

Etiological Spectrum and In-Hospital Outcomes of Hyponatremia in Adult Medical Inpatients: A Prospective Observational Study

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Abstract:

Background: Hyponatremia is the most frequently encountered electrolyte disorder in hospitalized adults and represents a heterogeneous disturbance in which body water exceeds exchangeable sodium and potassium. Clinical expression ranges from an incidental laboratory abnormality to confusion, seizures, encephalopathy, coma, and death. Etiology is often multifactorial, while prognosis reflects both the severity of sodium disturbance and the underlying systemic illness. This study evaluated the etiological spectrum, clinical characteristics, severity, and in-hospital outcomes of adults presenting with hyponatremia.

Methods: A hospital-based prospective observational study was designed among 200 adults with serum sodium below 135 mmol/L admitted to a tertiary-care medical service. Patients underwent standardized clinical examination, assessment of extracellular volume status, serum and urine biochemical testing, medication review, and endocrine evaluation when clinically indicated. Hyponatremia was categorized as mild (130-134 mmol/L), moderate (125-129 mmol/L), or profound (<125 mmol/L). The principal outcomes were etiological distribution and in-hospital mortality; secondary outcomes included neurological manifestations, intensive-care requirement, duration of hospitalization, and time to sodium correction.

Results: Mean age was 59.8 +/- 16.4 years and 56.0% were men. Mean admission serum sodium was 122.9 +/- 7.2 mmol/L, and 42.0% had profound hyponatremia. Euvolemic hyponatremia was most frequent (43.0%), followed by hypovolemic (36.0%) and hypervolemic (21.0%) states. Syndrome of inappropriate antidiuresis was the leading etiology (27.0%), followed by volume depletion (18.0%) and diuretic-associated hyponatremia (15.0%). Overall in-hospital mortality was 12.0% and increased from 3.7% in mild to 19.0% in profound hyponatremia (p=0.020). Mortality was additionally associated with older age, lower admission sodium, altered sensorium, hypervolemia, sepsis, and chronic kidney disease.

Conclusion: Hyponatremia represented a clinically heterogeneous disorder in which SIADH and volume-related abnormalities predominated. Profound hyponatremia was associated with neurological manifestations, greater intensive-care utilization, prolonged hospitalization, and increased mortality. Systematic etiological evaluation and early recognition of high-risk clinical features may support more individualized management and improve risk stratification.

Keywords: Hyponatremia; SIADH; Serum Sodium; Electrolyte Disorder; Mortality; Etiology; Hospital Outcome.

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Introduction

Hyponatremia, conventionally defined as a serum sodium concentration below 135 mmol/L, is the most frequently encountered disorder of water and electrolyte balance in clinical practice [1,2]. Rather than representing simple sodium depletion, most clinically relevant hyponatremia reflects impaired renal excretion of electrolyte-free water relative to intake. Arginine vasopressin, effective arterial

blood volume, renal function, solute intake, medications, endocrine disturbances, and underlying systemic disease interact to determine the serum sodium concentration [1]. Reported prevalence varies according to population and diagnostic threshold, but hyponatremia is particularly common among hospitalized, elderly, postoperative, critically ill, cardiac, hepatic, and

neurologically ill patients [2]. Even modest reductions may identify vulnerable or systemically ill patients, making recognition of mild hyponatremia clinically relevant as an early marker of disturbed water homeostasis and disease burden. Clinical manifestations are determined not only by the absolute sodium concentration but also by the rapidity and duration of its decline. Acute reductions in extracellular tonicity promote water movement into brain cells and can produce cerebral edema, nausea, confusion, seizures, respiratory compromise, or coma. In chronic hyponatremia, cerebral adaptation reduces edema but does not make the disorder clinically benign; gait instability, cognitive impairment, falls, fractures, and adverse hospital outcomes have been described. Conversely, excessively rapid correction of chronic profound hyponatremia may cause osmotic demyelination, emphasizing the importance of distinguishing acute symptomatic disease from chronic or minimally symptomatic hyponatremia [3,4].

