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Hyponatremia in Critically Ill Patients and its Relation to the Outcomes

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ABSTRACT

Background: Hyponatremia, defined as a serum sodium (Na^+) concentration below 135 mmol/L, is the most encountered electrolyte abnormality.

Objectives: This study aimed to ascertain causes, outcomes (duration and mortality) of hyponatremia and management in critically ill cases in intensive care unit (ICU).

Subjects and methods: The current study conducted at Faculty of Medicine, Mansoura University, and was performed on 50 patients with hyponatremia with serum above 18 years old. Laboratory investigations included complete blood count (CBC), Arterial Blood gases, liver functions tests, renal functions tests, serum electrolytes (Na^+, K^+, Mg^{2+}, Phosphorus and Calcium), coagulation profile, C-reactive protein (CRP), fasting Blood sugar levels, serum uric acid, fasting lipid profile, serum osmolality and urine analysis.

Result: The median age was 66 years, indicating a predominantly elderly population. The median SOFA score was 9. Serum osmolality was decreased, indicating true hypotonic hyponatremia. The median Urinary sodium was 27 mmol/L. On univariate analysis, three predictors were statistically significant which are SOFA score > 9, platelet count ≤ 57 per mm3, and AST > 48 IU/L. On multivariate analysis, only one was statistically significant independent predictor which is SOFA score > 9. The model was statistically significant ($\chi 2$ [5] = 30.022, p<.001). The model correctly classified 88% of cases, with 87.5% sensitivity, and 88.2% specificity.

Conclusion: This study demonstrates that SOFA score is the only independent predictor of ICU mortality, highlighting the importance of overall organ dysfunction over sodium level alone in outcome prediction.

Keywords: Hyponatremia, Critically Ill Patients, ICU.

INTRODUCTION

Hyponatremia, a frequent electrolyte abnormality is defined as a serum sodium (Na) level below 135 mmol/L [1]. Hyponatremia divided into mild (Na^+ level 135–130 mmol/L), moderate (130–125 mmol/L), and severe (less than 125 mmol/L). This may be classified as acute (ongoing for two days or less) versus chronic (ongoing for more than two days) [2].

The cause of hyponatremia is classified based on the volume condition of the extracellular fluid (ECF). Sodium is the primary solute of ECF. According to ECF volume, a patient could be classified into hypovolemic, euvolemic, or hypervolemic [3]. Symptoms accompanied by hyponatremia range from mild non-specific manifestations (dizziness, or headache) to fatal cerebral oedema. Secondly, due to the effective osmolality difference between the brain and plasma, cerebral cells start to swell when water moves from the extracellular compartment to the intracellular compartment. This typically happens when rapid hyponatremia occurs, causing the brain to have limited time to adjust to its hypotonic environment. [4].

Isotonic hyponatremia has to raise suspicion of pseudohyponatremia, which could be met owing to paraproteinemia or hyperlipidemia. The term pseudohyponatremia refers to a incorrectly low measurement of serum Na levels secondary to expansion of the solid phase of the plasma by a high protein level (in paraproteinemia) or lipid level (in hypertriglyceridemia and hypercholesterolemia) [5].

Management of hyponatremia is based on the onset and severity of hyponatremia and presenting manifestations. When a case with hyponatremia presents with extensive neurologic manifestations, which include fits or disturbed conscious level causing coma, an increase in serum Na by five mEq/L by administration of hypertonic saline is required, generally by administration of 100-ml triple saline [3].

Water restriction and isotonic saline have been considered the main treatment options for hyponatremia; however, their efficiency is restricted. In addition, stoppage of diuretic agents, management of hypokalemia, and usage of vasopressin receptor antagonists have been tried to treat mild and moderate hyponatremia; however, their efficiency and safety are still to be confirmed. Hypertonic saline is indicated for cases with severe hyponatremia, which include fits and altered mental status [6].

