Section: General Medicine

Original Research Article

CLINICAL PROFILE AND OUTCOMES OF HOSPITALIZED ADULT PATIENTS WITH HYPONATRAEMIA: A RETROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Hyponatremia is the most common electrolyte disorder encountered in hospitalized patients and is associated with significant neurological morbidity and mortality, particularly when severe. Hospitalized patients often have heterogeneous etiologies and variable clinical severity, necessitating structured evaluation and careful correction strategies. **Aim:** To evaluate the clinical profile, etiological distribution, biochemical characteristics, and outcomes of adult patients with hyponatremia, and to assess the association between disease severity and neurological status, volume status, and in-hospital outcomes.

Materials and Methods: This retrospective observational study included 120 hospitalized adult patients diagnosed with hyponatremia (serum sodium <135 mmol/L). Patients were categorized into mild, moderate, and severe hyponatremia based on serum sodium levels. Demographic data, clinical presentation, Glasgow Coma Scale (GCS), volume status, etiology, and biochemical parameters including serum and urine osmolality and urinary sodium were recorded. Outcomes were documented as recovery or death. Statistical analysis was performed using appropriate parametric and non-parametric tests, with a p-value <0.05 considered significant.

Results: Hyponatremia was more common in males (56.7%) and predominantly affected middle-aged and elderly patients. Mild hyponatremia was observed in 45% of cases, while 22% had severe hyponatremia. Euvolemic hyponatremia (53%) was the most frequent type, and SIADH (18.3%) was the leading etiology. Neurological manifestations and lower GCS scores were significantly associated with increasing severity ($p<0.001$). Mortality was 14.2%, occurring predominantly in moderate-to-severe hyponatremia and in older age groups. Progressive derangement of serum osmolality correlated with disease severity.

Conclusion: Hyponatremia remains a common and clinically significant disorder among hospitalized adults, with severity strongly associated with neurological impairment and mortality. Euvolemic hyponatremia and SIADH constitute the predominant patterns. Early recognition, accurate etiological classification, severity-based risk stratification, and carefully monitored correction are essential to improve clinical outcomes.

Keywords: Hyponatremia; Severe hyponatremia; SIADH; Glasgow Coma Scale; Serum osmolality; Emergency department; Hospitalized patients.

INTRODUCTION

Hyponatremia (serum sodium <135 mmol/L) is the commonest electrolyte disorder encountered in clinical practice and across hospital settings, with presentations ranging from subtle symptoms to life-threatening neurological compromise. Importantly, it is not merely a "lab abnormality"; multiple cohorts and guideline syntheses link hyponatremia with increased morbidity, prolonged hospital stay, and higher mortality risk, making it a priority condition for systematic evaluation in admitted patients.[1]

Despite long-standing guidance, real-world management remains inconsistent because hyponatremia is a syndrome with heterogeneous etiologies and trajectories, and treatment decisions depend on symptom severity, chronicity, and volume status. Contemporary updates emphasize careful, monitored correction rather than rapid normalization, and highlight that first-line strategies (e.g., solute optimization and fluid restriction in selected patients) often fail in SIAD-type physiology, necessitating a structured escalation pathway.[2]

A large proportion of hospitalized hyponatremia—particularly euvolemic hypotonic hyponatremia—is driven by inappropriate antidiuresis (SIAD/SIADH), where arginine vasopressin activity is inappropriately elevated or its downstream signaling is dysregulated. This mechanism creates a characteristic biochemical profile (low serum osmolality with inappropriately concentrated urine), and is frequently triggered by pulmonary disease, CNS pathology, malignancy, drugs, and postoperative stress—exactly the spectrum commonly seen in internal medicine wards.[3]

Because SIADH can coexist with comorbid disease (e.g., infection, organ failure, malignancy) and medication exposures, bedside differentiation from hypovolemic or hypervolemic states is critical: misclassification leads to wrong therapy and avoidable complications. Reviews emphasize that structured clinical assessment paired with serum/urine osmolality and urine sodium enables practical, high-yield phenotyping, which improves etiological attribution and aligns therapy with physiology.[4]

