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Hyponatremia following a cerebral abscess: cerebral salt wasting syndrome or syndrome of inappropriate anti-diuretic hormone secretion?

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ABSTRACT: Cerebral Salt Wasting Syndrome (CSWS) and Syndrome of Inappropriate Anti-Diuretic Hormone Secretion (SIADH) are rare causes of hyponatremia during cerebral disorders. It is important to correctly differentiate between the two syndromes to determine the right therapeutic path.
We present a case of SIADH secondary to a cerebral abscess in a patient with chronic sinusitis who underwent an endovascular cerebral procedure for the treatment of a giant saccular internal carotid artery aneurysm. His hyponatremia responded to sodium supplementation, raising doubts about the diagnosis.
Application of the Maesaka's algorithm is a useful tool to determine the right diagnosis.

Keywords: CSWS, SIADH, Syndrome of Inappropriate Anti-Diuretic Hormone Secretion, ADH, Case-report, Cerebral salt wasting syndrome, Hyponatremia, Cerebral abscess, Endovascular, Stent, Internal carotid, aneurysm, Anti-diuretic hormone, Antidiuretic hormone, Vasopressin.

INTRODUCTION

Hyponatremia can be considered a public health problem concerning both in- and out-patients. A correct diagnostic approach should consider a physical and radiological examination, to rule out the presence of edema, and both serum and urinary osmolality, to discriminate among the different causes and establish an adequate management[1]. Cerebral Salt Wasting Syndrome (CSWS) and Syndrome of Inappropriate Anti-Diuretic Hormone Secretion (SIADH) are rare causes of hyponatremia during cerebral disorders, with CSWS being more common than SIADH according to literature. It is important to correctly differentiate between the two syndromes to determine the right therapeutic pathway: an unrecognized CSWS can result in unnecessary hyponatremia-related morbidity[2]. Moreover, choosing an incorrect management can lead to an excessive and needless expense[1]

How can we correctly differentiate between the two clinical identities? Literature offers a list of features characterizing both SIADH and CSWS, only one difference between them emerged from several studies, although, it was proved not to be diagnostic, useful or scientifically attainable[3].

CASE REPORT

A 65-year-old man came to the Emergency Ward of our hospital complaining of fever and headache a week after the endovascular treatment of a giant saccular internal carotid artery aneurysm in the ophthalmic segment. He was previously affected by chronic sinusitis, arterial hypertension, panhypopituitarism and progressive visual impairment for 5 years before the admission, due to the aneurysm slow growth. He had a fever (temperature over 38°C) at the admission, the physical examination was otherwise unremarkable. The blood count was within normal limits. Abnormal laboratory findings were a slight hyponatremia (sodium 129 mmol/L, normal 130-149) and a mild increase in C-Reactive Protein (CRP, 1.00 mg/dl, normal < 0.5). A CT head scan performed at the emergency ward highlighted a bilateral infection of the maxillary sinuses, and

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a dual antimicrobial treatment with Ampicillin-Sulbactam and Moxifloxacin was started. 9 days after the admission the patient still complained fever and headache, and a cerebral Magnetic Resonance Imaging (MRI) was performed, highlighting an accentuation in the intensity of the sub-arachnoid space set between the aneurysm and the cerebral peduncles, of suspected infectious origin. The patient's general conditions gradually worsened. He presented loss of consciousness (Glasgow Coma Scale 8 points out of 15, for eye opening response to pain, best verbal response of incomprehensible sounds and flexion withdrawal for pain as best motor response). The blood count was abnormal, with WBC 11,750/mm^3 (Neutrophils 75%). Abnormal laboratory findings were hyponatremia (Sodium 122 mmol/L) and hypocalcemia (Calcium 7.89 mg/dl). Meropenem 2 g q 8 h and Linezolid 600 mg q 12 h were started, while the previous antibiotic treatment with Ampicillin/Sulbactam and Moxifloxacin was stopped. Another cerebral MRI was performed which pointed out a worsening of the lesions previously highlighted, set in the right frontal Sylvian region and the perimesencephalic region bilaterally. The MRI also showed an increased contrast enhancement of the right leptomeninges.

Meropenem 2 g q 8 h and Linezolid 600 mg q 12 h were continued. After a neurosurgical consultation, mannitol was started, and the patient's conditions gradually improved.

A nephrologist was consulted for the persistent hyponatremia (< 120 mmol/L) only partially responding to intravenous (IV) hypertonic sodium supplementation. Serum and urine cortisol and serum and urine osmolality were studied to understand the cause. Due to a serum osmolality constantly under 275 mOsm/kg H_2O and a normal urinary one, associated with the patient's clinical history, a week after the first consultation he was diagnosed with a Syndrome of Inappropriate Anti-Diuretic Hormone (SIADH) Secretion secondary to the cerebral abscess, although the urine Sodium was 17 mEq/L (common finding in both SIADH and CSWS is a urine Sodium > 20 mEq/L)[3]. Therapy with Tolvaptan was prescribed but never started, because of the progressive normalization of the natremia following the antibiotic therapy and the IV sodium supplementation, leading us to question the diagnosis: was it SIADH or CSWS?

DISCUSSION

CSWS was firstly described in 1950, and it is defined as the development of extracellular volume depletion due to a renal sodium transport abnormality in patients with intracranial disease and normal adrenal and thyroid function[4]. On the other hand, SIADH is defined by the hyponatremia and hypo-osmolality resulting from inappropriate, continued secretion or action of the hormone despite normal or increased plasma volume, which results in impaired water excretion[5].

There is a considerable overlap in the clinical presentation, and distinguishing between these two disorders is of crucial importance because using the treatment of choice in SIADH, fluid restriction, in CSWS, which therapy of choice is sodium and volume replacement, can result in severe negative clinical consequences[6].

In our case, SIADH was diagnosed because of a low serum osmolality, a clinically estimated euvolemia of extracellular fluid (ECF), a normal serum cortisol and the patient's history of cerebral abscess. However, the clinical assessment of the extracellular volemia (ECV) is a user-dependent method, not repeatable, thus not trustworthy, and an objective way to determine it is often not attainable[3]. Moreover, the patient's hyponatremia progressively improved with the gradual resolution of the abscess and to the sodium supplementation, defining by the response to treatment a CSWS.

Maesaka et al[7] introduced in 2012 the determination of the relationship between serum sodium and fractional excretion of urate (FEurate) to differentiate SIADH from CSWS. Determining serum urate would have been useful to establish the cause of hyponatremia using the algorithm proposed by Maesaka et al[3].

CONCLUSIONS

Hyponatremia is a common occurrence after cerebral procedures or in the case of traumatic brain injuries. More studies are required to determine differences between CSWS and SIADH. It is necessary to keep in mind a path to the correct diagnosis.

Conflicts of Interest:
The Authors declare that they have no conflict of interests.

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