Patent Ductus Arteriosus

Hagop D. Yacoubian*

THIS article details our experience with three cases of Patent Ductus Arteriosus treated at Salmaniya Medical Center between December 1979 and July 1980. A brief discussion of the pathogenesis, symptomatology and management of this condition follows.

CASE PRESENTATIONS

CASE NO. 1

M.A.H. a 12 year old Bahraini female child was admitted to Salmaniya Medical Center on December 12, 1979 with a history of patent ductus arteriosus discovered at the age of seven years. She had been admitted to the Paediatrics service on multiple occasions for pulmonary infections. She was known to have a sickling trait, and in August 1979 was treated for coliform septecaemia. On her present admission she was a febrile, had a hemoglobin of 13.3 gms % and looked clinically well. Physical examination revealed a machinery murmur in the left upper parasternal area, prominent second pulmonary sound and a blood pressure of 110/70 mm of Hg. Chest film showed a prominent pulmonary artery. Electro-cardiogram revealed left ventricular hypertrophy. On December 29, 1979 a 6 mm wide patent ductus was divided and sutured. She did very well post-operatively. Her blood pressure immediately after division of the ductus rose to 130/80 and within 12 hours reached 150/120. She was discharged on the seventh post-operative day in good condition. Two weeks later the blood pressure had declined to 100/60 and it remained at around that level.

CASE NO. 2

D.M.A. a three year old female Bahraini patient was transferred to the Department of Surgery from Paediatrics on May 22, 1980 with a diagnosis of patent ductus arteriosus and glucose phosphate dehydrogenase deficiency. On examination she had a faint machinery murmur over the left upper parasternal area. B.P. was 110/55 mm of Hg. Chest film showed increased pulmonary vacularity. EKG revealed left ventricular hypertrophy. HB was 12.9 gms %. On May 24, 1980 a 7 mm wide ductus was divided and sutured. Post-operatively the systolic pressure was unchanged but the diastolic rose to 70 mm of Hg. She did well post-operatively and was discharged on the 8th post-operative day. She has remained well.

CASE NO. 3

S.M.A. a nine year old Pakistani male child was admitted to Salmaniya Medical Center on July 3, 1980 with a diagnosis of asymptomatic patent ductus arteriosus. The characteristic murmur was discovered at the age of five while the child was admitted to the Paediatrics Service for infectious hepatitis. On examination, during this admission, there was a machinery murmur heard over the left upper parasternal area. There was a prominent second Pulmonary sound. Chest x-ray was normal, B.P. was 120/70 mm of Hg. and EKG was normal. On July 7, 1980 a small 3 mm wide ductus was divided and sutured. The patient did very well. B.P. remained unchanged. He was discharged on the seventh post-operative day and has remained well.

DISCUSSION

Figure 1 : illustrates the primitive aortic arches and their evolution into permanent vessels in the fetus. The darkened areas are vessels which are retained. The white areas indicate the portions of the arches which atrophy and disappear. The shaded areas indicate the portions which develop into the pulmonary arteries.

As can be seen, arches No. 1 and 2 disappear. Arch No. 3 on both sides, becomes the common carotid artery and its branches. On the right side arch No. 4 becomes the innominate artery proximally and the right subclavian artery distally. On the left side arch No. 4 becomes the aortic arch. Arch No. 5, on either side, is rudimentary and disappears early. Arch No. 6, on either side becomes the pulmonary arterial system; on the right, its connection with the aortic arch

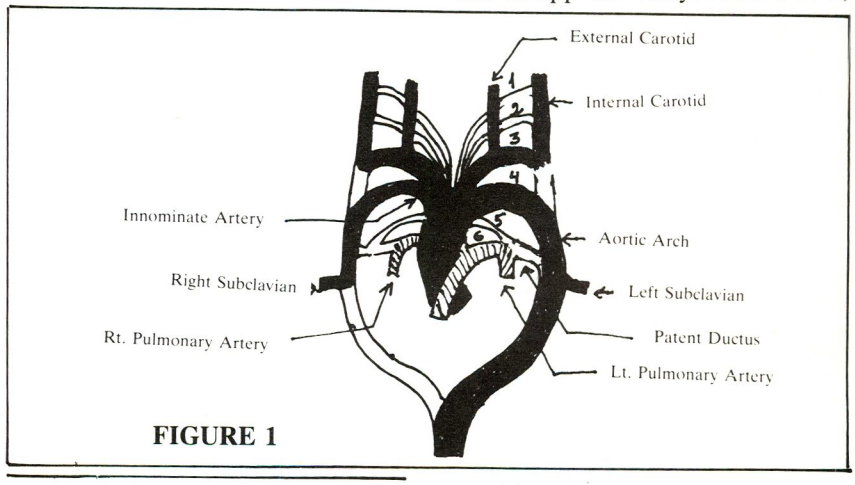

FIGURE 1

* Chairman
Department of Surgery
Salmaniya Medical Centre

system disappears but on the left this connection remains patent until birth as the ductus arteriosus. In this illustration, the truncus arteriosus has already subdivided into the root of the aorta and the main pulmonary artery.

