M-Mode and Cross-Sectional Echocardiographic Features of Flail Pulmonary Valve

A Case Report

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SUMMARY

In a patient with patent ductus arteriosus, a pulmonary regurgitant murmur developed after the treatment of endocarditis. M-mode echocardiograms showed abnormal echo mass, characterized by fine diastolic fluttering, in the right ventricular outflow tract during diastole which were continuous with anterior pulmonary leaflet and thick pedunculated lesions attached to the anterior pulmonary leaflet were seen to be moving freely by cross-sectional echocardiography. At autopsy the destruction of the pulmonary valve was confirmed.

This study suggests that diagnosis of flail pulmonary valve is feasible by M-mode and cross-sectional echocardiography.

Additional Indexing Words:

Cross-sectional echocardiography Infective endocarditis Flail pulmonary valve

ALTHOUGH there have been an increased number of reports which indicated an increased frequency of tricuspid valve involvement by infective endocarditis recently,¹⁾⁻³⁾ pulmonary valve involvement has been thought to be uncommon. M-mode and cross sectional echocardiographic features of infective endocarditis involving the aortic, mitral, and tricuspid valves are well known, however, there have been a few reports dealing with pulmonary valve involvement by endocarditis.^{4),5)}

CASE REPORT

A 40-year-old male was admitted to the Sakakibara Heart Institute in March, 1978, because of dyspnea and jaundice. In childhood heart murmur was detect-

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ed, but he had enjoyed a relatively healthy life until August 1970, when he noticed intermittent fever associated with progressive weakness. At another hospital blood cultures grew *Streptococcus viridans*, and he had treatment of infective endocarditis and responded to massive dosage of some antibiotics which had been continued for the following 6 months.

After that he was transferred to the Heart Institute of Japan for cardiac catheterization. Cardiac catheterization demonstrated a patent ductus arteriosus with a pulmonary to systemic flow ratio of 2.9:1 and pulmonary arterial pressure was elevated to 75/20 mmHg. A pulmonary arteriogram demonstrated massive pulmonary regurgitation as shown in Fig. 1. At that time corrective surgery was refused. In the 4 weeks before his present admission, he noticed progressive weakness, shortness of breath, and jaundice.

Significant physical findings on admission included an ill appearing man in acute respiratory distress. There was marked vein and liver distension with massive pitting edema over both the presacral area and legs. The blood pressure was 150/40 mmHg and the heart rate was 120 beats/min and regular. The point of maximal impulse was felt in the 6 left intercostal space on the anterior axillary line. The first sound was diminished and a grade 3/6 blowing (low frequency) diastolic murmur, increased on inspiration, was noted in the pulmonary valve area and a grade 2/6 soft regurgitant murmur was also audible at the left sternal border and apex. Crepitant rales were heard at the lung bases bilaterally.

Pertinent laboratory data on admission included an electrocardiogram revealing normal sinus rhythm and left ventricular hypertrophy. Chest X-ray films

Fig. 1. Angiogram. Although pulmonary arteriogram demonstrated massive pulmonary regurgitation, the specific details of pulmonary valve anatomy were obscured by the dense simultaneous opacification of both the pulmonary artery and right ventricle. PA=pulmonary artery; RV=right ventricle.

Fig. 2. M-mode echocardiogram of pulmonary valve taken on admission. Posterior pulmonary leaflet showed mid-systolic closure with normal a-dip, but anterior pulmonary leaflet disclosed coarse diastolic fluttering (arrow). Multiple echoes were observed in diastole between anterior and posterior pulmonary leaflets.

Fig. 3. M-mode scan from right ventricular outflow tract to pulmonary artery. Abnormal mass echoes (arrow) were recorded in the right ventricular outflow tract during diastole which were continuous with fluttering anterior pulmonary leaflet. A cross-sectional scan later showed it to be a portion of a destructed pulmonary leaflet. PA=pulmonary artery; AO=aorta; LA=left atrium; RVOT=right ventricular outflow tract.

Fig. 4. A diastolic stop-frame image of the pulmonary valve in the long axis view with a schematic diagram. Note the pedunculated lesion (arrow), attached to the anterior pulmonary leaflet, moving back into the right ventricular outflow tract during diastole. APL=anterior pulmonary leaflet; PPL=posterior pulmonary leaflet; RVOT=right ventricular outflow tract.

Fig. 5. Autopsy photograph of the pulmonary artery and valve from above showing destroyed anterior pulmonary leaflet (arrow). APL=anterior pulmonary leaflet.

showed generalized cardiomegaly and pulmonary congestion. The white blood cell count was 18,000/mm³ with a marked shift to the left and GOT and GPT levated.

M-mode echocardiograms taken on admission showed mild both right and left ventricular dilatation, and the thick posterior leaflet of the pulmonary valve showed midsystolic closure with normal a-wave in spite of pulmonary hypertension and in diastole anterior leaflet disclosed coarse fluttering, and multiple linear echoes were observed between anterior and posterior pulmonary leaflets (Fig. 2). In addition M-mode scan from right ventricular outflow tract to pulmonary artery demonstrated an abnormal echo mass, characterized by fine fluttering, in the right ventricular outflow tract during diastole which were continuous with the fluttering anterior pulmonary valve leaflet (Fig. 3). Cross-sectional echocardiography disclosed abnormal mass attached to the anterior pulmonary leaflet which appeared to enter the pulmonary artery completely in systole and moved back into the right ventricular outflow tract in diastole (Fig. 4). Two weeks later he died because of hepatic coma.

At autopsy there was a gross incompetence of the pulmonary valve with increased thickness of all leaflets, and the anterior pulmonary leaflet was partially destroyed with pediculated tissues (Fig. 5, arrow). Perforation of the right and non-coronary cusp was also observed.

DISCUSSION

Successful management of infective endocarditis depends on the early recognition and the prompt institution of therapy.

Several recent reports have indicated that cross-sectional echocardiography has been more useful in detecting the vegetations on the leaflet and complications of endocarditis such as flail valve and ruptured chordae tendineae.

Recently there have been some reports that shaggy and extra thick echoes on the pulmonary valve are helpful in diagnosing the pulmonary valve vegetations in patients with endocarditis as in other valves.^{4),5)} Pulmonary valve tumor and calcification might produce abnormal echoes in the valve, and therefore these lesions should be considered when abnormal echoes are observed in the pulmonary valve, especially in view of the negative blood culture.

Flail pulmonary valve can also be diagnosed when abnormal fluttering echoes are present during diastole in the pulmonic valve and right ventricular outflow tract as described by Wray on the aortic valve.⁶⁾

In this case M-mode and cross-sectional echocardiography provide anatomic informations in two different ways. The rapid fluttering of the diseased anterior pulmonary leaflet and abnormal echoes in the right ventricular outflow tract seen in Figs. 2 and 3 suggested the presence of a vegetative lesion or disruption of leaflet, or both. Although it was difficult to appreciate such

subtle fluttering of the diseased valve leaflet with cross-sectional echocardiography, it provided spatial anatomic information that was specific in characterizing the nature of the disordered valve anatomy.

Although cardiac catheterization and angiogram of the pulmonary artery permitted an assessment of the patient's hemodynamic state and the severity of the pulmonary regurgitation, the flail nature of the pulmonary valve was more fully appreciated with cross-sectional echocardiography.

We concluded that M-mode and cross-sectional echocardiography, in combination with the clinical condition of the patient, proved useful in the delineation of the pulmonic valve changes in infective endocarditis.

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