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REVIEW ARTICLE

Diagnosis and treatment of hyponatraemia in neurosurgical patients

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Abstract Hyponatraemia is the most common electrolyte imbalance in neurosurgical patients. Acute hyponatraemia is particularly common in neurosurgical patients after any type of brain insult, including brain tumours and their treatment, pituitary surgery, subarachnoid haemorrhage or traumatic brain injury. Acute hyponatraemia is an emergency condition, as it leads to cerebral oedema due to passive osmotic movement of water from the hypotonic plasma to the relatively hypertonic brain which ultimately is the cause of the symptoms associated with hyponatraemia. These include decreased level of consciousness, seizures, non-cardiogenic pulmonary oedema or transtentorial brain herniation. Prompt treatment is mandatory to prevent such complications, minimize permanent brain damage and therefore permit rapid recovery after brain insult. The infusion of 3% hypertonic saline is the treatment of choice with different rates of administration based on the severity of symptoms and the rate of drop in plasma sodium concentration.

The pathophysiology of hyponatraemia in neurotrauma is multifactorial; although the syndrome of inappropriate antidiuresis (SIADH) and central adrenal insufficiency are the commonest causes encountered. Fluid restriction has historically been the classical treatment for SIADH, although it is relatively contraindicated in some neurosurgical patients such as those with subarachnoid haemorrhage. Furthermore, many cases admitted have acute onset hyponatraemia, who require hypertonic saline infusion. The recently developed vasopressin receptor 2 antagonist class of drug is a promising and effective tool but more evidence is needed in neurosurgical patients. Central adrenal insufficiency may also cause acute hyponatraemia in neurosurgical patients; this responds clinically and biochemically to hydrocortisone. The rare cerebral salt wasting syndrome is treated with large volume normal saline infusion. In this review, we summarize the current evidence based on the clinical presentation, causes and treatment of different types of hyponatraemia in neurosurgical patients.

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PALABRAS CLAVE
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Pérdida de sal cerebral;
Insuficiencia suprarrenal

Diagnóstico y tratamiento de la hiponatremia en pacientes neuroquirúrgicos

Resumen La hiponatremia es el desequilibrio electrolítico más común en los pacientes neuroquirúrgicos. La hiponatremia aguda es especialmente frecuente en los pacientes neuroquirúrgicos después de alteraciones cerebrales de cualquier tipo, incluidos tumores cerebrales y su tratamiento, cirugía hipofisaria, hemorragia subaracnoidea o lesión cerebral traumática. Supone una urgencia, ya que origina edema cerebral debido al movimiento osmótico pasivo de agua desde el plasma hipotónico al cerebro relativamente hipertónico, que es en última instancia la causa de los síntomas asociados con la hiponatremia. Estos incluyen el descenso del nivel de conciencia, crisis convulsivas, edema pulmonar no cardiogénico o herniación cerebral transtentorial. Es imperativo el tratamiento inmediato para prevenir esas complicaciones, limitar el daño cerebral permanente y, en consecuencia, permitir una recuperación rápida después de la afectación cerebral. La infusión de solución salina hipertónica al 3%, a velocidades de administración diferentes en función de la gravedad de los síntomas y del ritmo de descenso de la concentración plasmática de sodio, es el tratamiento de elección.

La fisiopatología de la hiponatremia en los neurotraumatismos es multifactorial, aunque las causas encontradas con más frecuencia son el síndrome de antidiuresis inapropiada y la insuficiencia suprarrenal central. La restricción de líquidos ha sido tradicionalmente el tratamiento clásico del síndrome de antidiuresis inapropiada, aunque está relativamente contraindicada en algunos pacientes neuroquirúrgicos, como los que sufren hemorragia subaracnoidea. Además, muchos pacientes ingresados tienen hiponatremia de comienzo agudo y precisan infusión de solución salina hipertónica. El grupo de fármacos antagonistas del receptor de vasopresina 2, desarrollados recientemente, es una herramienta prometedora y eficaz, pero se necesitan más pruebas que lo demuestren en pacientes neuroquirúrgicos. La insuficiencia suprarrenal central, que también puede causar hiponatremia en pacientes neuroquirúrgicos, responde clínica y bioquímicamente a la hidrocortisona. El raro síndrome de pérdida de sal cerebral se trata con infusión de grandes volúmenes de solución salina normal. En esta revisión se resumen las pruebas disponibles actualmente basándose en la presentación clínica, las causas y el tratamiento de distintos tipos de hiponatremia en pacientes neuroquirúrgicos.

Introduction

Hyponatraemia (plasma sodium <135 mmol/l) is the most frequent electrolyte disturbance encountered in clinical practice, and it is particularly common in a variety of neurosurgical conditions. Between 15–20% of patients who are subject to traumatic brain injury (TBI) of moderate severity or worse develop hyponatraemia[1,2] whereas approximately 50% of patients with non-traumatic subarachnoid hemorrhage (SAH)[3,4] will develop hyponatraemia during hospitalization. Furthermore, many other neurosurgical conditions present with or develop hyponatraemia postoperatively. Around 10–20% of patients admitted with intracranial hematomas, brain tumors or pituitary surgery will develop hyponatraemia[5].

Severe hyponatraemia, particularly when the onset is rapid (< 48 h), is a life-threatening condition, associated with increased mortality[6–8]. Perhaps because of the high background mortality attributable to the neurosurgical insult, a number of studies suggest that hyponatraemia is not necessarily associated with increased mortality[9,10] in this setting, and indeed, hypernatraemia might be stronger predictor of mortality in neurosurgical patients[11]. Furthermore, there is good evidence highlighting that hyponatraemia is associated with prolonged length of hospital stay[12,13]. Although hyponatraemia is an important factor related to clinical outcome, its relevance is frequently overlooked in neurosurgical patients[14].

In this review, we consider the differential diagnosis of neurosurgical hyponatraemia, particularly in the setting of subarachnoid hemorrhage and TBI, and offer recommendations on treatment.

Pathophysiology and classification of hyponatraemia in neurosurgical patients

The differential diagnosis of hyponatraemia in neurosurgical patients is simplified in Table 1. However, formulating a correct diagnosis is not always straightforward, as hyponatraemia is often multifactorial and there are multiple confounding factors which make diagnosis complex. Many patients in the neurosurgical intensive care unit are treated with large volumes of intravenous fluid, either to prevent cerebral vasospasm or to combat hypotension. The widespread use of inotropes complicate the assessment of haemodynamic factors influencing blood volume. Adrenocorticotrophin deficiency make manifest as hyponatraemia, though pituitary injury following head injury may be very transient[1] and so the contribution effects on plasma sodium