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Device Closure of Patent Ductus Arteriosus in Adults

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ABSTRACT

Patent ductus arteriosus (PDA) persisting into adulthood is an uncommon but clinically significant congenital heart condition, representing the third most common congenital cardiac lesion in adults. Clinical presentation varies widely, from asymptomatic cases with incidental murmurs to complications such as left ventricular (LV) volume overload, pulmonary arterial hypertension(PAH), endarteritis, and Eisenmenger physiology. Classification is based on duct size and hemodynamic impact, guiding management decisions. Untreated PDA in adults carries risks including arrhythmias, LV dysfunction, progressive PAH, and endarteritis, which remains a leading cause of mortality. Current guidelines recommend closure when technically feasible, particularly in the presence of LV overload or favorable pulmonary vascular resistance.While surgical closure was historically the gold standard, transcatheter device closure is now the first-line treatment due to lower morbidity, shorter recovery, and high success rates. However, adult PDA closure presents unique challenges, including calcification, tortuosity, and complex anatomy, necessitating precise imaging and appropriate device sizing. Complications such as device embolization, residual shunt, and LV dysfunction may occur but are infrequent.

Keywords: Patent ductus arteriosus; Adult congenital heart disease; Transcatheter device closure; Pulmonary arterial hypertension; Left ventricular volume overload; Eisenmenger syndrome.

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INTRODUCTION

The ductus arteriosus connects the left pulmonary artery near its origin to the descending aorta just distal to the left subclavian artery.[1] Patent ductus arteriosus (PDA) is considered a congenital heart disease when it persists beyond three months in term infants.[2] PDA in adults is uncommon, as most symptomatic lesions are identified and treated during infancy or childhood.[3,4] Nevertheless, it remains the third most common congenital heart lesion in adults,[5] with an approximate prevalence of 0.05%.[5] Adult PDAs are often isolated lesions.[1] Adults with PDA may present with a wide spectrum of signs and symptoms,[6,7,8] including: Silent, incidental lesions, Endarteritis, Left-to-right shunting leading to Left ventricular (LV) dilation and Systolic dysfunction, Pulmonary arterial hypertension (PAH) and Eisenmenger physiology. Some patients may be asymptomatic, but on examination may have: a continuous murmur (heard during both systole and diastole), best heard at the upper left sternal border, often described as a "machinery murmur".[9]

Classification of PDA

Classification and clinical presentation of Adult PDAs may be classified by size and hemodynamic effect:[10]

1. Small: no significant hemodynamic effect (normal LV volume and pulmonary artery pressure).
2. Moderate-to-large: LV volume overload without markedly increased pulmonary vascular resistance.
3. Large: associated with increased pulmonary vascular resistance and symptoms (including differential hypoxemia/cyanosis).

Based on the size of the PDA, it was classified as silent, very small, small, moderate and large PDA by Fernando et al.[11]as shown in Table 1. Small PDAs are often asymptomatic. Patients with a small PDA generally have a normal life expectancy.[12,13,14] Complications of untreated PDA in adults include: endarteritis, arrhythmias, left ventricular dysfunction, progressive pulmonary arterial hypertension, calcifications, aneurysms and rarely acute aortic dissection.[7,15,16] Patients can tolerate very high pulmonary artery pressure for long periods without clinical deterioration.[4] Those with minimal or reactive pulmonary hypertension and limited myocardial changes may still have a normal life expectancy.[4] Hemodynamically significant PDA with a left-to-right shunt can lead to left ventricular volume overload and signs and symptoms of heart failure.[13,14] Before the availability of surgical and percutaneous closure, untreated PDA had an annual mortality rate of 1% (ages 20–29 years), 1.8% (≥30 years).[13] In patients with adult PDA, 34% die before age 40 and 61% die before age 60.[9,12,17,18]

Management of Adult PDA

Treatment of PDA in adults remains controversial.[7] The Dutch guidelines recommend closure of PDA when a murmur is present, primarily to prevent endarteritis.[12,13,14,19,20] Endocarditis is the most common cause of death in patients with PDA.[19,20] It accounts for nearly half of the deaths in untreated cases. [21]The incidence of endarteritis in adults with PDA is reported to be >0.45% per year.[13]Recent guidelines recommend closure of PDA when it is technically feasible, particularly in patients with LV volume overload (cardiac remodeling), patients with PAH, provided, 1)pulmonary vascular resistance (PVR) is < 3–5 Wood units (WU) and 2) dominant shunt is left-to-right. [22] There is increasing recognition of PDA in patients over 60 years of age.[23]Diagnosis and management in elderly patients remain challenging.[24]

