Management of Patent Ductous Arteriosus: A Short Review

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ABSTRACT

Patent ductus arteriosus (PDA) is one of the most common congenital heart defects. Failure of the ductus arteriosus (DA) to close within 72 hours after birth result in PDA. Clinical signs of ductal patency are murmur, tachycardia, bounding peripheral pulses, and congestive heart failure. Complications of a persistently patent DA after birth are heart failure, renal dysfunction, necrotizing enterocolitis (NEC) and altered postnatal nutrition and growth. Also risk of developing of chronic lung disease. Pharmacologic closure can be done by administration of intravenous indomethacin or ibuprofen. Other management strategies are cath intervention, and surgical ligation.

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Introduction
The ductus arteriosus is a normal fetal artery connecting aorta and pulmonary artery [Figure 1]. Every baby is born with a ductus arteriosus. After birth, the ductus arteriosus is no longer needed and it usually closes within three days. In term infant, The PDA results if it fails to close after birth within 72 hours in term infant and seven days in preterm infant. Some children can have associated heart defects along with the PDA. Normally the heart’s left side only pumps blood to the body, and the right side only pumps blood to the lungs. In a child with PDA, extra blood gets pumped from aorta into the pulmonary arteries. Small PDAs often close on their own within the first few months of life. If the PDA is large, the extra blood being flowing into the lung arteries makes the heart and lungs work harder and the lungs can become congested. This research article give insight into the review of incidence, sign and symptom, investigation and management of PDA.

Sign and Symptoms: The sign and symptoms of a PDA results from the left-to-right shunt causing pulmonary edema, which may cause respiratory failure [Table 1]. Low diastolic pressure contributes to hypotension and systemic hypoperfusion. Depending on the organ affected, hypoperfusion may lead to renal dysfunction, feeding intolerance, intraventricular haemorrhage and congestive heart failure. Neonates may also experience multiple episodes of apnea and exhibit ventilator dependence.7,8

Investigations: Chest X-ray shows LV type apex due to volume overload and pulmonary plethora. 2D Echocardiography easily detect PDA in majority of patients. Sometimes the older patients with severe pulmonary hypertension may require cardiac catheterisation study to decide whether the pulmonary hypertension is reversible or irreversible and to decide whether PDA is feasible for device closure or not. Cardiac CT is helpful to know any additional cardiac defect.

Treatment: Treatment of congestive cardiac failure due to PDA is same that of any other congestive cardiac failure fluid restriction and ionotropicsupports. Main treatment options are pharmacologic management, cath interventions [device closure or coil embolization] and surgical ligation.

Pharmacologic Management
Currently only two nonselective COX inhibitors indicated in the closure of PDAs. Both IV indomethacin and IV ibuprofen lysine are equally effective in the closure of PDA, achieving closure rates of 75%-93%.9 Responsiveness to PGE2 decreases with increased age; therefore, delaying treatment may result in diminished efficacy of COX inhibition. The side effects of indomethacin like renal toxicity, neurological impairment and necrotising enterocolitis are less with use of ibuprofen lysine10,11.

Cath Intervention
Cath intervention is reserved for patients who cannot be managed pharmacologically. Most children can have the PDA closed by inserting catheters into the blood vessels of leg to reach the heart and the PDA, and a coil or other device can be inserted through the catheters into the PDA like a plug [Figure 2].

Table 1: Symptoms and signs of PDA.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Signs</th>
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<tbody>
<tr>
<td>Episode of apnea</td>
<td>Crescendo systolic murmur</td>
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<tr>
<td>Respiratory distress</td>
<td>Wide pulse pressure</td>
</tr>
<tr>
<td>Unexplained metabolic acidosis</td>
<td>Hyperkinetic apex</td>
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<tr>
<td>Poor weight gain</td>
<td>Cardiomegaly</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Hepatomegaly</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>Diastolic hypotension</td>
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Surgery

Surgical ligation is reserved for patients who cannot be managed pharmacologically and by cath intervention. Surgical repair of a PDA typically involves either ligation or a combination of ligating and dividing the DA using surgical clips or nonabsorbable sutures via left posterior lateral thoracotomy. Video-assisted thoracoscopic surgery permits surgeons to safely and effectively ligate the DA with minimal invasiveness.

Conclusion

If the PDA is large, the child may breathe faster and harder than normal. Infants may have trouble feeding and growing at a normal rate. High pressure may occur in the blood vessels in the lungs because more blood than normal is being pumped there. Over time this may cause permanent damage to the lung blood vessels. Most children can have the PDA closed by pharmacological management specially premature infants, some may require cath intervention and device or coil closure. If surgery is needed, an incision is made in the left side of the chest, between the ribs. The ductus is closed by tying it with suture (thread-like material) or by permanently placing a small metal clip around the ductus to squeeze it closed. The long-term outlook is excellent, and usually no medicines and no additional surgery or catheterization are needed.

References


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