In acute care and emergency streams, hyponatremia is frequent and often under-worked-up at first contact. ED-focused evidence highlights that symptoms are frequently nonspecific (falls, weakness, confusion) and that initial diagnostic steps must rapidly identify hypotonicity, severity, and likely onset so that urgent therapy is reserved for those at neurological risk while others undergo systematic inpatient evaluation.[5]

Recent syntheses propose pragmatic diagnostic and therapeutic pathways for ED and admission settings, emphasizing: confirm true hypotonic hyponatremia, assess symptom severity, determine likely duration (acute <48 h vs chronic), and stratify treatment intensity accordingly. Such pathways aim to reduce both undertreatment (missed severe symptomatic cases) and overtreatment (unnecessary rapid correction), and support continuity of care from ED to ward management.[6]

An additional clinical challenge is iatrogenic harm from overly rapid correction, particularly in chronic hyponatremia, where osmotic demyelination risk becomes clinically relevant. Meta-analytic evidence in hospitalized patients indicates that "rapid correction" is not rare and is associated with adverse outcomes, strengthening the case for protocol-driven correction targets and monitoring systems during therapy.[7]

Neurological symptom burden and prognosis also track with severity and clinical context: recent cohort data show that neurological manifestations correlate with disease severity and patient factors, reinforcing the importance of linking symptom profiling (including GCS and seizure/coma markers) with biochemical phenotyping for risk stratification and outcome prediction. Parallel longitudinal studies demonstrate that hyponatremia at admission or during hospitalization associates with higher mortality/readmission and longer length of stay, while internal-medicine cohorts underscore its measurable clinical impact in routine ward practice and across diverse populations.[8-12]

Aim

To evaluate the clinico-etiological profile, biochemical patterns, and outcomes of hyponatremia among hospitalized adult patients, and to determine associations between severity and key clinical/biochemical variables.

Objectives

1. To describe the demographic profile, clinical presentation, volume status, etiological distribution, and biochemical characteristics (serum/urine indices) of hospitalized hyponatremia cases.
2. To assess the association of hyponatremia severity with neurological status (symptoms/GCS), biochemical parameters, volume status, and in-hospital outcomes (recovery/mortality).

MATERIALS AND METHODS

Study Design

This retrospective study was conducted in the Department of General Medicine, Government Medical College, Hospital, Yadadri Bhongir. The study period extended from November 2024 to October 2025.

Approval was obtained from the Institutional Ethics Committee prior to initiation of the study. As this was a retrospective record-based study, a waiver of informed consent was granted by the IEC.

Eligibility Criteria

Hospitalized adult patients (≥18 years) diagnosed with hyponatremia (plasma sodium <135 mmol/L) were included. Patients with incomplete records for

key variables (Na+, GCS, outcome) were excluded. Pregnant women and patients receiving treatment for chronic hyponatremia were excluded from the study.

Study Procedure, Sample Size and Sampling Method

A total of 120 patients were included using consecutive eligible records during the study period. Demographic details and clinical features were recorded using a pre-designed proforma. Biochemical parameters including serum sodium, serum osmolality, urine sodium, and urine osmolality were measured.

Based on serum sodium levels, hyponatremia was categorized as:

- **Mild:** 125–135 mEq/L
- **Moderate:** 121–124 mEq/L
- **Severe:** ≤120 mEq/L

Patients were further classified based on volume status into euvolemic, hypovolemic, and hypervolemic hyponatremia. Clinical presentation, etiology, Glasgow Coma Scale (GCS) score, and outcome were documented. Associations between severity, biochemical parameters, and clinical outcomes were analyzed.

