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ORIGINAL ARTICLE

Community-Acquired Versus Hospital-Acquired Hyponatremia in Medical ICU: Clinical Characteristics and Outcomes

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ABSTRACT

Background: Hyponatremia is common in the Intensive Care Unit (ICU) and is linked to increased mortality. Hyponatremia may be community-acquired (CAH) or develop after hospital admission (HAH); however, there are limited studies that compare the characteristics of hyponatremia in both settings and whether this is reflected in the short-term outcome. We aimed to compare community-acquired (CAH) and hospital-acquired (HAH) hyponatremia regarding clinical and laboratory features and short-term outcomes, including in-hospital mortality and length of stay (LOS).

Methods: In this prospective cohort study, critically ill patients with hyponatremia, defined as serum sodium concentrations <135 mmol/L, were included and were grouped into the CAH group and the HAH group that developed during hospitalization. Demographic, laboratory, and clinical data and outcomes (mechanical ventilation, length of stay, and ICU mortality) were compared between both groups.

Results: The CAH group included 71 patients: 39 (54.9%) males and 32 (45.1%) females with a mean age ±SD of 56.54 ± 15.92 years, whereas the HAH group included 19 patients: 9 (47.1%) males and 10 (52.6%) females with a mean age ± SD of 56.21 ± 18.37 years, with no statistically significant difference between both groups. In HAH, compared to CAH, the most common primary diseases were cardiopulmonary (26.3%) vs. (31%), and gastrointestinal disease (26.3%) vs. (25.4%), with no statistically significant difference between both groups. This non-significant difference between both groups extended to include clinical severity scores, the need for mechanical ventilation, and length of stay. However, mortality was significantly higher in the CAH group, and having hyponatremia on admission was associated with a 4-fold increased risk of mortality compared to the HAH group (odd ratio 4.62, confidence interval (0.99–21.63) ($p<0.05$).

Conclusions: Community-acquired hyponatremia is more frequent in ICU settings and shares many characteristics with hospital-acquired hyponatremia; however, CAH is associated with a worse outcome, specifically a higher risk of mortality compared to HAH.

Key words: Hyponatremia, ICU, Community acquired, Hospital acquired.

INTRODUCTION

Hyponatremia, defined as a serum sodium concentration of <135 mEq/L, is commonly seen in critically ill patients. Hyponatremia has been linked to worse outcomes and mortality, especially If moderate or severe (sodium levels <130 and 125 mEq/L, respectively) [1]. Hyponatremia was linked to increased mortality ranging from 5% to 50% in different studies [2].

Hyponatremia may develop in the hospital; it may be hospital-acquired or may be present on

admission; it may be community-acquired. Limited comparative studies were done and showed conflicting results. Hyponatremia in hospital admissions was seen in up to 39.4% or even higher in ICU patients **[3]**. Whereas hospital-acquired hyponatremia was more prevalent and represented up to two-thirds of hyponatremia cases in another study [4]. Also, Hawkins [5] showed that hyponatremia occurred in 42% of hospitalized patients, with 14% hospital-acquired and 28% at admission. Hoorn et al. [6] reported that severe hospital-acquired hyponatremia was associated with inadequate management, such as the use of diuretics, hypotonic intravenous fluids, and drugs that stimulate the secretion of antidiuretic hormone.

Many studies analyzed either community-acquired hyponatremia (CAH) or HAH in unselected hospitalizations separately and linked both to increased hospital length of stay (LOS) and increased in-hospital mortality; however, to date, limited studies have compared hospital-acquired hyponatremia (HAH) and community-acquired hyponatremia. The aim of this prospective cohort study was to evaluate, characterize, and compare community-acquired and ICU-acquired hyponatremia regarding clinical and laboratory characteristics and outcomes, including ICU mortality and LOS, in patients admitted to a university hospital medical ICU.

