Left Main Coronary Artery Compression Syndrome with an Incomplete Atrioventricular Septal Defect Presenting as Angina Induced by Hyperthyroidism

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Abstract

We herein report the case of a 29-year-old woman who was diagnosed with incomplete atrioventricular septal defect and extrinsic compression of the left main coronary artery (LMCA) with chest pain due to postpartum thyroiditis. She exhibited chest pain with ST elevation, and coronary computed tomography showed that the LMCA was compressed between the dilated pulmonary artery and aorta. After her hyperthyroidism was treated, her chest pain resolved. Surgical repair of endocardiosis and coronary bypass grafting were performed. On the one-year follow-up visit, the dilation of the pulmonary artery and right heart was decreased. It is important to consider the possibility of compression of the LMCA in patients presenting with pulmonary hypertension and chest pain.

Key words: left main compression syndrome, congenital heart disease, postpartum thyroiditis

(Intern Med 53: 2083-2085, 2014)  
(DOI: 10.2169/internalmedicine.53.2403)

Introduction

Extrinsic left main coronary artery (LMCA) compression can be caused by pulmonary artery (PA) enlargement in the setting of pulmonary artery hypertension (PAH) (1). Due to the proximity to the LMCA, a dilated PA trunk or aneurysm can compress the LMCA, thus resulting in narrowing of the LMCA and angina pectoris, termed LMCA syndrome (2). The etiology of PAH in patients with LMCA syndrome includes idiopathic PAH, congenital heart disease [atrial septal defect (ASD), ventricular septal defect (VSD)], chronic thrombosis disease and advanced lung disease. The diagnosis of extrinsic LMCA compression can be made using either invasive coronary angiography or multidetector computed tomographic (CT) coronary angiography; in this case, we used multidetector CT. We herein report the case of a patient with angina complicated by severe LMCA stenosis resulting from PA dilation due to postpartum thyroiditis.

Case Report

A 29-year-old woman came to our hospital complaining of exertional left chest pain with a systolic murmur and abnormalities on electrocardiography. Five months before the referral to our hospital, she had delivered her first baby. Three months after the delivery, she began to feel chest pain, and a systolic murmur was detected by her family physician. At our hospital, the diagnosis of incomplete atrioventricular septal defect (AVSD) was made using echocardiography. Two days later, a syncope attack lasting for several minutes occurred following strong chest pain on defecation. The patient experienced chest pain not only on effort, but also at rest. She was therefore admitted to our hospital for an extensive examination. A clinical examination revealed a systolic murmur with fixed splitting of the second heart sound. The SpO2 was 96% and the patient’s thyroid was slightly enlarged. A chest X-ray showed mild cardiomegaly. Laboratory tests demonstrated a decreased total cholesterol level and elevation of the free T3 and T4 lev-
vealed O2 step up in the right atrium, with Qp/Qs = 2.17. The dilated. Right heart catheterization with blood sampling re-
tion. The RV outflow tract and pulmonary artery were both dilated. Right axis deviation, incomplete right bundle branch block and first degree atrioventricular block. During hospitalization, the patient experienced severe chest pain. Significant ST ele-
vation in the aVR lead, slight ST elevation in the III and V1 leads and ST depression in the other leads subsequently de-
developed. On electrocardiography, right-axis deviation was detected in association with chest pain (Fig. 1). Emergent coronary angiography showed severe stenosis of the left main coronary artery without significant stenosis of the other coronary arteries (Fig. 2A). Cardiac multidetector 256-
slice CT angiography revealed that the LMCA was thrust between the dilated pulmonary artery and the aorta (Fig. 2B), and compression of the LMCA was diagnosed. Following treatment of hyperthyroidism using a beta blocker and iodine blockade, the patient’s chest pain resolved. We decided to repair the endocardiosis and performed coronary bypass grafting of the left internal mammary artery to the left anterior descending artery. No chest symptoms recurred, and the patient’s clinical course was favorable. On the one-
year follow-up visit, the dilation of the pulmonary artery, right atrium and right ventricle was decreased and the com-
pression of the LMCA had improved (Fig. 2C).

Discussion

This case involved an AVSD patient with mild pulmonary hypertension and a dilated PA who exhibited hyperthyroid-
ism on admission. It was suspected that postpartum hyper-
thyroidism had induced the augmentation of myocardial oxygen consumption and dilated PA observed in this case due to hyper-hemodynamics. On electrocardiography with chest pain, right axis deviation was noted resulting from an increased workload of the right ventricle. We suspected that the dilated PA also induced compression of the LMCA. Therefore, a myocardial oxygen supply (compression of the LMCA)-demand (augmentation of myocardial oxygen con-
sumption) imbalance was the cause of the patient’s chest pain. Treatment of the hyperthyroidism improved this bal-

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Thyroid abnormalities after pregnancy and delivery are not rare, and the condition may either increase or decrease the thyroid function (3). This case was diagnosed as involving painless thyroiditis due to the negative titer of TRAb, elevation of the free T4 level and depression of the TSH level. The gold standard for the diagnosis of LMCA compression is coronary angiography with intravascular ultrasound (IVUS); however, a prompt diagnosis can be made using non-invasive techniques, such as coronary CT (4). As to successful treatments of LMCA compression, surgical correction of ASD and LMCA stenting has been reported (5). In this case, both surgical AVSD closure and coronary artery bypass grafting were performed, because it was unclear whether the compression had improved following closure of the AVSD. Postoperatively, the patient’s chest pain completely resolved, the PHT and PA dilation decreased and the extrinsic compression improved. In conclusion, we recognize that it is important to consider the possibility of compression of the LMCA in patients presenting with pulmonary hypertension and chest pain.

The authors state that they have no Conflict of Interest (COI).

References