Dynamic Left Ventricular Outflow Tract Obstruction in a Patient with Pheochromocytoma

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Summary
Symmetric left ventricular hypertrophy or asymmetric septal hypertrophy associated with pheochromocytoma simulating hypertrophic obstructive cardiomyopathy have been rarely reported.1-3 In this report, we present a case with pheochromocytoma that had dynamic left ventricular outflow tract obstruction without asymmetric septal hypertrophy. A surface echo revealed resolution of the systolic anterior motion of the mitral valve and all Doppler evidence of left ventricular outflow tract obstruction following removal of the tumor. Dynamic left ventricular outflow tract obstruction seen in this patient was probably due to excessive secretion of cathecolamines by the tumor. (Jpn Heart J 40: 831–835)

Key words: Outflow tract obstruction, Dynamic, Pheochromocytoma

PHEOCROMOCYTOMA is rare and usually presents as paroxysmal or sustained hypertension. Symmetric left ventricular hypertrophy or asymmetric septal hypertrophy associated with pheochromocytoma simulating hypertrophic obstructive cardiomyopathy have been rarely reported.1-3 In this report, we present a case with pheochromocytoma which showed dynamic left ventricular outflow tract obstruction without asymmetric septal hypertrophy.

Case
A 37 year old Turkish woman with a history of hypertension and headache for five years was admitted to hospital. On physical examination, her blood pressure was 180/120 mmHg and the heart rate 112 beats/min. There was a grade 3/6 systolic ejection murmur heard best at the left sternal border. On further questioning, the patient revealed that she had experienced episodes of sweating and palpitations. Chest roentgenogram was normal. ECG showed left
Figure 1.  A: Preoperative echocardiogram showing systolic anterior motion of anterior mitral leaflet (arrow).  B: Postoperative echocardiogram showing resolution of the systolic anterior motion of the anterior mitral leaflet.

<table>
<thead>
<tr>
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<th>Preoperative</th>
<th>Postoperative</th>
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<tbody>
<tr>
<td>Interventricular Septum (cm)</td>
<td>1.1</td>
<td>1.1</td>
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<tr>
<td>Posterior Wall (cm)</td>
<td>1.1</td>
<td>1.1</td>
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<tr>
<td>End-diastolic Diameter (cm)</td>
<td>3.8</td>
<td>4.0</td>
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<tr>
<td>End-systolic Diameter (cm)</td>
<td>1.9</td>
<td>2.5</td>
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<tr>
<td>Fractional Shortening (%)</td>
<td>50</td>
<td>38</td>
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Table I. Echocardiographic Findings

anterior hemiblock. The echocardiogram revealed systolic anterior motion of the mitral valve, an end-diastolic septal thickness of 1.1 cm, and an end-diastolic posterior wall thickness of 1.1 cm (Figure 1A and Table I). Doppler study from the apex showed a classic late-peaking, high velocity jet in the left ventricular outflow tract with a peak systolic gradient of 68 mmHg (Figure 2A). Mitral regurgitation was not determined. The urine vanillylmandelic acid level was signifi-
Figure 2. A: Preoperative Doppler study from the apex showing a classic late-peaking, high velocity jet in the left ventricular outflow tract with a peak systolic gradient of 68 mmHg. B: Postoperative Doppler echocardiogram showing the marked reduction of pressure gradient (B).

Figure 3. Abdominal CT scan showing the right adrenal tumor (arrows), 8 × 7 × 8 cm in diameter.
cantly elevated (112.5 mg/24 hours). The urine metanephrine level was within normal limits (303 µg/24 h) and the urine normetanephrine level was elevated (541 µg/24 h). Abdominal CT scan revealed a right adrenal tumor, 8 × 7 × 8 cm in diameter (Figure 3). After preoperative stabilization, the adrenal tumor was removed. A histologic study confirmed the features of a pheochromocytoma. Postoperatively, the blood pressure, urine vanillylmandelic acid, and normetanephrine levels returned to normal. The murmur had disappeared. Three months after the operation, a surface echo revealed resolution of the systolic anterior motion of the mitral valve (Figure 1B). All Doppler evidence of left ventricular outflow tract obstruction had resolved (Figure 2B).

**DISCUSSION**

Specific cardiac complications of pheochromocytoma include left ventricular hypertrophy, catecholamine-induced myocarditis, and dilated cardiomyopathy. In experimental animals, repeated injections of catecholamines can induce hemodynamic abnormalities suggesting left ventricular outflow tract obstruction. In the literature, a few patients with pheochromocytoma had changes simulating hypertrophic obstructive cardiomyopathy. All previously reported cases with pheochromocytoma had left ventricular outflow tract obstruction, symmetric left ventricular hypertrophy or asymmetric septal hypertrophy. In our patient, there was no asymmetric septal hypertrophy (interventricular septal thickness was 1.1 cm). In patients with pheochromocytoma, the net cardiovascular effect of the physiologic variables (increased catecholamines, relative hypovolemia, increased left ventricular afterload, and tachycardia) on the dynamics of left ventricular outflow tract obstruction would be complex, variable and unpredictable. Dynamic left ventricular outflow tract obstruction seen in this patient could be secondary to the left ventricle hypercontractile state. Removal of the tumor resulted in relief of symptoms, normalization of blood pressure, and complete resolution of the echocardiographic findings.

This report has showed that dynamic left ventricular outflow tract obstruction could be induced by pheochromocytoma. The echocardiographic features regressed completely following successful tumor removal.

**REFERENCES**


