

Hyponatremia in the Patient with Subarachnoid Hemorrhage

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Abstract: Hyponatremia commonly occurs in patients with aneurysmal subarachnoid hemorrhage. Two mechanisms have been proposed as causes: syndrome of inappropriate anti-diuretic hormone and cerebral salt wasting. Physical examination and laboratory results can assist a clinician in identifying which mechanism is responsible and thus determine proper treatment. When hyponatremia is treated promptly and appropriately, patients' sodium levels return to normal without detrimental effects.

Hyponatremia is seen in 10%–40% of the patients with subarachnoid hemorrhage (SAH) admitted to the neuro critical care unit (NCCU; Mayberg et al., 1994; Woo & Kale-Pradhan, 1997). Controversy exists regarding both cause and treatment. Nonetheless, it is imperative that nurses, whether in an advanced practice role or at the bedside, understand the potential causes and effective treatment. Missing diagnostic clues or providing inappropriate treatment could be detrimental to the patient. This article reviews the probable causes of, suggested treatment for, and the role of the nurse in managing hyponatremia in the patient with SAH.

Hyponatremia

Hyponatremia is a serum sodium concentration level of less than 135 mEq/L (< 135 mmole/L, SI) for at least 1 day (Kurokawa et al., 1996). A value of less than 120 mEq/L is considered a critical value requiring immediate intervention (Larsen, Kronenberg, Melmed, & Polonsky, 2003; Nicoll, McPhee, Pignone, Detmer, & Chou, 2001). Signs and symptoms associated with hyponatremia are secondary to cellular hypo-osmolality. Fever, headache, nausea and vomiting, muscle cramps weakness, and confusion occur when serum sodium values are 115–120 mEq/L. Stupor, seizures and coma are more typically associated with serum sodium values of less than 110 mEq/L (Andreoli, Carpenter, Griggs, & Loscalzo, 2001; Diring, 2001). Sudden decrements in serum sodium are more likely to elicit severe symptoms than a gradual decrease over

days to weeks. In the presence of SAH, hyponatremia can be especially dangerous to patients since low serum sodium has been linked to decreased intravascular volume and vasospasm, one of the leading causes of morbidity and mortality in SAH (Suarez, 2004; Wijdicks, Ropper, Hunnicut, Richardson, & Nathanson, 1991).

Hyponatremia usually occurs several days post-hemorrhage (Mayberg et al., 1994). The incidence is increased in patients with a poor clinical grade of SAH (i.e., a Hunt-Hess grade of 3 or more, indicating severe neurological symptoms at the onset of SAH) and hydrocephalus (Mayberg et al.). The etiology of SAH-related hyponatremia has been controversial for decades. The two most common causal hypotheses are cerebral salt wasting (CSW) and syndrome of inappropriate antidiuretic hormone (SIADH).

Syndrome of Inappropriate Anti-diuretic Hormone

According to Palmer (2000), SIADH is an expansion of extracellular fluid volume resulting from the superfluous release of antidiuretic hormone (ADH) or increased renal sensitivity to ADH. SIADH is characterized by decreased serum osmolality with inappropriate urinary concentration (Larsen et al., 2003). ADH acts on the distal collecting duct tubules resulting in increased water reabsorption and increased intravascular volume (Albanese, Hindmarsh, & Stanhope, 2001; Andreoli et al., 2001). In response to the increased intravascular fluid volume, glomerular filtration rate and renal plasma flow increase, and proximal sodium reabsorption decreases in patients with SIADH. As a result, sodium excretion in the urine increases, resulting in low serum sodium values (Palmer, 2000).

