CASE REPORT

Syringomyelia associated with inappropriate antidiuretic hormone secretion

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SUMMARY

Hyponatraemia is the most common fluid-electrolyte disorder, and the most frequent related aetiologies are syndrome of inappropriate secretion of antidiuretic hormone (SIADH), which accounts for up to 38%. SIADH has been linked to multiple pathologies that affect the central nervous system; these disorders generally originate in the brain and, more rarely, in the spinal cord. It is often observed in patients undergoing neurosurgery and in patients with head injuries or intracranial tumours, and less common in those with spinal pathologies, especially traumatic. We describe an SIADH case associated with syringomyelia, in a patient admitted for severe, symptomatic hyponatraemia.

BACKGROUND

The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is defined by the presence of hypotonic hyponatraemia in a context of inadequately diluted urine and hypo-osmolality in plasma, with normal circulating volume and after excluding renal failure, adrenal insufficiency, severe hypothyroidism, drugs and physiological stimuli of ADH secretion. SIADH has been found to be one of the most frequent causes of hyponatraemia in the hospitalised patient.

Most SIADH are diagnosed as idiopathic and later a causal process is found, often a neoplastic process. To diagnose idiopathic SIADH, an exhaustive study is needed to discard other causes and even so, the patient should be strictly watched, for the next few years. In our patient, we found syringomyelia as a cause of SIADH after a rigorous study and after discarding other aetiologies of euvoalaemic hypo-osmolality. As far as we know, this is the first case described in the literature of a chronic SIADH case related with syringomyelia.

CASE PRESENTATION

A 62-year-old hypertensive woman with type 2 diabetes, anxious-depressive syndrome and rheumatoid arthritis was treated with Metilprednisolona 1 mg a day. The patient had generalised weakness, asthenia, nausea, unsteady gait and anorexia that started approximately 1 month ago. As laboratory tests made at her health centre revealed severe hyponatraemia, she was referred to our department. Hyponatraemia already appeared in previous inspections carried out since 2008, although the figures corresponded to mild hyponatraemia (serum sodium between 130 and 135 mEq/L).

When the patient was admitted to the hospital, she had serum sodium 116 mEq/L, serum potassium 4.5 mEq/L, plasma osmolality 273 mOsm/L, urine

osmolality 415 mOsm/L, urinary sodium 37 mEq/L and urinary potassium 15 mEq/L. Creatinine levels and blood urea concentration were normal. There were no signs of hypovolaemia like blood pressure decrease by orthostatic tachycardia or decreased skin turgor and dry mucous. There were no signs of hypervolaemia (ascites, oedema) either. We concluded, therefore, that the patient was euvoalaemic.

Thyroid hormones were normal. We have determined cortisol and adrenocorticotrophic hormone to discard adrenal insufficiency and we obtained normal values, so we discarded other causes of euvoalaemic hypo-osmolality different SIADH. Furthermore plasma renin values were slightly high, with normal aldosterone, uric acid 4 mg/dL and inappropriately high vasopressin levels regarding natraemia and osmolality carried out at that moment (vasopressin: 1.3 pg/mL, with plasma sodium: 131 mEq/L and plasma osmolality: 285 mOsm/L).

There was no antecedent of cranial trauma, and brain, chest and abdominal CT scans were carried out, discarding ongoing neoformative disease and confirming SIADH diagnosis.

During admission, impaired thermal sensitivity was observed in arms and shoulders on a rigorous neurological examination, although tactile sensitivity remained normal. Brain and spinal cord NMR scans were therefore requested, revealing the presence of an intramedullary cavity affecting the spinal cord to approximately D9, not associated with Chiari malformation (figure 1).

TREATMENT

After withdrawing concomitant drugs that could cause hyponatraemia (such as escitalopram, olmesartan and hydrochlorothiazide), hypertonic saline (3%) was started (0.5 mL/kg/h), and it was stopped once serum sodium level had increased above 120 mEq/L.

