Parachute Accessory Anterior Mitral Valve Leaflet
Causing Left Ventricular Outflow Tract Obstruction

Report of a Case with Emphasis on the Echocardiographic Findings

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SUMMARY A case of left ventricular outflow tract obstruction caused by a parachute accessory anterior mitral valve leaflet is presented. The abnormality was detected in an asymptomatic patient by systolic murmur and thrill and was demonstrated by echocardiography, as well as by cardiac catheterization and surgery. These findings are presented. Several previously reported cases of a similar nature are reviewed. An identical case was not found in the English literature.

Subaortic Stenosis is usually associated with asymmetric hypertrophy of the ventricular septum (also called idiopathic hypertrophic subaortic stenosis, IHSS) or a subaortic diaphragm (fixed or discrete subaortic stenosis). We have documented a case in which the obstruction was caused by an accessory mitral valve leaflet which parachuted into the left ventricular outflow tract in systole.

Case Report

The patient was an asymptomatic nine-year-old boy who was seen by his physician with a complaint of enuresis. On physical examination, he was a well developed young male with no abnormalities except those related to the cardiovascular system. Peripheral pulses were of normal rate and quality. The blood pressure in the right arm supine was 86/60. There was a systolic thrill over the neck, suprasternal area, right upper sternal border, and base. The second heart sound was finely split; both components were unremarkable, and no click was present. There was a grade IV/VI harsh systolic murmur at the upper right sternal border radiating well to the base of the heart and less well to the neck and back. There was a grade III/VI systolic murmur at the lower left sternal border radiating to the apex but not a true and having a high pitched squeak. It did not vary with position. No diastolic murmurs were present.

The chest X-ray showed slight prominence of the left ventricle, borderline heart size and normal pulmonary vasculature. The ECG and vectorcardiogram showed left anterior...
hemiblock and intraventricular conduction delay. There was left ventricular hypertrophy. There was no early septal activation and the pattern resembled an anteroseptal infarct.

Echocardiogram — Preoperative

The preoperative echocardiogram showed an abnormal echo in the left ventricular outflow tract that was separate from the anterior mitral valve leaflet (fig. 1a). This echo followed the normal motion of the anterior mitral leaflet in diastole, but in early systole it abruptly separated from the true anterior mitral echo and moved anteriorly. It remained in an anterior position in apposition to the septum until the end of systole when it moved posteriorly to again follow the echo of the anterior mitral leaflet. The aortic valve tracing showed early partial closure of the noncoronary cusp (fig. 2a). The interventricular septum and the posterior left ventricular wall measured 11 mm in thickness. There was evidence of increased, symmetrical thickening toward the apex. The left ventricular cavity was not dilated, measuring 47 mm at end diastole. The interpretation of the echocardiogram was that there was an abnormal, mobile structure in the left ventricular outflow tract causing left ventricular outflow obstruction.

Cardiac Catheterization

Right and left cardiac catheterization were performed under anesthesia of nitrous oxide, oxygen and halothane. In the pulmonary capillary wedge position, there was a V wave of 30 mm and a mean pressure of 18 mm Hg. The pressure in the body of the left ventricle was 150/6-15, subvalvular area 100/2-15, and aorta 70/55. Neither isoproterenol nor propranolol had any effect upon the gradient. The contour of

![Figure 1](http://circ.ahajournals.org/)

*Figure 1. Echocardiogram through the area of the mitral valve and left ventricular outflow tract (LVOT). a) Preoperative: showing the abnormal echo in the LVOT. b) Postoperative: showing disappearance of the abnormal echo.*

![Figure 2](http://circ.ahajournals.org/)

*Figure 2. Echocardiogram through the aortic valve and left atrium. a) Preoperative: showing early closing movement of the noncoronary cusp (arrow). b) Postoperative: showing normal appearance of the aortic valve echo.*
the pressure tracings resembles that seen in IHSS. Left ventricular angiography showed a small left ventricular cavity with very hypertrophied walls. A poorly defined structure was noted in the left ventricular outflow tract which appeared to move back and forth in systole.

Surgical Findings

At surgery, no abnormality was noted through an aortotomy. The aortic valve appeared to be normal. A left ventriculotomy was performed and a membrane was found attached to the anterior surface of the mitral valve ring; the membrane had chordae tendineae that attached to the septum and papillary muscles (fig. 3). It had a cup or parachute configuration convex toward the aortic valve so that in systole it could produce marked obstruction to the left ventricular outflow tract. The membrane and chordae were excised and the mitral valve apparatus was left intact.

Postoperative Course

The patient had an uneventful postoperative course and was free of symptoms and physical findings following the surgery. The patient was seen five months postoperatively for follow-up and was found to be well, with normal physical findings.

Echocardiogram — Postoperative

The postoperative echocardiogram showed that the abnormal echo in the left ventricular outflow tract had disappeared (fig. 1b) and the early systolic closure of the non-coronary cusp of the aortic valve was no longer present (fig. 2b).