SIADH occurs in patients with SAH for a variety of reasons. Both intracranial hemorrhage and neurosurgery for aneurysmal repair interrupt neuronal communication and hormonal feedback mechanisms (Albanese et al., 2001). The release of ADH can also be stimulated by pain, stress, increased intracranial pressure, and hypovolemic states (Diring, 2001). Drugs such as carbamazepine (Tegretol) and lamotrigine (Lamictal) can enhance the action of ADH (Albanese et al.). Although NCCU patients have multiple stimuli for the release of ADH, the physical examination and fluid status of many patients with decreased sodium levels is not always consistent with what would be expected in SIADH (Diring; Palmer, 2000). Thus, clinicians and researchers looked for other explanations; CSW has been proposed as a more immediate cause of hyponatremia in SAH patients.

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Cerebral Salt Wasting

CSW is a transient phenomenon in which kidneys are unable to conserve sodium. CSW leads to serum hyponatremia and hypovolemia as a result of SAH or other intracranial disease (Harrigan, 2001). CSW was first described in 1950, after symptoms of volume contraction and decreased sodium in neurological patients were noted (Peters, Welt, Sims, Orloff, & Needham, 1950). Since that initial observation, clinical and experimental data suggest that patients with SAH and other intracranial diseases experience hypovolemia rather than the euvoolemia or hypervolemia of SIADH. Derangements of sympathetic nervous system stimulation of kidneys, production of digoxin-like peptides, and excess natriuretic factors have all been implicated in CSW.

A surge of sympathetic nervous system (SNS) hormones, norepinephrine and epinephrine, may cause renal sodium excretion. Stimulated by the stress response during brain injury, the SNS hormones stimulate both arterial and venous contraction, leading to increased preload, inotropy, and systemic blood pressure. The kidneys could respond to these cardiovascular changes with a pressure-induced natriuresis (Singh et al., 2002). This neural mechanism of hyponatremia is not the most likely scenario, though it may be a contributing factor. Although a pressure natriuresis is associated with an acute stress response, ongoing sodium excretion is less likely with sustained SNS stimulation in the presence of hypovolemia unless renal vasodilation is also sustained. It may be that the initial brain injury and sympathetic response may, instead, contribute to the development of CSW.

Two additional molecular factors have been implicated in the onset of hyponatremia. One potential factor causing CSW is a digoxin-like peptide that has been found in the plasma of a series of patients with SAH. How this peptide causes renal sodium excretion has yet to be elucidated, but it was determined that infusing digoxin-specific antibodies directly into the ventricles of the rat brain, blocks the central nervous system response to natriuresis (Wijdicks, Vermulean, van Brummelen, den Boer, & van Gijn, 1987).

A second molecular factor is endogenous natriuretics. Both atrial and brain (or b-type) natriuretic factors have been linked to CSW. Both factors lead to natriuresis or excretion of sodium with subsequent serum hyponatremia. Renal sodium excretion, in turn, leads to a concurrent fluid diuresis and hypovolemia.

Atrial natriuretic peptide (ANP) was the first natriuretic suggested to have a potential role in causing hyponatremia in patients with SAH. It is a polypeptide that is produced in the atria of the heart and activated when the atrial stretch receptors become stimulated in response to hypervolemia, increased sodium, and/or an expanded preload (Braunwald et al., 2001; Svirni, Feinsod, & Soustiel, 2000). Atrial natriuretic peptide results in large amounts of sodium and fluid excretion. The

increased excretion of urine occurs due to inhibition of reabsorption of sodium in the collecting duct (Palmer, 2000). At the same time sodium is being blocked from returning to the bloodstream, there is an increased glomerular filtration rate contributing to natriuresis and diuresis. In addition, with the increased glomerular filtration rate, the secretion of renin and aldosterone diminish (Braunwald et al.). The reason for the decrease in aldosterone release is twofold. Its release from the adrenal gland is directly inhibited, and then there is an indirect inhibition due to the suppression of renin release from the juxtaglomerular kidney cells (Palmer). It is this decrease in circulating aldosterone levels that is thought to prevent potassium wasting from occurring in conjunction with the sodium loss (Palmer).

