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Sex-Related Differences in Patient Characteristics, Hemodynamics, and Outcomes of Cardiogenic Shock: INOVA-SHOCK Registry

Kelly C. Epps, MD, MSHP[a,*], **Behnam N. Tehrani, MD**[a], **Carolyn Rosner, NP-C**[a], **Pramita Bagchi, PhD**[b], **Annunziata Cotugno, MD**[c], **Abdulla A. Damluji, MD, PhD**[a], **Christopher deFilippi, MD**[a], **Shashank Desai, MD**[a], **Nasrien Ibrahim, MD**[a], **Mitchell Psotka, MD, PhD**[a], **Anika Raja, BS**[a], **Matthew W. Sherwood, MD, MHS**[a], **Ramesh Singh, MD**[a], **Shashank S. Sinha, MD, MSc**[a], **Daniel Tang, MD**[a], **Alexander G. Truesdell, MD**[a,d], **Christopher O'Connor, MD**[a], **Wayne Batchelor, MD, MHS**[a]

[a]Inova Heart and Vascular Institute, Falls Church, Virginia

[b]Department of Statistics, George Mason University, Fairfax, Virginia

[c]Department of Medical and Surgical Specialties, Radiological Sciences and Public Health, Institute of Cardiology, University of Brescia, Brescia, Italy

[d]Virginia Heart, Falls Church, Virginia

Abstract

Background: Little is known about sex-related differences in outcomes of patients with cardiogenic shock (CS) treated within a standardized team-based approach (STBA).

Methods: We evaluated 520 consecutive patients (151 women and 369 men) with CS due to acute myocardial infarction (AMI) and heart failure (HF) in a single-center registry (January 2017–December 2019) and examined outcomes according to sex and CS phenotype. The primary outcome was in-hospital mortality. Secondary outcomes included major adverse cardiac events, 30-day mortality, major bleeding, vascular complications, and stroke.

Results: Women with AMI-CS had higher baseline acuity (CardShock score: female [F]: 5.5 vs male [M]: 4.0; $P = .04$). Women with HF-CS more often presented with cardiac arrest (F: 12.4% vs M: 2.4%; $P < .01$) and had higher rates of vasopressor use (F: 70.8% vs M: 58.0%; $P = .04$)

*Corresponding author: kelly.epps@inova.org (K.C. Epps).

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Declaration of competing interest

Behnam Tehrani is a consultant to Medtronic and Abiomed. Alexander Truesdell is a consultant to and on the speakers bureau for Abiomed. Wayne Batchelor is a consultant to Medtronic, Abbott, Edwards LifeSciences, Boston Scientific, and Abiomed and receives research support from Boston Scientific and Abbott. All other authors report no financial interests.

Ethics Statement and Patient Consent

This study reports on a registry approved by the Inova Institutional Review Board.

Supplementary material

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and mechanical circulatory support (F: 46.1% vs M: 32.5%; $P = .04$). There were no sex-related differences in in-hospital mortality for AMI-CS (F: 45.2% vs M: 36.9%; $P = .28$) and HF-CS (F: 28.1% vs M: 24.5%; $P = .56$). Women with HF-CS experienced higher rates of major bleeding (F: 25.8% vs M: 13.7%; $P = .02$) and vascular complications (F: 15.7% vs M: 6.1%; $P = .01$). However, female sex was not an independent predictor of these complications. No sex differences in survival were noted at 1 year.

Conclusions: Within an STBA, although women with AMI-CS and HF-CS presented with higher acuity, they experienced similar in-hospital mortality, major adverse cardiac events, 30-day mortality, stroke, and 30-day readmissions as men. Further research is needed to better understand the extent to which historical differences in CS outcomes can be mitigated by an STBA.

Keywords

cardiogenic shock; mechanical circulatory support; multidisciplinary care; sex differences; shock team

Introduction

Despite advances in early reperfusion, regionalized systems of care, and mechanical circulatory support (MCS) devices, clinical outcomes in cardiogenic shock (CS) have been stagnant for nearly 2 decades, with mortality rates hovering at 50%.[1–4] Although both sexefs are afflicted by this lethal syndrome, women face higher in-hospital mortality,[5–8] major bleeding complications,[6,9] and higher 30-day readmission rates,[6,9–15] due at least in part to women presenting at an older age and with a greater burden of comorbidities.[5,6,16] However, women across the entire age spectrum presenting with acute myocardial infarction complicated by CS (AMI-CS) are also less likely to receive invasive diagnostic testing, percutaneous coronary intervention, and MCS, suggesting that treatment gaps may also contribute to disparate outcomes.[7,8,13,16] New treatment paradigms are emerging from single and multicenter registries suggesting that standardized protocols, predicated on early invasive hemodynamics, tailored MCS use, and multidisciplinary care in level 1 cardiac intensive care units, may improve outcomes in CS, irrespective of shock severity and phenotypes.[17–20] The primary objective of this study was to assess sex-related differences in patient characteristics, hemodynamics, and clinical outcomes for CS patients presenting with both AMI-CS and heart failure complicated by CS (HF-CS). We also aimed to evaluate the extent to which the uniform adoption of a standardized team-based approach (STBA) to CS impacted outcomes in women versus men within our CS program over 3 years.

Materials and Methods

Patients and setting

We evaluated 520 consecutive patients with a diagnosis of CS admitted to our institution from January 3, 2017, to December 31, 2019 (Figure 1). Clinical and hemodynamic criteria for CS diagnosis were based on the SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock) trial.[1] Clinical criteria included a systolic blood pressure <90 mm Hg for ≥30 minutes (or vasopressors to maintain systolic blood pressure ≥90 mm Hg) and evidence of end-organ hypoperfusion. Hemodynamic

criteria were Fick cardiac index ≤1.8 L/min/m^2 without vasopressors (or ≤2.2 L/min/m^2 with vasopressors) and a pulmonary capillary wedge pressure ≥15 mm Hg. Lactic acid levels were measured as surrogate markers of end-organ perfusion at baseline and 24 hours following the implementation of therapies. Patients with both AMI-CS and HF-CS were evaluated. The severity of illness was described using the validated CardShock score.[21] Higher scores indicate a higher risk of intensive care unit mortality, with a score >6 classified as "high-risk" (≥70% mortality). All patients were treated using a previously described standardized CS protocol[20] that emphasizes early diagnosis and treatment, multidisciplinary team-based care, and tailored pharmacologic and/or MCS use (Supplemental Figure S1). The study was approved by the Inova Health System institutional review board.

