Pulmonary Valve Cusp Rupture Due to Cardiac Catheterization: A Case Report

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We present a patient with iatrogenic rupture of a right pulmonary cusp. During the cardiac catheterization, iatrogenic rupture of a pulmonary valve is a rare case. We could not find any case in the literature. Preoperatively ventricular septal defect and right ventricular infundibular stenosis were documented echocardiographically and at cardiac catheterization. At operation there was a ventricular septal defect with right ventricular outflow tract obstruction. The pulmonic annulus was found normal in size and other two cusps were normal.

Key words: Ventricular septal defect, Ventricular outflow obstruction, right pulmonary valve abnormalities.

Erhenhaft(1), and Faruqui, Silverman(2) reported two cases of isolated severe pulmonary valvarular destruction probably as a result of endocarditis. Without endocarditis pulmonary valve cups rupture or destruction must be extremely rare. There has been no information regarding the iatrogenic rupture of pulmonary valve in the literature.

CASE REPORT
A 14-year-old was admitted with a complaint of palpitation and exertional dyspnea. On physical examination a grade 4/6 systolic murmur were heard along the left sternal border. Murmur was the loudest at the 2nd left intercostal space.

Electrocardiogram showed sinus rhythm and right ventricular hypertrophy. Prominence of pulmonary conus and plethoric lung was observed on his telecardiography. Two-Dimensional echocardiography revealed ventricular septal defect, pulmonary valvular insufficiency and right ventricular outflow tract obstruction (Fig. 1). The mean transvalvular gradient was 49 mm with an obvious hypertrophied right ventricle. Cardiac catheterisation confirmed the presence of a membranous type ventricular septal defect, pulmonary valvular insufficiency and right ventricular outflow tract obstruction. Pressures were 27/6 mm Hg in the main pulmonary artery (mean, 16), 78/0-8 mm Hg in the right ventricle, 3 mm Hg in the right and 8 mm Hg in the left atriums.

Operation was performed under cardiopulmonary bypass, moderate hypothermia, potassium cardioplegic myocardial preservation and topical cooling with iced saline. A longitudinal incision was made in the outflow tract of the right ventricle after resection of the fibrotic obstructive bands and the pulmonary valve was visualised. The pulmonary annulus was normal in size. Anterior and left cusp of the pulmonary valve were normal, but the right cusp could not be seen from the right ventriculotomy. Subsequently a vertical pulmonary arteriotomy was made. A piece of fibrous tissue was floating in the lumen. This was deformed and detached from the right cusp annulus of the pulmonary valve. It was only anchored at the side of the commissure of the anterior cusp. A right cusp was created by a semicircle shaped pericardial gusset and was sewn to the annulus by continuous 6-0 prolene. The ventricular septal defect was closed by a
Teflon patch with interrupted horizontal mattress sutures. Thereafter, the pulmonary artery and ventriculotomy were closed by continuous sutures.

Routine histologic section of the resected cusp showed fibrotic and calcific valvular tissue.

Postoperative period was uneventful and the patient was discharged ten days after the operation. Two months after the operation he was completely asymptomatic. He is now without medication.

**COMMENT**

A small proportion of patients with large ventricular septal defect and large left-to-right shunt develops in time infundibular pulmonary arteriosclerosis. Right ventricular outflow tract obstruction can complicate the natural history of patients with isolated ventricular septal defect. In this case despite the rupture of the cusp, probably because of subvalvular stenosis there was no clinical and laboratory signs of pulmonary valve regurgitation. Experimentally in dogs, the partial excision of cusp of the normal pulmonic valve has produced no significant hemodynamic changes at rest but some changes after induced anoxia.

However, there was no complication during cardiac catheterization. On the other hand the patient did not change clinically after the cardiac catheterization. It is difficult to explain how the cusp of the pulmonary valve was ruptured. There are two possibilities: Moreover, it is almost impossible to tear off a strong fibrous leaflet tissue from the annulus by a straight catheter.

In the histopathologic examination of ruptured cusp, no sign of endocarditis was observed. Isolated pulmonic endocarditis is very unusual and seen most commonly in addicts. While in this case, the only remaining explanation for the reason of the rup-
turing of this pulmonic cusp is probably during the cardiac catheterization.

Another peculiar situation was that in spite of ruptured cusp how could physical and laboratory examination reveal pulmonary valvular stenosis. We think that the ruptured cusp probably passed to the sub-valvular area from time to time and obstructed the valve.

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