Collapse and hypothermia in an elderly woman in early summer

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A 74-year-old woman was admitted during a warm spell, having been found at home collapsed. She was conscious but confused (Abbreviated Mental Test score (AMTS) 4/10). Her sister related her medical history which included an underactive thyroid diagnosed 8 years before, an operation to close an atrial septal defect as a young woman, a small stroke and several admissions over the last 3–4 years with difficulty coping and confusion. In fact, she had been told by one doctor that her sister had dementia. The patient was taking digoxin 62.5 µg, co-amilofruse 2 tablets, and thyroxine 50 µg, all once daily. On examination she was pale and drowsy and her rectal temperature was 33.6°C. Body hair was absent. The pulse rate was 60 beats/min, irregularly irregular, and her blood pressure was 120/60 mmHg. Although the heart was clinically enlarged, auscultation was normal. There were fine crackles audible at the lung bases. Apart from the cognitive impairment, central nervous system examination was normal.

Initial investigations showed haemoglobin 10.4 g/dl (mean cell volume 88 fl), white cell count $3.9 \times 10^9/l$, platelets $88 \times 10^9/l$, urea, electrolytes and thyroid function tests were normal. Plasma glucose was 1.4 mmol/l. An electrocardiogram revealed slow atrial fibrillation with a prolonged QT interval and right axis deviation. A chest X-ray confirmed cardiomegaly. The patient was given 50 ml of intravenous 50% dextrose with some improvement in her confusion and she was slowly re-warmed. She was then able to eat and drink. The following day her serum sodium was 123 mmol/l and blood glucose 2.0 mmol/l. Her old notes at this time revealed that during the admissions with confusion and not coping over recent years, her sodium had often, and her glucose had occasionally, been low. The notes also confirmed that she had had primary hypothyroidism diagnosed 8 years before. Computed tomography (CT) was performed and, following injection of contrast medium, a coronal image through the pituitary fossa obtained (figure).

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Figure Coronal CT scan showing view through pituitary fossa

Questions

- 1 What further investigations would confirm the diagnosis and what is the significance of the CT findings?
- 2 Why did she present with hypothermia?
- 3 What is the most probable mechanism causing the hypoglycaemia?
- 4 What is the cause of the hyponatraemia?

Answers

QUESTION 1

The further investigations which were carried out are shown in the table.

Table Investigations

	Time after ACTH (min)	Control (nmol/l)	Expected (nmol/l)
Short synathen test	0	139	
	30	228	
	60	285	>500
Follicle-stimulating hormone (U/l)	0.7		Post-menopausal range 30–72.6
Luteinising hormone (U/l)	<0.3		27.6–132.9 Normal range 0–700
Prolactin (mU/l)	518		0.2–13
Growth hormone (mU/l)	1.1		93–309
Insulin growth factor (µg/l)	87		

The diagnosis is hypopituitarism in association with an empty sella. The patient also has primary hypothyroidism. The empty sella syndrome (ESS) may be primary (idiopathic) or secondary. Idiopathic ESS is thought to arise as a consequence of congenital incompetence of the diaphragma allowing herniation of the arachnoid mater and cerebrospinal fluid (CSF) into the fossa, ultimately compressing the pituitary gland. This is most commonly seen in middle-aged obese women and may be associated with raised intracranial pressure but endocrine dysfunction is rarely observed.¹ In secondary ESS the meninges and CSF herniate downwards to fill the void created by pituitary involution as a result of other pathology, eg, surgery, irradiation, infarction, haemorrhage or tumour necrosis. Although the occurrence of ESS in this patient may be an incidental finding, it is possible that she developed secondary ESS as a consequence of thyroxine-induced shrinkage of thyrotroph cell hyperplasia following treatment for her primary hyperthyroidism. This effect has been previously reported.²

1 Braatvedt GD, Corral RJM. The empty sella syndrome: much ado about nothing. *Br J Hosp Med* 1992;47:523–5.

2 Kelestimur F, Selcuklu A, Ozcan N. Empty sella developing during thyroxine therapy in a patient with primary hypothyroidism and hyperprolactinaemia. *Postgrad Med J* 1992;68:589–91.

3 Haskell RM, Boruta B, Rotondo MF. Hypothermia. *AACN Clin Issues* 1997;8:368–82.

QUESTION 2

The patient may have been hypothermic because thyroid and adrenal hormones are involved in non-shivering thermogenesis and deficiency of one or both may predispose to hypothermia.³ Hypoglycaemia is thought to predispose to hypothermia independently of insulin, either as a direct effect or secondary to a glycopenic effect on the thermoregulatory centre.⁴ Pituitary disorders, through anatomical proximity to the thermoregulatory centre, may cause disturbances in thermoregulation. The patient's age may also have predisposed to hypothermia.

QUESTION 3

Deficiencies in the anti-insulin hormones: adrenocorticotropin and growth hormone are responsible for the hypoglycaemia seen in hypopituitarism. It was felt that the patient's periods of confusion were related to episodes of hypoglycaemia.

QUESTION 4

Cortisol deficiency results in increased secretion of vasopressin and dilutional hyponatraemia develops as a consequence. Renal water excretion is also impaired independent of this antidiuretic effect because of decreased tubular fluid delivery to the diluting site.⁵ This is in contrast to the salt-wasting state seen in Addison's disease.

Outcome

The patient responded well to treatment with hydrocortisone. In particular, her cognitive function improved to an AMTS of 9/10 and has been maintained at that since.

Final diagnosis

Hypopituitarism in association with empty sella syndrome.

Keywords: hypopituitarism; empty sella syndrome; hypothermia; hypoglycaemia

4 Freinkel N, Metzger BE, Harris E, *et al.* The hypothermia of hypoglycaemia. *N Engl J Med* 1972;287:841–5.

5 Kageyama Y, Hirose S, Terashi K, *et al.* A case of Sheehan's syndrome associated with severe hyponatraemia and congestive heart failure. *Jpn J Med* 1988;27:337–41.