Outcome

The primary outcome was in-hospital mortality overall and within AMI-CS and HF-CS. Secondary outcomes included major adverse cardiac events (MACE), 30-day mortality, major bleeding, vascular access complications, stroke, length of stay, and 30-day readmission and were adjudicated by the CS research team. MACE was defined as a composite of death, stroke, and readmission at 30 days. Major bleeding was defined as a composite of the Bleeding Academic Research Consortium (BARC) type 3a, 3b, 3c, and type 5.[22] Vascular access complications were adjudicated as major vascular complications as described by the second Valve Academic Research Consortium (VARC-2).[23] The primary outcome and the associated aforementioned adverse events were analyzed following clinical and/or hemodynamic diagnosis of CS.

Statistical analysis

Data are presented as mean ± SD, median (quartiles [Q1, Q3]), or frequency and percent. Comparisons were made via *t* test, Wilcoxon rank-sum test, 1-way analysis of variance, χ^2, or Fisher exact tests, where appropriate. We built separate multivariable logistic regression models for each of 5 outcomes for all CS patients in the registry: (1) in-hospital mortality, (2) major bleeding, (3) vascular access complications, (4) stroke, and (5) MACE to evaluate the effect of sex-adjusted for age (years), vasopressor duration (hours), time to MCS (hours), AMI-CS phenotype, diabetes mellitus, baseline kidney function (estimated glomerular filtration rate), need for renal replacement therapy, right atrial pressure at 24 hours (threshold cut point 24 mm Hg), pulmonary artery pulsatility index (PAPi) at 24 hours (threshold cutpoint 1), cardiac power output (CPO) at 24 hours (threshold cutpoint 0.6 W), lactate at 24 hours (threshold cutpoint 3 mg/dL) and CardShock score (continuous). These variables were chosen based on historical literature demonstrating their relationship with outcomes in CS.[1,2]. Time to MCS and vasopressor duration were log-transformed before using in the model due to their skewed distributions. For patients missing time to MCS, right atrial, CPO, lactate, PAPi, and vasopressor duration, imputation was employed using fully conditional specifications based on the other covariates to impute missing values. In order to study the change in outcomes over time, we built separate multivariate logistic regression models with each of the 4 outcomes as response and year as an independent variable adjusting for sex and all other covariates used in the previous models. Years were treated as a categorical variable with 3 possible values 2017, 2018, and 2019. The missing values and non-normally

distributed variables were dealt with similarly. Odds ratios and 95% CIs are presented. Group comparisons were accomplished with fixed effects for both sexes (male [M] vs female [F]) and shock type (AMI vs HF). We also performed a 1-year unconditional survival analysis for both M and F. All analyses were performed using R version 4.0.2 software.

Results

Patient characteristics

A total of 520 patients presented to our institution with CS over a 36-month period (Table 1), with 57.2% of patients transferred from another hospital with no difference in transfer rate by sex (F: 60.9% vs M: 52.8%; $P = .09$). Mean age was 61 ± 13 years, 29% of patients were F, 43.6% had diabetes mellitus, 64% had baseline renal insufficiency, and 20% required dialysis. Seventy-three percent of patients required vasopressor support at the time of diagnosis (F: 75.5% vs M: 68.3%; $P = .14$), and 53.3% required MCS (F: 56.3% vs M: 52.0%; $P = .33$). Twenty-five percent of patients received upfront intra-aortic balloon pump support (F: 24.5% vs M: 25.7%; $P = 1.00$) with 40.2% requiring escalation of MCS (F: 48.9% vs M: 38.9%; $P = .33$). In this cohort, 89.2% (n = 464) of all patients underwent baseline right heart catheterization (RHC).

Among AMI-CS patients, women were of similar age to men (67 vs 65 years, respectively; $P = .15$). Women with AMI-CS were more likely to be transferred from another hospital (F: 80.6% vs M: 60.5%; $P < .01$) and had higher hemo-metabolic acuity (index CardShock score, F: 5.5 vs M: 4.0; $P = .04$) and lower CPO at 24 hours following implementation of therapies (F: 0.7 W vs M: 0.9 W; $P = .02$). Among HF-CS patients, women more often presented with out-of-hospital cardiac arrest (F: 12.4% vs M: 2.4%; $P < .01$) and required higher rates of vasopressor use (F: 70.8% vs M: 58.0%; $P = 0.04$) and MCS (F: 46.1% vs M: 32.5%; $P = .04$). Women presented with lower rates of baseline renal insufficiency than men (glomerular filtration rate <60 mL/min, F: 53.9% vs M: 73.1%; $P < .01$). There were no sex-related differences in serial lactate levels or baseline hemodynamic parameters by RHC. However, at 24 hours, women with AMI-CS had lower CPO compared to men (F: 0.7 W vs M: 0.9 W; $P = .02$), and women with HF-CS had lower PAPi (F: 1.9 vs M: 2.3; $P = .05$) but higher cardiac index (F: 2.4 L/min/m^2 vs M: 2.1 L/min/m^2; $P = .02$) compared to men.