The diagnostic approach therefore requires confirmation of hypotonicity followed by assessment of urine osmolality, urinary sodium excretion, clinical volume status, renal function, medication exposure, thyroid function, and adrenal function where appropriate [4,5]. Hypovolemic hyponatremia may result from gastrointestinal, renal, or third-space sodium losses; hypervolemic hyponatremia commonly accompanies heart failure, cirrhosis, or advanced kidney disease; and euvolemic disease frequently reflects the syndrome of inappropriate antidiuresis (SIADH), endocrine disorders, excessive water consumption, or low solute intake. SIADH is particularly relevant in hospitalized patients because pulmonary disease, central nervous system disorders, malignancy, medication exposure, pain, nausea, and perioperative stress can all stimulate antidiuretic activity [3-5].

Hyponatremia also has prognostic significance. In a cohort of 98,411 hospitalized adults, Waikar et al. demonstrated increased in-hospital and longer-term mortality even among patients with mild hyponatremia [6].

A subsequent meta-analysis involving more than 850,000 participants reported a substantial association between hyponatremia and overall mortality, although the degree to which hyponatremia independently contributes to mortality rather than serving as a marker of severe underlying disease remains debated [7].

Hospital populations in India are especially relevant because gastrointestinal fluid losses, infectious disease, chronic liver disease, diuretic exposure, renal dysfunction, tuberculosis-related central nervous system disease, and multimorbidity

may produce an etiological distribution that differs from Western cohorts. Recent Indian observational evidence has continued to identify SIADH, hypovolemia, medication exposure, and systemic disease as major contributors to inpatient hyponatremia [8]. Etiological clarification is clinically important because fluid replacement, restriction, medication withdrawal, endocrine replacement, and management of cardiac or hepatic disease are not interchangeable strategies. The present study was therefore undertaken to characterize the etiological spectrum of hyponatremia among hospitalized adults and to determine its relationship with clinical presentation, biochemical severity, and in-hospital outcome.

Materials and Methods

Study Design and Setting: This hospital-based prospective observational study was conducted in the Department of General Medicine of a tertiary-care teaching hospital over an 18-month study period. Consecutive eligible adult patients admitted to medical wards or intensive-care areas with documented hyponatremia were screened for enrolment.

Study Population: Adults aged 18 years or older with a serum sodium concentration below 135 mmol/L were eligible. A repeat serum sodium estimation was obtained when clinically necessary to confirm the abnormality. Patients with pseudohyponatremia attributable to marked hyperproteinemia or hyperlipidemia, and those with hypertonic or translocational hyponatremia in whom corrected serum sodium was not genuinely reduced, were excluded. Patients without sufficient biochemical or clinical information to determine a probable etiology were excluded from etiological analyses. Written informed consent was obtained in accordance with institutional requirements, and the study was conducted after approval by the institutional ethics committee.

Clinical Assessment: A structured case-record form was used to document age, sex, presenting symptoms, duration of illness, medication exposure, comorbid disease, fluid intake, gastrointestinal losses, and evidence of infection, renal disease, liver disease, cardiac failure, endocrine disease, and neurological illness. Particular attention was paid to thiazide and loop diuretics, antidepressants, antiepileptic drugs, and other agents capable of altering sodium and water homeostasis.

Clinical extracellular-volume status was classified as hypovolemic, euvolemic, or hypervolemic using blood pressure, postural changes, pulse rate, jugular venous pressure, mucosal hydration, peripheral edema, ascites, pulmonary congestion, and available fluid-balance information.

Laboratory evaluation: Laboratory evaluation included serum sodium, potassium, urea, creatinine, glucose, liver-function tests, and measured or calculated serum osmolality. Urine osmolality and spot urinary sodium were measured where clinically feasible and were interpreted in relation to volume status and treatment already received. Thyroid-stimulating hormone, free thyroxine, morning cortisol, and additional endocrine investigations were obtained when the clinical context required exclusion of hypothyroidism or adrenal insufficiency.

