

PATENT DUCTUS ARTERIOSUS

Definition

The patent ductus arteriosus (PDA) connects the proximal descending aorta to the roof of the main pulmonary artery near the origin of the left pulmonary artery.¹ This fetal structure normally closes spontaneously within the first days after birth. It rarely closes after infancy.

Epidemiology

PDA accounts for approximately 10% of all congenital heart disease in the adult with a 2:1 female to male ratio.

Aetiology

PDA occurs in genetic syndromes with chromosomal aberrations (such as trisomy 21 and 4p syndrome), single-gene mutations (such as Carpenter's syndrome and Holt-Oram syndrome), and X-linked mutations (such as incontinentia pigmenti).^{1,2} Although most cases of PDA are seemingly sporadic, many may be due to multifactorial inheritance. In a family having one sibling with a PDA, there is a 3% chance of a PDA in a subsequent offspring. Rubella infection during the first trimester of pregnancy, children born in high altitude and prematurity are associated with a high incidence of PDA.¹

Pathophysiology and Natural History

The shunt flow depends on the ductal resistance and the pressure gradient between the aorta and the pulmonary artery.³ Left-to-right shunting through the ductus arteriosus results in increased pulmonary fluid volume and left atrial and ventricular volume overload. If the ductus is large enough, the diastolic runoff may result in "steal" phenomenon with impaired coronary perfusion. When pulmonary vascular resistance approaches and exceeds systemic vascular resistance, ductal shunting reverses and becomes right to left with eventual development of Eisenmenger's syndrome.² Patients with Eisenmenger's syndrome are cyanotic and typically have **differential cyanosis** (cyanosis and clubbing of the toes but not the fingers because the right-to-left ductal shunting is distal to the subclavian arteries). Cyanosis may be more profound when systemic vascular resistance is reduced, such as in hot weather or after exercise. **Infective endarteritis** and **aneurysm of ductus arteriosus** are the most common complications. Rarely, the ductus arteriosus aneurysm may rupture or present with symptoms of a thoracic mass, including hoarseness due to left vocal chord paralysis from recurrent left laryngeal nerve impingement and left bronchial obstruction. In previous series, one third of patients with not repaired PDA died of heart failure, pulmonary hypertension or endarteritis by the age of 40 years and two thirds died by the age of 60 years.⁴

Presentation

The clinical significance of the PDA depends on its size. Patients may be completely asymptomatic with a heart murmur or present due to exercise intolerance, endovascular infection or atrial fibrillation. Although most patients compensate well even with a moderate left-to-right shunt and remain asymptomatic during childhood, they may develop congestive heart failure or Eisenmerger syndrome in adulthood.¹

Physical Examination

Patients with **very small** patent ductus have no abnormal physical findings. A continuous murmur may be heard.

In **moderate** or **large** ductus:

Machinery murmur Continuous murmur in upper left sternal border below the left clavicle. It is louder in systole, may radiate into the back, and a thrill may be present. **S₂** may be inaudible.

LV apex prominent and collapsing **peripheral pulses**.

Pulmonary ejection click and **PR** appear as pulmonary hypertension develops.

Investigations

Chest Radiograph may be normal or display cardiomegaly (LA and LV enlargement) with increased pulmonary vascular markings. The main pulmonary artery is enlarged, and in older adults with pulmonary hypertension calcification of the ductus may be evident.

ECG Normal or AF, LV hypertrophy, and LA enlargement in patients with moderate or large ductus shunts.

Echocardiography Color Doppler is used for detecting the presence of a PDA and estimating the degree of ductal shunting. In patients with high pulmonary vascular resistance and PDA with low velocity or right-to-left flow, the ductus arteriosus may be very difficult to demonstrate. Findings such as septal flattening, unexplained right ventricular hypertrophy, and high-velocity pulmonary regurgitation should prompt a thorough investigation for a PDA.

Contrast echocardiography may be helpful.

Measurement of **oxygen saturation** in feet and both hands in adults to assess for the presence of right-to-left shunting (Table 1).

Cardiac magnetic resonance is useful in patients with unusual PDA geometry and associated abnormalities of the aortic arch.

ECG-gated multi-detector row computed tomography can also visualize PDA as two dimensional and three dimensional images, and, when combined with electrocardiogram (ECG)-gated acquisition.⁵

Cardiac Catheterization Diagnostic cardiac catheterization for uncomplicated PDA with adequate noninvasive imaging is not indicated (Table 1). Detailed assessment of the ductal anatomy by angiography is essential for the selection of the proper device size for transcatheter closure. In patients with elevated pulmonary artery pressure, assessment of pulmonary vascular resistance and its response to vasodilating agents may be helpful in determining possibility of ductus closure.

Therapy

PDA is associated with various complications of prematurity and cyclooxygenase-inhibitors such as high-dose ibuprofen are the first-line intervention for closure of the PDA.^{6,7}

Indications for Closure of PDA are presented in Tables 2 and 3 and Figure 1. Routine closure of even small PDAs is now recommended by most experts. The rationale is that endarteritis of clinically silent PDA has been reported and device closure is now effective and safe.^{8,9} In patients with borderline pulmonary arterial hypertension, a post-trial occlusion systolic pulmonary:systemic pressure ratio >0.5 is a useful hemodynamic parameter for identifying patients who have a high risk of postprocedural pulmonary arterial hypertension after transcatheter patent ductus arteriosus closure.¹⁰ Patients with PDA and pulmonary vascular disease who are considered unacceptable candidates for definitive closure may be managed with pulmonary vasodilating agents such as chronic oxygen, PGI₂, calcium channel blockers, endothelin antagonists, and phosphodiesterase inhibitors. One strategy in such patients is to accomplish partial closure of the ductus by surgery or transcatheter techniques, to make it "restrictive" but not completely closed, followed by long-term therapy with pulmonary vasodilating agents. If in follow-up the pulmonary vascular resistance decreases, then complete closure may be considered.¹ Follow-up is recommended every 3 to 5 years for patients with a small PDA without evidence of left-heart volume overload.

Transcatheter closure has become the treatment of choice in children and adults, especially in cases of calcified ductus arteriosus with increased pulmonary vascular resistance. The most commonly used occluders are the Nit-Occlud coil occlusion system and the Amplatzer duct occluder. Complications of transcatheter closure such device embolization of the patent ductus are rare. Other rare complications are flow disturbance in the proximal left pulmonary artery or descending aorta from a protruding device, hemolysis from high-velocity residual shunting, and infection.⁸⁻¹⁰

Surgical repair remains the treatment of choice for the rare very large ductus.

Early treatment of preterm infants with ibuprofen is not associated with a lower incidence of death or moderate or severe bronchopulmonary dysplasia at 36 weeks of postmenstrual age than placebo.¹¹

Infective endocarditis prophylaxis is not recommended any more for unrepaired PDAs.

For closed PDAs, prophylaxis is recommended for 6 months after the procedure (see also Chapter 1).

Pregnancy does not impose a significantly increased risk on patients without pulmonary hypertension.^{12,13}

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