Clinical course and outcomes

In-hospital mortality was similar between women and men with AMI-CS (F: 45.2% vs 36.9%; $P = .28$) and HF-CS (F: 28.1% vs 24.5%; $P = .56$). Similarly, no sex-related differences were noted in 30-day mortality, stroke, length of stay, or 30-day readmission in patients with AMI-CS or HF-CS (Table 2). Men with HF-CS were more likely to need renal replacement therapy (F: 18.9% vs M: 13.5%; $P = .04$). Women with HF-CS, on the other hand, experienced higher rates of major bleeding (F: 25.8% vs M: 13.7%; $P = .02$) and vascular access complications (F: 15.7% vs M: 6.1%; $P = .01$), differences that were not seen in the AMI-CS population. There were also no differences in nonaccess site bleeding (gastrointestinal hemorrhage, hemothorax, and genitourinary bleeding) between men and women with HF-CS. In multivariate logistic regression models, F sex was not an

independent predictor of in-hospital mortality, bleeding, vascular complications, stroke, or MACE (Fig. 2).

Survival to discharge improved for the entire cohort over 3 years (2017: 57.9% vs 2018: 74.2% vs 2019: 71.5%, $P < .01$) (Figure 3). Although the absolute improvements in survival to discharge were similar between men and women, the improvement was only statistically significant for men (survival in men 2017: 59.4% vs 2018: 75.0% vs 2019: 73.3%, $P = .03$; survival in women 2017: 54.6% vs 2018: 72.7% vs 2019: 67.2%, $P = .19$, and particularly men with HF-CS. This may have been in part because of the larger proportion of M patients in our registry. One-year survival was similar between women and men, both within the entire CS cohort and within each CS phenotype (Figure 4).

Discussion

This prespecified subgroup analysis of the INOVA-SHOCK registry provides insights into sex-based differences in patient characteristics and outcomes for patients with CS managed with an STBA. We note the following key findings: (1) women with all phenotypes of CS presented with higher baseline clinical and hemo-metabolic acuity of illness; (2) women with HF-CS were more likely to be supported with temporizing MCS and more prone to major bleeding and vascular complications; (3) despite these baseline differences in clinical characteristics and bleeding risks, within an STBA to CS, we observed no sex-related differences in in-hospital mortality, stroke, length of stay, and 30-day readmission and 1-year survival (Central Illustration).

Women comprised 29.0% (n = 151) of this registry, with similar sex distribution within AMI-CS (28.3% F; n = 62) and HF-CS (29.6% F; n = 89) phenotypes. This is consistent with data reported in the SHOCK,[1] IABP-SHOCK II (Intra-aortic Balloon Pump in Cardiogenic Shock II),[12] Catheter Based Ventricular Assist Device (cVAD),[9] and PROTECT (Placebo-controlled Randomized Study of Selective A1 Adenosine Receptor Antagonist Rolofylline for Patients Hospitalized with Acute Decompensated Heart Failure and Volume Overload to Assess Treatment Effect on Congestion and Renal FuncTion) studies.[24] As a registry of all-comer CS patients, we believe that our study population is more representative of real-world clinical practice, thereby addressing concerns about F underrepresentation and other selection biases noted in prior CS studies.[24,25]

In our registry, women with both AMI-CS and HF-CS presented with higher risk baseline clinical and hemodynamic characteristics than men. Women with AM-CS had higher CardShock Scores and lower CPO at 24 hours, parameters that are strongly associated with short-term mortality.[21,26] In addition, women with HF-CS more often presented with cardiac arrest and had higher vasopressor and MCS utilization rates, suggesting a more critical presentation than men. These findings are consistent with established literature demonstrating worse clinical profiles in women and are likely because of an increased burden of comorbidities, prolonged duration of symptom-onset to first medical contact, and system delays in disease recognition and initiation of care[5,6,9,16,24,25,27,28] Despite this, women in our study experienced similar rates of mortality, stroke, and rehospitalization compared to men. Although unproven, we hypothesize that this may be because of

implementing an STBA to CS, which appeared to afford a similar prognostic benefit, independent of sex, across our comprehensive CS program.

In our registry, 89.2% (n = 464) of all patients underwent baseline RHC. Strom et al[29] queried the National Inpatient Sample to assess interhospital variations in clinical practice patterns for AMI-CS and noted that RHC was performed to inform clinical decision making only 5.9% of the time in the highest quartile MCS utilizing institutions. This may represent a significant missed opportunity in real-world practice to improve outcomes in CS patients, as recent data from the CS Working Group revealed that using pulmonary artery catheter-derived hemodynamics improves survival in all stages of CS.[30] We believe that our findings highlight the potential merits of a protocolized, time-sensitive approach to CS recognition and treatment, a strategy that may mitigate previously observed sex gaps in this critically ill and vulnerable patient population.[4]

Notably, MCS was utilized in 53.3% of all patients treated in our registry, with higher utilization in women with HF-CS compared to men (46.1% vs 32.5%; $P = .04$). This is noteworthy, particularly given previous reports demonstrating lower or equivalent rates of MCS use in women with HF-CS, despite a more critical INTERMACS profile.[25,31] We believe that the higher use of MCS in women with HF-CS in our study was protocol driven since our standardized treatment algorithm emphasizes early RHC to identify and treat patients with severely compromised hemodynamics. These findings highlight the importance of minimizing practice variation through the implementation of an STBA to CS care. However, the higher rates of MCS utilization in women with HF-CS may have also contributed to the increased risk of major bleeding and vascular complications observed in this cohort, a finding consistent with previous reports.[6,9,32,33] It is interesting that we did not detect significant sex-based differences in MCS utilization in our AMI-CS cohort, nor did we find differences in bleeding in this setting. These findings are in contrast to data from the National Inpatient Sample, which showed that in real-world practice, women with AMI-CS were less likely to receive short-term MCS than men.[7,8] We believe that employing standardized best practices with ultrasound guidance for vascular access and radial arterial access for coronary angiography in AMI-CS patients may have helped lower bleeding and vascular complications in the AMI-CS women in our registry.[34–37] It has also been proposed that early identification and remediation of CS may halt the progression to end-stage shock, a condition characterized by irreversible renal and hepatic impairment and enhanced risk for bleeding.[38]