Three studies support the role of ANP in CSW in patients with SAH. Kurokawa et al. (1996) measured daily sodium and water balance and concentrations of ANP, ADH, and plasma renin activity (PRA) in 31 patients with aneurysmal SAH. Because ANP levels can be affected by arrhythmias, they excluded patients experiencing arrhythmias and also those with heart failure or kidney dysfunction. Their results showed a consistent abnormal increase in ANP values up until 14 days after the initial SAH; hyponatremia occurred in nine of these patients. ADH levels also increased within the first few days, but the rise was short-lived. Because sodium levels were low despite adequate sodium and fluid intake in the setting of elevated ANP, the authors concluded that ANP induced natriuresis in their subjects.

Wijdicks et al. (1991) also found elevated levels of ANP in a study done on 14 patients who had experienced SAH. In more than 50% of these patients, there was a sudden increase in ANP level followed by natriuresis with an accompanying net sodium loss. There was also a rise in ANP within the first few days of hospitalization. The researchers reasoned that fluid loss did not occur since there was a coinciding rise in vasopressin. During the natriuretic period, the vasopressin levels were low. Isotani et al. (1994) found similar results. Despite the correlation found between ANP levels and hyponatremia, no explanation of the increased levels of ANP has been established.

Two additional studies did not show a significant correlation between decreased sodium levels and elevated ANP plasma concentration, leading to a suggestion of an alternative contributor to CSW (Diringer, 2001; Okuchi et al., 1996).

Brain natriuretic peptide (BNP), a polypeptide consisting of 32 amino acids, is similar to ANP in many ways (Tomida, Muraki, Uemura, & Yamasaki, 1998). In addition to being found in the atria of the heart and stored in the myocardium of the heart's ventricles, it is stored in the hypothalamus of the brain (Braunwald et al., 2001; Svirni et al., 2000). Its action is similar to ANP; it is a potent vasodilator, causes sodium and fluid excretion, and leads to reduced circulating levels of renin and

aldosterone (Braunwald et al.). While it is well known that increased load on the ventricles can result in the release of BNP (Tomida et al.), there is no definitive explanation for its activation after SAH. Palmer (2000) suggested that it may be released as a protective measure for increased intracranial pressure, while Tomida et al. theorized that it may be activated as a stress response to surgery or the intensive care setting or as a result of damage in the hypothalamic region. Sviri et al. reported that unlike the variable findings analyzing the relationship of ANP to hyponatremia, studies measuring levels of BNP have demonstrated more consistent results.

Tomida et al. (1998) studied 18 patients with aneurysmal SAH and found that hyponatremia occurred in 11 patients with a corresponding rise in BNP levels. The rise that occurred between days seven and nine that resulted in natriuresis was not, however, statistically higher than in the patients with normal sodium levels. Despite this, it was concluded that BNP was more than likely responsible for the hyponatremia that resulted from the natriuresis and diuresis after SAH.

Sviri et al. (2000) also found elevated levels of BNP after SAH with the greatest rise between days one and three and seven and nine. While their study focused on the association of BNP levels with cerebral vasospasm, their findings were supportive of SAH-induced elevated levels of BNP and associated hyponatremia. Additional studies of small samples support these findings (Berendes et al., 1997; Nelson, Seif, Maroon, & Robinson, 1981). One study with contradicting results showed no difference between BNP levels in 20 patients with SAH compared to five normal subjects (Isotani et al., 1994). However, this may be the result of different procedures and clinical conditions of patients compared to the other investigations (Tomida et al., 1998).

Thus, three humoral and one neural derangement mechanisms have been suggested as possible causes of CSW. Data support the role of natriuretic factors, particularly BNP, as a mechanism for CSW; further investigation is needed in the role of digoxin-like peptide and sympathetic nervous system stimulation as well as the interaction between these four derangements. Determining the mechanisms of CSW may lead to targeted treatment. To explore current treatment approaches, a case study is presented.

