Acute Myocardial Infarction Following Hormone Replacement in Hypothyroidism

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SUMMARY

Patients with hypothyroidism have an increased risk of coronary artery disease because of significant changes in lipid metabolism and arterial hypertension. We report a 67-year-old man who developed acute myocardial infarction following hormone replacement in hypothyroidism in spite of no previous cardiac symptoms and no ischemia in intravenous dipyridamole myocardial perfusion imaging. Careful examination for ischemic heart disease should be performed before hormone replacement in hypothyroidism. (Int Heart J 2007; 48: 107-111)

Key words: Acute myocardial infarction, Hypothyroidism, Hormone replacement

It has been suggested that patients with hypothyroidism have an increased risk of coronary artery disease because of significant changes in lipid metabolism and arterial hypertension. We report here a case of acute myocardial infarction following hormone replacement in hypothyroidism even though the patient had no previous cardiac symptoms and no ischemia was present in intravenous dipyridamole myocardial perfusion imaging.

CASE REPORT

A 67-year-old man was admitted to our hospital because of gait disturbance. He was a cigarette smoker and had no history of angina pectoris. He presented with facial and foot edema, and muscle weakness of the proximal lower extremities. Blood pressure was 136/86 mmHg. Chest x-rays showed no cardiomegaly or pleural effusion. A 12-lead electrocardiogram revealed sinus bradycardia and no ST-T changes. The wall motion of the left ventricle on echocardiography was normal. His thyroid function indicated primary hypothyroidism (serum free thyroxine < 0.40 ng/dL, serum free tri-iodothyronine < 1.00 pg/mL, serum thyrotropin 24.81 µIU/mL). Ultrasonography of the thyroid showed diffuse atrophy and a small calcified nodule. Antibodies to thyroglobulin and thyroid peroxidase were
elevated. He was diagnosed as having autoimmune hypothyroidism. Serum total cholesterol was 429 mg/dL, low-density lipoprotein cholesterol 338 mg/dL, and hemoglobin A1c 5.8%. To check for ischemic heart disease, perfusion imaging by 99mTc tetrofosmin with dipyridamole infusion was performed, however, no ischemia or transient left ventricular dilatation was observed (Figure 1). Levothyroxine was started from 25 µg per day, and titrated up to 50 µg per day after 2 weeks and 75 µg per day after 4 weeks. He became euthyroid and his serum total cholesterol has been 190 mg/dL without lipid-lowering therapy. On the 80th day following initiation of levothyroxine, he suffered from persistent chest pain and visited our hospital. His blood pressure was 96/56 mmHg and chest x-rays

Figure 1. Myocardial perfusion imaging by 99mTc tetrofosmin with dipyridamole infusion.

Figure 2. Emergent coronary angiography. RCA indicates right coronary artery and LCA, left coronary artery.
showed no pulmonary congestion. An electrocardiogram revealed ST elevation in V1 to V4 and an echocardiogram showed akinesis in the antero-septal wall and apex of the left ventricle. The ejection fraction of the left ventricle was determined to be 27% using the area-length method. Acute myocardial infarction was suspected and emergency coronary angiography was performed. Total occlusion of the left descending artery (mid portion) and right coronary artery (proximal portion) and severe stenosis of the left circumflex (mid portion) and high lateral branch were detected. Good collateral flow from the septal branch to the posterior descending branch, and from the conus branch to the left descending artery were present (Figure 2). We inserted an intraaortic balloon catheter and coronary artery bypass graft surgery was performed. His postoperative course was good. The peak value of creatine kinase was 1160 IU/L.

**DISCUSSION**

It has been suggested that patients with hypothyroidism have an increased risk of coronary artery disease. Several mechanisms might be involved in association with hypothyroidism and ischemic heart disease. Abnormalities of lipid metabolism (increased serum total cholesterol and low density lipoprotein cholesterol) and arterial hypertension occur with increased frequency in hypothyroidism. Thyroid hormone causes the relaxation of smooth muscle cells and inhibits platelet aggregation, and hypothyroidism induces a hypercoagulable state and increases blood viscosity. Furthermore, it has been suggested that pathological immune reactivity in autoimmune thyroiditis may be important. In this case severe hypercholesterolemia may be the primary cause of severe coronary artery disease. The results of previous health care examinations were unknown, therefore, the exact durations of the hypothyroidism and hypercholesterolemia were unclear.

This patient could not perform the exercise stress test because of muscle weakness in the lower extremities. We believe that this muscle weakness was due to muscle deconditioning after low physical activity or hypothyroid myopathy. We examined the patient for ischemic heart disease by intravenous dipyridamole myocardial perfusion imaging, but it showed no ischemia despite the presence of 3-vessel disease. The sensitivity of stress myocardial perfusion imaging is enhanced with an increasing number of lesions and 3-vessel disease is rare in patients with a negative result. In this case, pharmacologic stress by dipyridamole infusion was not combined with exercise stress, therefore, the cardiac load may have been insufficient. Other possible causes of the false negative result in this case was decreased oxygen demand on the myocardium in hypothyroidism or good collateral flow. For patients with hypothyroidism, a usual stress proto-
Control may be insufficient because of decreased oxygen demand on the myocardium, and a more aggressive stress protocol may be necessary. Coronary angiography and multislice CT were not performed because we were concerned about deterioration of the hypothyroidism due to the iodine contrast agent. Although a previous study reported that none of the cardiac asymptomatic patients with untreated hypothyroidism showed angina pectoris or signs of ischemia during dobutamine stress echocardiography (DSE), DSE might be useful for the detection of ischemia in this case.

Caution must be exercised in treating hypothyroid patients who are elderly and who may have underlying heart disease to avoid precipitating myocardial infarction or severe congestive heart failure. In this case, acute myocardial infarction occurred after the serum cholesterol level had become lower and normalized. We speculated that the possible mechanism of acute myocardial infarction following hormone replacement was the occlusion of epicardial coronary arteries by plaque rupture. Although no previous study has reported the relation between thyroid hormone and plaque rupture, relative tachycardia, increased shear stress, and augmentation of the effect of epinephrine may relate to plaque rupture following thyroid hormone replacement. In addition, other than plaque rupture, vasospasm or an increase in myocardial oxygen demand may be possible mechanisms. In this case, we used the usual protocol for hormone replacement, although more gradual replacement might be adequate for such an elderly patient.

**Conclusion:** We experienced a case of acute myocardial infarction following hormone replacement in hypothyroidism. Only a negative finding in intravenous dipyridamole myocardial perfusion imaging may not be sufficient to determine the absence of ischemic heart disease, and we must be cautious when administering thyroid hormone to patients with hypothyroidism who are elderly and have severe hypercholesterolemia.

**References**