Our experience suggests but does not prove that implementing an STBA to CS management may be an effective strategy for reducing sex-based disparities in CS outcomes. Despite higher bleeding rates in women with HF-CS, we observed no sex-based differences in shortterm mortality, stroke, length of stay, or 30-day readmission. This is an important observation in light of historical data demonstrating higher in-hospital mortality and 30-day readmission rates in women with CS.[6–8,13–15] We believe that by reducing heterogeneity of care, an algorithm-driven STBA to CS care reduces the potential for sex-based bias in treatment and disparities in outcomes. Although there is precedent for implementing health system strategies and protocols to minimize treatment disparities between women and men with acute coronary syndromes,[39] our study is the first to suggest that a multidisciplinary

STBA to CS may mitigate the difference in short and long-term outcomes of men and women presenting with CS, regardless of baseline clinical and hemo-metabolic risk. Given the paucity of data regarding intermediate and long-term outcomes in CS, further research is needed in the form of multicenter registries and pragmatic clinical trial designs to better understand the potential lasting benefits of standardized care for CS in women and men afflicted with this highly lethal syndrome.

Limitations

There are several limitations to this study. First, as a single-center registry, findings may be influenced by center-specific patient characteristics and practice patterns. However, we believe implementing an STBA with regionalized care coordination for all-comers with CS reduces selection bias, variations in care, and potential concerns about the generalizability of our findings. Second, women represented only one-third of the patients in our registry. Although we found no statistically significant differences in MACE, mortality, stroke, or 30-day readmissions between the sexes, we cannot definitely rule out subtle yet important differences in clinical characteristics and outcomes, thus raising the possibility for type II errors.[40] Third, since this study is observational in nature, causality cannot be assumed, and therefore, we cannot assert definitively that the implementation of an STBA was the sole reason for comparable outcomes between women and men. Larger scale multicenter registries and adequately powered randomized control trials are needed to further understand which therapies and strategies improve outcomes of CS in both women and men.

Conclusion

In this single-center CS registry utilizing an STBA approach to care, women presented with higher acuity than men. However, we found no sex-related differences in in-hospital mortality, stroke, length of stay, 30-day readmissions, and 1-year survival. Further research is warranted to better understand the extent to which standardized multidisciplinary and hemodynamically tailored care may reduce existing sex-based disparities in CS outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations:

AMI	acute myocardial infarction
CS	cardiogenic shock
HF	heart failure
MCS	mechanical circulatory support
STBA	standardized team-based approach

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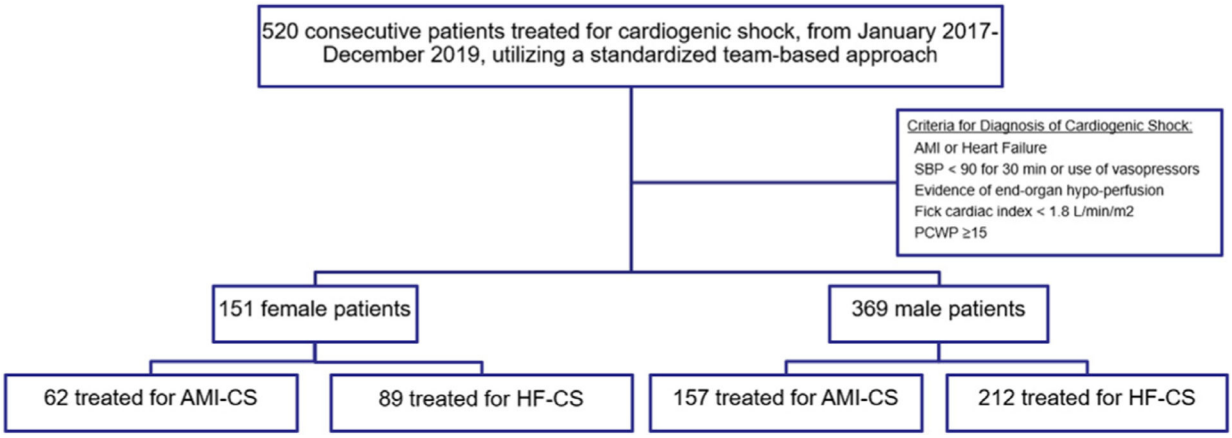


Figure 1. CONSORT flow diagram and study design.

ADHF, acute decompensated heart failure; AMI, acute myocardial infarction; CS, cardiogenic shock; PCWP, pulmonary capillary wedge pressure; SBP, systolic blood pressure.

