

A disappearing act in the pituitary fossa with recovery from hypopituitarism

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A 36-year-old, previously healthy, gentleman presented with several weeks' history of gradually worsening frontal headache. He attended the Emergency Department after he was awoken by sudden worsening of the headache, associated with vomiting and pre-syncope symptoms. He did not report any visual problems.

On further questioning, he described decreased libido for several weeks. On examination, he was alert and orientated, hypothermic at 35.1°C and bradycardic - 55bpm. He had no evidence of meningeal irritation but examination of vision revealed an right upper, lateral quadrantanopia.

Investigations:

- Severe hyponatraemia with a serum sodium 109mmol/L. He was also profoundly hypocortisolaemic with a random serum cortisol of 16 nmol/L
- CT head, and later MRI, pituitary demonstrated a 17mm suprasellar, complex, cystic pituitary lesion compressing the optic apparatus (panel A); there was no calcification within the mass on CT (not shown).

	Baseline
Free T4 (pmol/L)	6.1
TSH (mUnit/L)	0.68
Cortisol (nmol/L)	16
ACTH	19
Prolactin (0 -324 mUnit/L)	101
Testosterone (8-29 nmol/L)	0.8
SHBG (nmol/L)	38
Insulin-like growth factor ref. range 99-367(mcg/L)	94

Table 1: Pituitary profile at presentation

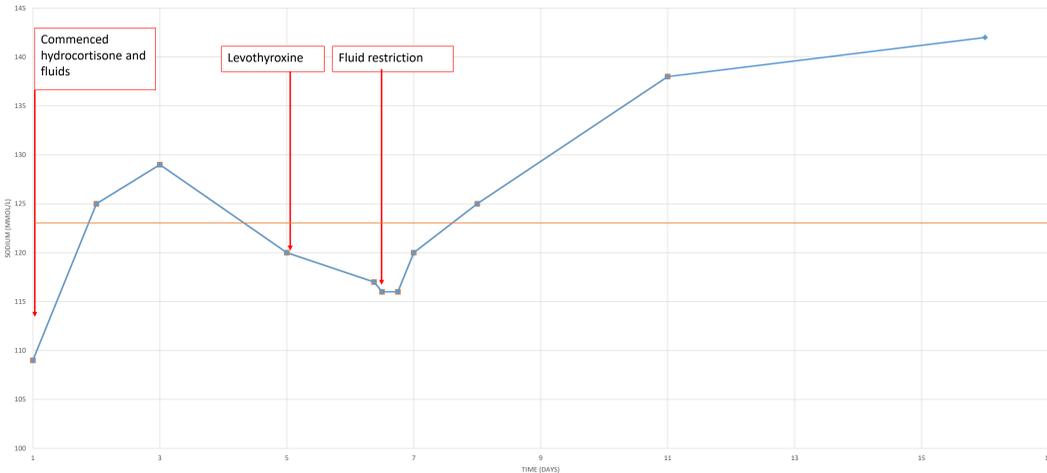
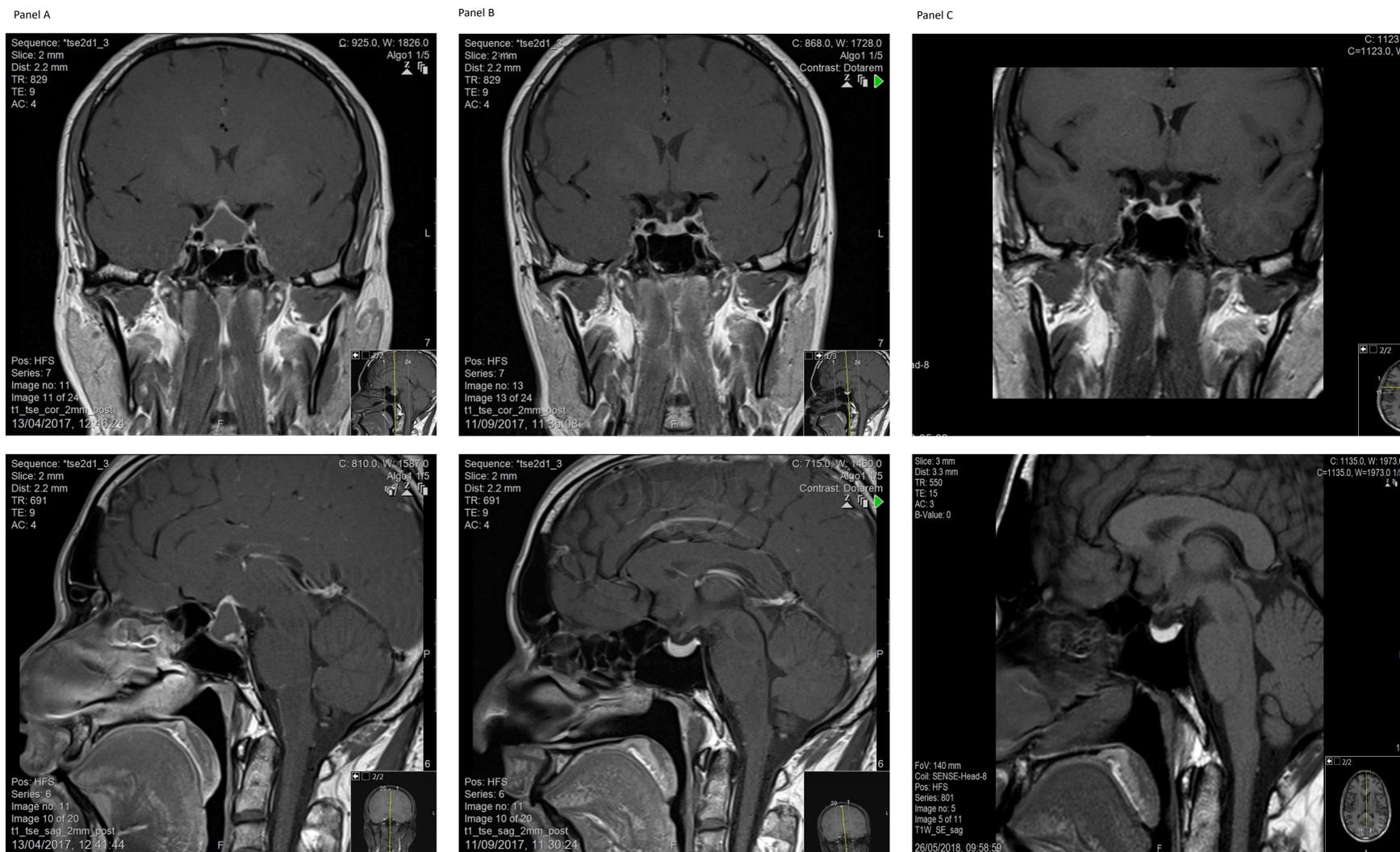


