**Conclusion**

This is the first reported case of a black patient in sub-Saharan Africa presenting with Riedel’s thyroiditis accompanied by extensive fibrosis.

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**CASE REPORT**

**A pituitary macroadenoma presenting with hyponatraemia**

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A non-functional pituitary macroadenoma commonly presents with headaches and/or visual field defects, which may even extend to blindness. Although symptoms are often present before diagnosis they are frequently not appreciated because they are nonspecific, require a focused history and are therefore erroneously attributed to other causes. Hyponatraemia is a common electrolyte disturbance with many different causes, but is rarely due to hypopituitarism. Yet hyponatraemia is potentially life-threatening, requiring a prompt diagnosis and initiation of appropriate therapy. In the setting of hypopituitarism this may merely be hormone replacement, obviating the need for meticulous fluid replacement to avoid central pontine myelinolysis (CPM).

We report on a patient presenting with hyponatraemia secondary to hypoadrenalism and hypothyroidism due to a pituitary macroadenoma.

**Case report**

A 67-year-old man presented to the medical emergency department with a 2-week history of left-sided lower abdominal pain and constipation. He did not complain of vomiting but did have a 1-year history of weight loss. There was no significant past medical history and he was not taking any medication.

Clinical examination revealed a well-looking patient who was not confused, was apyrexial, and had normal hydration. A chest radiograph was normal; a blood pressure of 167/92 mmHg with no postural drop, a pulse rate of 54 beats/min and a respiratory rate of 18 breaths/min. Apart from mild tenderness in the left lower abdomen, systemic examination was unremarkable. The patient was an elderly man with a change in bowel habits and loss of weight, the concern at this stage was that he might have a bowel malignancy and he was admitted to the emergency medical admission ward.

The findings on baseline biochemical investigation were as follows: sodium 108 mmol/l (normal 135 - 141 mmol/l), potassium 4.5 mmol/l (3.3 - 5.3 mmol/l), urea 1.5 mmol/l (2.6 - 7.0 mmol/l), creatinine 46 µmol/l (60 - 120 µmol/l), urinary sodium on a random urine sample 149 mmol/l, haemoglobin 10.2 g/dl (13.0 - 17.0 g/dl), mean cell volume 103.8 fl (79.1 - 98.9 fl), white cell count 4.42×10⁹/l (4.00 - 10.00×10⁹/l), platelets 191×10⁹/l (137 - 373×10⁹/l). A chest radiograph was normal. A working diagnosis of syndrome of inappropriate antidiuretic hormone secretion (SIADH) was made and the patient was placed on fluid restriction. Since his chest radiograph was normal a magnetic resonance image (MRI) scan of the brain was done and showed a well-defined pituitary lesion measuring 18 mm by 18 mm, compatible with a pituitary macroadenoma (Fig. 1). The patient was then referred to the endocrine...
service. Clinical review by an endocrinologist added the following important information and clinical findings. The patient reported fatigue, axillary hair loss, a reduction in the frequency of shaving and an increasing breast size. Examination confirmed that he had gynaecomastia, decreased axillary hair and soft, smooth facial skin; his visual fields were normal and he had no clinical features of hormone hypersecretion.

The results of hormonal assessment were as follows: free T4 6.2 pmol/l (normal 12 - 22 pmol/l), thyroid stimulating hormone (TSH) 2.88 mIU/l (0.27 - 4.2 mIU/l), 08h00 cortisol 71 nmol/l, follicle-stimulating hormone (FSH) 4.3 IU/l (1.7 - 8.6 IU/l), testosterone 9.8 nmol/l (9.9 - 27 nmol/l), prolactin 50.8 µg/l (4.6 - 21.4 µg/l), and random growth hormone <0.9 µg/l.

A diagnosis of panhypopituitarism secondary to a pituitary macroadenoma was made and the patient was started on hormonal replacement therapy with hydrocortisone 10 mg at 08h00, 5 mg at 12h00 and 5 mg at 18h00 followed after 24 hours with levothyroxine 25 µg daily and testosterone 50 mg intramuscularly monthly. By 10 days after admission his serum sodium had normalised. He was referred to the Neurosurgical Department for assessment for transsphenoidal surgical debulking of this non-functional pituitary tumour.

