Pituitary enlargement due to hypothyroidism

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A forty-year-old female presented to the neurologists with a 6-months history of episodic left-sided retro-orbital headaches not responding to analgesia. Brain MRI was reported as showing a 10 mm pituitary adenoma. Subsequently, she was referred to endocrinology. She did not have any visual problem, galactorrhoea or features of acromegaly or of Cushing’s. She had a history of primary hypothyroidism treated with Levothyroxine and her TSH had been high on many occasions for the last 5 years, despite reporting compliance during the first years of the diagnosis. Later, as she did not observe any change in her symptoms, she was not taking it regularly. Her past history included bowel resection for infected diverticulum (since then has had episodes of diarrhoea), adhesiotomy and cholecystectomy. She was on colestyramine, codipar and had a mirena coil. Her BMI was 22 kg/m². She had a moderate diffuse goitre. The remaining of the physical examination did not reveal remarkable findings.

Investigations
TSH >150 mU/L (0.35-5.5), FT4 7.6 pmol/L (11.5-22.7), FT3 2.8 pmol/L (3.5-6.5), Prolactin 301 mU/L (60-620), AntiTPO Abs >3000 IU/ML (0-60). Short Synacthen test showed normal response, IGF-1 8.11 nmol/L (10.5-35). The review of her scans suggested generalised enlargement of the pituitary gland, but no discrete mass.

Treatment
Thyroxine replacement was started at 50 μg/day and titrated up to 175 μg/day. TFTs gradually normalised and MRI scan after 6 months of adequate thyroxine replacement showed normal pituitary gland.

Discussion
This is a case of long-standing non-successfully treated primary hypothyroidism leading to pituitary enlargement. The difficulty in achieving biochemical euthyroidism was attributed to malabsorption (bowel surgery and colestyramine), as well as to non-compliance. This case illustrated the importance of differentiating pituitary hyperplasia due to primary hypothyroidism and a pituitary tumour.