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Echocardiographic Correlates of Hyponatremia in Critically Ill Patients With Heart Failure

Anan Younis, MD[1,2], Meir Tabi, MD[3,4], Kianoush B. Kashani, MD, MS[5], Garima Dahiya, MBBS[5], Maan Jokhadar, MD[5], Dustin B. Hillerson, MD[5], Ruben Crespo-Diaz, MD, PhD[5], and Jacob C. Jentzer, MD, MS, FACC, FAHA[5]

[1]Leviev Heart Center, Sheba Medical Center, Ramat Gan, Israel
[2]Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel
[3]Heart Institute, H'aEmek Medical Center, Afula, Israel
[4]Faculty of Medicine, Technion Israel Institute of Technology, Haifa, Israel
[5]Department of Cardiovascular Medicine, Mayo Clinic, Rochester, MN, USA

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Correspondence to
Jacob C. Jentzer, MD, MS, FACC, FAHA
Department of Cardiovascular Medicine, The Mayo Clinic, 200 First Street SW, Rochester, MN 55905, USA.
Email: jentzer.jacob@mayo.edu

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ABSTRACT

Background and Objectives: Few data have examined cardiac function correlates of hyponatremia in heart failure (HF) patients. We examined the association between hyponatremia and echocardiographic parameters with outcomes among HF patients in the cardiac intensive care unit (CICU).

Methods: Retrospective analysis of 3,372 Mayo Clinic CICU patients with HF from 2007 to 2018 grouped according to admission serum sodium: severe hyponatremia (sodium <130 mEq/L, 6%), mild hyponatremia (sodium 130–134 mEq/L, 16%), normal (sodium 135–144 mEq/L, 78%). Echocardiographic findings and mortality were compared across groups.

Results: The median age was 71.8 years old, and 39% were females. Patients with hyponatremia had worse echocardiographic parameters reflecting left ventricular (LV) systolic function and forward flow, particularly stroke volume index and LV systolic work index, with higher right atrial pressure and worse right ventricular-pulmonary artery coupling. In-hospital mortality occurred in 12.4% and increased with more severe hyponatremia: ≥135 mEq/L, 10.8%; 130–134 mEq/L, 16.7%; <130 mEq/L, 21.6% (p<0.001). One-year mortality occurred in 32.1% and was higher in patients with hyponatremia: ≥135 mEq/L, 29.6%; 130–134 mEq/L, 40.7%; <130 mEq/L, 40.7% (p<0.001). Patients with hyponatremia had significantly higher in-hospital (adjusted odds ratio, 1.80; 95% confidence interval [CI], 1.44–2.25; p<0.001) and one-year (adjusted hazard ratio, 1.51; 95% CI, 1.32–1.72; p<0.001) mortality, both overall and when stratified by echocardiographic measures. Additive increases in mortality were seen in patients with hyponatremia and poor echocardiographic hemodynamics.

Conclusions: CICU patients with HF and hyponatremia have worse hemodynamics reflected by echocardiographic parameters of LV systolic and diastolic function and forward flow, plus worse right ventricular function, with resultant worse clinical outcomes.

Keywords: Hyponatremia; Echocardiography; Heart failure; Mortality; Sodium

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INTRODUCTION

Abnormal sodium levels, particularly hyponatremia, have been associated with increased risk of mortality and morbidity among patients hospitalized with acute cardiovascular disease.[1-3] Notably, hyponatremia has consistently been shown to be associated with a significant increase in mortality among patients hospitalized with acute and chronic heart failure (HF).[4-6] Dysnatremia, including both hyponatremia and hypernatremia, is associated with adverse outcomes among patients hospitalized in the intensive care unit.[7,8] Recently, we showed that hyponatremia is independently associated with increased all-cause death among patients hospitalized in the cardiac intensive care unit (CICU).[9] Among the many potential causes of hyponatremia, non-osmotic vasopressin release due to low cardiac output and congestion have been proposed as potential etiologies in HF patients.[10]

Transthoracic echocardiography (TTE) is the standard noninvasive modality used to evaluate critically ill patients with suspected cardiac disease.[11,12] Bedside TTE performed at admission to CICU can provide valuable information regarding biventricular function, hemodynamics, and volume status to guide decision-making, prognostication, and management.[11-13] Yet, there are few if any data regarding the TTE abnormalities that might be associated with hyponatremia in hospitalized HF patients. Specifically, no studies have examined the association of sodium levels with TTE measures of cardiac function and non-invasive hemodynamics among HF patients in the CICU. In this study, we sought to investigate the echocardiographic characteristics and survival outcomes associated with hyponatremia in HF patients admitted to the CICU. We hypothesized that hyponatremia would be an indicator of poor cardiac function and impaired hemodynamics.

METHODS

Study population

This study was approved by the Institutional Review Board of Mayo Clinic as minimal risk. It was conducted under a waiver of informed consent for patients who had provided Minnesota Research Authorization. We analyzed data from the index admission of consecutive unique adult (aged ≥18 years) patients admitted to the CICU at Mayo Clinic Hospital St. Mary's Campus who were admitted from January 1, 2007, to April 30, 2018, with an admission diagnosis of HF (including all forms) and a TTE performed within one day of CICU admission.[14] Admission sodium level was defined as the sodium value closest to CICU admission, and we excluded patients who either did not have an admission sodium value or who had hypernatremia (defined as a sodium ≥145 mEq/L). Patients were grouped based on admission sodium level as follows: normal sodium (admission sodium, 135–144 mEq/L); mild hyponatremia (admission sodium, 130–134 mEq/L); severe hyponatremia (admission sodium, <130 mEq/L).

Data sources

Demographic, vital signs, laboratory, clinical, and outcome data were extracted electronically from the medical record using the Multidisciplinary Epidemiology and Translational Research in Intensive Care DataMart. Admission diagnoses were defined as all International Classification of Diseases (ICD)-9 and ICD-10 diagnostic codes recorded on the day of CICU admission and the day before or after CICU admission; these admission diagnoses were not mutually exclusive, and the primary admission diagnosis could not be determined.[15] The Acute Physiology and Chronic Health Evaluation-III score, and Day 1 Sequential Organ Failure Assessment (SOFA) score were calculated automatically using data from the first 24 hours of CICU admission, with missing variables imputed as normal as the default.[16] In-hospital mortality, including CICU mortality, was determined using electronic chart review. All-cause mortality within one year of CICU admission (including in-hospital deaths) was ascertained using electronic chart review supplemented by State of Minnesota and Mayo Clinic databases.

Echocardiographic parameters

Data were extracted from the Mayo Clinic Echocardiography Database, using the TTE performed within one day of CICU admission, including vital signs at the time of TTE (**Supplementary Table 1**). One best left ventricular ejection fraction (LVEF) value for each patient was determined using a guideline-based hierarchical approach as per Mayo Echocardiography laboratory protocol: volumetric LVEF calculated using Simpson's biplane method was preferred, followed by monoplane volumetric approach, followed by linear methods and finally by visual estimation if these other methods were unavailable. LVEF was used to grade the severity of left ventricular systolic dysfunction (LVSD), as per current American Society for Echocardiography guidelines.[17] Right ventricular systolic dysfunction (RVSD) was graded using guideline-based multiparametric analysis including the visual estimate by the interpreting physician.[18] Patients were classified based on the presence of moderate or greater LVSD and/or RVSD as having no/mild ventricular dysfunction, isolated LVSD, isolated RVSD, or biventricular dysfunction.