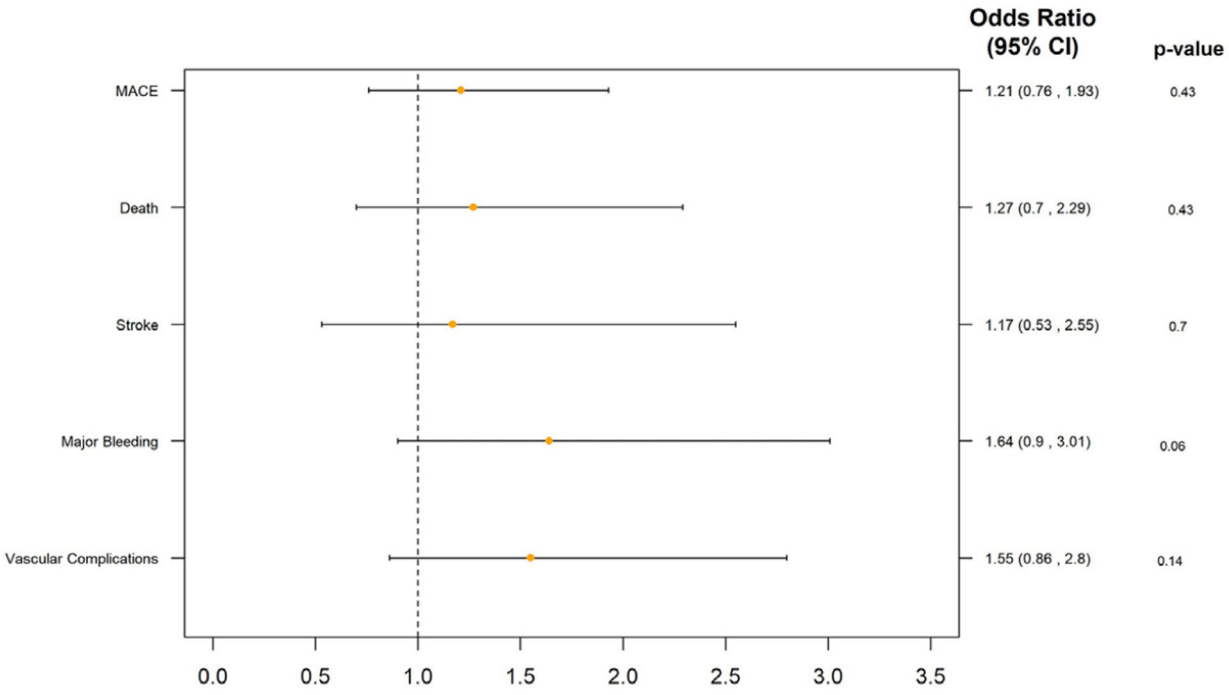


Figure 2.
Sex-related risk-adjusted 30-day outcomes.

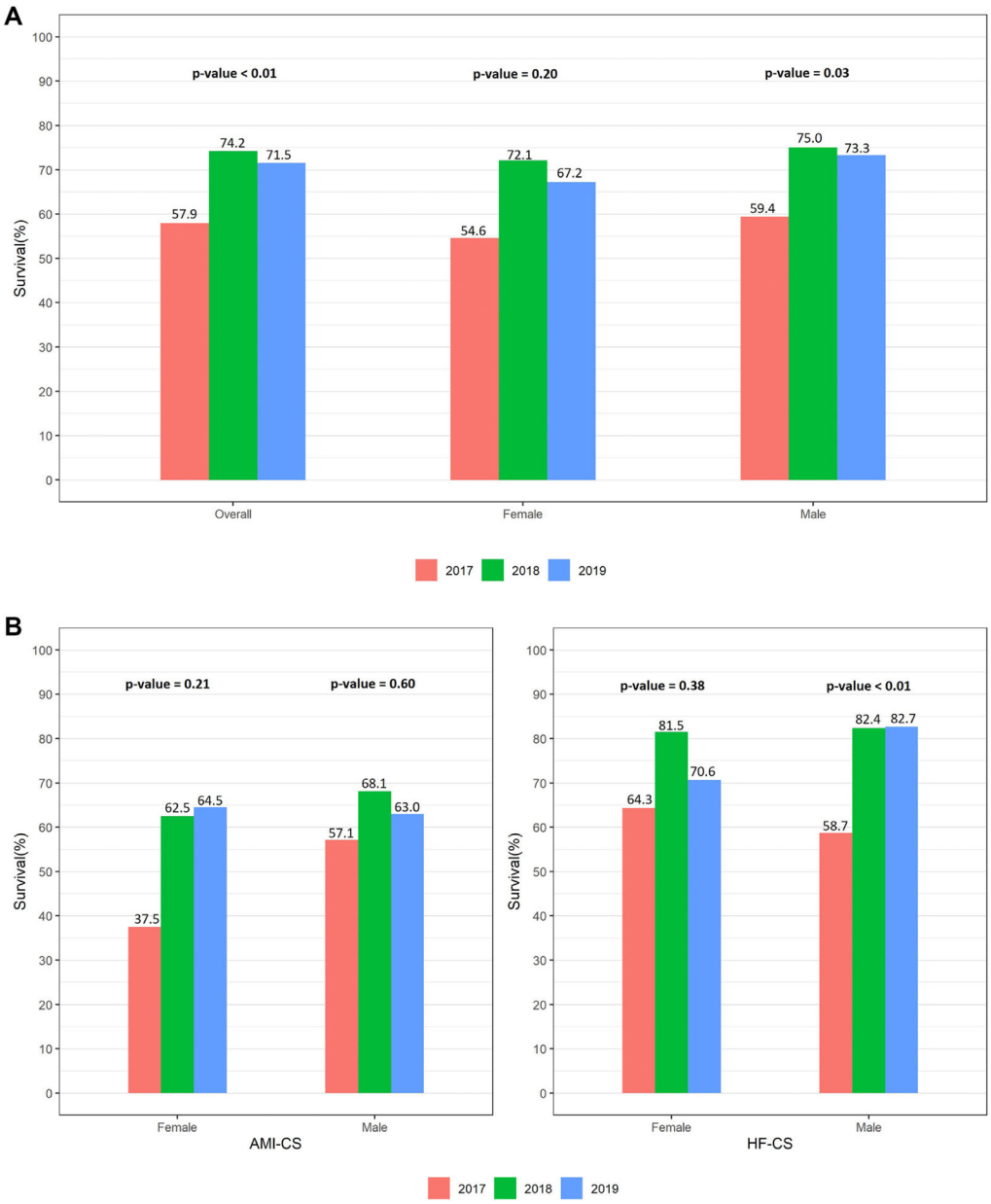


Figure 3.
Survival to discharge.

