

Percutaneous closure of patent ductus arteriosus in an asymptomatic adult

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The ductus arteriosus allows deoxygenated blood from the right ventricle through the descending aorta and to the placenta in the fetal period. Functional and anatomical closure is complete by several weeks after birth. Patients with ductus-dependent physiology generally have advanced congenital malformations and require surgical intervention early in life. However, some patients will present as adults with a patent ductus arteriosus (PDA). We describe a 43-year-old woman who was found to have a PDA during evaluation for a precordial murmur, and she underwent successful percutaneous closure.

CASE REPORT

A 43-year-old woman with mild systemic hypertension was referred after a precordial murmur was detected by her primary physician. Her only complaint was occasional palpitations. Her systemic blood pressure was well controlled on lisinopril 10 mg daily. A continuous murmur was present in the left first and second intercostal spaces.

Echocardiogram revealed normal-sized cardiac chambers and normal left and right ventricular systolic function with normal valvular structure and function. Using color-flow Doppler, an atypical jet with mosaic pattern was seen in the pulmonary trunk and the descending thoracic aorta (*Figure 1*). Mild coarctation of the aorta also was observed. A computed tomography scan also confirmed the PDA and the mild coarctation at the aortic isthmus (*Figure 2*). The PDA was 3.1×5.9 mm.

Elective percutaneous closure of the PDA was recommended. Initial catheterization revealed normal right-sided heart pressures and no pressure gradient across the mild coarctation of the aorta. A large PDA was seen with aortic angiography (*Figure 3*) and was successfully closed with an 8×6 -mm Amplatzer ductal occluder (*Figure 4*). She was discharged with aspirin 325 mg daily for at least 3 months, clopidogrel 75 mg daily for 1 month, and infective endocarditis prophylaxis for 12 months. At 2-month follow-up, the patient was doing well, and follow-up echocardiogram found no evidence of residual PDA.

DISCUSSION

The clinical presentation of PDA in adults is dependent on the degree of left-to-right shunting. As the degree of shunting

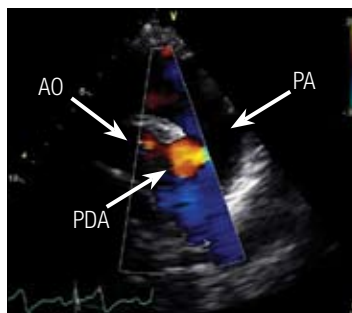


Figure 1. Echocardiographic representation of the patent ductus arteriosus (PDA), showing the color Doppler flow (red-orange) from the aorta (AO) to the pulmonary artery (PA).

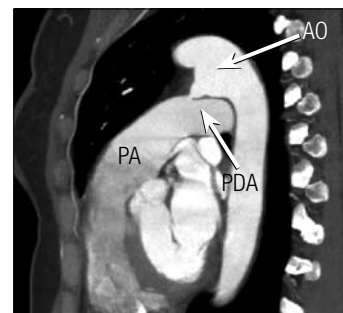


Figure 2. Sagittal cross-section computed tomography image showing contrast entering the pulmonary artery (PA) from the aorta (AO) through the patent ductus arteriosus (PDA).

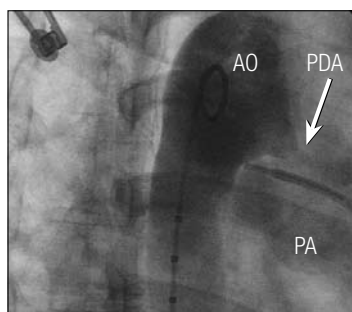


Figure 3. Angiogram depicting contrast extrusion from the aorta (AO) through the patent ductus arteriosus (PDA) into the pulmonary artery (PA).

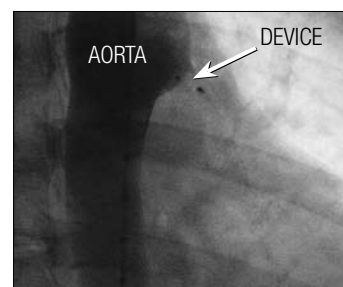


Figure 4. Postdevice implantation angiogram demonstrating absence of contrast in the patent ductus arteriosus and pulmonary artery. Amplatzer ductal occluder (arrow).

increases, pulmonary circulation also increases and may lead to increased work in breathing, exercise intolerance, and occasionally reactive airways disease. Increasing amounts of return to the left heart can eventually lead to hypertrophy, diastolic dysfunction, and systolic failure with its associated symptoms. Palpitations and atrial fibrillation are not infrequent with moderate to

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large PDAs. The chronic exposure of the pulmonary circulation to elevated pressures will result in Eisenmenger's syndrome in patients with large, uncorrected PDAs.

Currently, PDA is categorized as silent, small, moderate, or large based on presentation and noninvasive and invasive evaluation (1). Silent PDA is not associated with long-term clinical complications and in most cases is detected incidentally on imaging (2). If the PDA is small, patients will occasionally present with an endovascular infection. These patients are rarely symptomatic otherwise. In the case of a moderately sized PDA, patients frequently present with dyspnea or palpitations and occasionally atrial fibrillation from progressive left-sided volume overload, chamber dilation, and failure. Patients with large PDAs present with differential hypoxemia, differential cyanosis, and Eisenmenger physiology. Based on our patient's presentation, she had a small PDA.