Table 1. Patent Ductus Arteriosus (PDA) Classification Based on Size and Hemodynamic Significance

Type	Description
Silent PDA	Usually < 1.5 mm without audible murmur of PDA
Very small PDA	< 1.5 mm; a murmur of PDA is present
Small PDA	1.5–3.0 mm; a murmur of PDA is present
Moderate PDA	3–5 mm; a murmur of PDA is present
Large PDA	> 5 mm; a murmur of PDA is present

Note: Minimal ductal diameter based on lateral cine angiographic view.

Surgical closure of Adult PDA

Surgical closure of PDA has been considered the "gold standard" since 1939.[25,26] Surgical closure of PDA is traditionally performed via left lateral thoracotomy. In complex cases, it may require: median sternotomy (anterior approach), Cardiopulmonary bypass (CPB), Extracorporeal circulation and Aortic cross-clamping.[27] Surgical closure in adults can be complicated by: calcified ductus, aortic fragility due to atherosclerotic (atheromatous) lesions, aneurysm or diverticulum formation, shortening and friability of the ductus, LV dysfunction and PAH.[28,29,30] Outcomes may be further affected by the presence of coronary atherosclerosis and renal disease.[31]

About 15% of adult PDA patients present with severe calcification or tortuosity [32] and require special surgical care. Many such cases require cardiopulmonary bypass and patching of the main pulmonary artery (MPA).[15] Surgical complications include pneumothorax, bleeding and recurrent laryngeal nerve injury. Due to the above factors, surgical closure in adults carries a higher risk compared to children. With the advent of percutaneous device closure, surgical closure is now reserved for very large PDAs not suitable for device closure[33] and unfavorable anatomy,[30] especially aneurysmal PDA.[31,34] The availability of large Amplatzer Duct Occluder (ADO) type devices (Lifetech, Lepu and Cocoon) has made it possible to successfully close large PDAs, particularly in adults.

Device closure of adult PDA

Device closure has become the first-line treatment for most PDAs in adults[27,35,36] as it is safe and effective with good short- and long-term outcomes compared to surgical closure.[6,37,38,39,40, 41,42] Device closure is the preferred therapeutic option due to several benefits: shorter hospital stays, avoidance of surgery (no thoracotomy), no surgical scar, often performed without general anesthesia and fewer complications compared to surgery.[43] Device closure in adults differs from that in children due to: larger PDA size, greater anatomical variations and associated conditions such as: PAH, LV dysfunction, infective endocarditis, calcification, and aneurysm formation.[30] Transcatheter closure of PDA in adults can be more challenging, technically more difficult [39, 44,45] than in children and requires specialized expertise and careful planning [15] and requires longer fluoroscopy time.[39]

Adults with persistent PDA have difficult anatomy like calcification, tortuosity and eccentric ductal lumen.[46] These factors can: increase the difficulty of crossing the duct with a guidewire and complicate the placement of the delivery sheath.[32] Proper device sizing is crucial for successful device closure and requires accurate delineation of the PDA, typically using an angiogram.

Current sizing methods are largely based on pediatric experience using calibrated angiography. [47,48] Anatomical factors, such as size, configuration (shape) and relationship to adjacent structures, are important factors for successful PDA closure.

Krikenco described the angiographic classification that helps to guide the transcatheter closure procedure.[49] PDA can be classified into five types based on Krichenko's angiographic classification as shown in Table 2 and Figure 1.[49]

Angiogram is considered the gold standard for ductal size and morphology, but delineation of PDA using conventional angiography can be difficult in adults.[50] It is due to the distorted anatomical relationship between the aorta and pulmonary artery, calcification of the

Table 2. Krichenko Classification of PDA Based on Shape and Orientation

Type	Anatomy	Topology
A	Conical	Narrowest segment at the pulmonary artery end with a well-defined aortic ampulla
B	Window	Short duct with narrowing at the aortic insertion
C	Tubular	No constrictions present
D	Complex	Multiple constrictions present
E	Elongated conical	Long ductus with constriction at the pulmonary artery end, remote from the anterior tracheal edge