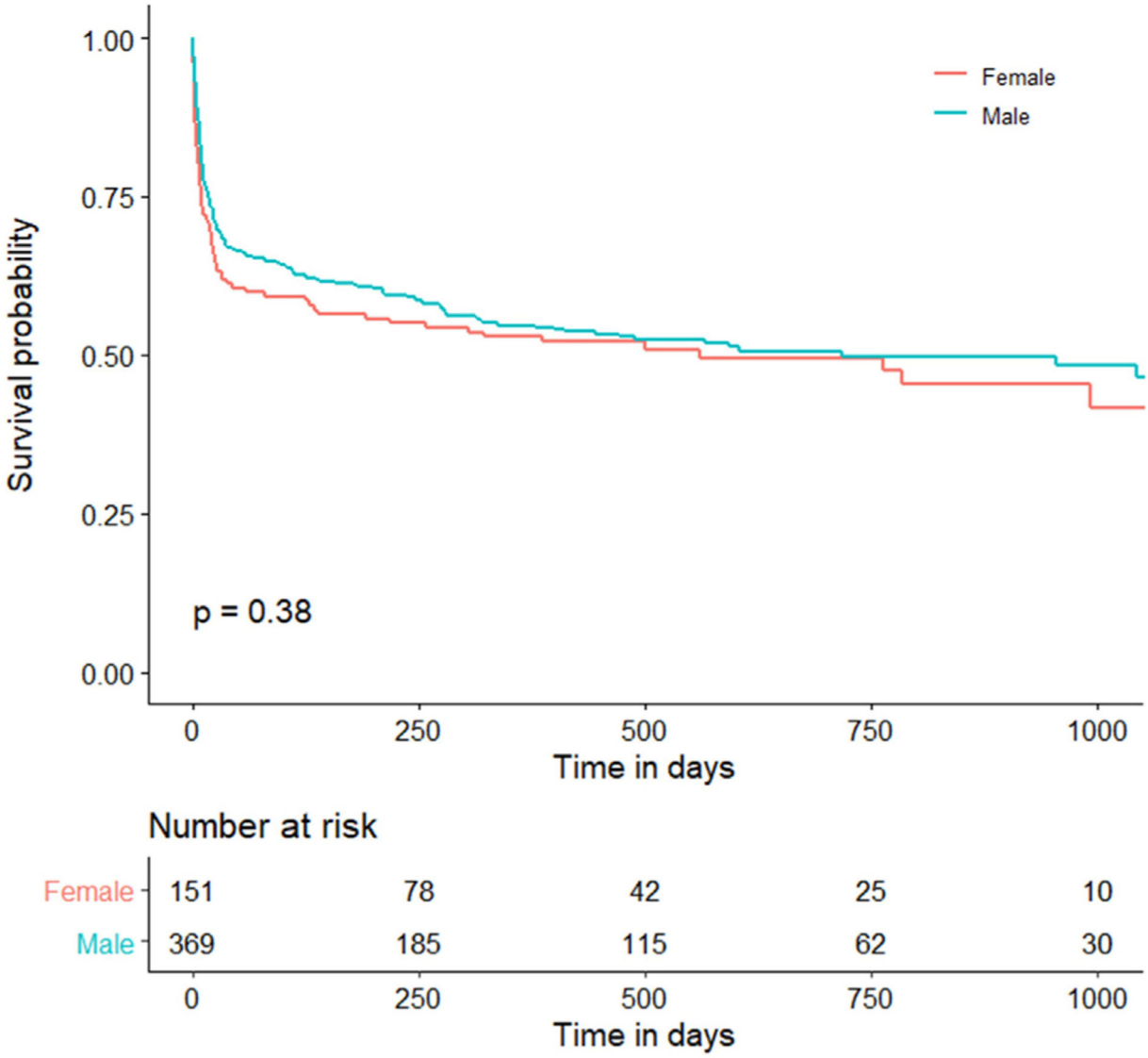
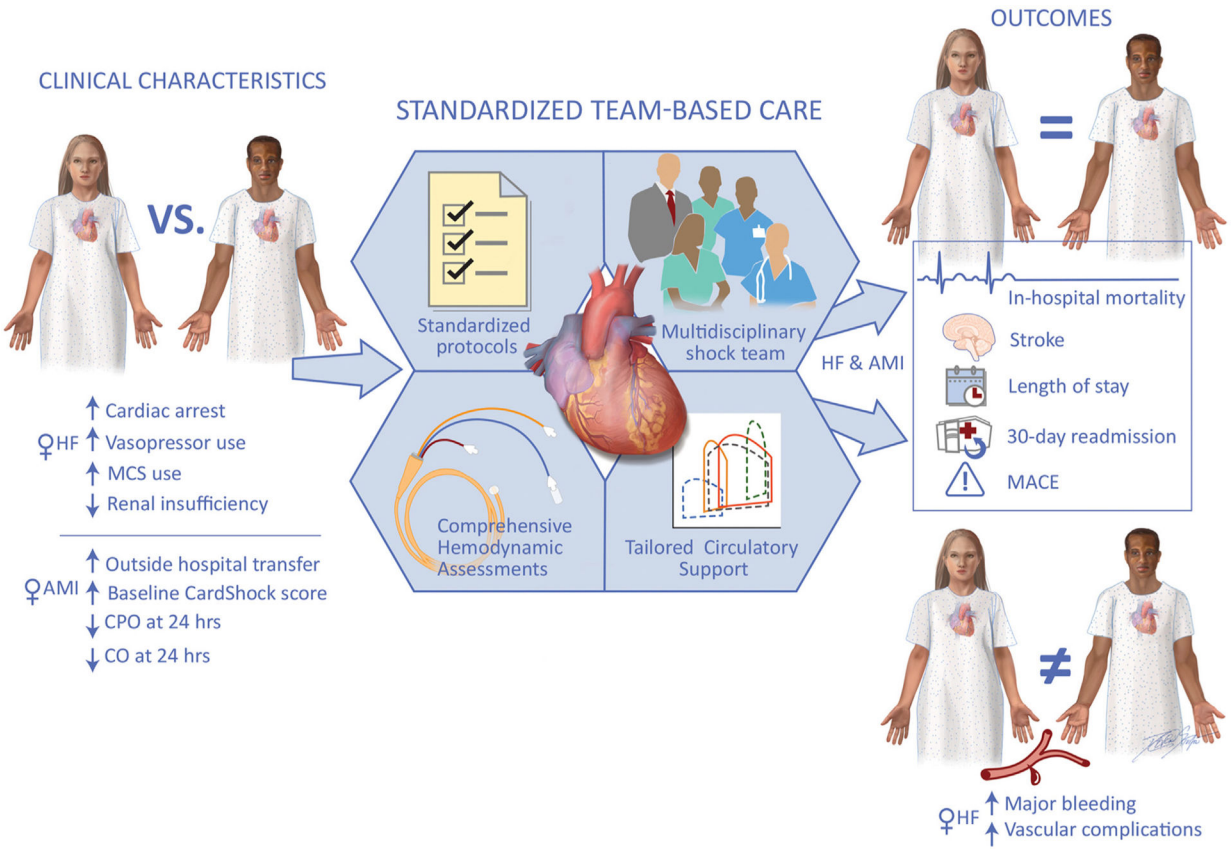