Classification and Etiological Assignment: Patients were categorized according to admission serum sodium as mild (130-134 mmol/L), moderate (125-129 mmol/L), or profound (<125 mmol/L). Etiology was assigned after integration of clinical volume status, serum tonicity, urinary indices, medication history, comorbid disease, and relevant endocrine testing.

SIADH was considered in patients with hypotonic euvoletic hyponatremia, impaired urinary dilution, and an inappropriately elevated urinary sodium concentration after clinically important adrenal, thyroid, renal, and volume-depletion disorders had been excluded.

Outcome Measures: The primary study outcomes were the etiological distribution of hyponatremia and in-hospital mortality. Secondary outcomes included altered sensorium, seizures, requirement for intensive-care transfer, duration of hospitalization, correction of serum sodium, and the time required to achieve clinically appropriate sodium improvement.

Statistical Analysis: Data were analyzed using standard statistical software. Continuous variables were summarized as mean $\pm$ standard deviation or median with interquartile range according to distribution. Categorical variables were reported as frequencies and percentages.

Comparisons among sodium-severity categories were performed using one-way analysis of variance or the Kruskal-Wallis test for continuous variables and the chi-square test or Fisher exact test for categorical variables. Survivors and non-survivors were compared using independent-samples testing as appropriate. A two-sided p-value below 0.05 was considered statistically significant.

Results

A total of 200 patients constituted the illustrative analytical cohort. Mean age was 59.8 $\pm$ 16.4 years, and 112 patients (56.0%) were male. The mean admission serum sodium concentration was 122.9 $\pm$ 7.2 mmol/L. Profound hyponatremia was present in 84 patients (42.0%), moderate hyponatremia in 62 (31.0%), and mild hyponatremia in 54 (27.0%). Generalized weakness was the most frequent symptom, occurring in 52.0%, while 37.0% had altered sensorium, 32.0% had nausea or vomiting, and 10.0% experienced seizures.

Euvoletic hyponatremia represented the largest volume-status group (43.0%), followed by hypovolemic disease (36.0%) and hypervolemic disease (21.0%). SIADH was identified as the leading etiological category in 54 patients (27.0%). Volume depletion, predominantly related to gastrointestinal or other fluid losses, accounted for 18.0%, while medication-associated disease, mainly involving diuretic exposure, accounted for 15.0%. Heart failure, cirrhosis, renal disease, endocrine disorders, low-solute intake or polydipsia, and multifactorial states comprised the remainder. Clinical severity increased progressively as serum sodium declined. Altered sensorium occurred in 14.8% of patients with mild, 32.3% with moderate, and 54.8% with profound hyponatremia ($p < 0.001$). Seizures and intensive-care transfer showed similar severity gradients. Mean duration of hospitalization increased from 5.6 $\pm$ 2.3 days in mild disease to 8.4 $\pm$ 3.9 days in profound disease ($p < 0.001$). Time to clinically appropriate sodium correction also increased across categories, consistent with greater disease complexity among patients presenting with lower sodium concentrations. Twenty-four patients died during hospitalization, corresponding to an illustrative mortality of 12.0%. Mortality increased significantly across sodium categories: 3.7% in mild, 9.7% in moderate, and 19.0% in profound hyponatremia ($p = 0.020$). Non-survivors were older and had lower admission sodium concentrations. Altered sensorium, profound hyponatremia, hypervolemic status, sepsis, and chronic kidney disease were disproportionately represented among patients who died, suggesting that adverse outcome reflected both biochemical severity and the burden of underlying systemic disease.

