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Review

Hyponatremia

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ABSTRACT

Hyponatremia, a prevalent electrolyte disturbance in hospitalized patients, particularly affects the elderly and ICU populations. This study highlighted hypertension, diabetes mellitus, and thiazide diuretics as significant risk factors. Complex etiologies involving chronic liver disease, congestive heart failure, and syndrome of inappropriate antidiuretic hormone secretion were also notable contributors. Effective management strategies, including judicious use of hypertonic saline for severe cases, emphasize the importance of gradual sodium correction to prevent osmotic demyelination syndrome. Vigilant monitoring and management, especially in patients with neurological symptoms, are crucial. The study underscores the need for a systematic diagnostic approach integrating comprehensive history-taking, clinical examination, and precise laboratory assessments. Such an approach promises to enhance evaluation and therapeutic management of hyponatremia, potentially reducing morbidity and mortality rates, particularly in patients with complex medical backgrounds like advanced cirrhosis. In conclusion, advancing our approach to managing hyponatremia is essential for optimizing patient care and safety in hospitals, reflecting ongoing efforts to improve clinical outcomes through proactive medical practices.

KEYWORDS: Electrolytes' disturbances, SIADH, Antidiuretic hormone

INTRODUCTION

Hyponatremia, characterized by serum sodium levels below 135 mEq/l, is the predominant electrolyte condition in hospitalized patients[1-5]. It is linked to death and morbidity rates between 5% and 50%, contingent upon severity and initial acuity[6].

Hyponatremia manifests in a wide range of patients. It affects both sexes and all age groups equally, but it is most frequently observed in elderly individuals due to an increased prevalence of comorbidities that can reduce serum sodium levels, such as cardiac, hepatic, or renal failure[7-9].

Patients who have a serum sodium concentration of 130 mEq/L or higher are typically asymptomatic, while those with lower values may experience symptoms. Anorexia, nausea, and vomiting are typically the initial symptoms of hyponatremia. Some patients may experience irritability and headaches. Patients develop neuropsychiatric symptoms as serum sodium levels continue to decrease. Restlessness, altered consciousness, lethargy, seizures, and coma comprise these

symptoms. Hyponatremia and its management are associated with severe neurological sequelae[10].

The diagnosis of hyponatremia is challenging to establish due to the significant variation in symptomatology. Accurate history-taking, clinical examination, and a variety of investigations are necessary for a comprehensive evaluation of hyponatremia. The early identification and optimal management of hyponatremia in hospitalized patients may reduce in-hospital mortality and symptom severity, enable less intensive hospital care, reduce the duration of hospitalization and associated costs, and enhance the treatment of underlying co-morbid conditions and the quality of life of the patient.

Depending on the patient's volume status, hyponatremia is not a uniform condition; it can be classified as either euvolemic, hypervolemic, or hypovolemic11. Different treatment strategies are necessary for each type and etiology of hyponatremia. Depending on the region, the etiological profile may be influenced by the climate, demography, personal behaviors, health education, availability of health care services, and disease distribution pattern of the associated systemic illness.

Consequently, this investigation was conducted to ascertain the prevalence, varieties, and consequences of hyponatremia at the time of admission in patients who were visiting our tertiary care hospital, which is situated in the southern region of Rajasthan.

REVIEW OF LITERATURE

A serum sodium content of less than 135 mEq/L is considered hyponatremia, albeit the precise definition varies according on the set values used by the particular laboratory. Twelve An excess of total body water relative to total body sodium content causes hyponatremia, a common electrolyte imbalance.[12] Edelman supported the idea that the ratio of total body solutes (such as total body potassium and sodium) to total body water determines serum sodium concentration rather than total body sodium.[13] An imbalance in this ratio, where the total body water exceeds the total body solutes, is what defines hyponatremia. One-third of total body water (TBW) is made up of extracellular fluid (ECF), with the other two-thirds being made up of intracellular fluid (ICF). Whereas potassium is the main solute in ICF, sodium is the main solute in ECF.

ETIOLOGY

The etiology of hyponatremia can be categorized according to the volume status of the extracellular fluid. Sodium is the predominant solute in extracellular fluid (ECF). A patient can be categorized as hypovolemic, euvolemic, or hypervolemic based on the volume of extracellular fluid (ECF)[14].

Hyponatremia may be caused by physiological causes that cause vasopressin release in conjunction with increased fluid intake. An increase in vasopressin secretion may result from hypothyroidism and adrenal insufficiency. Hypovolemic hyponatremia, or intravascular volume depletion, and hypervolemic hyponatremia, or decreased effective intravascular volume, are physiological stimuli for vasopressin release. The etiologies of different forms of hyponatremia are delineated below:

Drugs causing hyponatremia are following:

- Analogs of vasopressin, such as oxytocin and desmopressin
- Several antidepressants, morphine, other opioids, selective serotonin reuptake inhibitors, and other medications can either increase the effects of vasopressin or cause its release.
- Thiazide diuretics are among the medications that prevent urine dilution.
- Carbamazepine and its analogs, vincristine, nicotine, antipsychotics, chlorpropamide, cyclophosphamide, and nonsteroidal anti-inflammatory drugs are among the drugs that cause hyponatremia.
- Illegal drugs like ecstasy or methylene dioxy-methamphetamine (MDMA)[20]

Table 1: Causes of Hyponatremia Based on Types

Hypovolemic Hyponatremia (A reduction in total body sodium is more significant than a fall in TW.)[5]
• Loss of digestive fluids (vomiting or diarrhea) • The third fluid interval (small bowel obstruction, hypoalbuminemia, and pancreatitis) • Diuretics • Osmotic dieresis (mannitol, glucose) • Nephropathies caused by salt waste • Urinary salt wasting, which may be brought on by elevated brain natriuretic peptide, is known as cerebral salt wasting syndrome. • Deficiency in mineralocorticoids
Hypervolemic Hyponatremia (TBW rises more than the total sodium in the body does.)[16]
• Nephrotic syndrome, acute renal failure, and chronic renal failure are examples of renal causes. • causes outside the kidneys (cirrhosis, congestive heart failure) • Iatrogenic
(TBW rises when total body sodium remains constant.)
Non-osmotic, As with euvolemic hyponatremia, pathologic vasopressin release can happen when volume status is normal. Causes of euvolemic hyponatremia include: • Drugs • Syndrome of inappropriate antidiuretic hormone(SIADH) • Addison's disease • Hypothyroidism • High fluid intake in conditions like primary polydipsia; or potomania, caused by a low intake of solutes with relatively high fluid intake • Excessive fluid-related medical testing, like heart catheterization or colonoscoy[17-19] • Iatrogenic

EPIDEMIOLOGY

Hyponatremia is the predominant electrolyte condition, occurring in 20% to 35% of hospitalized patients. Hyponatremia is prevalent among critically ill patients in the intensive care unit (ICU) and postoperative individuals. This phenomenon is particularly prevalent among elderly patients because to several comorbidities, polypharmacy, and limited access to meals and beverages[19].