Discussion

Hyponatraemia is a common electrolyte disorder with numerous causes. It is often present in patients with intracranial pathology or those recovering from neurosurgery. SIADH has often been perceived to be the main culprit; however, it is increasingly becoming appreciated that the cerebral salt wasting syndrome (CSWS) may be more common than previously thought. Distinguishing between the two is essential as treatment differs, SIADH being treated with fluid restriction and CSWS with aggressive fluid replacement. Since both conditions probably occur with a similar frequency and in both there is an increased loss of sodium in the urine, other parameters are required to help differentiate the two.

The main distinguishing clinical feature is euvoalaemia in patients with SIADH and hypovolaemia in patients with CSWS. However, it is often difficult to determine fluid status clinically. Guerrero et al. suggest other clinical and biochemical parameters that may be useful in distinguishing between the two (Table I).

| Clinical parameters | CSWS | SIADH |
|---------------------|------|------|
| Plasma volume       | ↓    | ↑    |
| Signs/symptoms of volume depletion | Present | Absent |
| Weight              | ↓    | ↑ or no change |
| Central venous pressure | ↓    | ↑ or normal |
| Haematocrit         | ↑    | ↓ or no change |
| Serum osmolality    | ↑    | ↓ |
| Serum protein level | ↑    | Normal |
| Urinary sodium level | ↑ or no change | ↓ or no change |
| Urinary potassium level | ↑ or no change | ↓ or no change |
| Uric acid level     | Normal | ↓ |

*Adapted from Guerrero et al.*

The hyponatraemia in SIADH is caused by inappropriately excessive ADH resulting in free water reabsorption in the terminal distal tubule and medullary collecting ducts of the kidney. Less understood is the mechanism of natriuresis in this condition, which has been attributed to an increase in the glomerular filtration rate or a reduction in the tubular reabsorption of sodium. A diagnosis of SIADH is made in the presence of a plasma osmolality <275 mOsm/kg H₂O in addition to: inappropriate urine concentration (urine osmolality >100 mOsm/kg H₂O), clinical euvoalaemia and an elevated urinary sodium excretion in the presence of a normal salt and water intake. It is important to note that hypothyroidism, hypoadrenalism and diuretic use must be excluded prior to making a diagnosis of SIADH.

CSWS results from excessive natriuresis in patients with intracranial disease. The mechanism underlying this natriuresis is unknown but has been proposed to involve the release of natriuretic factors (atrial natriuretic factor (ANF), brain natriuretic factor (BNP), c-type natriuretic peptide and ouabain-like peptide) together with decreased sympathetic input to the kidney (the sympathetic nervous system is involved in salt and water handling by the kidney). Of the natriuretic factors, BNP is thought to be the most likely candidate, resulting in excessive sodium loss by the kidney.

Apart from SIADH and CSWS, hyponatraemia occurring in the setting of a pituitary mass lesion could also be due to hypoadrenalism and
hypothyroidism. Cortisol is an inhibitor of ADH secretion, hence secondary hypoadrenalinism results in increased ADH secretion and consequently there is decreased excretion of water by the kidney and hyponatraemia. Although primary hypothyroidism is more commonly associated with hyponatraemia, it may also occur in severe secondary hypothyroidism. The underlying pathophysiological mechanism/s of hyponatraemia in hypothyroidism remains unclear, but is thought to involve a reduction in GFR due to decreased renal perfusion and retention of water in the tissues thereby increasing total body water. A 20-year retrospective analysis by Diederich et al. reported that 28 of 139 patients with hyponatraemia seen in their endocrine unit were found to have hypopituitarism and secondary hypoadrenalinism. Furthermore, replacement with hydrocortisone was sufficient treatment for the hyponatraemia, suggesting that concurrent secondary hypothyroidism played a secondary role. Recognising the association of hyponatraemia with hypopituitarism is clinically important, as treatment involves replacement of cortisol and thyroid hormone using hydrocortisone and levothyroxine respectively. This obviates the need for meticulous fluid control to prevent central pontine myelinolysis (CPM). In chronic hyponatraemia it is generally advised not to increase the serum sodium by more than 8 - 12 mmol/l per day because of the risk of precipitating CPM. Interestingly, there are no data on the rapidity with which the serum sodium may rise in response to cortisol or thyroid hormone replacement. Furthermore, there are no case reports of CPM in this group of patients.

In summary, our patient with a pituitary macroadenoma presented to an emergency unit with nonspecific symptoms and hyponatraemia. This report highlights the importance of considering hypopituitarism in this setting, as treatment can be life-saving.

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