This study aimed to ascertain causes, outcomes (duration and mortality) of hyponatremia and management in critically ill cases in intensive care unit (ICU).

PATIENTS AND METHODS

This prospective observational study included 50 patients with hyponatremia admitted to the Critical Care Medicine Unit, Mansoura University, from December 2023 to June 2025.

Hyponatremia, defined as a serum sodium (Na^+) concentration below 135 mmol/L, is a relatively frequent electrolyte abnormality, affecting about 30% of hospitalized patients [4].

Inclusion criteria: Patients aged ≥18 years, of either sex, with hyponatremia in the medical ICU at Mansoura

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University Hospital, either within 24 hours of admission or during ICU stay after 24 hours.

Exclusion criteria: Patients with hyperlipidemia, paraproteinemia, those receiving mannitol or radiographic contrast agents, and those with diabetes mellitus.

All cases were subjected to the detailed history taking including age, gender, different medical comorbidities and drug history (diuretic use) from patients or their relatives. Complete clinical examination was done to all patients including the assessment of vital signs which include systolic, diastolic and mean arterial blood pressure, pulse, respiratory rate, temperature, and continuous saturation. Full systemic Examination included cardiac, neurological, respiratory, abdominal and lower limbs examination.

Laboratory investigations included CBC, arterial blood gases, liver functions tests (ALT (alanine transaminase), AST (aspartate transaminase), total, direct & indirect bilirubin)), renal functions tests (serum Urea, BUN and Serum creatinine), serum electrolytes (Na^+, K^+, Mg^{2+}, Phosphorus and Calcium (total and ionized)), coagulation profile, CRP, fasting Blood glucose levels, serum uric acid, fasting lipid profile (serum total cholesterol, serum triglycerides), serum osmolality and urine analysis.

Serum osmolality was a laboratory test that measures the concentration of dissolved particles (solutes) such as Na, glucose, and urea in the blood. It reflects the body's water–electrolyte balance and is crucial in evaluating conditions like hyponatremia, hypernatremia, dehydration, and poisoning.

Urinary sodium testing was done using midstream and clean catch urine on ion-selective electrode (ISE). Bedside twelve leads were used for any evidence of arrhythmia. Bedside Ultrasound was used to assess Inferior Vena Cava (IVC) Diameter.

Urinary sodium concentration was measured using a spot urine sample or a 24-hour urine collection. The most employed analytical methods are ion-selective electrodes (ISE), flame photometry, and indirect potentiometry. Urine samples were analyzed within one hour. Sequential organ assessment score (SOFA) system was used for all patients on admission [7]. Total SOFA Score ranged from 0 to 24, Mortality <10%: SOFA Score 0 to 6, 15–20%: SOFA Score 7 to 9, 40–50%: SOFA Score 10 to 12, 50–60%: SOFA Score 13 to 14, >80%: SOFA Score 15 and >90%: SOFA Score 15 to 24. Laboratories follow internal and external quality control protocols to ensure accuracy and reproducibility.

Ethical Consideration

The study protocol was approved by the Institutional Research Board (IRB) of the Faculty of Medicine, Mansoura University. Approval was also obtained from the hospital administration where the study was conducted. Confidentiality was maintained, and the collected data were not used for any other purpose. Written informed consent was obtained from all participants. The study protocol adhered to the Declaration of Helsinki, the ethical standard of the World Medical Association for research involving human subjects.

Statistical Analysis

Data was analyzed using SPSS version 22. Qualitative data were presented as number and percent; Quantitative data were tested for normality by Shapiro-Wilk test then described as mean and standard deviation for normally distributed data and median and range for non-normally distributed. The proper statistical test was used based on data type.

RESULTS

Table **(1)** shows that females represented a slightly higher proportion of the cohort (54%) compared to males (46%). In terms of occupation, housewives constituted the largest group (46%), followed by manual workers (24%), farmers (20%), and employees (10%). According to Severity of hyponatremia. Mild type (130-135) represented (16%), moderate type (125-130) represented (38%), severe type represented (46%), Notably, 44% of patients were found to be using diuretics.