Figure No. 2 : illustrates the fetal circulation in the later stages of development. Arterialized blood returning from the placenta through the umbilical vein, passes through the liver and reaches the right atrium after mixing with blood in the inferior vena cava. Most of this blood is shunted directly into the left atrium through the patent foramen ovale and is pumped into the aorta by the left ventricle. Blood returning via the superior vena cava is shunted predominantly into the right ventricle and pumped into the pulmonary artery. Because of the high pulmonary vascular resistance in the collapsed lungs a very small portion of this blood reaches the left atrium through the pulmonary circulation; most of it passes directly into the aorta through the ductus arteriosus. The above facts have been substantiated by radioactive tracers in the circulation of fetal sheep and by blood gas analyses (1, 2, 3). At birth, with expansion of the lungs pulmonary vascular resistance starts dropping markedly.Pulmonary artery blood now preferentially flows through the pulmonary vasculature. With increase in blood return to the left atrium, pressure in the latter increases. This results in closure of the foramen ovale by apposition of its flap like mechanism. Closure becomes permanent later by fusion. Therefore all blood reaching the right atrium has to pass through the right ventricle and pulmonary circulation. Flow in the patent ductus is reversed and is now from the aorta to the pulmonary artery. The ductus constricts rapidly decreasing the flow through it. The causes of this initial constriction are not known. Full obliteration due to intimal proliferation, may take up to months and when complete the pulmonary and systemic circulations are effectively separated. However, it has been thought that maternal rubulla during the first three months of pregnancy may be a contributory factor in maintaining the patency of the ductus.

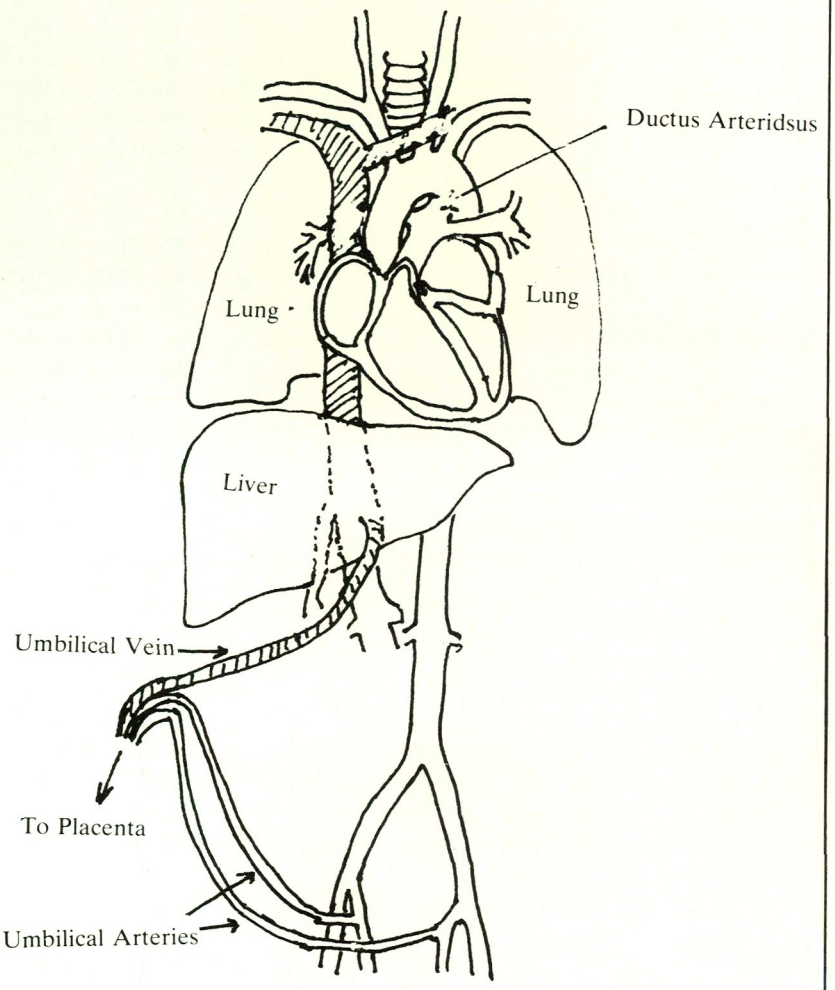

FIGURE 2

Christie, in his study of 558 infants found the ductus to be patent at 2 weeks in 65 pervent, at 32 weeks in 2 percent and at one year in only 1 percent (4). Most of the latter were small and functionally insignificant openings.

