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Review Article

An Overview on Patent Ductus Arteriosus in Children

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ABSTRACT

Background: Patent ductus arteriosus (PDA) accounts for 5–10% in all congenital heart diseases in term infants. It is one of the most prevalent congenital heart problems. PDA is more common in preterm newborns and has an inverse relationship with gestational age and weight. The fetus's normal development depends on the preservation of ductal patency. On the other hand, prolonged patency of the ductus arteriosus (DA) in a newborn is linked to a high rate of morbidity and death. Normally, the DA constricts at birth, causing intraluminal ischemia hypoxia, which ultimately causes the ductus to close and remodel. PDA is typically linked to immaturity in preterm infants, although it is typically associated with a functional impairment in mature infants. Prematurity modifies the normal physiological factors that contribute to closure, such as reduced prostaglandins and oxygen tension. Murmurs, tachycardia, bounding peripheral pulses, congestive heart failure, and its accompanying symptoms are clinical indicators of ductal patency. Since symptoms may not usually manifest, diagnostic imaging is essential if a PDA is suspected based on clinical criteria.
Conclusion: There are now three approaches for managing PDA: medicinal intervention, surgical ligation, and fluid restriction and diuretics (when clinically appropriate). The administration of intravenous indomethacin or ibuprofen lysine can result in pharmacologic closure. While the efficacy of both medications is comparable, ibuprofen lysine has shown a better safety profile than indomethacin, especially when it comes to renal consequences.
Keywords: Patent ductus arteriosus; Congenital heart; Children.

INTRODUCTION

The neonatal persistence of a conduit connecting the aorta to the pulmonary artery is known as patent ductus arteriosus (PDA). This shunt, which closes by two to three days of life in full-term neonates, is crucial to fetal circulation. Persistent ductus can cause systemic hypoperfusion and troublesome pulmonary overcirculation. Preterm birth is the main risk factor for PDA; its occurrence is inversely correlated with gestational age. According to estimates, the ductus arteriosus (DA) will persist in 80% of infants born between 25 and 28 weeks gestation [1].

Additional risk factors include septicemia, phototherapy, respiratory distress, excessive fluid delivery, and furosemide therapy. Long-term morbidities linked to patent ductus arteriosus include retinopathy of prematurity (ROP), intraventricular hemorrhage, renal impairment, diastolic cardiac dysfunction, bronchopulmonary dysplasia, and necrotizing enterocolitis (NEC) [2].

Because newborn care has significantly improved, patent ductus arteriosus is rarely a direct cause of death. The DA's function in a clinical setting differs greatly. In order to supply oxygen to the lower limbs during fetal life, the DA permits blood to circumvent the high-resistance pulmonary circulation. A patent ductus is necessary for the continuation of blood flow to the lungs following birth in cases of ductal dependent congenital heart abnormalities throughout the interim until surgical repair can be performed [3].

On the other hand, pulmonary overcirculation may result from a chronic PDA in a patient with otherwise normal cardiac architecture.

When the shift from fetal to neonatal circulation was thoroughly studied in the early 1900s, this association was found. Neonates with a PDA were identified by the presence of clinical symptoms such as cardiomegaly on radiograph, pulmonary edema, murmur, and bounding pulses even before the widespread use of echocardiography. [4]

1938 saw the execution of the first surgical ligation did not occur until 1972, when doctors Elliot and Starling accurately described the function of prostaglandins in preserving ductal patency. Chloroquine was tested as a prostaglandin intended to relieve these symptoms. The initial attempts at pharmacologic treatment antagonist in 1976, and by 1977, newborns with a significant left-to-right shunt were receiving indomethacin in modest trials. PDA is being treated with both medication and surgical ligation today. The best course of treatment, though, and when to start it are still up for debate. [3]

To encourage PDA closure, a variety of prostaglandin antagonists are utilized, and less invasive surgical methods are becoming accessible. Furthermore, there is controversy regarding the necessity of treatment and when it is best to begin treatment because both treated and untreated PDA are linked to morbidities. [4]

Physiology of Fetal Circulation and Postnatal Period

Because the unexpanded lungs and the pulmonary arteries have a significantly higher vascular resistance than the ductus and the descending aorta, only 7% of the combined ventricular output enters the pulmonary circulation in the intrauterine circulation, making the patency of the DA crucial. Because of placental circulation, there is no systemic resistance and the ductus, which is as big as the aorta, offers no resistance. The muscles' vasodilatory response to low blood oxygen tension, prostaglandins produced locally and in circulation, and nitric oxide derived from the ductal endothelium all contribute to the patency being maintained. The elevated amounts of prostaglandins (PG E2 and PGI2) in fetal life are caused by a combination of impaired lung tissue clearance and enhanced placental tissue synthesis [5]. They produce muscular relaxation by acting on certain G-protein coupled receptors on the muscle cells. According to Parkerson et al. [2], PGE2 is thought to be the primary endogenous prostaglandin regulating DA patency.