Table (1): Characteristics of the 50 cases at the time of enrollment regarding demographic data, severity and diuretic use

Characteristic	N	%
Sex		
Male	23	46
Female	27	54
Occupation		
Farmer	10	20
Housewife	23	46
Employee	5	10
Manual worker	12	24
Severity of hyponatremia		
Mild	8	16
Moderate	19	38
Severe	23	46
Diuretic use	22	44

Table **(2)** shows the median age of the studied patients was 66 years (IQR: 58.8–74), indicating a predominantly elderly population. The inferior vena cava (IVC) diameter was within the normal range, with a median of 2 cm (1.8–2.2), suggesting relatively preserved volume status in most cases. Neurological assessments showed that patients had high levels of consciousness at presentation, with a median Glasgow Coma Scale (GCS) of 15 and a FOUR score of 16, both indicating minimal to no neurological compromise. the SOFA score, which had a median of 9 (IQR: 5.8–11), reflects moderate to severe organ dysfunction in many

cases. The median serum sodium level was 125 mmol/L (120–128.2), confirming moderate hyponatremia across the cohort.

Both corrected sodium values (by Katz and Hillier formulas) aligned closely with the measured sodium, suggesting no significant pseudohyponatremia due to hyperglycemia (median fasting glucose: 98.5 mg/dL). Serum osmolality was also low (median: 268.8 mOsm/kg), indicating true hypotonic hyponatremia. Urinary sodium had a median of 27 mmol/L, with a range supportive of either hypovolemic or euvolemic hyponatremia depending on the clinical context. Hematological parameters revealed mild anemia (hemoglobin: 9.7 g/dL) and borderline thrombocytopenia (platelet count: 130.5 $x10^3/\mu L$). White blood cell count was within normal limits. Electrolyte levels such as serum potassium (3.9 mmol/L) and serum creatinine (1.6 mg/dL) suggest mild renal impairment or hypoperfusion in some cases.

Liver function parameters showed moderate hypoalbuminemia (serum albumin: 2.6 g/dL) and elevated AST (median: 54.5 IU/L), with a preserved ALT level, yielding an AST/ALT ratio >1, which may suggest non-alcoholic liver stress or systemic inflammation.

The direct-to-total bilirubin ratio was elevated (66%), suggesting possible cholestasis or hepatocellular injury. Lipid profile and metabolic markers including triglycerides (130 mg/dL) and total cholesterol (206.5 mg/dL) were within or slightly above normal ranges.

Table (2): Characteristics of included cases regarding age, IVC Diameter, clinical scores, laboratory tests

Characteristic	**Median**	**Q1-Q3**
Age (years)	66	58.8-74
IVC diameter (cm)	2	1.8-2.2
Glasgow Coma Scale (GCS)	15	14-15
FOUR (Full outline of unresponsiveness) score	16	15-16
SOFA score	9	5.8-11
laboratory tests		
► Serum sodium (mmol/L)	125	120-128.2
► Fasting blood glucose (mg/dl)	98.5	90-110.2
► Corrected Sodium (Katz, 1973)	125	120-128
► Corrected Sodium (Hillier, 1999)	125	120-128
► Serum osmolality	268.8	262.2-282
► Urinary sodium (mEq/L)	27	21.2-43.4
► WBC count ($x10^9$ /l)	9	7.4-15
► Hemoglobin (g/dl)	9.7	8.4-10.7
► Platelet count	130.5	73.3-198.3
► Serum potassium (mmol/L)	3.9	3.4-4.9
► Serum creatinine (mg/dl)	1.6	0.8-2.5
► Serum uric acid (mg/dl)	7	5-9
► Triglycerides (mg/dl)	130	90-177.2
► Serum total cholesterol (mg/dl)	206.5	170-250
► Serum albumin (g/dl)	2.6	2.1-3
► AST (IU/L)	54.5	38.8-86.8
► ALT (IU/L)	29.5	19.5-50.3
► AST/ALT ratio	1.7	1.3-2.5
► Serum total bilirubin (mg/dl)	2.7	0.7-10.6
► Serum direct bilirubin (mg/dl)	1.5	0.3-8.5
► Direct bilirubin / total bilirubin percentage	66	42.1-77.8

