Cerebral salt wasting: a forgotten diagnosis in district general hospitals?

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Introduction

Hyponatraemia is an electrolyte disorder with an incidence of between 4% and 15% among hospitalised patients. In older patients, the most common cause of hyponatraemia is the syndrome of inappropriate antidiuretic hormone secretion. However, cerebral salt wasting is a rare cause of hyponatraemia, accompanied with hypovolaemia and increased urine sodium levels, that is often overlooked in district general hospitals. Cerebral salt wasting is most commonly diagnosed after injury to the CNS so, when making a diagnosis of

Case report

An 81-year-old man presented with a fall and trauma to the head following a syncopal episode. His past medical history consisted of atrial fibrillation, hypothyroidism and prostate cancer (treated 18 years ago with brachytherapy). His drug regimen consisted of rivaroxaban and levothyroxine. Following the fall, new onset confusion and escalating right hip pain led to his admission to the district general hospital via ambulance.

Upon examination, the patient's right leg was externally rotated and shortened. Auscultation of the chest revealed left-sided coarse crepitations. Blood work revealed low sodium level (131 mmol/litre; normal range 135–145 mmol/litre), elevated white cell count (15.7×10^9/litre; normal range $4.0–11.0\times10^9$/litre), raised C-reactive protein level (199 mg/litre; normal range <10 mg/litre) and normal renal function (estimated glomerular filtration rate >90 ml/min/1.73m^2). Chest X-ray was unremarkable. Pelvic X-ray indicated an intertrochanteric fracture of the right femur, while a computed tomography scan of his head showed an 18 mm right cerebellar haemorrhagic focus with adjacent oedema (Figure 1). An electrocardiogram confirmed the previous diagnosis of atrial fibrillation with normal ventricular rate. His blood pressure was stable at 115/95 mmHg. The patient was commenced on intravenous co-amoxiclav to treat a lower respiratory tract infection as inferred by his chest examination findings, and elevated white cell count and C-reactive protein level. He subsequently tested positive for COVID-19. Orthopaedic review was sought for the fracture and external neurosurgical advice suggested withholding rivaroxaban, alongside conservative management of his cerebellar haemorrhage.

On day nine of admission, following a right dynamic hip screw fixation, his blood work deranged with his postoperative sodium level reduced to 128 mmol/litre. His sodium levels decreased further to a low of 124 mmol/litre by day eleven of admission. A postural hypotension-induced syncopal episode led to the initiation of midodrine. Following a geriatric review, a working diagnosis of hyponatraemia secondary to syndrome of inappropriate antidiuretic hormone secretion was proposed, which saw the patient placed on a 1.5 litre/day fluid restriction and commenced on demeclocycline. Despite 8 days of fluid restriction, his sodium levels did not improve and remained at 125 mmol/litre. A specialist opinion from an endocrinologist was sought and the patient's biochemical markers were reviewed with the following results: serum osmolality 261 mOsm/kg, urine osmolality 776 mOsm/kg, urine sodium >60 mmol/litre, thyroid-stimulating hormone 0.8 mU/litre, cortisol levels 640 nmol/litre. In view of his recent intracranial bleed, low sodium of 124 mmol/litre, hypovolaemia and clinical dehydration, the endocrinologist suggested cerebral salt wasting as a likely diagnosis. At this point, intravenous normal saline (0.9% solution) was administered (>3 litres/day), demeclocycline was stopped and the patient was encouraged to eat and drink as normal.

After 2 days of intravenous fluids, the patient's sodium levels increased to 131 mmol/litre. An increase in blood pressure alleviated his postural hypotension, allowing him to undergo physiotherapy and regain his mobility. After 27 days in hospital, the patient was discharged with normal sodium levels and recommended to undergo monthly sodium monitoring by his GP. At follow up, his sodium levels were maintained and magnetic resonance imaging of his head revealed resolution of his cerebral haematoma.

