Implications of Normal Left Ventricular Wall Thickness in Critical Aortic Stenosis

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It is standard practice for clinicians to consider echocardiographically-measured left ventricular wall thickness when estimating the severity of aortic stenosis. Most consider the degree of wall thickness above normal limits is in proportion to ventricular hypertrophy. Employment of wall thickness information to assess aortic stenosis severity, while generally reliable, can occasionally be misleading. Two cases are presented with findings of severe, critical aortic stenosis and normal wall thickness. In each case, left ventricular contractile function was markedly impaired and the patient markedly symptomatic.

Left ventricular hypertrophy is an important adaptation to significant aortic valvular stenosis. Echocardiographers utilize the degree of hypertrophy to estimate the severity of obstruction. The most widely used approach to judging hypertrophy is to measure the thickness of the left ventricular walls. Increased left ventricular wall thickness is then used as a guide for further diagnostic and invasive evaluation. This is a report of two patients with proven hemodynamically critical aortic stenosis who, nevertheless, had normal wall thickness. The possible mechanisms and implications of these findings are discussed.

Methods

Left ventricular wall and cavity dimensions, volumes, mass, and ejection fraction were determined from quantitative twodimensional echocardiography by established methods. In brief, the methods include the use of paired orthogonal two and four chamber apical views and off-line analysis by a commercially available preprogrammed microcomputer controlled light pen digitizing system. For volume determination, the peak of the R-wave was used as a marker of end-diastole and the frame before mitral opening as a marker for end-systole. All volumes were determined by tracing the orthogonal long axis apical two and four chamber views and combining them by a modified Simpson's rule algorithm. For determination of wall thickness and mass, stop frame images of the precordial short axis (papillary muscle tip level) were analyzed at the end of passive filling with the onset of the ECG p-wave as a marker of this portion of the cardiac cycle. The endocardial and epicardial outlines were traced with the above light pen system which provided the areas subtended by the epicardium and endocardium, respectively. The radius of each area was back calculated and the difference of the radii used to compute mean short axis wall thickness. This measurement is equivalent to that made from a standard precordial M-mode of the left ventricle.

Doppler tracings were obtained from the suprasternal notch, right parasternum, apex, and left parasternum using both pulsed and continuous wave methods. For determination of peak transaortic flow velocity, continuous wave methodology was employed.

Case Reports

Case 1

An 82-year-old woman was referred to Moffitt/Long Hospital for evaluation of congestive heart failure. Her initial complaints consisted of fatigue, shortness of breath, and intermittent episodes of exertional substernal chest pain occasionally accompanied by syncope or near syncope.

On examination, blood pressure was 94/70 mm Hg, JVP was 17 cm and carotid pulses were diminished and slightly delayed. The heart was enlarged with a sustained apex impulse displaced 2 cm lateral to the midclavicular line. The aortic component of S2 was diminished and both a soft S3 and intense S4 were heard. There was a grade 3/6 late peaking systolic crescendo-decrescendo murmur best heard along the right sternal edge, but there were no diastolic murmurs.

The ECG showed sinus tachycardia, left atrial conduction defect (left atrial enlargement), old inferior and anterior myocardial infarctions, and left ventricular hypertrophy. Echocardiography revealed severe left ventricular dilation, mitral annular calcification, moderate right ventricular dilation, left atrial enlargement, and a thickened immobile aortic valve. Quantitative echocardiography revealed left ventricular end-diastolic volume 178 ml (normal mean 80 ml, 106-90 percent upper confidence limits), ejection fraction 17 percent (normal 60), left ventricular mass 192 (normal mean 99 g, 140-90 percent upper confidence limits) and wall thickness 1.0 cm (normal 1.1 or less) (Fig 1). Left ventricular segmental wall motion abnormalities were not present. Cardiac Doppler study revealed a 2-2.2 M/s transaortic valve jet which, by modified Bernoulli equation, is equivalent to a peak systolic gradient of approximately 20 mm Hg.

Cardiac catheterization demonstrated moderate pulmonary hypertension, depressed cardiac index (1.3 L/min/M), mixed venous O₂ saturation 29 percent, and a peak-to-peak systolic gradient of 26 mm Hg across the aortic valve. With a calculated mean gradient of 20, the valve area was 0.4 cm². Left ventriculography was not performed because of adequate echocardiographic data, but fluoroscopy revealed calcification of the aortic valve, mitral annulus, and coronary artery tree. Coronary angiography showed critical proximal stenosis of all three vessels. Aortic valve replacement and coronary revascularization were recommended, but the patient was reluctant and died three days after catheterization while considering this option.

Case 2

A 43-year-old male computer scientist was admitted to Moffitt/Long Hospital with the chief complaint of severe shortness of breath. In 1979, it was noted that he had a heart murmur. A 1980 echocardiogram demonstrated mild left atrial enlargement, mild to moderate left ventricular hypertrophy (LV posterior wall thickness 1.3-1.5).