Right atrial pressure was either recorded at the time of TTE (if measured invasively) or estimated by TTE based on inferior vena cava size and collapsibility.[18] The left ventricular outflow tract (LVOT) velocity-time integral (VTI) was used to estimate stroke volume (SV) and cardiac output (CO). The ratio of mitral valve E velocity to medial mitral annulus e′ velocity (E/e′ ratio) was

used to estimate left ventricular (LV) end-diastolic pressure for the calculation of left ventricular stroke work index (LVSWI) and to estimate pulmonary capillary wedge pressure (PCWP) via the Nagueh formula.[15] We calculated several novel hemodynamic parameters reflecting cardiac function that have been validated in the CICU population (**Supplementary Table 2**), including the myocardial contraction fraction (MCF), cardiac power output (CPO), tricuspid annular s′ velocity (TASV)/right ventricular systolic pressure (RVSP) ratio (reflecting right ventricular [RV]-PA coupling), and pulmonary arterial (PA) elastance.[19] Congestion was defined using estimated right atrial pressure (RAP) ≥14 to define RV congestion and estimated PCWP ≥24 to define LV congestion.

Statistical methods

Data are reported as median (interquartile range) for continuous variables and numbers (%) for categorical variables. Groups were compared using the Kruskal-Wallis rank-sum test for continuous variables and the Pearson χ^2 test for categorical variables. Correlations between admission sodium and TTE parameters of interest were evaluated using both Pearson r and Spearman rho correlation coefficients and pairwise adjusted p values. Logistic regression was used to calculate odds ratio (OR) and 95% confidence interval (CI) values for predictors of in-hospital mortality, before and after multivariable adjustment. Univariable logistic regression was used to evaluate the associations between TTE variables and hyponatremia <135 mEq/L, including assessment of discrimination using area under the receiver-operator characteristic curve (AUC) values. Kaplan-Meier curves were used to evaluate one-year survival, with groups compared using the log-rank test. Cox proportional-hazards regression was used to calculate hazard ratio (HR) and 95% CI values for predictors of one-year mortality, before and after multivariable adjustment. Multivariable logistic regression and Cox models evaluating mortality were adjusted for age, sex, race, Charlson comorbidity index, Day 1 SOFA, number of vasoactives, admission diagnoses of shock/cardiac arrest/respiratory failure, and use of dialysis and cardiac catheterization. Statistical analyses were performed using BlueSky Pro version 10.3 (BlueSky LLC, Chicago, IL, USA). No extramural funding was used to support this research.

RESULTS

Study population baseline and clinical characteristics

Out of a database of 12,428 unique CICU patients, 6,008 had an admission diagnosis of acute or chronic HF. Among these, 3,598 had a TTE within one day of CICU admission. We excluded 67 patients without available admission sodium data and 159 with hypernatremia, yielding 3,372 patients in the final study population. The median age was 71.8 (61.8–81.0) years old and 39.4% were females (**Table 1**). Concomitant admission diagnoses included acute coronary syndrome (ACS; 47.0%), shock (27.4%), cardiac arrest (15.9%), and respiratory failure (41.1%). Vasoactive drugs were used in 35.3%, invasive ventilation in 25.4%, and 7.2% of patients required hemodialysis (**Table 1**). The median serum sodium was 138 (135–140) mEq/L. Patients were grouped according to admission sodium as follows: severe hyponatremia <130 mEq/L, 199 (5.9%) patients; mild hyponatremia 130–134 mEq/L, 558 (16.5%) patients; and normal 135–144 mEq/L, 2,615 (77.5%) patients. These groups differed substantially in terms of age, length of stay, severity of illness, and interventions performed in CICU (**Table 1**).

TTE parameters according to admission sodium

TTE was performed on the day of CICU admission in 1,874 (55.6%) patients (**Table 2**). The prevalence of severe left LVSD and moderate/severe RVSD were higher among patients with severe hyponatremia compared to those with either normal or mildly reduced sodium levels (**Supplementary Figure 1**). Most TTE parameters reflecting LV systolic function were significantly and incrementally worse among patients with lower sodium levels (**Table 2**), including LVEF and measures of forward flow (stroke volume index [SVI], CO, MCF, and LVSWI). Moreover, patients with lower sodium levels had worse LV diastolic function and higher filling pressures, as reflected by a higher E/e′ ratio. RV dysfunction was more common in those with lower sodium, and these patients had worse markers of RV-PA coupling (e.g., TASV/RVSP ratio and PA elastance) (**Table 2**).

Sodium correlation with TTE parameters

Overall, the various TTE parameters did not correlate strongly with sodium levels, with correlation coefficients below |0.2| for all TTE variables. However, there were modest but significant correlations between several TTE parameters and sodium levels (**Supplementary Table 3**). The majority of these were TTE parameters reflecting LV systolic function and forward flow, which were directly correlated with sodium levels, and markers of RV congestion, which were inversely correlated (**Supplementary Table 3**). The strongest correlations were noted for LVSWI (r=0.15), Pressure adjusted HR (r=−0.16), SV/SVI (r=0.12), RAP (r=−0.13), LV wall motion score index (r=−0.13) and PA elastance (r=−0.13). TTE parameters reflecting diastolic function were not significantly correlated with sodium levels, and LVEF correlated modestly (r=0.08). Similar results were observed for Pearson and Spearman correlations, as well as for patients (n=2,300) who were documented to be in sinus rhythm at the time of TTE. While several TTE parameters had significant univariable associations with hyponatremia <135 mEq/L (**Table 3**), only pressure-adjusted

Table 1. Baseline characteristics and outcomes of study population, according to sodium subgroups

