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Percutaneous closure of hypertensive ductus arteriosus

Carlos Zabal, José Antonio García-Montes, Alfonso Buendia-Hernández, Juan Calderón-Colmenero, Emilia Patiño-Bahena, Antonio Juanico-Enriquez, Fause Attie

ABSTRACT

Background The Amplatzer duct occluder (ADO) has been used with success to close large patent ductus arteriosus (PDA), but some problems exist especially with hypertensive PDAs, such as incomplete closure, haemolysis, left pulmonary artery stenosis, obstruction of the descending aorta and progressive pulmonary vascular disease.

Methods and results We analysed a group of 168 patients with isolated PDA and pulmonary artery systolic pressure (PASP) ≥50 mm Hg. Mean age was 10.3 ± 14.3 years (median 3.9), PDA diameter was 6.4 ± 2.9 mm (median 5.9), PASP was 63.5 ± 16.2 mm Hg (median 60), Qp/Qs was 2.7 ± 1.2 (median 2.5), total pulmonary resistance index (PRI) was 3.69 ± 2.15 (median 3.35) and vascular PRI was 2.73 ± 1.72 (median 2.37). We used ADOs in 145 (86.3%) cases, Amplatzer muscular ventricular septal defect occluders (AMVSDO) in 18 (10.7%), Amplatzer septal occluders (ASO) in three (1.8%) and the Gianturco-Grifka device in two (1.2%) cases. Device diameter was 106.3% ± 51% higher than PDA diameter. PASP decreased after occlusion to 42.5 ± 13.3 mm Hg (p<0.00001).

Immediately after closure, no or trivial shunt was present in 123 (74.5%) cases. Immediate complications were device embolisation in five (3%) cases and descending aortic obstruction in one case. The overall success rate was 98.2%. Follow-up in 145 (86.3%) cases for 37.1 ± 24 months (median 34.1) showed further decrease of the PASP to 30.1 ± 7.7 mm Hg (p<0.0001).

Conclusions Percutaneous treatment of hypertensive PDA is safe and effective. ADO works well for most cases, but sometimes other devices (AMVSDO or ASO) have to be used. When cases are selected adequately, pulmonary pressures decrease immediately and continue to fall with time.

INTRODUCTION

Percutaneous closure of patent ductus arteriosus (PDA) is now considered a first-line alternative to surgery with very good rate of closure and almost no complications with different types of devices.1–15 However, closure of hypertensive PDA is still a challenge.13–15 The Amplatzer duct occluder (ADO) has been used with success to close large PDAs, but some problems exist especially with hypertensive PDAs, such as incomplete closure, haemolysis, left pulmonary artery (LPA) stenosis, obstruction of the descending aorta and progressive pulmonary vascular disease.13 16 17

We analyse our experience in a group of patients with isolated PDA and pulmonary artery systolic pressure (PSAP) ≥50 mm Hg.

Methods

Patients

From September 1999 to January 2008 we intended to treat 845 patients with PDA. From this group we selected the cases with isolated PDA and PASP ≥50 mm Hg. The final study group consisted of 168 cases (19.9% of the whole PDA population).

Catheterisation procedure

The procedure was performed under mild sedation in adults and moderate sedation in children. The first step was to perform an aortic angiography in plain lateral and right anterior oblique views to delineate the PDA. A complete cardiac catheterisation was done and pressures and flows were measured breathing room air and 100% oxygen. Pulmonary vascular resistance was measured estimating oxygen uptake from standardised tables. We also did a pulmonary wedge angiography (PWA) in all cases.

In cases where pulmonary pressure did not decrease at least 20% when breathing 100% oxygen, we performed a test occlusion with a compliant balloon for 10–15 minutes and repeated the measurements. Test occlusion was performed using a 20-mm or 24-mm Amplatzer sizing balloon (AGA Medical Corporation, Golden Valley, MN, USA) hand-inflated with a diluted contrast solution, until a waist could be seen and occlusion of the arterial duct was confirmed by aortic angiography. If response to 100% oxygen and occlusion was non-conclusive, pharmacological challenge with adenosine (10 μg/kg/min for 3 minutes, with increments of 10 μg/kg/min, until significant response or a limit dose of 50 μg/kg/min) or inhaled NO (40 ppm for 10 minutes) was done. Reactivity of pulmonary hypertension (PHT) was established when at least one of the following parameters was achieved: more than 20% decrease in PASP, more than 20% increase in Qp/Qs or a PWA with more than five monopodial arteries and a uniform capillary blush. If reactivity was established, we continued with the selection of the device. In general, if the pulmonary pressure decreased under 60% of the systemic pressure, an ADO (AGA Medical Corporation) at least 50% larger than the smallest PDA diameter was selected (ex: an 8.5-mm PDA with PASP of 42% systemic after oxygen, a 14/12 ADO was used). If pulmonary pressures remained over 60% of the systemic pressure, then an Amplatzer muscular ventricular septal defect occluder (AMVSDO, AGA Medical Corporation) at least 50% larger than the smallest PDA diameter.
Congenital heart disease

