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HYPONATRAEMIA IN HOSPITALISED PATIENTS: CAUSES AND MANAGEMENT

Internal Medicine

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ABSTRACT

Hyponatraemia, defined as a serum sodium concentration <135 mmol/L, is the most common electrolyte disorder encountered in hospitalised adults. It is associated with substantial morbidity, prolonged hospital stay, increased healthcare utilisation, and mortality. The condition arises from disturbances in water balance, often mediated by inappropriate antidiuretic hormone activity and influenced by comorbid illness, medications, and clinical interventions. This review summarises the pathophysiology, diagnostic approach, and evidence-based management of hyponatraemia across hypovolaemic, euvolaemic, and hypervolaemic states. Particular emphasis is placed on safe correction thresholds, prevention of osmotic demyelination syndrome (ODS), and current guideline-driven recommendations. Emerging therapies, precision monitoring systems, and research directions aimed at improving outcomes in hospital settings are also discussed.

KEYWORDS

Hyponatraemia, SIADH, Hypertonic saline, Osmotic demyelination syndrome, Hospitalised patients

INTRODUCTION

Hyponatraemia, defined as serum sodium below 135 mmol/L, is the most frequent electrolyte disorder in hospitalized patients, affecting up to 30% of inpatients. It signifies underlying pathophysiological disturbances and is linked to increased morbidity, mortality, and prolonged hospital stay. Most cases result from impaired renal water excretion driven by arginine vasopressin (ADH). Effective management requires structured clinical assessment, targeted biochemical evaluation, and cautious correction to prevent complications such as osmotic demyelination syndrome.

Aetiology Of Hyponatraemia In Hospitalized Patients

Hyponatraemia occurs when water retention exceeds sodium levels, usually due to excess ADH or impaired renal water excretion. In hospitalized patients , It is classified by volume status: **hypovolemic** (sodium loss from renal[2] or extrarenal causes), **euvolemic** (often SIADH, medications[2,3] endocrine disorders, or low-solute intake), and **hypervolemic** (heart failure, cirrhosis, nephrotic syndrome causing reduced effective arterial volume and ADH-driven water retention).

Diagnostic Evaluation

The evaluation process initiates with an assessment of clinical volume, subsequently proceeding to biochemical investigations.[2,3] **Serum osmolality** serves to differentiate true hypotonic hyponatraemia, characterized by values below 275 mOsm/kg, from conditions such as pseudohyponatraemia or hypertonic states, including hyperglycaemia and mannitol administration.

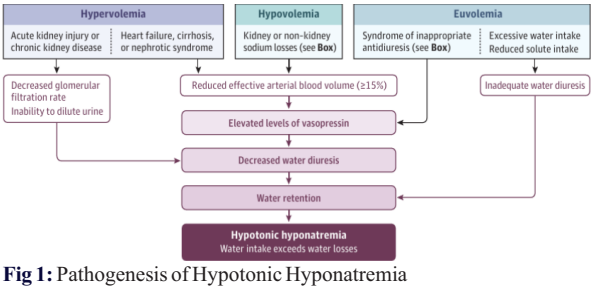

Fig 1: Pathogenesis of Hypotonic Hyponatremia

Urine Osmolality:

Values less than 100 mOsm/kg indicate an excess intake of water, whereas values greater than 100 mOsm/kg suggest water retention mediated by antidiuretic hormone (ADH).

Urine sodium levels below 30 mmol/L suggest hypovolaemia due to extrarenal sodium loss, while levels exceeding 30 mmol/L are indicative of SIADH or renal sodium losses.

Endocrine evaluation involves measuring thyroid-stimulating hormone and cortisol levels to exclude the presence of hypothyroidism and adrenal insufficiency.

Accurate diagnosis is crucial, as incorrect treatment, such as the administration of isotonic saline in cases of SIADH, may inadvertently exacerbate hyponatraemia.

