Acute Coronary Syndrome and Hyperthyroidism — Two Cases Report

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We report on two patients with hyperthyroidism presenting as acute coronary syndrome. Successful treatment of hyperthyroidism resulted in subsidence of angina and the overall prognosis is good. We suggest that routine tests of thyroid function may be warranted, especially in anginal patients without risk factors for atherosclerotic disease.

Key Words: Acute coronary syndrome • Hyperthyroidism

INTRODUCTION

The involvement of the cardiovascular system in hyperthyroidism has been recognized for many years. In aged patients, symptoms and signs of heart failure and complicating atrial fibrillation may dominate the clinical picture.[1] In young patients, acute coronary syndrome is a rare but severe and possibly life-threatening manifestation of hyperthyroidism.[2]

We report on two patients with hyperthyroidism presenting as acute coronary syndrome. In case 1, it was a precipitating factor of acute coronary syndrome, whereas in case 2, it was a causative factor.

CASE 1

A 55-year-old woman (160 cm, 56 Kg) was admitted to our intensive care unit because of sudden onset of persistent chest tightness and pain with radiation to the left arm and back, associated with palpitation, near syncope and cold sweating. She had history of hypertension and diabetes mellitus regularly controlled at our OPD. She did not smoke or drink alcohol. There was family history of hypertension and diabetes mellitus.

The blood pressure was 194/110 mmHg, heart rate 102 beats/min, respiration 22/min, and the temperature was 37°C. There was exophthalmos and diffuse goiter with bruit in the neck. The lungs were clear bilaterally. The heart rhythm was regular, and a grade 1 to 2/6 systolic murmur was heard over the left sternal border. There was no hand tremor. The remainder of the physical examination was normal. The electrocardiogram showed sinus tachycardia with left ventricular hypertrophy and ST-T changes. Chest X-ray showed borderline cardiomegaly. Echocardiogram showed concentric hypertrophy, with minimal mitral and tricuspid regurgitation and normal left ventricular systolic function. Complete blood count and urine routine were normal. Blood glucose and HbA1C were 166 mg/dL and 6.7%, respectively. Serum triglyceride and cholesterol were 123 and 268 mg/dL, respectively. Creatine kinase and CKMB were 82 and 19 IU/L. T3, T4 and TSH were 0.87 (normal: 0.87-1.78 ug/mL), 25.4 (normal: 6.09-12.23 ug/dL) and 0.02 (normal: 0.34-5.6 uIU/mL), respectively. T4 conversion into T3 may not become exaggerated, or be impaired by systemic illness or drugs. Ab-TSH receptor was 47.6 (normal = < 15%). Tc99m thyroid scan showed increased uptake, suggesting diffuse thyrotoxicosis. Thyroid sonogram showed thyroid gland enlargement with hypoheterogenous echogenicity compatible with

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Graves' disease. Tl201 myocardial perfusion SPECT showed mild myocardial ischemia in the lateral wall.

The patient was treated initially with nitroglycerin and heparin infusion, together with regular insulin, anti-hypertensive and antianginal drugs. Her blood pressure and heart rate became normal after 3 days (130/60 mmHg, 72 beatsmin^{-1}). However, chest distress persisted and intermittent morphine injection was required. After the initiation of carbimazole 20 mg three times daily, her symptoms became dramatically relieved after 10 days and she was subsequently discharged in good condition.

CASE 2

A 26-year-old man was admitted because of chest tightness and pain for two days. The patient was not obese (170 cm, 72 Kg). He denied previous and family history of hypertension, diabetes mellitus, hyperlipidemia, systemic or heart disease. He did not smoke or drink alcohol.