METHODS

This is a prospective cohort study that included adult patients 18 years of age or older who were admitted to the medical ICU of the Internal Medicine Department of Zagazig University Hospital, Egypt. The study was approved by the Institutional Review Board of the Faculty of Medicine at Zagazig University [IRB#: 1872/15-2-2015]. Informed verbal consent was obtained from patients or their relatives, where available.

Definitions

Hyponatremia was defined as a serum sodium level less than 135 mmol/L [7]. CAH was defined as a serum sodium level of <135 mEq/L on the day of ICU admission, whereas HAH was defined as a serum sodium level of <135 mEq/L that developed >24 hours after hospitalization with a normal serum sodium value of ≥ 135 mEq/L on admission. In this study, hospital-acquired and ICU-acquired hyponatremia terms were used interchangeably. Patients were followed up during hospitalization, the study endpoint was patient death, or discharge.

A total of 90 hyponatremic patients were enrolled in this study. Those patients were admitted to the medical intensive care unit with different medical conditions. They were divided into two groups according to the timing of hyponatremia. Hyponatremia was further subdivided according to volume status evaluation clinically and by measuring CVP into three different patterns: hypovolemic, euvolemic, and hypervolemic, and according to severity of hyponatremia into three grades: mild, moderate, and severe, corresponding to serum sodium concentrations of 130–134 mmol/L, 125–129 mmol/L, and < 125 mmol/L, respectively [8].

Exclusion criteria

Patients with hyperlipidemia or a prior diagnosis of paraproteinemia were excluded from the study. Patients receiving mannitol or radiographic contrast agents were excluded from the study. Patients with hyperglycemia (blood glucose levels greater than 200 mg/dL) were excluded from the study.

Demographics, biochemical analyses, and follow-Up

Data regarding demographics (age, gender), medical history, and clinical and laboratory parameters associated with hyponatremia (such as sodium, glucose, creatinine, urea, proteins, cholesterol, and triglycerides in serum) were collected. The presence of symptoms was assessed at admission and included all symptoms associated with hyponatremia (for example, nausea, vomiting, and dizziness). Patients were followed up during their stay in the ICU, and patient outcomes included mortality and length of stay in days.

A full medical history was obtained, including drug history, especially the use of thiazide diuretics and selective serotonin reuptake inhibitors (SRRIs), and the history of any comorbidities. Full clinical examination with special attention to blood pressure, heart rate, temperature, and evaluation of the volume status by measurement of CVP and clinically; peripheral edema (pedal or sacral), skin turgor, and capillary refill time; lung crepitations or rales. Clinical severity was measured by the commonly used scores in the ICU: Acute physiology assessment and chronic health evaluation (APACHE II) [9] and the Sequential organ failure assessment score (SOFA) [10].

Statistical analysis

All data from the present study were coded, checked, entered, and analyzed using SPSS software released in 2017. IBM SPSS Statistics for Windows, Version 25.0 Armonk, NY: IBM Corp. Continuous data were expressed in the form of mean ± SD, while categorical data were expressed

in the form of count and percent. Comparisons of continuous data were performed using the student T-test or one-way ANOVA, as appropriate, while categorical data were done using the Chi-square test. Relationships between variables were investigated by Pearson's correlation coefficient. A Kaplan-Meier plot was used to show the relationship between overall survival and the timing of hyponatremia among the studied patients. A p-value less than 0.05 was considered statistically significant.

RESULTS

Patient characteristics are presented in Table 1. In this study, we included a group of 71 patients with community-acquired hyponatremia; their average age was 56 ± 15 years, with 32 females (45.1%) and 38 males (54.9%); compared to a group of 19 patients with hospital-acquired hyponatremia, with an average age of 56 ± 18 years, with 10 females (52.6%) and 9 males (47.1%). The two groups did not show statistically significant differences regarding sex distribution and age.

In this study, cardiopulmonary disease was the most common disease (26% HAH vs. 31% CAH), followed by gastrointestinal disease (26.3% HAH vs. 25.4% CAH). Infection, hematological, metabolic, neurological, and renal diseases showed no significant difference between HAH and CAH regarding the frequency of these associated diseases.

