3. PATENT DUCTUS ARTERIOSUS (PDA)

The ductus arteriosus is a normal pathway in the fetal circulatory system, connecting the left pulmonary artery with descending aorta. During the fetal life, blood flow is shunted away from lungs through ductus arteriosus directly into the systemic circulation. Functional closure of ductus arteriosus usually occurs spontaneously during first 10-15 hours after birth. Permanent closure occurs within 5-7 days in most infants but may take up to several weeks.

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If the closure of ductus arteriosus does not occur even by 2-3 weeks of age it is known as Patent ductus arteriosus.

Incidence
- Most common defects of heart.
- It is twice more common in females as compared to males.
- PDA is common in infants who weigh less than 1500gm & accounts for 5-10% of all congenital heart diseases.

Risk factors.
* Premature birth
* Family history & other genetic conditions
* Rubella infection during pregnancy.
* Being born at a high altitude.
* Being female.

Complications
* Pulmonary hypertension
* Heart failure
* Endocarditis.

Pathophysiology
- Failure of closure of ductus arteriosus leads to shunting of blood from high pressure aorta to low pressure pulmonary artery (left to right shunt).
- This results in increased blood flow to pulmonary tree and increased blood return

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to left side of heart causing volume loaded left ventricles.

Clinical Features
The clinical features of PDA dipend on the size of PDA & age of infant. Preterm infants are usually symptomatic earlier. In preterm infants pulmonary vascular resistance falls more rapidly, allowing more left to right shunting & their myocardium is immature & less able to handle the extra load so they go into congestive heart failure easily.

In term infants symptoms depend on the size of ductus. The term infants with small PDA are usually asymptomatic, whereas those with large PDA may present with congestive heart failure. These children may have growth retardation & easy fatigability.

Symptomatic manifestations are
- Bounding pulse
- Tachypnoea
- Pulsation in the neck & frequent respiratory infections.
- There is increased systolic pressure & low diastolic pressure with wide pulse pressure.
- Feeding difficulties, slow weight gain are common features of PDA

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Diagnosis

The diagnosis of patent ductus arteriosus is made on the following basis:

i) Cardiac examination :- Systolic murmur or continuous murmer may be present, which is best heard in second to third left intercostal space. With large PDA, a diastolic rumble & a gallop may be heard. Pulse is usually bounding in these children

ii) Electrocardiogram :- The ECG is usually normal. However, it may show left ventricular hypertrophy & / left atrial dilatation in older children.

iii) Chest radiograph :- The chest radiograph shows increased pulmonary vascularity with normal or increased heart size.

iv) Echocardiogram :- A color flow 2D echo helps in visualization of patent ductus arteriosus. With a Doppler, the amount of blood flow across the PDA can be estimated.

Therapeutic Management

- Medical management :- In symptomatic patients with PDA, Indomethacin, a prostaglandin inhibitor a prostaglandin inhibitor. By inhibiting prostaglandin, indomethacin allows PDA to close. This agent is more effective in preterm than term infants & should be administered before the age of 10 days. It is given orally

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or intravenously in dose of 0.2 mg/kg/dose may be repeated up to three times, at an interval of 12 to 24 hours.

Supportive care is provided with rest, adequate intake of calories for weight gain & normal growth & development.

- Surgical Management:- Ligation of PDA via lateral thoracotomy. If a closed heart intervention is performed. It is preferably between 3-10 years of age in a symptomatic patient & in asymptomatic patients it should be done irrespective of age.

(B) Disorders With Decreased Pulmonary blood Flow:

1. COARCTATION OF AORTA.

Coarctation of aorta is a discrete narrowing of aortic arch, usually in juxtaductal position (in the region of ductus arteriosus & left subclavian artery).

Incidence & Types.

It constitutes approx. 7% of all congenital heart defects.

It is of 2 types:

a. Infantile or preductal Type:- There is constriction of aorta between left subclavian artery & ductus arteriosus.

b. Post ductal type:- There is constriction

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at or distal to ductus arteriosus.

Pathophysiology
* Coarctation of the aorta imposes significant afterload on the left ventricle (LV) which results in compensatory ventricular hypertrophy.
* As the ductus (aortic end) constricts, the left ventricular afterload rapidly increases, with a resultant increase in left ventricular pressures (systolic & diastolic).

Risk factors
* Bicuspid aortic valve
* Patent ductus arteriosus
* Holes in the wall between left & right sides of the heart.
* Aortic valve stenosis or regurgitation
* Mitral valve stenosis or regurgitation

Complications:
* Aortic stenosis
* High blood pressure
* Stroke
* Aneurysm
* Aortic rupture or tear (dissection)
* Heart failure

Clinical Features
There are two groups of patients with coarctation:
(1) Those who are symptomatic in infancy.

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ii) Those who remain asymptomatic & are diagnosed during routine physical examination in later years.

Symptomatic children present with the following features:

* Increased blood pressure in the upper part of body, resulting in headache, dizziness, fainting, nose bleed (epistaxis) & cerebrovascular accident.
* Occassionally the child may present with complaints of weakness or pain in their legs on exercise. Their legs may be colder than arms.
* Mottling is often seen in the lower extremities
* Infants usually present with CHF & failure to thrive. Symptoms include respiratory distress, poor weight gain, feeding problems, irritability and tachycardia.

Diagnostic Evaluation.

i) Cardiac examination:- Systolic murmur may be heard along the left mid to upper sternal border that radiates to the back.

ii) ECG shows left or right ventricular hypertrophy

iii) Echocardiogram:- The presence of a coarctation & the degree of narrowing as well as presence of other cardiac defects may be determined by the

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echocardiogram.
iv) MRI & Cardiac catheterization are useful in clearly defining the area & extent of narrowing.

Therapeutic Management
* Coarctation may be surgically corrected using numerous approaches which are as follows:
• End to end anastomosis :- In this procedure the narrowed portion of aorta is removed & two normal parts are joined.
• Subclavian flap aortoplasty :- In this procedure a longitudinal incision is made in the aorta across the coarctated site & continued to the end of distally divided left subclavian artery. The left subclavian artery is used as a patch or flap to increase the diameter of the aorta.
• Patch aortoplasty :- In this procedure coarctated area is excised & a patch graft is placed on aorta to widen it.
• Balloon aortoplasty :- It is a special procedure in which a balloon catheter is introduced into the aorta during cardiac catheterization & inflated at the site of coarctation to relieve the obstruction.
* Medical management for CHF & HTN is needed. Continued antibiotic prophylaxis is necessary for certain procedures and

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cardiology follow up, at least every 1-2 years is recommended.