PATHOPHYSIOLOGY

Blood pressure (BP), fluid and electrolyte balance, and the preservation of normal cellular homeostasis all depend on sodium, an essential nutrient. Because of its significant osmotic activity, it plays a critical function in regulating the volume of ECF. It is also essential for the excitability of muscle and nerve cells as well as for the movement of nutrients and substrates across plasma membranes[50].

In extracellular fluid, sodium is the most abundant cation [ECF2 (1 mmol, or molar equivalent, amounting to 23 mg of sodium)]. An adult male's average sodium content is 92g, of which 46g are found in the extracellular fluid at a concentration of 135–145 mmol/L, 10 mmol/L in the intracellular fluid, and 35 g in the skeletal system. By actively moving sodium and potassium across the cell membrane against their concentration gradients and using energy from ATP, the sodium-potassium pump preserves the concentration gradient between the extracellular fluid and intracellular fluid. Through certain channels or transport mechanisms, sodium enters the polarized cells of the intestinal wall or renal tubular epithelium from the tubular lumen or gut. A pump that is primarily found on the cell's basolateral surfaces then extrudes the sodium into nearby capillaries. In these cells, the movement of sodium is mostly associated with the movement of other substrates, including glucose, galactose, amino acids, and phosphates.

Sodium absorption transpires nearly entirely in the distal small intestine and the colon. The body's sodium balance is intricately connected to water balance and is meticulously regulated by the kidneys. The sodium filtered by the glomeruli is reabsorbed in a range of 0.5% to 10%, contingent upon tubular requirements, with angiotensin II, norepinephrine, aldosterone, and insulin promoting reabsorption, while dopamine, cAMP, cardiac natriuretic peptides, and prostaglandins induce a natriuretic effect. Typically, little sodium losses transpire through feces and perspiration; these losses escalate with heightened sodium consumption, but a portion is essential.

An imbalance in water regulation, where there is usually an excess of bodily water in relation to the total sodium and potassium content, is the core characteristic of hyponatremia. Disruptions in vasopressin, sometimes referred to as antidiuretic hormone, which controls water balance, are frequently the cause of this illness. Vasopressin activity is typically required for the development of hyponatremia, even in circumstances involving renal salt loss. Serum sodium levels and osmolality are controlled by the stimulation of thirst, the release of antidiuretic hormone (ADH), and renal control of filtered salt. The normal range of plasma osmolality is between 275 and 290 mOsm/kg. Changes in sodium concentration and related anions are the main cause of variations in serum osmolality. Distinguishing "effective osmolality" or tonicity from "total osmolality" is crucial. Total osmolality denotes the concentration of all solutes per unit of water, regardless of their capacity to traverse biological membranes.

Effective osmolality or tonicity refers to the osmoles that affect water flow between intracellular and extracellular compartments. This notion relies on the permeability of the membranes that delineate these fluid compartments. Effective solutes, such as sodium, generate osmotic pressure gradients across cell membranes, facilitating the transport of water between intracellular and extracellular compartments.

To sustain appropriate osmolality, water consumption must equal water elimination. An imbalance between water intake and excretion results in hyponatremia or hypernatremia. The thirst system regulates water intake, with osmoreceptors in the hypothalamus activating thirst when body osmolality attains 295 mOsm/kg. Antidiuretic hormone (ADH), which is made in the hypothalamus and kept in reserve in the posterior pituitary gland, carefully controls water excretion. ADH secretion is either increased or inhibited by variations in tonicity. Renal water reabsorption is caused by increased ADH secretion, whereas the opposite occurs when it is decreased. Although they are less sensitive than osmoreceptors, baroreceptors in the carotid sinus can cause the release of ADH. In reaction to pharmacological drugs, pain, nausea, stress, and decreased effective circulation volume, baroreceptors trigger the release of ADH[21].

Cells are fundamentally immersed in a solute solution of water, potassium, and sodium. Sodium is predominantly extracellular and expelled from the cell via Na+-K+ ATPase in favor of potassium, which is mainly intracellular. Water traverses the cell membrane through aquaporin channels; under normal physiological conditions, osmotic gradients are rapidly dissipated by the diffusion of water across the membrane[43].