Table 2 shows comparable clinical and laboratory data between both groups. Tables 3 and 4 show that in both CAH and HAH, euvolemic hyponatremia and moderate-severity hyponatremia patterns were the most common. There is no significant difference between both groups regarding the distribution of hyponatremia in relation to severity or volume status.

Table 5 compares morbidity and mortality scores between both groups. It shows non-statistically significant differences regarding APACHE II score, SOFA score, requiring mechanical ventilation, or length of stay. However, higher mortality was observed in the CAH group compared to the HAH group: 25 (35.2%) and 2 (10.5%) patients died, respectively, p = 0.037. The timing of hyponatremia added more risk to mortality, as having hyponatremia on admission (CAH) increased mortality risk by more than 4 folds (Odd Ratio (95% CI) = 4.62 (0.99–21.63), p <0.05). Table 6 shows a comparison of overall survival between the CAH and HAH groups. There was a statistically significant association between overall survival and timing of hyponatremia, as survival time was 17.09 ± 0.55 days in patients with CAH, which was significantly lower compared to patients with HAH (20.0 ± 1.12) days, p = 0.043.

Figure 1 shows the Kaplan-Meier plot that illustrates the relationship between overall survival and the timing of hyponatremia. Among the studied patients, the Kaplan-Meier plot shows a significant difference between CAH (admission) and HAH (hospital-acquired) patients regarding in-hospital mortality.

Table 1: Comparison of demographic and etiological data between hospital-acquired and community-acquired hyponatremia:

Parameter	Hyponatremia		Test	
	HAH N (19)	CAH N (71)	t/χ^2	p
Age (year) Mean ± SD	56.21 ± 18.37	56.54 ± 15.92	-0.076	0.939
Gender	N=19	N=71		
Female	10 (52.6%)	32 (45.1%)	0.344	0.557
Male	9 (47.1%)	38 (54.9%)		
Primary disease:				
Cardiopulmonary	5 (26.3%)	22 (31%)		
Gastrointestinal	5 (26.3%)	18 (25.4%)		
Infection	1 (5.3%)	9 (12.7%)	MC	0.744
hematological	1 (5.3%)	6 (8.5%)		
Metabolic	1 (5.3%)	4 (5.6%)		
Neurological	3 (15.8%)	4 (5.6%)		
Renal	3 (15.8%)	8 (11.3%)		

t independent sample t test; χ^2 Chi square test; MC Monte Carlo test.
HAH: hospital acquired hyponatremia, **CAH**: community acquired hyponatremia.

Table 2: Comparison of clinical and laboratory data between hospital-acquired and community-acquired hyponatremia

Parameter	Hyponatremia		Test	
	HAH	CAH	t	p
	Mean ± SD	Mean ± SD		
Temperature °C	37.54 ± 0.49	37.63 ± 0.64	-0.524	0.601
Pulse /minute	94.26 ± 9.08	99.99 ± 11.94	-1.942	0.055
Respiratory rate / minute	16.95 ± 3.01	18.27 ± 15.57	-1.398	0.166
Mean blood pressure	87.32 ± 13.95	83.76 ± 15.57	0.903	0.369
CVP (cm H2O)	11.11 ± 2.73	11.04 ± 3.4	0.075	0.941
Sodium (mmol/L)	127.79 ± 4.02	126.44 ±4.81	1.124	0.264
Potassium (mmol/L)	3.67 ± 0.76	3.87 ± 0.77	-1.013	0.314
Hematocrit %	31.7 ± 9.4	30.71 ± 6.87	0.429	0.672
Hemoglobin (g/dl)	10.8 ± 3.18	10.31 ± 2.36	0.626	0.537
PT (seconds)	15.33 ± 5.41	16.82 ± 5.57	-1.041	0.301
PTT (seconds)	40.83 ± 11.68	43.81 ± 9.99	-1.114	0.268
Protein (g/dl)	5.58 ± 0.93	5.95 ± 0.86	-1.631	0.106
Albumin (g/dl)	2.78 ± 0.62	2.71 ± 0.6	0.465	0.649
Parameter	**Hyponatremia**		**Test**	
	HAH	**CAH**		
	Median (IQR)	**Median (IQR)**	**Z**	**p**
Serum creatinine (mg/dl)	1.11 (0.88 – 1.45)	1.14 (0.84 – 2.36)	-0.138	0.89
Blood urea (mg/dl)	35 (22.5 – 46.4)	39.3 (22.95 – 59.5)	-0.771	0.441
WBCS ($\times 10^3/mm^3$)	12.4 (8.95 – 16.1)	9.6 (7.65 – 12.95)	-1.449	0.147
Platelet ($\times 103$ $/mm^3$)	229 (117.5 – 261)	180 (119 – 260)	-0.138	0.89
Total Bilirubin (mg/dl)	0.96 (0.62 – 1.2)	0.94 (0.66 – 1.58)	-0.282	0.778
AST (IU/L)	33 (28 – 38)	32 (22 – 54)	-0.139	0.89
ALT (IU/L)	26 (21 – 49)	25 (16 – 43.5)	-0.99	0.322