AST: Aspartate transaminase, ALT: Alanine transaminase, IVC: Inferior vena cava, WBC: White blood cells

Table (3) shows no statistically significant association between sex, occupation, between Severity of hyponatremia, Diuretic use, Use of 3% saline infusion, Timing of hyponatremia, Hyponatremia type and the outcome (favorable vs. worse).

Table (3): Comparisons of worse and favorable outcomes according to demographic data, severity, types, timing, causes and treatment of hyponatremia

Characteristic	Survivors		Deceased		Sig
	N	%	N	%	
Sex					.108*
Male	13	38.2	10	62.5	
Female	21	61.8	6	37.5	
Occupation					.315**
Farmer	6	17.6	4	25	
Housewife	18	52.9	5	31.3	
Employee	4	11.8	1	6.3	
Manual worker	6	17.6	6	37.5	
Severity of hyponatremia					
Mild	6	17.6	2	12.5	.876*
Moderate	13	38.2	6	37.5	
Severe	15	44.1	8	50	
Diuretic use	16	47.1	6	37.5	.525*
Use of 3% saline infusion	15	44.1	7	43.8	.981*
Timing of hyponatremia					
On admission	26	76.5	14	87.5	.468*
After admission	8	23.5	2	12.5	
Hyponatremia type					
Hypovolemic	2	5.9	1	6.3	.889**
Euvolemic	17	50	9	56.3	
Hypervolemic	15	44.1	6	37.5	

*Fisher's exact test, and **Fisher-Freeman-Halton Exact Tests

Table **(4)** shows no statistically significant association between age, IVC Diameter, GCS, FOUR Score and shows a statistically significantly higher SOFA score in deceased vs. survivors. There was a statistically significantly higher triglyceride level in deceased vs. survivors, and a marginally significantly higher AST and lower platelet count in deceased vs. survivors.

Table (4): Comparisons of worse and favorable outcomes regarding age, IVC Diameter, clinical scores and laboratory tests

	Survivors Median	Q1-Q3	Deceased Median	Q1-Q3	Sig
Age (years)	67	58.8-73.3	65	57.8-74.8	.909
IVC diameter (cm)	2	1.8-2.3	2	2-2.3	.824
GCS	15	13.8-15	14.5	14-15	.826
FOUR score	16	15.8-16	16	9.5-16	.118
SOFA score	7	4-9	12	10-13.8	<.001
Laboratory tests					
Serum sodium (mmol/L)	126	119.5-128.3	123	121-128.8	.992
Fasting blood glucose (mg/dl)	99	90-111	98.5	90-110	.925
Corrected Sodium	126	119.5-128.3	123.5	121-128	.958
Corrected Sodium	126	119.5-128.3	123.5	121-128	.975
Serum osmolality	266.1	258.9-282.3	272	265-281.3	.170
Urinary sodium	27	20.5-51.3	26.5	21.5-37	.499
WBC count	8.7	7-16.3	10.3	8-14	,499
Haemoglobin (g/dl)	9.5	8.2-10.6	10	8.9-10.9	.392
Platelet count	148	95.8-214.3	98	43-154.8	.072
Serum potassium (mmol/L)	3.8	3.3-4.9	4.1	3.5-4.9	.595
Serum creatinine (mg/dl)	1.5	.8-2.1	2	1.2-3	.166
Serum uric acid (mg/dl)	7	4.9-8.3	7.4	5.6-11.4	.358
Triglycerides (mg/dl)	112.5	86-162.5	170	122.5-180	.030
Serum total cholesterol (mg/dl)	202.5	151.5-242.5	221	192.5-250	.387
Serum albumin (g/dl)	2.6	2-3	2.5	2.3-2.9	.587
AST (IU/L)	51.5	31.5-74.5	64.5	50.3-111.5	.094
ALT (IU/L)	28.5	18-50.3	33	25.5-51	.333
AST/ALT ratio	1.7	1.2-2.4	1.9	1.4-2.8	.220
Serum total bilirubin (mg/dl)	1.9	.7-10.6	4	1.3-10.7	.293
Serum direct bilirubin (mg/dl)	1.2	.3-8.5	2.6	.8-8.8	.359
Direct bilirubin/totalbilirubin (%)	62.5	40-77.6	67.1	60-82.2	.441

