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Review

Management of Sodium Imbalance

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ABSTRACT

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Sodium imbalance consists of hyponatremia and hypernatremia. Acute and severe hyponatremia and hypernatremia are life-threatening conditions. Rapid and slow correction should be considered for optimal outcomes, as aggressive management is often associated with the danger of demyelination and cerebral edema. This review aims to determine the safe management of sodium imbalance so that it is hoped that in overcoming critical conditions it does not transfer to other critical conditions that are equally fatal. The data search strategy uses the PubMed and Google Scholar databases between 2010 and 2022 with free full text that can be downloaded. The findings emphasize that acute and severe hyponatremia and hypernatremia are often life-threatening conditions. Management of these conditions requires caution, as aggressive correction in hyponatremia can result in osmotic demyelination, while hypernatremia can lead to cerebral edema. Despite theoretical risks, the study found no significant difference in 30-day mortality, seizures, cerebral edema, or decreased consciousness between fast and slow correction methods. The conclusion underscores the importance of tailored and cautious management strategies to mitigate the risks associated with sodium imbalance corrections.

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INTRODUCTION

Sodium imbalance consists of hyponatremia and hypernatremia. Hyponatremia, defined usually as serum sodium concentration ([Na]) either below 135 mmol/L or above 145 mmol/L for hypernatremia, represents the most frequently encountered electrolyte disorder in a variety of clinical settings¹. The prevalence of hyponatremia is most common in hospitalized patients at 5-35%², while hypernatremia is 1-4%³. Sodium imbalance is often life-threatening conditions that require rapid management, but aggressive management is often associated with the danger of demyelination and cerebral edema^{2,4}.

Most patients' hyponatremia should be managed by treating their underlying disease and according to whether they have hypovolemic, euvolemic, or hypervolemic⁵. Hypotonic hyponatremia is the most common electrolyte disorder encountered in clinical practice. While

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managing patients with hyponatremia, it is very important to keep in mind their risks for complications from the acute state as well as risks of demyelination in the chronic state⁶.

Hypernatremia is a disorder of the homeostatic status regarding body water and sodium contents. This imbalance is the basis for the diagnostic approach to hypernatremia⁷. Hypernatremia increases the risk of mortality in the general population. Acute hypernatremia causes lesions in the brain, such as cell shrinkage, petechial and subarachnoid haemorrhages, hematomas, subdural fluid collections, vascular congestion, and venous thrombosis¹. Hypernatremia is an electrolyte disorder, which may lead to fatal consequences under improper management⁷.

The purpose of this literature review is to determine the safe management of sodium imbalance so that it is hoped that in overcoming critical conditions it does not transfer to other critical conditions that are equally fatal.

METHOD

This study employs a literature review methodology to analyze the management of sodium imbalance. The PubMed and Google Scholar database was utilized for the literature search, using specific keywords including "hyponatremia, hypernatremia, treatment, management, correction, guideline". The inclusion criteria were articles published in PubMed and Google Scholar between 2010-2022, focusing on the management of sodium balance disorders, and specifically encompassing clinical trials, randomized controlled trials, and reviews. Articles that could not be accessed were excluded from the study. The selection process involved several key steps to ensure the quality and relevance of the included studies. Following this, data extraction was performed from the selected articles, gathering information on study methods, population, interventions, main outcomes, and recommendations.

The extracted data was then qualitatively analyzed to identify common patterns, compare findings across different studies, and highlight gaps in the current literature. This comprehensive analysis allowed for a synthesis of the findings to provide a thorough overview of best practices in the management of sodium imbalance, including approaches to diagnosis, treatment, and prevention of complications. By employing this rigorous methodology, the study aims to offer evidence-based recommendations for the effective and safe management of sodium imbalances.

RESULTS

The results of the literature search in the PubMed and Google Scholar databases were extracted as needed.

