Short reports

Abnormal movements associated with severe hyponatraemia

Nages Nagaratnam, Evelyn Icao, Helen Peric

Summary

An elderly woman with severe hyponatraemia manifested transient choreo-athetoid movements of the upper extremities and dyskinetic movements of the face and mouth. She showed more than one type of hyponatraemia and a precise diagnosis was not possible. The movements were abolished with treatment of the hyponatraemia with no recurrence or sequelae.

Keywords: hyponatraemia, diuretic, abnormal movements

Hyponatraemia (serum sodium level <130 mmol/l) is the most common electrolyte abnormality, especially in the elderly, and occurs with a wide variety of medical conditions. Asymptomatic hyponatraemia often goes undetected and with severe hyponatraemia the clinical presentation can be diverse and non-specific such that early recognition can be delayed.¹ A wide variety of neurological manifestations has been associated with hyponatraemia and severe hyponatraemia can have damaging effects on the central nervous system.² Generalised disturbances such as inappropriate behaviour, hallucinations (both visual and auditory), unresponsiveness, seizures, and coma are the more frequent manifestations.³ We describe an elderly woman with severe hyponatraemia with choreo-athetoid and dyskinetic movements.

Causes of hyponatraemia
<ul style="list-style-type: none">• extra-renal: vomiting, diarrhoea, sweating, fever, ileus• renal: diuretics, chronic renal failure• dilutional: intravenous fluid overload, cardiac failure, syndrome of inappropriate secretion of antidiuretic hormone

Initial symptoms of hyponatraemia
<ul style="list-style-type: none">• weakness, faintness• nausea, anorexia• thirst, cramps• poor skin turgor• postural hypotension

Case report

A 77-year-old woman presented with intermittent dizziness and vomiting for over three months which had worsened over the past week. She was said to have Ménière's disease and had been on nicotinic acid, a combined diuretic (amiloride and hydrochlorothiazide) and prochlorperazine (5 mg tid) for several weeks. The subsequent diagnosis was benign paroxysmal positional vertigo. The medical history was of no clinical significance, but for diet-controlled diabetes. She had never been on neuroleptics and is a non-drinker and a non-smoker.

On admission the vital signs were unremarkable. The cardiovascular, respiratory and central nervous system were normal. The serum sodium was 134 mmol/l, potassium 3.1 mmol/l, blood glucose 11.1 mmol/l, calcium 2.37 mmol/l, magnesium 0.62 mmol/l, urea 5.8 mmol/l and creatinine 6.5 µmol/l. Computed tomography (CT) scan of the brain was normal. She remained on the diuretic and prochlorperazine. Four days following admission vomiting worsened and three days later she was found groaning at 08.00 h. She was conscious, but did not respond to verbal commands. She had mouthing movements involving the face, mouth and tongue, simulating tardive dyskinesia and writhing with sudden jerky movements of the upper limbs. The lower limbs were in extension with slight increase in tone. The pupils were equal and reactive. The plantars were flexor. There were no abnormalities in the other systems. The chest X-ray was normal. Routine haematology and liver function tests were normal. Her medications were stopped.

By 11.00 h her level of consciousness had worsened. She was unresponsive, but the movements continued and the plantars were extensor. The serum sodium level was found to be 101 mmol/l, potassium 3.7 mmol/l, urea 6.2 mmol/l, creatine 68 µmol/l, plasma osmolality 217 (275–295) mosm/kg, glucose 12.2 mmol/l, cholesterol 4.5 mmol/l, triglycerides 2.8 (0.1–1.85) mmol/l, sodium 117 mmol/l, potassium 6.1 mmol/l, urine osmolality 478 (300–600) mosm/kg. CT scan of the brain revealed no abnormality. She was commenced on normal saline. She had a generalised seizure, but was not given anticonvulsants. At 20.00 h she was still unconscious, the plantars were flexor and no movements were seen. Her serum sodium was 112 mmol/l. She was then given 3%

Department of
Medicine
(Geriatrics),
Blacktown Hospital,
NSW 2148 Australia
N Nagaratnam
E Icao

University of
Sydney, Sydney,
Australia
H Peric

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sodium chloride 500 ml over 10 h and by 24.00 h the sodium was 116 mmol/l, potassium 3.7 mmol/l.

On day 2, her level of consciousness had improved. The serum sodium was 118 mmol/l, potassium 2.6 mmol/l and magnesium 0.58 mmol/l. The last two were included in the infusion. Normal saline was then commenced, a litre 8-hourly. After 48 h, serum sodium was 124 mmol/l, potassium 3.4 mmol/l and magnesium 1.09 mmol/l. On day 3, she opened her eyes spontaneously and responded to verbal commands. There were no focal or lateralising neurological signs. Thereafter her condition improved and remained stable and on day 7 her sodium was 136 mmol/l and potassium 4 mmol/l; she was mobilising independently and was discharged home on day 11. Blood sugar levels from days 1 to 11 ranged from 3.0–12.2 mmol/l with no treatment. She was prescribed prochlorperazine (Stemetil) with no recurrence of symptoms. When reviewed six weeks later there was no overt abnormality and prochlorperazine was stopped. On further follow-up over 18 months she remained well and her blood sugar was 5.5 mmol/l.

