Atypical presentation of Addison's disease

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Abstract

Hyponatraemia is a relatively common electrolyte problem encountered in hospitalised patients and it carries significant morbidity and mortality. It is challenging to spot the exact cause of hyponatraemia especially when it is associated with multiple comorbidities, such as hypothyroidism, obesity and lymphoedema. We present a case of hyponatraemia in a patient with known hypothyroidism and lymphoedema. Initially the patient was fluid restricted, considering hypervolemic hyponatraemia due to lymphoedema and hypothyroidism, but following a continued drop in sodium levels, the diagnosis was reconsidered. Thorough examination showed hyperpigmentation which directed the measurement of cortisol levels. That resulted in a diagnosis of Addison's disease and rapid recovery of the patient occurred after steroid replacement.

Case presentation

An 81-year-old woman presented with a complaint of generalised weakness and lethargy for 3 weeks. She had a background of hypothyroidism, osteoarthritis, bilateral long-standing lymphoedema and tuberculosis. Her initial bloods showed the following levels: sodium (Na) 114 mmol/L, potassium 4.8 mmol/L, urea 6.7 mmol/L, creatinine 83 µmol/L and thyroid stimulating hormone 31 mIU/L, T4 was 8 pmol/L. She was managed with fluid restriction based on lymphoedema secondary to hypothyroidism developing due to poor compliance with thyroxine. Her Na levels failed to improve, but rather worsened to 112 mmol/L on the second day post-admission. Her serum and urine osmolalities were 248 mOsmol/kg (low) and 360 mOsmol/kg, respectively. On closer examination she had tanning of the skin, and her 9am cortisol level was low (238 nmol/L). A short Synacthen test was arranged and showed no improvement at all after 15, 30 and 60 minutes post-Synacthen injection. A diagnosis of Addison's disease was further supported with computed tomography of the abdomen that showed bilateral adrenal calcification post-infection. She was treated with intravenous hydrocortisone and made a rapid recovery. Thyroxine was started from the next day. Her Na improved to 118 mmol/L and later to 124 mmol/L. Her adrenocorticotrophic hormone (ACTH) level came back as high (108 ng/L) and confirmed the diagnosis of Addison's disease.

Discussion

To diagnose hyponatraemia, the first step is to confirm hypotonic hyponatraemia by serum osmolality, followed by assessment of the volume status and urine osmolality. In our patient, assessment of her volume status was complicated due to lymphoedema. And under substitution of levothyroxine could have contributed to hyponatraemia.

The usual initial management for hyponatraemia is fluid restriction, which is not helpful in hypocortisolism where aldosterone deficiency leads to increase urinary excretion of Na, which results in a decrease of intravascular volume. Hypotension and decreased cardiac output cause an increase in secretion of antidiuretic hormone (ADH) from posterior pituitary to absorb more water, leading to further reduction in Na concentration. There is no negative feedback to suppress ADH from the low level of cortisol, hence fluid restriction alone doesn't improve the serum Na level and hydrocortisone replacement is needed to improve the Na level.

The most common cause of Addison's disease is autoimmunity in the UK, although tuberculosis is still the leading cause worldwide. ■

Learning points

- In hyponatraemia, serum osmolality, urine osmolality and urine Na should be tested, then assess the volume status of the patient to identify the cause of hyponatraemia.
- If the patient is not responding to fluid restriction, Addison's disease should be considered.
- Tuberculosis is the most common cause of Addison's disease worldwide, although autoimmune causes are common in the UK.

Conflicts of interest

None declared.

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