

CASE REPORT

Traumatic damage to the mitral valve during percutaneous balloon valvotomy for critical aortic stenosis

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Abstract

Percutaneous balloon valvuloplasty is now a widely accepted alternative to surgical valvotomy for patients with congenital aortic valve stenosis. Mitral valve anomalies are well known to coexist and influence the prognosis from all palliative procedures. Two cases of mitral valve injury occurring during balloon aortic valvuloplasty are reported, one an 11 month old boy, the other a 2 day old baby boy. Both cases were characterised by an unusually posterior position of the guidewire, over which the balloon was deployed. The wire, and hence the balloon, may have been placed through the tension apparatus of the mitral valve with subsequent damage to its free edge on inflation. This is at least conceptually more likely to occur if the orifice of the valve is posterior, if there is a small left ventricular cavity, or if the mitral valve itself is abnormal—features present in both cases. Possible strategies for decreasing the incidence of such damage are considered.

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Keywords: critical aortic stenosis; mitral valve damage; percutaneous balloon valvotomy

Congenital aortic valve stenosis accounts for 3–6% of congenital heart disease^{1,2} and percutaneous aortic balloon valvuloplasty (PABV) is becoming the treatment of choice in many institutions.^{3,4} Results of percutaneous aortic balloon valvuloplasty have been shown to be comparable to those of surgery in children outside the neonatal period^{5,6}; but the use of PABV in critical aortic stenosis in neonates remains controversial.³ Abnormalities of the mitral valve, often associated with left ventricular hypoplasia, have been shown to be a highly significant independent predictor of survival after surgical intervention in neonates with critical aortic stenosis,⁷ and we consider that they may also make the mitral valve more susceptible to damage during PABV.

Mitral valve injury has been described in two older children undergoing PABV,⁸ and it was noted in one case that the guidewire was posi-

tioned posteriorly in the left ventricle. We report two cases of mitral valve injury occurring during balloon aortic valvuloplasty, one an 11 month old boy, the other a 2 day old baby boy.

Case 1

An 11 month old boy with asymptomatic but severe aortic stenosis, was referred from overseas. Initial echocardiography showed a severely stenotic tri-leaflet aortic valve with a peak instantaneous systolic gradient of 130 mm Hg across it. There was an eccentric valve orifice between the right and left coronary leaflets and consequent severe left ventricular hypertrophy. The mitral valve had short chordae between the hypertrophied papillary muscles and the leaflet tips but there was no mitral stenosis.

He underwent a balloon dilatation of his aortic valve via a right femoral arterial approach under general anaesthesia. Aortography confirmed a severely stenotic aortic valve, which was crossed using a 5 F multipurpose catheter over a 0.025" guidewire. A 4 cm × 10 mm balloon was inflated across the aortic valve (echo diameter 11 mm). No ventriculography was performed and the immediate postprocedural Doppler gradient fell to 35 mm Hg. There was no aortic regurgitation and no mitral regurgitation was seen on a further echocardiogram performed 24 hours after the procedure.

On routine follow up at three months he was increasingly breathless. The Doppler derived systolic gradient across the aortic valve was 40 mm Hg but there was severe mitral regurgitation. Transoesophageal echocardiography revealed a tear in the anterior leaflet of the mitral valve. At surgery the anterior leaflet was detached from the annulus over 5 mm and there was a tear from the annulus to the free edge of the leaflet. This was successfully repaired with a patch of autologous pericardium.

The angiograms from the PABV were reviewed. With hindsight, there was a second more distal indentation on the inflating balloon, which we now consider to represent the passage of the balloon through the shortened chordae of the mitral valve, and that on

