Left pulmonary artery stenting for relief of left pulmonary artery stenosis following ductal closure using Amplatzer Duct Occluder II

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ABSTRACT

A 6-month-old infant with moderate-sized patent ductus arteriosus (PDA) and inadequate weight gain underwent closure of the duct using Amplatzer Ductal Occluder II (ADO II). She developed severe progressive left pulmonary artery (LPA) stenosis due to protrusion of the disc at the pulmonary end of the ADO II. She was subjected to balloon angioplasty of the LPA stenosis with suboptimal result. Hence, she was subjected to stenting of the LPA using a Formula stent which could be subsequently postdilated to keep up with the growth of the child. Immediate and short-term results were excellent anatomically as well as physiologically.

Keywords: Amplatzer Duct Occluder II, left pulmonary artery stenosis, left pulmonary artery stent

INTRODUCTION

Percutaneous device closure of patent ductus arteriosus (PDA) was first reported by Porstmann *et al.* in 1971.^[1] The first-generation Amplatzer Ductal Occluder (ADO) device has emerged as the most widely used device for percutaneous ductal closure. The second generation of the device – ADO II was introduced with a view to close nonconical ducts in infants. In the initial experience with the ADO II, the incidence of left pulmonary artery (LPA) stenosis was found to be around 0%–8%.^[2-4] Most of these cases of LPA stenosis showed spontaneous improvement during the follow-up without any intervention.^[5] We report a case of progressive worsening of LPA stenosis caused by ADO II device impingement, which was successfully treated with pulmonary artery stenting.

CASE REPORT

Our patient was a preterm girl child, born at 35 weeks of gestation who was detected to have a PDA at the age of 3 months. Her two-dimensional (2D)

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echocardiography (Echo) showed a moderate-sized PDA, with no other structural disease. On follow-up, she had features of congestive cardiac failure with shortness of breath on feeding and inadequate weight gain. Her chest X-ray showed cardiomegaly with increased vascularity. She was initiated on appropriate medical therapy, but her weight for age continued to be under the third centile. Repeat Echo at 6 months demonstrated persistence of moderate-sized PDA with volume overload of the left atrium and left ventricle. Her weight remained at 5.5 kg ($<3^{rd}$ centile). In view of her symptoms and presence of moderate-sized PDA, she was taken up for the device closure. Angiography demonstrated Krichenko et al. Type C^[6] PDA with a diameter of 4 mm and length of 6 mm. A 6 mm \times 6 mm ADO II (St. Jude Medical, St. Paul, MN, USA) was implanted across the duct by antegrade approach. Pressure measurements taken in the aorta after device release demonstrated no pressure gradient across the device and thoracic aorta. Angiography demonstrated no residual PDA flow and no evidence

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On follow-up examination 6 weeks after device closure (8 months of age), the patient's respiratory status and weight gain showed significant improvement. However, a transthoracic 2D Echo showed significant stenosis of the LPA origin due to protrusion of the disc of the ADO II. The child continued to be asymptomatic with adequate weight gain but with persistent stenosis of the LPA by the device. At the age of 2 years, the Doppler



Figure 1: (a) Two-dimensional echocardiography and color Doppler-Amplatzer Ductal Occluder II device *in situ* with left pulmonary artery stenosis. (b) Computed tomography scan shows Amplatzer Ductal Occluder II device impinging on left pulmonary artery causing severe origin stenosis. (c) Fluoroscopic image showing left pulmonary artery origin stenosis. (d) Three-dimensional rotational angiography showing left pulmonary artery origin stenosis

velocity across the LPA was 3.5 m/s with diastolic tail [Figure 1a]. A radionuclide lung perfusion scan was performed which showed significant mismatch of right and left lung perfusion. The right: left lung perfusion ratio was 80:20.

