Life threatening hyponatraemia following evacuation of spontaneous chronic subdural haematoma

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ABSTRACT

We report the case of a 43-year-old woman who developed life threatening hyponatraemia 4 days following burr hole drainage of a spontaneous chronic subdural haematoma (CSDH). Syndrome of inappropriate secretion of antidiuretic hormone was confirmed. This is the first report of delayed life threatening hyponatraemia developing postoperatively in CSDH. The mechanism remains unclear but may involve brain shift on the pituitary stalk following subdural evacuation.

KEYWORDS

Subdural haematoma – Chronic subdural haematoma – Syndrome of inappropriate secretion of antidiuretic hormone

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Spontaneous chronic subdural haematomas (CSDHs) are rare, especially in young adults.¹ Although hyponatraemia secondary to syndrome of inappropriate secretion of antidiuretic hormone (SIADH) in patients with traumatic subdural haematomas and severe traumatic brain injury is well documented,² we report the first case of spontaneous CSDH associated with life threatening hyponatraemia due to postoperative SIADH.

Case History

A 43-year-old woman presented with a headache 15 days after a long haul flight. The headache, which followed a single episode of vomiting during the flight, had increased gradually in severity. There were subtle memory problems and the headaches were refractory to simple analgesia. She was fully conscious with a Glasgow coma scale score of 15/15 and a mild pronator drift in the left upper limb. Computed tomography (CT) of the head revealed a right-sided CSDH with marked midline shift (Fig 1A). Blood biochemistry revealed a sodium level of 141mmol/l and a normal coagulation profile. There was no significant medical history and no history of trauma, clotting disorders, anticoagulant therapy or antiplatelet medication.

Surgical procedure

Right frontal right parietal burr holes were performed under general anaesthesia and the dura opened. A subdural membrane was incised to release the CSDH under significant pressure. The subdural space was washed

gently with warmed Ringer's solution. The brain did not re-expand fully and a 7mm Exudrain™ (Wellspect Health-Care, Mölndal, Sweden) was left in the subdural space with gravity drainage only.

Postoperative course

Postoperatively, there was resolution of the pronator drift. CT of the brain on postoperative day 1 showed resolving midline shift (Fig 1B). The patient was kept lying flat for 48 hours and the subdural drain removed prior to mobilisation. On day 4, she was slightly more lethargic and her serum sodium level, which had fallen from 137mmol/l on day 1 to 120mmol/l on day 4, dropped further to 118mmol/l with a serum osmolality of 250mosmol/l. Urinary sodium of 112mmol/l and osmolality of 628mosmol/l was consistent with a diagnosis of SIADH. Repeat CT showed further resolution of the subdural haematoma.

In view of the severe hyponatraemia, the patient was restricted to 1l of fluid per day. Endocrinological investigations revealed a normal pituitary axis with normal cortisol (386nmol/l). Serum sodium failed to respond to fluid restriction over the next 48 hours and on day 6 it was 117mmol/l. Owing to the spontaneous nature of the CSDH, magnetic resonance imaging and magnetic resonance cerebral angiography were carried out to exclude vascular abnormality. No other cause for the SIADH was found and the patient's chest x-ray was normal. When fluid restriction was increased to 750ml/day, her serum sodium level corrected precipitously to 136mmol/l over 24 hours and careful observation was carried out to monitor for central

Figure 1 Computed tomography of the brain of a 43 year-old woman presenting with progressive headaches 15 days following a long haul flight: Imaging at presentation showing a right-sided chronic subdural haematoma with midline shift (A); imaging on postoperative day 1 with resolving midline shift, subdural drain in situ and some intracranial air (B); and six-week follow-up imaging showing complete resolution of the subdural haematoma (C)

pontine myelinolysis. She improved clinically and was discharged on day 10 with no further hyponatraemia, remaining well at the six-week follow-up visit when CT of the brain revealed complete resolution of the CSDH (Fig 1C).

Discussion

Hyponatraemia is a common complication of brain injury, the main differential diagnosis of severe hyponatraemia being SIADH or cerebral salt wasting, with SIADH appearing to be the most common cause in severe traumatic brain injury.³ Sodium abnormalities secondary to hypothalamopituitary dysfunction might be expected in traumatic CSDH because of concomitant trauma at the time of the primary injury. Even in spontaneous CSDH, dysfunction, midline shift and brain compression could lead to distortion and

vascular compromise of the hypothalamopituitary unit. In a study of 59 patients undergoing surgical evacuation of CSDH, the only pituitary abnormalities detected acutely were hypogonadism or growth hormone deficiency.⁴ No cases of acute SIADH were found.

We present here the first report of life threatening hyponatraemia due to SIADH following evacuation of a CSDH. The CSDH developed spontaneously, there being no post-operative trauma to the hypothalamopituitary axis. The mechanism of development of the CSDH remains elusive although it is possible that a subdural bridging vein sheared during the flight vomiting episode. We hypothesise that the SIADH resulted from transient disruption of the hypothalamopituitary apparatus due to restoration of midline shift following evacuation of the subdural haematoma. Fragility of the hypothalamopituitary system to brain shifts is suggested by the finding that hypopituitarism may develop after intracranial surgery for benign primary brain tumours distant from the hypothalamopituitary system.

Cerebral atrophy in the elderly may protect the hypothalamopituitary apparatus from mechanical disruption owing to more gradual brain expansion and resolution of midline shift. Nonetheless, SIADH may develop in elderly patients with untreated CSDH and cases of CSDH presenting as SIADH in the elderly were reported in the pre-CT era.⁵ The patient described here a young with no cerebral atrophy, and the rapid, almost complete resolution of brain expansion and midline shift within 24 hours of surgery may have caused the postoperative SIADH and severe hyponatraemia.

Conclusions

Life threatening hyponatraemia is a rare postoperative complication of surgical evacuation of chronic subdural haematoma. The diagnosis of acute SIADH should be considered in patients deteriorating following surgery for CSDH, particularly in younger patients.

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