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CASE REPORT

Thyrotropin-secreting pituitary tumor presenting with congestive heart failure and good response to dopaminergic agonist cabergoline

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Hyperthyroidism is an important inducing factor in patients with atrial fibrillation, and may trigger heart failure. Thyrotropin (thyroid stimulating hormone, TSH)-secreting pituitary tumors are rare causes of hyperthyroidism. Here, we report a 66-year-old man with a pituitary TSH-secreting tumor who presented with hyperthyroidism and congestive heart failure. Endonasal trans-sphenoidal pituitary adenomectomy was performed. After the operation, the symptoms of hyperthyroidism and congestive heart failure were relieved, associated with normalization of thyroid function tests. Unfortunately, hand tremor and progressively elevated free T4 and TSH concentrations recurred 5 months after surgery. A dopaminergic agonist, cabergoline was administered and euthyroidism was restored for at least 11 months.

Introduction

Heart failure[1] is a major public health problem in Taiwan that continues to grow, with a recent prevalence rate of about 5% in the Taiwanese population.[2] The association and complex interaction between atrial fibrillation (AF) and heart failure is well understood.[3] Etiologies of AF include hypertension, ischemic heart disease, and hyperthyroidism. As a reversible cause of AF-induced heart failure, checking thyroid function to exclude hyperthyroidism is important in the assessment of patients with AF.[4]

The prevalence rate of hyperthyroidism varies from 0.5 to 2.3%.[5,6] In the elderly group, the prevalence rate increases to 6.98%, based on a recent survey in Taiwan.[7] The most common cause is Graves' disease, an autoimmune disease caused by stimulating antibodies directed

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against the thyroid stimulating hormone (TSH) receptor.[8] However, other rare conditions including TSH-secreting tumors also cause hyperthyroidism.

Initially described in 1960 by Jailer and Holub,[9] TSH-secreting pituitary tumors are found to account for about 1% of the functioning pituitary tumors and for <1% of all the causes of hyperthyroidism.[10] Patients usually present with goiter and hyperthyroidism, along with inappropriately normal or elevated TSH concentrations.[11] Such cases are easily misdiagnosed as Graves' disease, despite the fact that the treatment modality is totally different. First-line therapy of patients with TSH-secreting pituitary tumors is trans-sphenoidal resection of the tumor by which about one-third of all patients can be cured completely.[10] However, if surgery is contraindicated, pituitary radiotherapy and/or administration of somatostatin analogs or dopaminergic agonists should be considered.[12,13]

Here, we report a Taiwanese patient with pituitary microadenoma-induced goiter and hyperthyroidism. The pathology of pituitary tumor confirmed the diagnosis of TSH-secreting pituitary tumor. The symptoms and signs were relieved after trans-sphenoidal pituitary adenomectomy. However, recurrent hyperthyroidism occurred without obvious tumor regrowth in the pituitary gland based on magnetic resonance image (MRI) studies. Dopaminergic agonist was administered, the symptoms and signs were relieved and biochemical euthyroidism was restored for at least 11 months.

Case report

A 66-year-old man had a history of hypertension for 10 years, type 2 diabetes mellitus for 1 year, and atrial fibrillation for 1 year. Goiter was incidentally found, associated with elevated serum freeT4 and TSH levels in 2007 at a local hospital. None of his family had thyroid or other endocrine disorders. He suffered from dizziness, palpitation, and exertional dyspnea for 2 days, followed by sudden onset syncope on March 10, 2008. He was then sent to the emergency department of National Taiwan University Hospital. Noncontrast brain computed tomography and MRI did not show evidence of recent intracranial hemorrhage or ischemic stroke. Electrocardiography revealed atrial fibrillation with rapid ventricular response (heart rate 190/min). Chest plain film revealed cardiomegaly with bilateral pleural effusion and enlargement of bilateral hilar regions. Cardiac ultrasonography on March 11 revealed severe