t independent sample t test; Z Mann Whitney test. **IQR** interquartile range. **CVP**: central venous pressure, **PT**; prothrombin time. **PTT** partial thromboplastin time. **WBCs**: white blood cell count. **AST**: aspartate transaminase. **ALT**: alanine transaminase. **HAH:** hospital acquired hyponatremia, **CAH:** community acquired hyponatremia.

Table 3: Distribution of different pattern of hyponatremia according to volume status, in both groups

Parameter	Hyponatremia		Test	
	HAH	CAH	χ^2	p
	N=19 (%)	N=71 (%)		
Hypovolemic	3 (15.8%).	13 (18.3%)		
Euvolemic	11 (57.9%)	36 (50.7%)	0.015	0.903
Hypervolemic	5 (26.3%)	22 (31%)		

t independent sample t test. **HAH**: hospital acquired hyponatremia, **CAH**: community acquired hyponatremia

Table 4: Distribution of different patterns of hyponatremia according to severity in both groups

Parameter	Hyponatremia		Test	
	HAH	CAH	χ^2	p
	N=19 (%)	N=71 (%)		
Severe (<125) Moderate125 – 130) Mild (>130)	1 (5.3%). 13 (68.4%) 5 (26.3%)	5 (7 %) 53 (74.6%) 13 (18.4%)	0.57	0.45

t independent sample t test. **HAH**: hospital acquired hyponatremia, **CAH**: community acquired hyponatremia

Table 5: Comparison of morbidity and mortality scores between both groups

Parameter	Hyponatremia		Test	
	HAH	CAH	t	p
	Mean ± SD	Mean ± SD		
APACHE II score	16.63 ± 5.8	18.85 ± 6.59	-1.331	0.187
LOS	12.74 ± 4.99	12.54 ± 4.68	0.165	0.87
	Median (IQR)	Median (IQR)	Z	p
SOFA score	5 (3 – 5)	4 (3 – 6.5)	-0.667	0.505
MV No Yes s	 16 (84.2%) 3 (15.8%)	 61 (85.9%) 10 (14.1%)	Fisher	>0.999
Mortality outcome	N=19 (%)	N=71 (%)	χ^2	p
Died (27) Survivors (63)	2 (10.5%) 17 (89.5%)	25 (35.2%) 46 (64.8%)	4.349	**0.037***
Crude odds ratio for mortality outcome				
COR (95% CI)	**4.62(0.99-21.63)**			**<0.05**

t independent sample t test; Z Mann Whitney test; IQR interquartile range; χ^2 Chi square test. **APACHE II:** Acute physiology assessment and chronic health evaluation. **COR** crude odds ratio. **CI** confidence interval. **MV**: mechanical ventilation. **LOS** length of stay. **SOFA:** Sequential organ failure assessment score.

Table (6) Comparison of overall survival between both groups.