A. HYPONATREMIA

PREVALENCE OF HYPONATREMIA

Plasma sodium concentration is determined by the external balance (intake minus output) of sodium, potassium, and water and by the internal exchange between sodium free in solution and sodium bound to polyanionic proteoglycans in bone, cartilage, and skin⁴. Hyponatremia is the most frequent electrolyte disturbance in hospitalized patients ². Acute and severe hyponatremia is sometimes a life-threatening condition. The prevalence of hyponatremia in all degrees of hyponatremia increases with the age of the patient and the mortality rate increases with lower sodium levels. The mortality rate also increases with the severity of hyponatremia and increasing age (Figure 2)².

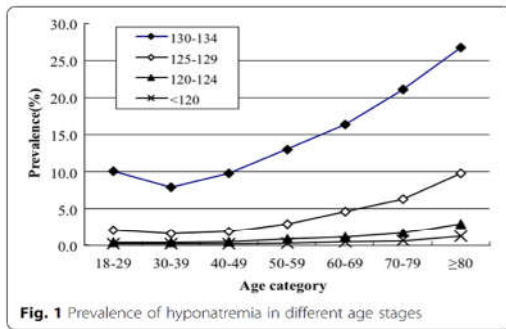


Fig. 1 Prevalence of hyponatremia in different age stages

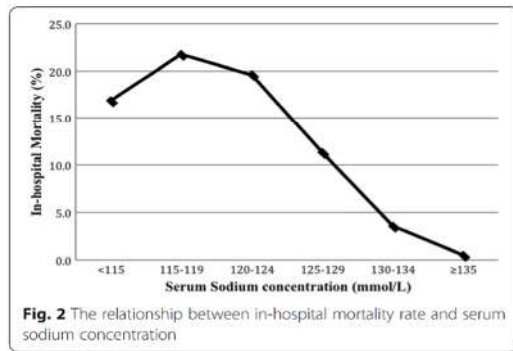


Fig. 2 The relationship between in-hospital mortality rate and serum sodium concentration

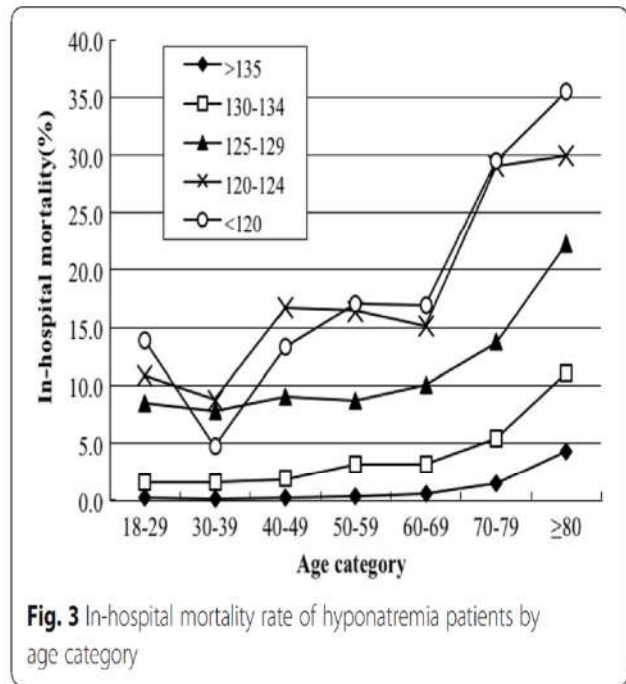


Fig. 3 In-hospital mortality rate of hyponatremia patients by age category

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Figure 1. Prevalence of hyponatremia by age (A), mortality rate by degree of hyponatremia (B), mortality rate by degree of hyponatremia and age (C)^{26,8}.

DIAGNOSIS OF HYPONATREMIA

Hyponatremia is diagnosed using an algorithm based on the type of hyponatremia. Hyponatremia is differentiated based on serum osmolality into hypertonic hyponatremia (S osm >295 mOsm/L), hypotonic hyponatremia (S osm < 275 mOsm/L), and isotonic hyponatremia (S osm 275-295 mOsm/L). Hypotonic hyponatremia is true hyponatremia and with further

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examination of urine osmolality and urine sodium, the type of hyponatremia can be determined, whether hypervolemic hyponatremia, hypovolemic hyponatremia, or euvolemic hyponatremia and its possible causes (Figure 3)^{6,8,9}.