Once the serum sodium achieved normal levels (more than 135 mEq/L), the patient was discharged, but in subsequent checkups hyponatraemia reappeared. Given the difficulty that fluid restriction entailed for the patient, and whereas natraemia was less than Natriuria plus Kaliuria, treatment was instigated with Tolvaptan 15 mg/day, achieving adequate serum sodium control a few days later, so it was decided to reduce the dose gradually under supervision to 15 mg twice weekly.

OUTCOME AND FOLLOW-UP

After treatment adequate serum sodium control has been observed on an outpatient basis, without any other symptoms that could justify SIADH in more than 12 months of follow-up.

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Figure 1 Brain and spinal cord NMR scan showing the presence of an intramedullary cavity affecting the spinal cord to approximately D9, not associated with Chiari malformation.

DISCUSSION

Hyponatraemia association with intracranial diseases is well documented, less frequently reported is the association of transient SIADH and spinal injuries.¹ This is the case of spinal fusion in patients with severe scoliosis or spondylolisthesis, who suffer SIADH postsurgery.² Hyponatraemia can develop after any type of surgery and more commonly in brain or spinal cord surgery, which is the case of surgery with spinal or epidural anaesthesia. Frisbie³ reported that higher levels of spinal injury were more often associated with hyponatraemia, while Peruzzi *et al*⁴ suggested that the most important predictor for hyponatraemia development is the spinal injury type.

Thus, a Frankel class A injury, with loss of sensitivity and strength below the lesion, is more often associated with hyponatraemia than a class E injury, without loss of either sensitivity or strength.⁴

Our patient had syringomyelia, with a cavity from the cervical spine to D9. Furlan and Fehlings⁵ postulated a specific, multifactorial cause for hyponatraemia following spinal cord injury, among which could be impairment of the descending renal sympathetic fibres, responsible for renal water and sodium retention, mediated by renin release; which does not seem to be the most accurate hypothesis, since the values of plasma renin levels are normal or even slightly high.

Presence of potentially causal neurological diseases does not always imply responsibility for clinical condition; in fact, there are injuries rarely associated with the syndrome, and therefore screening should be performed for other potentially serious causes that could be responsible, such as neoplasia. In our case screening was carried out and no other possible SIADH cause was found. However, given that SIADH aetiology can be multifactorial, other intercurrent factors should also be taken into account, such as drugs, intravenous fluids and also various

osmoregulatory mechanisms that influence antidiuretic hormone secretion. After drugs discontinuation, symptoms appeared again, meaning that syringomyelia was causing the syndrome.

After hypertonic saline, when levels of natraemia are more than 120 mEq/L, the treatment of choice for SIADH is, when possible, fluid restriction. This usually happens in reversible situations, being more difficult to maintain for long periods of time due to the persistence of thirst reflex, as well as patient's comorbidities that often make it unfeasible. In addition, in situations where urinary sodium plus potassium is higher than serum sodium (Furst formula⁶) fluid restriction does not operate.

In our case, it was decided to initiate an oral selective arginine vasopressin V2 receptor antagonist Tolvaptan, at a dose of 15 mg/day. Later, it was possible to reduce the dose to 15 mg twice weekly, a dose lower than that currently recommended for management of chronic SIADH.⁷

In summary, we present an SIADH case associated with cervicodorsal syringomyelia, which debuted with clinically severe symptomatic hyponatraemia, and which responded effectively and was maintained at a low Tolvaptan dose. This is the first syringomyelia case described associated with chronic SIADH.

Learning points

- ▶ Hyponatraemia is the most common fluid-electrolyte disorder (over 30% of hospitalised patients), and syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is one of the most frequent causes.
- ▶ SIADH is caused by multiple aetiologies. To diagnose idiopathic SIADH, an exhaustive study is needed to discard other causes, and even so, the patient should be strictly observed, during the next few years.
- ▶ After fluid restriction or when fluid restriction is not adequate, vaptans are the unique and effective therapeutic option.

Competing interests None.

Patient consent Obtained.

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