Physical examination is highly variable and is helpful at the extremes of severity but not in discriminating small from moderate PDAs. The classic physical examination findings include a murmur that is continuous, often described as machinery-like, best heard in the second left anterior intercostal space. The second heart sound is often obscured. It may radiate to the back and have an associated thrill. If the shunt is moderate or large, there may be a diastolic rumble at the apex and bounding peripheral arterial pulses. The point of maximal impulse may be displaced laterally, and the pulse pressure may be increased. As pulmonary hypertension develops and progresses, the diastolic component of the murmur disappears. As Eisenmenger's syndrome develops, differential cyanosis is present, and the murmur may disappear altogether as shunting becomes insignificant.

Echocardiography is the initial procedure of choice to diagnose and characterize a PDA (2). Color-flow Doppler is extremely sensitive. Other suggestive findings include septal flattening, pulmonary insufficiency, and right ventricular hypertrophy. Contrast echocardiography can confirm the presence of shunting into the descending aorta. Computed tomography can provide clarity if the echocardiographic findings are uncertain. In addition, aortic arch anatomy and unusual PDA geometry can be detected.

Cardiac catheterization allows for complete assessment of the hemodynamic importance of the PDA. The shunt fraction (Q_p/Q_s) can help establish severity, with small shunts having a $Q_p/Q_s < 1.5/1$; moderate shunts, $1.5-2.2/1$; and large shunts, $> 2.2/1$. The size and length of the ductus can be estimated with angiography. This is also important in order to choose the appropriate type and size of device if closure is to be performed. Pulmonary vascular resistance can be measured to assess for irreversible pulmonary hypertension.

Closure of an isolated, clinically detectable PDA without irreversible pulmonary hypertension is indicated in all symptomatic patients and in asymptomatic patients with left-to-right shunting (3). It is controversial whether closure of a small ductus is necessary, and to date no large randomized trials exist that address this question (4, 5). However, arteritis and aneurysm have been reported in these patients (6-8). The actual incidence of endarteritis is difficult to estimate and changes depending on

the age of the population surveyed and the era in which estimates are made. Prior to routine closure and antibiotic use and prophylaxis, the rate of infective arteritis may have approached 1.0%, but there are no reliable estimates. It was a frequently fatal complication of PDA. It is now extremely rare, and the current estimate of 0.45% annually after the second decade of life is probably an overestimate (5). The improvement in dental hygiene, the use of prophylactic antibiotics, and a practice of routine closure of PDAs in children have likely all contributed to the decrease in incidence of endarteritis and in the prevalence of PDA in the adult population (5). Despite the very low rate of infective endarteritis, closure of a small PDA is reasonable given the safety and efficacy with percutaneous techniques. The only instance in which it is contraindicated is in the presence of severe pulmonary hypertension or active endarteritis.

A transcatheter technique for closure is currently the preferred method. These techniques have evolved significantly over the past 40 years since the first transcatheter occlusions in 1967 with Gianturco coils (3). Early experience with coils, umbrellas, and plugs was complicated by high rates of embolization, prolonged fluoroscopy times, and early postprocedural failure, especially when used in moderate or large PDAs (9, 10). These complications have been dramatically reduced with the use of the Amplatzer ductal occluder and related devices (11-15).

In the largest multicenter trial of over 400 patients with a wide range of ductal anatomical characteristics, varying PDA sizes, and ages ranging from 0.2 to 70.7 years, efficacy rates were $> 98\%$ at 12 months as defined by the absence of residual PDA on echocardiography when closed with an Amplatzer PDA occlusion device (11). The rate of major complications was 2.3%; such complications included device embolization into the pulmonary artery and distal aorta, partial obstruction of the aorta or pulmonary artery resulting in a gradient above 20 mm Hg, femoral artery pseudoaneurysm, and bleeding. No procedures resulted in death. Most patients were closed with a 6F system, and the median fluoroscopy time was 7.1 minutes—both improvements over previous devices and systems. Hospital stay was generally < 24 hours, which is significantly shorter than most surgical stays. The most frequently used devices include Gianturco coils, Amplatzer septal occluders, and the more recent Nit-Occlud device. Moderate and large PDAs, previously felt to be more suited to open repair, are now safely treated percutaneously (11, 12).

Surgical ligation and division had been the gold standard for PDA treatment since it was introduced in 1939 by Gross and Hubbard. However, with the advent of safer, more easily delivered percutaneous implants, surgery has largely been supplanted by catheter-based therapy (16, 17). The surgical procedural success rate is 100% with a morbidity rate of 4.4% and mortality rate of 0% in a single-institution cohort over a 46-year period (18). Similar morbidity and mortality rates have been seen in other patient cohorts, and a general estimate of the surgical mortality rate is $< 0.5\%$ (18-20). To date, there have been no direct trials comparing surgery with percutaneous closure, and much of the surgical literature predates the advent of newer percutaneous technologies (17, 19, 20).

Adolescents and adults typically have larger PDAs and are often best suited for an Amplatzer ductal occluder or possibly some of the newer devices designed for larger ductus diameters. The PDA can become calcified over time, and the tissues may become friable from previous infective endarteritis. Surgical repair in adults is therefore associated with a higher complication rate (21). Simple surgical ligation may not be possible, and cardiopulmonary bypass with aortic cross-clamping and patch closure may be necessary (9, 19–21). Though patient numbers are small, adults and adolescents with calcified PDAs have safely undergone percutaneous closure (13, 15). It is therefore the recommended closure technique for adults with PDA.

There is little data to guide clinical management after the procedure, and current recommendations are based on what has typically been done in most of the small cohorts followed in the literature. Strict endocarditis prophylaxis, aspirin, and clopidogrel are generally recommended for 6 months and up to 1 year. Repeat echocardiogram is usually done at the first follow-up visit to assess for residual shunt and at 1 year. Some groups have reassessed the closure by performing yearly echocardiograms, though if no residual shunt is detected at 1 year, the benefit in further echocardiography is debatable.

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