Discussion

Left ventricular outflow tract obstruction may be caused by abnormalities at the supravalvular level, valvular level (acquired or congenital aortic valvular stenosis), or by sub-

![Figure 3. Photograph of the excised specimen. Note the parachute shape. The site of attachment to the mitral valve ring is at the upper left of the picture.](image)
aortic stenosis (hypertrophic or discrete). The discrete form of subaortic stenosis is usually due to a narrow or elongated fibrous or fibromuscular band surrounding the subaortic region of the left ventricular outflow tract.2

In the present case, the left ventricular outflow tract obstruction was caused by an accessory anterior mitral valve leaflet. The leaflet was attached at the base to the mitral ring and had chordae tendineae attached to the septum and papillary muscle. Presumably, the membrane was originally flat and as a result of the hemodynamic forces in the left ventricular outflow tract which filled the underside of the membrane causing it to balloon toward the aortic valve, the parachute deformity was acquired. Two similar cases have been described in the literature. In one, two diverticula were found on the anterior mitral leaflet which, as in our case, ballooned into the left ventricular outflow tract to cause subaortic obstruction.3 The abnormality in that case was diagnosed at surgery. Another case, diagnosed at post-mortem examination, had five spherical balloon-like masses of accessory tissue 0.5–2 cm in diameter attached to the anterior mitral leaflet and chordae.4 Left ventricular outflow tract obstruction has also been described due to a tricuspid valve leaflet ballooning through a ventricular septal defect,4 and several cases of subpulmonic stenosis have been reported due to abnormal atrioventricular valvular tissue in transposition of the great arteries.5

The echocardiographic features noted in this case are similar to the features noted with other causes of subaortic stenosis in that left ventricular hypertrophy was demonstrated and early closure of a cusp of the aortic valve was seen.6 The lack of asymmetric septal hypertrophy rules out IHSS, either as a distinct entity, or as has been reported, coexisting with fixed outflow tract obstruction.7 The additional feature in this case was an abnormal echo in the left ventricular outflow tract that moved anteriorly in systole and was seen to be separate from the anterior mitral valve leaflet echo. The systolic anterior motion was presumably due to the free edge of the membrane moving anteriorly as the accessory leaflet filled with blood.

A similar case has been described echocardiographically by Popp et al.8 In that case discrete subaortic stenosis was diagnosed clinically and by catheterization. The abnormal echo seen in the left ventricle was virtually identical to the one seen in our case and the abnormality was likely to have been similar if not identical. However, the patient’s condition did not warrant surgery so there is no anatomical proof. Another example of an abnormal echo in subaortic stenosis was reported by Kronzon et al.9 In their case, the abnormal left ventricular outflow tract echo differed significantly from that in our case and that reported by Popp et al.; the obstruction was caused by the usual type of subaortic fibrous ring.

In our case, difficulty was encountered at surgery in defining the anatomy in the region of the mitral valve. Since the accessory tissue was collapsed, it was difficult to distinguish it from the true mitral valve leaflet. Echocardiography has proven extremely useful in the diagnosis and evaluation of patients with all types of left ventricular outflow tract obstruction. In this case and other specific types of subaortic stenosis, echocardiography can be extremely useful in making the diagnosis and helping to direct the surgical approach.
CORD-LIKE VEGETATION BY ECHO/Yoshikawa et al.

References


Cord-like Aortic Valve Vegetation in Bacterial Endocarditis
Demonstration by Cardiac Ultrasonography. Report of a Case

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SUMMARY The ulcerative features in a case of bacterial endocarditis in which a highly mobile, cord-like vegetation, attached to the noncoronary cusp of the aortic valve, are reported. The echocardiogram demonstrated abnormal echo patterns in the left ventricular outflow tract in diastole which was continuous with similar echoes in the aortic root. While these findings did not differ from the previously reported manifestations in cases of flailing aortic valve leaflets, the cardiac ultrasonogram distinguished this vegetative growth. Cardiac ultrasonography revealed a cord-like characteristic, showed the movement of the entire growth into the left ventricular outflow tract in diastole and into the aorta in systole, and allowed estimation of the size of the vegetative. We conclude that cardiac ultrasonography can help in differentiating aortic valve leaflet flail and vegetations in endocarditis.

EARLY RECOGNITION OF ENDOCARDITIS and its complications should lead to improved mortality and morbidity figures. Aggressive surgical intervention in selective cases may alter the catastrophic consequence of aortic valve destruction. Cardiac catheterization may be hazardous because of potential serious embolic complications. Therefore, noninvasive methods such as echocardiography and cardiac ultrasonography must be developed for safe, reliable identification of the pathologic process. This report illustrates the use of these techniques to demonstrate a highly mobile, cord-like aortic valve vegetation in a case with bacterial endocarditis.

Ultrasonic Techniques
All cardiac echograms were performed with an Aloka SSD-90 echograph using a 2.25 MHz transducer of 10 mm diameter. Records of sector M-mode scan were made with a Fukuda Denshi ECO-125 strip chart recorder.
Cardiac ultrasonograms were performed utilizing an ultrasonic contact scanner gated to an electrocardiogram. The plane of the scanning arm was adjusted to correspond to the cardiac long axis. A gating circuit triggered by the patient's electrocardiogram was used to activate the recording storage oscilloscope for a preselected brief interval during successive cardiac cycle. A permanent record was made on 35 mm film from the oscilloscope image.

Case Report
A 31-year-old woman was admitted to the Kobe Municipal Central Hospital on August 23, 1975, for progressive dyspnea. She had been hospitalized two times in that year because of apparent congestive heart failure and fever, which had been alleviated by treatment with digitalis, diuretics, and antibiotic agents. There was no known prior history of rheumatic fever or chest trauma.
Physical examination on admission revealed an agitated woman in respiratory distress, with a blood pressure of 140/40 mm Hg and a pulse rate of 90/min. The neck veins were distended and carotid pulses were bounding and had a typical collapsing quality (fig. 1). The cardiac apex was displaced to the left, and a grade 4/6 blowing diastolic murmur was heard at the left sternal border and cardiac apex. The first heart sound was diminished and a high-pitched ejection systolic murmur was heard at the upper left sternal border. The liver edge was palpable two finger breadths beneath the

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