was selected (as in the preceding example if the pulmonary pressure after oxygen was 75% systemic, a 14-mm AMVSDO was used). Complete catheterisation was repeated after closure and a control aortogram was done as the last step. No heparin was used during most of the procedures.

Follow-up
Patients were discharged the following morning on prophylactic antibiotics if a residual shunt was present. Follow-up visits were scheduled 1 month and 6 months after the procedure and yearly thereafter with a clinical examination, x-ray and transthoracic echocardiogram. Pulmonary pressures were measured by echo with a measurable tricuspid regurgitation jet, assuming RA pressure was 5 mm Hg and, when we could not find any measurable regurgitation jet, we calculated it with the Doppler upstroke in the main pulmonary artery. In cases where pulmonary systolic pressure measured by echo was >40 mm Hg, follow-up cardiac catheterisation was performed.

Statistical analysis
Statistical analysis was performed with statistical software (SPSS 13.0 for Windows). Quantitative data are expressed as mean ± SD (median). A paired t test or Wilcoxon test was performed as properly indicated to compare two mean values. Factors associated with acute procedural outcomes were sought with t tests and Kruskal-Wallis analysis of variance. Linear regression was performed to compare some results. A p < 0.05 was set as the level of statistical significance.

RESULTS
Demographic data
Mean age of the 168 patients was 10.3 ± 14.3 (3.9) years (limits, 2 months–59 years). There were 137 children (81.5%) and 31 adults (18.5%). From the group of children, 97 (57.7%) were younger than 5 years. There were 117 female (69.6%), and two adults had previous surgical ligation of the PDA in another institution. NYHA class was I in 61 (36.3%) cases, II in 98 (58.3%) and III in nine (5.4%). Some degree of cardiac enlargement was present in all but four cases (CTI ≤ 65 in 148 cases, CTI > 65 in 16).

Procedure data
At angiography, the smallest PDA diameter was 6.4 ± 2.9 (5.9) mm (limits, 1.4–17.9) with 66.7% of cases ≥ 5 mm. The type of PDA was A in 79.8%. Baseline haemodynamic data are summarised in Table 1. Test occlusion was performed in 27 cases with a PASP of 72.1 ± 16.8 mm Hg and a PDA with a smallest diameter of 8.7 ± 4 mm that increased to 12.1 ± 4.7 mm for mean 38% increase; therefore we used the stretched diameter to select the device. PASP decreased 36.1 ± 15.4% (median 40%).

In eight cases pulmonary pressure decreased less than 10% despite test occlusion. The PDA diameter in these cases was 7.5 ± 5 mm, PASP was 65.9 ± 9.2 mm Hg, Qp/Qs was 2.5 ± 1.14 and total PRI was 4.9 ± 2.6. Device occlusion was carried out because of the left to right shunt (all but one Qp/Qs > 1.3), total PRI < 6 in six of them and a PWA with more than five monopodial arteries and a uniform capillary blush. In two cases total PRI was 7.4 and 9.4 and decreased after test occlusion to 6.9 in both.

We used ADOs in 145 (86.3%) cases, AMVSDOs in 18 (10.7%), Amplatzer septal occluders (ASO, AGA Medical Corporation) in three (1.8%) and the Gianturco-Grifka device (Cook Inc, Bloomington, IN, USA) in two (1.2%) cases. AMVSDOs used were three of 22 mm, five of 20 mm, two of 18 mm, four of 16 mm, three of 14 mm and one of 12 mm. ASO devices were used in two cases because a 24-mm AMVSDO embolised or passed freely through the ductus and 28-mm devices closed the PDAs safely (figure 1). In the other case, a 16/14 ADO embolised, it was retrieved and changed to a 18-mm AMVSDO that protruded into the LPA causing stenosis, the device was retrieved and changed to a 18-mm ASO that closed the PDA safely with no protrusion to the LPA.