Variable	Patients groups*				
	<130 MEq/L (n=199)	130–134 MEq/L (n=558)	≥135 MEq/L (n=2,615)	Total (n=3,372)	p value
Demographics					
Age (years)	66.9 (56.9–76.3)	70.6 (61.7–80.4)	72.5 (62.0–81.2)	71.8 (61.6–81.0)	<0.001
Female	82 (41.2%)	232 (41.6%)	1,013 (38.7%)	1,327 (39.4%)	0.395
White race	174 (87.4%)	511 (91.6%)	2,417 (92.4%)	3,102 (92.0%)	0.041
Comorbidities					
Charlson comorbidity index	2.0 (1.0–4.0)	2.0 (1.0–4.0)	2.0 (1.0–4.0)	2.0 (1.0–4.0)	0.319
Myocardial infarction	43 (21.6%)	98 (17.6%)	575 (22.0%)	716 (21.3%)	0.070
Heart failure	65 (32.7%)	184 (33.1%)	783 (30.0%)	1,032 (30.7%)	0.290
Diabetes mellitus	61 (30.7%)	210 (37.8%)	895 (34.3%)	1,166 (34.6%)	0.139
Lung disease	49 (24.6%)	120 (21.6%)	576 (22.1%)	745 (22.1%)	0.663
Cancer	38 (19.1%)	135 (24.3%)	576 (22.1%)	749 (22.3%)	0.283
Chronic kidney disease	50 (25.1%)	140 (25.2%)	671 (25.7%)	861 (25.6%)	0.957
Haemodialysis	17 (8.5%)	46 (8.2%)	167 (6.4%)	230 (6.8%)	0.175
Admission diagnoses					
Acute coronary syndrome	59 (29.6%)	237 (42.5%)	1,290 (49.3%)	1,586 (47.0%)	<0.001
Respiratory failure	82 (41.2%)	230 (41.2%)	1,073 (41.0%)	1,385 (41.1%)	0.996
Shock (any)	67 (33.7%)	183 (32.8%)	675 (25.8%)	925 (27.4%)	<0.001
Cardiogenic shock	62 (31.2%)	164 (29.4%)	572 (21.9%)	798 (23.7%)	<0.001
Cardiac arrest	26 (13.1%)	86 (15.4%)	425 (16.3%)	537 (15.9%)	0.464
Sepsis	26 (13.1%)	58 (10.4%)	264 (10.1%)	348 (10.3%)	0.414
Admission laboratory values					
Creatinine (mg/dL)	1.2 (0.9–2.3)	1.3 (0.9–2.0)	1.2 (0.9–1.7)	1.2 (0.9–1.7)	<0.001
BUN (mg/dL)	33.0 (18.0–58.0)	29.0 (20.0–47.0)	24.0 (17.0–37.0)	25.0 (17.0–40.0)	<0.001
Bicarbonate (mEq/L)	23.0 (19.0–26.0)	23.0 (20.0–25.0)	24.0 (21.0–26.0)	23.0 (21.0–26.0)	<0.001
Anion gap (mEq/L)	14.0 (10.0–17.0)	13.0 (11.0–16.0)	13.0 (11.0–15.0)	13.0 (11.0–16.0)	0.130
Sodium (mEq/L)	127.0 (124.0–128.0)	133.0 (132.0–134.0)	139.0 (137.0–141.0)	138.0 (135.0–140.0)	<0.001
Chloride (mEq/L)	90.0 (87.0–93.0)	97.0 (94.0–100.0)	102.0 (100.0–105.0)	101.0 (98.0–105.0)	<0.001
Potassium (mEq/L)	4.4 (3.9–5.0)	4.4 (3.9–5.0)	4.3 (3.9–4.7)	4.3 (3.9–4.7)	<0.001
Severity of illness					
Apache 3 score	68.0 (56.0–85.0)	71.0 (58.0–85.0)	64.0 (51.0–80.0)	66.0 (53.0–81.0)	<0.001
Braden score	17.0 (15.0–19.0)	17.0 (14.0–19.0)	17.0 (15.0–20.0)	17.0 (15.0–20.0)	0.049
Day 1 SOFA score	5.0 (3.0–8.0)	4.0 (2.0–7.0)	4.0 (2.0–6.0)	4.0 (2.0–7.0)	<0.001
M-CARS	3.0 (2.0–5.0)	3.0 (2.0–5.0)	3.0 (1.0–5.0)	3.0 (1.0–5.0)	<0.001
Therapies & procedures in CICU					
Vasoactive drug use	104 (52.3%)	230 (41.2%)	856 (32.7%)	1,190 (35.3%)	<0.001
Vasopressor drug use	90 (45.2%)	204 (36.6%)	758 (29.0%)	1,052 (31.2%)	<0.001
Ionotropic drug use	58 (29.1%)	99 (17.7%)	289 (11.1%)	446 (13.2%)	<0.001
Coronary angiography	80 (40.2%)	179 (32.1%)	812 (31.1%)	1,071 (31.8%)	<0.001
Percutaneous coronary intervention	27 (13.6%)	120 (21.5%)	762 (29.1%)	909 (27.0%)	<0.001
Non-invasive ventilation	63 (31.7%)	128 (22.9%)	640 (24.5%)	831 (24.6%)	0.045
Invasive ventilation	43 (21.6%)	140 (25.1%)	672 (25.7%)	855 (25.4%)	0.436
Hemodialysis	33 (16.6%)	56 (10.0%)	155 (5.9%)	244 (7.2%)	<0.001
IABP	31 (15.6%)	87 (15.6%)	344 (13.2%)	462 (13.7%)	0.230
PAC	45 (22.6%)	96 (17.2%)	361 (13.8%)	502 (14.9%)	<0.001
Outcomes					
CICU length of stay (days)	3.1 (1.8–5.0)	2.7 (1.4–4.5)	2.0 (1.1–3.7)	2.2 (1.2–3.9)	<0.001
Hospital length of stay (days)	7.8 (4.6–16.0)	7.8 (4.5–12.8)	6.2 (3.8–10.8)	6.7 (3.9–11.4)	<0.001
CICU mortality	23 (11.6%)	56 (10.0%)	169 (6.5%)	248 (7.4%)	<0.001
Hospital mortality	43 (21.6%)	93 (16.7%)	283 (10.8%)	419 (12.4%)	<0.001

Data displayed as numbers (%) for categorical variables and median (interquartile range). The p value is for comparison of sodium subgroups, using Kruskal-Wallis rank-sum test for continuous variables and Pearson χ^2 test for categorical variables.
BUN = blood urea nitrogen; SOFA = Sequential Organ Failure Assessment; M-CARS = Mayo Clinic CICU Admission Risk Score; CICU = cardiac intensive care unit; IABP = intra-aortic balloon pump; PAC = pulmonary artery catheter.
*Patients were grouped based on admission sodium level as follows: normal sodium (admission sodium, 135–144 mEq/L); mild hyponatremia (admission sodium, 130–134 mEq/L); severe hyponatremia (admission sodium, <130 mEq/L).

heart rate (0.603) and left ventricular stroke work (0.601) had AUC values >0.6 reflecting good discrimination. Among clinical and laboratory values, only chloride (0.832) and bilirubin (0.603) had AUC values >0.6 (data not shown).

