Images in Cardiovascular Medicine

Transient Left Ventricular Apical Ballooning Complicated by a Mural Thrombus and Outflow Tract Obstruction

in a Patient with Pheochromocytoma

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A 53-year-old postmenopausal woman presented at the emergency department shortly after an episode of intense emotional stress. She was experiencing angina-like chest pain. An electrocardiogram (ECG) showed ST-segment elevation in the anterolateral and inferior leads (Fig. 1). Immediate coronary angiography revealed normal coronary arteries.

Two-dimensional echocardiography showed extensive mid-ventricular and apical akinesis and a large mural thrombus in the apex (Fig. 2) of the left ventricle (LV). Basal segments of the LV were hyperkinetic, generating a LV outflow tract (LVOT) gradient of 144 mmHg (Fig. 3). The LV ejection fraction (LVEF) was 0.32, in accordance with the Simpson rule.

The following hormone levels were highly elevated in the urine: epinephrine, 8,720 (reference range, 0–190); norepinephrine, 7,650 (reference range, 0–620); and dopamine, 8,020 (reference range, 425–2,610). The cardiac enzymes were moderately elevated (peak creatine kinase, 1,690 U/L; and peak troponin I, 2.74 μg/L). Results of serum tests for viral infections were negative. Computed tomography revealed a cystic mass of the left adrenal gland (Fig. 4).

The patient was treated with heparin (intravenous), aspirin, a diuretic, and an α-adrenergic blocker. Two weeks later, echocardiography showed normal LV regional systolic function (Fig. 5), absence of the mural thrombus, and no LVOT obstruction. The LVEF was 0.60. Histopathologic examination after surgery confirmed a diagnosis of pheochromocytoma (Fig. 6).

Comment

Transient LV apical ballooning, first described as takotsubo cardiomyopathy in 1990 by Sato and colleagues,[1] is a syndrome characterized by a reversible balloon-like asyn-

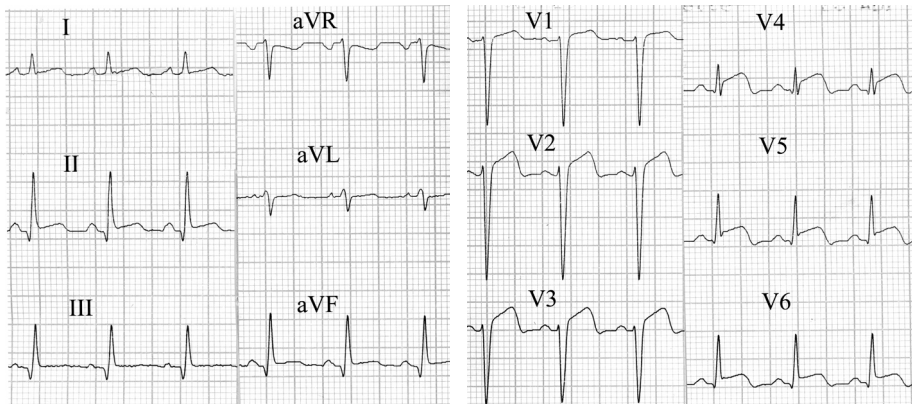

Fig. 1 *Electrocardiogram shows ST-segment elevation in the anterolateral and inferior leads.*

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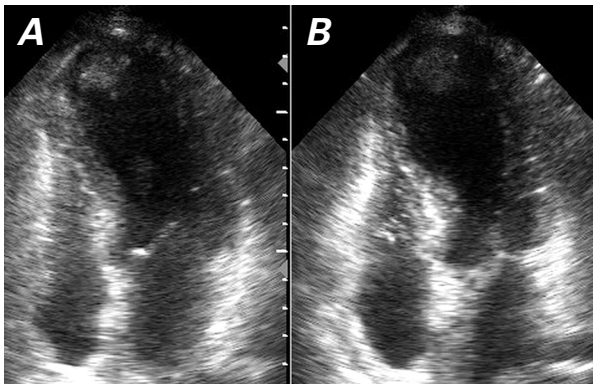

Fig. 2 *A) End-diastolic and* ***B****) end-systolic 2-dimensional echocardiograms show left mid-ventricular and apical akinesis of the left ventricle, with a large mural thrombus in the apex.*

Real-time motion image is available at www.texasheart.org/journal.

