Neurologic Intensive Care Unit Electrolyte Management

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KEYWORDS

• Dysnatremia • Hyponatremia • Hypernatremia • Neurologic surgery • Intensive care

KEY POINTS

- Alterations in sodium levels are common among intensive care patients, and have been associated with poor outcomes in certain intracranial processes.
- Differentiating between the syndrome of inappropriate antidiuretic hormone and cerebral salt wasting is imperative in the correct treatment of neurologic intensive care patients.
- Diabetes insipidus is a common postsurgical finding following pituitary surgery.

DEFINITION

Dysnatremia is a common finding in the intensive care unit (ICU) and has been suggested to be a predictor for mortality and poor clinical outcomes.[1–3] Depending on the time of onset (ie, on admission vs later in the ICU stay), the incidence of dysnatremias in critically ill patients ranges from 6.9% to 15%, respectively.[4,5] The symptoms of sodium derangement and their effect on brain physiology make early recognition and correction paramount in the neurologic ICU (NICU). Hyponatremia in brain injured patients can lead to life-threatening conditions such as seizures and may worsen cerebral edema and contribute to alterations in intracranial pressure.[6]

DIAGNOSIS

Critical illness may result in activity fluctuations of antidiuretic hormone.[7,8] Patients with certain neurologic diseases, such as subarachnoid hemorrhages or traumatic brain injuries, are at an additional risk of dysnatremias. The patient population in an NICU are widespread and include, but are not limited to, traumatic brain injuries (TBIs), hemorrhagic and ischemic strokes, neoplasms, and infections; each has its own prevalence for sodium alterations (**Box 1**). For example, neurosurgery (specifically transsphenoidal pituitary surgery) has an incidence of postoperative diabetes insipidus (DI) that ranges from 1.6% to 31%.[9–11] The incidence of hypernatremia

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Box 1
Common causes of hypernatremia and hyponatremia in neurologically critically ill patients

Traumatic brain injury

Infections
- Meningitis
- Encephalitis

Neurovascular
- Thrombotic or embolic stroke
- Hemorrhagic stroke

Neoplasm
- Pituitary adenoma
- Meningioma
- Germinoma

Adapted from Faridi AB, Weisberg LS. Acid-base, electrolyte, and metabolic abnormalities. In: Parrillo JE, Dellinger, RP, editors. Critical care medicine: principles of diagnosis and management in the adult. 3rd edition. Philadelphia: Mosby; 2008. p. 1225.

following TBI ranges from 16% to 40%, whereas the prevalence of dysnatremias in aneurysmal subarachnoid hemorrhage (SAH) can range from 19% to 30%.[12,13] Distinguishing disorders affecting specifically the hypothalamic-neurohypophyseal system (ie, pituitary neoplasm/surgery) is important in diagnosis, duration, and management compared with systemic brain processes such as hemorrhagic strokes or TBI.

HYPERNATREMIA

Hypernatremia in critical illness has numerous causes and can be multifactorial. The diagnostic approach when dealing with dysnatremias should focus on the patient's volume status.[2,4,13] This article discusses euvolemic hypernatremia; specifically, DI.

Tonicity refers to the effect of plasma on cells. Hypernatremia always indicates hypertonicity, which results in cell shrinkage.[14] Plasma hypertonicity is a powerful stimulus for thirst (polydipsia), but, in acute brain injury, oral intake may be limited because of a decline in mental status.

DI is characterized by polydipsia and polyuria leading to an imbalance of water in the body. It can be caused by the decreased secretion of antidiuretic hormone (ADH), also known as central DI (CDI) or by a lack of renal responsiveness to the hormone, termed nephrogenic DI.[15] Providers in the NICU commonly come into contact with the neurologic form, CDI, which affects the hypothalamic-neurohypophyseal system. Clinical manifestations of CDI include large amounts of dilute urine, causing hypernatremia if the patient is unable to match the total fluid losses with water intake. The degree of the symptoms is proportional to the magnitude of the increase in sodium concentrations in the plasma, with chronic hypernatremia having milder symptoms than acute hypernatremia. Patients with chronic hypernatremia may experience weakness and lethargy, whereas acute hypernatremia may present as acute-onset headache, seizures, and even coma.[14,16]

CDI may follow 1 of 3 pathways following neurosurgery: either transient, permanent, or following a triphasic pattern. The first 2 phases occur in 2.7% to 13.6% of patients whereas the triphasic pattern has been described to occur in 3.4% of patients undergoing transsphenoidal pituitary surgery.[11,17] The pathophysiology of the triphasic phase begins with early hypothalamic dysfunction and inhibition of ADH resulting in