Notes: AST: Aspartate transaminase, ALT: Alanine transaminase GCS = Glasgow Coma Scale. IVC: Inferior vena cava, FOUR score = Full Outline of Unresponsiveness. Sig. = statistical significance (p-value). The test of significance is chi-square, *Fisher's exact test, and **Fisher-Freeman-Halton Exact Tests for categorical data and Mann-Whitney U-test for numerical data.

Table **(5)** displays the results of binary logistic regression analysis run to demine the effects of male sex, serum sodium ≤ 118 mmol/L, SOFA score > 9, platelet count ≤ 57 per mm^3, and AST > 48 IU/L on the likelihood that participants will exhibit ICU mortality. On univariate analysis, three predictors were statistically significant which are SOFA score > 9, platelet count ≤ 57 per mm3, and AST > 48 IU/L. On multivariate analysis, only one was statistically significant independent predictor which is SOFA score > 9. The mode waters statistically significant ($\chi 2$ [5] = 30.022, p<.001). The model correctly classified 88% of cases, with 87.5% sensitivity (Sn), and 88.2% specificity (Sp).

Table (5): Predictors of ICU mortality

Predictor	**Univariate**				**Multivariate**			
	Sig.	**COR**	**95% CI**		**Sig.**	**AOR**	**95% CI**	
			Lower	**Upper**			**Lower**	**Upper**
Male sex	.113	2.692	.790	9.173	.496	1.864	.311	11.170
S. sodium ≤ 118 mmol/L	.168	0.217	.025	1.905	.133	.104	.005	1.988
SOFA score > 9	<.001	27.00	4.938	147.630	.002	22.338	3.136	159.098
Platelet count ≤ 57 per mm^3	.016	5.833	1.387	24.539	.062	8.922	.893	89.136
AST > 48 IU / L	.028	6.222	1.222	31.677	.173	4.977	.495	50.076

Notes: Sig. = statistical significance (p-value). COR = crude odds ratio. AOR = adjusted odds ratio. CI = confidence interval.

Figure **(1)** shows the performance of serum sodium in predicting ICU mortality. A cutoff value ≤ 118 mmol/L has an area under the curve (AUC) (95% CI) of 0.501 (0.356-0.646), p = .992, with 6.25% sensitivity and 76.5% specificity.

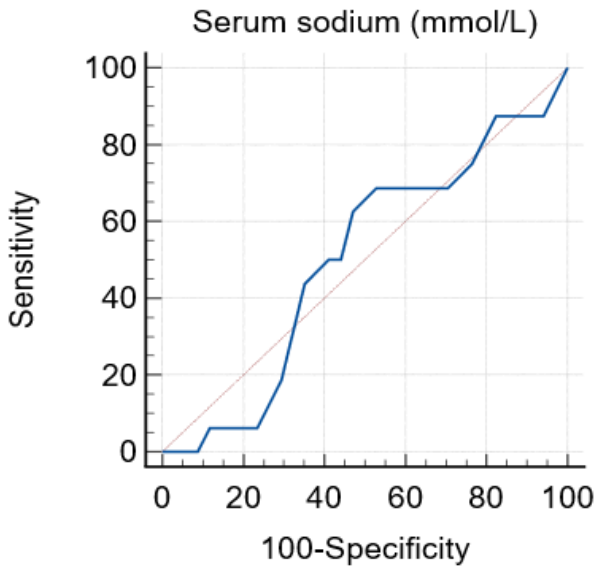

Figure (1): ROC curve for serum sodium as a predictor of ICU mortality.