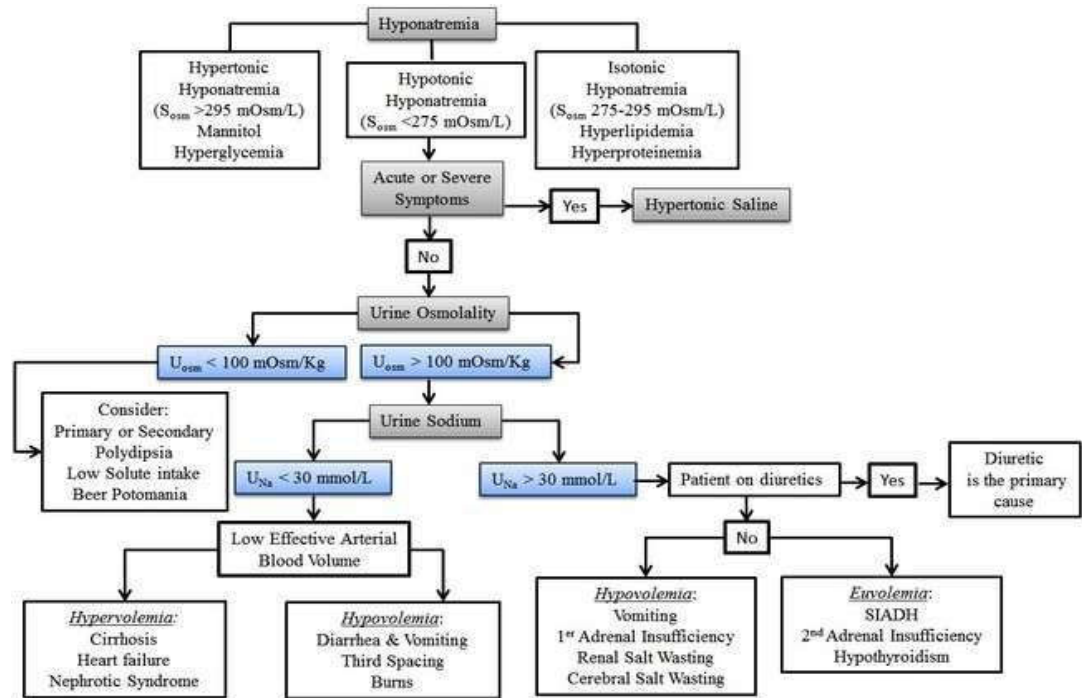


Figure 2. Hyponatremia diagnostic algorithm¹⁰

This classification of hyponatremia still has limitations in clinical application. Determining the severity of hyponatremia is based on SNa absolute concentration criteria but symptoms do not always correlate with the degree of hyponatremia. Determining acute or chronic based on the time of onset criteria (cutoff 48 hours) but the time of onset is not always known. Determining hypovolemic, euvolemic, and hypervolemic is also based on the criteria of clinical measurements of volume status and these measurements have low sensitivity and specificity⁸.

MANAGEMENT OF HYPONATREMIA

The general approach to treating hyponatremia is based on American and European guidelines (table 1)¹¹. For acute symptomatic hyponatremia, both guidelines emphasize the need for urgent correction to prevent brain herniation, with slight differences in the recommended increase in serum Na⁺ levels. For symptom-based treatment, the US guidelines suggest a more aggressive approach with continuous infusion for moderate symptoms, while the European guidelines recommend bolus doses with careful monitoring. In chronic hyponatremia, both guidelines stress the importance of avoiding rapid correction to prevent osmotic demyelination syndrome, but they differ in the specific correction rates and management of overcorrection. For additional treatment options, both regions agree on fluid restriction for SIADH but differ in the specifics of using

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isotonic saline and handling hypervolemia with furosemide and NaCl. Overall, while both guidelines share common goals in the safe management of hyponatremia, they exhibit differences in specific protocols and correction rates, reflecting regional variations in medical practice.