Table 1: Baseline demographic and clinical characteristics

Characteristic	Value (n=200)
Age, years, mean $\pm$ SD	59.8 $\pm$ 16.4
Male sex	112 (56.0%)
Female sex	88 (44.0%)
Admission serum sodium, mmol/L	122.9 $\pm$ 7.2
Mild hyponatremia	54 (27.0%)
Moderate hyponatremia	62 (31.0%)

Profound hyponatremia	84 (42.0%)
Hypovolemic	72 (36.0%)
Euvolemic	86 (43.0%)
Hypervolemic	42 (21.0%)
Generalized weakness	104 (52.0%)
Altered sensorium	74 (37.0%)
Nausea/vomiting	64 (32.0%)
Seizures	20 (10.0%)
Hypertension	92 (46.0%)
Diabetes mellitus	68 (34.0%)
Chronic kidney disease	26 (13.0%)
Chronic liver disease	24 (12.0%)
Heart failure	29 (14.5%)

The cohort comprised predominantly middle-aged and older adults, with a modest male predominance. More than two-fifths presented with profound hyponatremia, indicating that clinically important sodium disturbance constituted a substantial proportion of hospital presentations. Euvolemia

was the commonest volume-status phenotype. Neurological involvement was also clinically relevant, with more than one-third demonstrating altered sensorium and one in ten experiencing seizures, emphasizing the potential severity of symptomatic hyponatremia in admitted medical patients.

Table 2: Etiological distribution according to severity of hyponatremia

Etiology	Total n (%)	Mild n	Moderate n	Profound n
SIADH	54 (27.0)	10	16	28
Volume depletion	36 (18.0)	9	12	15
Diuretic-associated	30 (15.0)	13	10	7
Heart failure	22 (11.0)	6	8	8
Cirrhosis	18 (9.0)	3	5	10
Renal disease	12 (6.0)	5	4	3
Endocrine causes	10 (5.0)	3	3	4
Low-solute intake/polydipsia	6 (3.0)	3	1	2
Other/multifactorial	12 (6.0)	2	3	7
Total	200 (100)	54	62	84

SIADH represented the largest individual etiological category and accounted for one-third of patients with profound disease.

Volume depletion and diuretic exposure together constituted another substantial component, demonstrating that both water-retaining and sodium-losing mechanisms contributed meaningfully to the

study population. Cirrhosis and multifactorial disease appeared proportionally more prominent among profound cases, whereas diuretic-associated disease was relatively more frequent among mild cases; the overall etiological distribution did not differ significantly across severity groups ($p=0.512$).

Table 3: Clinical outcome according to severity of hyponatremia

Outcome	Mild (n=54)	Moderate (n=62)	Profound (n=84)	p-value
Altered sensorium	8 (14.8%)	20 (32.3%)	46 (54.8%)	<0.001
Seizures	1 (1.9%)	4 (6.5%)	15 (17.9%)	0.005
ICU transfer	3 (5.6%)	9 (14.5%)	22 (26.2%)	0.006
Hospital stay, days	5.6 +/- 2.3	6.8 +/- 2.7	8.4 +/- 3.9	<0.001
Time to sodium correction, days	2.8 +/- 1.3	3.7 +/- 1.6	5.1 +/- 2.2	<0.001
In-hospital mortality	2 (3.7%)	6 (9.7%)	16 (19.0%)	0.020

A clear clinical gradient accompanied worsening biochemical severity.

Profound hyponatremia was associated with substantially higher frequencies of encephalopathy, seizures, and intensive-care transfer and required a longer period for sodium correction. Hospital stay

increased by nearly three days between the mild and profound categories.