Figure (2) shows the performance of SOFA score in predicting ICU mortality. A cutoff value > 9 has an AUC (95% CI) of 0.889 (0.764-0.958), p <.001, with 87.5% sensitivity and 79.4% specificity.

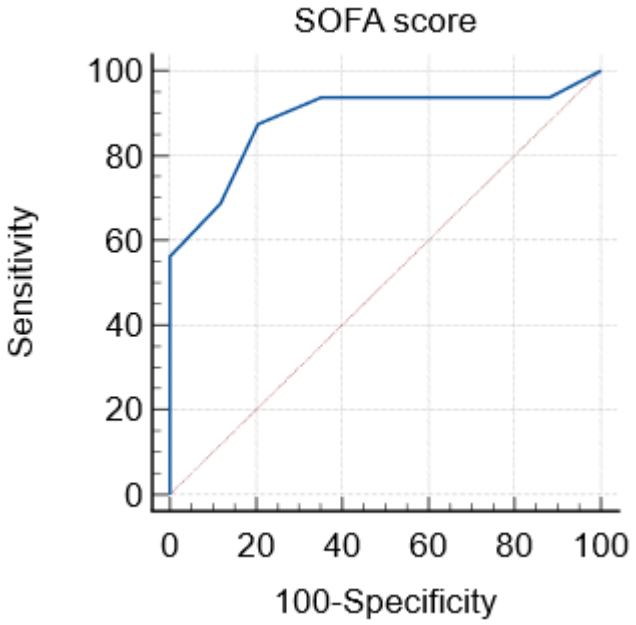

Figure (2): ROC curve for SOFA score as a predictor of ICU mortality.

DISCUSSION

Hyponatremia, defined as a serum sodium (Na^+) level below 135 mmol/L, is the most frequently encountered electrolyte abnormality. It is particularly prevalent among critically ill patients, with reported incidence rates of up to 30% in intensive care units (ICUs). Despite its high frequency, hyponatremia is often underdiagnosed or underestimated, even though it is closely linked to increased morbidity, prolonged hospital stays, elevated healthcare costs, and higher mortality rates [8].

Mild or slowly developing hyponatremia is often asymptomatic, or may be presented by vague symptoms such as headache, nausea, fatigue, or cognitive impairment. However, when serum sodium drops rapidly and severely, the consequences can be life-threatening. These include fits, coma, irreversible neurological insult, respiratory arrest, brainstem herniation, and death. In cases of chronic or slowly progressive hyponatremia, the brain has time to adapt by regulating osmotic pressure, which may reduce symptom severity even when sodium levels are critically low. Nevertheless, this compensatory mechanism is not always protective, especially in the presence of additional risk factors [9].

Several comorbid conditions and metabolic disturbances can exacerbate the clinical effects of hyponatremia. These include acidosis, hypoxia, potassium depletion, hepatic failure, renal dysfunction, and malnutrition. Each of these factors can worsen the severity of symptoms and increase the risk of complications. Furthermore, they often coexist in the ICU population, making the management of hyponatremia more complex and nuanced [9].

This study aimed to ascertain causes and outcomes (ICU stay duration and mortality) of hyponatremia and management in critically ill cases in ICU. The current study conducted at Faculty of Medicine, Mansoura University, performed on 50 patients with hyponatremia with serum above 18 years old. Our findings showed that females represented a slightly higher proportion of the cohort (54%) compared to males (46%). In terms of occupation, housewives constituted the largest group (46%), followed by manual workers (24%), farmers (20%), and employees (10%). Notably, 44% of patients were found to be using diuretics, indicating a significant potential contributing factor to the development of hyponatremia among this population.