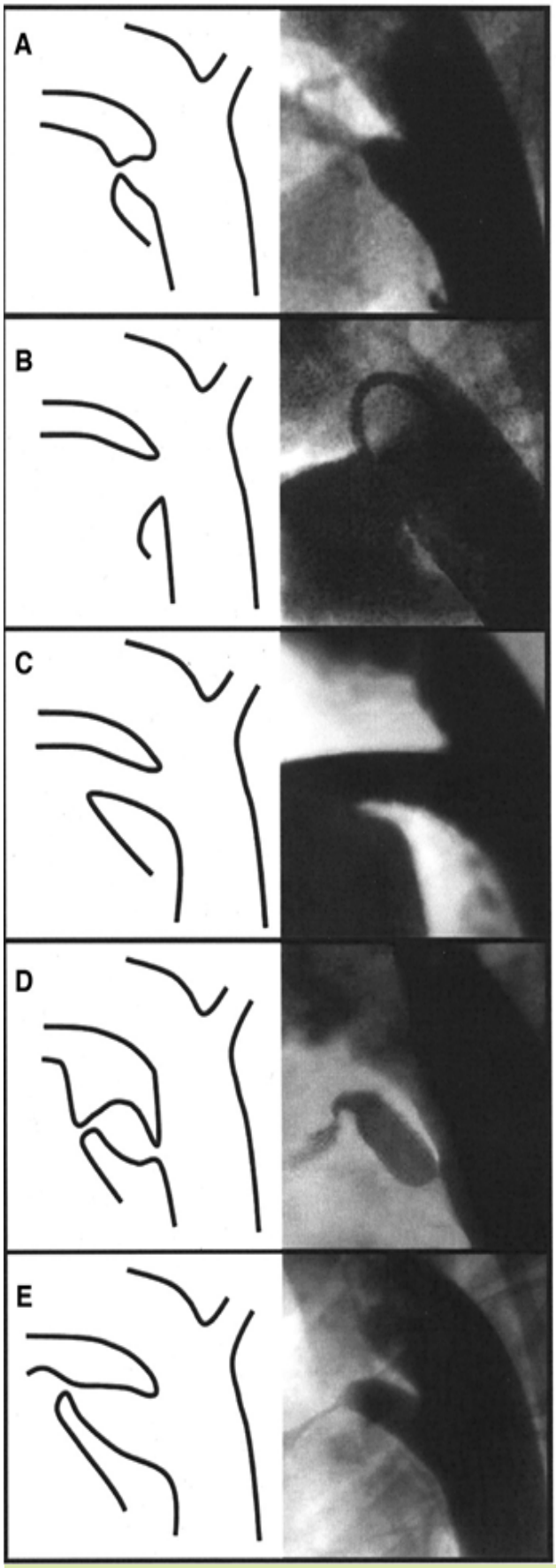

Figure 1. Krichenko's angiographic classification

ductus,[50] high-flow states and large aortic diameters, causing contrast streaming and poor visualization (suboptimal definition).[51]The traditional left lateral view may be inadequate to properly delineate the PDA. The right anterior oblique (RAO) view, which can improve visualization as suggested by Garg and Moorthy, [52] is a good alternative. When angiographic assessment is difficult, balloon sizing is useful because it: accurately outlines the defect diameter, allows morphological analysis and provides precise measurement for appropriate device selection.[15]Angiography itself may underestimate the true size of the ductus due to: assessment only at the moment of contrast injection, inability to measure the fully distended (elastic) diameter. Some PDAs may appear small or severely narrowed (especially at the pulmonary end), but still allow easy passage of a catheter, indicating a larger actual size.[15]However, if 2D Echocardiography is able to reproduce all those variables with precise measurements, aortogram via femoral artery puncture could be avoided in many patients, especially in children.[53] In adults, if we can get a good image to evaluate the PDA, the size of the PDA can be determined by echocardiogram, thus avoiding the aortogram. Some authors use CT angiography for PDA sizing in selected cases. Although it's very accurate, it has an untoward risk of radiation to the younger patient population, who are