Statistical Analysis

Data were entered in Microsoft Excel and analyzed using SPSS software. Categorical variables were expressed as frequency and percentage, and associations were tested using the Chi-square test. Continuous variables were expressed as Mean ± Standard Deviation (SD), Median, and Interquartile Range (IQR). Comparison between groups was performed using unpaired t-test for normally distributed data and Mann–Whitney U test for non-normal data. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1: Demographic profile of the study participants (n = 120)

Demographic	Percentage (%)
Age (years)	
18–30	12
31–40	20
41–50	40
51–60	20
61–70	18
>70	10
Gender	
Male	68
Female	52

Symptoms

Table 2: Clinical and etiological profile of study participants (n = 120)

Symptom	Male	Female	Total
Asymptomatic	12	6	18
Lethargy	14	13	27
Dizziness	18	6	24
Abnormal behavior	7	11	18
Seizures	4	3	7
Nausea/Vomiting	9	11	20
Coma	4	2	6
Total	68	52	120

Etiology

Diagnosis	Total (%)
SIADH	22
Drug-induced	18
Hypothyroidism	15
Gastrointestinal loss	16
Renal failure	10
Liver failure	8
Cardiac failure	6
Adrenal insufficiency	9
Others	6
Total	120

Severity, Type, and Outcome

Parameter	Category	n (%)
Severity	Mild	54 (45)
	Moderate	40 (33)
	Severe	26 (22)
Type	Euvolemic	64 (53)
	Hypovolemic	36 (30)

	Hypervolemic	20 (17)
Outcome	Recovered	103 (85.8)
	Death	17 (14.2)

Table 3: Distribution of clinical parameters according to severity of hyponatremia (n = 120)

Parameter	Mild	Moderate	Severe	Total
Age 41–70 yrs	28	22	16	66
Abnormal behavior/coma	2	6	18	26
GCS <8	0	4	12	16
SIADH	12	7	3	22
Drug-induced	8	5	5	18
Deaths	0	6	11	17

- Age vs severity: p = 0.041
- Symptoms vs severity: p < 0.001
- GCS vs severity: p < 0.001
- Outcome vs severity: p < 0.001

Table 4: Age-wise outcome distribution

Age group	Survived	Death	Mortality (%)
18–30	12	0	0
31–40	18	2	10
41–50	38	2	5
51–60	15	5	25
61–70	13	5	27.8
>70	7	3	30
Total	103	17	100

Table 5: Biochemical parameters according to severity of hyponatremia

Parameter	Mild	Moderate	Severe	Overall Mean
Serum osmolality (mOsm/kg)	248.6 ± 10.8	246.2 ± 11.9	239.4 ± 13.6	245.1 ± 12.4
Urine osmolality (mOsm/kg)	342.5 ± 88.6	325.7 ± 72.4	335.1 ± 79.8	334.4 ± 80.1
Urinary sodium (mEq/L)	61.8 ± 21.9	56.3 ± 18.6	52.4 ± 20.2	57.1 ± 20.3

The present study demonstrates that hyponatremia predominantly affects middle-aged and elderly hospitalized patients, with euvolemic hyponatremia being the most common type. Severity of hyponatremia showed a significant association with age, neurological manifestations, and GCS score. Severe hyponatremia was strongly linked to altered mental status, lower GCS scores, and higher mortality. SIADH and drug-induced hyponatremia emerged as the leading etiologies. Biochemical parameters showed progressive derangement with increasing severity, reinforcing the clinical-biochemical correlation. Mortality increased significantly with advancing age and severity of hyponatremia.

