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REVIEW

Hyponatremia in the neurocritical care patient: An approach based on current evidence☆

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Abstract In the neurocritical care setting, hyponatremia is the commonest electrolyte disorder, which is associated with significant morbimortality. Cerebral salt wasting and syndrome of inappropriate antidiuretic hormone have been classically described as the 2 most frequent entities responsible of hyponatremia in neurocritical care patients. Nevertheless, to distinguish between both syndromes is usually difficult and useless as volume status is difficult to be determined, underlying pathophysiological mechanisms are still not fully understood, fluid restriction is usually contraindicated in these patients, and the first option in the therapeutic strategy is always the same: 3% hypertonic saline solution. Therefore, we definitively agree with the current concept of ''cerebral salt wasting'', which means that whatever is the etiology of hyponatremia, initially in neurocritical care patients the treatment will be the same: hypertonic saline solution.
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PALABRAS CLAVE
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Hiponatremias en el paciente neurocrítico: enfoque terapéutico basado en la evidencia actual

Resumen En el paciente neurocrítico la hiponatremia es la distonía más frecuente, comportándose como un predictor pronóstico. Clásicamente, el cerebro perdedor de sal y la secreción inadecuada de hormona antidiurética han sido las 2 entidades responsables de explicar

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Secreción inadecuada de hormona antidiurética;
Cloruro de sodio hipertónico

la mayor parte de los casos de hiponatremia en estos pacientes. Sin embargo, en virtud de la dificultad en establecer el estado de la volemia en el paciente crítico, el diagnóstico diferencial es con frecuencia difícil de establecer. Por otra parte, en el paciente neurocrítico el diagnóstico diferencial entre ambos síndromes no ha demostrado ser de utilidad debido a que el cloruro de sodio hipertónico es la piedra angular en el tratamiento de ambos cuadros, y la restricción hídrica con frecuencia está contraindicada. Es por ello que ha surgido el concepto de «cerebro falto de sal», lo cual traduce la necesidad del aporte de sodio como estrategia terapéutica en todos los casos.
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Introduction

Hyponatremia is defined as a serum sodium concentration of <136 mmol/l, and is the most common electrolyte disorder in hospitalized patients (affecting 15–20% of all individuals requiring hospital admission).[1,2] Likewise, hyponatremia is a mortality predictor in critical patients. In this regard, Stelfox et al.[3] have shown that hyponatremia acquired in the Intensive Care Unit (ICU) increases hospital mortality by 16–28% ($p < 0.001$). More recently, Sturdik et al.[4] have demonstrated that an age of over 65 years, the presence of dilutional hyponatremia, and its inadequate correction are three independent risk factors associated to increased hospital mortality in hyponatremic patients. Corona et al.,[5] in a systematic literature review and metaanalysis including 80 randomized clinical trials (RCTs), showed hyponatremia–even when mild–to be associated to a significant increase in mortality in the ICU (RR: 2.60; 95%CI: 2.31–2.93, $p < 0.0001$).[5] Likewise, inadequate treatment of this electrolyte disorder, failing to observe the required correction range (overcorrection), as well as insufficient treatment, imply added risk that further worsens the prognosis of critical patients with hyponatremia.[6,7]

In neurocritical patients, hyponatremia is also the most common electrolyte disorder, having been reported in up to 50% of all cases of serious neurological injury.[8] Among the acute brain conditions, severe traumatic brain injury (TBI) and aneurysmal subarachnoid hemorrhage (SAH) are those with the highest incidence of hyponatremia. In this regard, Sherlock et al.[9] showed hyponatremia to be more frequent in patients with hypophyseal disease (5/81; 6.25%), TBI (44/457; 9.6%) and intracranial tumors (56/355; 15.8%).[9] In their study the authors found that among 316 patients with SAH, 179 (56.6%) developed hyponatremia, which proved severe (natremia <130 mmol/l) in 62 cases (19.6%). In SAH, hyponatremia can be due to different causes, including syndrome of inappropriate antidiuretic hormone secretion (SIADH), so-called cerebral salt wasting (CSW)–also known as renal salt wasting–and pressure natriuresis or acute corticosteroid deficiency (hypocortisolism).[10–13] Up until now, CSW has been regarded as the most frequent cause of hyponatremia in the evolutive course of aneurysmal SAH, though recently this has been strongly questioned. However, on the basis of current knowledge, the differential diagnosis between SIADH and CSW is often difficult to establish, and both syndromes moreover have been suggested to be part of one same disease condition–manifesting successively in the same patient.[10,11]

Considering the above, the present review was carried out to provide an update on the etiological and physiopathological aspects of hyponatremia in the neurocritical patient, placing special emphasis on the treatment strategies adjusted to current evidence.

Hypoosmolarity and neuronal adaptation mechanisms

Hyponatremia/hypoosmolarity is a primary cause of water entry to the cells, resulting in an increase in cell volume (volumetric variation secondary to osmotic change).[14] This change in turn triggers a volume-regulating mechanism called regulatory volume decrease,[15] characterized by a rapid potassium, chloride and sodium outflow phase with the purpose of quickly ''buffering'' the osmotic change, and a second outflow phase of organic osmolytes (''non-perturbing'' osmolytes) which accumulate in the neurons without producing deleterious effects upon cell structure and function (cytoprotective function). These compensating mechanisms are incomplete in acute hyponatremia (evolution under 48 h) and complete in chronic hyponatremia (evolution longer than 48 h). In the presence of cell swelling, a first phase (called the rapid phase) is observed, involving osmolyte outflow, followed by a second phase (or slow phase) characterized by inhibition of the synthesis of these osmolytes.[16] However, in acute neurological injury these protective mechanisms carried out by the neuroglia in response to plasma hypotonicity are altered. Likewise, the increase in circulating antidiuretic hormone (ADH) levels observed in the two characteristic conditions (CSW and SIADH) results in action upon the V1a receptors of vascular smooth muscle. The stimulation of these receptors generates vasoconstriction independent of the endothelium, due to an increase in the calcium levels through activation of the phosphatidylinositol-bisphosphonate cascade.[17] This cerebral vasoconstrictor effect reduces cerebral blood flow, oxygen supply to the astroglia, and the production of ATP and phosphocreatine.[17]

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