Figure 1. Serum sodium concentration at time points from presentation with annotations of interventions

Management: Treated with IV 0.9% saline and glucocorticoids. Serum Na initially improved to 129mmol/L and he felt better. However, after 4 days, he complained of headache with nausea - serum sodium declined again (Figure 1). Clinically euvoelaemic and repeat biochemical assessment confirmed a persistent state of antidiuresis despite replacement of glucocorticoids - serum sodium 116, urine osmolality 726mOsm/kg, urine Na 62mmol/L. He was then treated with fluid restriction, while continuing glucocorticoids and T4, and serum sodium gradually returned to the normal range.

Progress: He had panhypopituitarism, without polyuria (table 1). Serum prolactin was not elevated, so the neurosurgical team planned transsphenoidal debulking; however, repeat pituitary imaging, six weeks later, demonstrated substantial shrinkage of the lesion, now confined to the sella (panel B).

Figure 2 Serial pituitary MRI scans. Panel A: Presentation- Enlarged pituitary fossa containing a 17mm cystic mass lesion, suprasellar extension deforming the chiasm. Enhancement of the cyst wall and stalk appears foreshortened. **Panel B:** 6 weeks – Cystic tissue is completely within the sella and significantly reduced in size with no chiasm compression. **Panel C:** 6 months- Normal volume of pituitary tissue which is of diffuse T1 high signal suggesting proteinaceous or old haemorrhagic content. Optic chiasm is normal and stalk midline.



Recovery: Six months after presentation, the lesion had regressed further and dynamic endocrine testing, including insulin tolerance test (ITT), demonstrated almost complete recovery of pituitary function; FT4 18.6 pmol/L, TSH 0.03 mUnits/L, testosterone 16.2 nmol/L, IGF-1 182 mcg/L. Insulin tolerance test demonstrated normal ACTH reserve and persistent, isolated growth hormone deficiency - peak cortisol of 846 nmol/L and peak growth hormone of 2.07ug/L. Eighteen months after presentation, he remains well, on no medication

Discussion: Differential diagnosis includes cystic pituitary adenoma or Rathke's cleft cyst. Craniopharyngioma is less likely. This case highlights the occurrence of spontaneous, almost complete, recovery from hypopituitarism which is rare in patients presenting with a suprasellar pituitary mass. Long-term surveillance is required due to the risk of tumour recurrence. Furthermore, the case highlights the complexity of the diagnosis and management of severe, symptomatic hyponatraemia, in patients with a newly discovered sellar lesion.