Al Yaqoubi *et al.* [10] found that most patients admitted with hyponatremia were elderly and had multiple comorbidities, including hypertension and diabetes. This demographic overlap with housewives, who often have multiple health issues and may be more likely to use diuretics, suggests a potential link between occupation and the risk of hyponatremia.

Also, **Hendriksen *et al.*** [11] confirmed whether these sex differences in hyponatremia-related hospital admissions in diuretic users are still following adjusting for multiple confounding variables which include age, dose, and concomitant medications. They found that women using diuretics had a significantly greater risk

of hospital admission due to hyponatremia compared to men.

The median age of the studied patients was 66 years (IQR: 58.8–74), indicating a predominantly elderly population. The IVC diameter was within the normal range, with a median of 2 cm (1.8–2.2), suggesting relatively preserved volume status in most cases. Neurological assessments showed that patients had high levels of consciousness at presentation, with a median GCS of 15 and a FOUR score of 16, both indicating minimal to no neurological compromise. However, the SOFA score, which had a median of 9 (IQR: 5.8–11), reflects moderate to severe organ dysfunction in many cases.

The median serum sodium level was 125 mmol/L (120–128.2), confirming moderate hyponatremia across the cohort. Both corrected sodium values (by Katz and Hillier formulas) aligned closely with the measured sodium, suggesting no significant pseudohyponatremia due to hyperglycemia (median fasting glucose: 98.5 mg/dL). Serum osmolality was also low (median: 268.8 mOsm/kg), indicating true hypotonic hyponatremia. Urinary sodium had a median of 27 mmol/L, with a range supportive of either hypovolemic or euvolemic hyponatremia depending on the clinical context. Hematological parameters revealed mild anemia (hemoglobin: 9.7 g/dL) and borderline thrombocytopenia (platelet count: 130.5 $x10^3/\mu L$). White blood cell count was within normal limits. Electrolyte levels such as serum potassium (3.9 mmol/L) and serum creatinine (1.6 mg/dL) suggest mild renal impairment or hypoperfusion in some cases. Liver function parameters showed moderate hypoalbuminemia (serum albumin: 2.6 g/dL) and elevated AST (median: 54.5 IU/L), with a preserved ALT level, yielding an AST/ALT ratio >1, which may suggest non-alcoholic liver stress or systemic inflammation. The direct-to-total bilirubin ratio was elevated (66%), suggesting possible cholestasis or hepatocellular injury. Lipid profile and metabolic markers including triglycerides (130 mg/dL) and total cholesterol (206.5 mg/dL) were within or slightly above normal ranges.

Literature supported our findings of preserved neurological function yet significant organ dysfunction in hyponatremic ICU patients; for example, SOFA scores were reported to be significantly higher in hyponatremic patients compared to normonatremic ones, despite otherwise similar demographic characteristics, including age (median in the mid-60s) [12]. Additionally, ultrasonographic measurement of the IVC has been shown to effectively differentiate between volume states in hypotonic hyponatremia supporting our interpretation of IVC diameter as reflective of volume status [13].

Hyponatremic patients demonstrated significantly higher SOFA scores, reflecting greater organ dysfunction, as reported by **Sim *et al.*** [12]. Additionally, this study noted significant differences in serum potassium and creatinine levels between hyponatremic and non-hyponatremic groups, suggesting altered renal function or electrolyte imbalance associated with hyponatremia.

In our study, although a higher ratio of deceased patients had severe hyponatremia (50%) compared to survivors (44.1%), the difference in outcomes across severity levels was not statistically significant ($p = 0.876$). This suggests that while severe hyponatremia was more common among non-survivors, severity alone did not significantly influence mortality in our patient population.