Table 2. Echocardiographic findings by admission sodium group

Variables	Patients groups*				p value
	<130 MEq/L (n=199)	130–134 MEq/L (n=558)	≥135 MEq/L (n=2,615)	Total (n=3,372)	
Vital signs					
SBP at time of TTE (mmHg)	106.50 (96.00–123.00)	109.00 (97.00–125.00)	113.00 (100.00–129.00)	112.00 (100.00–128.00)	<0.001
DBP at time of TTE (mmHg)	62.00 (53.00–72.00)	62.00 (53.00–70.00)	63.00 (54.00–73.00)	63.00 (54.00–73.00)	0.023
Heart rate (b/min)	82.50 (71.75–95.00)	80.00 (70.00–98.00)	78.00 (67.00–92.00)	79.00 (67.00–93.00)	<0.001
MAP (mmHg)	77.17 (68.17–86.00)	77.33 (69.67–87.33)	80.00 (71.00–90.67)	79.67 (70.67–90.00)	<0.001
SI score	0.78 (0.62–0.96)	0.75 (0.61–0.91)	0.69 (0.55–0.85)	0.70 (0.57–0.87)	<0.001
PP (mmHg)	45.50 (34.75–57.00)	47.00 (36.00–59.00)	49.00 (37.00–64.00)	48.00 (37.00–62.00)	0.003
LV systolic function parameters					
LVEDD (mm)	55.50 (48.00–64.00)	53.00 (47.00–60.00)	54.00 (48.00–59.00)	54.00 (48.00–60.00)	0.022
LVESD (mm)	45.00 (33.50–57.00)	41.00 (32.00–50.00)	41.00 (33.00–50.00)	41.00 (33.00–50.00)	0.040
Fractional shortening (%)	17.65 (11.29–31.82)	20.58 (13.43–30.68)	21.82 (15.00–31.70)	21.31 (14.55–31.58)	0.008
LVEF (%)	33.50 (20.00–52.00)	35.00 (25.00–54.00)	37.00 (26.00–54.00)	37.00 (25.00–54.00)	0.003
MCF	0.27 (0.20–0.40)	0.30 (0.22–0.41)	0.33 (0.25–0.42)	0.32 (0.24–0.42)	<0.001
LVOT VTI (cm)	16.00 (12.00–20.00)	17.00 (13.70–20.00)	18.00 (14.80–21.00)	17.40 (14.00–21.00)	<0.001
LV stroke volume (mL)	58.00 (44.00–80.00)	63.00 (49.25–80.00)	69.00 (56.00–85.00)	68.00 (54.00–83.00)	<0.001
LV stroke volume index (ml/m^2)	29.50 (22.00–41.00)	33.00 (26.00–40.00)	36.00 (29.00–44.00)	35.00 (28.00–43.00)	<0.001
LV cardiac output (L/min)	4.86 (3.79–5.89)	4.98 (3.99–6.36)	5.25 (4.28–6.32)	5.16 (4.19–6.31)	<0.001
LV cardiac index (L/min/m^2)	2.51 (1.93–3.01)	2.60 (2.12–3.24)	2.69 (2.23–3.21)	2.66 (2.20–3.20)	<0.001
LV WMSI	2.31 (2.00–2.56)	2.13 (1.75–2.50)	2.06 (1.69–2.38)	2.06 (1.69–2.44)	<0.001
CPO (W)	0.84 (0.63–1.00)	0.86 (0.66–1.14)	0.94 (0.74–1.17)	0.92 (0.72–1.16)	<0.001
CPI (W/m^2)	0.43 (0.32–0.52)	0.45 (0.35–0.58)	0.48 (0.39–0.60)	0.47 (0.38–0.59)	<0.001
LV stroke work index (g·min/m^2)	24.98 (17.76–33.91)	27.68 (20.95–37.72)	31.62 (23.99–39.90)	30.51 (23.04–39.38)	<0.001
API	1.92 (1.49–3.09)	2.28 (1.48–3.14)	2.45 (1.70–3.42)	2.40 (1.65–3.36)	<0.001
Diastolic function parameters					
MV E wave peak velocity (cm/s)	0.90 (0.70–1.20)	0.90 (0.70–1.12)	0.90 (0.60–1.10)	0.90 (0.70–1.10)	0.003
MV E/A ratio	1.57 (0.87–2.33)	1.17 (0.80–1.80)	1.11 (0.75–1.60)	1.13 (0.75–1.67)	<0.001
MV medial e′ peak velocity (cm/s)	5.00 (4.00–6.00)	5.00 (4.00–6.00)	5.00 (4.00–6.00)	5.00 (4.00–6.00)	0.672
E/e′ ratio	18.61 (13.33–26.50)	17.50 (13.33–25.00)	16.67 (12.00–24.00)	17.14 (12.00–24.00)	0.003
Right ventricular function parameters					
Peak TR velocity (m/s)	2.90 (2.50–3.20)	2.90 (2.50–3.20)	2.85 (2.50–3.24)	2.88 (2.50–3.22)	0.915
Estimated RAP (mmHg)	14.00 (10.00–20.00)	14.00 (10.00–15.75)	10.00 (5.00–15.00)	10.00 (5.00–15.00)	<0.001
Estimated RV systolic pressure (mmHg)	46.00 (39.00–56.00)	46.00 (37.00–56.00)	45.00 (35.00–55.00)	45.00 (36.00–56.00)	0.085
TASV (cm/s)	9.00 (7.00–12.00)	10.00 (8.00–13.00)	11.00 (8.00–13.00)	10.00 (8.00–13.00)	0.003
TASV/RVSP ratio	0.19 (0.15–0.27)	0.22 (0.15–0.30)	0.23 (0.16–0.32)	0.22 (0.16–0.32)	<0.001
PA elastance (mmHg/mL)	0.75 (0.57–1.13)	0.72 (0.52–0.98)	0.65 (0.47–0.88)	0.67 (0.49–0.92)	<0.001
Congestion phenotype					<0.001
Biventricular	43 (37.1%)	89 (26.7%)	296 (20.1%)	428 (22.3%)	
Predominant LV	23 (19.8%)	75 (22.5%)	391 (26.5%)	489 (25.4%)	
Predominant RV	26 (22.4%)	81 (24.3%)	250 (17.0%)	357 (18.6%)	

Data are reported as median (interquartile range) for continuous variables, with groups compared using the Kruskal-Wallis rank-sum test.
SBP = systolic blood pressure; TTE = transthoracic echocardiography; DBP = diastolic blood pressure; MAP = mean arterial pressure; SI = shock index; PP = pulse pressure; LV = left ventricular; LVEDD = left ventricular end-diastolic dimension; LVESD = left ventricular end-systolic dimension; LVEF = left ventricular ejection fraction; MCF = myocardial contraction fraction; LVOT = left ventricular outflow tract; VTI = velocity-time integral; WMSI = wall motion score index; CPO = cardiac power output; CPI = cardiac power index; API = aortic pulsatility index; MV = mitral valve; E/e′ ratio = ratio of mitral valve E velocity to medial mitral annulus e′ velocity; TR = tricuspid regurgitation; RAP = right atrial pressure; RV = right ventricular; TASV = tricuspid annular s′ velocity; RVSP = right ventricular systolic pressure; PA = pulmonary arterial.
*Patients were grouped based on admission sodium level as follows: normal sodium (admission sodium, 135–144 mEq/L); mild hyponatremia (admission sodium, 130–134 mEq/L); severe hyponatremia (admission sodium, <130 mEq/L).