Table 1. Hyponatremia Management Guideline¹¹

United States Guidelines		European Guidelines
Symptomatic Acute Hyponatremia < 24-48 hours		
Urgent correction goal to aim to prevent brain herniation	Increase serum Na ⁺ by 4-6 mmol/L	Increase serum Na ⁺ by 5 mmol/L
Treatment based on symptoms		
Severe symptoms	Bolus 100 mL of 3% NaCl over 10 minutes x 3 as needed	Bolus 150 mL of 3% NaCl over 20 minutes, 2- 3 times as needed, checking Na every 20 minutes (First-hour management, regardless of acute or chronic condition)
Moderate symptoms with low risk of herniation	Continuous infusion of 3% NaCl at 0.5-2 mL/kg/h	Bolus 150 mL 3% NaCl over 20 minutes, x 1 to prevent further decrease in Na
Limit not to exceed	None in true acute hyponatremia	None in true acute hyponatremia
Chronic Hyponatremia > 48 hours		
Correction rate		
Goal in symptomatic patients using hypertonic saline	4-8 mmol/L/d if low risk for ODS 4-6 mmol/L/d if high risk of ODS For patients with severe symptoms, the first day's increase can be accomplished during first 6 h	Avoid > 10 mmol/L in the first 24 h And > 8 mmol/l during every 24 h thereafter
Limit to avoid potential harm in asymptomatic patients	10-12 mmol/L/d, but max 18 mmol in 48 h if at low risk for ODS 8 mmol/L/d if at high risk of ODS	10 mmol/L in the first 24 h and 8 mmol/l during every 24 h thereafter
Managing Overcorrection of Chronic Hyponatremia		
	Baseline serum Na ⁺ ≥ 120 mmol/L: Intervention probably unnecessary Baseline serum Na ⁺ < 120 mmol/L: Replace water orally or as D5W at 3 mL/kg/h with or without desmopressin (2-4 µg every 8 h parenterally) Withhold any vasopressin receptor antagonists (vaptans) used Consider dexamethasone, 4 mg every 6 hr for 24 hr following excessive correction	Start once the above-mentioned limits are exceeded Consult an expert to discuss infusion containing electrolyte-free water (10 mL/kg) over 1 h with or without 2 µg desmopressin IV every 8 h
Other Treatment Options		
Hypovolemic hyponatremia	Isotonic saline	Isotonic saline or balanced solution at 0.5-1.0 mL/kg/h
Euvolemic hyponatremia (SIADH)	Fluid restriction of 500 mL/d below the 24-h urine volume (first-line treatment) Urea, vaptan, or demeclocycline (second-line treatment)	Fluid restriction (first-line) Urea or loop diuretics + oral NaCl (second-line) Do not recommend vaptans Recommend against lithium or demeclocycline
Hypervolemic hyponatremia	Fluid restriction, loop diuretic Vaptans	Fluid restriction Recommend against vaptans and demeclocycline

Management of Sodium and Potassium Imbalance (Barkah Djaka Purwanto)

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B. HYPERNATREMIA

CAUSE AND DIAGNOSIS OF HYPERNATREMIA

Table 2. Causes of hypernatremia¹²

Cause	Proximate Cause	Findings Supporting Diagnosis
Inadequate water intake	Lack of access to water	<ul style="list-style-type: none"> Altered sensorium, immobility, endotracheal intubation Chronic care facility residence Fluid prescription that does not take into account insensible losses UOsm > 600 mOsm/kg H₂O
Extrarenal hypotonic uid loss	GI losses or perspiration	<ul style="list-style-type: none"> History of diarrhea, febrile illness, gastric suction, or enteric stula UOsm > 600 mOsm/kg H₂O
Renal concentrating defect	Diuretics	<ul style="list-style-type: none"> History of loop diuretic use Isosthenuric urine Hyperglycemia with glucosuria
	Osmotic diuresis	<ul style="list-style-type: none"> Urea-induced osmotic diuresis Isosthenuric urine (eg, recovery from ATN) Presence of brain trauma, surgery, tumor, in ltrative disease, or infection including tuberculosis
	Central diabetes insipidus	<ul style="list-style-type: none"> Maximally or submaximally dilute urine Persistently dilute urine during water deprivation test Low copeptin levels UOsm increases in response to desmopressin Treatment with lithium or demeclocycline, hypercalcemia, hypokalemia, renal tubulointerstitial disease, especially sickle cell nephropathy and obstructive uropathy
	Nephrogenic diabetes insipidus	<ul style="list-style-type: none"> UOsm < 300 mOsm/kg H₂O Persistently dilute urine during water deprivation test High copeptin levels UOsm fails to increase in response to desmopressin
Excessive salt intake	Hypertonic uid administration	<ul style="list-style-type: none"> Receipt of hypertonic sodium bicarbonate solution dur-ing cardiac arrest or hypertonic saline solution History of dilution error for powdered feeding formulas in infants Administration of TPN or concentrated enteral tube feeds UOsm > 600 mOsm/kg H₂O UNa > 100 mEq/L

