

Transient Apical Ballooning in Hypertrophic Obstructive Cardiomyopathy

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A 79-year-old white woman with known hypertrophic obstructive cardiomyopathy (HOCM) presented with sudden-onset chest pain and no identifiable stressor. Examination suggested a new apical 3/6 systolic murmur and pulmonary edema. A 12-lead electrocardiogram showed anterolateral wall ischemia. The patient's peak troponin T level was 2.5 ng/mL. Echocardiography revealed a basal hypertrophied septum (Fig. 1) with a resting left ventricular (LV) outflow gradient of approximately 20 mmHg, severe mitral valve regurgitation, and apical akinesis. No obstruction of the coronary arteries was seen on arteriography. Simultaneous LV and aortic pressure tracing showed dynamic LV outflow tract (LVOT) obstruction as evidenced by the Brockenbrough-Braunwald-Morrow sign¹ (Fig. 2). Severe mitral regurgitation, anteroapical ballooning, and basal hyperkinesis with a low LV ejection fraction (LVEF, 0.25) were noted on ventriculography (Fig. 3). The patient was stabilized with oxygen, diuretics, and β -blockers, and she was discharged from the hospital on the 4th day. At her 2-month follow-up visit, the apical ballooning had completely resolved (LVEF, 0.65), and the dynamic LVOT obstruction was relatively less severe.

Comment

Apical ballooning syndrome is a newly described pattern of transient LV apical or mid-ventricular wall motion abnormality that is commonly associated with physiologic or psychological stress in postmenopausal women and is believed to be catecholamine-mediated.² In patients with HOCM and LVOT obstruction, only 1 previously published report of apical ballooning syndrome appears in the medical literature.³ The mechanism of transient apical ballooning is difficult to explain in a patient who had HOCM, with pre-existing dynamic LVOT obstruction and the absence of a preceding stressor. It is possible that an aggravated dynamic LVOT obstruction led to a sympathetic surge and increased wall stress.^{3,4} This, coupled with decreased coronary

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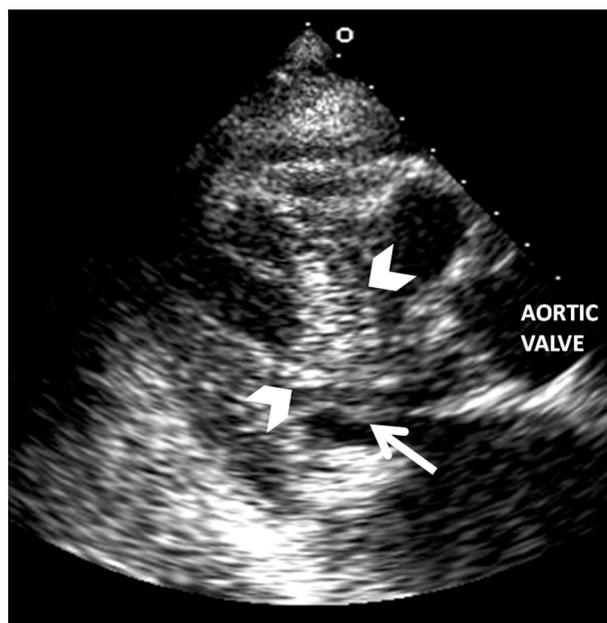


Fig. 1 Transthoracic echo-
cardiogram (parasternal long-
axis view) shows a thickened
basal septum (22-mm, arrow-
heads) and systolic anterior
motion of the mitral leaflet
(arrow).

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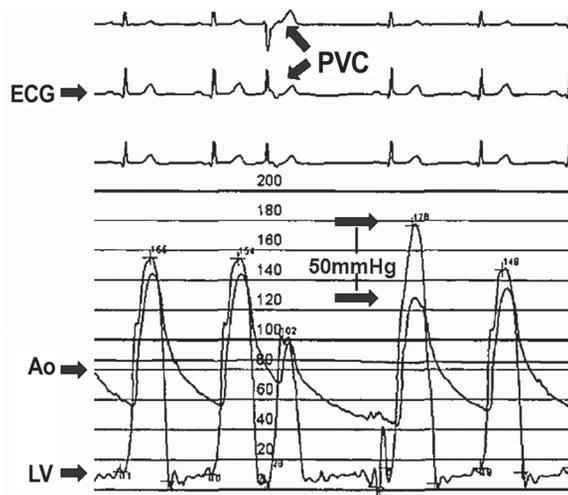


Fig. 2 Pressure tracings show a sharp rise in LV outflow gradient that follows the pause associated with PVC. A dynamic obstruction leads to a concomitant fall in aortic pressure and a disproportionate (12- to 50-mmHg) increase in gradient. This phenomenon, known as the Brockenbrough-Braunwald-Morrow sign, is part of the classical description of hypertrophic obstructive cardiomyopathy.

Ao = aorta; ECG = electrocardiogram; LV = left ventricle; PVC = premature ventricular complex

reserve and systolic coronary squeezing,^{5,6} could have caused wall-motion abnormalities and electrocardiographic changes. Treatment of such a patient is mainly supportive, and a reversal of the dysfunction is likely.

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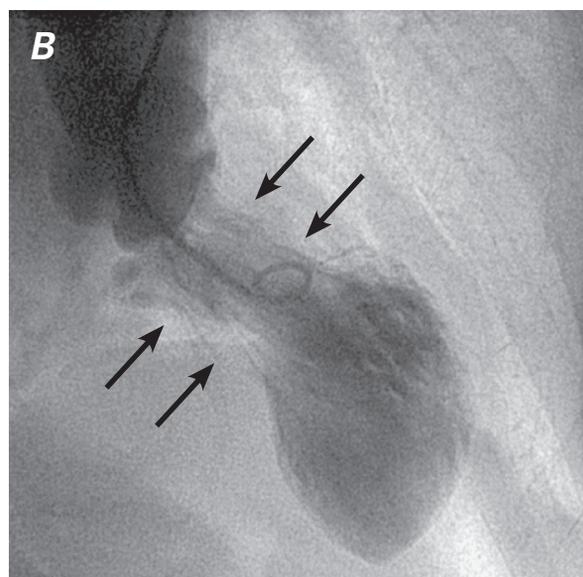
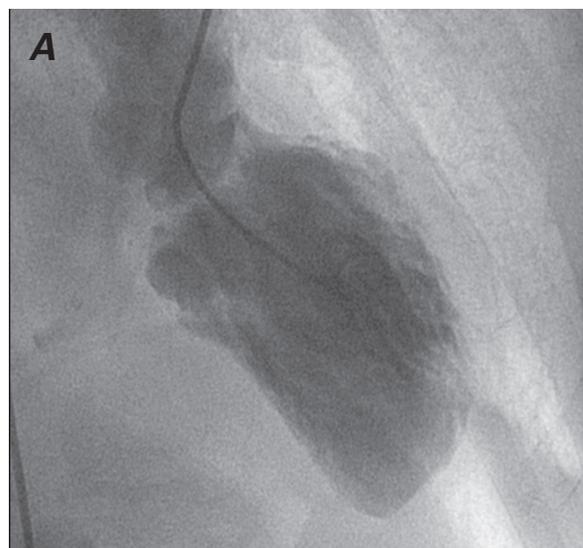


Fig. 3 End-diastolic (A) and end-systolic (B) ventriculograms (right anterior oblique view) show anteroapical akinesia or "ballooning" with basal hyperkinesis (arrows).

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