CASE REPORT

An interesting case of acute, severe postoperative hyponatraemia following carotid endarterectomy

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SUMMARY

A 62-year-old man with no major comorbidities became acutely hyponatraemic on the second postoperative day following a routine carotid endarterectomy. He developed a headache, became hypertensive and confused, and then had a seizure and required intubation and admission to the intensive care unit. A CT angiogram of his head and carotid arteries was normal, as was a subsequent MRI head. His serum and urine osmolality were low. He was treated by fluid restriction and his hyponatraemia resolved over 3 days. On discontinuation of sedation the patient woke up appropriately. The cause of his hyponatraemia was initially a mystery but when questioned by the medical team he admitted that he drank about 5 litres of water in the afternoon on the second postoperative day. At this point the diagnosis of dilutional hypervolaemic hyponatraemia secondary to water intoxication could be made.

BACKGROUND

Hyponatraemic encephalopathy owing to acute hypervolaemic hyponatraemia is an uncommon finding postoperatively. The cause of this diagnosis was initially unclear despite involvement of the surgical, intensive care, medical, endocrine and cardiology teams. Only when the patient was able to give the history that he drank excessively in the postoperative period, after oral fluids were encouraged, did the cause become apparent. This highlights the importance of monitoring type and quantity of fluid intake (intravenous as well as oral), and the importance of checking a patient's understanding of instructions from doctors.

CASE PRESENTATION

A 62-year-old man was admitted for a left carotid endarterectomy to treat a transient ischaemic attack which was attributed to a left internal carotid artery stenosis (50–59% North American Symptomatic Carotid Endarterectomy Trial criteria). His only comorbidities included mild gastro-oesophageal reflux disease and urinary hesitancy which had resolved a year ago. His renal function was normal.

A standard, uncomplicated carotid endarterectomy with a Javed shunt was performed. The plaque was soft with significant intraplaque haemorrhage. The artery was closed with a Dacron patch. Three-thousand units of intravenous heparin were administered intraoperatively along with antibiotic prophylaxis. There was no intraoperative hypertension or hypotension. The patient received 500 ml of Hartmann's solution intraoperatively.

On day 1 postoperatively the patient was generally well. His serum sodium had dropped from a normal level preoperatively to 127 mmol/l. He developed urinary retention and was treated with a urinary catheter which was removed the following morning. Oral fluids were encouraged by the surgical team. That evening the patient started complaining of headache and nausea. His systolic blood pressure was 180 mm Hg. He was recatheterised, and oral antihypertensive medication was administered. He deteriorated further and became confused and dysphasic. His serum sodium was rechecked and had dropped to 107.

He was admitted to the intensive care unit. Soon after admission he suffered a tonic-clonic seizure and required endotracheal intubation. His blood pressure was labile throughout this period. Four hours after the seizure he had a pulseless electrical activity cardiac arrest and required 3 min of cardio-pulmonary resuscitation including one dose of epinephrine, after which his regained spontaneous cardiac output.

His hyponatraemia was treated with fluid restriction. His renal parameters remained normal with a urinary output >1 ml/kg/h throughout his intensive care unit admission. His serum sodium increased over 3 days back to normal levels.

Three days after admission to intensive care sedation was stopped and as he woke up appropriately. His Glasgow Coma Scale was 15/15 and he was transferred to the ward the following day. Postextubation he was questioned regarding his fluid intake and admitted that in trying to avoid urinary retention he drank between 5 and 7 litres of water on the second postoperative day.

INVESTIGATIONS

When the patient became confused an urgent CT angiogram was performed, and excluded any intracranial bleed or ischaemia. The CT also excluded any operative site complications with a patent left internal carotid artery.

Once stable a cerebral MRI was performed, which was normal (no pituitary injury).

An echocardiogram excluded significant cardiac failure.

On the intensive care unit his serum sodium was 107 mmol/l, his other electrolytes, urea, creatine and a random serum cortisol level were all normal. Urine osmolality was 251 mmol/l and serum osmolality was 233 mmol/l. These low values indicated a hypervolaemic hyponatraemia with a diluted urine.