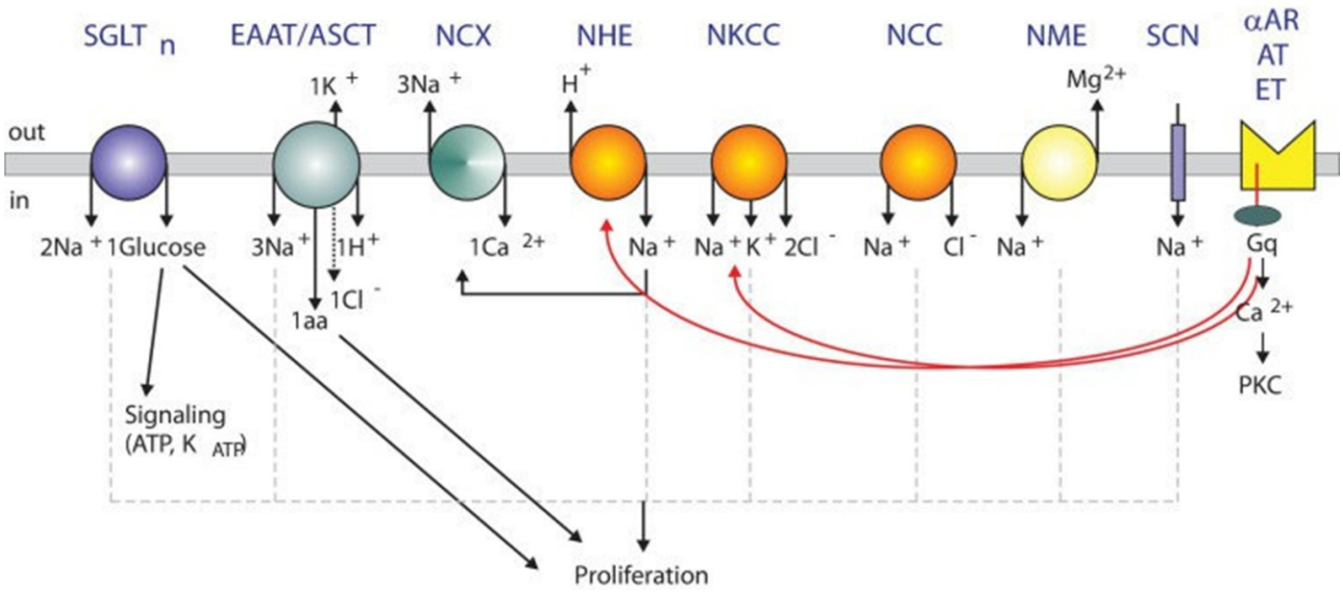

Figure 1: Sodium Dependent Transporters

Sodium readily passes through capillary membranes in peripheral blood vessels. However, in brain capillaries, tight endothelial junctions prevent sodium from crossing. Alterations in sodium levels cause water to move in to brain cells due to this restriction on sodium diffusion out of the cells. This can result in brain cell swelling or shrinkage in response to changes in plasma sodium levels.

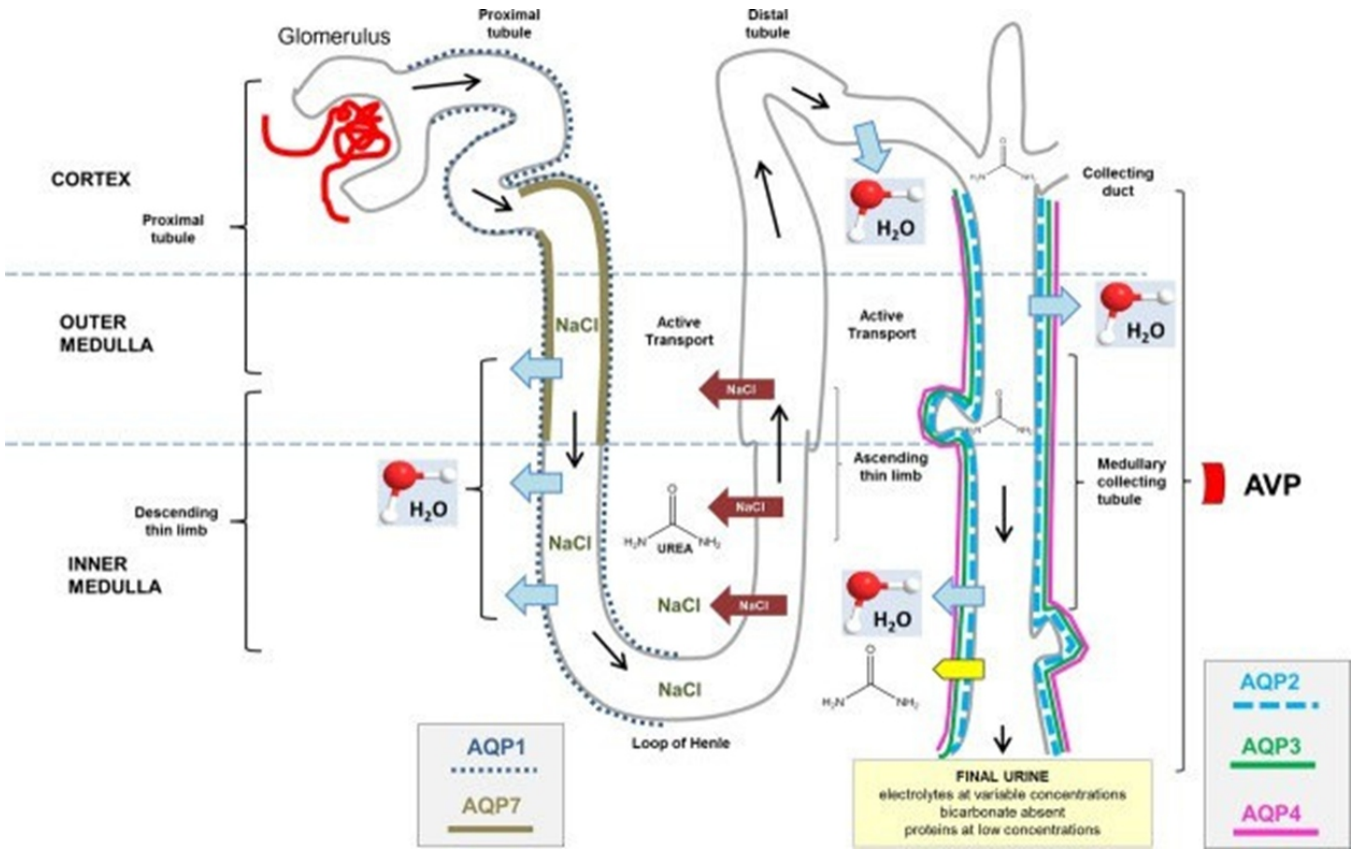

Figure 2: Nephron and Collecting Duct Anatomy, as well as the Distribution of several Aquaporins (AQPs) in the Kidneys that are impacted by Vasopressin (AVP).
Water and Sodium chloride (NaCl) Reabsorption Sites are shown.
In the Collecting Duct, Type-intercalated Cells' Internal Vesicle Membranes contain AQP6[46].

