CASE REPORT

Thyrotoxicosis with Coronary Spasm that Required Coronary Artery Bypass Surgery

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Abstract

We describe a 47-year-old woman with severe coronary vasospasm induced by hyperthyroidism. The patient complained of anginal chest pain without specific characteristics of thyrotoxicosis. Coronary angiography was performed and revealed 90% stenosis of both the left and right coronary os. She was treated with emergent coronary artery bypass graft surgery. Postoperatively, she exhibited a comatose mentality. Severe thyrotoxicosis was indicated on thyroid function tests and thyrotoxic storm was diagnosed. Nineteen days after the surgery and following the initiation of propylthiouracil treatment, coronary angiography revealed entirely normal coronary arteries.

Key words: thyrotoxicosis, coronary vasospasm, coronary artery bypass, angina

Introduction

Thyroid hormones act on the cardiovascular system by increasing the heart rate and stroke volume (1). Thyrotoxicosis increases the risk of heart diseases such as atrial fibrillation, heart failure, angina, and myocardial infarction (1). Anginal chest pain was found to be a frequent symptom in thyrotoxicosis, occurring in 0.5-20% of patients (usually 10-12%), and almost all of the patients had previous coronary artery disease (CAD) or atherosclerotic lesions (2). A study in 1950 found that thyrotoxic patients frequently complain of anginal chest pain (3), and a relationship between thyrotoxicosis and coronary vasospasm was demonstrated in 1979 (4). A few cases were reported about their thyrotoxicosis complicated with angina or myocardial infarction induced by coronary obstructions and recovering normal coronary arteries after antithyroid drug therapy (5-9). However, we are not aware of any previous reports on coronary artery bypass graft (CABG) surgery due to thyrotoxicosis.

We present a case of hyperthyroidism with unstable angina complicated by severe coronary artery stenosis that prompted CABG surgery, developed thyrotoxic storm, and finally revealed normal coronary arteries after antithyroid medication.

Case Report

A 47-year-old woman visited the cardiology clinic because of a 2-week history of chest pain and dyspnea. The chest pain was characterized as a squeezing sensation in the retrosternal area with a normal duration of about 5 min and radiation to both shoulders. The chest pain was aggravated by exercise and initially relieved by rest, but the chest pain and dyspnea even persisted during rest over the previous few days. She had no history of hypertension, diabetes mellitus, smoking or alcohol consumption, or family history of premature CAD.

She was 156 cm tall and weighed of 53 kg, yielding a body mass index of 21.7. Her vitals were as follows: blood pressure, 140/90 mmHg; pulse rate, 96 beats/min; respiration rate, 18/min; and body temperature, 36.5°C. Physical examination of the head and neck by the cardiologist did not reveal a palpable neck mass or exophthalmos. On auscultation of the chest, her heart sounds were regular without murmurs. Her lung sounds were clear without rales. No significant findings were obtained from the examination of other systems.

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Routine laboratory tests revealed the following: white blood cell count, 6.1×10^9/L; hemoglobin, 12.1 g/dl; hematocrit, 35.0%; and platelets, 251×10^9/L. Biochemical testing showed the following: total protein, 71 g/L (reference range: 55-80); albumin, 42 g/L (reference range: 35-55); total cholesterol, 3.83 mmol/L (reference range: 3.10-5.17); low density lipoprotein, 2.59 mmol/L (reference range: <2.59); high density lipoprotein, 1.15 mmol/L (reference range: >1.55); triglyceride, 0.9 mmol/L (reference range: <1.8); alkaline phosphatase, 1.1 nkat/L (reference range: 0.5-2.0); creatinine, 79.8 μmol/L (reference range: <133). All cardiac enzymes such as myoglobin, creatinine phosphokinase, and troponin-I were within normal levels. Chest X-ray and echocardiogram were normal. Less than 1 mm of ST-segment depressions were observed at the III, aVF, and V4 to V6 leads on electrocardiogram (Fig. 1). Coronary angiograms showed 90% narrowing of both the right and left main coronary arteries (Figs. 2A, 3A).

