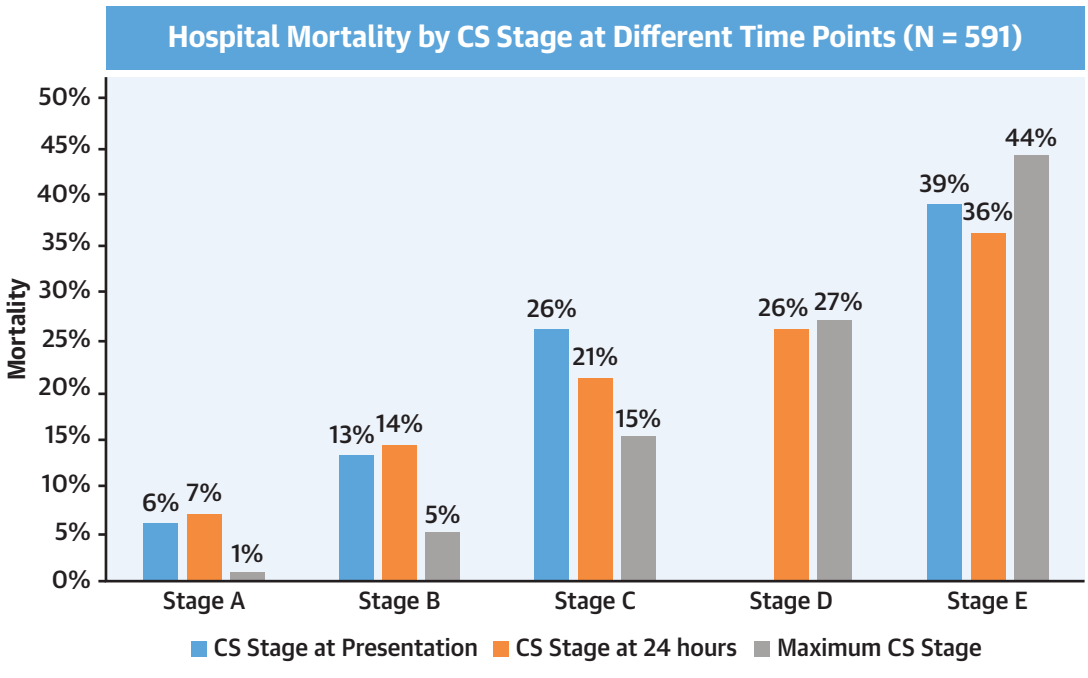
STUDY LIMITATIONS. First, we sought to define CS using clinical evidence of hypoperfusion, including standard criteria of elevated lactate, cool extremities on examination, and systemic hypotension. However, due to the study's retrospective design, serum lactate concentrations were not always measured in patients with ADHF, presumably because clinical suspicions of CS were low. This may have led to an underestimated incidence of CS in our cohort. Additionally, there is controversy regarding whether isolated hypotension should be classified as shock in adults. Second, serial blood pressure measurements and invasive hemodynamic data were not collected, limiting our determination of hypotension to a single value and prohibiting an analysis of continuous hemodynamic measurements. Third, unlike some studies in adults, we did not include urine output in our definition of CS. Fourth, we did not have information on socioeconomic status to ascertain what role, if any, it might play in patients' clinical acuity at presentation or subsequent outcomes. Fifth, our cohort included patients with readmissions, which appeared to artificially lower the observed mortality rates when compared with unique patient analysis. Finally, because we included patients admitted for ADHF based on the inpatient HF team census, we would have missed patients who may have presented in extremis with undifferentiated shock in the

FIGURE 4 90-Day In-Hospital Survival Estimates for Children Admitted in ADHF



Kaplan-Meier figure depicting estimates of 90-day in-hospital survival for individual children admitted in acute decompensated heart failure with and without cardiogenic shock, including only most recent hospitalization, censored at time of hospital discharge. Approximately one-half of CS deaths occurred within 2 weeks of admission. The shaded areas indicate the confidence intervals. Abbreviations as in Figures 1 and 2.

FIGURE 5 Hospital Mortality Stratified by CS Stage and Time Point of Classification



Bar chart depicting the hospital mortality for each stage of cardiogenic shock (CS) per the modified Society for Cardiovascular Angiography and Interventions criteria. Each hospitalization was assigned a CS severity based on parameters at admission and at 24 hours, and the highest severity stage attained in the hospitalization. There was an increase in the mortality rate with increasing shock severity at each time point of classification ($P < 0.001$).

emergency room or general pediatric intensive care unit and died shortly thereafter prior to CS being identified.

CONCLUSIONS

In a large, heterogeneous cohort of children hospitalized for ADHF, we found that a state of CS was commonly present at admission and that cardiomyopathy and myocarditis comprised the majority of etiologies of HF. Compared with children without CS, those with CS presented with worse systolic function, higher BNP concentration, and more frequent early end-organ injury. Resource utilization was high with nearly one-half of CS patients receiving MCS and nearly 9 in 10 receiving mechanical ventilation. The inpatient mortality rate for children with CS was also high, worse than what is described in children with other forms of shock, and increased with escalating shock stage. Future prospective studies are necessary to validate risk stratification criteria for children presenting in CS or compensated CS, compare safety and efficacy of short-term MCS systems, and test the value of rapid response multidisciplinary care teams in the initial triage of children presenting in CS.

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PERSPECTIVES

COMPETENCY IN PATIENT CARE AND PROCEDURAL SKILLS: CS in children hospitalized with ADHF is associated with a high risk of mortality. Short-term MCS can be a bridge to recovery and heart transplantation.

TRANSLATIONAL OUTLOOK: Risk stratification studies are needed to inform early use of rescue therapies to improve survival.

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KEY WORDS cardiogenic shock, children, heart failure, mortality, pediatrics

APPENDIX For supplemental tables and a figure, please see the online version of this paper.