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Case Report

Can device closure of AP window be made easy?

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ABSTRACT

Aortopulmonary window is an uncommon congenital cardiac defect. Most infants presenting with aortopulmonary window will undergo conventional surgical repair. Experience with transcatheter closure of aortopulmonary window is limited. We report the case of a 5-year-old boy with aortopulmonary window, in whom transcatheter closure was performed successfully using Amplatzer ductal occluder device.

Keywords: Amplatzer ductal occluder, Aortopulmonary window, Congenital heart disease

INTRODUCTION

An aorto-pulmonary window (APW) is a communication between the pulmonary artery and the ascending aorta in the presence of two separate semilunar valves.[1] The window is usually a large oval defect and in about 10% of cases it is small.[2] APW accounts for approximately 0.1% of all congenital cardiac anomalies.[3] Associated anomalies occur in 50% of cases and include interrupted aortic arch, right pulmonary artery from aorta, coarctation of aorta, coronary artery from main pulmonary artery, tetrology of Fallot, or even pulmonary atresia or aortic atresia. In most cases, surgical repair is undertaken during infancy using different techniques like ligation without cardiopulmonary bypass (CPB), division and over-sewing between clamps on CPB, or transaortic patch closure.[1] The experience with trans-catheter closure of APW is limited. We report a case of successful closure of APW using Amplatzer ductal occluder device.

CASE REPORT

A 5-year-old boy presented to us with history of recurrent episodes of lower respiratory tract infection and congestive heart failure during infancy and childhood. On clinical examination, he had a high volume collapsing pulse with a rate of 96 beats per minute. His blood pressure was 104/50mmHg. Apex beat was felt in the left 6th intercostal space 1cm outside the mid-clavicular line. There was grade VI/VI systolic murmur. It was best heard over the left 2nd and 3rd intercostal space. Hemoglobin was 11.3gm/dl and other baseline biochemical parameters were normal.

Table 1: Pressure and oximetry data.

	Pressure data (mmHg)	Oximetry (%)
SVC	-	70%
RA (mean)	6	70%
RV	81/04	70.1%
MPA	65/31	87.7%
PA wedge (mean)	10	-
LV	104/08	97.7%
Aorta	107/50	97%

Two-dimensional echocardiogram showed turbulent flow (jet width of 9.2mm) across APW. Left atrium and ventricle were dilated. There was no patent ductus arteriosus or coarctation of aorta. The pulmonary valve

was thick, doming with a gradient of 25mmHg. Cardiac catheterization and selective aortogram were performed. The step-up in oxygen saturation was 17% (absolute value, 24% of RV) in the main pulmonary artery. Pulmonary artery pressure was 65/31mmHg (mean 47mmHg). Pulmonary to systemic flow ratio (Qp / Qs) was 2.7:1 (Table 1). Selective ascending aortogram in AP view showed the presence of APW measuring 10mm in diameter (Figure 1).

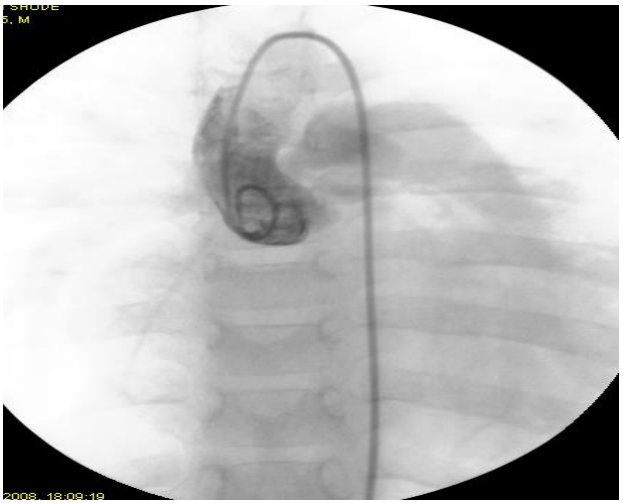

Figure 1: Ascending aorta shoot showing AP window.