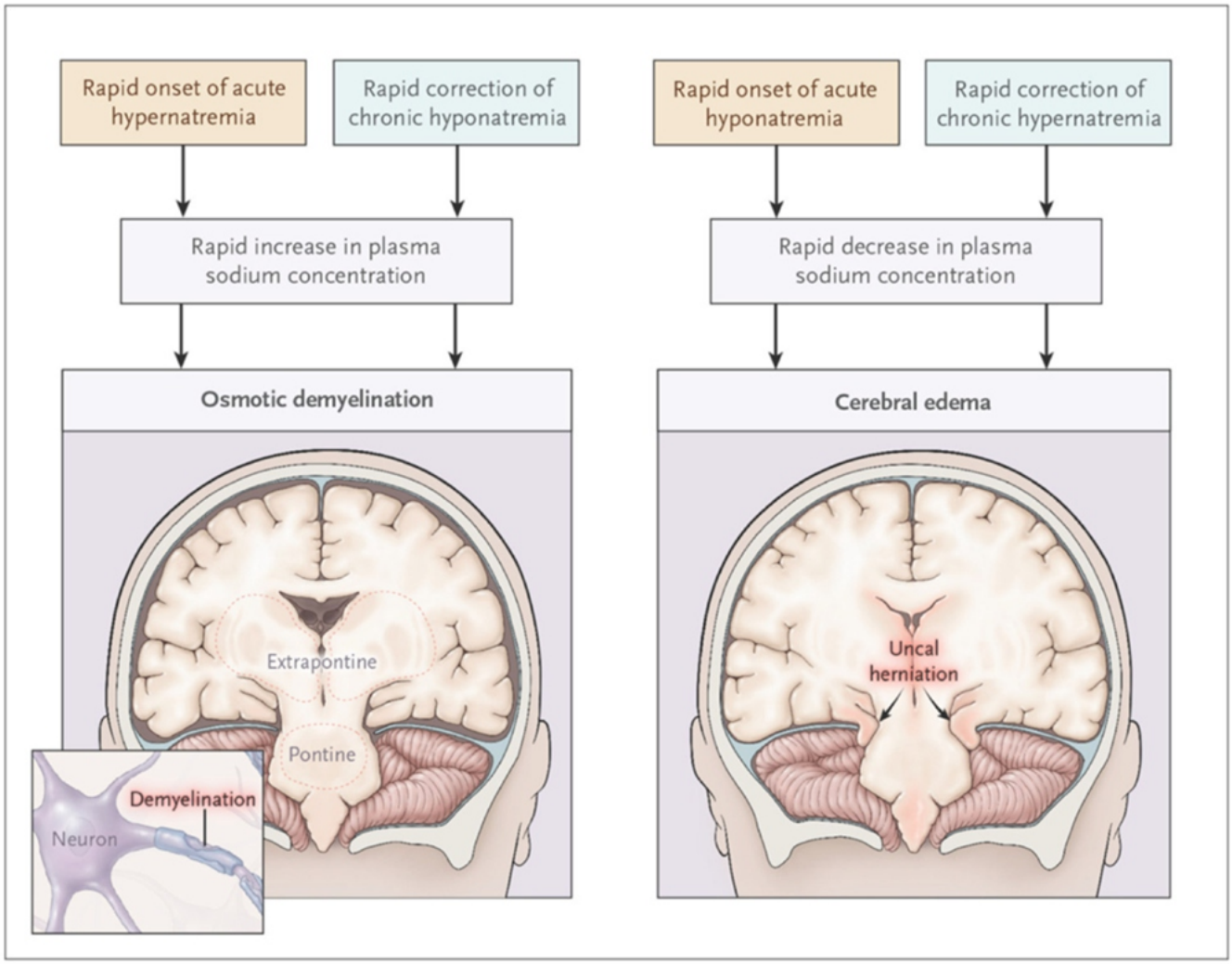

Figure 3: Consequence of Rapid Change in the Plasma Sodium Concentration

Table 2: Types of Hyponatremia based on Tonicity

Hypertonic Hyponatremia (Serum osmolality exceeding 290 mOsm/kg)
• Hyperglycemia • Mannitol
Isotonic Hyponatremia Serum osmolality ranging from 275 mOsm/kg to 290 mOsm/kg
• A laboratory artifact is pseudo-hyponatremia. Hypertriglyceridemia, hyperproteinemia (monoclonal gammopathy, intravenous immunoglobulin [IVIG]), and cholestasis (lipoprotein X) are the most frequent causes. Because two-thirds of clinical labs still employ direction-selective electrode technology, this problem still exists.
• Non-conductive irrigation solutions containing sorbitol, glycine, or mannitol are used in urological and gynecological operations, such as transurethral resection of the prostate (TURP).[22]
Hypotonic Hyponatremia (Serum osmolality of less than 275 mOsm/kg)
An excess of free water is represented by hypotonic hyponatremia. There are two possible causes for this excess free water: • Increased intake of free water: The patient consumes more free water than the kidneys can eliminate (more than 18 liters per day or more than 750 milliliters per hour). Psychogenic polydipsia, marathon running, water - drinking contests, and ecstasy are a few examples of this. • Decreased free water excretion: Patients ingest abnormal volume of free water, yet the kidneys cannot eliminate the water for whatever reason.

There are three mechanisms involved in the inability of kidneys to excrete water:

1. **Elevated ADH Activity: High ADH can result from three distinct mechanisms:**
   - Decreased effective arterial blood volume (EABV): Antidiuretic hormone (ADH) is released when the EABV is lowered by 15% or more. This occurs when there is hypovolemia (as in vomiting or diarrhea), decreased cardiac output (as in heart failure), or vasodilation (as in cirrhosis).
   - SIADH: Autonomous secretion of ADH. Brain diseases, lung disorders, medications (like SSRIs), and other conditions (including pain and nausea) are the four main causes of this. Deficiency in cortisol: Cortisol inhibits the release of ADH. Large amounts of ADH are released when cortisol levels are lowered. This process is due to adrenal insufficiency[23] .
   - Deficiency in cortisol: Cortisol inhibits the release of ADH. Large amounts of ADH are released when cortisol levels are lowered. The mechanism is due to adrenal insufficiency[23].
2. **Low Glomerular Filtration Rate (GFR):** The kidney's capacity to eliminate water would be hampered by a low GFR. Acute kidney injury (AKI), chronic kidney disease (CKD), and end-stage renal disease (ESRD) are common examples.

3. **Low Solute Intake**: Patients on a normal diet eat 600 mOsm to 900 mOsm of solute every day. When it comes to water, solutes are substances that the glomeruli may effortlessly filter but that the tubules find either entirely or partially difficult to reabsorb. The two main solutes are urea, which is created during protein metabolism, and electrolytes, including salt. The solute burden is not influenced by carbohydrates. Solute intake and urine solute burden are equal under steady-state conditions. As a result, 600–900 mOsm of solute should be excreted in the urine by these patients. The urine solute load affects urine volume and, consequently, water excretion. The volume of urine produced must increase with the amount of solute that must be eliminated. The volume of urine that must be produced decreases with the amount of solute that must be eliminated. Under steady-state conditions, patients who consume a small quantity of solute daily (e.g., 200 mOsm/day) will also excrete a small amount of solute in their urine, which means they will do so in a lesser volume of urine. The kidneys' ability to eliminate water will be restricted by this reduced urine volume. The tea-and-toast diet and beer potomania are typical instances of this.