		Total N.	N. of Events	Censored		Survival time, days		p
				N.	%	Mean		
						Estimate ± SD	95% CI	
Timing	HAH	19	2	17	89.5%	20.0 ± 1.12	17.81 – 22.19	0.043*
	CAH	71	25	46	64.8%	17.09 ± 0.55	16.01 – 18.17	
Overall		90	27	63	70.0%	17.64 ± 0.52	16.63 – 18.65	

*p<0.05 is statistically significant. **HAH**: hospital acquired hyponatremia, **CAH**: community acquired hyponatremia

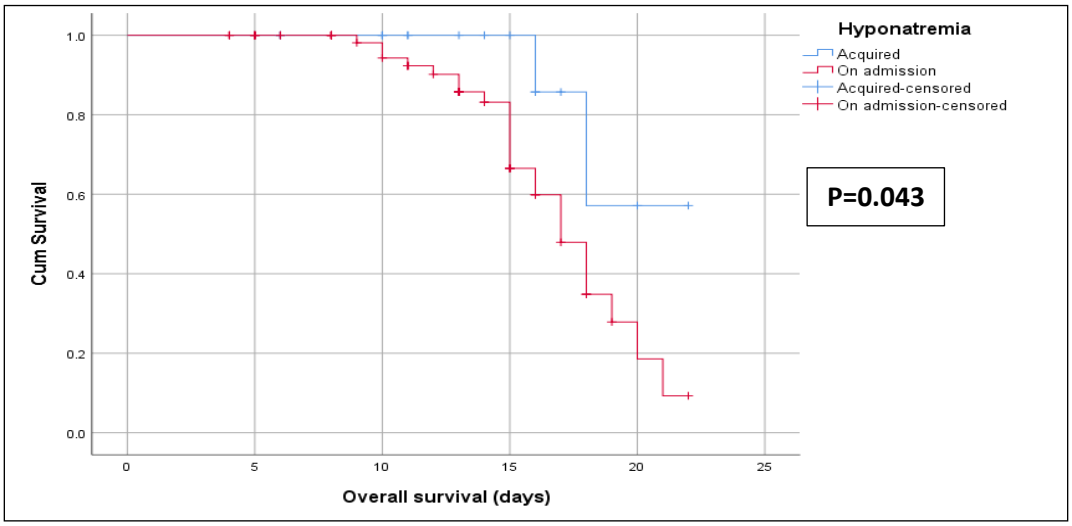

Figure 1: Kaplan Meier plot showing relation between overall survival and timing of hyponatremia among studied patients.

DISCUSSION

This analysis of hyponatremic patients admitted to our medical ICU revealed that CAH is more frequent. CAH is associated with a higher risk of ICU mortality compared to ICU-acquired hyponatremia. During the study period, we included 90 hyponatremic patients; CAH was 4 times more frequent than HAH; 71 patients (78.9%) had CAH on admission, and 19 patients (21.1%) had HAH after admission. Like our study, in a recent retrospective analysis of 6,539 hospitalizations; overall hyponatremia occurred in 32.5%, and CAH was more common than HAH (24.7% compared to only 10.3%), respectively. Most cases of hyponatremia were mild (69.1%), with 20.2% being moderate and 10.7% being severe [13]**.** Also, Hawkins [5] showed that CAH was two-fold more frequent than HAH**.** On the contrary, another study showed that hospital-acquired hyponatremia was more prevalent and represented up to two-thirds of hyponatremia cases [4]. Similarly, Shchekochikhin et al. [14] showed that HAH was more common, as it occurred in 30.2% of patients compared to CAH, which occurred in 19.4%. In an analysis of 53,236 hospitalizations, Wald et al. [15] showed that both types of hyponatremias occurred at a comparable frequency: community-acquired hyponatremia occurred in 37.9% of hospitalizations, and hospital-acquired hyponatremia developed in 38.2% of hospitalizations, worth mentioning that they used a higher cutoff for the definition of hyponatremia: a serum sodium less than 138 mEq/L.

