v. 5, n. 14, 2025

International Journal of Health Science

SYNDROMES RELATED TO SODIUM AND ARGININE-VASOPRESSIN ALTERATIONS IN NEUROSURGERY AND NEUROINTENSIVISM

Acceptance date: 25/03/2025

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Abstract: Postoperative dysnatremias, characterized by imbalances in serum sodium levels, have been associated with increased resource use and mortality in surgical and intensive care patients. The treatment of dysnatremias can involve medical interventions based on changes in sodium levels. In this sense, hyponatremia is an important and common electrolyte disorder in critically ill neurosurgical patients that has been reported in association with several different primary diagnoses. Making an accurate diagnosis between syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and cerebral salt loss (CSW) in patients in whom hyponatremia develops is important because treatment differs greatly between the conditions. Whereas, correctly predicting postoperative hypernatremia can alert the postoperative nursing team to initiate preventive procedures such as providing adequate hydration and frequent monitoring and potentially reduce the risk of hypernatremia in postoperative neurosurgery patients. The aim of this article is to develop a literature review on syndromes related to hydroelectrolytic disorders and arginine-vasopressin alterations in the neurosurgical setting. This original article constitutes a bibliographic review, with several original articles, which were observed in scientific databases such as LILACS, *Scientific Electronic Library Online* (Scielo), *National Library of Medicine* (NIH), Nature, Medline, during the period from 2020 to 2024. SIADH is an expanded volume condition, while CSW is a contracted volume state involving renal sodium loss. The treatment for patients with SIADH is fluid restriction and the treatment for patients with CSW is usually salt and water replacement. Therefore, hyponatremia is commonly found in patients who have undergone neurosurgery, specifically those with traumatic brain injury, aneurysmal subarachnoid hemorrhage, recent transsphenoidal surgery for pituitary tu-

mors and postoperative cranial vault reconstruction for craniosynostosis. With regard to hypernatremia, although less frequent, it can have a high incidence after surgical resection such as craniopharyngioma, which can be associated with hypothalamic lesions. It can be concluded that hyponatremia is a serious comorbidity in neurosurgical patients that requires special attention, as its treatment varies according to the cause and its consequences can affect the neurological outcome.

Keywords: Neurosurgery; Hyponatremia; Hypernatremia; Inappropriate Antidiuretic Hormone Secretion Syndrome; Cerebral Salt Loss;

INTRODUCTION

Hyponatremia is the most common electrolyte disorder encountered in clinical practice and has been associated with an increased risk of stroke and ischemic heart disease. Brain adaptation to plasma hypotonicity occurs through the removal of solute to mitigate the additional influx of water into brain cells. Acute hyponatremia begins within 48 hours, usually resulting in severe symptoms, while chronic hyponatremia develops over 48 hours. Metabolic disorders are described as the second cause of stroke mimicry and the most frequent electrolyte disorder found in stroke mimicry is hyponatremia.

Around 21% of patients with severe hyponatremia manifest with neurological features such as confusion, abnormal consciousness, but also focal impairment, even though this is uncommon. Hyponatremia is defined as a serum sodium concentration below 135 mmol/L. It is classified as hypotonic or non-hypotonic hyponatremia (HONG *et al.*, 2022). While a previous study reported that for every 1 mEq/L reduction in sodium level below 135 mEq/L, mortality increases by 23%. In addition, hyponatremia is correlated with perioperative complications, including pneumonia, wound infections and major coronary events and prolonged hospitalization (KINOSHITA *et al.*., 2022)

According to He *et al.*,(2022) the incidence of hyponatremia in stroke has been reported to be between 11% and 35% in the literature, while the discrepancy in serum sodium is the most commonly observed disorder, and can also be described as a marker of worse prognosis and cardiovascular diseases. In the context of acute stroke, the appearance of hyponatremia can be attributed to the administration of hypotonic solutions and medications such as mannitol, inadequate intake of solutes, infections, or complications such as syndrome of inappropriate secretion of antidiuretic hormone (SIADH), cerebral salt loss syndrome and secondary adrenal insufficiency (BARKAS et al., 2023a)such as diabetes, chronic kidney disease and heart failure, along with diuretics, antidepressants and proton pump inhibitors are the most common causes of hyponatraemia in community. In the setting of acute stroke, the emergence of hyponatraemia might be attributed to the administration of hypotonic solutions and drugs (ie. mannitol and antiepileptics. In relation to drug intake, Gupta *et al.*,(2015) reported that treatment with esclicarbazepine acetate for focal generalized eplepsia can cause hyponatremia in around 1.3% of patients.