In-hospital mortality

A total of 419 (12.4%) patients died during hospitalization, including 248 (7.4%) deaths in the CICU. In-hospital mortality increased incrementally with lower sodium: ≥135 mEq/L (n=2,615), 10.8%; 130–134 mEq/L (n=558), 16.7%; <130 mEq/L (n=199), 21.6% (p<0.001). In-hospital mortality was higher with decreasing sodium levels (adjusted OR, 0.95 per 1 mEq/L higher sodium; 95% CI, 0.93–0.98; p<0.001). In-hospital mortality was higher in patients with mild (adjusted OR, 1.58; 95% CI, 1.17–2.13; p=0.002) or severe (adjusted OR, 2.29; 95% CI, 1.48–3.49; p<0.001) hyponatremia, including in patients with and without a diagnosis of cardiogenic shock (**Figure 1**). There was a gradient of increased mortality with worsening hyponatremia across groups divided by the severity of LV systolic dysfunction

Table 3. Echocardiographic predictors of hyponatremia (sodium <135 mEq/L)

Variable	Unit OR	Lower 95% CI	Upper 95% CI	No. of patients	AUC	p value
Vital signs at TTE						
Systolic BP	0.9904	0.9865	0.9942	3,205	0.5590	<1e-04
Diastolic BP	0.9917	0.9859	0.9974	3,200	0.5334	0.0045
Mean BP	0.9873	0.9815	0.9930	3,200	0.5528	<1e-04
Pulse pressure	0.9925	0.9883	0.9968	3,200	0.5393	0.0007
Heart rate	1.0086	1.0044	1.0128	3,114	0.5568	<1e-04
Shock index	1.6400	1.2624	2.1958	3,079	0.5775	0.0005
LV structure and systolic function						
LVEDD	1.0047	0.9958	1.0137	2,838	0.5033	0.2996
LVESD	1.0080	1.0002	1.0158	2,364	0.5172	0.0437
Septum	0.9590	0.9209	0.9978	2,362	0.5377	0.0409
Posterior wall	0.9663	0.9218	1.0121	2,350	0.5346	0.1499
RWT	0.6902	0.3012	1.5454	2,326	0.5343	0.3737
LV mass index	0.9986	0.9961	1.0011	2,306	0.5142	0.2812
Fractional shortening	0.9898	0.9811	0.9986	2,361	0.5402	0.0241
LVEF	0.9933	0.9884	0.9983	3,152	0.5367	0.0085
LV WMSI	1.7853	1.4087	2.2707	2,013	0.5759	<1e-04
Medial s′	0.9615	0.9085	1.0162	1,803	0.5335	0.1696
Lateral s′	0.9563	0.9036	1.0103	1,491	0.5350	0.1165
MCF	0.4097	0.1964	0.8391	2,156	0.5511	0.0160
LV diastolic function						
Mitral E velocity	1.7186	1.2832	2.2997	2,255	0.5472	0.0003
Mitral E/A ratio	1.2492	1.0937	1.4258	1,615	0.5620	0.0010
Medial e′	0.9935	0.9478	1.0401	2,243	0.5125	0.7832
Lateral e′	0.9842	0.9444	1.0246	1,695	0.5181	0.4446
Medial E/e′	1.0162	1.0064	1.0260	2,086	0.5499	0.0011
Lateral E/e′	1.0177	1.0030	1.0324	1,593	0.5513	0.0167
Mitral DT	0.9955	0.9933	0.9976	1,895	0.5850	<1e-04
Left heart hemodynamics						
LVOT peak velocity	0.6369	0.4138	0.9739	2,714	0.5308	0.0388
LVOT VTI	0.9537	0.9366	0.9709	2,712	0.5684	<1e-04
Cardiac output	0.9270	0.8771	0.9783	2,645	0.5501	0.0066
Cardiac index	0.8505	0.7588	0.9498	2,623	0.5450	0.0048
SV	0.9876	0.9834	0.9917	2,683	0.5866	<1e-04
SVI	0.9753	0.9669	0.9835	2,655	0.5864	<1e-04
CPO	0.5686	0.4324	0.7423	2,605	0.5661	<1e-04
CPI	0.2989	0.1700	0.5178	2,588	0.5667	<1e-04
LVSW	0.9862	0.9816	0.9906	1,962	0.6013	<1e-04
LVSWI	0.9734	0.9643	0.9823	1,949	0.5971	<1e-04
SVR	0.9941	0.9780	1.0088	2,380	0.5102	0.4583
SVRI	0.9955	0.9864	1.0035	2,366	0.5110	0.3044
API	0.8561	0.7886	0.9269	2,081	0.5616	0.0002
Right heart hemodynamics						
TR peak velocity	0.9604	0.8138	1.1316	2,515	0.5032	0.6311
RAP	1.0547	1.0378	1.0719	2,733	0.5808	< 1e-04
RVSP	1.0052	0.9994	1.0110	2,475	0.5281	0.0753
TASV	0.9584	0.9292	0.9879	1,985	0.5505	0.0064
TAPSE	0.9640	0.9299	0.9984	721	0.5493	0.0433
TASV/TR velocity	0.8912	0.8176	0.9694	1,692	0.5520	0.0080
TASV/RVSP	0.1705	0.0611	0.4603	1,671	0.5577	0.0006
TAPSE/RVSP	0.2590	0.0833	0.7513	640	0.5464	0.0159
PA elastance	1.7988	1.4347	2.2559	2,209	0.5760	<1e-04
PAHR	1.0459	1.0337	1.0583	2,582	0.6029	<1e-04

OR = odds ratio; CI = confidence interval; AUC = area under the receiver-operator characteristic curve; BP = blood pressure; LV = left ventricular; LVEDD = left ventricular end-diastolic dimension; LVESD = left ventricular end-systolic diameter; RWT = relative wall thickness; LVEF = left ventricular ejection fraction; WMSI = wall motion score index; MCF = myocardial contraction fraction; DT = deceleration time; LVOT = left ventricular outflow tract; VTI = velocity-time integral; SV = stroke volume; SVI = stroke volume index; CPO = cardiac power output; CPI = cardiac power index; LVSW = left ventricular stroke work; LVSWI = left ventricular stroke work index; SVR = systemic vascular resistance; SVRI = systemic vascular resistance index; API = aortic pulsatility index; TR = tricuspid regurgitation; RAP = right atrial pressure; RVSP = right ventricular systolic pressure; TASV = tricuspid annular s′ velocity; TAPSE = tricuspid annulus plane systolic excursion; TASV = tricuspid annular s′ velocity; PA = pulmonary arterial; PAHR = pressure-adjusted heart rate.

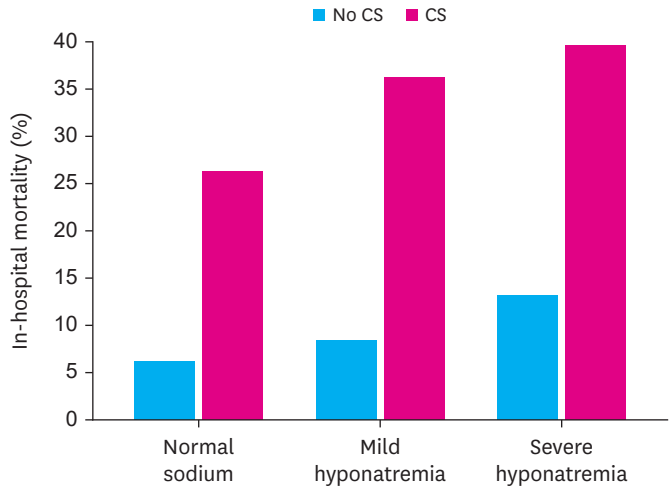

Figure 1. In-hospital mortality according to admission sodium group in patients with and without a concomitant diagnosis of CS. The p value for comparison of in-hospital mortality across sodium groups was 0.006 without CS and 0.009 with CS.
CS = cardiogenic shock.