Singh *et al.* [14] did a prospective cross-sectional study of 375 adult hyponatremic patients found that the severity of hyponatremia didn't significantly influence outcomes, Correction of hyponatremia was significantly accompanied by better outcomes ($p = 0.003$). Also, **Farah *et al.*** [15] found that there was no significant difference in 6-month mortality between moderate and severe hyponatremia groups (11.1% vs. 16.2%, $p = 0.163$).

According to comparison of worse and favorable outcomes, our result showed a statistically significantly higher SOFA score and triglyceride level in deceased vs. survivors, and a marginally significantly higher AST and lower platelet count in deceased vs. survivors.

In agreement with our results, **Sipahioglu** & **Bahcebası** [16] found that in critically ill geriatric patients with sepsis, the SOFA score was the only independent predictor of mortality, with an odds ratio of 1.886 with p value < 0.001. Also, agreed with **Zheng *et al.*** [17] who explored the correlation between the triglyceride-glucose (TyG) index and the risk of in-hospital mortality in critically-ill cases with sepsis. They showed that higher AST and lower platelet count compared to survivors (both $P < 0.05$), in addition to elevated SOFA scores.

In contrast with **Huang** & **Sun** [18] who examined how triglyceride (TG) levels relate to 30-day ICU mortality, they found that patients with TG levels in the second quartile (74–103 mg/dL) had a higher risk of death. The findings suggest a non-linear relationship between TG levels and prognosis in critically ill septic patients.

Concerning ROC curve for SOFA score as a predictor of ICU mortality, this study showed the performance of SOFA score in predicting ICU mortality. A cutoff value > 9 has an AUC (95% CI) of 0.889 (0.764-0.958), p <.001, with 87.5% sensitivity and 79.4% specificity.

In the same line, **Do *et al.*** [19] conducted a multicenter study across 15 intensive care units in Vietnam demonstrated that the Sequential Organ Failure Assessment (SOFA) score is a useful predictor of ICU mortality in critically ill patients. They reported an AUC of 0.713 $p < 0.001$. An optimal SOFA score cutoff of ≥9.5 was identified, which yielded a Sn of 53.6% and a Sp of 80.1%, suggesting that higher SOFA scores are accompanied by increased mortality risk and

that the score can be effectively used for early risk stratification in ICU settings.

Also, **Gershengorn *et al.*** [20] conducted in Vietnamese ICUs assessed the predictive performance of the SOFA score for ICU mortality. They revealed an AUC of 0.713 $p < 0.001$, indicating a fair discriminatory ability. The optimal cutoff value for SOFA was identified as ≥9.5, with a Sn of 53.6% and Sp of 80.1%.

Concerning ROC curve for serum sodium as a predictor of ICU mortality, the current study showed the performance of serum sodium in predicting ICU mortality. A cutoff value ≤ 118 mmol/L has an AUC (95% CI) of 0.501 (0.356-0.646), p = .992, with 6.25% sensitivity and 76.5% specificity.

In supporting with **Marahrens *et al.*** [21] who evaluated whether serum Na, measured at various time points throughout the in-hospital treatment period, offered prognostic data in AKI. They found that sensitivity and specificity around 60%. Also, **Huang *et al***. [22] who assessed the correlation between Na trajectories in cases with AKI and mortality at 30-days reported that serum sodium was significantly accompanied by mortality in cases with acute kidney injury.

In contrast with **Liu *et al.*** [23] who explored the correlation between hypernatremia and 28-day mortality in elderly sepsis cases. They showed that the optimal cutoff was 154.9 mmol/L, offering 73.2% sensitivity. However, they agree with our findings in specificity that they were 78.1%.

CONCLUSION

This study demonstrates that SOFA score is the only independent predictor of ICU mortality, highlighting the importance of overall organ dysfunction over sodium level alone in outcome prediction.

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