Mature duct is more sensitive to changes in oxygen tension and less susceptible to PGE2, whereas immature duct is more receptive to the vasodilatory effects of prostaglandins and less responsive to oxygen stress. Within 10 to 15 hours of delivery, full-term infants experience the first "functional" closure of the ductus. This is followed by a permanent "anatomic" occlusion, which is replaced over the course of the next two to three weeks by the ligamentum arteriosum, a fibrous ligament. There are several factors that contribute to the functional closure, the primary one being elevated arterial PO2. Arterial PO2 rises significantly as a result of blood oxygenation and lung tissue expansion. [6].

The ductal smooth muscle cells' voltage-dependent potassium channels are inhibited by oxygen, which causes an internal calcium overabundance and vasoconstriction. Reduced levels of circulating PGE2 and a loss of the opposing vasodilatory response result from the removal of the placenta and functional lung tissue with enhanced pulmonary blood flow. A decrease in blood pressure within the ductus lumen causes hemodynamic alterations that further aid in the ductus's functional closure. Following this constriction, the subendothelial region swells, smooth muscle cells move into the area, and the ductal endothelial cells separate. Growth factors are released in response to a considerable hypoxic insult sustained by the endothelium and the innermost layer of smooth muscle cells. [7].

Growth factors cause endothelial cells to proliferate, which results in a large amount of neointimal thickening and infolding. Cells then gradually die and are replaced by fibrous tissue. If the typical reaction is disrupted at birth due to alterations in the oxygen tension or developmental abnormalities in the histopathologic architecture of the ductal tissue, the duct will stay open. The precise mechanics behind the duct's refusal to seal in certain full-term newborns are still unknown, though. [8]

PATHOPHYSIOLOGY

By the eighth week of pregnancy, the distal dorsal sixth aortic arch, from which the DA is derived, has fully developed. Its function is to divert blood from the nonfunctioning fetal lung by means of its attachment to the proximal descending aorta and the major pulmonary artery. The blood with a relatively low oxygen concentration can be transported from the right ventricle to the descending

aorta and ultimately to the placenta, where gas exchange takes place, thanks to this right-to-left shunt [9].

Roughly 90% of the right ventricular output passes via the DA prior to birth. The function of the DA in rerouting fetal circulation in contrast to neonatal circulation is seen in the figure 1. Fetal hydrops may arise from premature closure in the fetus, which is linked to serious morbidities such as right-sided heart failure. After a full-term birth, the DA normally closes in 24 to 72 hours. If the ductus is still open after 72 hours, chronic PDA may be the cause [10].

Low fetal oxygen tension and prostanoids generated from COX's metabolism of arachidonic acid, of which PGE2 produces the most profound ductal relaxation, are the main factors regulating the patency of the DA. PGE2-induced activation of the G-coupled prostaglandin receptor EP4 causes smooth muscle relaxation of the DA. The activation of prostaglandin receptor EP4 initiates a series of actions that culminate in decreased myosin light chain kinase, increased protein kinase A, and buildup of cyclic adenosine monophosphate. These events ultimately lead to DA patency and vasodilation. The preterm ductus is more vulnerable to prostaglandins' vasodilatory actions, which makes ductal closure less successful. Reduced DA sensitivity to prostaglandins and circulating PGE2 levels help induce DA closure in term newborns as delivery draws near.[11]

The DA shuts 24–72 hours following a full-term birth due to decreased prostacyclin (PGI2) and PGE2 circulation, as well as increased oxygen tension. Voltage-dependent potassium channels in smooth muscle are suppressed when oxygen tension rises. Ductal constriction is facilitated by an inflow of calcium through this inhibition. Preterm newborns fail this oxygen-induced constriction, may be because their oxygen-sensing receptors are still developing. The elimination of the placental source and higher metabolism in the newly functioning lung result in lower levels of circulating PGE2 and PGI2. The DA can tighten because there are fewer of these powerful vasodilators in the blood. All of these elements work together to cause smooth muscle constriction, which results in ischemia hypoxia of the DA's inner muscle wall [1].

