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A Young Female with Generalised Weakness and Exertional Dyspnoea A Case of PDA

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Mrs. E. R. 23 years old married female from Lahore was admitted in the department of Cardio Throracic Surgery Shaikh Zayed Hospital on the Ist of March, 1987.

At the time of admission she had the complaints of :–

Generalised weakness	5 years
Progressive shortness of breath on exertion.	5 years

Present Illness.

According to patient, she was completely well til about five years ago when she started to have generalized weakness, palpitations and shortness of breath on performing household chors. She also gives history of spells of peripheral cyanosis. There is no history of orthopnoea.

Past Illness:

The patient was diagnosed as a case of patent ductus arteriousus when she was six years old. She has been leading a symptom free life till five years ago when she developed the above mentioned complaints, which were progressive in nature.

General Physical Examination:

On general physical examination the patient was found to be a well developed adult female. she was very comfortable in her bed and did not look ill at all.

Pulse:	84/min.
B.P.	120/90 mm.
Temp.	98.6 F^{o}

Cardio Vascular System:

Examination of the cardio vascular system revealed that her pulse was regular and of good volume. The JVP was not raised. Ankle edema was negative. A thrill was palpable on the precordium. On auscultation, both heart sounds were audible and the characteristic machinery murmur could be heard all over the precordium with maximum intensity at the pulmonary valve area.

Perspiratory System

Normal vesicular breath sounds were audible in both lungs. Her gastrointestinal, genitourinary and other systems were also normal. She was clinically diagnosed as a case of patent ductus arteriousus and it was decided to carry out series of investigations.

ECG normal

X-ray Chest slightly increase in pulmonary vascularity and prominence of pulmonary conus.

Echocardiography

2 D echo showed mild pulmonary hypertension, some suspicion of mitral valve prolapse, but no other congenital abnormalities. The dopplar studies confirmed the presence of turbulance above the pulmonary valve, and these findings were Compatible with our clinical diagnosis of PDA.

After carrying out these investigations the diagnosis was confirmed and it was decided to operate upon her and ligate the PDA. A postero lateral left 4 I.C.S. thoracotomy was done on the 3rd of March, 1987.

Paraoperatively it was seen that adhesions were present between the (L) lung and pariatel pleura. A patent doctus arteriosus 1.5 cm in diameter and 1/4 cm in length was noted. The P.A. pressure was raised. The lung was mobilised first and then the aorta was mobilized from the arach to 1 cm below the doctus. Tapes were passed around it to gain proximal and distal control. Both pulmonary and aortic ends of the duct were identified & No. 1 silk ligatures were passed around it to secure it. The PDA was double tied at aortic and pulmonary ends respectively after proximally cross clamping the arch of aorta to reduce tension on the ductus. The total crossclamping time was approximately 20 seconds. Total occlusion was confirmed by the absence of thrill. A chest drain was placed and the chest was closed in layers. The patient was shfited to the I.C.U. for overnight monitoring and was transferred to her room next day.

Her post Op. course was smooth and without any complications. She was discharged a week later in completely satisfactory state.

DISCUSSION

The anatomy and closure of the patent ductus arteriousus was first described by Galen in 181 AD. We can see how beautifully he described the correct anatomy of this rare lesion. Though the anatomical terminology used in those days was slightly baffling.

Persistent Ductus Arteriosus

Ductus Arteriousus is a vascular communication between the beginning of the left pulmonary artery and the descending thoracic aorta. The arterial duct arises from the posterior superior aspect of the junction of the pulmonary trunk and pulmonary artery. It courses posteriorlay and slightly leftwards to join the junction of the aortic arch and descending aorta just distal to and opposite the left sub clavian orifice. Its pulmonary end is covered by a fold of pericordium and its oartic end by parietal pleura.

Now briefly I would like to talk about foetal circulation and the effects of a persistent ductus arteriosus on patient's haemodynamic status.

The Fetal Circulation

The lungs are functionless during fetal life and the blood supply to them is therefore, minimal. Oxygenated blood from the placenta reaches the fetus by was of the umbilical vein, thence through the ductus venosus into the inferior vena cava. The IVC also receives deoxygenated blood from the trunk and the lower extremities.