It was round in shape, and was located in the left lateral wall of the ascending aorta, 12mm away from the origin of the left main coronary artery (LMCA). It communicated with the main pulmonary artery on its right wall before origin of right pulmonary artery. The APW was crossed from the venous side via the right femoral vein, inferior vena cava, right atrium, right ventricle and pulmonary trunk with a 5 F Judkins Right 4 (JR4) catheter and 0.032" angiography wire. The JR 4 catheter was then advanced over the wire and its tip positioned in the descending aorta. The angiography wire was exchanged with a super stiff Amplatzer J-tipped 0.035" wire leaving its tip in the descending aorta. A long 7 F Cook's sheath and dilator were advanced over the wire and its tip positioned across the APW into the descending aorta. A 12×10mm Amplatzer duct occluder device was deployed across the APW. A check aortogram was done to ensure complete occlusion of the defect without impinging upon LMCA. The device was delivered and the sheath was withdrawn in the right ventricle. An aortic root angiogram performed after 10 min showed the device occluding the APW completely (Figure 2).

There was no impingement of left coronary artery, or aortic and pulmonary regurgitation. There was gradient of 30 mm of Hg across pulmonary valve with RV pressure of 45/05mm of Hg. 2 D echocardiography done at 3 months showed no residual shunt and valvular pulmonary stenosis was mild (Gradient across pulmonary valve 28mmHg).

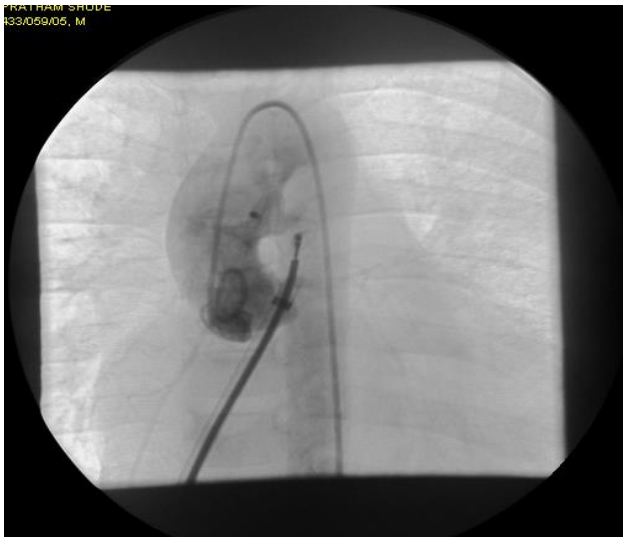

Figure 2: After closure of AP window, no opacification of pulmonary artery.

DISCUSSION

This case report demonstrates the feasibility of non-surgical closure of APW using Amplatzer duct occluder device in carefully selected cases. Transcatheter closure of APW should be considered when anatomy is favourable in terms of location and size of the defect, in the absence of associated anomalies.[5] Initial report of transcatheter closure of APW describing the use of umbrella occluder system in a child resulted in incomplete closure.[6] Subsequently, Rashkind double umbrella occluder systems were successfully used in a child with post-operative APW and an infant with native APW.[2,7] A buttoned device was used to close post-operative APW in an adult. In our patient, we used an Amplatzer duct occluder device. Literature review revealed three case reports of closure of APW using Amplatzer device; one in a child with post-operative APW using a custom-made Amplatzer device and one in native APW.[8,9] In the third report, a septal occluder device and a ductal occluder device was used in two patients.[6]

All reported cases were done using retrograde approach through arterial end to cross AP window and then snaring the wire through venous end. In present case we used venous approach for crossing AP window. Initial attempts with terumo wire failed to cross as high pressure jet was not giving stability to wire tip. Slightly stiffer wire like 0.035" coronary angiography wire though JR4 catheter was more stable and wire could be advanced against jet of AP window flow. We crossed AP window from venous side, and this is an option with a sizable hole but the alternative to cross from artery is equally good. Using a super stiff wire may sometime cause tissue damage especially to tricuspid valve.

Most infants presenting with APW will require conventional surgical repair. Mori et al classified APW as

Type I (proximal defect between ascending aorta and main pulmonary artery), Type II (distal defect between ascending aorta and right pulmonary artery), and Type III (all the defects inclusive, Type I+II).[4] The type of APW most suitable for device closure is small APW located in the middle of the two great arteries, away from the origin of the left coronary artery and the right and left main pulmonary arteries, with no associated congenital anomalies.[3] Significantly less discomfort, avoidance of CPB and surgical scar, and shorter duration of hospital stay make device closure the treatment of choice in such selected cases of APW.

CONCLUSION

In percutaneous device closure procedure with higher pressure gradient across the defect, use of transevenous approach to cross defect retrogradely reduces procedure time. Slight stiffer wire gives more stability while crossing this type of defect.

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