Adrogué HJ, Tucker BM, Madias NE. Diagnosis and management of hyponatremia: A review. JAMA. 2022;328(3):280-291. doi:10.1001/jama.2022.10217

Pathophysiology Of Hyponatraemia

Hyponatremia reflects a disturbance in serum osmolarity, as small shifts can cause cellular swelling or shrinkage.[4] Because sodium is the main determinant of osmolarity, low serum sodium (<135 mmol/L) leads to clinical hyponatremia, with prevalence ranging from 5–35% depending on definition, testing frequency, and healthcare setting.[5-7] It is classified as mild (130–135 mmol/L), moderate (125–129 mmol/L), or severe (<125 mmol/L),[8-9] and may be asymptomatic or life-threatening. Classification by volume status—hypovolemic, euvolemic, or hypervolemic—guides diagnosis and management.[8]

Hyponatremia arises from excess water, sodium loss, or both.[6] Common causes include CKD, heart failure, cirrhosis, gastrointestinal losses, endocrine disorders, malignancies, and SIADH due to pulmonary or neurological diseases or medications[8]. Neurologic manifestations result from cerebral edema. Acute hyponatremia (<48 hours) carries a higher risk because the brain cannot adapt quickly, whereas chronic hyponatremia allows cerebral adjustment[7] but still increases risks. Severe cases may present with encephalopathy[4], seizures, coma, or herniation, while moderate cases cause lethargy and confusion, and mild cases present with headache, nausea, or subtle cognitive impairment.

Even mild chronic hyponatremia increases morbidity through gait disturbances, falls, fractures, and reduced quality of life. Management includes hypertonic saline for severe symptomatic cases, and isotonic saline or diuretics for chronic asymptomatic cases based on volume status. Correction should not exceed 6 mmol/L to avoid osmotic demyelination. Identifying and treating the underlying cause[4,10] is essential, as hyponatremia increases hospitalizations, healthcare costs, and overall resource use.[11-13]

Principles Of Management

Management of hyponatraemia relies on assessing symptom severity, duration, and volume status. Severe neurologic symptoms require urgent therapy; acute cases risk cerebral edema, while chronic cases risk osmotic demyelination if corrected rapidly. Treatment principles include addressing the underlying cause, limiting correction to ≤10–12 mmol/L per 24 hours, monitoring sodium every 2–4 hours[1], tracking urine output, and using desmopressin clamping to prevent overcorrection.

Treatment Strategies

6.1 Hypovolaemic Hyponatraemia[2]

Management focuses on correcting both the sodium deficit and the underlying volume depletion:

Isotonic saline (0.9% NaCl) is the cornerstone therapy. Restoring

intravascular volume suppresses ADH secretion, allowing renal excretion of free water and gradual sodium normalisation.

Identify and reverse contributing factors, including discontinuation of thiazides, treatment of adrenal insufficiency, or management of GI losses.

Potassium repletion is essential, as hypokalaemia aggravates hyponatraemia by driving potassium into cells and sodium out.

Monitoring: Frequent assessment of urine output and serum sodium is crucial, as rapid suppression of ADH may unexpectedly accelerate correction.

Euvolaemic Hyponatraemia (SIADH and Related Causes)

Treatment aims to increase water excretion while addressing the cause:

Fluid restriction (<1 L/day) remains first-line. Stricter limits (500–750 mL/day) may be required in patients with a high urine osmolality (>500 mOsm/kg).

Increased solute intake enhances osmotic diuresis:

- Oral **urea (15–30 g/day)** is highly effective, safe, and recommended in recent guidelines.
- **Salt tablets** combined with **loop diuretics** improve excretion of electrolyte-free water.

Vaptans (vasopressin receptor antagonists):[3]

- **Tolvaptan** is effective in chronic SIADH but must be used cautiously due to risk of overcorrection.
- Close inpatient monitoring is mandatory during initiation.

Endocrine causes (e.g., adrenal insufficiency, hypothyroidism) must be addressed before diagnosing SIADH.

Demeclocycline is largely obsolete due to nephrotoxicity and delayed onset of action.

Hypervolaemic Hyponatraemia

Seen in heart failure, cirrhosis, and nephrotic syndrome:

Fluid restriction combined with **sodium restriction (1–2 g/day)** helps reduce free-water retention.

Loop diuretics (e.g., furosemide) promote free-water excretion.

Vaptans may benefit patients with heart failure or cirrhosis unresponsive to conventional therapy.