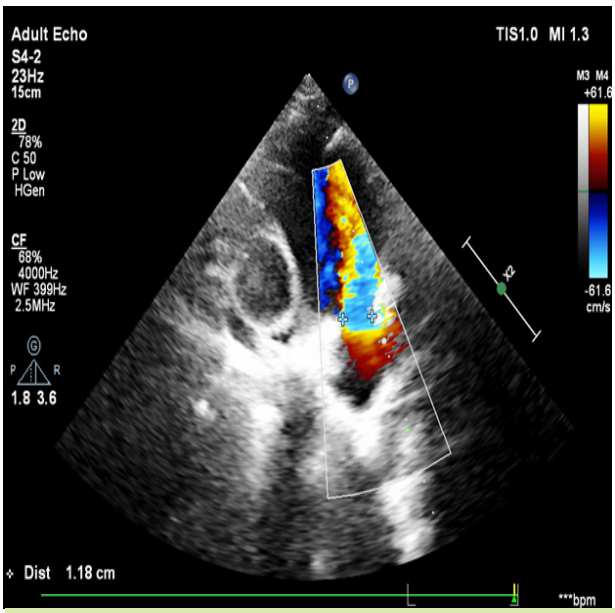

Figure 2. Echo of large PDA in adult

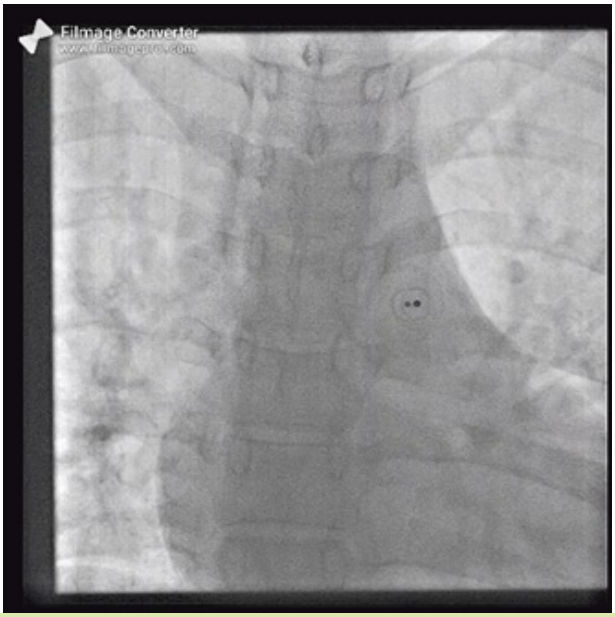

Figure 3. PDA device used to close an adult PDA

commonly referred for PDA device closure. A routine CT scan is not considered for PDA device closure.[51]

Challenges of PDA device closure in adults

Device closure of t he PDA in adults is often difficult due to difficulty in crossing the duct from the pulmonary end.[50] This is a well-described difficulty among elderly patients with PDA.[45,54,55] It's because of gross size mismatch between: moderate-sized arterial duct (typically 3–6 mm) and markedly dilated pulmonary artery due to long-standing shunt, the guidewire tends to enter dilated pulmonary artery branches instead of entering the narrow ductal opening.[46] In adults, the use of JR (Judkins Right) catheter or MP (Multipurpose) catheter with straight-tip Terumo wire or standard guidewire helps in directing the wire into the ductal opening through the pulmonary ends known as the antegrade technique. If antegrade fails, retrograde crossing is useful. PDA is crossed from the aortic end, advance the wire retrogradely into the pulmonary artery, snare the wire in the pulmonary artery, create an arteriovenous (AV) loop, then advance the delivery sheath antegradely and deploy the device to close PDA.[50] Retrograde technique requires more hardware and increases procedure time.

Selection of PDA device in adults

Use of single or multiple coils of various sizes is generally safe and effective for complete occlusion in small PDAs [56] but has reduced success in achieving complete closure in large PDAs.[57] PDA closure with coils can lead to coil embolization as well. Introduction of the Amplatzer Duct Occluder (ADO) and ADO-like devices enables closure of moderate to large PDAs effectively, regardless of ductal morphology. It is proven to be safe and highly effective.[37,58] ADO and ADO-type devices are the most widely used devices suitable for all age groups.[59] The device should be compressed at its mid portion (waist), which corresponds to the narrowest diameter of the ductus arteriosus, leaving the pulmonary end slightly dilated, preventing slippage of the prosthesis into the descending aorta.[15]