Hyponatremia in critically ill patients is associated with poor clinical outcomes such as prolonged hospital stays, increased hospital costs, increased mortality, and even mild or chronic hyponatremia, which represents an economic burden [11]**.** In a study that included 600 ICU patients**,** Mahmoud et al. [12] found that ICU-acquired hyponatremia was common in the ICU. Hyponatremia occurred in 18% of patients and was associated with a 2.5-fold increased risk of mortality compared to normonatremic patients.

This difference in prevalence of both types of hyponatremias might be related to different medical practices. Hospital-acquired hyponatremia is related to the administration of hypotonic intravenous fluids, and this was shown in a recent study by Sindahl et al. [16], which was A cross-sectional survey to analyze the Prescribing practices of Danish emergency department physicians. In that survey, they found that most physicians were unaware of the effect of hypotonic fluids on serum sodium in acutely ill patients, and they concluded that HAH is related to the limited knowledge of a great number of physicians associated with inappropriate intravenous fluid prescribing practices, they also suggested that further interventions should be implemented to decrease the risk of HAH.

The frequency of hyponatremia in terms of severity and volume status was comparable between both groups. Most patients in both groups were clinically euvolemic (50.7%) in CAH vs. (57.9%) in HAH, and moderate hyponatremia was the most common type regarding severity of hyponatremia (74.6%) in CAH vs. (68.4%) in HAH, a non-statistically

significant difference. One study showed similar findings; euvolemic hyponatremia was the most common type of hyponatremia [17], but mild hyponatremia was the most common (66.0%).

Many studies have reported that stroke, heart failure, pneumonia, liver cirrhosis, and other diseases are commonly associated with hyponatremia [18–20]. The results of our study show that cardiopulmonary disease and gastrointestinal diseases are commonly associated with hyponatremia. So monitoring serum sodium in those patients may offer early detection and correction of hyponatremia, which may reflect an improved mortality outcome.

In this cohort, markers of clinical severity were comparable between the CAH and HAH groups, as were APACHE and SOFA scores. The percentage of patients who were mechanically ventilated and LOS all showed non-statistically significant differences; however, the mortality outcome was significantly different.

Many studies have shown that hyponatremia is a predictor of mortality and a direct cause of increased mortality. In a study by Wald et al. [15], survival rates were reduced in patients with either CAH or HAH. The mortality risk was higher in the HAH group (23% compared to the CAH group's 8%) for every 1-mmol/L drop in serum sodium level. Compared to our study, Wald et al. [15] included a large number of patients compared to ours (53236 patients); second, for some reason, they used a higher level of sodium to define hyponatremia (they defined hyponatremia as serum sodium less than 138 mEq/L), so it seems that they included many normonatremic patients; third, although mortality was higher in the HAH group, the comorbidity index score surprisingly showed a non-significant difference between HAH and CAH, so again, higher mortality in HAH was not explained by morbidity scores ,and that was similar to our study, as we found no difference in morbidity scores between both groups**.** Also, Tzoulis et al. [21] found that the risk of in-hospital mortality increased 3.3 times and the mortality rate increased 17.3% in patients with serum sodium concentrations < 128 mmol/L**.** These findings suggest that hyponatremia is both an independent predictor and a direct cause of increased mortality. Many studies linked the severity of hyponatremia to an increase in both hospitalization time and mortality [17, 22, 23].

In our study, mortality was higher in the CAH group compared to the HAH group (35.2% vs. 10%) and having hyponatremia on admission (CAH) increased mortality risk by more than 4 folds. Similar to our study, Omar and Guglin [24] have shown in their analysis that they compared hospitalized patients with acute heart failure with either CAH or HAH. CAH is slightly more common (23.8%) compared to HAH (19.9%); however, patients with CAH had worse post-discharge outcomes compared with HAH. They explained that finding by citing the nature of HAH, which likely represents the transient effect of medications (diuretics), whereas they considered CAH to be a more reliable reflection of the overall disease status. Surprisingly, they found no association between HAH and poor outcomes, even though they concluded that HAH has a favorable intermediate-term outcome in acute systolic HF compared with CAH.