On physical examination, the patient appeared well. He was conscious and afebrile. The temperature was 37°C, the pulse was 84 beats/min and respiration was 20/min. Blood pressure was 126/80 mmHg. The head and neck were normal. There was no exophthalmos. The lungs were clear bilaterally. Heart rhythm was regular, and a grade 1 to 2/6 systolic murmur was heard over the left sternal border. The abdomen was soft and non-tender. The liver and spleen were not felt. There was no hand tremor. There was no peripheral edema. Neurologic examination was unrevealing. The complete blood count and blood chemistry profile were normal. The urine was normal. Radiographs of the chest were normal. The electro-cardiogram showed sinus rhythm with Q waves and ST segment elevation at II,III and aVF leads. Echo-cardiogram showed minimal mitral and tricuspid regurgitation and hypokinesis of inferior wall with normal ejection fraction. Tc99m myocardial infarct scan showed positive findings for myocardial infarction at the inferior wall. Creatine kinase and CKMB were 335 and 27 IU/L. Blood glucose was 95 mg/dL. Serum triglyceride and cholesterol were 156 and 116 mg/dL, respectively. T4 and TSH were 22.72 ug/dL and 0.04 uIU/mL, respectively. Thyroid sonogram showed a tiny cyst-like lesion (0.2 cm) in right lobe of thyroid.

The patient was treated initially with nitroglycerin and heparin infusion, together with other antianginal drugs, followed by carbimazole 10 mg three times daily after hyperthyroidism was confirmed. Two days after admission, chest pain subsided and the electrocardiogram showed sinus rhythm with Q waves and T wave inversion at II,III, and aVF leads. He was discharged the following day. One month later, the electrocardiogram showed sinus rhythm with small q waves at leads II, III, and aVF with no ST-T changes. Follow-up Tl201 myocardial perfusion SPECT one year later showed suspicious myocardial ischemia at the inferior wall, and echocardiogram showed normal ventricular systolic function with no hypokinesis at inferior wall.

DISCUSSION

The diagnosis of hyperthyroidism is now generally made early, so that fewer cardiologists have had experience with severe thyrocardiac disease. It has been known that patients with thyrotoxicosis frequently complain of anginal chest pain. The relationship and association between coronary artery, thyroid function and angina is complex and poorly understood. Reports of myocardial infarction[3] and also of unstable angina[4] with hyperthyroidism are scant in the recent literature. Because of the rarity of this condition, its pathogenicity, and its therapeutic interest, we report here another two cases so that clinicians may keep it in mind in the diagnosis of similar cases.

Myocardial infarction is unusual in thyrotoxicosis, with an incidence of 1.8%.[3] Unstable angina occurs in 20% of patients with hyperthyroidism.[4] The cause of ischemia and infarction in thyrotoxic patients is unclear. It may be due to in situ coronary thrombus,[5] to a direct metabolic effect of thyroid hormone on the myocardium,[6] be secondary to supraventricular tachycardia or atrial fibrillation,[7] or be because of coronary vasospasm.[8,9] As our two patients denied coronary angiography because of clinical improvement, we cannot delineate the exact etiologies of their acute coronary syndrome. However the clinical interest of our cases remains in the empirical fact that treatment of hyperthyroidism resulted in subsidence of angina and the overall prognosis is good. We are of the opinion that optimal treatment of acute coronary syndrome in relation to hyperthyroidism requires rapid and definite antithyroid therapy. Moreover, since cardio- vascular

complications continue to cause substantial morbidity and mortality from hyperthyroidism, early detection is mandatory. In our cases, the presentations of hyperthyroidism were not evident, as happens not infre- quently with hyperthyroidism alone. In view of the great difficulty in diagnosing underlying hyperthyroidism clinically, the routine tests of thyroid function may be warranted, especially in patients with no obvious risk factors.

We conclude that hyperthyroidism should be considered as a precipitating or causative factor of life-threatening acute coronary syndrome, particularly in patients without risk factors for atherosclerotic disease. Treatment with antithyroid and antianginal drugs results in good pro- gnosis.

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急性冠狀動脈症候群與甲狀腺功能亢進 — 二病例報告

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我們報告二位甲狀腺功能亢進併急性冠狀動脈症候群病人，服用抗甲狀腺藥物後心絞痛緩解而預後良好，我們建議在沒有冠心病危險因子的心絞痛病人，應常規檢查其甲狀腺功能。

關鍵詞：急性冠狀動脈症候群、甲狀腺功能亢進。