Complications of PDA device closure in adults

Although device closure of the PDA is safe and effective, some complications have been reported, and the overall rate is low [40,60,61,62,63,64] Device embolization, narrowing of the Left Pulmonary Artery (LPA), aortic obstruction, residual leak, hemolysis and infective endocarditis are commonly reported.[65,66,67] Device Embolization is the most important complication of device closure of PDA, with the incidence ranging from 0% to 3.1%.[40,60, 65,66,67] and may require transcatheter retrieval. Surgical removal of the device and PDA closure should be done if transcatheter retrieval is unsuccessful. Proper sizing of a PDA is crucial for procedural success and the prevention of complications such as residual leak and device embolization.[15,51]

Undersized devices may lead to: residual shunt (leak), hemolysis, and device embolization, whereas oversized devices may cause: device malformation, erosion and vascular injury.[5] Device closure may be performed based on non-invasive assessment of PDA and its size and device size selection can be used when echo images are enough.[68] It is suggested that oversizing the device by ~4 mm compared to the pulmonary end diameter of the PDA can increase procedural safety and reduce the risk of device

embolization. There is no fear of obstruction of the descending aorta or left pulmonary artery in adolescents or adults.[50] This way of oversizing the PDA device used by Zhang et al. [69] and Francisco et al.[15] In a study by Zhang et al,[69] occlusions of large PDA in patients with pulmonary hypertension. The study was conducted on 137 patients (age ≥12 years) and oversizing of the occluder was preferred and the device chosen for closure had a diameter at the aortic end that was twice the duct diameter. The stringent oversizing of devices increases procedural safety, preventing embolization.[15]

Left Pulmonary Artery (LPA) Obstruction is one of the most significant complications of PDA device closure. However, in adults, it is usually not a major concern due to the large diameter of the pulmonary artery branches. [7] The PDA is a remnant of the 6th aortic arch and typically forms a ~30° angle with the descending aorta. The ADO has a retention disc positioned at a right angle. As a result, the retention disc may partially protrude into the aorta. This is more commonly seen in Type B morphology and in Type A morphology with a small ampulla. In adults, this protrusion is usually not hemodynamically significant.[70]

Calcification is an important risk factor for residual shunt, as a rigid, calcified duct prevents optimal device conformity.[71] Calcified PDA increases the risk of residual shunt and hemolysis. A residual shunt was observed in 18.8% of adult patients with a calcified PDA and 6.2% of them had mild hemolysis.[45] Device sizing for calcified PDA requires careful, intentional oversizing based on angiographic measurements, usually by 2-4 mm, to accommodate the rigidity and friability of the calcified ductal tissue. For heavily calcified and fragile ducts, less rigid, more flexible devices such as the Amplatzer Duct Occluder II (ADO II), ADO II AS, or vascular plugs are preferred to prevent tissue rupture or shear stress.[55]Adult PDA closure devices that are undersized can result in inadequate closure, hemolysis, and increased embolization rates, whereas oversized closure devices may increase the risk of device malformation, erosion, or vascular injury. Furthermore, the large size and high flow state seen in adult PDAs can make defect and device sizing by conventional thoracic angiography challenging and imprecise.[51]

LV systolic dysfunction of PDA device closure in adults

LV Systolic Dysfunction is consistently diagnosed after PDA device closure in adults.[42,72] with the incidence of 11% to 22.8% of adult patients after PDA closure.[53,73] The Proposed Mechanism of LV dysfunction after device closure is likely due to a reduction in preload and an increase in afterload, resulting from the elimination of the low-resistance pulmonary circulation.[74] Various Studies report[42,72] variable outcomes after LV systolic dysfunction. Some showed incomplete recovery of Left Ventricular Ejection Fraction (LVEF) and others reported no significant difference between baseline and follow-up LVEF.[40] According to a study by Agha et al., PDA diameter is a predictor of post-closure LV systolic[75]

CONCLUSIONS

Adult PDAs present distinct anatomical and hemodynamic challenges that influence therapy choice and procedural strategy. Transcatheter closure is the preferred first-line approach for most adult PDAs, offering lower morbidity than surgery, though careful preprocedural assessment, accurate sizing (often with balloon techniques), and awareness of calcification/ tortuosity are essential. Long-term outcomes are generally favorable, but adult patients-especially those with long-standing shunts, calcification, or PAH-require individualized assessment and follow-up for LV function and residual flow.

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