SIADH (Syndrome of Inappropriate Antidiuretic Hormone Secretion)[24]

This disease results in hyponatremia due to poor kidney water excretion caused by incorrect ADH secretion despite normal or increasing plasma volume.

Since there is not a single test that can validate the diagnosis, SIADH is an exclusionary diagnosis. The patients are euvolemic[25] and hyponatremic.

Causes of SIADH :

- Any condition affecting the central nervous system (CNS)
- Ectopic ADH production (most often lung small cell cancer)
- Medication (including carbamazepine, oxcarbazepine, and chlorpropamide)
- HIV
- TB and pneumonia are examples of pulmonary illnesses.
- Patients recovering from surgery (pain medication)

Treatment includes fluid restriction and the use of vasopressin-2 receptor inhibitors[26,27].

EVALUATION

The severity and chronicity of hyponatremia determine the symptoms. Minimal symptoms are seen in patients with mild-to-moderate hyponatremia (more than 120 mEq/L) or a progressive drop in sodium (longer than 48 hours). Individuals who have severe hyponatremia (less than 120 mEq/L) or a sharp drop in sodium levels exhibit a wide range of symptoms[28].

From headache, muscle cramps, nausea, vomiting, and anorexia to altered mental status, agitation, seizures, and even coma, symptoms can vary widely[29].

In addition to symptoms, it is critical to collect a thorough medical history that includes any history of pulmonary and central nervous system illnesses, all prescription drugs taken at home, and any social history of increased beer consumption, MDM usage, or ecstasy use.

Assessing neurological and volume status is part of the physical examination.

To avoid irreversible brain damage, patients with neurological symptoms and indicators must receive treatment right away[26].

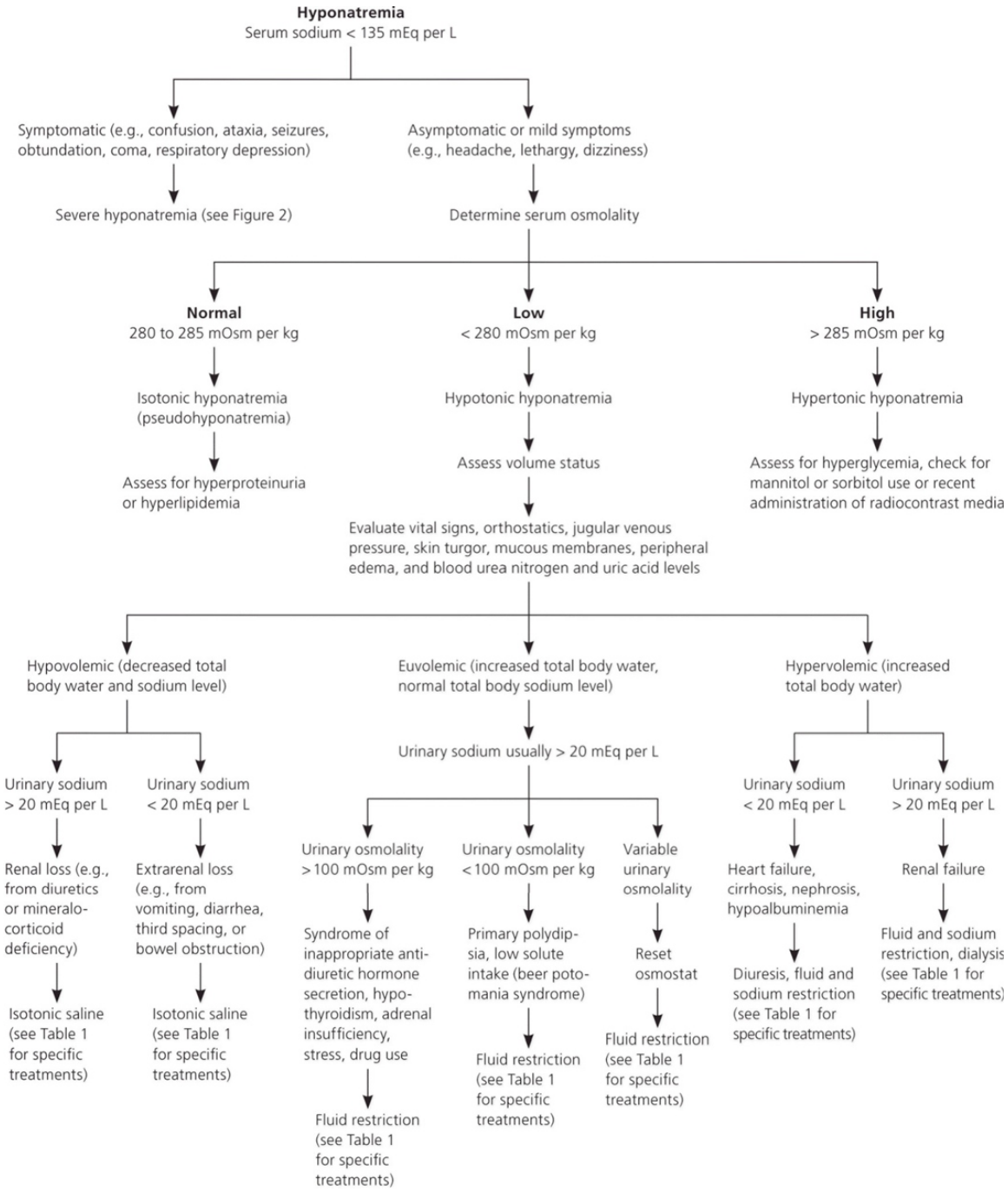

Figure 4: Diagnostic algorithm of Hyponatremia[49]

Table 3: Laboratory Investigations Aiding in Diagnostic Algorithm of Hyponatremia