The luminal area decreases with ductus constriction, leading to thicker vessel wall and blocked flow through the vasa vasorum, the vital capillary network that supplies the vessel's outer cells with nourishment. This may results in an increased diffusion distance for nutrients, such as glucose, glycogen, and adenosine triphosphate (ATP), and oxygen, which creates a nutritional deficit and oxygen starvation that ultimately kills cells. Infants born before their due date have insufficiently deep ductal constriction. As a result, premature babies are less able to withstand smooth muscle hypoxia, which is crucial for inducing the cell death and remodeling needed for the DA to close permanently. Preterm neonates exhibit less extensive inhibition of prostaglandin and nitric oxide as a result of tissue hypoxia than term infants, which may further explain the preterm infant's resistance to DA closure. [12].

The lumen supplies the majority of the nutrients to the DA, while the vasa vasorum also contributes significantly to the ductus's outer wall. The vasa vasorum extends 400–500 μm from the ductus's outer wall and develops toward the lumen. The maximum distance permitted for efficient nutrient transport is known as the avascular zone, and it lies between the lumen and the vasa vasorum (40–500 μm). This avascular zone expands beyond the effective diffusion distance in full-term newborns, which subsequently contributes to cell death. [13].

The avascular zone fails to enlarge enough in preterm newborns, which preserves ductal patency and cell survival. Closure is aided if PGE2 and other prostaglandin levels in the blood are lowered via COX inhibition. The figure 2 shows the aforementioned variations in the thickness of the ductal vessel wall in fetuses, term neonates, and preterm neonates. Together with other inflammatory mediators, vascular endothelial growth factor and transforming growth factor beta—both of which promote endothelial proliferation—help the DA remodel into the non-contractile ligament known as the ligamentum arteriosum in response to the nutritional deficiency and ischemic hypoxia. [2]

CLINICAL PRESENTATION AND ASSOCIATED COMPLICATIONS

A PDA has a distinct pattern of clinical presentation and has the potential to cause serious consequences. Physical indicators should be noted during assessment even though they do not always indicate the hemodynamic significance of the ductus. The gastrointestinal (GI), cardiovascular, renal, and respiratory systems exhibit the most prevalent symptomology. [2]

Cardiovascular

Wide pulse pressures, an active precordium, hypotension, and a distinctive coarse systolic murmur at the left sternal border are the most prevalent cardiovascular symptoms of PDA. Wide pulse pressures are caused by compromised systemic

perfusion during diastole, and the distinctive murmur is caused by turbulent blood flow through a patent ductus. As pulmonary overcirculation increases blood return to the left side of the heart during diastole, elevated preload results in an active precordium. [14].

During systole, blood flow is diverted from the aorta to the pulmonary artery, which affects organ perfusion and results in systemic hypotension. During diastole, the decrease in perfusion pressure is most noticeable. Notably, aortic diastolic pressure affects coronary artery perfusion and can cause some myocardial ischemia. The GI and renal systems' symptoms are also brought on by this ductal theft. [15].

Renal

PDA can cause oliguria (urine production <1 mL/kg/h), metabolic acidosis due to renal hypoperfusion, and fluid retention in the cardiopulmonary circuit. Increased creatinine and hyponatremia are possible related test results. Doppler investigations may reveal diastolic flow to the renal arteries that is missing, reduced, or reversed [16].

