CASE REPORT

Severe coronary vasospasm associated with hyperthyroidism causing myocardial infarction

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Abstract

A 48 year old woman presented with angina after an anterior myocardial infarction and was found to be hyperthyroid. Coronary angiography showed a stenosis of the left coronary os and a long, severe stenosis of the left anterior descending artery which was partially relieved by glyceryl trinitrate. Three months later, after radioactive iodine treatment had rendered her euthyroid, repeat coronary angiography showed entirely normal coronary arteries. This unusual case establishes an association between hyperthyroidism and coronary vasospasm resulting in myocardial infarction.

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Keywords: coronary vasospasm; hyperthyroidism; myocardial infarction

Hyperthyroidism is a rare cause of coronary artery spasm. We present an unusual case in which severe coronary spasm resulting in myocardial infarction was associated with hyperthyroidism.

Case report

A previously well 48 year old woman, with no risk factors for cardiovascular disease, presented to her local hospital with a 2 day history of severe chest pain and increasing dyspnoea, preceded by a 2 month history of exertional angina and palpitation. On direct questioning she gave a history of resting tremor, heat intolerance, and weight loss of 5 kg. Physical examination showed a blood pressure of 120/70 mm Hg, a regular pulse of 110/minute, and a prominent apical impulse. A small nodular goitre was palpable and lid lag noted. Her electrocardiogram showed a sinus tachycardia, Q waves, and ST elevation in the anterior leads and T wave inversion in the lateral leads suggesting recent anterior myocardial infarction. Short runs of atrial fibrillation were seen. Two days after admission she developed further chest pain associated with ST depression and was transferred to the University Hospital of Wales for further investigation.

Routine biochemical and haematological tests, liver function tests, and serum cholesterol and triglyceride concentrations were normal. Serum thyroid stimulating hormone was undetectable (normal 0.05-4.2 mU/l) and free thyroxine was > 100 pmol/l (normal 12-28) indicating hyperthyroidism. Anti-thyroid antibodies were not detectable and a radioactive iodine thyroid scan showed an enlarged gland with increased uptake, compatible with multinodular goitre.

Treatment with carbimazole, slow-release propranolol (160 mg twice a day), isosorbide mononitrate (60 mg once a day), and aspirin (150 mg once a day) was started. Coronary angiography (figure A) showed a significant narrowing of the left main stem and a 1 cm long, concentric 90% stenosis in the proximal segment of the left anterior descending artery with delayed distal filling. The right coronary artery was normal. After intra-coronary nitrate was given there was partial resolution of the narrowing in the anterior descending artery, but no resolution of the main stem stenosis. Left ventriculography showed antero-apical akinesia.

Coronary angiography was repeated 3 months later, when she was euthyroid (free thyroxine 28 pmol/l, free triiodothyronine 6.9 pmol/l (normal 3.0-9.0) and thyroid stimulating hormone 0.05 mU/l) and symptom free. There was complete resolution of all coronary abnormalities (figure B).

Discussion

Angina occurs in up to 20% of patients with thyrotoxicosis, usually because of coronary atheroma and increased sympathetic activity.¹ The cause of ischaemia and infarction in thyrotoxic patients with normal coronary arteries is unclear. It may be due to in situ coronary thrombosis² or to a direct metabolic effect of thyroid hormone on the myocardium³ or be secondary to supraventricular tachycardia or atrial fibrillation.⁴ More recently, coronary vasospasm has been described in patients with myocardial ischaemia and hyperthyroidism;⁵⁻⁹ its cause, however, is unknown.

This case is unique because: (a) a close association was established between coronary vasospasm induced by hyperthyroidism and

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Figure (A) Left coronary angiogram in the right anterior oblique projection (at presentation). A long concentric stenosis of the proximal left anterior descending coronary artery is seen (black arrow) and the left main stem is abnormally narrow at the catheter tip (white arrow). (B) Left coronary angiogram in the right anterior oblique projection (3 months later). There is resolution of all abnormalities. The calibre of the entire left coronary artery appears larger.

myocardial infarction, (b) severe narrowing of the left coronary os was demonstrated, indicating the potentially serious nature of this condition; and (c) repeat coronary angiograms were normal after the patient had been rendered euthyroid. In common with previous observations¹⁻⁶ our case was notable for mild clinical manifestations of thyrotoxicosis despite considerable biochemical changes. We conclude that hyperthyroidism should be considered as a cause of life-threatening myocardial ischaemia, particularly in patients without risk factors for atherosclerotic disease.

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