Plasma Osmolality (275 mOsm to 290 mOsm/kg)	• The distinction between hypotonic, isotonic, and hypertonic hyponatremia can be aided by it. • Patients who are hypotonic are truly hyponatremic.
Osmolality of Urine	• Primary polydipsia or reset osmostatis is indicated by urine osmolality below 100 mOsm/kg. • A high ADH condition is typically indicated by urine osmolality greater than 100 mOsm/kg.
Volume Status (ECF status)	• euvolemic, hypervolemic, and hypovolemic.
Urine Sodium Concentration	• Extra renal fluid loss (from remote vomiting and diuretic treatment) is indicated by urine sodium levels below 10 mmol/L. • Greater than 20 mmol/L of urine sodium indicates renal loss of urine (salt-wasting nephropathies, vomiting, diuretics, and cortisol deficit).
Other Investigations	• Thyroid-stimulating hormone (TSH) in serum • Adrenocorticotropic hormone (ACTH) in serum • Tests for liver function and serum urea • CT scan of the head or computed tomography (CT) scan of the chest

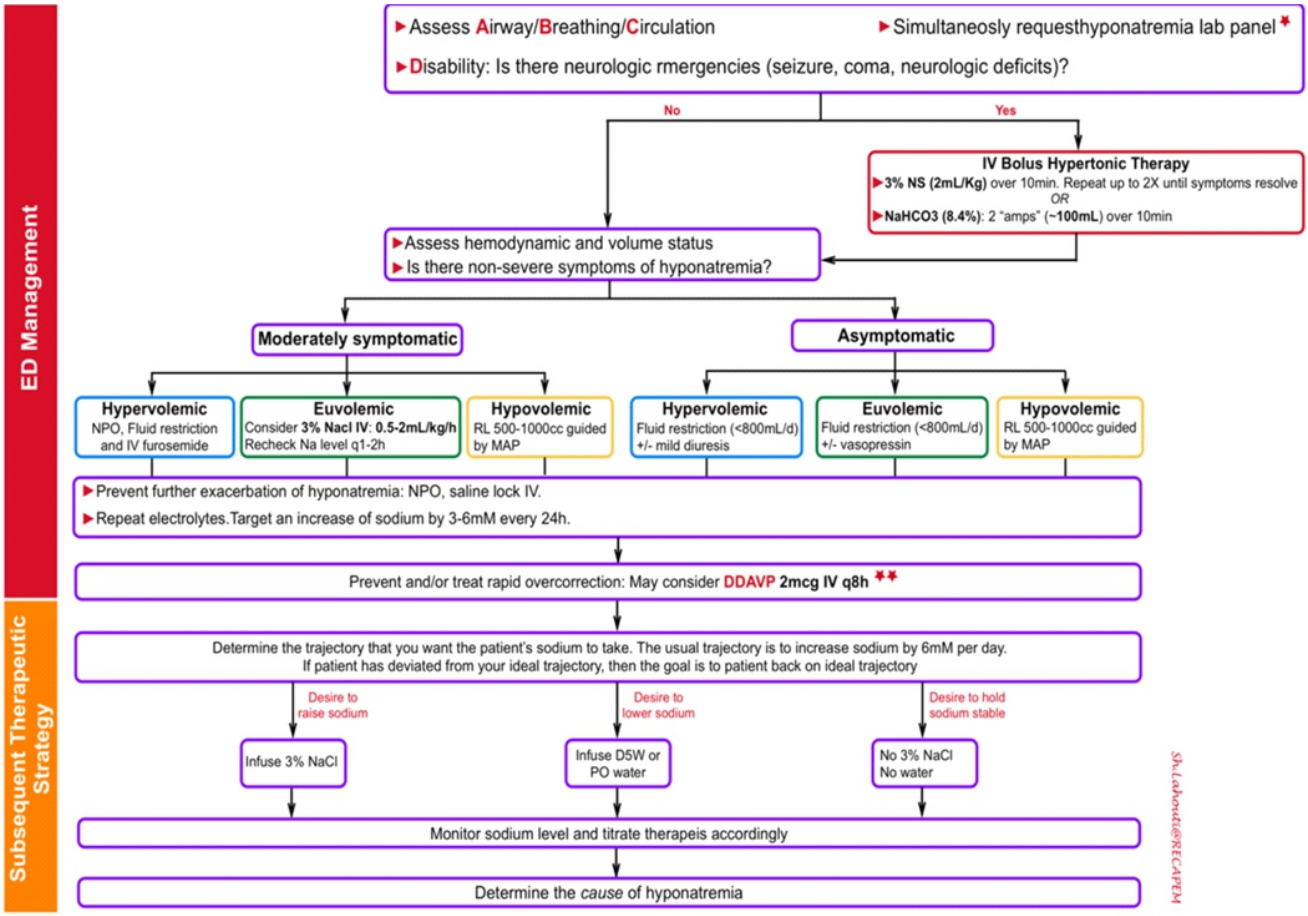

Figure 5: General Approach to Management of Hyponatremia[50] - Acute Symptomatic Hyponatremia

TREATMENT AND MANAGEMENT

The intensity of symptoms, volume status, duration, and degree of hyponatremia all affect how hyponatremia is treated.

In cases of severe hyponatremia, provide a 100 mL intravenous (IV) bolus of 3% sodium chloride (repeat up to thrice if symptoms persist).

Hyponatremia characterized by mild to moderate symptoms: 3% sodium chloride, gradual infusion (compute the infusion rate using the sodium deficiency formula, then recalculate it with regular sodium monitoring).

Chronic Asymptomatic Hyponatremia

- Hypovolemic hyponatremia: the provision of isotonic fluids and the retention of any diuretics.
- Treat the underlying ailment, limit fluid and salt intake, and use loop diuretics to treat hypervolemic hyponatremia.
- Fluid restriction to less than one liter per day is known as euvolemic hyponatremia.

Medication: Recently, selective vasopressin-2 receptor antagonists have been employed. They raise serum sodium levels by increasing the kidneys' excretion of water without influencing sodium levels. If the aforementioned measures are ineffective, these drugs are utilized in patients with euvolemic and hypervolemic situations (apart from liver failure)[30,31].

Correcting sodium by no more than 10 mEq/L to 12 mEq/L in any 24-hour period is the aim of the correction. Osmotic Demyelination Syndrome (ODS) risk factors include alcohol consumption, liver illness, hypokalemia, and malnourishment.

Limit of Correction

- ODS high-risk: less than 8 mEq/Lin over a 24-hour span
- ODS risk is typically less than 10 mEq/Lin over a 24-hour period.