Case Example

C.L. is a 37-year-old female admitted to the NCCU with a SAH resulting from the rupture of an anterior communicating artery aneurysm, grade 2. Upon admission she was loaded with phenytoin (Dilantin) and started on nimodipine (Nimotop) and aminocaproic acid (Amicar). Gastrointestinal ulcer and deep vein thrombosis prophylaxis was instituted. On day two of hospitalization, she underwent surgical clipping of the aneurysm without incident. Postoperatively she was treated with large amounts of fluid to maintain her blood pressure

and promote therapeutic hypervolemia to prevent or minimize cerebral vasospasm.

Her examination on the seventh day postoperatively was as follows:

- *Morning vital signs:* temperature 37.2°C, pulse 112 beats/minute, respirations 24 breaths/minute, blood pressure 115/56 mm Hg with a peripheral oxygen saturation of 99% on room air. Central venous pressure (CVP) measurement was 3 mm Hg. Weight was 56.3 kg (admission weight was 57.1 kg). Intake and output for previous 24 hours: 6,200 ml and 4,300 ml respectively.
- *Laboratory values:* glucose, 113 mg/dL; sodium, 128 mEq/L; potassium, 4.8 mEq/L; chloride, 100 mEq/L; BUN, 21mg/dL; creatinine, 1.0 mg/dL; uric acid, 3.0mg/dL; white blood cell count (CBC), 7.3; hematocrit, 48%.
- *Basic physical examination:* Neurologically intact, heart rate rapid but regular, pulses weak, respirations even and unlabored, abdomen soft with positive bowel sounds, continent for clear yellow, urine, and skin intact, but dry.

Differentiating Between CSW and SIADH

When all other potential causes of hyponatremia have been ruled out (Table 1), differentiation between CSW and SIADH should occur in the patient with SAH. Hyponatremia alone is not a reliable diagnostic indicator for either SIADH or CSW. Table 2 summarizes diagnostic criteria for SIADH and CSW.

One of the most basic and most important steps in differentiating SIADH from CSW is the physical examination. CSW is associated with a decreased fluid volume with symptoms of hypovolemia. In contrast, hyponatremia in SIADH is associated with either euvolemia or hypervolemia. In this case study, there are many "clues" to the presence of hypovolemia: C.L. is tachycardic with an elevated respiratory rate, weak peripheral pulses, dry skin, and a decreased body weight from admission. While C.L.'s weight loss is not large, with the amount of fluid that is usually administered postoperatively, a loss in body weight is not expected. Another finding is a CVP reading of 3 mm Hg. CVP readings and pulmonary capillary wedge pressure (PCWP) readings are believed to be critical in differentiating between CSW and SIADH (Suarez, 2004). In this case study, this value is within normal limits. However, with the large amount of fluid received, a higher reading would be anticipated. Not only would it be expected, but it would be desired as a preventative measure against vasospasm for which CVP levels are frequently kept above 8 mm Hg (Suarez, 2004).

The basic physical examination alone points toward the diagnosis of CSW rather than SIADH. Additional confirmatory steps include checking for orthostatic changes in blood pressure and heart rate if the patient's condition permits (Palmer, 2000). C.L.'s findings from physical examination are strongly indicative of CSW.

Table 1. Potential Causes of Hyponatremia (Andreoli, Carpenter, Griggs, & Loscalz, 2001; Braunwald et al., 2001)

- Gastrointestinal losses
- Burns
- Obstruction
- Diuretics
- Hypoaldosteronism
- Osmotic diuresis
- Nonoliguric acute tubular necrosis
- Chronic renal insufficiency
- Nephrotic syndrome
- Primary polydipsia
- Intravenous fluid overload
- Decreased solute intake
- Glucocorticoid deficiency
- Hypothyroidism
- Heart failure
- Hepatic cirrhosis
- Peritonitis
- Pancreatitis
- Internal bleeding
- Positive pressure ventilation
- Brain injury