Renal hypoperfusion triggers the release of renin from the kidney's juxtaglomerular cells, which sets off the compensatory renin-angiotensin-aldosterone pathway. Renin initiates a signaling cascade that causes peripheral vasoconstriction in an effort to hold on to water and salt and increase intravascular volume. This compensatory route exacerbates and increases cardiac circulation in the case of extensive PDA with left-to-right shunting, but renal perfusion stays optimal. [17]

Respiratory

Pulmonary edema, a protracted requirement for assisted breathing, pulmonary bleeding, and elevated rates of bronchopulmonary dysplasia (BPD) are indicative of pneumonia-related dysplasia (PDA). These aftereffects stem from lower pulmonary resistance leading to pulmonary overcirculation. As postnatal circulation transitions, decreasing resistance leads to increased flow from the aorta to the pulmonary artery. Because surfactant insufficiency and low serum oncotic pressures facilitate faster interstitial fluid accumulation, preterm infants are more susceptible to overcirculation and pulmonary edema. Alveolarization is hampered by prolonged breathing and oxygen therapy, and the likelihood of extended hospital stays is raised. [18]

Gastrointestinal

Necrotizing enterocolitis (NEC) risk is elevated in cases of PDA-related gastrointestinal problems, such as eating intolerance. These consequences originate from shunting blood flow away from the GI tract and systemic hypoperfusion. It is thought that during times of hypoperfusion, the stomach becomes ischemic. The gut then suffers from harm from reperfusion and reoxygenation, and the risk of NEC rises. When clinical indications of a PDA are observed, a diagnosis must be determined in order to guide treatment recommendations. [19]

DIAGNOSIS

Numerous indicators that are not diagnostic may point to the existence of a PDA. These indicators consist of the results of chest radiographs, laboratory markers, and assessment aspects. The cardiac echocardiography, which shows the direction of shunting and pressure variations in different chambers, is still the gold standard for diagnosing PDA, nevertheless. [17]

1. Non-specific diagnostics:

Physical evaluation signs such as decreasing respiratory status, widening pulse pressures, hypotension, feeding intolerance, and a characteristic holosystolic murmur may raise strong suspicions of a PDA. As was previously mentioned, abnormal blood flow is the cause of these characteristic clinical symptoms [16].

Elevated C-reactive protein, deteriorating blood gases, and metabolic acidosis are common test values. Furthermore, it has been demonstrated that increases in a number of biochemical markers, such as cardiac troponin T, B-type natriuretic peptide, and the segment of the amino terminal B-type natriuretic peptide, correspond with the existence of PDA. These indicators are not now widely utilized to determine diagnosis or treatment; their clinical value is still being investigated. [14].

A patient with PDA may have cardiomegaly, left atrial dilatation, and pulmonary congestion on their chest radiograph. These results, however, do not provide a conclusive diagnosis. Infants with aorticpulmonary collateral vessels, an aortic-pulmonary window, a right pulmonary artery originating from the aorta, or a fistula between the coronary artery and the right ventricle may present with similar physical and radiological characteristics. Doppler echocardiography is necessary for a conclusive diagnosis. [18]

2. Doppler Echocardiography

Within the DA, Doppler echocardiography is the gold standard for evaluating hemodynamics and

patency. Doppler studies work by using the reflection of sound waves from the heart's myocardium and blood as it passes through it to show the direction and speed of blood flow visually. This makes it possible to evaluate relevant factors such ductal diameter, ductal flow patterns, left atrial size in relation to the aorta, left ventricle filling pressure, and blood flow patterns to the middle cerebral and renal arteries. [2]

Blood flow velocity and ductal diameter measurements enable the estimate of total blood flow across the DA. According to Kluckow and Evans (quoted in Wylie), the strongest early predictor of hemodynamic importance in infants under 29 weeks of age was a ductal diameter larger than 1.5 mm, measured in the first 31 hours of life. The sensitivity and specificity of this indicator are 83% and 90%, respectively, within a confidence interval of 71%-94%) and 81%-98%), respectively. [13].

The bulk of the most recent studies set the treatment threshold at 1.5 mm or 1.4 mm in diameter based on echocardiographic parameters. When the ductus diameter and blood flow velocity are taken into account combined, the significance of the two increases. While fast flow combined with a high ductal diameter implies more severe hemodynamic impairment, quick flow through a small ductus may imply a relatively minor shunt. Echocardiography is commonly used to evaluate additional parameters, such as ductal flow patterns, left atrial size in relation to the aorta, left ventricular filling pressure, and absence or reversal of enddiastolic flow to the superior mesenteric artery or middle cerebral artery. [1].