Discussion

Our patient had severe hyponatraemia (serum sodium 101 mmol/l) with abnormal movements. She responded to sodium replacement with a correction of 24 mmol/l in the first 48 h. She recovered without any recurrence or sequelae. She had been on a combined diuretic until the onset of the abnormal movements.

More than one type of hyponatraemia could have contributed to her clinical state. The biochemical findings at the onset were that of the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). SIADH is common in the elderly and has many causes (eg, infection/malignancy), although there was no obvious cause in our patient other than for

the thiazide therapy. There are, however, other mechanisms which may have contributed in some degree to the hyponatraemia, namely (a) the intractable vomiting and diuretic, (b) hyperglycaemia, and possibly (c) pseudohyponatraemia due to mildly elevated triglycerides.

It had been suggested that rapid correction of the hyponatraemia could lead to central pontine myelinolysis. It is now accepted that central pontine myelinolysis is almost never seen with hyponatraemia and is associated with medical conditions such as alcoholism, sepsis, advanced liver disease or malignancies.³ In hyponatraemia the observed lesions were diffuse areas of cerebral infarction. The rate of correction is said not to be a factor in the genesis of hyponatraemic brain injury but the absolute increase should be within 25 mmol/l in the first 48 h of treatment.³ Brain damage often occurs in untreated patients with hyponatraemia.² Two different mechanisms induce brain injury in hyponatraemic patients, one associated with treatment and the other unrelated to it.⁴ In severe hyponatraemia cerebral hypoxia results from swelling of the brain and an increase in intracranial pressure giving rise to a reduction in cerebral blood flow and pressure of the brain against the rigid skull.⁴ In hyponatraemia, neuroimaging and autopsy findings have demonstrated infarction.

Tardive dyskinesia may develop with prochlorperazine and the risk increases with duration of the treatment (cumulative dose) and less commonly after brief treatment at low doses. In our patient the syndrome occurred acutely with no recurrence on re-commencement of the medication. It seems most unlikely that the abnormal movements were caused by prochlorperazine. Sethia *et al*⁵ have described generalised chorea due to basal ganglia infarcts and they postulated that ischaemia rather than infarction gives rise to the movements. Abnormal and involuntary flinging movements, principally unilateral, of sudden onset lasting a few hours have been described with a striatal infarct.⁷ Episodic dyskinesias have been documented with cerebral ischaemia⁶ and are a rare manifestation of transient ischaemic attack in the carotid artery territory. Choreoathetosis has been reported after insulin-induced hypoglycaemia⁸ and with non-ketotic hyperosmolar coma.⁹

In conclusion, it is hypothesised that the transient hypoxia and ischaemia that occurs with severe hyponatraemia could affect the basal ganglia and give rise to abnormal movements. Although the CT scan was normal, CT is not particularly sensitive at identifying lesions in the region of the basal ganglia.

Symptomatic hyponatraemia
<i>Investigations</i> <ul style="list-style-type: none">● serum sodium● serum sodium concentration● urea and creatinine● urine osmolality● plasma osmolality
<i>Treatment</i> <ul style="list-style-type: none">● aim of treatment is a sodium concentration of >130 mmol/l● correction of not more than 25 mmol/48 h

1 Booker JA. Severe symptomatic hyponatraemia in elderly outpatient. *J Am Geriatr Assoc* 1984; 32: 108.
2 Arieff AI, Llach F, Massary SL, Kerian A. Neurological manifestations and morbidity of hyponatraemia: correlation with brain water and electrolytes. *Medicine (Baltimore)* 1976; 55: 121–9.
3 Arieff AI. Management of hyponatraemia, fortnightly review. *BMJ* 1993; 309: 305–8.
4 Tien R, Arieff AI, Kucharczyk W, Wasik A, Kucharczyk J. Hyponatraemic encephalopathy: is central pontine myelinolysis a component? *Am J Med* 1992; 92: 513–22.
5 Sethi KD, Nichols FT, Yaghamai F. Generalised chorea due to basal ganglia lacunar infarcts. *Mov Disord* 1987; 2: 61–6.

6 Margolin DI, Marsden CD. Episodic dyskinesias and transient cerebral ischaemia. *Neurology* 1982; 32: 1379–80.
7 Defebvre L, Destec A, Cassim F, Muller JP, Vermersch E. Transient hemiballism and frontal infarct (letter). *Stroke* 1990; 21: 967–8.
8 Newman RP, Kinkel WR. Paroxysmal choreoathetosis due to hypoglycaemia. *Arch Neurol* 1974; 41: 341–2.
9 Rector WG, Herlong HF, Moses H. Non-ketotic hyperglycaemia appearing as choreoathetosis or ballism. *Arch Intern Med* 1982; 142: 154–5.