Meanwhile, the severity of the neurological symptoms associated with hyponatremia remains the most clinically relevant indication for immediate treatment. The symptoms of hyponatremia can range from mild and non-specific to severe and fatal, such as headache, fatigue, nausea, dizziness, gait disturbances, muscle cramps, falls and attention deficits. However, if left uncorrected, patients can develop moderate to severe symptoms such as seizures, coma or death (MEZZINI *et al.*., 2023)

Furthermore, among the hydrolytic disorders most commonly seen in neurosurgery, the basic mechanisms involved in hypernatremia are water deficit and solute excess. Hypernatremia is usually associated with hypovolemia, which can occur in conditions that cause combined water and solute loss, where water loss is greater than sodium loss or free water loss. Combined loss can be seen in extra-renal conditions such as gastroenteritis, vomiting, prolonged nasogastric drainage, burns and excessive sweating. Renal losses can be seen in intrinsic kidney disease, post-obstructive diuresis and with the use of osmotic or loop diuretics. Hyperglycemia and mannitol are common causes of osmotic diuresis. Free water loss is seen with central or nephrogenic diabetes insipidus (DI) and also in conditions with increased insensible loss (WU *et al*., 2022). Therefore, the aim of this article is to develop a literature review on syndromes related to hydroelectrolytic disorders and arginine-vasopressin alterations in the neurosurgical field.

METHODOLOGY

This original article is a bibliographical review of several original articles, which were looked at in scientific databases such as LILACS, *Scientific Electronic Library Online* (Scielo), *National Library of Medicine* (NIH), Nature and Medline. The terms used in this study referred to therapeutic and innovative methods on neuroplasticity and neuropsychiatric disorders. The descriptors used in this research were: "Hyponatremia", "Hypernatremia", "Cerebral Salt Loss Syndrome", "Syndrome of Inappropriate Secretion of Antidiuretic Hormone", "Diabetes Insipidus".

The data was collected between 2020 and 2024, over the last 4 years. The inclusion criteria were studies available in full and free online, the articles used were originals such as literature reviews, randomized and double-blind studies, systematic reviews on different therapeutic methods, their technologies and the impact of their interventions, articles in Portuguese and English were used. While the exclusion criteria for this article were the exclusion of duplicate articles, incomplete works, paid-for works and articles that were not in English and Portuguese. A total of 82 original articles were found, of which 37 were used to develop this article.

RESULTS AND DISCUSSION

HYPONATREMIA IN NEUROSURGICAL PATHOLOGIES

Dynatremias, such as a post-operative decrease in sodium, were correlated with various complications, such as deep vein thrombosis, pneumonia, intracranial infection, urinary infection, seizures, myocardial infarction and prolonged hospital stay. On the other hand, post-operative increases in sodium were associated with acute kidney injury, deep vein thrombosis, pneumonia, intracranial infection, urinary infection, surgical site infection, seizures, myocardial infarction and prolonged hospital stay (LI *et al*., 2024)

Tonicity disorders, hyponatremia and hypernatremia are common in neurosurgical patients. Tonicity is detected by the circumventricular organs, while volume status is detected by the kidneys and peripheral baroreceptors, which are integrated in the hypothalamus. The drop in serum sodium concentration results in a proportional drop in extracellular osmolarity, so water will flow from the extracellular to the intracellular compartment, which will reduce the compartment, this can result in cellular edema and consequent rupture (GANKAM KENGNE, 2023). Consequently, brain cells have developed significant mechanisms to prevent the increase in cell volume during hyponatremia. These mechanisms are collectively called regulatory volume decrease

mechanisms, because their goal is to restore the initial volume after hypotonicity-induced swelling (GANKAM KENGNE; DECAUX, 2018). While, hyponatremia can be described as the most common electrolyte disturbance in stroke patients and can exacerbate seizures or cerebral edema in the acute phase of stroke (QIAN *et al*., 2025). While, according to Abla *et al*, (2011) vasopressin can be released from damaged hypothalamic-neurohypophyseal tracts and the posterior pituitary gland, which can cause isolated postoperative hyponatremia.

