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Case Report

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PHEOCHROMOCYTOMA MIMICKING AN ACUTE MYOCARDIAL INFARCTION

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ABSTRACT

We are reporting a 34-year-old female who presented with diffuse chest pain, dyspnoea, headache, tremor and palpitation. Electrocardiography suggested a recent anterior myocardial infarction. Paroxysmal hypertension raised the suspicion of a pheochromocytoma. Abdominal ultrasonography and computed tomography revealed a mass in the right adrenal gland. Elevated levels of plasma and urine catecholamines supported the diagnosis of pheochromocytoma. After surgery, all antihypertensive medication was discontinued and the blood pressure returned to normal within several days. Currently, the patient is asymptomatic, has normal catecholamine levels and the electrocardiographic signs of ischaemia have resolved entirely. This case illustrates that a rare clinical entity such as pheochromocytoma should be put in the differential diagnosis of acute coronary syndrome.

KEYWORDS: Pheochromocytoma, myocardial infarction, left ventricular hypertrophy.

INTERODUCTION

Pheochromocytomas are rare catecholamine secreting neuroendocrine tumours arising from chromaffin cells in the adrenal medulla or extraadrenal paraganglia (paraganglioma). The classical triad of symptoms consists of episodic headache, palpitations and diaphoresis.[1] However, patients may not have these symptoms and several alternative clinical manifestations have been described. Most presenting symptoms of pheochromocytoma are related to the paroxysmal excess release of catecholamines into the circulation, including anxiety, headache, abdominal pain, nausea, fever, as well as hyperglycaemia and weight loss. In addition to hypertension, various other cardiovascular manifestations such as cardiomyopathy, arrhythmia, left ventricular hypertrophy and congestive heart failure have been observed in patients with a pheochromocytoma. However presentation like acute coronary syndrome is very rare.[3-5]

In this case report, we are describing a young female who presented with retrosternal pain, headache, sweating, nousea and electrocardiographic changes in favour of acute myocardial ischaemia. Finally, a pheochromocytoma was diagnosed.

CASE REPORT

A 34-year-old female was admitted to the cardiology department of our hospital with recent episodes of diffuse chest pain, dyspnoea, palpitations, tremer, headache, diaphoresis and nausea. She had a one year history of anxiety attacks and headache. With regard to cardiovascular risk factors, there was no history of smoking and she had a normal body mass index. However, she did have hyperglycemia (HbA1c 6.92%). On physical examination she had a blood pressure of 210/130 mmHg, a regular heart rate of 130 beats/min and normal heart sounds on auscultation. No additional abnormalities were found. The electrocardiogram showed a left-axis deviation, left ventricular hypertrophy, significant ST segment elevations in precordial leads V_1 to V_4 suggesting a recent anterior myocardial infarction (figure 1). Biochemical analysis demonstrated elevated levels of high sensitivity troponin T (390 µg/l, normal range 01- 100 µg /l) with normal levels of total creatine kinase (CK, 90 U/l, normal range 0 to 170 U/l).

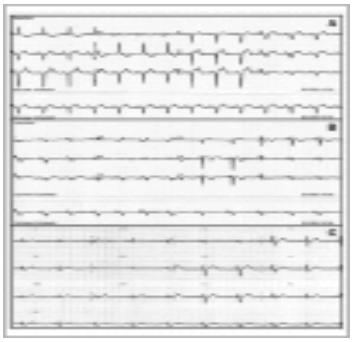

Figure: 1

She was admitted to the coronary care unit for further observation.Urgent coronary angiography was done and was normal and 2D echo did not show any signs of wall motion abnormalities except LVH. Treatment was started with a single dose of 300 mg acetylsalicylic acid followed by 100 mg once daily, carvedilol 6.5 mg twice daily, ramipril 2.5 mg once daily and atorvastatin 80 mg once daily. During the next days, the marked signs of acute ischaemia in the electrocardiogram gradually improved (figure 1B). In addition, the initially elevated troponin T returned to normal without any further increase, with normal levels of total CK and CK-MB at all times. However, the patient still experience tremors, headache and palpitations. As the electrocardiogram was highly suggestive of a stenosis of the left anterior descending coronary artery, coronary angiography was performed. It revealed normal coronary flow, no valvular abnormalities and normal left ventricle function (ejection fraction 55%).

During her stay on the coronary care unit, repeated fluctuation of blood pressure were noticed, with blood pressure values up to 140/110 mmHg and a heart rate up to 120 beats/min. In the background of the lack of findings during echocardiography, these episodes of fluctuating hypertension prompted the suspicion of a pheochromocytoma. This diagnosis was supported by the finding of pronounced urinary excretion of catecholamines (epinephrine 14,2011 nmol/24 hours, normal 250 to 1000 nmol/24 hours, norepinephrine 11,230 nmol/24 hours, normal 800 to 2800 nmol/24 hours). Abdominal ultrasound and computed tomography revealed a 11×9×6.5 cm mass in the right adrenal gland. A right adrenal pheochromocytoma was diagnosed and the α- adrenergic blocker doxazosine was started (16 mg daily) with subsequent normalisation of the blood pressure (110/73 mmHg).

