Case Report

Pheochromocytoma-Related Myocardial Damage Following Delivery

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We present the case of a 35-year-old woman whose pregnancy was complicated by the rare condition of transient pheochromocytoma-related myocardial damage. Short-duration left ventricular dysfunction was apparently caused by acute non-transmural myocardial infarction provoked by coronary artery vasospasm rather than catecholamine-induced cardiomyopathy. Forty-eight days after onset, a 50×55×35 mm tumor was excised and histologically confirmed to be a pheochromocytoma. (*Hypertens Res* 1996; 19: 291-293)

Key Words: pheochromocytoma, pregnancy, myocardial damage

Pheochromocytoma is commonly seen in young to mid-adult life, with a slight female preponderance (1). Pheochromocytoma occurs in approximately 0.1 % of the hypertensive population and may be fatal if undiagnosed or mistreated. Rarely, cardiac involvement may occur due to the direct impact of excess catecholamines secreted by the tumor. A review of the literature revealed 5 cases of pheochromocytoma and congestive heart failure in pregnant women (2-6). We report our experience with a woman who had acute heart failure following delivery in whom a pheochromocytoma was subsequently diagnosed.

Case History

A 35-year-old woman was in good health until 1990, when headaches and palpitations developed. At that time, she was given a diagnosis of migraine headache, and the highest recorded blood pressure (BP) was 136/80 mmHg. In 1993, she gave birth at 40 weeks' gestation and was given methylergometrine maleate (0.25 mg/d) as a uterine-stimulating agent. On the next day, she experienced severe left chest pain, headache, and palpitation. The BP was 218/114 mmHg. The patient was immediately transferred to Wakayama Medical College Hospital. The BP was 186/102 mmHg and the heart rate (HR) was 100 beats per min. An electrocardiogram (ECG) showed sinus tachycardia with ST-segment elevations in leads II, III, and aVF and depressions in leads I, aVL, and V₁₋₆ (Fig. 1). Plasma fraction-free catecholamines, creatine kinase (CK), and CK-MB levels were abnormally high: norepinephrine, 18.51 ng/ml (normal range, 0.07 to 0.31); epinephrine, 12.27 ng/ml (normal range,

<0.10); dopamine, 1.40 ng/ml (normal range, <0.10); CK, 879 U/l (normal range, 20 to 200); and CK-MB, 139 mg/l (normal range, 5 to 20). A chest radiograph showed widespread alveolar filtration; the size of the heart was normal. Echocardiography showed hypokinesis of the antero-septal and posterior walls and fractional shortening of 20%. She was immediately treated with nitroglycerin, but BP and HR were extremely variable. Doxazocin mesilate and atenolol were therefore also given. Eighty minutes after onset, the ECG showed only ST-segment depressions in leads V₁₋₄. Subsequently, a 24-h urine specimen revealed high catecholamine levels: norepinephrine, 4,167 µg/d (normal range, 10 to 90); epinephrine, 5,749 µg/d (normal range, <12); and dopamine, 1,432 µg/d (normal range, 100 to 700). An abdominal computed tomographic scan showed a right suprarenal mass measuring 5.5 cm in diameter (Fig. 2). The mass contained high amounts of radioiodinated metaiodobenzylguanidine (¹³¹I-MIBG), 24 h after intravenous administration. Three weeks after admission, her condition remained stable while receiving the following drugs: doxazocin mesilate, 1 mg/d; atenolol, 25 mg/d; and isosorbide dinitrate, 80 mg/d. On the 39th hospital day, she underwent cardiac catheterization, which revealed normal coronary arteries. A provocation test with methylergometrine maleate (5, 10, 20 µg into each coronary artery) induced neither coronary arterial spasm nor ECG changes similar to those seen at admission. Left ventricular angiography showed an ejection fraction of 65% with slightly reduced contraction of the apical and postero-basal walls.

On the 48th hospital day, the tumor was excised. Pathologically, the tumor was a hemorrhagic, necro-

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tic, 58-g pheochromocytoma ($50 \times 55 \times 35$ mm) with an intact capsule and tumor cells that not extend into the blood vessels. The postoperative course was uneventful. On the 12th postoperative day, she was discharged, and drug therapy was gradually

withdrawn. Three months after the operation, BP and fractionated urinary catecholamines levels had normalized, and no metastatic pheochromocytoma was noted.

Discussion

Pheochromocytoma originates from chromaffin tissue, which produces its systemic effects through the release of the large quantities of catecholamines. Pheochromocytoma-related myocardial diseases have been well characterized and include angina, acute myocardial infarction, and cardiomyopathy (1), but pheochromocytoma-related myocardial damage on delivery is extremely rare (2-6).

Myocardial ischemia may occur even in the absence of coronary artery disease. Possible causes include a catecholamine-induced increase in myocardial oxygen consumption and coronary vasospasm. This patient's condition was characterized by acute heart failure with ST-segment changes, which rapidly improved before the initiation of treatment with an α - or β -blocker. Pharmacological treatment of pheochromocytoma can improve left ventricular contraction in patients with catecholamine-induced cardiomyopathy, but complete restoration of function required 0.5 to 18 months and was most dramatic after surgical removal of the tumor (2, 4, 7-15). In such patients, catecholamine toxicity may gradually diminish after the elimination of excessive catecholamines. Therefore, the cardiac event in our patient cannot be explained by this mechanism. Although a provocative test was normal, acute non-transmural myocardial infarction following coronary vasospasm induced by catecholamines and ergometrine might have contributed to the left ventricular dysfunction.

Fig. 1. *Electrocardiogram at admission. ST-segments were elevated in leads II, III, and aVF, and depressed in leads I, aVL, and V₁₋₆.*

Fig. 2. *Location of pheochromocytoma in abdominal computed tomographic scan.*

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