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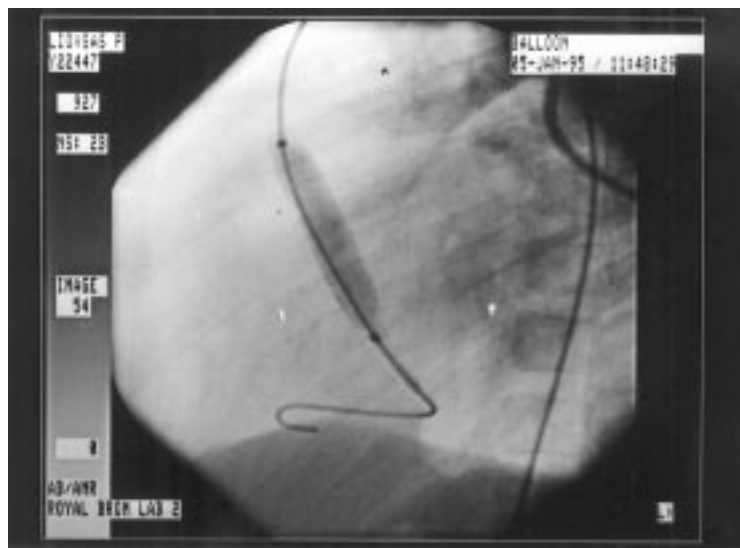


Figure 1 Angiogram showing the posterior position of the guidewire in the left ventricle of case 1.

inflation the tension caused the tear. It is also evident that before insufflation the guidewire occupied a profoundly posterior position in the left ventricle (fig 1).

Case 2

A 2 day old baby boy was referred with cardiogenic shock associated with severe metabolic acidosis. Treatment with positive pressure ventilation and prostaglandin led to prompt resolution of the acidosis. Echocardiography showed critical aortic stenosis with profound left ventricular dysfunction (shortening fraction 5%). The mitral valve appeared normal. The peak instantaneous gradient across the aortic valve was only 35 mm Hg reflecting the poor left ventricular function.

Cardiac catheterisation was performed via the right femoral artery; a size 4 right coronary artery catheter was advanced over an 0.018" wire into the left ventricle. Initially there was a peak to peak gradient of 35 mm Hg between the left ventricle and aorta. A balloon aortic valvuloplasty was performed using a 6 mm balloon, reducing the valve gradient to 13 mm Hg.

Before discharge echocardiography showed a Doppler derived gradient of 25 mm Hg, mild aortic regurgitation, a small arterial duct, a 6 mm secundum atrial septal defect, and greatly improved left ventricular function. The mitral valve appeared normal and no mitral regurgitation was evident.

There was a gradual deterioration over the next 10 weeks, with increasingly severe congestive heart failure. Echocardiography showed trivial aortic stenosis and regurgitation, but profound dilatation of the left atrium with severe mitral regurgitation. There was also obvious left ventricular endocardial fibroelastosis, with a restrictive pattern to left ventricular filling on Doppler studies.

At surgery two tears were found in the posterior leaflet of the mitral valve. The aortic valve appeared normal. It was necessary to perform mitral valve replacement but the baby

died in the immediate postoperative period from low cardiac output.

On reviewing the initial angiogram the wire appears to occupy a posterior position in the left ventricle, and although a less convincing second waist appears on balloon inflation we consider that mitral valve damage may have occurred by a similar mechanism to that in the first patient.

Discussion

We have previously reported our institutional results of a non-randomised but contemporary series of children and neonates undergoing balloon dilatation or surgery for congenital aortic valve stenosis.⁴ The results were similar to other studies.^{3 5 6 8} There was essentially no difference in outcome of the two groups, but those treated by balloon valvuloplasty had a significantly shorter hospital stay. The secondary morbidity, however, of balloon dilatation and surgery must also be taken into account when comparing the two techniques. The two cases presented in this report highlight an important complication of balloon aortic valvotomy that may be avoidable.

In keeping with a similar case reported previously,⁸ both our cases were characterised by an unusually posterior position of the guidewire, over which the balloon was deployed. We speculate that the wire, and hence the balloon, may have been placed through the tension apparatus of the mitral valve with subsequent damage to its free edge on inflation. This is at least conceptually more likely to occur if the orifice of the valve is posterior, if there is a small left ventricular cavity, or if the mitral valve itself is abnormal—features present in each of our cases.

It is interesting that neither patient had demonstrably important mitral regurgitation immediately after the procedure, and it is conceivable that extension of a tear occurs over time or possibly that the mitral insufficiency became more pronounced as left ventricular function recovered.

We suggest that if a posterior wire position is noted on lateral screening it should be repositioned, even if this necessitates recrossing the aortic valve. Whether a transcarotid approach, with its less angled catheter position in relation to the valve, or intraprocedural transoesophageal echocardiography would reduce the likelihood of this complication is speculative, but certainly the latter seems an attractive way of confirming the position of the wire and balloon if there is doubt.

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