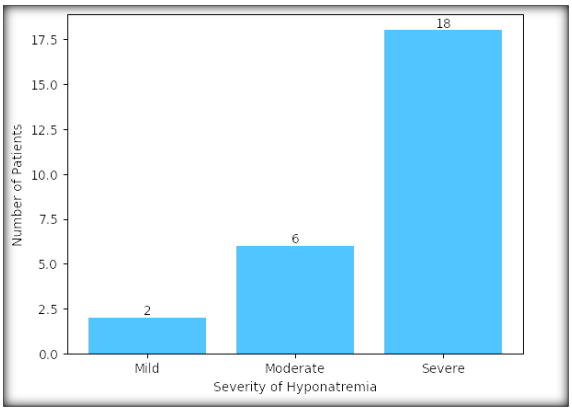

Figure 2: Distribution of patients with abnormal behavior/coma across severity levels

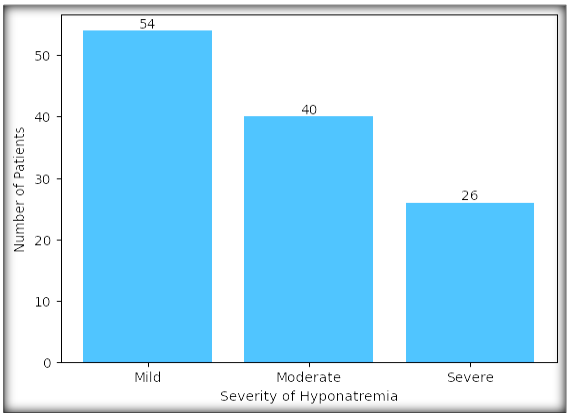

Figure 1: Distribution of patients according to severity of hyponatremia (n = 120)

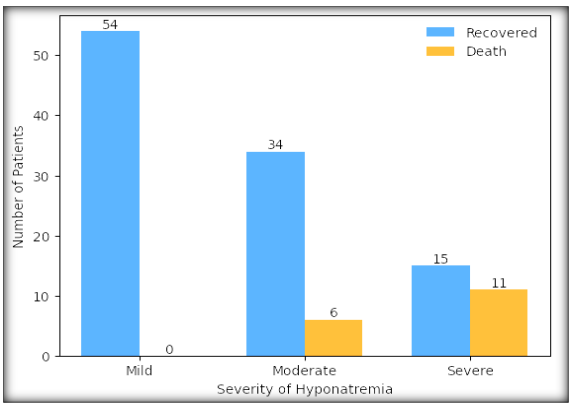

Figure 3: Clinical outcome of patients according to severity of hyponatremia

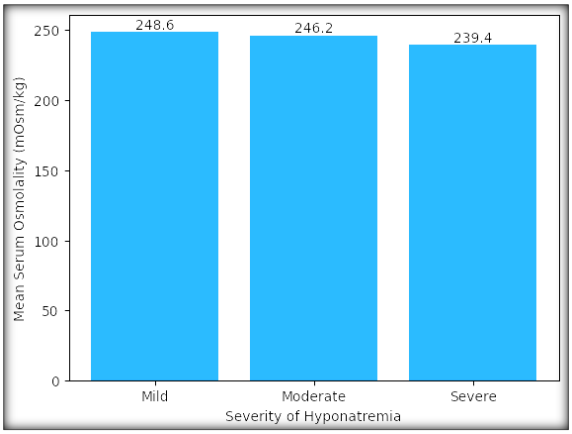

Figure 4: Mean serum osmolality across severity levels of hyponatremia

DISCUSSION

Hyponatremia is among the most frequent electrolyte abnormalities in hospitalized patients and is clinically important because it reflects impaired water excretion, most commonly due to failure to suppress antidiuretic hormone (ADH) and resultant water retention. In addition, excess free-water intake beyond renal diluting capacity (primary polydipsia or low-solute intake states) can contribute in selected settings. Kheetan et al., 2021,[13] highlight that hyponatremia is fundamentally a disorder of water balance and must be interpreted in relation to osmolality, volume status, and the clinical context.

