Autoimmune hypothyroidism in the presence of TSH secreting pituitary adenoma: a case report
AA Tahrani, J Ayuk, S Ranagan, MA Karamat, R Mitchell, N Gittoes and J Franklyn.
The Centre for Endocrinology Diabetes and Metabolism, School of Clinical and Experimental Medicine, University of Birmingham

A 69-year old man presented to another endocrine centre with weight gain and constipation. He had no family history of thyroid disease. Clinical examination was unremarkable. Thyroid examination showed no evidence of thyroid enlargement. Thyroid function tests (TFTs) showed evidence of autoimmune subclinical hypothyroidism [Thyroid stimulating hormone (TSH) 29.14 mIU/L (normal range: 0.4-4.5), Free T4 (FT4) 16.9 pmol/l (10-22), thyroid peroxidase (TPO) antibodies 3311 IU/ml (0-34)]. Due to the patients’ symptoms and the TSH levels, the patient was commenced on thyroxine. Repeated TFTs showed persistently elevated TSH levels and hence the dose of thyroxine was increased gradually to 250 micrograms daily over 2-year period. Despite that, TSH levels remained above the normal range and the FT4 levels became elevated (TSH: 15.7 mIU/L, FT4: 27.5 pmol/L) and thyroxine was stopped. The patient denied compliance issues. Heterophilic antibodies to TSH, T4 and T3 were undetectable.

On referral to our centre, TFTs showed evidence of subclinical hypothyroidism with significantly raised TSH levels [TSH 55.6 mIU/L, FT4 12.8 pmol/L, FT3 4.5 pmol/l (3.5-6.5)] and as a result the patient was started on thyroxine 50 micrograms daily. A year later, the patient was taking 100 micrograms daily and re presented with sweating and heat intolerance. TFTs showed elevated TSH with normal FT4 levels (25.6 pmol/l and 18.66 mIU/L respectively). α-subunit was elevated at 2.9 IU/L (<1.00). Further investigations showed evidence of secondary hypogonadism [testosterone: 6.2 nmol/l (9-28) without appropriate elevation of gonadotrophins]. Prolactin, IGF-1 and cortisol levels following ACTH stimulation were all normal. Pituitary MRI showed 1.7x1.5 cm pituitary macroadenoma. The patient was commenced on testosterone replacement and continued thyroxine 100 micrograms daily.

It was felt that the pituitary macroadenoma most likely represented a TSH-oma and the patient was started on octreotide 100 micrograms thrice daily. This resulted in normalisation of the TFTs and α-subunit (TSH: 0.66 mIU/L, FT4: 18.2 pmol/l, FT3: 13.4 pmol/l, α-subunit 0.35 IU/L) and the patient was switched to somatulin autogel 60 mg once monthly. Repeat pituitary MRI 9 months later showed enlarged pituitary fossa and bulky pituitary gland without identifiable focal lesion. Follow up TFTs and α-subunit levels remained normal up till now.

In summary, our case represents an unusual situation in which autoimmune hypothyroidism coexisted with a TSH secreting pituitary adenoma. The presence of hypothyroidism protected the patient from the thyrotoxic effects of the TSH-oma. Thyroxine treatment unmasked the underlying TSH-oma biochemically. This case suggests that exploring the possibility of a TSH-oma is indicated in patients with hypothyroidism and persistently elevated TSH levels in the presence of elevated FT4 levels on thyroxine replacement.