



Hyperthyroidism-associated coronary spasm: A case of non-ST segment elevation myocardial infarction with thyrotoxicosis

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Abstract

Hyperthyroidism is associated with many heart diseases. Thyrotoxic state has a relationship with coronary spasm. We present a case of a non-menopausal woman with hyperthyroidism who complained of chest pain. The diagnosis of coronary spasm was confirmed by coronary angiography (CAG). She is treated well with anti-thyrotoxicosis and anti-anginal medication. We recommend not use CAG as the first diagnostic choice among the patients with medication-uncontrolled hyperthyroidism and chest pain.

J Geriatr Cardiol 2011; 8: 258–259. doi: 10.3724/SP.J.1263.2011.00258

Keywords: hyperthyroidism; coronary spasm; coronary angiography; beta-blocker

1 Case report

A 39-year-old woman had recurrent chest pain for three years. The pain was located in the precordial area and lasted for several minutes. The symptom worsened in previous days. The patient was diagnosed of hyperthyroidism one year ago and stopped medication treatment by herself four months ago. Her only risk factor of coronary artery disease was obesity with a body mass index of 27.3. Her menstrual was regular. She had a sudden onset of chest pain while sleeping and was sent to the emergency room. Physical examination: blood pressure (BP): 110/60 mmHg, heart rate (HR): 88 bpm. Thyroid gland second degree enlargement, soft and without tenderness. Her CK-MB was 11 µg/L, C-TnI 1.58 µg/L. Electrocardiogram (ECG) showed wide range of ST-segment depression (Figure 1). Thyroid gland function: thyroid stimulating hormone (TSH): 0, free thyroxine 4 (FT4): 3.86 ng/dL and free thyroxine 3 (FT3): 11.85 pg/mL (normal range: FT3 1.8–4.1 pg/mL, FT4 0.81–1.89 ng/mL and TSH 0.38–4.34 µIU/mL). Emergent coronary angiography (CAG) revealed obvious ostial narrows of left main and right coronary artery, which showed

a beak sign, when the tip of catheter was inserted into the left main (Figure 2), accompanied with BP curve dampening. The spasm disappeared instantly when the catheter tip was pulled out, with BP turned to normal. No other obvious stenosis was observed. She was treated with aspirin, low molecular weight heparin (LMWH), bisoprolol, glycerin trinitrate, diltiazem and propylthiouracil (PTU). Three days later, she underwent cardiac magnetic resonance imaging (MRI) which revealed no stenoses at the ostium of coronary arteries. The patient still had paroxysmal chest pain, especially when her heart rate was over 100 bpm. She carried on intensive medical therapy, with LMWH switched to clopidogrel and bisoprolol dose doubled to 10 mg Qd. Repeated CAG was performed 10 days after the first procedure. A massive spasm was induced again at the ostium of left main. Thiamazole 5 mg Tid was added and bisoprolol was increased to 10 mg Bid. Then no chest pain occurred again during hospitalization with improved thyroid function at TSH 0.01, FT 42.22 ng/dL and FT 35.77 pg/mL. Her ECG before discharge was much better (Figure 3).

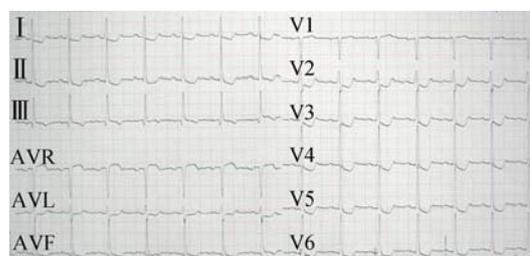


Figure 1. Wide range of ST-segment depression in electrocardiogram leads.

