Thymic hyperplasia and its spontaneous resolution with treatment of Graves’ hyperthyroidism

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DESCRIPTION

A 40-year-old man was referred to the maxillofacial team for dysphagia, change in voice and weight loss. As part of investigation, MRI and CT scan of the neck were performed, which showed mild diffuse thyroid enlargement with no obvious focal lesions, and a 5.5×2.1 cm soft tissue mass in the anterior mediastinum with configuration suggesting diffuse thymic enlargement (figure 1). There were no associated sinister radiological features. Biochemistry confirmed thyrotoxicosis with thyroid stimulating hormone (TSH): 0.01 µ/L (reference value 0.3–5.0), free T4: 79 pmol/L (8–19) and free T3: >30.8 pmol/L (2.1–6.0). The anti-TSH receptor antibodies (TRAB) were elevated at 34.5 IU/L (<0.9). A diagnosis of hyperthyroidism secondary to Graves’ disease (GD) and coexisting thymic hyperplasia was made based on the clinical, biochemical and benign radiological appearance of the thymus.

A titration regimen of carbimazole was initiated with an aim to achieve and maintain euthyroidism. A conservative active observational approach was taken to monitor thymic hyperplasia before planning further invasive diagnostic investigations. A repeat CT scan after maintaining a euthyroid state for 6 months showed significant reduction in thymus size, to 2.0×1.2 cm, and attenuation (figure 2). The patient relapsed after stopping antithyroid drugs after 18 months and hence proceeded to have radioactive iodine therapy, dosed as per local hospital guidelines.

Thymic hyperplasia in GD is not uncommon, though the true incidence is not known, as the mediastinum is not routinely imaged in GD. It is thought to be an autoimmune process and there is a correlation between degree of hyperplasia and severity of thyrotoxicosis, and also the level of TRAB.1 Improvement in hyperthyroidism and TRAB with treatment of GD is associated with reduction in size of the enlarged thymus.2

Learning points

▸ Graves’ disease is a rare endocrine cause for thymic hyperplasia and generally tends to take a benign course.
▸ The exact pathogenesis behind this association is not well established. A proposed theory is the presence of thyrotropin receptors on thymic epithelial cells, which may participate in the autoimmune response leading to hyperplasia.1
▸ Simple monitoring of the enlarged thymus during treatment for hyperthyroidism may be the appropriate course of clinical action. Spontaneous resolution could be the general outcome and hence further invasive investigation or surgical intervention may not be indicated.

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REFERENCES


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