Laboratory data are confirmatory. While nurses at the bedside are unable to order labs without a provider's order, they can utilize their knowledge by analyzing the labs that have been ordered. Serum osmolality, electrolytes, and uric acid, along with urine osmolality and electrolytes, are useful in distinguishing CSW from SIADH. Serum hematocrit, uric acid, and urine uric acid may also be helpful. In this scenario, C.L.'s hematocrit and blood urea nitrogen (BUN) in the presence of normal creatinine level, were both elevated. This is indicative of hypovolemia and thus CSW (Palmer, 2000). Potassium on the high end of normal is also consistent with a diagnosis of CSW; with SIADH an elevated potassium level is not expected (Harrigan, 2001). Plasma uric acid is in a low-to-normal range. In patients experiencing CSW, plasma uric acid levels are usually normal or low. It is important to know that plasma uric acid levels are also decreased in SIADH (Palmer, 2000). Patients with hypovolemia from causes other than CSW are more likely to exhibit elevated serum uric acid (Milionis, Liamis, & Elisaf, 2002).

Some laboratory values that would lead the clinician to suspect SIADH rather than CSW are dilute serum with an osmolality less than 280 mOsm/L, hyponatremia and low BUN with concurrent concentrated urine, and an elevated urine sodium greater than 25 mEq/L (Palmer, 2000; Woo & Kale-Pradhan, 1997). Serum hematocrit, albumin concentration, and potassium levels remain normal (Palmer, 2000). CVP and PCWP readings demonstrate hypervolemia. However, while SIADH does result in an increased fluid status, the patient is not automatically expected to have peripheral

edema upon physical examination. The explanation for this is that the excess free water is equally distributed and the hypo-osmolar state causes subsequent swelling of cells (Albanese et al., 2001) preventing an overexpansion of the interstitial space (Palmer).

Treatment

Once the differential diagnosis is made, treatment can be initiated. While many may believe it is the sole responsibility of the physician or APN to diagnose the hyponatremic patient, it is important for nurses at the bedside to understand and recognize when the diagnosis is not consistent with the clinical picture. Though nurses may not be responsible for ordering the treatment, they are responsible for implementing orders. Failure to recognize the appropriate diagnosis and instituting contraindicated treatments could have calamitous results.

In this scenario C.L. was experiencing CSW. Employing fluid restriction measures, as is sometimes utilized in the SIADH patient, could not only worsen her symptoms of hypovolemia, but also could increase the risk of cerebral vasospasm (Palmer, 2000; Suarez, 2003). In contrast, treating someone with SIADH with normal saline could create a symptomatic lowering of the serum sodium concentration (Palmer, 2000).

Overall, the treatment for hyponatremia after SAH is aimed at restoring normovolemia with normal serum sodium levels. Often if the patient is without symptoms, aggressive treatment is unnecessary, and if it is employed, it could have adverse outcomes (Mayberg et al., 1994; Suarez, 2004). If treatment is warranted, the serum sodium concentration level and the duration of time necessary for a safe decrease will determine the measures taken. Kurokawa et al. (1996) utilized a treatment approach based on the daily balances of fluid and sodium and found that by doing this, there were very few neurological deficits related to ischemia.

In general, patients with CSW will receive an order for intravenous normal saline. Depending on the sodium and fluid balance and the patient's symptoms, hypertonic 3% saline at an initial rate of 25–50 ml/hour, 325 mg salt tablets, and/or 1–2 mg daily of oral fludrocortisone (Florinef) may also be used (Palmer, 2000). Fludrocortisone (Florinef Acetate) treatment is sometimes difficult to use because of slow onset and long duration of action (Diringer, 2001). However, if this treatment option is chosen, careful monitoring is paramount because it has been linked with decreased serum potassium levels, hypertension, and pulmonary edema (Suarez, 2004).

In SIADH, the preferred treatment in the general population is fluid restriction (Palmer, 2000). However, in patients with aneurysmal SAH, great care must be taken because of the risk of vasospasm in these patients. One study found an increased incidence of infarction in patients treated for supposed SIADH with fluid

Table 2. Differentiating CSW and SIADH (Harrigan, 2001; Palmer, 2003; Woo & Kale-Pradhan, 1997)

Cerebral Salt Wasting

Serum sodium < 135 mEq/L
 Decreased extracellular fluid volume
 Increased hematocrit
 Increased plasma albumin concentration
 Normal or increased serum potassium
 Normal or decreased plasma uric acid
 Increased blood urea nitrogen/creatinine
 Signs of dehydration
 orthostatic changes
 flat neck veins
 dry mucous membranes
 poor skin turgor
 tachycardia
 weight loss
 negative fluid balance
 central venous pressure < 6 mm Hg and/or
 pulmonary capillary wedge pressure < 8 mm Hg

Syndrome of Inappropriate Antidiuretic Hormone

Serum sodium < 135 mEq/L
 Increased extracellular fluid volume
 Normal hematocrit
 Normal plasma albumin concentration
 Normal serum potassium
 Decreased plasma uric acid
 Urine sodium > 25 mEq/L
 Serum osmolality < 280 mOsm/kg
 Urine osmolality greater than serum osmolality
 Decreased urine output (400–500 ml/24 hours)
 Signs of hypervolemia
 increased body weight
 elevated central venous pressure and pulmonary
 capillary wedge pressure readings

monitoring would be every 6 hours for the next day and then every 12 hours until normalized.

Summary

Hyponatremia is a common phenomenon seen in patients after aneurysmal SAH. Although there is no definitive cause to date, it is frequently thought to be the result of SIADH, CSW, or a combination of both. While the mechanism for SIADH is understood, there continues to be controversy in respect to CSW, though natriuretic peptides do appear to play a prominent role. Obtaining an adequate

restriction (Wijdicks, Vermulean, Hijdra, & van Gijn, 1985). Other types of treatment include the infusion of hypertonic saline in conjunction with loop diuretics.

Regardless of the disorder and the treatment to be used, serum sodium levels should be corrected slowly. If too rapid correction of serum sodium occurs, the patient is placed at risk for developing central pontine myelinolysis (Braunwald et al., 2001, Suarez, 2004). Central pontine myelinolysis is a disorder in which the patient may present with confusion, dysarthria, gaze disturbances, quadriplegia, and pseudobulbar palsy as a result of demyelination in the base of the pons (Braunwald et al., 2001). To prevent this complication, serum sodium concentration levels should not be corrected at a rate exceeding 1.3 mEq/L/hour with a total correction of no more than 10 mEq/L in 24 hours, and sodium levels should be checked frequently throughout treatment. In this scenario, C.L. was treated initially 3% hypertonic saline at 30 ml/hr for 12 hours and then with normal saline at 125 ml/hr. Since C.L. was not yet experiencing neurological deficits related to the sodium deficit, an assessment alone would not be helpful in determining improvement. Also, due to the danger of central pontine myelinolysis, this would not be a safe approach. Therefore, serum sodium levels were checked every 6 hours for the first 24 hours and then every 12 hours until the sodium level was within normal levels. For this patient, the sodium gradually increased to normal levels over a period of 48 hours. The patient did not experience any side effects from the treatment prescribed. The sodium did decrease slightly before it began to improve; however, the patient remained without neurological deficits. If C.L. had been symptomatic, the frequency of laboratory assessment would be every 4 hours for 24 hours or until the symptoms resolved. Then

level of understanding of the pathophysiology of hyponatremia in the patient with SAH is key to differentiating between SIADH and CSW. Treatment varies for hyponatremia based on the presence of hypervolemia or hypovolemia. Nurses are valuable team members in recognizing the different findings and implementing the unique treatments so that outcomes for the patient with SAH are optimized and complications are avoided.

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