Symptoms in patients with patent ductus arteriosus vary greatly and depend on the size of the shunt, age and other factors which are not known. In extreme cases there may be severe heart failure at birth necessitating emergency surgery. Such procedures carry a very high mortality. Most children are asymptomatic and the condition is discovered

accidentaly during examination for some other reasons. Such children lead normal lives with no physical limitations. Most, however, exhibit retardation of physical growth. The fact that the majority of children are well in childhood should not lead one to conclude that the condition is compatiable with long life. Almost half the patients with untreated patent ductus will be dead by the age of 45 years (5).

The heart is usually normal in size but hyperactive. The shunt is unidirectional and continuous but more pronounced during systoly due to the higher gradient. This gives the typical machinery murmur heard best in the left upper parasternal area. The systolic pressure may be normal but the diastolic is characteristically low giving a wide pulse pressure. The shunt overloads the pulmonary circulation and the left size of the heart giving rise to a loud second pulmonary valve closure sound and left ventricular hypertrophy which is demonstrated by electrocardiography. Two of our patients had left ventricular hypertrophy and had ductuses of 6 and 7 mm. The third patient (S.M.A.) had a normal electro-cardiogram. This patient had a small 3 mm ductus and most probably had a small shunt. In very large shunts there may even be a diastolic rumble of relative mitral stenosis due to the very high flow through the left side of the heart.

X-ray studies show a normal sized heart in most cases. Any enlargement, if present, will be in the left atrium and left ventricle. The pulmonary arteries are prominent and the lungs reveal increased vasculature. Fluroscopy may demonstrate a hilar dance.

In untreated patients the increased pulmonary flow will eventually cause pulmonary arterial intimal proliferation with thickening of the wall and luminal narrowing resulting in gradually increasing pulmonry vascular resistance, pulmonary hypertension and right ventricular hypertrophy. With increasing pulmonary hypertension there will be equilibration and then eventual reversal of the shunt through the ductus. This will lead to the characteristic paradoxical cyanosis of the lower trunk.

In the vast majority of cases the diagnosis can be easily made on the basis of the typical clinical picture, electro-cardiogram and radiography without having to resort to special invasive studies. Surgical correction can be safely recommended on these grounds. Special studies may be necessary in equivocal cases. The condition most difficult to differentiate from patent ductus is aorto-pulmonary window but the latter is fortunately very rare. It is hardly ever necessary to measure the volume of the shunt.

Operative correction is simple and in asymptomatic children the risk should be no more than that of general anaesthesia. The procedure should be, ideally, carried out before the age of 5 years and in symptomatic children as soon as the diagnosis is made.

The decision is more difficult to make in older patients. With pulmonary hypertension and equilibrated shunts, surgery is probably indicated but with considerably increased risk. It has been shown that in some of these cases interruption of the shunt can have a beneficial effect on the pulmonary vascular resistance, particularly in younger patients.

When the shunt is predominantly right to left, there is general agreement that interruption of the shunt is contra-indicated. The shunt in these cases acts as a compensating safety valve. Not only is there nothing to gain by its closure but death is unavoidable from the rapid onset of right heart failure.

It is immaterial whether the ductus is closed by multiple ligation or by division and suture (6). Most surgeons use the division method made possible by the advent of modern and effective vascular clamps.

Immediately after closure systolic and particularly diastolic pressures are likely to rise. These patients have hypervolemia and it will take some time for the blood volume and increased cardiac output to adjust to the new hemodynamic situation. In our first patient (M.A.H.) blood pressure rose immediately after closure and settled down to a normal value in 2 weeks.

Few operations are more gratifying with such long term curative effects as closure of patent ductus. The operation can be performed with almost complete safety in children who are asymptomatic and have no pulmonary hypertension.

SUMMARY

Patent Ductus Arteriosus is a congenital cardiovascular abnormality resulting from failure of closure of the ductus arteriosus after birth. Symptoms depend on age and size of shunt. In the vast majority of children the condition is asymptomatic, is discovered accidentally and can be easily diagnosed clinically. Interruption of the shunt is indicated early in life prophylactically and can be carried out with almost complete safety in patient with no pulmonary hypertension.

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(Above) H.E. The Crown Prince and Commander-in-Chief of the Bahrain Defence Force opening the Jidhafs Maternity Hospital on October 10, 1980 (below) Guests at the opening of the Jidhafs Maternity Hospital – October 10, 1980