If there is no hypovolemic condition, including postural hypotension, faux hyponatremia, or false laboratory hyponatremia, the next step is to assess the urine's osmolarity and salt content. In the absence of quick water intake and low urine salt levels (less than 100 mOsm/kg), the potential for a high fluid, low protein diet, including beer potomania, should be examined. For patients whose severe hyponatremia is less than 120 mEq/L, the chronicity of the hyponatremia should be considered.

Therefore, intravenous 3 percent saline at a dose of 2 milliliters per kilogram of body weight (e.g., 150 milliliters) over 10 minutes is recommended for severe chronic hyponatremia. If severe symptoms continue, it can be repeated twice as needed.[49] Serum sodium levels rise by 2 mEq/L for every 100 mL of hypertonic saline. doing a thorough electrolyte panel 20 minutes after administering hypertonic treatment. Raising Na levels by roughly 3-5 mM is the goal in order to improve clinical outcomes. If the symptoms continue:

- Consider doing more rounds of hypertonic therapy to reach a total rise of roughly 4-6 mM if the sodium gain is less than 4 mM.
- Hyponatremia may not be the root cause if symptoms continue after a 6 mM increase in salt. To look at additional or different conditions, more assessment is necessary.

To avoid unduly quick correction, desmopressin (dDAVP) should also be given to certain patients.

There is now no evidence of vascular thrombosis or extravasation harm, and 3% saline can be safely administered via a peripheral vein. However, some facilities have policies that forbid peripheral infusion of hypertonic saline. These circumstances necessitate central venous infusions or infusions of a lower concentration at higher infusion rates. Tolvaptan is advised when hyponatremia is associated with increased anti-diuretic hormone (ADH) activity.

For patients with normovolemic hypotonic hyponatremia, fluid restriction is sufficient. Malnourished patients with the syndrome of inappropriate antidiuretic hormone secretion (SIADH) may require a high protein diet because it raises the solute load for renal excretion, which leads to a greater elimination of free water. Patients with SIADH have low serum osmolality (less than 280 mOsm/kg) and hyponatremia (plasma sodium level of less than 135 mEq/L), according to laboratory results. Additionally, urine osmolality (usually above 100 mOsm/L) and sodium levels (higher than 20 mMol/L) are elevated in SIADH patients[32].

Differential Diagnosis

- Hypo osmolality is linked to true hyponatremia. First, it is important to distinguish between the conditions that cause hyperosmolar hyponatremia and iso-osmolar hyponatremia (also known as pseudo-hyponatremia).[33]
- Elevated blood sugar levels
- Overdose of Mannitol
- Elevated cholesterol levels
- Elevated protein levels

Differential Diagnosis for Hypo-Osmolar Hyponatremia

- Gastroenteritis
- Diuretic use
- Congestive heart failure
- Liver failure
- Psychogenic polydipsia
- Renal causes
- SIADH
- Adrenal Crisis
- Hypothyroidism

Prognosis

The severity of hyponatremia and the underlying illness producing it determine the prognosis for patients with this disorder. Patients with acute hyponatremia, severe hyponatremia, and elderly patients have a bad prognosis[34].

Complications

Patients with hyponatremia may experience seizures, rhabdomyolysis, altered mental status, and even coma if they get insufficient or no treatment.

Osmotic demyelination syndrome may result from the quick correction of chronic hyponatremia (more than 10 mEq/L to 12 mEq/L of sodium in 24 hours).

Rapid sodium correction in individuals with persistent hyponatremia[35] can result in osmotic demyelination syndrome, originally termed as central pontine myelinolysis. Within 48 hours, the brain of hyponatremia patients adjusts to a drop in serum sodium levels without experiencing cerebral edema. If the initial salt is greater than 120–125 mmol/L, osmotic demyelination is uncommon[44].

Patients with chronic hyponatremia are therefore typically asymptomatic. Osmotic demyelination syndrome results from the brain's quick sodium correction once it has adapted to low serum sodium levels. Seizures, confusion, and even coma are among the irreversible neurological symptoms that make up the clinical manifestations, which are usually a few days later. Patients who are badly impacted may have "locked-in" syndrome. These patients are conscious, but they can only move or speak with the aid of their eyes[36].

Previous Evidence showing Clinical Spectrum of Hyponatremia

Sood et al. (2020) reported that 1.17% of hospitalized patients had hyponatremia. The average age of the study participants was 62.25±17.7 years. There were 1.25 times as many men as women. The most common neurological symptom was altered