Note: Even if not speci cally noted, impaired thirst or access to water is typically also present. Abbreviations: ATN, acute tubular necrosis; GI, gastrointestinal; TPN, total parenteral nutrition; UNa, urine sodium concentration; UOsm, urine osmolality.

The underlying cause of hypernatremia needs to be identified (table 2). Hypernatremia is caused by renal or non-renal water loss that exceeds the accompanying salt loss or by concentrated salt intake. Water loss is the most common cause. The resulting reduction in renal electrolyte-free water excretion and increased water intake prevents the development of hypernatremia if the individual can sense and act on thirst¹².

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The diagnosis of hypernatremia is based on elevated serum sodium concentration ($\text{Na}^+ >145 \text{ mEq/L}$). The prevalence of hypernatremia in hospitalized patients is reported to be between 1% and 4% consisting of community-acquired hypernatremia occurring in 21% and hospital-acquired hypernatremia occurring in 25.9% of patients ^{7,13}.

MANAGEMENT OF HYPERNATREMIA

There are no clear guidelines on the sodium correction rate for hypernatremia, some studies suggest a reduction rate not exceeding 0.5 mmol/L per hour. The goals of management in hypernatremia are symptom recognition, identification of underlying causes, correction of volume disturbances and correction of hypertonicity ^{7,13}. Fluid regimen can be chosen for hypernatremia with following steps. The steps are the calculation of free water deficit, calculation of ongoing fluid losses, choosing the suitable fluid regimen, and monitoring the serum electrolytes ^{14,15}.

Symptomatic Hypernatremia Recommendations

Symptom recognition is necessary to determine the onset of hypernatremia. Recommendations for symptomatic hypernatremia are as follows:^{7,13}

- a. Establish a documented onset (acute, < 24 hours; chronic, > 24 hours)
- b. In acute hypernatremia, correct serum sodium at an initial rate of 2-3 mEq/L/hour (for 2-3 hours) (maximum total, 12 mEq/L/day).
- c. Measure serum and urine electrolytes every 1-2 hours
- d. Perform serial neurological examinations and reduce the correction rate with improvement in symptoms
- e. Chronic hypernatremia with no or mild symptoms should be corrected at a rate not exceeding 0.5 mEq/L/hour and a total of 8-10 mEq/day (e.g. 160 mEq/L to 152 mEq/L in 24 hours).
- f. In case of volume deficit and hypernatremia, intravascular volume should be restored with isotonic sodium chloride before free water administration.

Correction of Volume Disturbance

To make corrections for volume disturbances, it is necessary to calculate fluid deficits and fluid requirements while still taking into account insensible water losses as shown in the following example (Figure 4)^{1,16}