The child was taken to the catheterization laboratory for balloon angioplasty of the LPA. She underwent balloon dilation of the LPA using a noncompliant, $10 \text{ mm} \times 40 \text{ mm}$ Mustang balloon (Boston scientific) at 15 atm pressure. There was immediate recoil of the LPA after deflation of the balloon. At this time, postdilatable stents were not available, and hence, stenting was not considered as one of the options. The family refused surgical removal of the device with or without LPA plasty. The child was medically followed up for another 1 year. At this point, the LPA peak velocity on continuous-wave Doppler increased to 4 m/s. Throughout the follow-up, the right ventricular pressure was not elevated, and the right ventricular function was normal. A computed tomographic scan was performed at this stage which showed severe focal stenosis the LPA due to device impingement with poststenotic dilation of distal segment [Figure 1b]. Fortunately, by this time, postdilatable stents were available, and hence, the child was taken up for LPA stenting using Formula stent (Cook). A 3D rotational angiography (3DRA) was performed for 3D evaluation of the LPA in the catheterization laboratory along with routine fluoroscopy [Figure 1c and d]. The exact plane of working was decided by the plane of 3DRA where the stenosis was best seen. The stenosed segment was crossed using a 5 Fr Judkin's right coronary catheter and 0.035 Terumo wire. An optimum distal wire position was secured and exchanged for 0.035 Amplatz super-stiff wire. A 7 Fr long sheath was introduced over this wire and was positioned distal to the point of LPA stenosis. The LPA origin measured 4 mm while the distal LPA measured 9 mm in diameter. The length of LPA before branching was 28 mm. Hence a $10 \text{ mm} \times 20 \text{ mm}$ premounted Formula stent (Cook) was advanced through the long sheath across the area of obstruction in the proximal LPA. The long sheath was retracted off the balloon-mounted stent and the stent position optimized using angiographic control. The stent was then deployed at an appropriate position.

Repeat fluoroscopy and 3DRA showed good stent position with no residual stenosis [Figure 2a and b]. The pulmonary disc of ADO II was squeezed away from the LPA without any recanalization of the ductus. A pull-back gradient from LPA to main pulmonary artery showed no residual gradient. The right ventricular pressure and function were normal. She was advised oral aspirin in the dose of 5 mg/kg once a day and continued for 1 year.



Figure 2: (a) Fluoroscopic image showing stent and Amplatzer Ductal Occluder II device *in situ* with good flow in left pulmonary artery. (b) Three-dimensional rotational angiography demonstrating relief of left pulmonary artery origin stenosis with stent angioplasty. (c) Two-dimensional echocardiography and color Doppler-stent and Amplatzer Ductal Occluder II device *in situ* with good flow in left pulmonary artery

At 6 months follow-up, 2D Echo showed no residual PDA, good stent position, and no residual LPA obstruction [Figure 2c].

DISCUSSION

LPA obstruction is a rare complication of percutaneous PDA occluder device placement.^[2-4] It is known that larger devices used in smaller children are more likely to cause pulmonary artery stenosis.^[7] It is more likely to occur with ADO II device due to the large pulmonary artery disc.^[5] We believe that in our case, small child with large pulmonary artery disc of ADO II and some contribution from the ductal tissue in LPA contributed to progressive stenosis. While there have been reports of spontaneous improvement of LPA stenosis, our patient demonstrated progressive LPA obstruction related to pulmonary artery disc of ADO II device.

This case demonstrates that the results of balloon angioplasty in these cases are suboptimal. Stent angioplasty is a feasible and effective method of relieving LPA obstruction caused by an ADO II device. The Cook Formula stent is a premounted balloon-expandable stent that can be significantly overdilated to double its original size with virtually no shortening allowing for precise placement and minimal protrusion into adjacent vessels.^[8] The stent has low profile and can be deployed through 7 Fr sheath which makes it ideal to be used in young children. The use of these newer Formula stent design will ensure that the stent can be left in place throughout childhood and eventually further dilated to a normal adult diameter of the LPA without significant shortening. The stent works by pushing away the pulmonary artery disc of device towards the vessel wall and thus relieving the obstruction. Dean and Slack^[9] reported a case of LPA stent angioplasty following ADO I implantation. However, there have been no reported cases of stent angioplasty following ADO II device. Stenting of the LPA provided excellent immediate relief; however, long-term outcome needs to be evaluated over period of time.

CONCLUSION

In our patient, stent angioplasty provided an effective method of relieving the LPA obstruction caused by an ADO II device. Stent angioplasty should be considered in selective patients who have LPA stenosis postdevice placement, which does not improve with time. However, more number of patients and long-term follow-up are needed to validate the use of postdilatable stents in all patients with pulmonary artery stenosis following device placement.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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