Optimisation of underlying disease-e.g., RAAS blockade in heart failure, paracentesis/albumin support in cirrhosis-is essential.

Severe Symptomatic Hyponatraemia

Patients presenting with seizures, severe confusion, vomiting, or signs of cerebral oedema require urgent correction:

Administer **100 mL of 3% hypertonic saline over 10 minutes**, repeated up to three doses.

Target an **initial rise of 4–6 mmol/L** in the first few hours to halt cerebral oedema.

After stabilisation, use slower correction strategies depending on the underlying aetiology.

Continuous monitoring in a high-dependency or ICU environment is recommended.

Treatment Goals And Correction Limits

Safe correction of serum sodium is the most crucial aspect of managing hyponatraemia:

Recent guidelines (ESE/ES, 2023; CJASN, 2024) recommend:
Chronic hyponatraemia (>48 h duration):
Limit increase to **≤10–12 mmol/L in 24 hours** and **≤18 mmol/L in 48 hours**.

High-risk patients (Na <105 mmol/L, alcoholism, malnutrition, liver disease):

Correction should not exceed **8 mmol/L/24 hours**.

Acute hyponatraemia (<48 h) with severe symptoms:
An initial rapid rise of **4–6 mmol/L** is appropriate, followed by controlled correction.

Monitoring Requirements
Serum sodium every 2–4 hours during active correction.

Urine output surveillance—a sudden high output suggests ADH suppression and risk of rapid sodium rise.

Desmopressin clamp: Proactively used to stabilise correction by preventing water diuresis. This is particularly beneficial in patients at high risk of ODS.

Why Limits Matter
Rapid correction in chronic hyponatraemia causes **osmotic demyelination syndrome**, a severe neurological condition. The brain adapts to chronic hypo-osmolar states by extruding osmolytes; rapid re-exposure to normal osmolality leads to irreversible demyelination.

Prevention and Management of Overcorrection[15]
Overcorrection is a medical emergency because it directly predisposes to ODS.

Risk Factors for Overcorrection

- Sudden water diuresis (e.g., after treating hypovolaemia or adrenal insufficiency)
- Discontinuation of ADH-stimulating drugs
- Postoperative ADH suppression
- Use of vaptans or hypertonic saline without adequate monitoring

Prevention Strategies
1. Set conservative correction targets based on patient risk profile.
2. Use desmopressin (DDAVP) proactively to prevent rapid rises in serum sodium when high aquaresis is expected.
3. Avoid large fluid boluses unless indicated for symptomatic cases.
4. Monitor sodium frequently—every 2 hours in high-risk patients.

Management of Overcorrection
If serum sodium rises too quickly:
Stop hypertonic saline or ongoing therapy immediately.

Administer desmopressin 2–4 µg IV to halt further free-water excretion.[16]

Re-lower sodium using:
D5W infusion, or
Hypotonic saline (0.45%) in selected cases.

Consult nephrology/critical care urgently.
Prompt reversal of overcorrection substantially reduces risk of ODS.

Emerging Therapies[17]

Hyponatraemia research is shifting toward precision-based, technology-supported, and pathophysiology-targeted therapies.

Precision Monitoring Systems

ICU-based closed-loop systems integrate real-time serum sodium measurements with automated saline or DDAVP dosing. Early studies show improved control of correction rates.

Pharmacological Advances

SGLT2 inhibitors facilitate osmotic diuresis and may serve as adjunct therapy in SIADH.

Selective aquaretics under development aim to reduce risks associated with current vaptans.

Novel biomarkers of cerebral adaptation (e.g., myoinositol levels) may help personalise correction thresholds.

Risk-Stratified Algorithms

Machine-learning models using variables such as baseline Na, comorbidities, urine osmolality, and volume status.

CONCLUSION

Hyponatraemia in hospitalised patients remains a complex clinical entity requiring nuanced understanding of pathophysiology, precise diagnostic interpretation, and cautious management. Tailored therapy guided by volume status, severity, and chronicity is fundamental. Integration of multidisciplinary (nephrology, endocrinology, critical care) expertise and adoption of standardised hospital protocols will continue to enhance patient safety and outcomes.

Declaration Of Conflicting Interests

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