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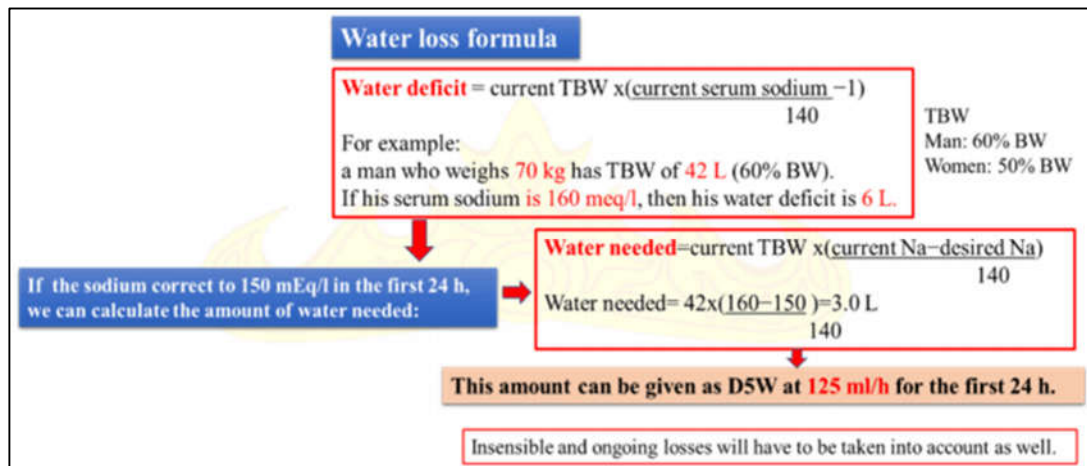


Figure 4. Example of calculation of fluid deficit and fluid requirement^{10,17}

Correction of Hypertonicity

If we use the example above (Na 160 mEq/L) the clinician can use D5W at 125 ml/hour to cover 3 L per 24 hours and to correct the deficit over 48 hours. Some things to note include:^{7,16}

- Correction of hypernatremia should be done slowly and at a rate not exceeding 12 meq/24 hours
- Can use hypotonic solutions such as D5W and NaCl 0.45 and serum sodium is checked every 6 hours
- A combination of D5W and intravenous furosemide can be used with frequent electrolyte monitoring
- If the patient is hypotensive, NaCl 0.9% is used first to stabilize blood pressure followed by hypotonic iv solution
- Patients with central Diabetes Insipidus (DI) are treated with desmopressin
- Thiazide diuretics may be useful in nephrogenic DI in addition to correcting the underlying cause if possible.

DISCUSSION

The clinician should try to ascertain when hyponatremia begins, as its duration is important in determining the appropriate speed of correction. The brain begins to adapt to hyponatremia immediately and cerebral adaptation is maximal within two to three days. Too aggressive correction of hyponatremia with hypertonic saline after adaptation has occurred will increase serum sodium levels so that the extracellular fluid is more concentrated than the intracellular fluid and will draw more water from brain cells and cause osmotic demyelination syndrome (figure 5)^{5,9,18}.

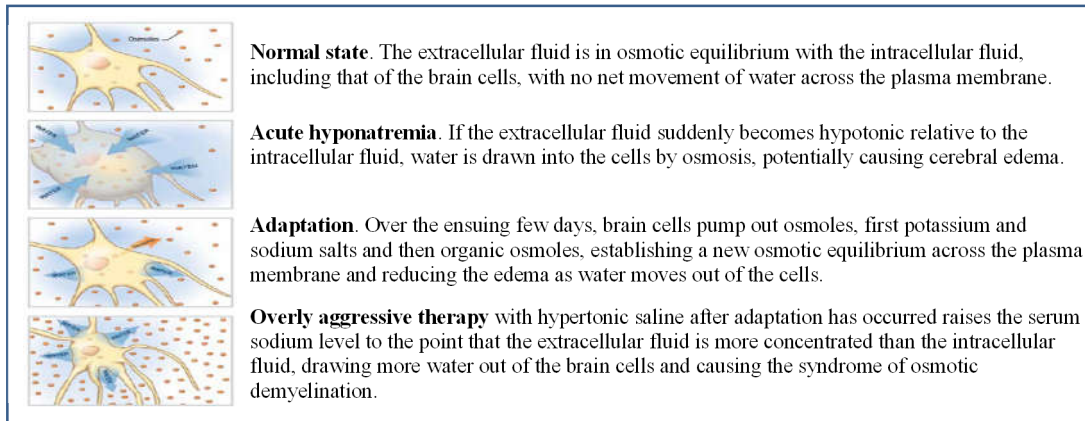


Figure 5. The danger of aggressive correction of hyponatremia¹⁸

Correction of hypernatremia must also be careful. The difference is that in the correction of hyponatremia that is too fast, demyelination will occur while in the correction of hypernatremia that is too fast, cerebral edema, damage and eventually death will occur (Figure 6)⁴.