In this sense, the disturbance of internal homeostasis can lead to permanent damage to biological functions and potentially fatal events, while the maintenance of normal neurological function requires precise distribution and concentration of electrolytes. With regard to patients with subarachnoid hemorrhage, hyponatremia can occur due to elevated plasma concentrations of vasopressin, or due to natriuretic peptides, consequently, the reduction in the concentration of sodium ions in the blood also affects the tone and regulation of cerebral blood vessels, In these patients, acute aneurysmal subarachnoid hemorrhage (SAH) can occur, associated with episodes of hyponatremia during treatment in the intensive care unit (ICU), while hyponatremia can be caused mainly by cerebral salt-wasting syndrome (CSWS) or syndrome of inappropriate secretion of antidiuretic hormone (SIADH) (KIENINGER *et al*., 2021). In addition, according to He *et al*, (2022) stroke--related hyponatremia can be caused by adrenal insufficiency due to pituitary ischemia or hemorrhage, SIADH and cerebral salt loss.

According to Qian *et al*.,(2025), malignant cerebral edema (MCE) is a complication of rapid neurological deterioration and correlates with cerebral herniation. Furthermore, even when procedures such as osmotic craniectomy or enhanced decompression are performed, there can be significant neurovascular and symptomatic implications for the patient. Hyponatremia is the most common electrolyte disorder in stroke patients and can exacerbate seizures or cerebral edema in the acute phase of the stroke. It can be seen that ischemia-induced altered permeability of the blood-brain barrier leads to greater disruption of intracerebral electrolyte homeostasis, and brain swelling can cause further ischemia by increasing intracranial pressure and decreasing capillary perfusion; there may also be an increase in intracellular Ca2+ levels and in the production of reactive oxygen species, which would reduce the recoverability of penumbral tissue.

In a complementary way, Pelouto *et al*.,(2024) describe that acute reperfusion therapy performed in patients with ischemic stroke aims to preserve healthy areas, preventing the conversion of the penumbra into an infarct core. Tissue viability can be assessed using perfusion computed tomography (PTC), which allows the ischemic core and penumbra volume to be distinguished and quantified.

According to Barkas *et al*, (2023) patients who have suffered a stroke with low serum sodium levels may show rapid correction of chronic hyponatremia, which can lead to cerebral dehydration and increased neurological damage that manifests itself as a biphasic disease called osmotic demyelination syndrome (ODS), Therefore, caution should be exercised in relation to serum Na+ concentrations of less than 106 mEq/l if the patient also has a history of hypokalemia, alcoholism, malnutrition and advanced liver disease, as these are predisposing factors.

Preoperative hyponatremia can be associated with dysregulated immune function and high rates of postoperative infection, warranting careful monitoring and observation in the perioperative period. Patients who have suffered a subarachnoid hemorrhage after

International Journal of Health Science ISSN 2764-0159 DOI https://doi.org/10.22533/at.ed.1595142512037

an aneurysm may have preoperative hyponatremia, which represents an increased risk of postoperative pneumonia and prolonged hospitalization. In this sense, treatment can be carried out through microsurgical clipping or endovascular coiling. Regarding hyponatremia, the guidelines relate maintenance of euvolemia and maintenance of hypertension as therapy (GANKAM KENGNE; DECAUX, 2018). In stroke, hyponatremia is mainly hypo-osmolar, among other factors it can also be caused by SIADH or CSW. In SIADH, there is an uncontrolled secretion of antidiuretic hormone (ADH) from the posterior pituitary gland in response to stimulation from the hypothalamus, which leads to hypotonicity of the body fluid and increased blood volume, while in CSW, large amounts of sodium are lost in the urine (EHTESHAM *et al.*., 2019)

