Transcatheter closure of hypertensive ductus with amplatzer post infarction muscular VSD occluder after percutaneous retrieval of embolized amplatzer duct occluder

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ABSTRACT

Transcatheter closure of a large hypertensive patent ductus arteriosus is challenging with scant data about it. Even more challenging is retrieval of an embolized Amplatzer duct occluder. We report successful closure of a 12 mm large ductus with the Amplatzer muscular VSD occluder (post myocardial infarction) after percutaneous retrieval of the embolized, largest available, 16/14 mm Amplatzer duct occluder.

Keywords: Closure, embolization, hypertensive PDA, retrieval

INTRODUCTION

Transcatheter closure of a large hypertensive patent ductus arteriosus (PDA) poses a challenge to the interventional cardiologist and there is scant data regarding the same. Even more challenging is percutaneous retrieval of a malpositioned Amplatzer duct occluder (ADO). In the multicenter U.S. experience of 439 implants, transcatheter closure of PDA with the ADO was safe and effective in most patients with PDA up to a diameter of 10.6 mm, with an embolization rate of 0.5% and a 50% percutaneous retrieval rate.[1] We report a case of successful closure of a large hypertensive PDA with the Amplatzer muscular VSD occluder (post-myocardial infarction) after successful percutaneous retrieval of an embolized ADO.

CASE REPORT

A 28-year-old female presented with New York Heart Association (NYHA) Class III dyspnea. She had been diagnosed to have congenital heart disease in early childhood, but had not been treated for the same. Physical examination revealed cachexia. There was no clubbing or cyanosis. An ejection systolic murmur and an early diastolic murmur could be heard in the pulmonary area. Chest radiography revealed cardiomegaly and increased pulmonary vascularity. Biventricular hypertrophy was evident on the electrocardiogram. The trans thoracic echocardiogram showed a large PDA with continuous flow [Figure 1], left atrial enlargement, left ventricular volume overload, right ventricular hypertrophy and moderate pulmonary regurgitation.

Transcatheter closure with ADO

Right femoral arterial and venous accesses were obtained. The plan to perform test balloon occlusion of the PDA

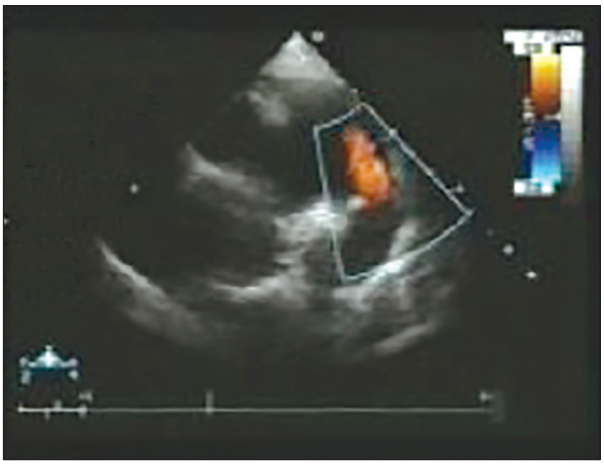

Figure 1: 2D Echocardiography-colour Doppler image showing a large non-restrictive patent ductus arteriosus shunting predominantly left to right

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was altered due to occlusion of left iliac vein. It was decided to 'test-occlude' the PDA with the ADO itself, and then measure the pulmonary artery (PA) pressure through the delivery sheath before ADO release. Right and left heart catheterization was performed. There was systemic pulmonary hypertension (Aorta 110/80, PA 110/70). The baseline saturations revealed only a left to right shunt (a step-up of 12% in the left PA with descending aortic saturation of 98%). A 6F-pigtail catheter was used to perform aortography in lateral [Figure 2] and right anterior oblique projections, which revealed a type A ductus with a diameter of 12 mm at the pulmonary arterial end. The duct was closed by the usual technique. Using a 9F Amplatzer delivery sheath (AGA Medical Corporation, Golden Valley, MN), the largest available (16/14 mm) ADO that was precisely deployed into the duct, after confirming a drop in PA pressure and performing check aortographic shoots before release. Following deployment of the device, the PA pressure dropped to 70/50 mm Hg with an aortic pressure of 110/80. There was a mild residual shunt through the device [Figure 3]. Post-procedure, the patient was doing well without any evidence of hemolysis. However, twelve hours later, she complained of right-sided back pain. Echocardiography and fluoroscopy revealed embolization of the ADO into the right pulmonary artery (RPA) [Figure 4].