The oxygenated blood reaching the inferior vena cava from the umbilical vein is prefrentially deflected through the foramen oval into the left atrium and to the head and neck vessels via left ventricle. aorta. The superior vena cava carries entirely deoxygenated blood from the head and upper limbs in the right atrium. This deoxygenated blood alongwith deoxygenated blood from lower limbs and trunk goes via RV into the pulmonary trunk.

Most of the pulmonary arterial blood passes through the large ductus arteriousus into the aorta and back to the lower limbs and trunk, which require blood with less oxygen saturation. A small amount of blood still is required for nutrition of lung tissue and escapes though pulmonary, artery branches into the lung and returns to the LA through the pulmonary veins. The LA thus receives a small amount of deoxygenated blood from the pulmonary veins but a much larger amount of predominantly oxygenated blood from the RA via the foramen ovale.

The PDA directs blood away from the non ventilating lungs into the descending aorta and into the placental circulation. In the fetus the PDA has the same diameter as the descending aorta and carries about 50% of the combined venticular output from the pulmonary trunk to the aorta.

It dilates in response to hypoxia and to the circulating prostaglandins E_1 and E_2. This fact is of immense clinical importance as it is considered in the treatment of PDA.

At birth with aeration and expansion of the lungs, a fall in pressure and resistance occurs in the pulmonary circulation, such that pressure in the pulmonary artery approximates that of the aorta, and this together with an increase in the partial pressure of O_2 in the blood causes the contraction of the smooth muscle in the ductus arteriosus, and flow through it virtuallly ceases and the blood in the pulmonary artery is directed to the lungs. This increased pulmonary circulation results in a greater return of blood to the LA via the pulmonary veins.

Because more blood flows in to the LA, the LA pressure rises above that of the RA and leads to the closure of the foramen ovale. Functional closure of the PDA usually occurs within 10 to 15 hours after birth, while anatomical closure may take between 1 to 12 weeks. Failure of this mechanism is thought to be due to immaturity of the histological structures of the PDA in combination with biochemical unresponsiveness.

Haemodynamics:

The shunt in an uncomplicated ductus is from the aorta to the pulmonary artery. Because both the sytolic and diastolic pressures in the aorta are always higher than those of the pulmonary artery, blood flows through the duct throughout the cardia cycle, resulting in a continuous murmur. The total pulmonary blood flow is increased. So is the return to LA. and LV. The left heart has to cope with this augmented flow resulting in left ventricular overload. The LV has to work more and over the years becomes hypertrophied. It finally dilates and fails. At the same time pulmonary artery resistance increases due to increased flow and lead to Rv failure and the reversal of the shunt.

Clinical Features:

At least twice as many females are affected as males. Usually no symptoms are produced and the classical machinery murmur is discovered as an incidental finding.

Physical development is only slightly affected. If the left-to-right shunt is large, then dyspnoea and pulmonary infections including bronchitis and bronchpuenuomia become common and the child may fail to thrive. The adults may develop heart failure and angina.

Physical Sings:

1. The machinery murmur. It is a continuous murmur and loudest heard over 2nd I/C spece Lt. of sternum. It accentuated during expiration and exercise.
   If heart failure occurs the typical murmur may disappear and if the shunt becomes reversed it invariably does so.
2. The scond sound is split and the pulmonary element is lound, but it tends to be hidden by the murmur. Which is at its maximum intensity just before and after the second sound.
3. A mid-diastolic murmur is commonly heard near the mitral valve.
4. The pulse is of collapsing type when a large shunt is present.

Complication:

Complications of a persistent ductus arterioses may be:

1. Bacterial infection of the ductus arteriosus.
2. Pulmonary hypertension leading to the reversal of the shunt
3. Congestive Cardiac failure

1. Bacterial infection of the PDA:

The frequency of infective endarteritis is about 10% and the mortality is 12%.

The vegetations are usually on the pulmonary arterial end of the duct. Small ducts are more prone to infections than large ones.

Treatment:

Indomethacin, a potent inhibitor of prostaglandin synthesis, has been used orally in the treatment of low birth weight infants with successful closure of the duct in most of the cases. The dosage is 0.2 mg/kg per dose via a nasogastric tube at 12 hourly intervals for three doses. If no improvement occurs after three doses, surgical ligation is carried out.

