Cerebral salt-wasting syndrome associated with bacterial meningitis☆

Síndrome pierde sal cerebral asociado a meningitis bacteriana

Sir,

We present the case of a patient with meningitis due to pneumococcus admitted to our department with subsequent hyponatraemia and polyuria.

The patient was a 64-year-old woman operated on for closed mastoidectomy with tympanoplasty due to chronic otitis media, with hearing loss in the left ear 3 months after admission.

The patient presented with pain and suppuration in the left ear lasting for one month. A few days before admission, the pain had become more intense and was accompanied by headache, vomiting, fever and, later on, a reduction in her level of consciousness, for which reason she was referred to the emergency department, where she was seen to have stiffness in the neck and was given a cranial tomography, which was normal, and a lumbar puncture, which produced a purulent liquid.

She was admitted to the intensive care unit with a diagnosis of acute bacterial meningitis with a likely focus in the ear. Empirical treatment was begun with cephotaxime, vancomycin, and corticosteroids. A few hours after admission, she required mechanical ventilation due to a diminished level of consciousness and increased respiratory labour. *Streptococcus pneumoniae* was isolated in the culture of the cerebrospinal fluid and, after identifying the antibiogram (intermediate sensitivity to cephotaxime), the dosage of cephotaxime was increased and rifampicin was added. She was extubated satisfactorily after 2 days, although she still presented a confused state. Three days later, she started with polyuria (>5 L/day) and progressive hyponatraemia down to 130 mEq/L, elevated natriuresis (148 mEq/L) with also elevated urinary osmolality (277 mOsm/kg), while maintaining good renal function. Suspicion then fell on a cerebral salt-wasting (CSW) syndrome, with additional evidence of hypouricaemia (1.2 mg/dL), elevated levels of cerebral natriuretic peptide (2012 pg/mL) and anti-diuretic hormone in a normal range (4.2 μg/L). Adequate restoration of volume with saline solution in the light of urinary sodium and water losses allowed her condition to be brought under control, and natraemia, diuresis, and neurological status returned to normal.

Hyonatraemia is one of the most common electrolytic disorders, both in habitual clinical practice and associated with diseases of the central nervous system (CNS).[1,2]

The causes of hyponatraemia are numerous, but most of them are synonymous with a reduction in plasma osmolality (SIADH), with water restriction forming the basis for treatment. Nonetheless, especially in cases of intracranial pathology, hyponatraemia may also be due to a different mechanism, with a loss of sodium through the kidneys of cerebral origin (CSW), in which case restricting water intake is contraindicated.[2]

Table 1 Differences between CSW and SIADH.[a]

	CSW	SIADH
Plasma volume	Diminished	Increased
Sodium balance	Negative	Variable
Water balance	Negative	Increased or normal
Signs of dehydration	Present	Absent
Central venous pressure	Decreased	Increased or normal
Serum osmolality	Increased	Diminished
Haematocrit[b]	Increased	No variation
Plasma:ureic nitrogen in blood/creatinine	Increased	Diminished
Sodium in urine	Highly increased	Increased
Volume of urine	Highly increased	Diminished or normal

[a] Taken from Cuadrado et al.[5]
[b] Not evaluable after surgery.

Suspicion of this syndrome required the presence of inappropriate natriuresis for the levels of circulating sodium and volume depletion.[3] Typically, the onset of hyponatraemia due to CSW occurs in the first 10 days following the neurological or neurosurgical event. Thus, in the context of an illness in the CNS, CSW is diagnosed in a patient with clinical evidence of hypovolaemia and with the following findings[4]:

- Hyponatraemia (<135 mEq/L) with low plasma osmolality.
- Inappropriately elevated urinary osmolality (>100 mOsm/kg and usually >300 mOsm/kg).
- Diminished uric acid in plasma due to loss of urate in urine.

Clinical evidence of hypovolaemia is fundamental as these same findings can be found in SIADH[4] (Table 1).

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Meningitis associated with spinal anaesthesia: not always bacterial☆

Meningitis asociada a anestesia espinal: no siempre bacteriana

Sir,

Laguna del Estal et al.[1] have recently reported a series of patients with bacterial meningitis associated with epidural analgesia and anaesthesia, and in their discussion they quite rightly pointed out that the differential diagnosis must include chemical meningitis.[1] It is important to stress that meningitis induced by the local administration of anaesthetics must also be suspected whenever the cultures are negative. The clinical picture produced is indistinguishable from that of bacterial meningitis, but what many clinicians are unaware of is that the cerebrospinal fluid (CSF) may also be negative, revealing intense pleocytosis and polymorphonuclear predominance. These situations are well documented with, for example, bupivacaine, which may trigger pleocytosis of several thousand leucocytes with a percentage of polymorphonuclear cells close to 100%.[2—4] Some important facts may help distinguish between bacterial and aseptic meningitis. First of all, the latency between epidural anaesthesia and the onset of symptoms as a time of less than 6 h suggests that it is chemical meningitis. Second, the presence of eosinophilia in CSF, which is ''never'' seen in bacterial meningitis but is in drug-induced meningitis, or else that the patient presents atopy. Third, the presence of hypoglycorrachia less than 30 mg/dL, typically occurring in bacterial forms (albeit also described in aseptic cases). And finally, a frank elevation of acute phase reactants, common only in bacterial meningitides.

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