The patient’s blood pressure during the angiography decreased to 70/40 mmHg, and she exhibited a drowsy mental state. Consequently, emergent CABG surgery was performed. After the operation, she displayed the following: pulse rate, 130-150 beats/min; body temperature, 38.0-39.0 °C; and a comatose mental state. Thyroid function tests revealed that her total thyroxine (T4) was 1280 nmol/L (reference range: 58-140), thyroid stimulating hormone (TSH) was 0.10 mU/L (reference range: 0.5-4.7), and free triiodothyronine (T3) was 30.80 pmol/L (reference range: 0.22-6.78). The endocrinologist was consulted on the second day after surgery. She had no exophthalmos, but a mild goiter and bruit on auscultation were noted on physical examination of the neck. The Burch and Wartofsky thyrotoxic crisis score was calculated to be 80 points; therefore, a thyrotoxic storm was diagnosed (<25: unlikely, 25-44: impending, >45-60: likely, and >60: highly likely) (10). She was treated with 1200 mg per day of propylthiouracil (PTU), Lugol’s solution five drops every 8 hours and 320 mg per day of propranolol by nasogastric tube after postoperative day 3. Her heart rate and body temperature stabilized, and her mental state improved. The following values were obtained on postoperative day 8: TSH, 0.06 mU/L; free T4, 46.8 pmol/L (reference range: 10.3-35.0); free T3, 9.46 pmol/L; TSH receptor antibody, 22.69 U/L (reference range: 0-1.50); antithyroglobulin antibody, 0.10 U/ml (reference range: 0-60); and antimicrosomal antibody, 148.0 U/ml (reference range: 0-60.0). A 99m-technetium thyroid scan showed mild diffuse enlargement of the thyroid gland, and the 2-h uptake was increased to 7.2% (reference range: 1.5-4.5%). Coronary angiography on postoperative day 19 showed entirely normal coronary arteries with complete resolution of the previous obstructive lesions (Figs. 2B, 3B). She was discharged with a decreased dose of PTU (450 mg/day). After 2 months, her thyroid function tests were as follows: total T3, 2.38 nmol/L (reference range: 0.92-2.78); TSH, 3.05 mU/L; and free T4, 5.8 pmol/L; her PTU was subsequently changed to methimazole. After 12 months, her thyroid function tests were as follows: free T3, 0.23 pmol/L; TSH, 1.19 mU/L; free T4, 21.26 pmol/L; and TSH receptor antibody, 0.72 U/L. We decided that her hyperthyroidism was in remission and terminated her medication. She has not experienced angina since taking the antithyroid medication nor has she had a recurrence of hyperthyroidism for 16 months after the operation.

**Discussion**

Excessive thyroid hormones are well known to induce coronary artery spasm, but several theories have been proposed for the mechanism. The main pathophysiologic mechanism was found to be an increase in the sensitivity of coronary arteries to vasoconstrictive agents such as catecholamine and 5-hydroxytryptamine under a thyrotoxic state (11, 12). In addition, coronary vasospasm was found to be influenced by increased levels of serotonin, thromboxane A2, endothelin, and prostaglandin F2α and by decreased levels of endothelin-induced vasodilator or prostacyclin (13). Moreover, thyrotoxicosis causes vasospasm through increases in the density and affinity of beta-receptors in the vasculature.
Figure 2. Left coronary angiograms: initial and after the antithyroid drug medication. The initial coronary angiography showed a 90% occlusion of the left main os in the left anterior oblique projection (A). On day 19 after the initiation of antithyroid drug medication, coronary angiography indicated normal coronary arteries with a patent previous graft (B). The arrows denote the same coronary artery lesion before and after the antithyroid medication.

Figure 3. Right coronary angiograms: initial and after antithyroid drug medication. The initial coronary angiography showed a 90% occlusion of the right coronary artery os (A). On day 19 after the initiation of antithyroid drug medication, coronary angiography indicated normal coronary arteries with a patent previous graft (B). The arrows denote the same coronary artery lesion before and after the antithyroid medication.

and decreases in alpha-receptors in animal models (14). Hyperthyroidism leads to vasoconstriction by inhibition of parasympathetic nerves that initiates vasodilation (15). Coronary vasospasm increases the risk of atherosclerotic events by accelerating thrombus formation and delaying fibrinolysis (16). Thyrotoxicosis can increase cardiac demands by producing a hypermetabolic state and promoting the development of relative myocardial ischemia due to an insufficient blood supply in response to the increased oxygen demands. Coronary vasospasm was thought to provoke coronary heart disease by a complex interaction of the above theories rather than through a single process.

CABG surgery has been considered preferential with the following conditions: a left main coronary artery lesion with unstable angina or non-ST segment elevated acute myocardial infarction, multiple coronary artery lesions, and left ventricular dysfunction (17). In the present patient, multiple indications for CABG surgery were present, including a left main coronary artery lesion with unstable angina, multiple coronary obstructions and cardiogenic shock during angiography. If the cardiologists had known that the chest pain and coronary lesions were caused by vasospasms due to thyrotoxicosis, they would have likely refrained from performing the CABG. The risk factors of CAD are well known, such as hypertension, smoking, high cholesterol, obesity, diabetes mellitus, left ventricular hypertrophy, a family history of premature coronary artery events, and estrogen therapy, but this patient did not have any of these specific risk factors.
(18). We estimated the risk of a CAD event by age, sex, total cholesterol, low density lipoprotein, hypertension, diabetes mellitus, and smoking according to the Framingham heart study (18). The estimated 10-year CAD event risks were about 1% in this patient. A noncardiac origin such as thyrotoxicosis should be considered when a patient with a low risk of CAD complains of anginal chest pain.

Anginal chest pain due to thyrotoxicosis has three characteristics that differentiate it from general angina pectoris: anginal pain occurring at rest, recent onset and rapid progression of symptoms, and abrupt cessation of angina with suppression of thyrotoxicosis (3). Coronary vasospasm caused by thyrotoxicosis has features that are similar to vasospasm caused by variant angina, with the main difference being that variant angina usually occurs at night or early in the morning, while chest pain due to vasospasm with thyrotoxicosis is not time-dependent and does occur during the day (19). The diagnosis of hyperthyroidism is difficult and is morning, while chest pain due to vasospasm with thyrotoxicosis is one of the manifestations of thyrotoxicosis, especially in patients who have a low risk of CAD events.

References


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