Unrestrictive, pulsatile flow across the ductus is indicative of the most severe hemodynamic impairment. A low flow during end diastole, which indicates that the pressures in the pulmonary artery and aorta are almost similar at this stage of the cardiac cycle, is an example of this pattern. The left atrium's dilation and filling pressure provide insight into the degree of pressure overloading the left side of the heart. If left untreated, the larger, more pronounced ductal shunting left atrium might result in greater pulmonary overcirculation and left-sided heart failure. The greatest threat to end-organ perfusion is indicated by the absence or reversal of end-diastolic flow to the superior mesenteric artery or middle cerebral artery.[17]

MANAGEMENT

Closing the ductus or continuing the patient with more evaluations is the initial choice in the care of an adult patient with PDA.[20]

According to the most recent guidelines from the American College of Cardiology, American Heart Association, and European Society of Cardiology, nearly all PDAs should be closed, with the exception of patients with pulmonary arterial hypertension (PAH) who have net right-to-left shunting (class III) and small, silent ducti without audible murmurs. The following are class I indications: development of PAH with pressure and resistance still less than two-thirds of the systemic levels; previous history of endarteritis; shunt associated with evidence of volume overload on the left atrium or left ventricle (left atrial or left ventricular enlargement). Because percutaneous closure carries a very low risk and a high success rate, small PDAs with normal PA pressures, normal heart size, and a shunt ratio less than 1.5/1 may be considered for closure (class IIa). Alternatively, they may be monitored with follow-up assessments every three to five years. [21]

When pulmonary pressures in patients with severe PAH exceed two-thirds of systemic levels or are even equivalent to systemic pressures, there is a quandary about the hazards involved. If the net shunt is still left to right and the QP/QS ratio is greater than 1.5/1, the ductus can be closed cautiously in these cases. Alternatively, one can show a decrease in PA pressures by using vasodilator therapy (no vascular reactivity) or balloon occlusion during cardiac catheterization (Class IIa). Although small studies have demonstrated that pulmonary arterial pressures would eventually decline, concerns persist if PAH is caused by a disorder other than PDA, such as primary PAH, and is unrelated to PDA [22].

Techniques for Closing PDAs

Unlike in children, the adult ductal tissue in PDA patients does not respond to COX inhibitors such as indomethacin. Consequently, percutaneous or surgical methods continue to be the two main methods of closure for PDA. [23]

Intradermal Techniques

Although surgery is still the treatment of choice for most premature infants with patent ductus arteriosus (PDA), transcatheter techniques have largely supplanted surgery for closure of PDA in children and adults. The ideal technique for adult patients (class I) is percutaneous closure of the PDA because adult surgery involves a higher perioperative risk because of ductal friability, calcification, and related concomitant diseases such as aortic atherosclerosis or coronary artery disease. Even in cases where cardiac procedures are required for other related cardiac diseases, device closure remains the preferred approach. [24].

In 1967, Portsmann and colleagues described the first transcatheter closure of the PDA; using a doubleumbrella device, Rashkind and Cuaso reported the same experience in 1979. Based on data from a multicenter registry, the US Food and Drug Administration approved the Amplatzer duct occluder device in 2003. Since then, coils in PDAs less than 3 mm and Amplatzer duct occluders for PDAs between 3 and 12 mm have been the two most widely utilized devices to close PDAs. It has been reported that Amplatzer (St. Jude Medical Inc., MN) septal occluders can be used to close type B PDAs that are shorter than 3 mm. Countries outside of the US can choose from additional sizes up to 16 mm. Trials are being conducted on the novel devices, such as ADO II (St. Jude Medical Inc., MN), because ductal geometry and size are highly diverse [25].

The fundamental method for percutaneous closure of PDA involves inserting coils or occluder devices down the length of the artery using a delivery catheter that is passed through either the aorta or the pulmonary artery. The Amplatzer duct occluder is now the preferred device for moderate-to large-sized PDAs in adult patients. A 7-Fr sheath is inserted into the femoral vein, and a 5- to 6-F sheath is positioned in the femoral artery, all while the patient is conscious sedation and local anesthesia is used. [26, 27]

In order to compute the pulmonary and systemic blood flows and ascertain the pulmonary and systemic vascular resistances, pressure readings and oxygen saturations are first taken from the right and left sides of the heart. The PDA's size and shape are then determined by performing an aortic angiography in a 90-lateral projection using a pigtail catheter in the proximal descending aorta. This allows the surgeon to choose the right kind and size of closure device. [28].

