Syndrome of inappropriate antidiuretic hormone caused by continuous lumbar spinal fluid drainage after transphenoidal surgery

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ABSTRACT

A 53-year-old acromegalic woman had cerebrospinal fluid rhinorrhoea following transphenoidal surgery for a pituitary microadenoma. A continuous lumbar spinal fluid drainage catheter was inserted and on the sixth postoperative day, she developed hyponatremia with features of syndrome of inappropriate antidiuretic hormone (SIADH) requiring hypertonic saline administration. Over-drainage is potentially hazardous and close biochemical monitoring is required. To our knowledge, this is the first reported case of SIADH caused by continuous lumbar drainage in an adult.

Keywords: cerebrospinal fluid rhinorrhoea, continuous lumbar spinal fluid drainage, hyponatremia, syndrome of inappropriate antidiuretic hormone (SIADH), transphenoid surgery

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INTRODUCTION

Cerebrospinal fluid (CSF) leak is a recognised complication of transphenoidal surgery. The incidence ranges from 0.5% to 15%[1], with the majority between 0.5% to 6.0%. The management of patients with postoperative CSF leak is still controversial. Since the publication by Vourc'h in 1963[2] describing the use of a plastic catheter inserted percutaneously for the drainage of lumbar CSF, the indication for spinal drainage are numerous but has not been very well researched. The success rate of continuous lumbar spinal fluid drainage (CLSFD) in the treatment of CSF leak was reported to be as high as 85-90% but endoscopic surgical repair has been reported to be superior with a 95% success rate[3]. The main problem with CLSFD is deficient flow and infection. Over-drainage is potentially dangerous, as illustrated in the current case.

CASE REPORT

A 53-year-old woman with acromegaly underwent transphenoidal surgery of a 0.5x0.5cm pituitary adenoma. In the immediate postoperative period, she was well, except for CSF rhinorrhoea that occurred on the second postoperative day. She was initially managed with a nasal pack. However, the CSF leak persisted and a CLSFD catheter was inserted. The CSF was drained at a rate of 10-15ml/hour. She complained of headache but there was no fever, polyuria nor visual disturbances.

At sixth postoperative day, hyponatremia with a serum sodium of 110mmol/L (normal 135-150) was noted. The serum potassium was 3.4 (normal 3.5-5.0) mmol/L, urea 3.4 (normal 2.5-6.4) mmol/L, and creatinine 46 (normal 62-133) umol/L. Her serum and urine osmolality were 236 (normal 275-295) mOsm/kg and 660 (normal 50-1200) mOsm/kg, respectively, with a urine sodium level of 48mmol/L, consistent with the diagnosis of SIADH.

She was not hypovolemic throughout the hospital stay as evidenced by a central venous pressure reading that was maintained at 8-10 cmH_20 and there was no vomiting, diarrhoea, nor any evidence of chest, central nervous system or meningeal infection. She was not hypothyroid or hypocortisol. Her free T4 and thyroid stimulating hormone were 14.8 (normal 10.3-24.5) pmol/L and 1.9 (normal 0.4-5.0) mIU/L, respectively. Her serum cortisol was 576 (normal 138-690) nmol/L. There was no record of taking any medication that predisposed the patient towards SIADH or natriuresis. Her urinalysis was normal and ultrasonography did not show any renal pathology to suggest a salt-losing nephropathy.

She was treated for SIADH with fluid restriction of 500-800ml/day. Despite three days of fluid restriction, the serum sodium was slow to increase. She was given hypertonic saline and the serum sodium normalised. Throughout the period of CSF rhinorrhoea, the CLSFD was inserted for five days and a total of 1,350ml of CSF was drained out.

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DISCUSSION

CLSFD is not without complication. These included meningitis, vocal cord paresis, posterior inferior cerebellar artery infarction, tension pneumoencephalus and coma from collapse of the intracranial cisterns[4] Hyponatremia has also been reported but this occurred in an infant who had post-haemorrhagic hydrocephalus which was treated with oral sodium supplement[5].

The sodium and potassium concentrations in the CSF are almost similar to those in plasma. Therefore, any CSF losses will result in equilibration between the serum and CSF sodium, thus maintaining the serum sodium within normal limits. With the patient developing hyponatremia, sodium losses by the CSF leak do not explain the drop in serum sodium. In addition, throughout this period, he was not given any hypotonic fluid that could have also lowered the serum sodium.

Cerebral salt wasting syndrome (CSWS), which shares similar serum and urinary biochemical characteristics, is also unlikely as the patient was never hypovolemic nor did he have any natriuresis throughout this period. Hypovolemia and marked natriuresis are essential findings in CSWS[6]. The low serum osmolality in this patient is also more suggestive of SIADH than CSWS which tend to have a normal or high serum osmolality. To our knowledge, this is the first reported case of SIADH caused by CLSFD in an adult following transphenoidal surgery. Hence, serum sodium concentration should be monitored closely in any patient requiring regular drainage of CSF.

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