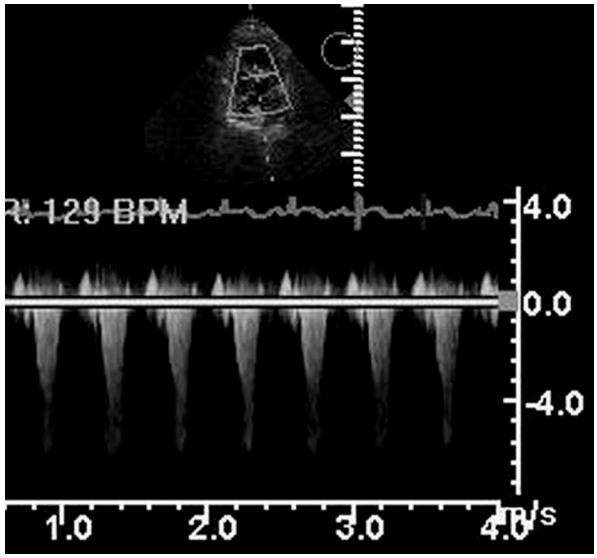

Fig. 3 *Continuous-wave Doppler echocardiogram shows a high-velocity flow of 6.0 cm/sec at the left ventricular outflow tract, with a pressure gradient of 144 mmHg.*

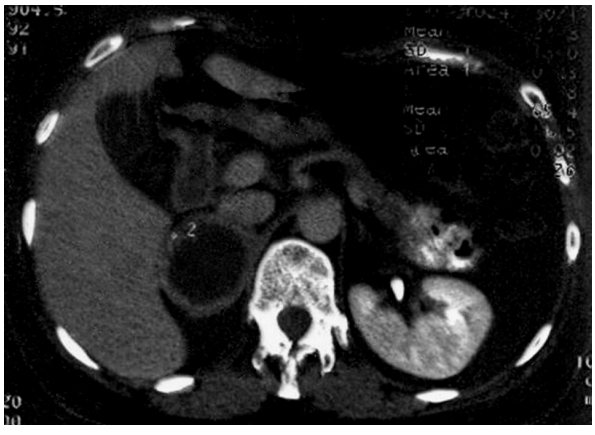

Fig. 4 *Computed tomographic scan reveals a solitary cystic mass of the left adrenal gland.*

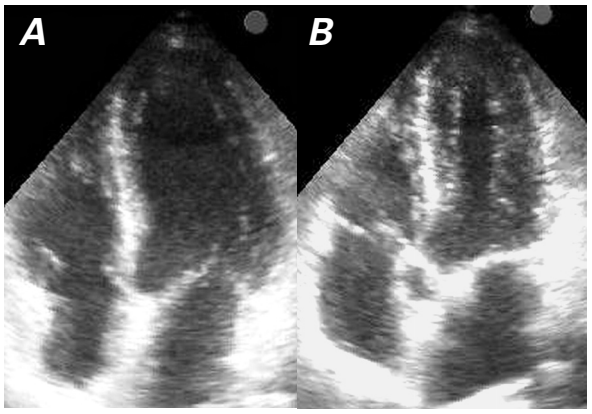

Fig. 5 *A) End-diastolic and* ***B****) end-systolic 2-dimensional echocardiograms show normalization of the left ventricular systolic function and the absence of apical thrombus.*

Real-time motion image is available at www.texasheart.org/journal.

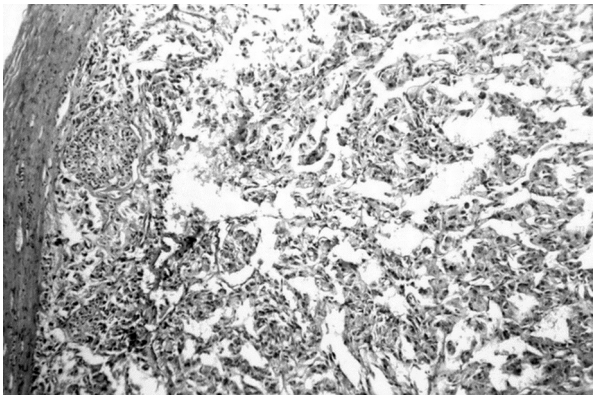

Fig. 6 *Postoperative results of histopathologic examination confirm the diagnosis of pheochromocytoma (H & E, orig. ×200).*

ergy in the apical regions of the LV and excessive contraction in the basal regions. It may account for 1% to 2% of patients who present with an acute myocardial infarction[2] and up to 1 in 30 cases of primary angioplasty in some institutions.[3] Several pathophysiologic mechanisms of transient LV apical ballooning have been proposed, such as coronary vasospasm, abnormalities in coronary microvascular function, and direct catecholamine-mediated myocardial stunning.

Transient LV apical ballooning can be categorized as primary (takotsubo cardiomyopathy, idiopathic, or stress-related), or secondary to catecholamine-related entities (takotsubo-like), including adrenal[4] or extra-adrenal[5] pheochromocytoma-induced cardiomyopathy. For clinical diagnosis of transient LV apical ballooning, all 4 criteria proposed by Prasad[2] (Mayo Clinic) should be present. For a diagnosis of primary transient LV apical ballooning, one of the criteria is the absence of a known catecholamine-mediated state.[2,5,6]

Transient LV apical ballooning syndrome has occasionally been associated with the development of LV

mural thrombus.[7] In addition, hemodynamically significant, dynamic LVOT obstruction has been observed in some patients.[8]

We present a case that encompasses all of the abovementioned features of transient LV apical ballooning syndrome. This case, to our knowledge, is unique.

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