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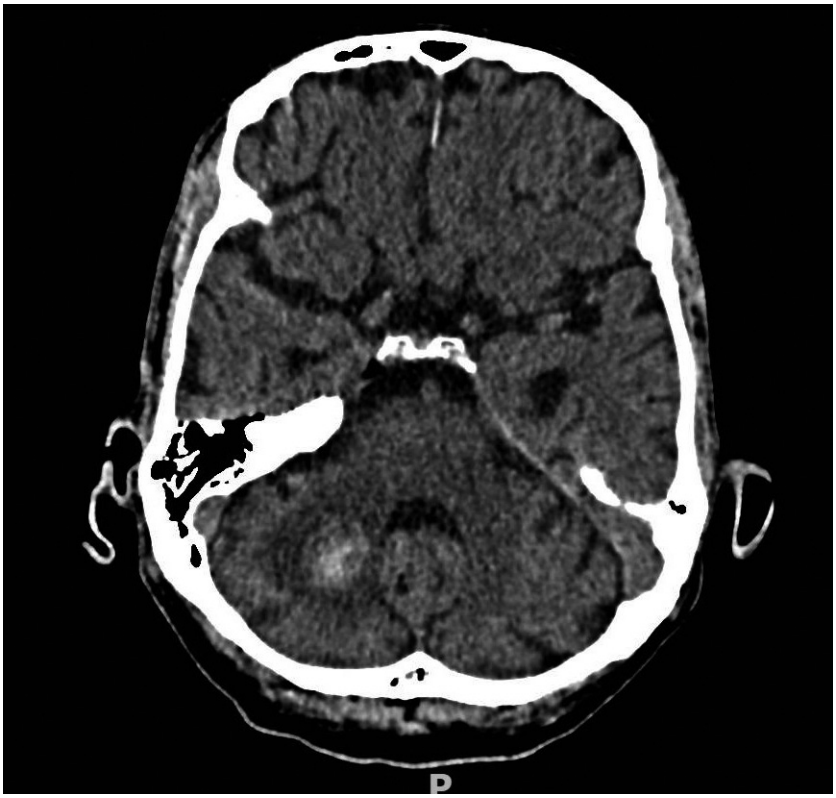

Figure 1. 18 mm right cerebellar haemorrhagic focus with adjacent oedema.

hyponatraemia, it is important to consider whether a patient has experienced a traumatic brain injury, an intracranial bleed or recent neurosurgery. Despite having somewhat similar laboratory results, the management of cerebral salt wasting is in direct contrast to that for syndrome of inappropriate antidiuretic hormone secretion, so reaching the correct diagnosis before treatment is started is essential. This article describes the case of a patient initially diagnosed with syndrome of inappropriate antidiuretic hormone secretion and then correctly diagnosed and treated for cerebral salt wasting with intravenous fluids.

Discussion

Cerebral salt wasting is the result of an insult to the CNS (Tenny and Thorell, 2020). The incidence of cerebral salt wasting is inconsistently reported, but appears rare among the general population (Orlik et al, 2019). The condition is most commonly diagnosed in those experiencing a traumatic brain injury, with a review estimating an incidence ranging from 0.8% to 34.6% within this cohort of patients (Tenny and Thorell, 2020).

Cerebral salt wasting presents with hypovolaemia, low blood sodium levels and normal renal function (Orlik et al, 2019). The correct diagnosis of cerebral salt wasting lies in being able to differentiate it from syndrome of inappropriate antidiuretic hormone secretion (Tenny and Thorell, 2020). The difficulty for physicians is that both conditions cause hyponatraemia, with similar biochemical and physical manifestations (Nakajima et al, 2017), but treatment regimens for these conditions are contradictory as a result of the differing aetiologies. For syndrome of inappropriate antidiuretic hormone secretion, treatment includes fluid restriction, while for cerebral salt wasting, because patients are hypovolaemic, the exact opposite is required (Tenny and Thorell, 2020). Once fluids have been administered, it is essential that the underlying causes of cerebral salt wasting are not exacerbated by this (Tenny and Thorell, 2020).

Centres offering neurosurgery report that cerebral salt wasting is a common occurrence following surgery. A 2015 review of the literature related to cerebral salt wasting found that approximately half of published papers were in neurology or neurosurgery-focused journals (Leonard et al, 2015). The condition is infrequently diagnosed within the district

Learning points

- While cerebral salt wasting is rarely seen in district general hospitals, it is imperative to consider it as a differential diagnosis and to not rule all cases of hyponatraemia as syndrome of inappropriate antidiuretic hormone secretion.
- Given the differing treatment regimens for cerebral salt wasting and syndrome of inappropriate antidiuretic hormone secretion, healthcare workers must be able to accurately diagnose and treat each condition.
- In cases of worsening hyponatraemia, especially after a traumatic brain injury, early endocrine review is key to ensuring appropriate treatment.

general hospital setting and, consequently, is often overlooked as a case of syndrome of inappropriate antidiuretic hormone secretion, as occurred in this case.

To improve the treatment of cerebral salt wasting and subsequent patient outcomes, it imperative that awareness of the condition is increased among healthcare professionals. This is particularly important in district general hospitals where the condition is more likely to be incorrectly diagnosed as syndrome of inappropriate antidiuretic hormone secretion, as a result of its rare nature. Understanding the fundamental difference between cerebral salt wasting and syndrome of inappropriate antidiuretic hormone secretion allows timely diagnosis and appropriate management of patients with each condition.

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