Indomethacin avoids surgery in tiny premature infants, but if they weigh less than 1000 gm the response is poor.

Surgical Treatment would be discussed by Dr. Shahkar We will have comments from faculty members.

PROF. ASHFAQUE A. KHAN *(Cardiologist)* Natural history of PDA in an adult is variable. It is much more common in females (F:M=6:1) and its incidence is much higher in the population living at high altitudes. A small PDA usually remains asymptomatic and spontaneous closure have been reported in the adult. Cardiovascular decompensation is related to the size of left to right (L-R) shunt with resultant hyperdynamic pulmonary flow which results in anatomical changes in the pulomnary vasculature resulting in pulmonary hypertension (P.H.) and subsequent right heart failure. This process of development of P.H. can be accelerated if there is any concurrent pathology in the lungs producing hypoxia with resultant pulmonary vasoconstriction.

Most common symptom with development of cardiovascular decompensation is early fatiguability. This is more pronounced in the legs and is primarily the result of reversal of the L-R shunt which is directed preferrantially to the lower extremities. Dyspnea on exertion is another common symptom and is due to pulmonary congestion.

Typical physical signs of a collapsing arterial pulse, hight pulse pressure and a machinery like murmur at the 2nd left intercostal space is usually found in those patient who do not have significant P.H. However, with the development of P.H. the murmur may acquire more phasic characteristics and in later cases is only confined to the early diastole like a Graham Steel murmur. In the later stages as the shunt reverses central cyanosis confined to the lower extremeties is also seen and can be easily over looked if the lower extremeties are not examined properly.

Most common complications of PDA besides cardiovascular decompensation are infective endocarditis and clacification or aneurysmal dilation/rupture of the duct in the later years. The common sight for infective endocarditis is on the pulmonary artery endothelium where the jet strikes. However, the duct itself may get spontaneously involved in the infective process and spontaneous closure have been reported during the resolution phase. Calcification and aneurysmal dilatation is a serious complication and can lead to spontaneous rupture with fatal out come and can cause serious problems during surgical closure if the diagnosis of this complication is not made preoperatively. The best alternative for such patient is to put them on cardiopulmonary bypass during surgery and to do the closure accordingly.

Closure of the duct, by a plug which is passed over, a guide wire introduced retrograde into the aorta and manipulated through the duct in to the pulmonary artery and backward through the right ventricle, right atrium and inferior vena cava is reported by Portman and two hundred such cases have been done successfully.

DR. NAUMAN *(Cons. Anaesth)* Cardiac anaesthesia is a specialised branch of anaesthesia. During thoracic to give good access to the heart and great vessals. Usually intermittent positive pressure ventilation is used with one lung anaesthesia. During this type of anaesthesia there is an increased venours admixture in the circulation and is

dealt with increase in inspired oxygenation.

One lung anaesthesia is achieved by introducing the endotracheal tube in the main bronchus of one lung with a subsequent collapse of the other lung. During surgery monioring of pulse, BP and ECG is mandatory

DR. SHAHKAR *(Cardio Thoracic Surgeon)* Operative intervention is suggested in all children, symptomatic or asymptomatic when the PDA is first encountered. Older age is not a contraindication to operative intervention, provided severe pulmonary hypertension with Eisenmengers changes is not present. The aim of surgery is to interrupt the communication between the pulmonary and systemic circuits, either by division or ligation of the duct.

The ductus arteriousus was first successfully ligated by Gross in Boston, USA in the year 1939.

Depending on the size of the ductus different operative techniques are used. If the duct is narrow, ligation is carried out with two thick ligatures. If the duct is wide, division and suturing is done'

After operation the systemic blood pressure tends to be slighlty raised for a week or so, and if the patient is already hypertensive it is best to ligate the ductus and then treat the resultant hypertension.

Few operations in Cardiac surgery carry the prognosis for total cure such as is carried by interuption of the uncomplicated PDA. Operative mortality during the past decade, in patients without associated anomalies has been less than 0.5 percent. Long term evaluation of these patients has clearly demonstrated that when the PDA is divided, there is acomplete absence of late morbidity.