Motion of Pulmonic Valve and Constrictive Pericarditis*

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Extreme respiratory variation in the depth of the “a” wave of the pulmonic valve echo was demonstrated in a patient with constrictive pericarditis; a mechanism for this finding is offered. Disparity in systolic and diastolic ventricular function in constrictio is also useful in ruling out restrictive cardiomyopathy.

Echocardiography has contributed greatly to the diagnosis and management of pericardial effusion, yet its value in constrictive pericarditis is not well established, although several echocardiographic features have been reported1-4 (ie, thickened pericardium, flattening of left ventricular posterior wall motion during diastole, abnormal septal motion, and premature opening of the pulmonic valve). We report a case of constrictive pericarditis with echocardiographic findings which were useful in diagnosing this condition and in differentiating it from restrictive cardiomyopathy.

CASE REPORT

A 76-year-old surgeon had coronary bypass grafts to the left anterior descending artery and obtuse marginal branches of the circumflex artery. A week later, he developed pain in the chest, a pericardial rub, and atrial fibrillation, followed gradually by systemic congestion and general weakness. Treatment with prednisolone and diuretics produced no improvement. Three months after surgery, the blood pressure was 132-128/78 mm Hg, and the pulse was small but regular at 70 beats per minute. Jugular venous distention was evident, with a prominent y descent. There was slight accentuation of the pulmonic component of the second heart sound and a dull third sound. The liver was palpable, and there was mild bilateral peripheral edema. The electrocardiogram revealed sinus rhythm, a QRS axis of +90°, low voltage, and nonspecific changes in the S-T segment and T waves. A chest x-ray film was unremarkable.

An echocardiogram (Fig 1 and 2) showed a small left ventricular end-diastolic dimension (18 mm) and end-diastolic dimension (35 mm). The stroke volume was small (37 ml), but with a large ejection fraction (86 percent). Septal motion was increased (8 mm), and left ventricular posterior wall motion was flat in diastole. The epipericardium appeared thickened, but no pericardial effusion was seen. The pulmonic valve showed extreme respiratory variation in the depth of the “a” wave.

Tracings of ventricular pressure (Fig 3) showed the classic “square-root” sign of early dip and plateau. Pulmonary arterial pressure was 35/17 mm Hg, right ventricular pressure was 33/15 mm Hg, and left ventricular pressure was 110/17 mm Hg. The mean right atrial pressure was 16 mm Hg, with the “a” wave equaling 17 mm Hg. A left ventricular angiogram showed excellent ventricular contraction (ejection fraction, 90 percent). The coronary grafts were patent.

DISCUSSION

The distinctive feature of this patient with subacute constrictive pericarditis was extreme respiratory variation in the depth of the “a” wave of the pulmonic valvular echo (Fig 1). It varied from 2 mm to more than 10 mm with inspiration. The only other condition in which such an abnormally deep “a” wave can be observed is valvular pulmonic stenosis; however, clinical and hemodynamic evidence excluded pulmonic stenosis.

Extreme variation in the depth of this pulmonic “a” wave may be explained by one of the most important characteristics of constrictive pericarditis, ie, markedly reduced ventricular compliance. Because of this, small changes in ventricular diastolic volume may produce exaggerated increases in ventricular diastolic pressure.6 This may be particularly important in producing an abnormally deep pulmonic “a” wave during inspiration. During inspiration a small increase in right ventricular volume can produce a disproportionate increase in right ventricular diastolic pressure, which may be further increased by atrial contraction at the end of diastole, producing a deep pulmonic “a” wave during inspiration. Although in constrictive pericarditis, respiratory effects on cardiocirculatory flows and pressures may be greatly damped, the degree to which this occurs depends on the severity of constriction. In fact, in our patient a small increase in right ventricular dimension was observed during inspiration, slightly diverting the septum toward the left ventricle.

Patients with constrictive pericarditis often show abnormal septal motion,1-4 although our patient did not. Premature opening of the pulmonic valve, mentioned by Feigenbaum,1 also was not present. Our patient did show a thickened pericardium and flattened posterior

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wall motion during diastole (Fig 2). None of these features appear to be specific, but in conjunction with them, extreme respiratory variation of the pulmonic "a" wave may further support the echocardiographic diagnosis of constriction.

Among other conditions characterized by reduced ventricular compliance, restrictive cardiomyopathy may be difficult to exclude because of clinical resemblance and hemodynamic similarity. The most common cause of restrictive cardiomyopathy in the United States is probably amyloidosis; echographic features of amyloid heart disease have been described. In differentiating these conditions, differences in systolic and diastolic ventricular function may be most important. In restrictive cardiomyopathy, because of coexistent poor contractile function and reduced ventricular compliance, echocardiograms may demonstrate reduced ventricular functional indices and a small left ventricle with a small cardiac output, associated with the reported echocardiographic features. By contrast, in con-

**Figure 1.** Pulmonic valvular (PV) echo during expiration (EXP) and inspiration (INS). Arrows indicate huge variation in depth of pulmonic "a" wave.

**Figure 2.** Left, poor motion of aortic root (AO) and gradual closure of aortic valve (arrow), demonstrating small stroke volume. LA, Left atrium. Right, Small left ventricle with hyperdynamic wall motion, flattening of posterior wall motion during diastole, and thickened epicardium (EPI) and pericardium (PER). EN, endocardium; and IVS, interventricular septum.
Partial Atrioventricular Canal in Association with Ebstein’s Anomaly*

Echocardiographic Diagnosis and Surgical Correction

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Echocardiography was used to diagnose the presence of both Ebstein’s anomaly and partial atrioventricular canal in the case of a 20-year-old man who presented for evaluation of supraventricular tachyarrhythmias. The diagnosis was confirmed at surgery with successful surgical repair.

Ebstein’s anomaly and atrioventricular canal malformation is a rare combination of congenital cardiac lesions. Kilby et al reported such a case in a 13-year-old girl who died on the day following repair by the “atrial well technique” in 1956. The diagnosis was made at surgery, and no autopsy was performed. Lev et al have described one pathologic specimen with tetralogy of Fallot, complete atrioventricular canal and a tricuspid valve “reminiscent of Ebstein’s complex.” No case of isolated atrioventricular canal defect and Ebstein’s anomaly diagnosed under cardiopulmonary bypass or by pathologic examination has been reported.

Reported is the combination of Ebstein’s anomaly and partial atrioventricular canal, ie, ostium primum atrial septal defect with a cleft anterior mitral valve leaflet which was diagnosed by wide angle, two-dimensional echocardiography and found at operation. Although cardiac catheterization revealed partial atrioventricular canal, this procedure did not confirm the presence of Ebstein’s anomaly.

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