

diverticula result from high intragastric pressure. Alternatively, prepyloric diverticula might lead to gastric stasis, pyloric ulceration and stenosis. In both instances local muscular weakness must play an important part since even longstanding pyloric stenosis is seldom associated with diverticulosis; gastric diverticula have lately been reported in a patient with Caroli's disease,<sup>6</sup> a deficiency of the fibromuscular matrix of the biliary tree.

In the present case, the true diagnosis became evident only after gastrectomy. A technique that might possibly have led to earlier diagnosis is endoscopic ultrasound-guided fine-needle aspiration biopsy.<sup>7</sup>

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## Low TSH in a patient with primary hypothyroidism

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For routine thyroid function testing many laboratories now confine themselves to assay of thyroid stimulating hormone (TSH). This will identify the great majority of affected patients<sup>1</sup> though it can miss secondary hypothyroidism.<sup>2,3</sup> TSH measurement is also used to monitor thyroxine replacement, and on rare occasions this too can be misleading.

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## CASE HISTORY

A woman of 66 was seen in the emergency department with a history of recurrent falls, urinary incontinence and new-onset confusion. She walked with a shuffling small-stepped gait and her abbreviated mental test score was 1 out of 10. 10 years earlier she had been found to have primary hypothyroidism, TSH 12 mU/L (reference range 0.3–4.2), and on treatment with thyroxine 100 µg daily the TSH had become normal. A year before the present episode her TSH was noted to have fallen to 0.49 mU/L, which in the absence of a free T4 was interpreted as suggesting that she was taking too high a dose of thyroxine. The dose was reduced to 75 µg daily and three months later the TSH was 1.26 mU/L. However, a further six months later the TSH had fallen to 0.2 mU/L and the thyroxine dose was once again reduced, to 50 µg daily.

CT of her head showed hydrocephalus and a large cystic lesion in the region of the pituitary. MRI confirmed these findings (Figure 1a,b). Her baseline pituitary hormonal profile showed a very high prolactin of 398 500 mU/L (<650). The TSH was now raised at 22.0 mU/L, with a free thyroxine of 9.6 pmol/L (9.0–26.0); she had not taken thyroxine for about two weeks because of her confusion. Further assessment revealed a follicle stimulating hormone of 0.4 IU/mL, undetectable luteinizing hormone, growth hormone 0.3 IU/L with an IGF-1 4.0 nmol/L (6.0–30.0), and cortisol 765 nmol/L. These results indicated partial hypopituitarism. Thyroid peroxidase antibodies were strongly positive. She had a bitemporal hemianopia on perimetry. She was restarted on thyroxine replacement.

Transcranial drainage of the intraventricular cystic lesion did not improve her clinical state or lessen the hydrocephalus (Figure 1c). She was therefore started on cabergoline 500 µg twice weekly, and over the subsequent nine months her tumour shrank (Figure 1d–f), her visual fields improved, and her confusion resolved completely, to the extent that she was able to proof-read this paper.

## COMMENT

This patient with primary autoimmune hypothyroidism went on to develop a giant macroprolactinoma. We propose that the continuous decline in her TSH despite reduction of the thyroxine dose was due to partial pituitary failure. Such a phenomenon has been observed by others;<sup>4</sup> the low TSH in this patient reflected the development of hypopituitarism, not over-replacement leading to thyrotoxicosis.

The routine measurement of TSH to monitor the adequacy of thyroxine replacement has in this case given a fascinating insight into the changes occurring in the thyroid axis as hypopituitarism developed. The use of TSH as the sole index of function led to erroneous interpretation and inappropriate reductions of thyroxine dosage. Only when

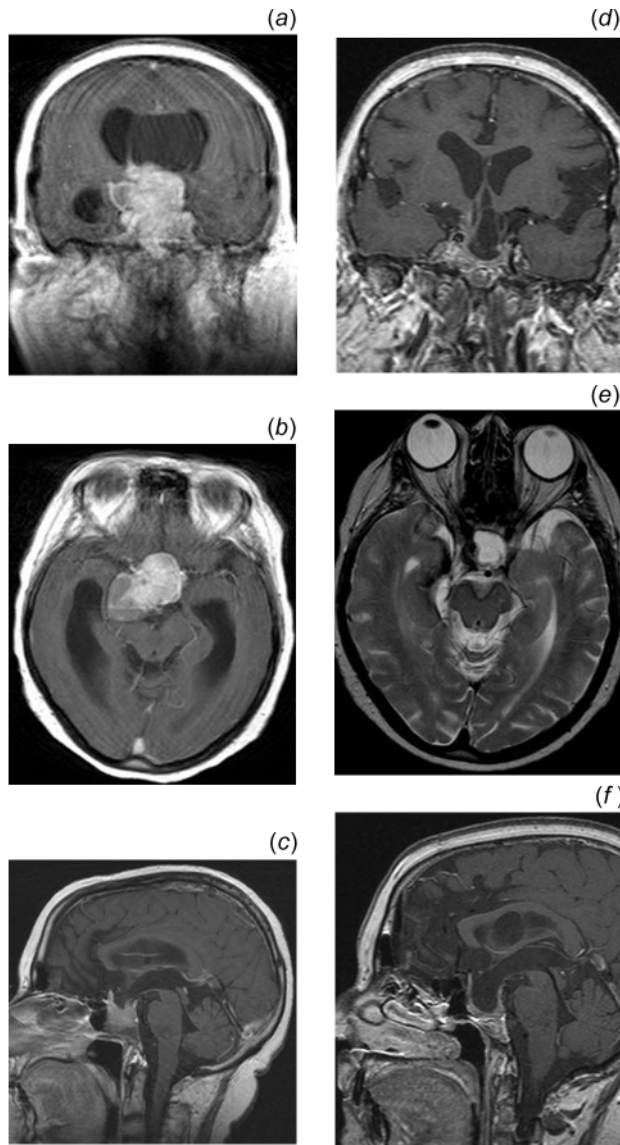


Figure 1 MRI of head (a, b) on admission (with movement artefact); (c) immediately after cyst drainage; (d-f) after nine months' treatment with cabergoline

the patient was taking half the correct thyroxine dose, and stopped taking thyroxine altogether for a short period, did the TSH rise, by which time the pituitary tumour had caused hydrocephalus severe enough to produce confusion, incontinence and falls. Although the dose of thyroxine required to maintain the euthyroid state has been reported to fall with age,<sup>5</sup> this would not have been a plausible explanation for the rapid decline in TSH seen in this patient. Had the free thyroxine also been measured, it might have prompted a search for other causes of a falling TSH during thyroxine replacement and led to diagnosis of the macroprolactinoma before the development of hydrocephalus.

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**Cold feet from adrenal leiomyosarcoma**

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ADRIAN TANNER PRIZE, JUNE 2004

Among the rarer causes of cold feet is inferior vena cava thrombosis.

**CASE HISTORY**

A previously fit man of 57 was seen by the general physicians after acute onset of right groin pain with cold feet and altered sensation in both legs. On examination the feet were cold and cyanosed and the leg pulses were detectable only by doppler sonography. Examination was otherwise unremarkable. An ultrasound scan revealed bilateral femoral vein thrombosis and he was started on anticoagulants. A duplex scan then demonstrated thrombosis of the inferior vena cava (IVC) and both iliac veins. On abdominal CT he was found to have a massive left adrenal tumour involving the left kidney with tumour thrombus extending from the left renal vein into the IVC as far as the right atrium (Figure 1). A team including endocrine, vascular and cardiac surgeons was assembled. Additional investigations included a hormonal screen to exclude phaeochromocytoma, coronary angiography and

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