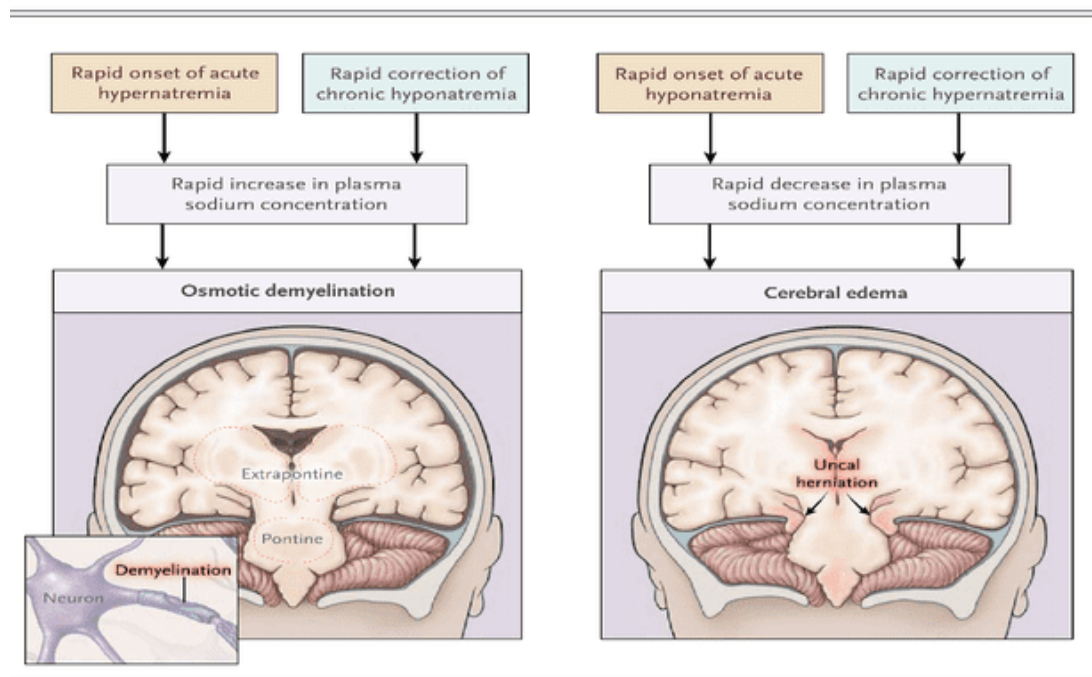


Figure 6. Consequences of Rapid Changes in Plasma Sodium Concentration⁴.

In the figure 6., On the left side, it depicts osmotic demyelination, which occurs due to a rapid increase in plasma sodium concentration. This can be caused by the rapid onset of acute hypernatremia or the rapid correction of chronic hyponatremia. The condition leads to the destruction of the myelin sheath of neurons, particularly in the pons and extrapontine regions of the brain, as shown in the inset diagram of a demyelinated neuron. On the right side, the image

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shows cerebral edema, resulting from a rapid decrease in plasma sodium concentration. This can be triggered by the rapid onset of acute hyponatremia or the rapid correction of chronic hypernatremia. Cerebral edema causes swelling of the brain tissue, which can lead to uncal herniation, where part of the brain is forced downward through openings in the dura, as indicated in the picture. Overall, the image emphasizes the critical importance of carefully managing the rate of sodium concentration changes in plasma to avoid severe neurological complications, such as osmotic demyelination and cerebral edema⁴. Patients with rapid development of hypernatremia, sodium can be corrected quickly with isotonic saline or water without increasing the risk of cerebral edema¹⁹.

Transient atrial fibrillation can also become the risk of hypernatremia during the correction. It may, due to atrial stretch, increase the propensity for rapid firing from the pulmonary veins as a result of stretch-sensitive ion channels. The occurrence of atrial fibrillation in these patients may be explained by the phenomenon of an increase in right atrial diameter, i.e. atrial stretch in response to the increase in preload during the course of treatment²⁰.