The device diameter was 106.5% ± 51% higher than the smallest diameter of the PDA (PDA diameter 6.4 ± 2.9 mm vs device diameter 12.3 ± 4.1 mm). This oversizing had no difference among age or pulmonary pressure quartile groups.

The device had to be changed in 15 cases because of embolisation, slippage, significant residual shunt or significant stenosis, for a device exchange rate of 7.7%.

The PASP decreased after occlusion from 65.5 ± 16.2 mm Hg to 42.5 ± 13.3 mm Hg (p < 0.00001) for a mean decrease of 32.7 ± 13.2%. Immediately after device deployment, PDA was completely occluded in 45 (26.8%) cases, there was trivial (through the device) residual shunt in 78 (46.4%), small (less than 1-mm jet) in 54 (20.2%) and moderate (1–2-mm jet) in 11 (6.5%) cases.

The procedure was completed in 62 ± 20.7 minutes with a fluoroscopy time of 11 ± 6.1 minutes.

Immediate complications were device embolisation in five (3%) cases, three were retrieved and changed and two were sent to surgery, and in one case a 10 mm Hg immediate gradient across the aorta increased to 50 mm Hg in the following days and the patient was sent to surgery. An LPA peak gradient <12 mm Hg was present in three (1.8%) cases. The overall success rate was 165/168 (98.2%).

Follow-up data
Follow-up was possible in 145 (86.3%) cases for a mean of 37.1 ± 24 (54.1) months (limits, 1–108). The 20 cases that were lost to follow-up had a post-procedure PASP of 41.5 ± 11.9 mm Hg and 17 had no or trivial residual leak.

On transthoracic echocardiography (echo), only two (1.4%) cases had trivial residual leak, the rest had complete closure of the PDA. Pulmonary pressures measured by echo at the first month of follow-up were very similar to those obtained by catheterisation at the end of the procedure. At their last visit (133 cases > 12 months), pulmonary pressure measured by echo (in 132 (91%) cases by tricuspid regurgitation jet and in 13 (9%) by Doppler upstroke) or cardiac catheterisation (14 patients) decreased further to 50.1 ± 7.7 mm Hg (p < 0.0001). In the eight cases where the immediate PASP decreased less than 10%, follow-up at least for 1 year showed further decrease of the PASP from 64 ± 10.5 mm Hg (limits, 50–75) to 58.9 ± 7.9 mm Hg (limits, 25–48, p < 0.0001) (figure 2). There were 14 cases with

Table 1 Baseline haemodynamic data

<table>
<thead>
<tr>
<th>Pulmonary artery pressure</th>
<th>Mean±SD (median)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic</td>
<td>63.5±16.2 (60)</td>
</tr>
<tr>
<td>Diastolic</td>
<td>31.8±11 (30)</td>
</tr>
<tr>
<td>Mean</td>
<td>42.4±12.2 (40)</td>
</tr>
<tr>
<td>Pulmonary to systemic flow ratio</td>
<td>2.73±1.2 (2.5)</td>
</tr>
<tr>
<td>Total pulmonary resistance index</td>
<td>3.69±2.15 (1.3)</td>
</tr>
<tr>
<td>Vascular pulmonary resistance index</td>
<td>2.73±1.7 (2.37)</td>
</tr>
</tbody>
</table>
There were four (2.7%) complications at follow-up; two vascular (femoral artery pseudoaneurysm and AV fistula) that required surgical treatment, one stroke 5 months after FDA occlusion in a child with protein S deficiency, and an LPA gradient of 20 mm Hg in a 5-year-old girl with an 8.6 mm type A FDA that was completely closed with a 16/14 ADO.

**DISCUSSION**

Percutaneous closure of FDA has been widely available since 1990 and is now considered a first-line alternative to surgery with very good rate of closure and almost no complications using different types of devices. Devices such as coils and the Nit-Occlud work very well for small PDAs, but have major disadvantages when used for large and especially hypertensive ducts. The ADO has been used with success to close large PDAs, but some problems exist especially with large and hypertensive PDAs, such as embolisation, incomplete closure, haemolysis, left pulmonary artery stenosis, obstruction of the descending aorta and progressive pulmonary vascular disease.

Masura *et al* reported the first human experience with ADO where only one patient had significant PHT (mean 46 mm Hg) with immediate reduction of the pulmonary artery mean pressure to 24 mm Hg. Thanopoulus *et al* reported the first use of AMVSDO in seven patients with severe PHT with 100% occlusion rate and decrease of pulmonary pressures. More recently, Yan *et al* published their experience with 29 adults, all with PHT using ADO and AMVSDO, and they achieved closure in 20 cases with clinical improvement at a maximum 6-month follow-up, but with no pulmonary pressure measurements.