Electroencephalography (EEG) studies can be used, according to Suárez *et al*., (2024) EEG can show disturbances in brain activity similar to those observed in epilepsy, and can only provide data from cortical areas. Consequently, in relation to these patients, therapeutic measures such as the use of hypertonic saline boluses to rapidly correct serum sodium levels can be carried out, as corrections as small as 5 mmol/L can improve symptoms and achieve seizure control. According to Barkas *et al.*,(2023) hypertonic saline solution of 3% NaCl is recommended for the treatment of acute or symptomatic hyponatremia, as it is an effective and potentially life-saving treatment for cerebral edema caused by hyponatremia, however, in the case of patients with hypervolemic hyponatremia, hypertonic saline solution can be combined with loop diuretics. Intravenous administration of 150 ml of 3% hypertonic saline solution can be carried out over 20 minutes, two or three times as necessary, until symptoms resolve or a target increase of 5 mmol/l Na+ is reached during the first hour.

According to the aforementioned author, chronic hyponatremia can also be treated with isotonic saline solution or balanced crystalloid solution. The treatment of chronic hyponatremia depends on reducing free water intake and increasing renal excretion of free water. Regarding SIADH, Krisanapan *et al*.,(2023) described the use of vaptans for treatment, although they demonstrate a notable increase in serum sodium concentrations when contrasted in randomized studies with placebos, there is a pronounced likelihood of an overcorrection. However, the risk associated with osmotic demyelination syndrome is not evident. Patients with SIADH have a combination of ADH-induced water retention and secondary solute loss. Therefore, in patients with mild to moderate symptoms, the basis of treatment is the restriction of oral water intake with a target of less than 800 mL/day. If hyponatremia is persistent, sodium chloride as an intravenous saline solution can be administered, loop diuretics such as furosemide at a dosage of 20 mg twice a day can also be added, as they help to decrease urine concentration and therefore increase water excretion (PINKHASOV *et al*., 2021). However, caution should be exercised, as no more than 8 mEq/L per 24 hours or 0.5 to 1 mEq/L per hour should be exceeded, as faster correction can result in osmotic demyelination of the central nervous system (CNS), leading to serious lethal complications, such as osmotic demyelination syndrome (KRISANAPAN *et al.*., 2023)

In neurosurgical procedures, such as intracranial surgery, a variety of postoperative complications occur, such as postoperative hyponatremia, the symptoms of which include a depressed mental state and lethargy, and can progress to convulsions and death. Hyponatremia can occur after surgery for lesions in the sellar and suprasellar region, in the context of patients with subarachnoid hemorrha-

ge with aneurysm and after craniotomies for other supratentorial lesions. Other potential causes of postoperative serum sodium disturbance include adrenocortical insufficiency, hypothyroidism, hypercorrection of hypernatremia and volume overload. (CALDWELL *et al.*, 2024). Upadhyay and Gormley (2012) also reported that neurological symptoms in patients with acute hyponatremia occur as a result of cerebral edema, can range from nausea and malaise to headache, lethargy, obnubilation and eventually seizures, coma and respiratory arrest can occur as the serum sodium concentration progressively falls below 120 meq/L, while non-cardiogenic pulmonary edema has also been described.

According to Castle-Kirzbaum *et al*,(2022) in transfenoid access surgery to remove pituitary tumors, SIADH can occur as a result of iatrogenic injury to the neurohypophysis during surgical exploration and leads to degeneration of magnocellular neurons and excessive release of antidiuretic hormone, the tumor can extend into the cavernous sinus, suprasellar space, sphenoid sinus or clivus. Complementary secondary CWS syndrome is also a common post-surgical complication, due to the increased release of atrial and brain natriuretic peptide.

The incidence of delayed hyponatremia in post-surgical patients with pituitary adenomas is between 4% and 23%, and the larger the tumor, the greater the rupture of the pituitary stalk during surgery and the greater the likelihood of developing delayed hyponatremia (LIN *et al.*, 2022), which is more common in elderly patients, while in children hyponatremia can be present in 25% of children after craniotomy (KEATING *et al.*, 2023). Preoperative hydrocephalus as an independent risk factor for hyponatremia after craniotomy. The main clinical manifestation of SIADH is delayed hyponatremia due to the uncontrolled release of ADH secretion following damage to the hypothalamic-neurohypophyseal system, leading to water retention, so when a pituitary adenoma grows, it can compress the pituitary stalk, resulting in its chronic distortion and can lead to changes in the position of the posterior pituitary gland, abnormal ADH secretion can occur from the second day after the procedure (LEE *et al.*., 2021)

With regard to other surgical procedures, craniotomy is also among the surgeries known to induce post-surgical hyponatremia, even without subarachnoid hemorrhage or rupture of the sellar region. Similarly, according to Lonjaret *et al*,(2017) conducted a study that analyzed postoperative complications and 30-day readmissions in 243 patients undergoing craniectomy or craniotomy; however, they did not identify any patients with sodium abnormalities as a complication or reason for readmission.

HYPERNATREMIA IN PRE- AND POST-SURGICAL PATIENTS

Hypernatremia is defined as serum sodium of more than 145 mmol L-1. It is less common than hyponatremia in neurosurgical patients or those with neurological disease. In most cases, patients with hypernatremia have undergone pituitary surgery, HSA or traumatic brain injury (TBI). Most patients are asymptomatic, and the analysis of serum electrolytes in the preoperative evaluation clinic may be the first indication of sodium impairment. Consequently, patients who undergo craniotomy are particularly susceptible to the effects of hypernatremia, which can cause dehydration and cell shrinkage, leading to abnormal brain cell shape and function, disrupt osmolarity in the brain, causing cerebral edema and further compromising brain function. This can result in neurological symptoms such as confusion, seizures and coma (LI et al., 2024b).

In patients with craniopharyngioma who underwent operative care, the incidence of hypernatremia observed was 53.4%, with a higher complication for Puget III classification, other factors such as preoperative adrenal insufficiency, preoperative hypernatremia, total resection were also associated with a higher risk of developing hypernatremia in these cases (LI *et al*., 2023). Hypernatremia is treated with hypotonic solutions after correction of the volume status as necessary with isotonic solution, with the addition of desmopressin or vasopressin in cases of central diabetes insipidus (BABA *et al*., 2022). While, according to Papadimitriou-Olivgeris *et al*, (2019) described the presence of hypernatremia and aspiration pneumonia in around 24% of 107 patients with subarachnoid hemorrhage, with the highest incidence within 72 hours of admission, with a mortality rate of 40%.

Hypernatremia can be seen in head trauma, due to dehydration caused by involvement of the hypothalamic-pituitary axis, which would lead to central diabetes insipidus, or due to the use of osmotic diuretics and hypertonic saline solutions (CARVALHO, 2019). Therefore, hypernatremia is caused by increased sodium intake, loss of free water or both. The thirst mechanism can be disrupted in critically ill patients because their consciousness is usually disturbed by sedation, delirium, altered mentality and hormonal abnormalities resulting from brain damage.

In addition, hypernatremia can be induced by treating elevated intracranial pressure (ICP) with hyperosmolar therapies such as mannitol and hypertonic saline, and ICP control can be correlated with serum sodium concentration (LEE; AHN; RYU, 2023). This therapy generates an osmotic gradient between the intravascular space and brain tissue, a high ICP is considered to be 5 to 15 mmHg, while intracranial hypertension can be defined as an ICP>20mmHg for a period longer than 5 to 10 minutes, this causes a decrease in cerebral blood flow and consequent perfusion (KAMEL; SCHREIBER; HAREL, 2022). In addition, normalizing tidal volume to maintain arterial carbon dioxide pressure (PaCO2) between 35 and 45 mmHg, and maintaining positive end-expiratory pressure (PEEP) at levels correlated to 5 to 10 mmHg, are essential factors for preventing perivascular acidosis, and for preventing hypoxemia in patients with TBI (CARVALHO, 2019).

After surgery in the pituitary or hypothalamic region, hypernatremia can develop in response to a reduction in ADH levels, in addition to rupture of the supraoptic and paraventricular nuclei of the hypothalamus, pituitary stalk or posterior pituitary gland. According to Kamabu *et al*.,(2023)the consequence of hypernatremia on occurrence of expansive hematoma (EH hypernatremia can be described in 25.2% of patients with traumatic brain injuries, and also the risk was 1.56 times higher of developing hypernatremia in the case of expansive hematoma.

Therefore, the appropriate therapeutic management of hypernatremia consists of identifying the underlying condition and correcting the hypertonicity. The aim of therapy is to correct both serum sodium and intravascular volume, so fluids should be administered orally or by feeding tube whenever possible. In patients with severe dehydration or shock, the initial step is fluid resuscitation with isotonic fluids before free water correction. Cerebral edema should be considered if seizures occur during correction of hypernatremia. This can occur due to rapid changes in osmolality, and the administration of hypotonic fluids should be stopped. The estimated free water deficit should be corrected over 48 to 72 hours with a decrease in serum sodium not exceeding 0.5 meq per hour, with careful monitoring of the rate of correction, urine output and ongoing losses (WU *et al*., 2022)

In this context, in acute symptomatic hypernatremia (within 48 hours) due to sodium overload, a more aggressive rapid correction of plasma sodium (1-2 mmol/L/h drop in the first 6-8 hours, restoring a Na concentration of 145 mmol/L within 24 hours) improves prognosis without increasing the risk of cerebral edema. However, patients with hypernatremia of longer duration (> 48 hours) or unknown should be corrected at a rate of < 0.5 mmol/h. However, several studies in adults have reported that rapid correction rates (> 0.5 mmol/L/h) are not associated with a high risk of mortality and damage (YUN; BAEK; KIM, 2023).

DIABETES INSIPIDUS AND ITS EFFECTS

Diabetes insipidus, both central and nephrogenic, can be hereditary or acquired. Central diabetes insipidus is considered the main cause of hypernatremia in patients with brain damage (CHRIST-CRAIN; WINZELER; REFARDT, 2021), because brain damage involving the hypothalamus-pituitary gland can cause decreased ADH secretion and damage to the osmoreceptor or thirst center, resulting in a large amount of urine discharge with low sodium content, causing hypernatremia, the increase in blood sodium caused by central diabetes insipidus is mainly normovolemic hypernatremia. If the thirst center were damaged, it would often cause moderate to severe hypernatremia, with a serum sodium concentration between 155-190 mEq/L (KINOSHITA *et al.*., 2022)

Therefore, identifying the cause of hypernatremia and assessing the patient's volume status are essential factors in the treatment of hypernatremia. Hypovolemic hypernatremia is accompanied by a loss of water and electrolytes. For patients with hemodynamic instability, an isotonic solution such as 0.9% NaCl solution should be used for resuscitation until their vital signs are normal. After hemodynamic stability, hypotonic solution is selected to correct the volume deficiency. Hypervolemic hypernatremia is usually associated with iatrogenic sodium loading, and the first step is to stop the infusion of hypertonic drugs. Alternatively, using diuretics alone to excrete sodium will lead to further water loss, which will aggravate the hypertonic state (ABLA *et al.*., 2011)

Diabetes insipidus is a disorder characterized by the excretion of large quantities of hypotonic urine. The different types of diabetes insipidus can be named: central diabetes insipidus resulting from a deficiency of the hormone arginine vasopressin (AVP) in the pituitary gland or hypothalamus, nephrogenic diabetes insipidus resulting from resistance to AVP in the kidneys, gestational diabetes insipidus resulting from an increase in placental vasopressinase and, finally, primary polydipsia, which involves excessive intake of large quantities of water despite normal secretion and action of AVP.

FINAL CONSIDERATIONS

Hyponatremia is associated with increased morbidity and mortality in hospitalized patients and is common in neurosurgical patients. It is an especially frequent occurrence after TBI, SAH and pituitary surgery. The most common cause of hyponatremia after neurotrauma is SIADH, with acute glucocorticoid insufficiency accounting for a smaller but significant number of cases. CSW is very rare after neurotrauma. Although hyponatremia is often mild and self-limiting, treatment with fluid restriction is generally unsatisfactory, while the use of hypertonic saline is still the treatment of choice for acute symptomatic hyponatremia. It is concluded that more studies are needed to develop the management of patients with hydroelectrolytic disorders in neurosurgery.

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