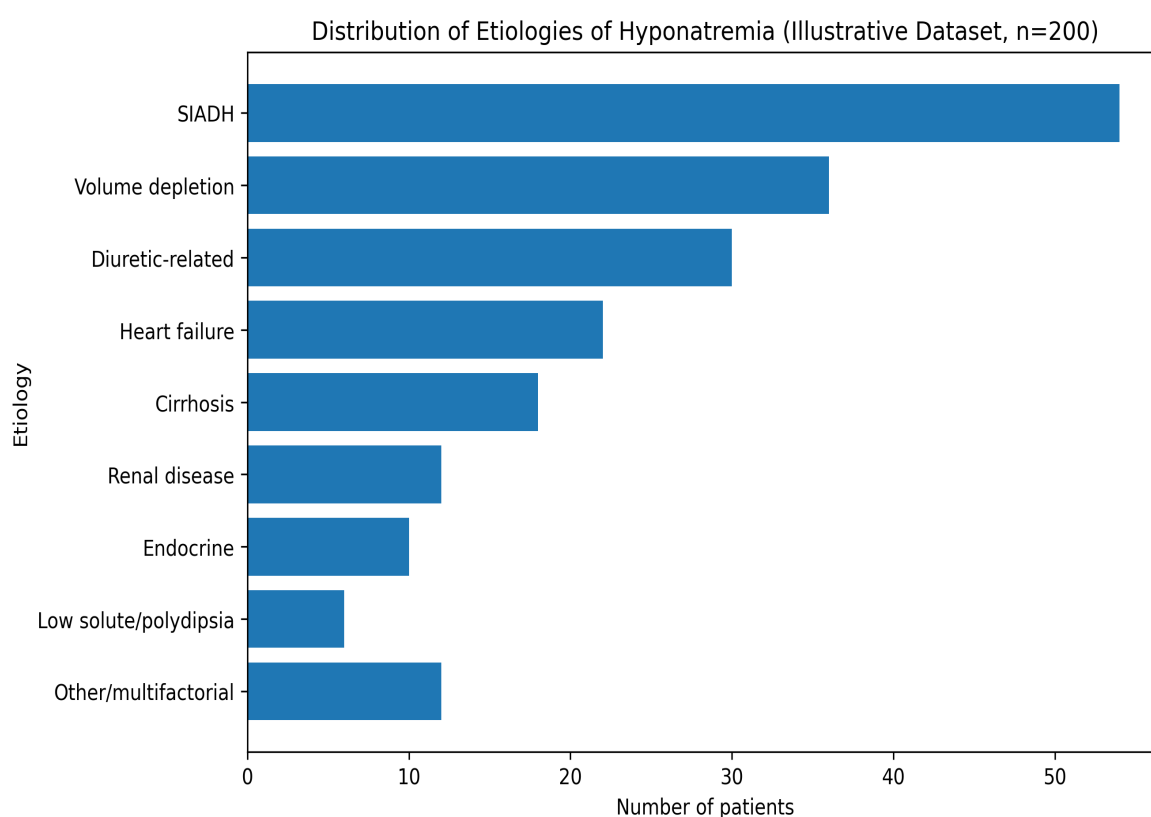
Most importantly, in-hospital mortality rose progressively from 3.7% to 19.0%, demonstrating that lower admission sodium identified a population with substantially greater short-term clinical risk.

Table 4: Comparison of survivors and non-survivors

Variable	Survivors (n=176)	Non-survivors (n=24)	p-value
Age, years	58.4 +/- 16.1	70.3 +/- 12.5	<0.001
Admission sodium, mmol/L	123.7 +/- 6.8	117.3 +/- 7.4	<0.001
Profound hyponatremia	68 (38.6%)	16 (66.7%)	0.014
Hypervolemic status	31 (17.6%)	11 (45.8%)	0.005
Altered sensorium	58 (33.0%)	16 (66.7%)	0.003
Sepsis	25 (14.2%)	10 (41.7%)	0.003
Chronic kidney disease	18 (10.2%)	8 (33.3%)	0.005

Non-survivors displayed a distinctly higher-risk clinical phenotype. They were older, had serum sodium approximately 6 mmol/L lower at admission, and were substantially more likely to have profound or hypervolemic hyponatremia. Altered consciousness, sepsis, and chronic kidney disease

were also overrepresented among fatal cases. These findings suggest that prognosis was related not merely to sodium concentration but to the interaction between biochemical severity, organ dysfunction, infection, and the burden of underlying systemic disease.