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DIFFERENTIAL DIAGNOSIS

The initial symptoms of headache, nausea, vomiting and confusion could have been caused by an encephalopathy because of various reasons. We considered alcohol withdrawal, malignant hypertension, reperfusion syndrome postcarotid endarterectomy or a cerebrovascular accident with increased intracranial pressure. These possibilities were excluded by his history, the normal CT and progression of his symptoms. Reperfusion syndrome is a very rare complication postcarotid endarterectomy. Cerebral hyperperfusion (>100% increase in cerebral blood flow compared with preoperative baseline) occurs in 9–14% of patients with carotid endarterectomy patients. Only a minority (0.75–3%) develop a cerebral hyperperfusion, or cerebral reperfusion, syndrome, characterised by ipsilateral headache, seizures and focal neurological symptoms.

Once severe, acute hyponatraemia was diagnosed the differential diagnosis included water intoxication, syndrome of inappropriate antidiuretic hormone (SIADH) secretion and cerebral salt wasting. We also considered whether he might have pituitary apoplexy.

TREATMENT

After the patient's seizure the patient was fluid restricted. He was subsequently arrested. Once he regained cardiac output the patient was reviewed by the consultant intensivist, endocrinologist and a medic, and discussed with the biochemist. At this point there was a debate regarding further management of the hyponatraemia. Acute management of severe acute hyponatraemia is typically 100 ml of 3% saline bolus. Ideally this would have been given immediately postseizure. However, postarrest serum sodium level had already increased to 114 mmol/l and the cause of the hyponatraemia was unknown at this point. The patient was diuresing >2.5 ml/kg/h of urine. Weighing up the improvement in serum sodium and the risk of osmotic demyelination syndrome with rapid correction of hyponatraemia, it was decided to continue fluid restriction and supportive treatment rather than active treatment with hypertonic saline.

OUTCOME AND FOLLOW-UP

Once the patient's serum sodium had returned to normal he recovered rapidly, and was discharged home with no neurological sequelae.

DISCUSSION

Hyponatraemic encephalopathy is a severe complication of hyponatraemia, characterised by symptoms of headache, nausea, vomiting and confusion. Seizures, respiratory arrest and non-cardiogenic pulmonary oedema can occur in severe cases.

Hyponatraemia is defined as a serum sodium <135 mmol/l. Sodium homeostasis is achieved by renal reabsorption of sodium, which is modulated by aldosterone, ADH and atrial natriuretic hormone. Acute hyponatraemia can either be hypervolaemic, normovolaemic or hypovolaemic. Mild postoperative hyponatraemia is relatively common—a prospective study of 1088 patients found 4.4% to be hyponatraemic postoperatively (serum sodium <130 mmol/l). Most of these patients (41%) will have normovolaemic hyponatraemia as a result of non-osmotic secretion of ADH which is related to the surgical stress response. Hypotonic fluid administration exacerbates the problem.¹

Non-osmotic secretion of ADH or the SIADH is secretion of ADH without appropriate osmotic or hypovolaemic stimulus.

It can instead be released as a result of other stimulating factors including subclinical volume depletion, pain, nausea and stress, which are all seen in the postoperative period. Studies have shown that ADH levels are universally elevated postoperatively when compared with preoperative values.²

Hypervolaemic hyponatraemia as a result of water intoxication is far less common than normovolaemic hyponatraemia postoperatively. It has been described in soldiers and endurance athletes, and in psychiatric patients with compulsive water drinking symptoms.^{3–5}

This is the first case of self-induced postoperative water intoxication described in the literature. This patient had been encouraged to drink oral fluids postoperatively once his intravenous fluids had been stopped. He was particularly anxious to avoid further urinary retention and so forced himself to drink excessively. This case highlights the importance of communication—particularly of clarifying the patient's understanding of instructions.

Postoperatively many patients become oliguric because of the increased secretion of ADH. During the postoperative period overhydration can occur even with a maintenance dose of hypotonic fluids. Overhydration with hypotonic solutions causes intracellular oedema, while isotonic fluids will cause oedema in the interstitial and extracellular spaces.⁶ Most patients tend to be given intravenous isotonic fluids postoperatively. Little attention, however, tends to be given to what fluids the patients consume orally which are mostly hypotonic. This patient had drunk a large volume of tap water, a hypotonic solution.

Learning points

- ▶ Improved understanding of perioperative fluid and electrolyte management by doctors.
- ▶ Importance of monitoring all fluids (intravenous and oral) in the immediate postoperative period.
- ▶ The importance of checking patients' understanding of instructions.
- ▶ The significant difference between acute and chronic hyponatraemia and their respective management.

Contributors All authors were involved in the care of the patient. FP and AH wrote the first draft of the manuscript. SE and CF edited the first draft to produce the final manuscript.

Competing interests None.

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