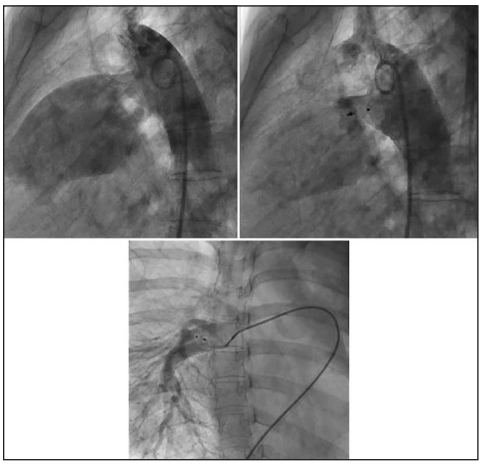

Figure 2: *Upper Left Panel*: Descending thoracic aortic angiogram done in the left lateral view showing a large type B PDA with a diameter of 12 mm at the pulmonary arterial end *Upper Right Panel*: Descending thoracic aortic angiogram done in the left lateral view 10 minutes after release of the ADO showed appropriate device position with only a trivial residual shunt *Lower Panel*: Fluoroscopic image 18 hours post ADO-deployment. The device has embolized (*arrow*) into the right pulmonary artery (RPA)

Retrieval of the ADO

After discussion with the surgeons and the patient's family, a decision was taken to attempt percutaneous device retrieval, in order to avoid the alternative of open-heart surgery. Under local anesthesia, a 14F access was taken in the right femoral vein; the right femoral artery and the right internal jugular vein were cannulated with 6F introducer sheaths. A 12F Mullins sheath (Cook Inc.) was introduced into the PA through which a 6F Judkins right coronary catheter (RCA) was passed past the device in the RPA over an angled tip glide wire. A tracker catheter was passed retrogradely through the duct and placed into the main pulmonary artery (MPA). A 3F vascular retrieval forceps (VRF) (Cook Inc.) was passed antegrade through the RCA catheter, and the side of the ADO was grasped by the alligator jaws of the VF [Figure 5]. The ADO was then retrieved into the MPA. Since the ADO was held by its side, it was difficult to snare it with the goose neck snare passed retrograde through the tracker catheter from the PDA. Further, attempts to hold the aortic disk of the ADO with a 5F bioptome (Cook Inc.) passed retrograde failed. Hence, an attempt was made to retrieve the ADO held by the VRF into the 12F Mullins sheath in the MPA. However, this led to the tip of the Mullins being crumpled [Figure 4 *Upper Panel*]. In addition, the ADO could not be released from the jaws of the VRF. Hence, the ADO was brought bare through the pulmonary valve and the tricuspid valve into the inferior vena cava at its junction with the right

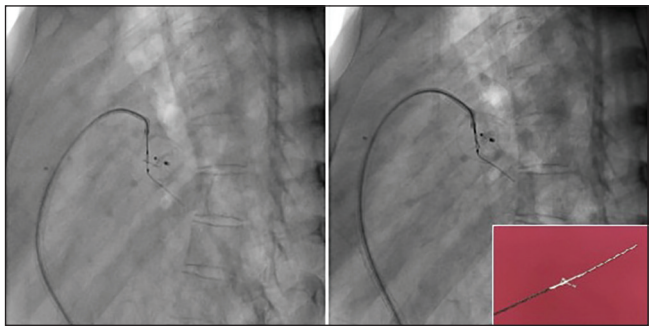

Figure 3: The device has been grabbed with the 3F Vascular retrieval forceps (*inset*). The left panel shows the VF with its jaws open, and the right panel shows the VF holding the device

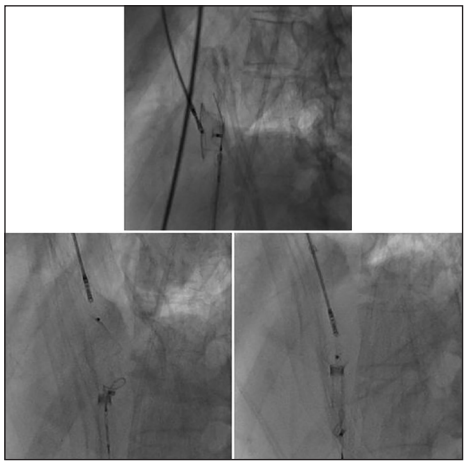

Figure 4: *Upper Panel*: The tip of the Mullin's sheath has been crumpled during the attempt to withdraw the device into it. *Lower Panel*: The device has been held at both ends (*left panel*) and Slenderized (*right panel*)

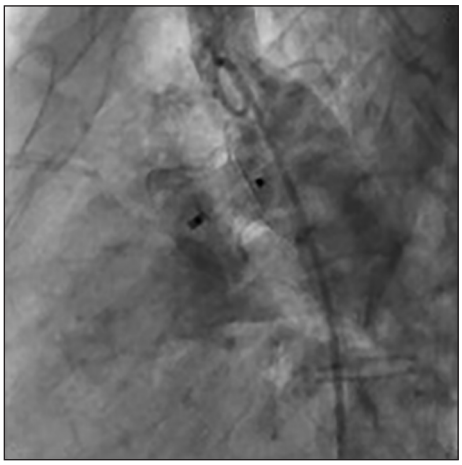

Figure 5: Descending thoracic angiogram following deployment of a 20 mm post-infarction VSD device shows a small residual shunt, which subsequently disappeared on follow-up

atrium. The device was then grabbed near the center of its aortic disk with a 5F bioptome passed through a snaring catheter from the right internal jugular vein. With the 5F bioptome holding the ADO firmly, the VRF could be freed from the ADO. The VRF and the crumpled Mullins sheath was now removed and replaced by a straight stiffer 11F sheath (Cook Inc). The VRF was again reintroduced with its snaring catheter into the straight 11F sheath and the ADO was gripped at its PA end [Figure 4 *Lower Left Panel*]. Thus held firmly at its two ends, the ADO could be slenderized [Figure 4 *Lower Right Panel*] and withdrawn into the straight long sheath and retrieved.

Transcatheter closure with the VSD device

Three weeks later, under local anesthesia using a 10F Amplatzer atrial septal defect (ASD) delivery sheath, an Amplatzer 20 mm post-MI VSD device could be precisely deployed in the PDA [Figure 5] after confirming correct position of the aortic and pulmonary disks on check aortography and pulmonary angiography. The PA pressures fell to 60/40 mm Hg. Post-deployment, there was a mild residual shunt, which completely disappeared 48 hours later. There was no hemolysis. At a follow-up period of 12 months, the patient was asymptomatic with no clinical or echocardiographic evidence of any residual shunt. The pulmonary artery pressures had fallen to 36/20 mmHg.

DISCUSSION

Transcatheter closure of the large hypertensive ductus in adults is challenging. Except for one large series[2] and another smaller one,[3] both from China, there are only anecdotal case reports.[4] Overall, embolization of the device is a rare complication — an incidence of only 0.5% being reported in the multicenter series of 439 patients mentioned earlier in this article. Factors predisposing to embolization include an inappropriately small device size, significant pulmonary hypertension, and 'abnormally' shaped ducts. One of the reasons for embolization could be related to the high distensibility of the hypertensive ductus. Yan *et al.*,[2] have therefore chosen devices 4 to 10 mm larger than the narrowest diameter of the hypertensive ductus. In the present case, the choice of an ADO sized16 × 14 mm device initially appeared reasonable. In hindsight, we now feel that a larger (18 mm) device size would have been more appropriate — although this would not have been available in the ADO series.

Size is not the only issue in selecting an appropriate device for closing a hypertensive ductus. The absence of a retention disc at the pulmonary end of the ADO may predispose to dislodgement of the device in large ducts with pulmonary hypertension. Indeed, Thanopoulos *et al.*,[5] have suggested that the designs of currently used devices in PDA closure may not be appropriate in cases with pulmonary hypertension, and may result in device embolization into the aorta. In their series of seven children with large hypertensive ducti, they have described a 100% successful occlusion rate with the use of the Amplatzer muscular ventricular septal defect occluder (AMVSDO). Larger studies with longer-term follow-up may help in resolving this issue.

Percutaneous retrieval of an embolized ADO by snaring its microscrew is technically more demanding than snaring the microscrew of the Amplatzer septal occluder (ASO). This is principally because the microscrew of the ADO is recessed well within the pulmonary artery end of the device. Additional difficulties encountered during this case included crumpling of the Mullins sheath when we tried to pull the VRF grasping the ADO back into the sheath, and subsequently a failure to release the grasped portion of the device from the VR, necessitating a withdrawal of the 'bare' ASO through the pulmonary and tricuspid valves — a maneuver which carries the potential risk of traumatizing the valvular apparatus. Once the device was brought into the IVC, we were able to grasp its aortic end with the bioptome passed from the internal jugular vein (IJV) route, thereby allowing the freeing of the VRF from the device. An alternative would have been to attempt to retrieve the device from the arterial access. We did not prefer this for two reasons — first, it would have necessitated pulling a large crumpled device through a relatively smaller PDA, with the potential risk of traumatizing the duct. Secondly, a much larger arterial access would be needed — up to 16 French.

When percutaneous device retrieval is contemplated, a general principle is that the largest possible and permissible access size should be chosen. Manipulations such as grasping and snaring the device should preferably be performed in a large chamber or vessel (such as the IVC), taking care to keep the 'bare' device well away from

valvular structures. In case of an ADO, snaring of the microscrew may be rendered difficult by the fact that this screw is recessed well within the pulmonary end of the device. Slenderization of the device using a bioptome introduced through the retrograde route can facilitate its withdrawal into the vascular sheath. Alternatively, the bioptome can be introduced via the IJV route. A straight stiff sheath may help avoid the problem of crumpling of the sheath tip, which was encountered in the present case. The 3F VRF has the advantages of a low profile and a distal spring-tip coil — the latter feature may help avoid accidental engagement of the vascular wall during manipulations. In fact, owing to its flexibility, a second 3F VRF may be used, instead of a bioptome, to hold the device during capture of the device back into the sheath.

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