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Received: June 27, 2011 **Revised:** August 10, 2011

Accepted: August 17, 2011 **Published online:** December 28, 2011

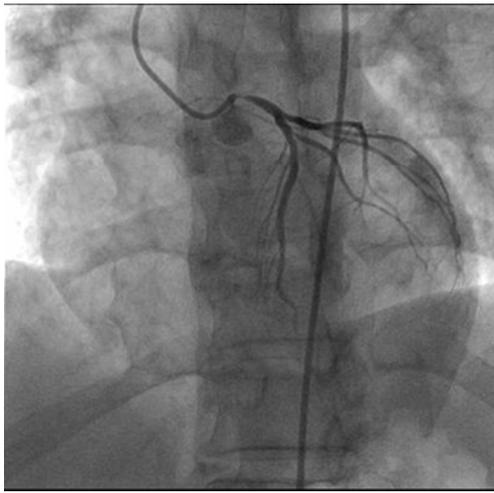


Figure 2. Vasospasm at ostium of left main.

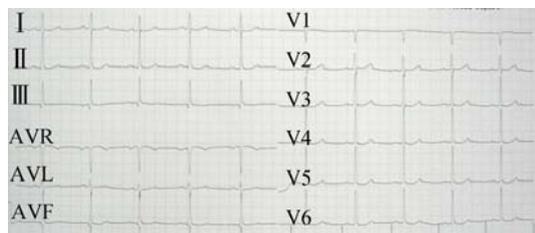


Figure 3. Electrocardiogram before discharge: no obvious abnormalities.

2 Discussion

Thyroid hormones affect cardiovascular system by increasing stroke volume and heart rate.^[1] Excessive thyroid hormones are linked to many heart diseases such as angina, myocardial infarction, arrhythmia and sudden death.^[2] The possible mechanisms of coronary occlusion with thyrotoxicosis include: significant underlying coronary atherosclerosis, direct damage to coronary artery and coronary embolization.^[3] Several hypothesis have been proposed for the mechanism of thyroid hormone and coronary artery spasm. The basic idea is that a higher sensitive state of coronary artery to vasoconstrictive agents and a decreased level of vasodilator under thyrotoxic state.^[4] In additional, coronary spasm produces a higher chance of atherosclerotic events owing to thrombus formation accelerating and fibrinolysis delaying. Thyrotoxicosis also leads to a hypermetabolic state and causes imbalance between blood supply and oxygen demand, resulting in cardiac symptoms.^[5]

There have been only a few cases reported for hyperthyroidism related coronary spasm. The diagnosis of hyperthyroidism may be overlooked or delayed in patients without typical symptoms. Lee *et al.*^[6] reported a patient who complained of chest pain without specific symptoms of thyrotoxicosis. She was treated with emergent coronary artery bypass graft surgery due to ostial stenoses of coronary

arteries on angiography. The patient had thyrotoxic storm postoperatively.

The patient we reported here had regular menstrual and no other risk factor of coronary heart disease except obesity. She had a history of hyperthyroidism and her angina might be induced by high levels of thyroid hormones. For such patient, we strongly recommend not to take CAG as the first diagnostic choice for chest pain, because the procedure manipulations, such as guidewire and catheter insertion, may not only cause a massive spasm, but also induce thyrotoxic storm due to excessive intake of iodine. Anti-thyrotoxicosis and anti-anginal medications should be used actively. Beta-block agents is strongly recommended and should be titrated gradually to the maximal doses that patients could tolerate. However, persistent and high dose use of beta-blockers in preventing thyrotoxicosis-induced heart remodeling in hyperthyroidism is still undetermined and worth further investigation. Bisoprolol is not the best choice in patients with hyperthyroidism, as it will induce alpha-receptor simultaneously while selectively inhibiting beta-receptor. Carvedilol, which can block alpha and beta receptors at the same time, may be better. Angiotensin-converting enzyme inhibitor is widely accepted as the mainstay of treatment of heart failure, but its effect to prevent thyrotoxicosis related heart remodeling is uncertain. If patients continue to have chest pain after high doses of anti-thyrotoxicosis therapy, non-invasive examination such as cardiac MRI can be used to rule out existed atherosclerosis coronary disease, especially in those who have multiple risk factors. Intravascular glyceryl trinitrate and calcium channel blocker are the best choices of antispasm. Patients with thyrotoxicosis and coronary plaques may have a higher chance of plaque rupture, inducing thrombosis events.

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