The patient was transferred to the Department of surgery. In preparation for removal of the pheochromocytoma, α-blockade was restarted using increasing doses of the non-competitive α-adrenergic blocker phenoxybenzamine, up to a total dose of 90 mg/day. Intravenous infusion of normal saline (1.5 litres a day) was started to prevent orthostatic hypotension. Nevertheless, mild reflex tachycardia occurred and the β-adrenergic blocker propranolol (20 mg four times a day) was added to the medication according to the pre-surgery protocol.

Two weeks after the initial hospital admission, a right sided adrenalectomy was performed. During surgery, the haemodynamics were unremarkable, except for a brief episode of increased blood pressure during tumour manipulation, which was managed effectively. Pathological examination of the tumour confirmed the presence of a pheochromocytoma. After surgery, antihypertensive medication was discontinued and within several days her blood pressure had normalised. In addition, plasma and urinary catecholamine returned to normal levels. At follow-up, after surgery, the patient was asymptomatic, she was normoglycaemic without medication and all electrocardiographic signs of ischaemia had resolved (figure 2).

DISCUSSION

A pheochromocytoma can result in various cardiac manifestations including ventricular hypertrophy and congestive heart failure due to prolonged hypertension,[6] myocarditis and dilated cardiomyopathy due to the toxic exposure to catecholamines,[7] or occasionally, as in the present case, acute coronary ischaemia.[3-5] During a pheochromocytoma crisis, a myocardial oxygen demand-supply mismatch can occur due to an increased afterload (vasoconstriction), catecholamine indused tachycardia, and coronary vasospasms. This can precipitate myocardial ischaemia with concomitant electrocardiographic abnormalities, even in the absence of coronary atherosclerosis.

In the Netherlands 200 to 400 out of 26,000 patients presenting with symptoms and electrocardiographic changes suggestive of an acute myocardial infarction are eventually diagnosed with unrelated disorders.[8] A pheochromocytoma will only be the underlying cause in a few cases. In the present case, all signs and symptoms including electrocardiographic abnormalities and elevated troponin levels suggested a myocardial infarction. The presence of ST elevations, inverted T waves and especially Q waves in V_{3-4} strongly suggestive for irreversible myocardial damage due to ischaemia. Surprisingly, however, all electrocardiographic changes including the Q waves improved dramatically after several days and reverted completely back to normal after surgery. Although reversible ST elevations and T-wave inversions have been described in patients with a pheochromocytoma mimicking an acute myocardial infarction, to our knowledge only one other patient showing reversible Q waves has been reported to date.[3] Interestingly, similarly reversible Q waves have been observed in patients with subarachnoidal haemorrhages[9] and a recently recognised form of cardiomyopathy, known as Takotsubo cardiomyopathy.[10] In these cases cardiac ischaemia appears to be related to increased, possibly toxic, levels of catecholamines causing a transient wall-motion abnormality on echocardiography. This aberrant wall motion has been described as the 'octopus sign' or 'Takotsubo cardiomyopathy'. Such abnormalities in wall motion were not observed in our patient, however.

Several mechanisms have been suggested for the cardiac abnormalities related to excess levels of circulating catecholamines. In pheochromocytoma patients excess norepinephrine impairs both endothelium dependent as well as smooth muscle-dependent vasodilatation, possibly leading to coronary spasms.[11] Experimental evidence suggests that norepinephrine causes inflammatory exudates, which in turn possibly lead to myocardial as well as endothelial damage.[12] Interestingly, increased fibrosis of the carotid intima and

media has been found in pheochromocytoma patients. These changes occurred independently of blood pressure induced hypertrophic remodelling.[13] Another hypothesis involves neurogenically mediated stunning of the cardiac apex as observed in Takotsubo cardiomyopathy, possibly due to the increased density of adrenergic receptors in that area. In addition, this would explain the transient nature of some of the observed effects on the myocardium.[10]

In conclusion, we describe an uncommon presentation of a pheochromocytoma mimicking a myocardial infarction. In general, β-adrenoreceptor blockers are recommended in patients with acute myocardial infarction; however they can have disastrous effects in patients with a pheochromocytoma. Thus, our report illustrates the importance of including pheochromocytoma in the differential diagnosis of patients presenting with signs and symptoms of an unexpected myocardial infarction, as early treatment may prevent serious morbidity and mortality.

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