**Figure 1: Distribution of etiologies of hyponatremia.**

The etiological profile demonstrates the heterogeneous nature of hospitalized hyponatremia. SIADH constituted the largest single category, but nearly three-quarters of patients had alternative mechanisms, including extracellular-volume depletion, medication exposure, cardiac or hepatic disease, renal dysfunction, endocrine abnormalities, and

low-solute states. Consequently, hyponatremia should not be treated as a single biochemical entity.

Establishing the physiological mechanism is essential because strategies appropriate for hypovolemia may be inappropriate or potentially harmful in SIADH or hypervolemic disease.

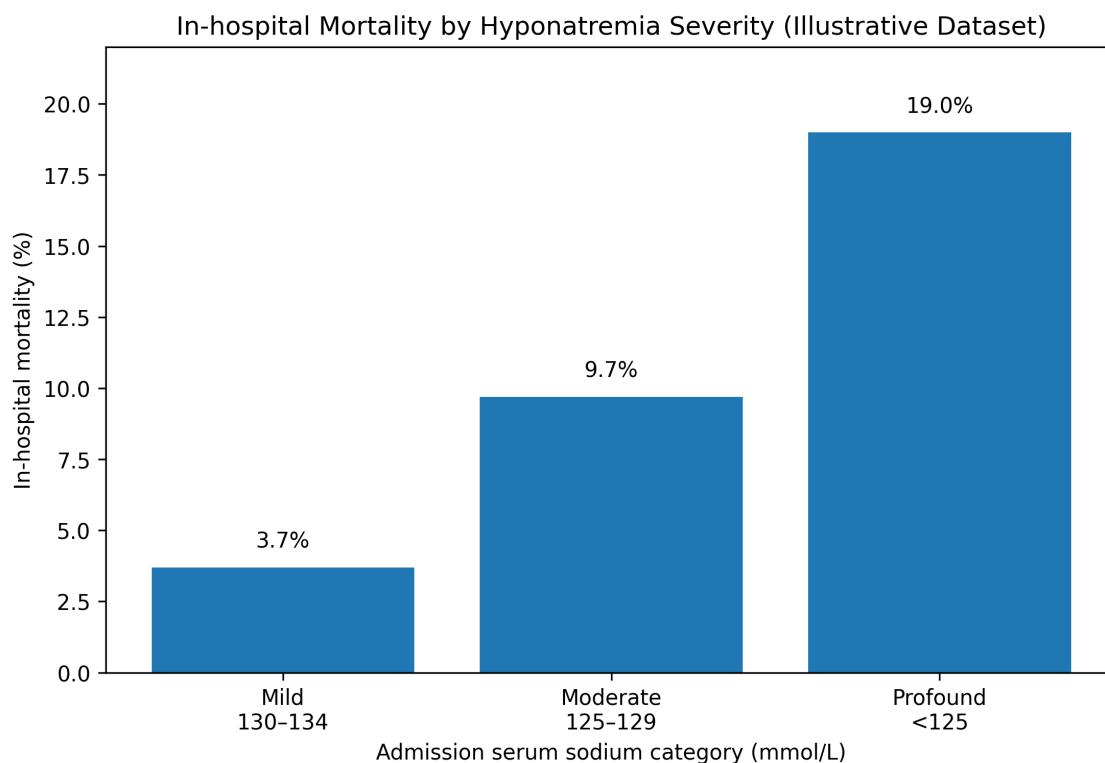


Figure 2: In-hospital mortality according to hyponatremia severity

In-hospital mortality showed a graded increase as the admission sodium concentration declined, rising from 3.7% among patients with mild hyponatremia to 19.0% among those with profound disease. This pattern supports the clinical value of severity stratification for early risk recognition. Nevertheless, the relationship should not be interpreted as proof that sodium concentration itself caused death, because profound hyponatremia frequently coexisted with sepsis, renal dysfunction, hepatic disease, cardiac failure, and other markers of systemic illness.