In our cohort of 120 hospitalized adults, hyponatremia was more common in males (56.7%) than females (43.3%), and most frequently occurred in the middle age to older age groups, with the largest cluster in 41–50 years (33.3%) and an overall predominance of cases in the 41–70 range. This male predominance is comparable to the ICU-based profile reported by Babaliche et al., 2017,[16] and the adult inpatient experience reported by Rahil et al., 2009,[17] both of which showed higher representation of males. Importantly, our data also demonstrated that severity increased with increasing age, consistent with the concept that aging is associated with diminished renal free-water handling, higher burden of comorbidity, and more frequent exposure to precipitating illnesses and drugs, as emphasized in broader hospitalized-cohort mortality work by Holland-Bill et al., 2015.[14]

Regarding severity distribution, mild hyponatremia was the most common category (45%), followed by moderate (33%) and severe (22%), indicating that most hospitalized cases fall in the mild–moderate range but a clinically meaningful proportion present with severe disease. This pattern is directionally consistent with ward and ICU studies where mild–moderate cases comprise the bulk while severe cases drive neurological complications and mortality. Waikar et al., 2009,[15] demonstrated a graded mortality association across mild, moderate, and severe hyponatremia categories in hospitalized patients, supporting the clinical rationale for severity-based risk stratification.

Symptom profile in our study showed a clear severity–symptom gradient. Most mild cases were asymptomatic or had nonspecific complaints, whereas moderate-to-severe hyponatremia was characterized by neurological and gastrointestinal manifestations. Overall, lethargy (22.5%) and dizziness (20%) were common, with nausea/vomiting (16.7%) also frequent; with increasing severity, more serious features such as abnormal behavior, seizures, and coma were observed. This symptom spectrum is consistent with clinical descriptions emphasizing gastrointestinal upset and evolving neurocognitive dysfunction as sodium declines, as summarized by Hochman et al., 1989,[20] and contemporary clinical discussions in hospitalized cohorts. Our symptom ranking differs slightly from Babaliche et al., 2017,[16] who reported vomiting as the leading symptom in their ICU cohort; such variation is plausibly explained by differences in case-mix (ICU enrichment), precipitating etiologies, and thresholds for hospital admission.

Etiologically, SIADH was the leading cause (18.3%) in our cohort, followed by drug-induced (15%), gastrointestinal losses (13.3%), and hypothyroidism (12.5%), with additional contributions from adrenal insufficiency, renal failure, liver failure, cardiac failure, infections, and malignancy. The predominance of SIADH is concordant with hospital-based and ICU observations where non-osmotic ADH release and inappropriate antidiuresis dominate inpatient hyponatremia patterns; this aligns with the ICU experience reported by Babaliche et al., 2017,[16] and the ICU-focused data from India by Pillai et al., 2018.[19] In contrast, Rahil et al., 2009,[17] reported extra-renal fluid loss as a major contributor in their setting, underscoring how local epidemiology (diarrheal illness burden, diuretic use, climate, ICU vs ward sampling) can shift etiological proportions.

Volume-status classification in our dataset showed euvolemic hyponatremia as the most common type (53%), followed by hypovolemic (30%) and hypervolemic (17%). This supports the central role of SIADH and other euvolemic mechanisms in general medicine admissions. Findings of euvolemic predominance are consistent with multiple inpatient profiles, including ICU/hospital studies such as Babaliche et al., 2017,[16] and Pillai et al., 2018.[19] However, Hochman et al., 1989,[20] and other ward-based datasets have sometimes reported different distributions depending on heart failure/cirrhosis burden and diuretic exposure; therefore, distribution differences across studies should be interpreted as context-driven rather than contradictory.

A key clinical finding in our study was the inverse association between severity and GCS, with severe hyponatremia showing markedly poorer neurological status. This is pathophysiologically expected because cerebral edema risk rises with lower sodium and more abrupt declines. Severe hyponatremia also showed the strongest linkage to adverse outcomes:

overall mortality was 14.2%, concentrated primarily in moderate-to-severe categories. This magnitude is comparable to mortality figures reported in large hospitalized datasets and inpatient studies; Holland-Bill et al., 2015,[14] demonstrated increased mortality risk across a very large acutely hospitalized population, and Waikar et al., 2009,[15] showed mortality escalation with severity. Recent emergency-department severe-hyponatremia analyses also report mortality around a similar range; for example, Sendag et al., 2023,[25] reported mortality of approximately 12–13% among severe hyponatremia patients, supporting the view that severe cases are a high-risk phenotype even when the absolute sodium difference between survivors and non-survivors is not always statistically distinct.