(**Figure 2A**) or RV systolic dysfunction (**Figure 2B**). Mortality varied according to serum sodium and degree of biventricular dysfunction (**Figure 2C**) and biventricular congestion (**Figure 2D**), generally being worse with lower sodium. Likewise, mortality varied according to serum sodium and established hemodynamic predictors, including medial mitral E/e′ ratio (**Figure 3A**), severity of PH (**Figure 3B**), SVI (**Figure 3C**), LVSWI (**Figure 3D**), MCF (**Supplementary Figure 2A**), and TASV/RVSP ratio (**Supplementary Figure 2B**). Mortality risk was generally additive for patients with poor hemodynamics and hyponatremia. Adjustment or stratification for these selected TTE variables did not substantially affect the association between hyponatremia and mortality. Indeed, stratification by medial mitral E/e′ ratio showed an even stronger association between hyponatremia and mortality in patients with high E/e′ ratio >20 (adjusted OR, 2.81 vs. 1.77 with hyponatremia <135 mEq/L for E/e′ >20 vs. <20, respectively).

One-year survival

A total of 1,083 (32.1%) patients died by one year, including in-hospital deaths. Among hospital survivors (n=2,953), 664 (22.5%) died by one year and 325 (11.0%) had follow-up duration less than one year but were alive at last follow-up. One-year mortality was higher in patients with hyponatremia: ≥135 mEq/L, 29.6%; 130–134 mEq/L, 40.7%; <130 mEq/L, 40.7% (p<0.001). Patients with hyponatremia (sodium <135 mEq/L) had higher one-year mortality (adjusted HR, 1.40; 95% CI, 1.22–1.60; p<0.001; **Figure 4A**), but there was not an incremental increase with severe hyponatremia versus mild hyponatremia (p=0.84 between groups; **Figure 4B**). This effect was similar in patients with and without LVSD, although severe hyponatremia appeared to confer an incrementally higher risk in patients without LVSD (**Supplementary Figure 3**). A gradient of mortality was observed when patients were stratified by LVEF or medial mitral E/e′ and hyponatremia (**Supplementary Figure 4**).

DISCUSSION

This novel analysis examined the cardiac function and noninvasive echocardiographic hemodynamic correlates of hyponatremia in a large cohort of critically-ill HF patients admitted to CICU. We found that HF patients with hyponatremia had worse noninvasive hemodynamic parameters by echocardiography including biventricular dysfunction, lower forward flow, higher ventricular filling pressures, and worse RV-PA coupling. Patients with hyponatremia had higher mortality during and after hospitalization, with incremental prognostication between the sodium level and TTE findings. Overall, our analysis suggests that poor hemodynamics are associated with hyponatremia in CICU patients with HF, but the associations between hyponatremia and adverse outcomes extend beyond what can be quantified by TTE abnormalities, highlighting the need to better understand hyponatremia in HF.

To our knowledge, this is the first study to investigate echocardiographic hemodynamic parameters among HF patients hospitalized in the CICU based on their sodium levels. Overall, sodium levels correlated modestly with traditional and novel echocardiographic markers of cardiac structure and function. This implies that although poor hemodynamics are associated with hyponatremia in critically ill patients with HF, other factors not quantified by TTE are likely to be more important correlates. Likewise, the common clinical and laboratory variables captured in this data set did not predict hyponatremia well, highlighting the importance of other factors in predicting hyponatremia in critically ill HF patients. Accordingly, while impaired hemodynamics should be suspected in hospitalized HF patients with hyponatremia, the sodium level is neither sensitive nor specific for any particular TTE abnormality. Furthermore, outcomes are worse for patients with hyponatremia even when accounting for TTE abnormalities in an additive manner.

Hyponatremia is common among patients with various cardiac diseases, and it has been shown to be associated with an increased risk of reinfarction at 30-days among patients with ACS.[10,20] As in our analysis, hyponatremia has been significantly associated with mortality among patients with acute and chronic HF.[6] In a previous study, we showed that hyponatremia but not hypernatremia is associated with increased in-hospital and post-discharge mortality among unselected patients hospitalized in the CICU after adjustment for established risk markers.[9] This suggested that

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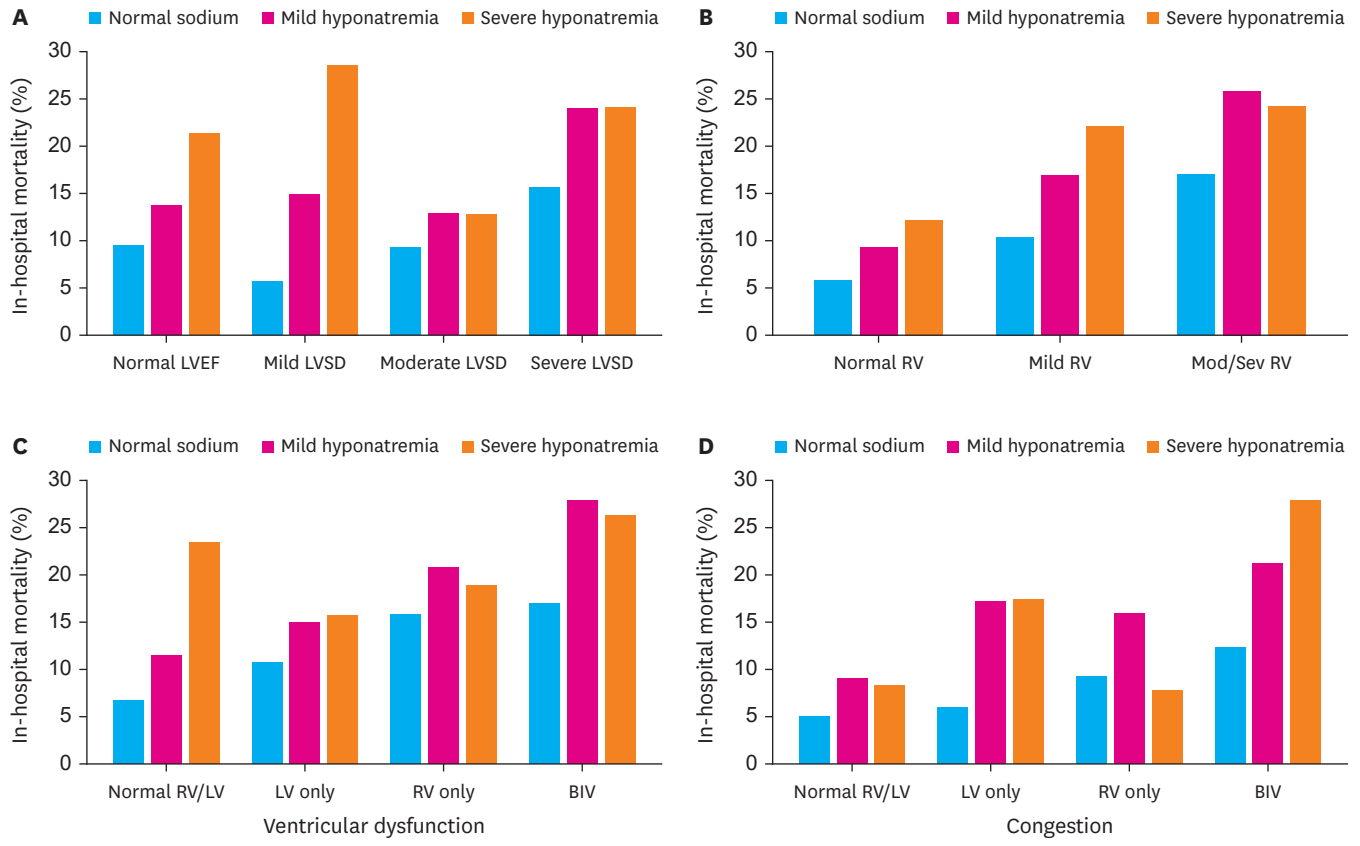