Figure 4.
One-year cumulative survival for patients hospitalized for cardiogenic shock.



Central Illustration.

Schematic diagram demonstrating salient sex-related differences in baseline clinical characteristics and short-term clinical outcomes following implementation of standardized team-based approach to the management of cardiogenic shock. AMI, acute myocardial infarction; CS, cardiogenic shock; HF, heart failure; CO, cardiac output; CPO, cardiac power output; MACE, major adverse cardiac events.

Table 1.

Baseline clinical and hemodynamic characteristics (n = 520).

Parameter	AMI-CS N=219			HF-CS N=301			Overall *P* value
	Female n = 62	Male n = 157	*P* value	Female n = 89	Male n = 212	*P* value	
Age, y	67.1 ± 12.3	64.9 ± 9.9	.15	56.3 ± 17.1	59.4 ± 13.3	.26	.90
Body mass index	27.9 (25.4, 31.3)	27.9 (24.4, 32.0)	.87	29.1 (23.4, 34.4)	27.25 (24.28, 32.03)	.42	.45
Race			.95			.98	.96
White	35 (56.5)	88 (56.1)		46 (51.7)	106 (50.0)		
Black/African American	6 (9.7)	12 (7.7)		28 (31.5)	68 (32.1)		
Asian	14 (22.6)	39 (24.8)		8 (9.0)	22 (10.4)		
Other	7 (11.3)	18 (11.5)		7 (7.9)	16 (7.5)		
Hispanic	6 (9.7)	13 (8.3)	.79	6 (6.7)	10 (4.7)	.57	.56
Diabetes mellitus	36 (58.1)	71 (45.2)	.10	32 (36.0)	88 (41.5)	.44	.70
Index GFR <60 mL/min	35 (56.5)	95 (60.5)	.58	48 (53.9)	155 (73.1)	<.01	<.01
Cerebrovascular disease	7 (11.3)	22 (14.0)	.66	16 (18.0)	38 (18.0)	1.00	.90
Prior MI/PCI/CABG/valve surgery	12 (19.4)	36 (22.9)	.72	26 (29.2)	81 (38.2)	.15	.17
Outside transfer	50 (80.6)	95 (60.5)	<.01	42 (47.2)	100 (47.2)	1.00	.10
Vasopressors at index diagnosis	51 (82.3)	129 (82.2)	1.00	63 (70.8)	123 (58.0)	.04	.11
Vasopressors at 24 h	29 (46.8)	79 (50.3)	.66	40 (44.9)	64 (30.2)	.02	.17
Intervention	47 (75.8)	130 (82.8)	.32	-	-		
PCI	37 (78.7)	115 (88.5)	.16	-	-		
CABG	10 (21.3)	15 (11.5)		-	-		
Out-of-hospital arrest	4 (6.5)	22 (14.0)	.16	11 (12.4)	5 (2.4)	<.01	.38
Shockable rhythm	4 (100)	15 (68.2)	.55	5 (45.5)	4 (80)	.31	.52
In-hospital arrest	14 (22.6)	44 (28.0)	.50	13 (14.6)	21 (9.9)	.24	1.00
Shockable rhythm	5 (35.7)	19 (43.2)	.76	8 (61.5)	6 (28.6)	.08	.61
Left ventricular ejection fraction, %	0.3 (0.2, 0.5)	0.3 (0.2, 0.4)	.20	0.3 (0.2, 0.4)	0.2 (0.1, 0.3)	.75	.21
Right heart catheterization	56 (90.3)	144 (91.7)	.79	75 (84.3)	189 (89.6)	.24	.21
Baseline hemodynamics							
Cardiac output, L/min	3.5 (3.1, 4.1)	3.8 (3.1, 4.8)	.18	3.2 (2.4, 4)	3.3 (2.7, 4.1)	.42	.23