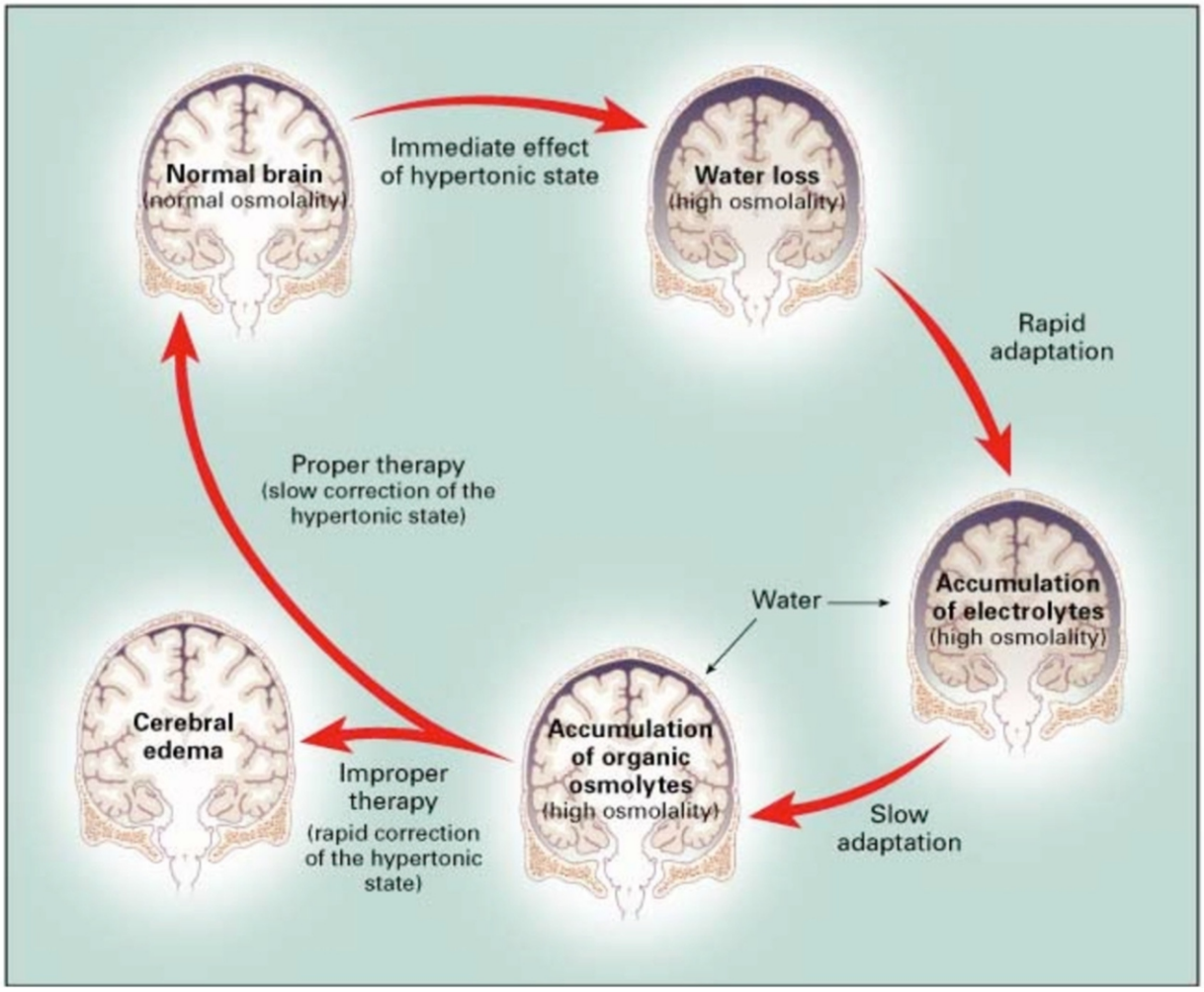

Figure 6: Effects of Hypernatremia on the Brain and Adaptive Responses

sensorium. Ninety percent of the patients had poor osmolarity. 38 (40%) of the 94 patients had euvolemia. Chronic obstructive pulmonary disease (COPD) with cor pulmonale and right-sided heart failure was the most common cause of hypervolemic hyponatremia (n = 9, 31%). The most common cause of hypo-volemic hyponatremia (n = 13, 48%) was acute gastroenteritis. Syndrome of inadequate anti-diuretic hormone secretion (SIADH) was the most common cause of euvolemic hyponatremia (n = 20, 53%). Out of the 106 patients, 11 (10.38%) passed away[37].

A study by Oommem et al. (2019) involved 904 patients who were admitted to AIMS, Kochi. They were divided into three groups (Mild, Moderate, and Severe) according to their serum sodium levels. The most typical manifestation of hyponatremia is altered sensorium. Compared to severe hyponatremia (20%), disorientation was more common in moderate hyponatremia (64%). The primary cause of hyponatremia was identified as the syndrome of inappropriate antidiuretic hormone secretion (SIADH). The most common causes of SIADH were respiratory conditions such as pneumonia, asthma, and obstructive airway disease (OAD). Of the several forms of carcinoma, genitourinary and lung cancer were the primary causes of SIADH. Cellulitis (14%), chest infections (15%), and urinary tract infections (UTIs) (68%) were the infections linked to hyponatremia. Of the 42.0% of people with hyponatremia who had diabetes mellitus, 64% had peripheral neuropathy, and 10% developed necrotizing fasciitis and diabetic foot[8].

One hundred hyponatremia patients were enrolled by Suresh et al. (2017). Of the patients, 46% had no symptoms. Lethargy affected 33% of patients, postural dizziness affected 28%, and aberrant behavior affected 19%. In hospitalized patients, the overall incidence of hyponatremia was 4.58%, however in intensive care unit patients, it was 22.4%. Hypertonic saline infusion was used to treat twelve patients with symptomatic severe hyponatremia, 25% of patients received loop diuretics with oral sodium chloride supplementation for free water excretion in SIADH cases, 44 patients were advised to restrict their fluid intake, 36 patients received oral sodium chloride supplementation, and 64 patients received normal saline. Five of the nine study participants who passed away had advanced liver cirrhosis as their underlying cause. Osmotic Demyelination Syndrome (ODS) occurred in one patient[39].

According to Rai et al. (2017), hyponatremia (serum Na+≤130 mEq/L) occurred in 100 out of 100 cases.The mean sodium level was 118.2 ± 8.1 mEq/L, and 56 patients had severe hyponatremia. 38 patients experienced vomiting, 7 experienced hiccups, 6 experienced seizures, 38 hyponatremic patients were asymptomatic, and 43 patients experienced altered levels of consciousness in the form of lethargy, disorientation, irrelevant talking, or coma. Our study found that euvolemic hyponatremia was the most prevalent kind, accounting for 71% of cases, followed by hypervolemic (27%) and hypovolemic (2%). SIAD was responsible for 94.4% of the euvolemic hyponatremia in our research[40].

In a cross-sectional study of 250 patients aged >18 with hyponatremia (<130meq/l), Mittal et al. (2016) discovered that 154 (61.6%) of the patients had euvolemic hyponatremia, 53 (21.2%) had hypervolemic hyponatremia, and 43 (17.2%) had hypovolemic hyponatremia. Acute gastroenteritis, CLD, and CNS infections were the most frequent causes of euvolemic, hypervolemic, and hypovolemic hyponatremia, respectively. Severe hyponatremia was associated with more neurologic symptoms than mild hyponatremia (69.7% versus 8.1%). 44 patients (17.6%) experienced seizures related to hyponatremia, and all of them had severe hyponatremia.

Mortality was 14% overall. Patients who had severe hyponatremia were more likely to die than those who had mild hyponatremia[41].

In 2012, Chatterjee et al. recruited 201 patients (16.4%) whose serum Na level was less than 135 meq/l. 75 (37.31%) of the patients were female, while 126 (62.69%) were male. Thirty patients (2.4%) had severe hyponatremia (Na < 120 meq/l). The largest proportion of hyponatremic patients were euvolemic [102 (50.74%)] and hypervolemic [54 (26.86%)]. and low blood sugar [45 (22.4%)]. Sixty-six patients fulfilled the criterion of SIADH. The most common underlying risk factor for hyponatremia in our case series was fluid loss from diarrhea or vomiting. During their hospital stay, 13.5% (15/201) of hyponatremic patients died, while 8.5% of normonatremic patients did the same[42].

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