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Anatomy

Patent Ductus Arteriosus: A Cadaveric Observational Study

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ABSTRACT Patent ductus arteriosus (PDA) is a condition wherein the ductus arteriosus fails to close after birth . Ductus arteriosus is a communication between the descending thoracic aorta and pulmonary artery seen in the fetal life and closes after birth. In a PDA, the vessel does not close and remains "patent" (open), resulting in irregular transmission of blood between the aorta and the pulmonary artery.This condition is frequently diagnosed in infants but in some it may be seen in the later stages of life . In isolated patent ductus arteriosus left to right shunting may be see. The incidence of PDA in term infants is about 1 in 2000 births, accounting for 5 to 10% of all congenital heart disease.

The female to male ratio is ~2:1. Histologically the ductus arteriosus contains more smooth muscle which helps in narrowing of the lumen and shortening . Physiologically the factors which maintain the patency of ductus is exposure to low partial pressure of oxygen , circulating prostaglandins and local nitric oxide production from endothelial cells. The availability of echocardiography has improved the detection of congenital PDA, resulting in earlier treatment. A continuous murmur is heard, located at the upper left sternal border, referred to as a “machinery” murmur. It radiates down the left side of the sternum and into the back, and a thrill may be present. Closure of PDA, by either surgery or transcatheter techniques, can now be achieved safely, resulting in a decrease in the incidence of severe complications of PDA.

KEYWORDS : Ductus arteriosus, Patent, Fetal circulation, Shunt

BACK GROUND :

During the fetal circulation the right ventricular blood with low oxygen content is discharged into pulmonary trunk. From the pulmonary trunk blood flow trifurcates

- Enters into corresponding lung via pulmonary artery.
- Enters into distal part of arch of aorta via ductus arteriosus and it continues into descending aorta .

The ductus arteriosus closes after birth and forms as ligamentum arteriosum. In some conditions it is patent and called as PDA – Patent ductus arteriosus.

The clinical spectrum of presentation of a PDA may range from a "silent" PDA, one with no clinical manifestations but which is incidentally discovered on echocardiogram for other purposes, to patients who present with congestive heart failure, pulmonary hypertension, signs of volume overload, endocarditis, atrial fibrillation, or "recurrent pneumonia." Patent ductus arteriosus (PDA) at childhood is one of the five major and frequent congenital abnormalities that its frequency was reported as 10-14.4% of all congenital defects[1]. PDA maybe presented in adulthood but most adult cases reported with pulmonary hypertension (PHTN). PHTN and other presentations such as heart failure and edema are the identified complications of longstanding PDA, but adult case with no permanent heart symptoms and PHTN was rare.

MATERIAL AND METHOD :

In the present study 36 heart specimens from adult cadavers were dissected over a period of 5 years irrespective of the sex, in the Department of Anatomy at Apollo Institute of Medical Sciences and Research, Hyderabad, Telangana State, India.

A complete midline incision is made from the suprasternal notch to the symphysis pubis. The fibrous pericardium is incised. The major blood vessels are clamped and insiced. The heart is separated and washed and preserved in 10 % formalin solution for further studies.

RESULTS:

Among the 30 specimens of heart, 1 heart was found to be having the patent ductus arteriosus.

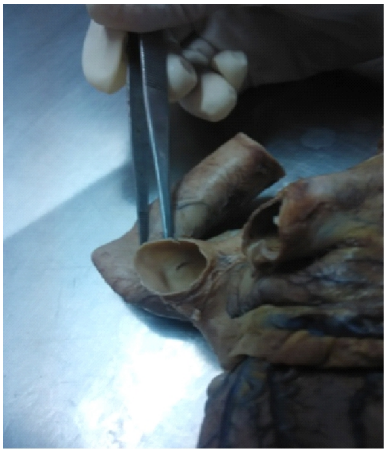
DISCUSSION

In the developing fetus, the DA is the vascular connection between the pulmonary artery and the aortic arch that allows most of the blood from the right ventricle to bypass the fetus' lungs, which are fluid-filled and compressed. When the newborn takes his or her first breath, the lungs open and pulmonary vascular resistance decreases. After birth, the lungs release bradykinin to constrict the smooth muscle wall of the DA and reduce blood flow as it narrows and then completely closes.

In normal newborns, the DA is substantially narrowed within 12–24 hours after birth, and seals completely after three weeks.The primary stimulus for closure of the DA is an increase in neonatal blood oxygen content. Withdrawal from maternal circulating prostaglandins also contributes to ductal closure. The residual scar tissue from the fibrotic remnants of the DA, called the ligamentum arteriosum, remains in the normal adult heart.

“PDA normally closes soon after birth, but in some newborns it does not close spontaneously, and there is continuous flow from the aorta to the pulmonary artery (i.e., left-to-right shunting)” [2] . On average, PDA occurs in about 8 out of every 1,000 premature babies, compared with 2 out of every 1,000 full-term babies. Premature babies also are more vulnerable to the effects of PDA.PDA is twice as common in girls as it is in boys[3].

PDA is mostly seen in preterm newborns. Therefore, it is expected that the prevalence of incidental PDA in adult's increase. Thus, primary care physicians need to be alert to the clinical situations suggesting a previously undiagnosed PDA [4]. It was previously reported that if the size of the defect is large (more than 2.5 mm), occurrence of PHTN and Eisenmenger syndrome will increase [5]. The normal pulmonary artery pressure in our patient despite a moderate defect could because of the potential ability of pulmonary vascular bed for tolerating longstanding high volume of blood from the childhood[6].



CONCLUSION

The present study was limited to the cadaveric dissection to percentage the occurrence of patent ductus arteriosus in adult specimens in view of current scenario of knowledge about the relationship of patent ductus arteriosus with unidentified blood vascular conditions in general population. Further well-designed studies are necessary to address several unresolved issues related to pathophysiology and complications of patent ductus arteriosus.

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