Figure 2. In-hospital mortality according to severity of hyponatremia and cardiac function by echocardiography. In-hospital mortality according to admission sodium group stratified by the severity of LVSD (A), RVSD (B), moderate or greater LVSD/RVSD (C), or congestion profile based on estimated RAP and PCWP (D). LVSD = left ventricular systolic dysfunction; RVSD = right ventricular systolic dysfunction; RAP = right atrial pressure; PCWP = pulmonary capillary wedge pressure; LVEF = left ventricular ejection fraction; RV = right ventricular; LV = left ventricular; BIV = biventricular.

hyponatremia is an independent risk factor for adverse outcomes and can identify patients with a high-risk phenotype or disease process. The mechanisms and directionality of these associations remain uncertain, but proposed pathophysiologic mechanisms may include increased neurohormonal activation (e.g., increased catecholamines, increased vasopressin), renal dysfunction, diuretic-induced disturbances, and worsened fluid retention with congestion.[21-23]

Consistent with previous studies, our analysis showed that more severe hyponatremia was incrementally associated with higher in-hospital mortality and a greater need for advanced treatment during CICU hospitalization among HF patients, as well as higher mortality out to one year after admission.[6,24] This may be expected as patients with low sodium levels had worse underlying cardiac function and hemodynamics, coupled with more severe critical illness. However, hyponatremia was not specific for poor LV function and was associated with worse diastolic function and RV hemodynamics. Notably, there was a synergy between TTE parameters and serum sodium for predicting mortality—patients with hyponatremia were at elevated risk, particularly when this was coupled with poor hemodynamics or cardiac function. Accordingly, CICU patients with HF who have hyponatremia are not only more likely to have hemodynamic compromise and poor cardiac function, but they are at elevated risk of adverse events in an additive manner that does not end at hospital discharge.

Hyponatremia appears to be a modest marker of more severe cardiac disease,[6] which is reflected by worse echocardiographic measurements of LV systolic function, forward flow, LV diastolic function, RV function, and congestion.[9,12] Hyponatremia itself does not correlate strongly with any echocardiographic parameters, although modest associations were observed, particularly for hemodynamic measurements related to stroke volume and blood pressure. Our findings emphasize that hyponatremia is an important parameter in risk stratification of patients with HF hospitalized in the CICU independent from cardiac structure and function.[9,25] Thus, using the bedside TTE to evaluate LV systolic function and especially systolic forward flow is appropriate in patients with severe hyponatremia.[12,25] Like previous studies, we observed increased mortality with the presence of diastolic dysfunction (elevated mitral E/e′ ratio) which was additive to the risk

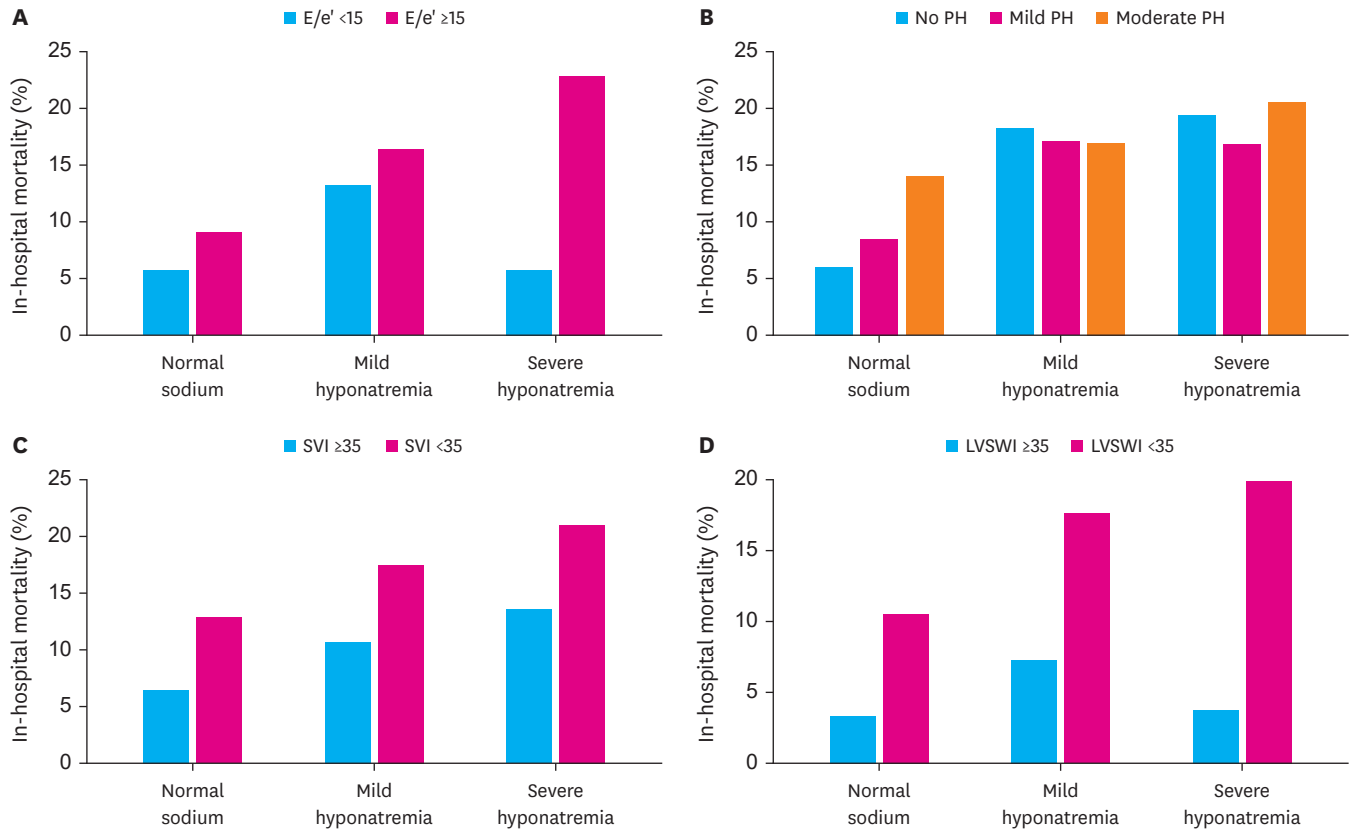

Figure 3. In-hospital mortality according to severity of hyponatremia and echocardiographic hemodynamics. In-hospital mortality according to admission sodium group stratified by medial mitral E/e′ ratio (A), PH severity based on RVSP (B), SVI (C), and LVSWI (D).
E/e′ ratio = ratio of mitral valve E velocity to medial mitral annulus e′ velocity; PH = pulmonary hypertension; RVSP = right ventricular systolic pressure; SVI = stroke volume index; LVSWI = left ventricular stroke work index.