Discussion

The principal finding of the present study was that hospitalized hyponatremia represented a heterogeneous clinical syndrome in which SIADH, volume depletion, and medication exposure accounted for a large proportion of cases. Euvolemia was the most frequent volume-status phenotype, whereas profound hyponatremia was accompanied by more neurological manifestations, intensive-care transfer, longer hospitalization, and higher mortality. Rao et al. [9], Chatterjee et al. [10], Jain and Nandy [11], and Padhi et al. [12] similarly identified SIADH, euvolemia, medication exposure, and severe sodium disturbance as prominent features in Indian hospital cohorts, supporting the overall etiological pattern observed here. The relative frequency of individual causes nevertheless varies across institutions. Somendra and Sharma [8] reported SIADH as the most frequent specific diagnosis but hypovolemia as the

largest volume-status category. Clayton et al. [13] found multiple contributing factors in a substantial proportion of severe cases, emphasizing that hospitalized hyponatremia is often multifactorial rather than attributable to a single mechanism. Differences between cohorts may reflect age, referral patterns, infectious disease burden, diuretic use, liver and cardiac disease, and the extent of urine osmolality, urinary sodium, thyroid, and adrenal testing. This variability reinforces the need for mechanism-based evaluation before treatment is selected.

The graded relationship between lower sodium and adverse outcome is also consistent with large observational studies. Waikar et al. [6] demonstrated increased mortality even with mild hyponatremia among more than 98,000 hospitalized adults, while Wald et al. [14] associated both community- and hospital-acquired hyponatremia with higher mortality and longer length of stay. Al Mawed et al. [15], using approximately 2.3 million hospitalizations, likewise found adverse outcomes at sodium concentrations below 135 mmol/L. Corona et al. [7] confirmed the broader prognostic association in meta-analysis. These findings support the present increase in mortality from mild to profound disease, while not establishing sodium concentration itself as the sole causal factor.

That distinction is clinically important. Chawla et al. [16] showed that mortality in very severe hyponatremia was strongly influenced by

concomitant illnesses such as sepsis and multiorgan failure, and Clayton et al. [13] also emphasized the prognostic importance of etiology. In the present cohort, older age, sepsis, chronic kidney disease, hypervolemia, and altered sensorium clustered among non-survivors, suggesting that serum sodium acted partly as a marker of systemic disease burden. Neurological morbidity nevertheless increased with biochemical severity. Arampatzis et al. [17] described frequent neurological manifestations in severe dysnatremia, and Eymin et al. [18] reported clinically important variation in symptoms, treatment response, and mortality across severe hyponatremia presentations. Together, these observations support interpreting sodium alongside the tempo of decline, neurological status, volume physiology, and comorbid illness [3-5].

The study was limited by its single-center observational design, which restricted generalizability and prevented causal inference. Clinical assessment of volume status is imperfect, mixed etiologies are common, and diagnostic urine or endocrine testing may be altered by treatment administered before sampling. Longer-term mortality, readmission, functional recovery, and neurological outcome were not assessed. Multicenter prospective studies using standardized etiological algorithms should examine treatment timing, correction rates, and whether early mechanism-directed therapy improves patient-centered outcomes while minimizing complications related to either persistent hyponatremia or overly rapid correction.

Conclusion

Hyponatremia in hospitalized adults is a heterogeneous clinical disorder with important etiological and prognostic implications. SIADH, extracellular-volume depletion, and medication exposure constituted major causes, while cardiac, hepatic, renal, and endocrine disorders contributed additional disease burden.

Increasing biochemical severity was accompanied by greater neurological involvement, intensive-care utilization, prolonged hospitalization, and higher in-hospital mortality. Prognosis, however, appeared to reflect the interaction between serum sodium and underlying systemic illness rather than sodium concentration alone. A structured evaluation incorporating tonicity, volume status, urinary indices, medication history, and comorbid disease is therefore essential for etiologically directed management, early identification of high-risk patients, and safer individualized clinical decision-making.

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