CASE REPORTS

Echocardiographic Diagnosis of Ruptured Aortic Valve Leaflet in Bacterial Endocarditis

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SUMMARY Aortic valve rupture, secondary to aortic valve endocarditis, was diagnosed echocardiographically and closely followed preoperatively.

The ruptured left coronary cusp of the aortic valve was seen as dense irregular echoes, located anteriorly during ventricular diastole, and protruding into the left ventricular outflow tract in an otherwise normally appearing aortic valve. These echocardiographic findings, when correlated with changes in the clinical status of the patient, prompted immediate cardiac catheterization and aortic valve replacement.

Early echocardiographic detection of abnormal aortic cusps and variation from normal aortic root echo features should alert the physician to proceed to cardiac catheterization, and aortic valve replacement if necessary.

THE DIAGNOSIS OF RUPTURED AORTIC CUSPS is an elusive one for which angiography provides only tentative evidence. Often diagnosis is made only at the time of surgery or autopsy.

Unusual echocardiographic findings early in the course of the disease in a patient we studied recently led us to suspect the diagnosis of a ruptured left coronary cusp. When these were noted to correlate with clinical deterioration, immediate cardiac catheterization and aortic valve replacement was undertaken.

The echocardiographic features of aortic valve endocarditis have been described.1,2 However, very little has been written on ruptured aortic cusps or flail aortic valve.3,4 The purpose of this paper is to describe the echocardiographic features of a ruptured left coronary cusp and to emphasize the value of early echocardiographic examination.

Echocardiographic Technique

The echocardiographic recordings were made with the patient in the supine position, with a Smith-Kline ultrasonoscope equipped with a 2.25 MHz transducer focused at 7.5 cm, and recorded on a Honeywell Visicorder. Complete studies were performed using the standard echocardiographic techniques.5

Case Report

A 20-year-old white male was admitted because of "shortness of breath." He had been in good health until three weeks before his admission when he had a fever for about 4–5 days accompanied by generalized body aching. At this time the patient noticed swelling and tenderness of the distal portion of his left thumb, and shortly thereafter a minimal amount of purulent material drained. Approxiately at the same time he noticed a slight urethral discharge with a burning sensation on urination lasting for about three days. Ever since the episode of fever the patient had felt tired, had had profuse nocturnal sweating, occasional chills, anorexia, a 15 pound weight loss, and occasional dyspnea at rest. He denied orthopnea. He had a questionable history of heart murmur at age 11 which was thought to be functional. He denied any use of intravenous drugs or a history suggestive of rheumatic fever.

On physical examination at the time of admission to the hospital he appeared to be chronically ill, in no respiratory distress, and with no features suggestive of Marfan's syndrome. His temperature was 100.8° F orally. His blood pressure was 140/30 mm Hg. There were bounding arterial pulses, visible and palpable, as well as pistol shot sounds over both femoral arteries. The first heart sound was within normal limits; the second heart sound was markedly decreased in intensity. A grade IV/VI systolic ejection murmur and a loud decrescendo diastolic murmur were heard over the aortic region. Also a very distinct, loud mid-to-late, diastolic rumbling murmur was heard at the apex, thought to represent an Austin-Flint murmur. There was an S3 gallop. Splenomegaly of one finger breadth below the costal margin was felt. There were no signs of peripheral embolization. Laboratory data on admission included a hematocrit of 36.9% and a white blood count of 9,600 per cu mm with neutrophilia. The chest X-ray on admission showed borderline cardiomegaly and clear lung fields. The electrocardiogram showed sinus rhythm and left ventricular enlargement by voltage criterion only.

The echocardiogram showed abnormal diastolic echoes in the aortic valve area located eccentrically close to the anterior aortic wall, which protruded into the left ventricular outflow tract during diastole (figs. 1, 2). The anterior mitral valve leaflet exhibited fine fluttering consistent with aortic regurgitation (fig. 3).

Six blood cultures on admission showed no growth. The urethral culture was positive for N. gonorrhoea. Indirect fluorescent tests for antibodies to N. gonorrhea done on the sixth day of hospitalization were positive to a titer of 1 to

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200 (a positive titer of less than 1/100 may be normal). Because staphylococci and gonorrhea were thought to be the most likely etiologies of bacterial endocarditis, the patient was treated with large doses of oxacillin and gentamicin. His condition remained stable until about five days after admission when he started having moderate dyspnea at rest, orthopnea, a loud S3 gallop, and the chest X-ray showed evidence of acute interstitial pulmonary edema.

On the eighth day of hospitalization cardiac catheterization was performed and showed + aortic regurgitation and no mitral regurgitation. The left ventricular pressure was 113/34 mm Hg and the aortic pressure was 113/55 mm Hg indicating severe aortic regurgitation.

The following day the patient underwent open heart surgery. Inspection of the aortic valve showed three leaflets; the left and right coronary cusps were the site of heavy vegetation and clots. The left coronary cusp was partly detached. There was an aneurysm of the right sinus of Valsalva. The leaflets were excised and a Bjork-Shiley valve was inserted. The operative specimen consisted of several fragments of aortic valve leaflets revealing fibrinous deposition, necrosis, and acute inflammation by histologic examination. Central myxoid degeneration was also noted. The patient was discharged on the 44th day of hospitalization.

**Discussion**

The echocardiographic manifestation of aortic valve endocarditis has been previously described. Aortic valve vegetations of bacterial endocarditis can be identified as disorganized, shaggy echoes attached to deformed cusps. It has also been reported that the flail aortic valve can be detected by the presence of vegetations within the aortic valve area and by the presence of irregular echoes protruding into the left ventricular outflow tract.

The echocardiographic characteristics in the present case — specifically the presence of dense, shaggy echoes appearing anteriorly during diastole (fig. 1) and protruding into the left ventricular outflow tract (fig. 2) in an otherwise normally appearing aortic valve — very strongly suggested the
SUMMARY A 29-year-old female with Uhl's anomaly developed complete atrioventricular (A-V) block. His bundle studies revealed a block distal to the His bundle recording site with narrow QRS complexes. Right ventricular capture could not be obtained and despite successful left ventricular epicardial pacing, the patient died. Autopsy revealed absence of myocardium in most areas of the right ventricle and the right side of the ventricular septum with a normal tricuspid valve. Conduction system examination revealed total destruction of both bundle branches. This is the first case where bilateral bundle branch block is shown to be present in Uhl's anomaly. Narrow QRS complexes probably reflected the absence of right ventricular forces.

UHL'S DISEASE is characterized by partial or complete absence of the myocardium of the right ventricle and replacement by fibroelastic and adipose tissue, in the presence of a normal tricuspid and pulmonary valve.1-18 In previous cases the ventricular septum has been spared. The present study is the first reported case with both marked involvement of the ventricular septum and atrioventricular (A-V) block. In addition, electrophysiologic studies correlated well with serial section findings in the conduction system.

Report of Case

The patient was a 29-year-old female admitted to Saint Francis Hospital, Lynwood, California on September 17, 1975 following a syncopal attack. The patient first became symptomatic nine years prior to admission during a pregnancy, at which time she noticed dyspnea on exertion and ankle edema. One year prior to admission, her symptoms increased markedly with episodes of breathlessness on mild exertion, and weakness and diaphoresis associated with episodes of rapid heart rate. An X-ray taken at this time revealed cardiomegaly; cardiac catheterization revealed normal pulmonary artery pressure. The cardiac output was 2.5 L/min and the cardiac index was 1.25 L/min/m². Angiocardiography revealed moderate tricuspid regurgitation and
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