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## **Case Report**

# Balloon pulmonary valvotomy — Not just a simple balloon dilatation



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#### ABSTRACT

Balloon pulmonary valvotomy is the preferred mode of treatment in patients with isolated pulmonary valvar stenosis and has shown good long term results. It is generally considered a safe procedure with few complications. There have been however, case reports of potentially fatal acute severe pulmonary edema occurring after the procedure in some patients. The cause of this complication and its pathophysiology is still not clear. Its occurrence is also infrequent with less than 5 cases reported till now. We report a case of pulmonary valvar stenosis which developed acute severe refractory pulmonary edema immediately after balloon pulmonary valvotomy.

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#### 1. Introduction

Isolated pulmonary valve stenosis is not uncommon comprising 7.5–9% of all congenital heart diseases. Balloon pulmonary valvotomy (BPV) is presently the treatment of choice for isolated pulmonary valve stenosis and has virtually replaced surgery as a treatment mode for this condition. It is a safe and effective procedure with very few complications and good long term results. While generally considered as safe, there are few case reports of acute severe pulmonary edema occurring immediately after the procedure which has at times

proved to be fatal.<sup>4</sup> We report a case where the patient developed acute severe pulmonary edema after BPV.

### 2. Case report

A 19-year-old girl was admitted with history of fatigue and dyspnea on exertion of 7–8 years duration. She was told to have some cardiac disorder at a peripheral medical centre but had never undergone a complete evaluation for the same. A complete evaluation done at our hospital revealed that she had severe pulmonary valvar stenosis. There was associated

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severe tricuspid regurgitation and the estimated RV pressure was 160 mmHg. The left side chambers and left ventricular function was normal. There were no associated shunt lesions. Her blood investigations did not reveal any significant abnormality. She was advised to undergo a balloon pulmonary valvotomy.

The oxygen saturation measured at rest was 88%. Right femoral vein and femoral artery access was taken. The mean right atrial pressure was 20 mmHg, the right ventricular systolic pressure was 183 mmHg with an end diastolic pressure of 20 mmHg. The left ventricular systolic pressure was 136 mmHg with an end diastolic pressure of 12 mmHg. Right ventriculogram was done with a 6F Berman catheter which confirmed the presence of isolated pulmonary valvar stenosis. The annulus measured 14 mm and we decided to use an 18 mm balloon for BPV. The pulmonary valve was crossed with a 150 cm angled tip 035" hydrophilic wire (Terumo corporation, Tokyo, Japan) and this was exchanged for a 260 cm 035" Amplatz extra stiff wire (Cook Inc, USA) using a 6F Cournand catheter. The mean pulmonary artery (PA) pressure was 12 mmHg as measured in the main pulmonary artery trunk. An 18 imes 40 mm Atlas PTA balloon (BARD peripheral vascular Inc, AZ, USA) was then positioned across the pulmonary valve and a balloon dilatation was done. Waist produced by stenotic valve was obliterated and valve opened well (Fig. 1). Post procedure the RV systolic pressure decreased to 90 mmHg and the PA pressure increased to 77/30 mmHg with a mean pressure of 56 mmHg (Fig. 2). Repeat right ventriculogram done showed that the valve had opened well with mild pulmonary insufficiency. The PA pressures however continued to remain high even after 10 min of the procedure. The patient remained hemodynamically stable with the oxygen saturation increasing to 100%.

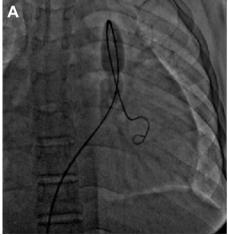
The patient was shifted to the post cath recovery room for monitoring and close observation. 20 min after the procedure, the patient complained of sudden onset of breathlessness with associated cough. The oxygen saturation started dropping and within 5 min dropped to 55%. The patient was visibly cyanosed and chest auscultation revealed B/L diffuse coarse crepitations with wheezes. At this stage the patient started having profuse hemoptysis with blood stained frothy sputum. She was given intravenous (IV) Frusemide and was

immediately intubated and put on ventilator support. The blood pressure remained normal. An echo was done immediately which showed a peak PA gradient of 30 mmHg. Other findings by echo were normal as noted in the pre BPV echo. The patient continued to have profuse bloody frothy sputum from the endotracheal tube and the PEEP in the ventilator setting had to be increased to 18 mmHg to maintain a saturation of 90% with 100% oxygen. The blood pressure continued to be normal. 6 h after the procedure the patient started developing hypotension. IV Ionotropes were started and she was eventually on the highest possible dose of Dopamine and Noradrenaline infusion. Her blood pressure however kept falling and the hourly urine output fell down to 30 ml per hour over the next 3 hours. Nine hour after the procedure the patient had a cardiac arrest and despite maximal efforts for cardiopulmonary resuscitation, she could not be revived.

#### 3. Discussion

BPV has replaced surgery as the treatment mode for isolated pulmonary valve stenosis. It has been performed in all age groups with very good long term results. It is a safe procedure with a mortality rate of <0.25% and very few major complications.<sup>1</sup> This case reports a very rare complication of BPV which unfortunately proved fatal for this patient.

Our literature search showed only four such cases reported earlier. Mattison et al<sup>5</sup> reported a case of pulmonary edema after pulmonary valvotomy, but the patient had a coexistent unrecognized mitral stenosis and so it could be presumed that the sudden increase in blood flow with stenosis at the mitral valve precipitated the pulmonary edema. Similarly Walker et al<sup>6</sup> reported pulmonary edema after BPV in an elderly patient, which resolved with diuretics, ventilation and minimal ionotropic support. This was presumed to be due to a sudden increase in blood flow in an otherwise unprepared pulmonary vascular bed. Both these cases show that the left side of the heart should be ready to handle a sudden increase in blood flow after BPV in severe pulmonary stenosis. The pulmonary edema in the elderly patient could have also been due to presence of LV diastolic dysfunction which is common in the elderly. Hence, physiologically a similar principle can act in



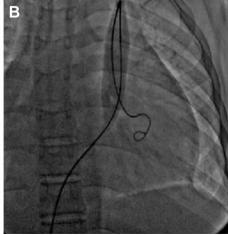


Fig. 1 – A Shows a waist over the balloon during BPV; B Fully dilated balloon. The balloon size was 18 imes 40 mm.

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