The measures for the most prevalent type A PDA include the vertical length of the ampulla, the length from the pulmonary end to the aortic end, and the narrowest diameter. Typically, the chosen device has a pulmonary end 2 mm bigger than the PDA's narrowest diameter. [29]

The pulmonary artery is bridged over using a multifunctional catheter and wire, and the catheter is then progressed into the distal descending aorta. The multifunctional catheter is replaced with the PDA delivery sheath using a strong guide wire. Pulling back on the sheath reveals the retention disc once the chosen device connected to the delivery cable is gradually advanced to the sheath's tip. [30, 31]

Now, the device and sheath are drawn back into the aortic ampulla together. Once the retention disc is securely fastened within the ampulla, the sheath is pulled back, allowing the remaining portion of the device to be positioned along the PDA's length. To verify the residual shunt and device position, an aortic angiography is conducted ten minutes later while the delivery cable is still in place. The delivery system is withdrawn, the device is freed, and the cable is retracted into the delivery sheath if the outcomes are satisfactory. To confirm immediate device stability, a follow-up aortogram may be necessary 10 to 15 minutes after the initial release. [32].

Usually, a chest radiograph and transthoracic echocardiography (TTE) are used to check the device location before the patient is released from overnight observation. Six months following the surgery, a TTE is carried out to document full closure of the PDA in addition to routine clinical follow-up. Should echocardiography still reveal any residual shunt, it ought to be done again after a year. Prophylactic treatment for subacute bacterial endocarditis should be administered for at least six months, and it should be continued until full closure is confirmed. [20]

There are a few nonrandomized comparative trials and a clinical series that provide the majority of the evidence about the effectiveness of percutaneous closure devices for PDA. With instantaneous closure at the moment of implantation at 76%, at day 1, at 89%, and at 6 to 12 months at 99% by echocardiography, the overall success rate of ADO implantations is 99%. Major complications are incredibly rare, accounting only 1 out of every 439 cases of mortality. [10].

Major events were recorded in 2.3% (10/439) of patients in the pivotal registry who had device embolization in 2 out of 439 individuals. In a day, the majority of patients are sent home. For closing all PDAs, the Amplatzer duct occluder is not the best option because of the differences in PDA sizes and designs. Nonetheless, most patients will undoubtedly be able to get percutaneous PDA care thanks to the modifications made to the Amplatzer device that are presently undergoing testing and other novel devices that are being developed. [33]

Surgical approaches

Since its introduction by Gross and Hubbard in 1939, surgical ligation has been the gold standard for treating PDA. Since safer catheter-based therapy is now available, surgery is only performed in extremely rare cases of very large PDA, atypical ductal anatomy, such as ductal aneurysms, or

substantial endarteritis or abscess at the PDA site, which renders them unsuitable for percutaneous closure. [34]

The methods include video-assisted thoracoscopic ligation, median sternotomy, and lateral thoracotomy. The incidence of successful closure is about 100%; however, in comparison to percutaneous closure, the surgical morbidity and duration of stay are higher. Damage to the phrenic or recurrent laryngeal nerves is one of the surgical complications. [2]

Long-term Management

Endocarditis prevention and routine follow-up examination are not recommended in individuals with silent PDA. It is permissible to monitor individuals with minor PDA (shunt ratio less than 1.5 to 1) at 3- to 5-year intervals with repeat TTE examination if there is no evidence of left atrial or ventricular enlargement. Patients who have percutaneous closure and are found to have no residual shunt after six months are not in need of any more routine monitoring. Patients with moderate-to-large PDAs may remodel left ventricular size if they receive treatment for left-sided volume overload and/or congestive heart failure; however, even after a successful percutaneous or surgical closure, these patients may still need to have their left ventricular dysfunction monitored and managed. [27]

Similar to this, persons who present with pulmonary hypertension might not regress the pulmonary arterial abnormalities and should be regularly monitored in adult congenital clinics with specialists in pulmonary hypertension, using the most recent therapy techniques for pulmonary hypertension as needed. Adult experts with expertise in congenital cardiac disease must monitor patients with Eisenmenger syndrome. [15]

Antibiotic prophylaxis is not advised by current guidelines for untreated PDA. However, if the full closure is confirmed by TTE at six months, antimicrobial prophylaxis is advised for both surgical and percutaneous closure for six months following the surgery. Future subacute bacterial endocarditis prevention will still be necessary for patients whose residual shunt was discovered by TTE. [2]

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Competing interests

The authors declare that they have no competing interest.

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