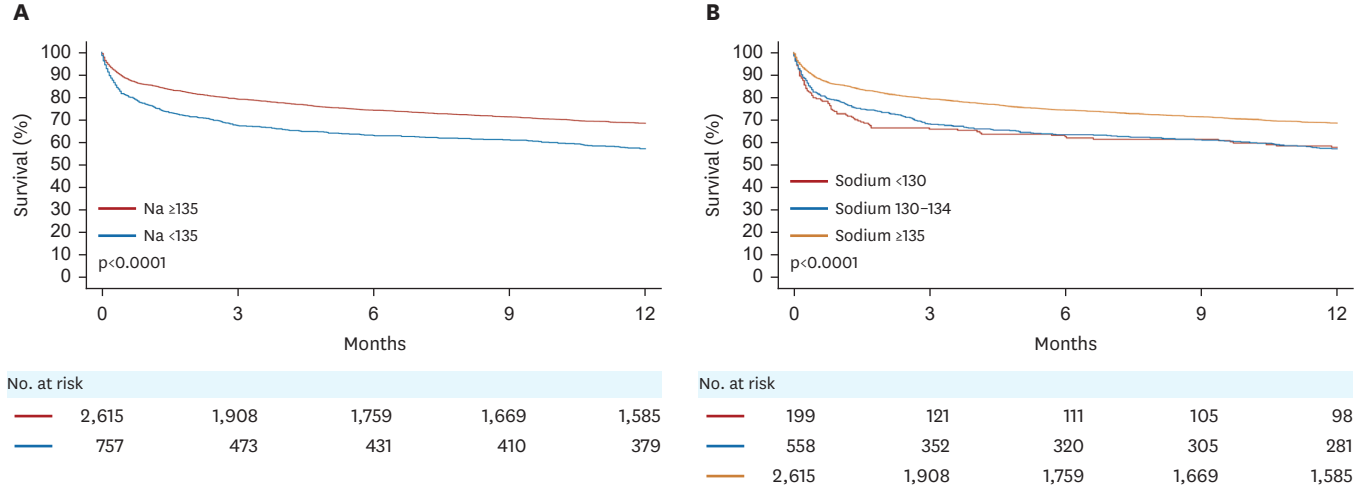

Figure 4. One-year survival according to the presence and severity of hyponatremia. Kaplan-Meier curves demonstrating all-cause one-year survival for patients with and without hyponatremia <135 mEq/L (A) and according to sodium group (B).

associated with hyponatremia.[11] The presence of RV dysfunction or worse RV-PA coupling, as reflected by low TASV/RVSP, predicted higher mortality across all sodium groups.

This study has limitations that are common to all retrospective studies, including the potential presence of unmeasured confounding factors and the inability to establish a causal

relationship between sodium levels and mortality. The Mayo Clinic CICU HF population may differ significantly from other populations, potentially limiting external applicability. We included a diverse HF cohort, which included patients with both acute and chronic HF as well as ACS. We had to exclude patients without sodium or TTE data, and many TTE variables had substantial missingness, resulting in potential selection bias as well as limited statistical power to detect weak effects. Clinical condition or diagnostic suspicion likely affected which TTE data were available, and TTE measurements were not over-read. We analyzed whole blood, plasma, and serum sodium values; we were unable to determine which patients had sodium levels measured by each assay, although the differences between the assays are unlikely to impact the main findings. We did not have data regarding diuretic therapy, fluid balance, or other factors that could have influenced both sodium levels and TTE findings. Likewise, we examined sodium levels at only a single time point which may not have been the time the TTE was performed and we did not have data on serial sodium testing, preventing us from evaluating the acuity or etiology of hyponatremia. This is a crucial limitation, insofar as changes in sodium levels over time, particularly in response to HF treatment, might provide a more useful evaluation of clinical status than a one-time assessment during critical illness. We anticipate that some patients might be reclassified based on subsequent improving or worsening hyponatremia during hospitalization, but that these changes could be prognostically important and might be more closely associated with cardiac function.

In conclusion, our study showed that hyponatremia correlates with more severe cardiac function abnormality and higher illness acuity in CICU patients with HF. Patients with hyponatremia have worse echocardiographic parameters reflecting LV and RV performance and systemic hemodynamics. Thus, the presence of hyponatremia could be a surrogate to guide clinicians in risk stratification of HF patients in the CICU and encourage the performance of rapid bedside TTE to identify those with a low-output state. Further research is needed to understand better the cardiovascular and non-cardiovascular contributions to hyponatremia in critically ill patients with HF.

ORCID iDs
Anan Younis
https://orcid.org/0000-0002-6486-8170
Meir Tabi
https://orcid.org/0000-0002-9199-9985
Kianoush B. Kashani
https://orcid.org/0000-0003-2184-3683
Garima Dahiya
https://orcid.org/0000-0003-1925-7722
Maan Jokhadar
https://orcid.org/0009-0009-0882-2133
Dustin B. Hillerson
https://orcid.org/0000-0002-9962-8855
Ruben Crespo-Diaz
https://orcid.org/0009-0001-9202-2922
Jacob C. Jentzer
https://orcid.org/0000-0002-6366-2859

Conflict of Interest
The authors have no financial conflicts of interest.

Author Contributions
Conceptualization: Younis A, Tabi M, Kashani KB, Jentzer JC; Data curation: Jentzer JC; Formal analysis: Jentzer JC; Investigation: Younis A, Tabi M, Kashani KB, Dahiya G, Jokhadar M, Hillerson DB, Crespo-Diaz R, Jentzer JC; Methodology: Younis A, Tabi M, Kashani KB, Dahiya G, Jokhadar M, Hillerson DB, Crespo-Diaz R, Jentzer JC; Project administration: Jentzer JC; Resources: Jentzer JC; Software: Jentzer JC; Supervision: Tabi M, Kashani KB, Jentzer JC; Validation: Jentzer JC; Visualization: Jentzer JC; Writing - original draft: Younis A, Jentzer JC; Writing - review & editing: Younis A, Tabi M, Kashani KB, Dahiya G, Jokhadar M, Hillerson DB, Crespo-Diaz R, Jentzer JC.

SUPPLEMENTARY MATERIALS

Supplementary Table 1
Measured and derived echocardiographic variables of interest

Supplementary Table 2
Formulas used to calculate echocardiographic hemodynamic parameters, using data from the time of the echocardiogram

Supplementary Table 3
Correlation coefficients with serum sodium

Supplementary Figure 1
Prevalence and severity of LVSD (A) and RVSD (B) according to admission sodium group.

Supplementary Figure 2
In-hospital mortality according to admission sodium group stratified by MCF (A), and TASV/RVSP ratio (B).

Supplementary Figure 3
Kaplan-Meier curves demonstrating all-cause one-year survival for patients according to sodium group stratified into those with LVSD (A) and without LVSD (B).

Supplementary Figure 4
Kaplan-Meier curves demonstrating all-cause one-year survival according to hyponatremia and either LVEF (A) or medial mitral E/e′ ratio (B).

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