Parameter	AMI-CS N=219			HF-CS N=301			Overall *P* value
	Female n = 62	Male n = 157	*P* value	Female n = 89	Male n = 212	*P* value	
Cardiac index, L/min/m^2	2.0 (1.7, 2.3)	1.9 (1.6, 2.4)	.57	1.8 (1.3, 2.2)	1.6 (1.3, 2)	.17	.10
CPO, W	0.7 (0.6, 0.8)	0.7 (0.5, 0.9)	.10	0.6 (0.4, 0.7)	0.6 (0.5, 0.8)	.23	.08
PAPi	1.1 (0.7, 1.7)	1.3 (0.8, 2.0)	.50	1.2 (0.9, 2.3)	1.6 (1.1, 2.4)	.19	.12
RA, mm Hg	13 (10, 17.5)	14 (10,18)	.80	15 (11,20)	15 (11,20)	.89	.99
PCWP, mm Hg	20 (15, 25)	23 (16, 28)	.07	22 (18, 30)	25 (20, 30)	.06	.01
Mean PA, mm Hg	25.5 (20.3, 32)	29 (23, 36.5)	.06	32.5 (27.5, 39.5)	35.7 (29, 41)	.17	.03
24-h hemodynamics							
Cardiac ouput, L/min	4.3 (3.5, 5.3)	4.9 (4.0, 6.4)	.03	4.5 (3.7, 5.6)	4.3 (3.7, 5.5)	.73	.24
Cardiac index, L/min/m^2	2.3 (2.0, 2.9)	2.4 (2.0, 3.1)	.48	2.4 (2.0, 2.9)	2.1 (1.8, 2.6)	.02	.20
CPO, W	0.7 (0.6, 0.9)	0.9 (0.7, 1.1)	.02	0.7 (0.6, 0.9)	0.8 (0.6, 0.9)	.70	.07
PAPi	1.6 (1.0, 2.5)	1.6 (1.0, 2.8)	.22	1.9 (1.3, 2.8)	2.3 (1.5, 3.8)	.05	.10
RA, mm Hg	11 (8, 15)	11.5 (8, 15)	.96	10 (7.3, 16)	10 (7, 14)	.42	.52
PCWP, mm Hg	18 (16, 23)	18 (14.5, 21.5)	.45	20 (16, 25)	21 (17, 26)	.40	1.00
Mean PA, mm Hg	25 (21.8, 29.5)	25 (21,29.6)	.86	28 (22, 35)	30 (24, 35.3)	.18	.41
Lactate, mg/dL at baseline	2.3 (1.6, 5.4)	2.7 (1.8, 5.1)	.69	2.4 (1.5, 4.6)	2.2 (1.3, 4.0)	.21	.63
Lactate, mg/dL at 24 h	1.6 (1,2.3)	1.9 (1.2, 3.5)	.17	1.7 (1.2, 3.0)	2.0 (1.4, 3.7)	.10	.04
Index CardShock score	5.5 (4, 7)	4 (3, 7)	.04	4 (3, 6)	4 (3, 6)	.74	.31
MCS	44 (71.0)	123 (78.3)	.29	41 (46.1)	69 (32.5)	.04	.39
IABP	26 (41.9)	72 (45.9)	.65	11 (12.4)	23 (10.8)	.69	.83
Escalation from IABP	11 (42.3)	29 (40.2)	1.00	5 (45.5)	8 (34.8)	.71	.70
pVAD only	21 (33.9)	58 (36.9)	.76	16 (18.0)	27 (12.7)	.28	.73
VA-ECMO only	3 (4.8)	12 (7.6)	.56	8 (9.0)	11 (5.2)	.30	.70
pVAD + VA-ECMO	7 (11.3)	26 (16.6)	.40	9 (10.1)	13 (6.1)	.23	1.00
Renal replacement therapy	10 (16.1)	43 (27.4)	.08	12 (13.5)	40 (18.9)	.32	.04

Values presented are mean ± SD, frequency (percent), or median (quartiles [Q1, Q3]) where appropriate.

AMI, acute myocardial infarction; BMI, body mass index; CABG, coronary artery bypass grafting; CPO, cardiac power output; CS, cardiogenic shock; GFR, glomerular filtration rate; HF, heart failure; IABP, intra-aortic balloon pump; MCS, mechanical circulatory support; MI, myocardial infarction; PAPi, pulmonary arterial pulsatility index; PCI, percutaneous coronary intervention; PCWP, pulmonary capillary wedge pressure; pVAD, percutaneous ventricular assist device; RA, right atrial; PA, pulmonary artery; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.

Table 2.

Clinical outcomes.

Parameter	AMI-CS N = 219			HF-CS N = 301			Overall *P* value
	Female n = 62	Male n = 157	*P* value	Female n = 89	Male n = 212	*P* value	
MACE	41 (66.1)	86 (54.8)	.13	44 (49.4)	102 (48.1)	.80	.25
In-hospital mortality	28 (45.2)	58 (36.9)	.28	25 (28.1)	52 (24.5)	.56	.25
30-day mortality	32 (51.6)	65 (41.4)	.48	30 (33.7)	67 (31.6)	.81	.57
Length of stay, d	9 (3, 15)	9 (5, 19)	.14	13 (7, 22)	13 (7, 25)	.73	.25
Access site bleeding	2 (3.2)	11 (7.0)	.27	6 (6.7)	8 (3.8)	.30	.97
Major bleeding[a]	16 (25.8)	41 (26.1)	1.00	23 (25.8)	29 (13.7)	.02	.10
Vascular complications[b]	11 (17.7)	29 (18.5)	1.00	14 (15.7)	13 (6.1)	.01	.11
Stroke	7 (11.3)	16 (10.2)	.81	6 (6.7)	13 (6.1)	.80	.86
Persistent shock (CPO <0.6 at 24 h)[c]	8 (25.8)	12 (11.2)	.08	19 (32.8)	40 (25.0)	.30	.04
Readmission within 30-d[d]	10 (16.4)	16 (10.3)	.32	17 (19.1)	39 (18.8)	1.00	.51
LVAD or transplant	1 (1.6)	5 (3.2)	1.00	9 (10.1)	30 (14.2)	.45	.39

Values are expressed as n (%) or median (IQR).

AMI, acute myocardial infarction; CPO, cardiac power output; CS, cardiogenic shock; HF, heart failure; LVAD, left ventricular assist device; MACE, major adverse cardiac events.

[a]Major bleeding = Bleeding Academic Research Consortium (BARC) type 3a, 3b, 3c, and type 5..

[b]Vascular complications = Vascular Academic Research Consortium.

[c]Nonmissing (denominator) for CPO 24 h: HF-CS: female 58, male 160; AMI-CS: female: 31, male: 107.

[d]Nonmissing (denominator) for Readmission 30 days: HF-CS: female: 89, male: 207; AMI-CS: female: 61, male: 155.