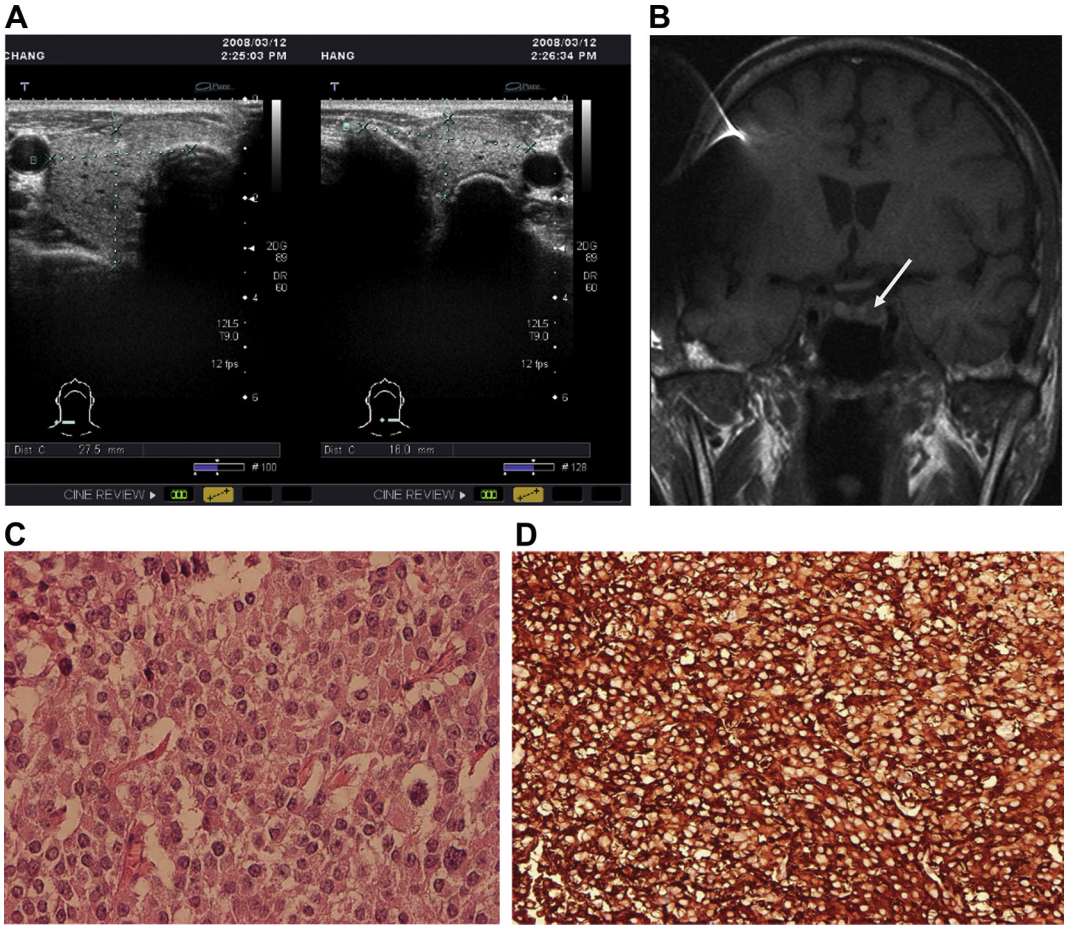

Figure 1 (A) Thyroid echo showed multinodular goiter without obvious autoimmune thyroid disease pattern. (B) Coronal T1-weighted magnetic resonance imaging with contrast of pituitary gland before operation (March 10, 2008) showed one 8 mm pituitary microadenoma. (C) Pathology of the pituitary adenoma showed hypervascularity without fibrosis, mitosis, or polymorphic nucleus (hematoxylin and eosin, 400×). (D) Immunohistochemical stain for TSH was positive in the cytoplasm. (TSH stain, 400×).