Szkutnik *et al* reported their experience in a high altitude environment with 13 patients, children and adults, 38% with PHT, all but one closed successfully with the ADO.

In this paper we report our experience, we believe one of the largest published to date, in a group of patients with isolated FDA and PASP ≥50 mm Hg.

The most important dilemma in these patients is whether to close or not to close the FDA, because of the risk of progressive pulmonary vascular disease. Patients with a pulmonary resistance >6 Wood units when breathing 100% oxygen have been considered unsuitable candidates for repair of congenital heart defects. As others do, we believe that temporary occlusion of the duct is a very good tool to assess the consequences of FDA closure and thereby evaluate the reactivity of severe pulmonary arterial hypertension. If we also perform a pulmonary wedge angiography, a reflection of the anatomical condition of the pulmonary vascular bed, and, in cases that have a non-conclusive response to 100% oxygen, a pharmacological challenge with adenosine or inhaled NO, we can enhance the possibility of ascertaining that pulmonary pressure will decrease after FDA closure, as we did in our cases where, even in the eight patients with an immediate very mild decrease in PASP, we achieved a very good result at follow-up with more than 90% of our cases normalising pulmonary pressures.

Although calculation of pulmonary vascular resistance is also important in assessing the reactivity of pulmonary arterial hypertension, it is just one of the multiple parameters that we have to measure in these patients. Also, we have available today a good number of pulmonary vasodilators that can be used before or after FDA closure.

We think that using all the tools to evaluate each patient as described, we are expanding the number of patients in whom the FDA can be closed with good results. The two patients in whom the FDA was closed with PRI >6 Wood units and PASP

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**Figure 1** Angiographic images of a 25-year-old woman with hypertensive patent ductus arteriosus (PDA). Pulmonary pressures were 90/50 (63) and Qp/Qs 1.3. (A) Aortogram in left lateral projection showing a type C large PDA that measured 17.9 mm. Test occlusion showed a decrease of pulmonary pressures to 45/30 (35) and a PDA stretched diameter of 22 mm. (B) A 24-mm Amplatzer muscular ventricular septal defect occluder (AMVSDO) was used and the control aortogram shows moderate residual shunt and not good apposition of the central waist of the device to the PDA walls. (C) After 10 minutes, the device embolised to the PA and was retrieved. (D) A 28-mm Amplatzer septal occluder (ASO) was then positioned and released. (E) Aortogram shows a better apposition of this device to the PDA walls with slight protrusion to the left pulmonary artery (LPA) and the aortic arch. (F) Control aortogram 8 months after closure showing complete closure with a good conformity of the aortic disc to the PDA ampulla. Pulmonary pressures were 40/18 (23) with no gradient in the LPA.
of 75 mm Hg and 80 mm Hg are doing well, with PASP measured by echo at 45 mm Hg and one has been recatheterised with a PASP of 42 mm Hg with sildenafil treatment. We have had three patients with PAH (>70), low Qp/Qs (<1.5), PVR over 6 Wood units, no reactivity even on test occlusion and a poor anatomy on the PWA that did not undergo closure. They are being treated with bosantan and are waiting to be recatheterised.

Oversizing of Amplatzer devices has been reported to have an important role in achieving a virtually 100% occlusion rate and decreasing the likelihood of embolisation. Oversizing has been reported from 22% to 83%.[19] In our institution from previous experience, we select at least a 50% larger device than the selected cases where PASP remained over 60% of the aortic pressure, we used the AMVSDO or the ASO. Despite using large tools we have available. ADO works well for most cases, but cases should be evaluated individually for reactive PHT with all the indirect measure. Three years of follow-up may not be enough time to ascertain that PHT will not recur.

CONCLUSIONS

Percutaneous treatment of hypertensive PDA is safe and effective. Cases should be evaluated individually for reactive PHT with all tools we have available. ADO works well for most cases, but sometimes other devices (AMVSDO or ASO) have to be used. When cases are selected adequately, pulmonary pressures decrease immediately in most of them and continue to fall with time.

Competing interests Dr Carlos Zabal is proctor and consultant for AGA Medical Corporation.

Ethics approval This study was conducted with the approval of the National Institute of Cardiology "Ignacio Chavez".

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES


