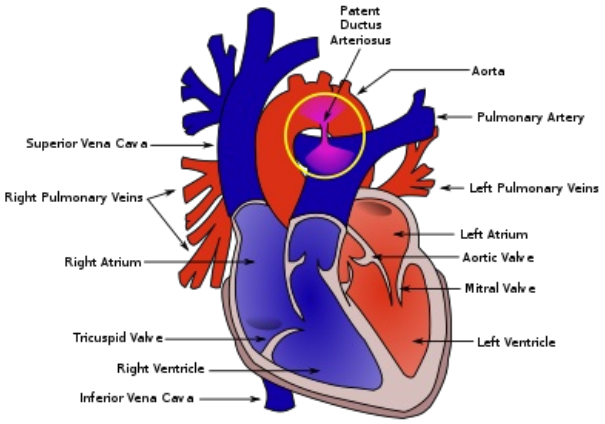
Open Botall's soul

Template:Infobox - VVV

Patent ductus arteriosus patens (*ductus arteriosus patens*, PDA) is acyanotic congenital heart defect in which the ductus arteriosus of Botalli, the *ductus arteriosus Botalli,* has not closed '. The duct of Botall is a vessel that connects the pulmonary artery to the aorta and is important in fetal circulation where it allows blood to bypass the lungs by shunting from the pulmonary artery to the aorta.

In healthy full-term newborns, a '*functional closure* of the duct occurs shortly after birth. Complete closure occurs in almost half of full-term newborns *within 24 hours* of birth, in 90% within 48 hours, and in *all within 96 hours* of birth. Failure to close and persistence of this fetal junction is called a patent or persistent ductus arteriosus (PDA).[1]

PDA is more common in premature. A patent ductus can vary in width and length, and there is a wide spectrum of severity of symptoms, from occasional murmurs to severe acute heart failure.[2]



Open duct of Botall

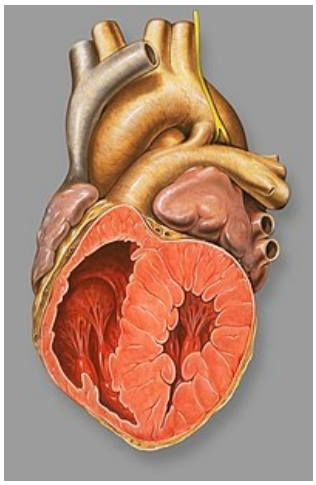
Risk Factors

A *higher incidence of PDA* is associated with the following factors:

- immaturity (for newborns with a birth weight of less than 1000 g, the incidence is 80%),[3]
- respiratory distress syndrome (RDS) and its treatment with surfactant,
- high intravenous fluid intake in the first days of life,
- asphyxia,
- congenital rubella, trisomy 13, trisomy 18, ...,
- high altitude,
- congenital heart defects (coarctation of the aorta, pulmonary atresia, transposition of the great arteries, ...).[3]

Conversely, a **lower incidence of PDA'** is associated with the following factors:

- antenatal administration of corticosteroids (induction of lung maturity in immature newborns),
- IUGR,
- premature outflow of amniotic fluid (PROM).[3]



Ductus arteriosus Botalli

File:Botall's persistent spirit.jpg

CT Botall's persistent spirit

Pathophysiology

The fetal ductus arteriosus diverts blood flow from the pulmonary artery to the *descending aorta*, thus bypassing the pulmonary basin in which there is high vascular resistance. The closure or persistence of the ductus after birth is determined by the action of various factors. Oxygen promotes airway closure, and prostaglandins E, on the other hand, open the airway. Susceptibility to these factors varies with **gestational age**. The younger the fetus, the smaller the contracting effect of oxygen, the more the dilating effect of prostaglandin E_2 increases and the sensitivity to indomethacin, which is used for pharmacological closure of the duct, increases.[1]

Clinical picture

Small duct is usually asymptomatic. Large airways result in slow growth, failure to thrive, collapsing peripheral pulsations, hypotension (may be the first symptom in ELBW), ventilatory difficulties, heart failure (with pulmonary edema and hepatomegaly). Peripheral pulsations are noticeably lively, the precordium is active. Resting tachycardia and tachypnea are conspicuous.

A wide short wind causes heart failure, pulmonary hypertension, manifestations of cyanosis of the lower half of the body. A moderately significant cough can cause repeated respiratory infections and failure of the child to thrive. Narrow duct is asymptomatic. **All patients** are at risk of infective endocarditis.

In premature infants with RDS (*respiratory distress syndr*ome*) open trachea can lead to dependence on artificial pulmonary ventilation. In infants, it causes circulatory failure, manifested by expiratory dyspnea from pulmonary edema.*

⚠: continuous systolic-diastolic locomotive **murmur** (so-called three-beat rhythm) *under the left clavicle*, the maximum of the murmur during II. sounds[4] (we hear the low-frequency sound of "coke spilling").

Diagnosis

We establish the diagnosis based on

- physical findings,
- echocardiography including Doppler,
- X-ray of the heart - cardiomegaly, pulmonary plethora[2].

Differential diagnosis

Patients with an **aortopulmonary window** (direct communication ascending aorta and truncus pulmonalis) have a similar clinical picture.

Therapy

- **Asymptomatic PDA'** - observation, most close spontaneously (mainly in premature infants)[2]
- **symptomatic PDA**;
  - limited fluid intake,
  - ensuring sufficient blood oxygenation,
  - treatment of heart failure: furosemide,
  - consider pharmacological closure: indomethacin, ibuprofen,
    - absolute contraindication: heart defects in which an open trachea is necessary for survival (e.g. coarctation of the aorta),
    - side effects: oliguria, fluid retention and hyponatremia due to reduced renal blood flow, reduced cerebral blood flow, gastrointestinal complications (bleeding, ulceration), bleeding (impaired platelet function), [[icterus]],
  - surgical treatment (ligature, transection)[2].
  - cardiac catheterization (Amplatz occluder)

Links

Related Articles

- Congenital heart defects
- Acquired heart defects

External links

- Open Botall's soul - Selest - Audio recordings (TECHmED) (https://www.techmed.sk/kontinualny-selest/)

References

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