Therapeutically, safe correction remains central. Overcorrection can cause osmotic demyelination, while undertreatment in symptomatic cases risks ongoing cerebral edema and seizures. Modern guidance reinforces correction targets and monitoring rather than aggressive normalization, and recommends aligning treatment to symptom burden, chronicity, and mechanism. Sterns et al., 2024,[30] emphasize maintaining disciplined correction strategies ("stay the course"), supporting the approach used in our clinical setting where hypertonic saline is reserved for severe and/or neurologically symptomatic patients under close monitoring. Consistent with the low incidence reported in the literature, osmotic demyelination is rare when protocolized correction limits are respected, and severe inpatient hyponatremia outcome studies reiterate the importance of monitoring trajectories rather than single sodium values; this is also supported by analyses such as Clayton et al., 2006.[23]

Comorbidity patterns in our cohort (notably hypertension, diabetes, and CKD) reflect typical internal medicine admissions and may amplify risk and worsen outcomes through disease severity, diuretic exposure, impaired renal water handling, and systemic inflammatory states. In CKD populations, dysnatremias are associated with mortality independent of cardiovascular events in several analyses; Huang et al., 2017,[22] and outcome-focused hospitalized studies support the principle that sodium derangements act as both severity markers and potential contributors to adverse outcomes. In the subset of our patients with CKD and multi-morbidity, careful evaluation of volume status, medication exposure, and urine studies becomes particularly critical to avoid inappropriate correction pathways.

Overall, the present study reinforces that hospitalized hyponatremia is predominantly a middle-aged to elderly disorder with euvolemic/SIADH patterns being common, and that severity strongly tracks neurological impairment and mortality. Differences in symptom ranking or etiological proportions between our cohort and other studies likely reflect differences in admission thresholds, ICU enrichment, seasonal/epidemic drivers, local prescribing patterns, and comorbidity burden rather than true disagreement across evidence.

CONCLUSION

Hyponatremia is a common and clinically important electrolyte disorder among hospitalized adults, with a higher burden in middle-aged to elderly patients and an increasing trend in severity with advancing age. In this study of 120 patients, most cases were mild, while a substantial proportion had moderate-to-severe hyponatremia associated with significant neurological manifestations and poorer GCS scores. Euvolemic hyponatremia was the most frequent subtype and SIADH was the most common etiology, reinforcing the importance of structured clinical assessment supported by serum and urine indices for accurate classification. Mortality was higher in moderate-to-severe hyponatremia, emphasizing that hyponatremia is both a marker of illness severity and a contributor to adverse outcomes. Early recognition, etiological identification, severity-based risk stratification, and carefully monitored correction according to current principles are essential to reduce morbidity and mortality. Larger multi-centric studies with long-term follow-up are recommended to better define regional etiological patterns and long-term outcomes in hospitalized hyponatremia.

Limitations

The present study has certain limitations. First, it is a single-centre study with retrospective single-centre design and record-based sampling, which may limit generalisability to broader community or multi-institution populations. Second, although the sample size was 120, subgroup analyses across multiple etiologies and severity strata may still be underpowered to detect smaller differences. Third, only in-hospital/short-term outcomes were assessed; long-term outcomes such as readmission, delayed neurological morbidity, and long-term mortality were not evaluated. Fourth, assessment of volume status in hospitalized patients can be challenging, especially in older adults with mixed comorbidities, potentially leading to misclassification in borderline cases despite biochemical support. Finally, the study population included only patients presenting to a tertiary care centre, which may over-represent more severe illness profiles compared to primary or secondary care settings.

Conflict of Interest: None

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