In hospitalized patients with heart failure, Shchekochikhin et al. [14] showed that HAH was more common compared to CAH (30% vs. 19%). Both HAH and CAH groups had the same poor prognosis regarding in-hospital mortality, with no statistically significant difference (9.7% vs. 9.1%; p = 0.58). They concluded that hyponatremia, whether community-acquired or hospital-acquired, was independently associated with increased in-hospital mortality**.** Nevertheless, in that study, although in-hospital mortality was similar, patients with HAH were more ill at baseline and had more comorbidities. HAH had a higher prevalence of chronic kidney disease, a significantly lower estimated glomerular filtration rate, more myocardial infarctions, and a higher comorbidity index score compared to patients with CAH. So, it seems that disease severity or added comorbidities are not associated with an increased risk of mortality. The study by Shchekochikhin et al. [14] shows that the severity of the associated disease or comorbidities is not a "sine qua non" for increased mortality risk in patients with either CAH or HAH. Similarly, we found that mortality was higher in patients with CAH despite similar morbidity scores (SOFA and APACH II) in both groups. Also, Wald et al. [15] showed that morbidity scores do not explain higher mortality in HAH. But it is still unclear what would cause increased mortality in relation to the timing of hyponatremia in CAH compared to HAH, but it may be related to the transient nature of hyponatremia in HAH, which was even associated with a favorable outcome compared to CAH, as explained by Omar and Guglin [24].

Saepudin et al. [25] showed contradictory results in patients hospitalized for heart failure, as they found that the prevalence of HAH was almost the same as that of CAH (22% and 19%, respectively). Surprisingly, in terms of in-hospital mortality, while CAH had no association with in-hospital mortality, HAH had an increased risk of mortality [odds ratio 3.473 (95% CI 1.899–6.351)].

In our study, mortality was high—up to 35% in CAH compared to 10% in HAH. We found no significant difference between both groups regarding the mean sodium level or frequency of hyponatremia in relation to severity or volume status. Moreover, clinical severity scores such as APACHE II and SOFA scores showed no significant difference. Although our results look different from the previously mentioned studies, Similar to our study, an analysis of 279508 acutely hospitalized patients showed that the risk of death increased with decreasing serum sodium from 139 to 132 mmol/L; however, that occurred only with mild hyponatremia, as the decrease in serum sodium below a threshold of 132 mmol/L did not show a further increase in overall mortality risk, and they concluded that hyponatremia is associated with increased 30-day and 1-year mortality risk, regardless of underlying disease and independent of hyponatremia severity [2]. Our results also, agree with Zheng et al. [27], who showed that severe hyponatremia was not associated with worsening mortality, as they found no significant difference in the risk of death between the severe and moderate groups. Patients with moderate or severe hyponatremia had a higher mortality rate than those with mild hyponatremia (moderate vs. mild group: OR 6.92, 95% CI 2.53–18.92, $p< 0.001$; severe vs. mild group: OR 4.54, 95% CI 1.05–19.58, $p = 0.043$). There was no significant difference in the risk of death between the severe and moderate groups.

This is one of the limited studies that compares CAH and HAH in ICU regarding patient characteristics and outcome, which pointed out the grave outcome of CAH compared to HAH. However, our study has several limitations, as it was a single center study with a small cohort of patients, especially the HAH group, which may limit the generalizability of our findings. Another limitation related to hyponatremia definition, which was identified by only one measurement as sodium level, was not followed up during the patient's ICU stay, and that may not reflect the course of hyponatremia, either normalized, persistent, or worsened. Finally, outcome measurement was only in-hospital, with no short- or long-term assessment after discharge.

CONCLUSIONS

CAH is more frequent in the ICU compared to HAH, and although both groups showed similarities in baseline characteristics, morbidity scores, severity of hyponatremia, and distribution of volume status, having hyponatremia on admission was associated with a higher risk of in-hospital mortality.

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