In the following study, it was found that rapid correction of hypernatremia with a serum sodium correction rate of >0.5 mmol/L per hour, the 30-day mortality rate in hypernatremia patients obtained from the community and in hospitalized patients did not get a significant difference with patients who performed slower hypernatremia correction with serum sodium levels <0.5 mmol/L per hour. Likewise, in the sub analysis with various levels of correction of hypernatremia >8 , >10 , and >12 mmol/L per 24 hours, there was also no significant difference in the incidence of seizures, cerebral edema and other disorders of consciousness with rapid correction of hypernatremia (Figure 7)¹⁰. It was found that hypernatremia treatment was inadequate in many cases with serum sodium either remaining constant or even increasing during the first 24 h after Emergency Department admission. Mortality in hypernatremia > 147 mmol/L was high at 25%, while patients with adverse outcome had significantly less decline in serum sodium than those who survived²¹.

The studies assessed the effects of slow correction rates. Under-correction was frequent on initial assessment and in the days following admission, with 42% having a worsened or identical natremia one day after admission and serum sodium was normalized in only 32% at 72 hours frequently associated with incorrect fluids administration and an in-hospital mortality rate of 24%¹⁴.

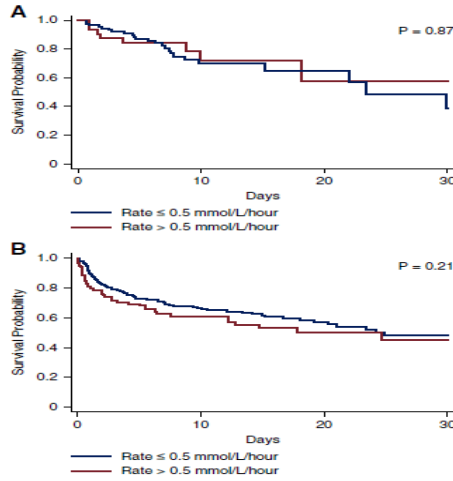


Figure 1. | Thirty-day survival curves for the rapid versus slow correction rate groups are not significantly different. Figure 1A and 1B represents admission and hospital-acquired hypernatremia patients respectively. Thirty-day survival patterns by rapid versus slow correction rate in patients with (A) admission and (B) hospital-acquired hypernatremia.

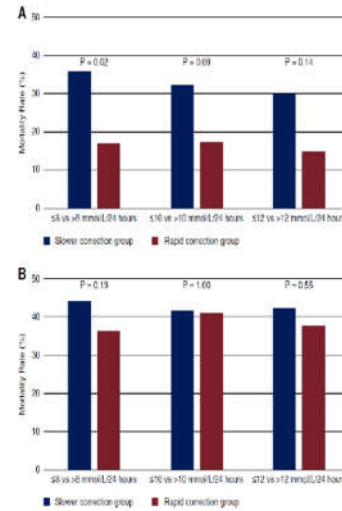


Figure 2. | Incidence of in-hospital mortality rates are lower in rapid correction rate group versus slow correction rate group but not significantly different by various cut-offs of correction rates Figure 2A and 2B represents admission and hospital-acquired hypernatremia patients respectively. In-hospital mortality proportions in patients with (A) admission and (B) hospital-acquired hypernatremia.

Figure 7. 30-day mortality rate with rapid and slow correction of hypernatremia in community-acquire hypernatremia and hospitalized patients¹¹

Lowest mortality in patients with sodium levels of 140 mEq/L and adjusted hazard ratios for the group, 130 and 130 to 135 mEq/L to be 1.93 and 1.28, respectively. Thus, gradual correction of serum sodium levels to 130 to 135 mEq/L appears to be a reasonable target in populations with kidney disease²².

CONCLUSION

Hyponatremia and hypernatremia in acute and severe conditions are often life-threatening. Caution in management is sought because theoretically aggressive management in hyponatremia can cause demyelination while in hypernatremia it can cause cerebral edema although in the study there was no significant difference between fast and slow correction in 30-day mortality, not even seizures, cerebral edema or decreased consciousness.

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