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and thickened calcified bicuspid aortic valve with a mobile posterior cusp and a fused thickened anterior cusp (left + right cusps). The patient was advised to be evaluated by a cardiologist at regular intervals. He remained asymptomatic until three months prior to his June 1983 hospital admission when he noted a mild decrease in his exercise tolerance and exertional dyspnea. In the two days prior to admission, he developed class 3/4 dyspnea but denied syncope.

Physical examination showed blood pressure of 117/90 mm Hg and sinus tachycardia at 120. The jugular veins were distended to 7 cm above right atrial level. Carotid pulses were weak and delayed, and the apical impulse was displaced into the fifth intercostal space at the anterior axillary line. A summation gallop was noted, and there was a grade 2/6 late peaking, harsh, systolic crescendo-decrescendo murmur best heard along the upper sternal edge. There were bifid or inspiratory rales and a lower extremity pitting edema. The ECG showed left axis deviation, left atrial enlargement, and left ventricular hypertrophy. Two-dimensional and M-mode tracings showed left ventricular end-diastolic volume of 190 ml (normal mean 111, 35th-90th percent upper confidence bounds), ejection fraction of 30 percent (normal 60), a left ventricular mass of 193 g (normal mean 71, 94-90 percent upper confidence bounds), and a left ventricular wall thickness of 0.9 cm (normal 1.1 or less). Left ventricular segmental wall motion abnormalities were not present. Cardiac Doppler flow study demonstrated a peak transaortic velocity by continuous wave Doppler between 3 or 4 m/s and an estimated peak systolic gradient by modified Bernoulli equation of 40 to 50 mm Hg. There was also aortic insufficiency by Doppler, estimated to be mild.

Catheterization revealed moderate pulmonic hypertension, cardiac index of 2.6 L/min/m², elevated end diastolic LV pressure of 36, and a peak to peak gradient of 50 mm Hg (mean 34). The calculated valve area was 0.7 cm². Cardiac fluoroscopy revealed a calcified aortic valve, and the aortogram showed minimal aortic insufficiency. Because of adequate echocardiographic data, a left ventriculogram was not performed.

Immediately after catheterization, the patient underwent aortic valve replacement and four months after surgery was asymptomatic. Examination of the heart, during surgery, grossly excluded previous myocardial infarctions, congenital heart disease, or cardiomyopathy. Repeat echocardiogram revealed end-diastolic left ventricular volume of 165 (was 190 preoperatively), an ejection fraction of 49 percent (30 preoperatively), a left ventricular mass of 163 (was 193), and a wall thickness of 1.0.

**DISCUSSION**

A popular approach in assessing the severity of aortic stenosis has been to use indirect echocardiographic findings such as the degree and nature of aortic valve opening and the ventricular wall thickness. In most "compensated" cases of aortic stenosis, resting echocardiography reveals a thick-walled, small cavity left ventricle with normal or even "hyperdynamic" wall motion. The apparent paradox of the excellent contractile function in the face of afterload excess is explained by calculating wall stress or its analogue, the radius/thickness ratio. As these calculations show, the wall thickness is increased beyond what would be expected to normalize resting wall stress resulting in an actual decrease in wall stress. The explanation for this overshoot in hypertrophy is that the hypertrophy represents a response to conditions which occur during exercise when systolic pressure in the ventricle can be expected to rise to much higher levels than seen at rest.

The two patients presented departed from the usual picture in aortic stenosis. In the first place, both patients had documented critical aortic stenosis and normal wall thickness; wall stress at rest was therefore increased. The consequence of increased wall stress was reduced ventricular performance, which was probably due to increased afterload. A further consequence of increased resting wall stress is diminished ventricular reserve which clinically translated into exercise intolerance. The actual aortoventricular systolic gradients were relatively low in these patients, also reflecting poor contactile performance. Pathophysiological, these patients can be viewed as demonstrating an inadequate hypertrophic response to the elevated systolic wall stress of aortic stenosis. There was no evidence of other mechanisms such as previous transmural myocardial infarction, congenital heart disease, or cardiomyopathy to explain the lack of increased wall thickness. Although the first patient might also have thinned her walls as a consequence of previously undiagnosed myocardial infarction, this was not confirmed. The coronary arteries were patent during coronary angiography, and there were no segmental wall motion abnormalities observed during echocardiographic evaluation. As inferred from these two cases, the consequences of an adaptive failure of this nature are dire; both cases are presented as subacute cardiac emergencies requiring timely surgical intervention.

Our goal in presenting these cases is to alert clinicians to this malignant subgroup of patients with aortic stenosis. Their cardinal features are congestive failure, low output, reduced contractile function, modest gradients, and normal wall thickness. If such a patient is encountered, it is well to remember that Doppler echocardiography, so reliable in the majority of patients with aortic stenosis, may provide mis-
leadingly low gradient estimations; hemodynamic estimation of aortic valve area by cardiac catheterization is the most rational way to document a critically reduced aortic valve area. Once documented, expeditious valve replacement is the only recourse.

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