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Case report

Cerebral salt wasting syndrome in the posterior fossa surgery post-operative period: Case report[☆]



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ABSTRACT

Hyponatremia is the most common electrolyte disorder following intracranial surgery. Its aetiology is multifactorial. We present a case of a patient taken to microvascular decompression (Janetta surgery) for the treatment of trigeminal neuralgia who developed a symptomatic magnification of basal hyponatremia in the immediate post-operative period. Cerebral salt wasting syndrome was diagnosed. The management of this condition poses a challenge for physicians involved in postoperative neurosurgical care.

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Síndrome pierde sal en el postoperatorio de cirugía de fosa posterior: reporte de caso

RESUMEN

La hiponatremia es el trastorno electrolítico más frecuente después de la cirugía intracraneal. Su etiología es multifactorial. A continuación presentamos un caso de un paciente sometido a una descompresión microvascular (cirugía de Janetta) como tratamiento de la neuralgia del trigémino que en el postoperatorio inmediato desarrolló una magnificación sintomática de su hiponatremia basal. Se diagnosticó un síndrome pierde sal cuyo manejo supone un reto para los médicos implicados en los cuidados neuroquirúrgicos postoperatorios.

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Introduction

Fluid and electrolyte disorders are one of the complications of head injury and neurosurgical procedures, hyponatremia being the most common. Except for excess fluid or drug administration, the most frequent cause of polyuria-associated hyponatremia is cerebral salt wasting syndrome (CSWS).

Clinical case

A 58-year-old patient with trigeminal neuralgia scheduled for microvascular sub-occipital decompression craniotomy, with a history of multiple sclerosis (MS), prior thermocoagulation procedures and home treatment with oxcarbazepine, tryptizol, tapentadol and transcutaneous fentanyl. The surgical procedure was performed under general anaesthesia with the patient in supine recumbent position and cervical rotation for a retromastoid approach. No osmotic diuretics were required. The patient was taken to the recovery unit for postoperative neurological monitoring. While in the unit, the patient developed intense headache, difficult-to-control nausea, muscle cramps, and a tendency to polyuria (3 ml/kg). The neurosurgeon did not find the need for an urgent CT scan. Follow-up biochemistry tests were requested, with the finding of plasma sodium at 117 mEq L^{-1} (pre-anaesthesia consultation 130 mEq L^{-1} and urine sodium at 170 mEq L^{-1}). Volume replacement was initiated using isotonic saline solution, and the fluid and electrolyte imbalance was addressed with 3% hypertonic saline solution. Twenty-four hours later, plasma sodium was at 128 mEq^{-1} and diuresis was at 150 ml h^{-1} , with total remission of the clinical picture.

Discussion

Trigeminal neuralgia (TN) consists of electrical paroxysmal discharges in the trigeminal nerve innervation area.¹ It may present in isolated form or as a result of major neurological diseases such as MS or benign posterior fossa tumours.¹ MS patients have a 20 times higher risk² of developing TN.¹ While the annual incidence of TN in the general population is 0.015%, it is 1.9-4.9% in MS.¹ The most probable hypothesis is the irritation caused by the superior cerebellar artery on the root of the Vth cranial nerve.^{3,4} In cases of TN in MS, there is also a lesion of the trigeminal presynaptic afferent pathway due to the presence of a demyelinating plaque.^{1,5,6} At present, there are four potential therapies,⁷ including pharmacological treatment, radiofrequency, thermocoagulation, glycerol or balloon percutaneous rhizotomy, or microvascular decompression (Jannetta procedure).³ This latter technique involves placing a layer of autologous or artificial material between the nerve and the impinging vessel through a retromastoid craniotomy.³

The picture of severe symptomatic hyponatremia that developed in this patient may be explained on the basis of multiple factors that need to be analysed and identified correctly. Hyponatremia is the most common electrolyte disorder in hospitalized patients.⁸ It is defined as sodium plasma

levels below 135 mmol L^{-1} . The severity of the clinical picture depends on whether it is acute (less than 48 hours) or chronic (more than 48 h) and on the intensity (severe if 115 mmol L^{-1}), the aetiology being variable (Table 1). The most frequent symptoms are somnolence, apathy, anorexia, muscle cramps, headache, nausea, vomiting and stupor, which may progress to Cheyne-Stokes breathing, seizures, coma and death. Perioperatively, diuretics, ACE inhibitors and carbamazepine,⁹ normally used in TN, are the most frequent pharmacological causes of hyponatremia. Oxcarbazepine⁹ used as monotherapy or in combination with other drugs, may lead to what is known as oxcarbazepine-induced hyponatremia, a dose dependent and usually asymptomatic condition with an incidence ranging from 23 to 73%.⁹ Aetiological mechanisms are varied, but the most commonly accepted hypothesis is the inadequate release and/or greater sensitivity of tubular vasopressin¹⁰ as well as increased water with secondary sodium loss. The picture of oxcarbazepine-induced hyponatremia is usually mild and asymptomatic, secondary to a process of neuronal hyperhydration,¹¹ and severity is associated with how fast the absolute drop in plasma sodium occurs, with numbers under 125 mmol L^{-1} being clinically significant. The association between the use of carbamazepine and its derivatives and the inappropriate secretion of antidiuretic hormone (SIADH) has been widely described in the literature.¹²

There are very limited data in the literature on severe symptomatic oxcarbazepine-induced hyponatremia.⁹ It may be fatal when associated with other frequent complications found in neurosurgical patients, including polyuria and hyponatremia.

The two conditions most frequently associated with polyuria in the neurosurgical patient are diabetes insipidus (DI) and salt wasting syndrome (SWS), while those associated with hyponatremia are SWS and SIADH¹³, both of which determine a picture of hypotonic hyponatremia with elevated natriuresis.⁸ Both DI and SWS share some clinical findings with SIADH such as hyponatremia or a history of brain injury, whereas other concomitant findings help establish a clear difference (Table 2), as is the case with hypovolemia, excess uris and significant natriuresis, with normal anti-diuretic hormone levels.¹⁴

The distinction between the three clinical pictures is critical because they each require different management and need to be dealt with quickly by means of fluid replacement in DI and SWS, and fluid restriction in SIADH. On the other hand, in SWS, DI and SIADH sodium, vasopressin and diuretics must be given, respectively.¹³ Analytical determinations confirmed our suspicion of SWS: high hourly diuresis, low plasma sodium, high urinary sodium and low plasma osmolality which exacerbated the underlying picture of oxcarbazepine-induced hyponatremia (preoperative plasma sodium of 130 mEq L^{-1}), which progressed to severe symptomatic subsidiary hyponatremia following ionic correction.

The salt wasting syndrome was initially described by Peters et al. in 1950.¹³ The mechanism by which a cerebral condition causes SWS is not well known,⁸ although several hypotheses have been described, such as lower adrenocorticotrophic hormone secretion.¹³ The most accepted mechanism at the present time is excess release of atrial natriuretic

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