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An adolescence girl with an anterior mediastinal mass

W S Chow, A W C Kung

A 14-year-old Chinese girl was referred to us for assessment of her thyrotoxicosis before corrective surgery of genu varus. On questioning, she mentioned significant weight loss with occasional palpitations. She had also experienced episodic lower limb proximal muscle weakness, but there was no history of paralysis.

Physical examination showed sinus tachycardia of 110 beats/min. There was no lid retraction or lid lag. Her goitre was enlarged three-fold with audible bruit, but there was no retrosternal extension. Eye examination revealed no evidence of Graves’ ophthalmopathy. Abdominal examination was normal. There was no lymphadenopathy, splenomegaly nor pretibial myxoedema. Her proximal muscle power was grade 4/5 and the jerks were all brisk.

Investigations showed a raised free thyroid hormone of >170 pmol/l (normal range 10–19 pmol/l) and a suppressed thyrotropin of <0.03 mIU/l (0.35–5.5 mIU/l). Chest X-ray, however, revealed a mediastinal shadow with clear lung field. Complete blood count and routine biochemistry were normal. Acetylcholine receptor antibody was normal. A computed tomography (CT) scan was arranged for further visualization of the mediastinal mass (figure 1). Carbimazole and propranolol were started for her thyrotoxicosis. Subsequently she became euthyroid and her muscle weakness disappeared. A follow-up CT scan, taken 8 months later, is shown in figure 2.
Answers

QUESTION 1
The initial CT scan showed an anterior mediastinal mass, which is a diffusely enlarged thymus. Since the normal configuration of the gland was preserved, the findings were compatible with thymic hyperplasia. There was no abnormal mediastinal lymphadenopathy.

QUESTION 2
The differential diagnosis of an anterior mediastinal mass includes a variety of clinical entities (box).

It is useful to recognise the clinical association of thymic hyperplasia with hyperthyroidism, although massive enlargement of the thymus detected radiologically has only rarely been reported. Some studies have shown that approximately 38% of patients with thyrotoxicosis have an abnormal thymus gland, and biopsy shows the formation of medullary lymphoid follicles.1 Michie et al showed that the enlarged thymus regresses upon treatment of hyperthyroidism with antithyroid agents.2 The decrease in thymic size on treatment with antithyroid drugs could be related to a direct immuno-suppressant effect of the antithyroid drugs or an indirect effect by lowering of circulating thyroid hormones. It has been reported that exogenous thyroxine treatment in animals could result in thymic enlargement.3

QUESTION 3
Recent studies have demonstrated mRNA and protein expression of the thyroid-stimulating hormone (TSH) receptor in thymic tissues of patients with Graves’ disease.4 As Graves’ disease results from the development of antibodies against the TSH receptor, the thymic TSH receptor may act as an auto-antigen responsible for the initiation or perpetuation of the autoimmune response. However, the underlying pathophysiology of thymic hyperplasia is still not well delineated.

Outcome
Bergman et al recommended deferring invasive diagnostic manoeuvres until the effects of specific antithyroid treatment could be evaluated.5 We adopted this approach with close imaging monitoring. The thymus soon returned to normal size after euthyroidism had been achieved with antithyroid drugs. Although there was no histological confirmation, thymic hyperplasia is the most probable diagnosis based on the sequence of events.

Final diagnosis
Thymic hyperplasia.

Keywords: thymic hyperplasia

References: