

A STUDY OF CLINICAL PROFILE AND MANAGEMENT OF PATIENTS WITH PATENT DUCTUS ARTERIOSUS

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ABSTRACT

BACKGROUND

PDA is an abnormal persistence of a patent lumen in the foetal ductus arteriosus that usually connects the upper descending thoracic aorta with the proximal portion of the left pulmonary artery. The aim of the study is to study the clinical profile of patients with patent ductus arteriosus, role of various investigation modalities, various surgical treatment options available and overall morbidity and mortality.

MATERIALS AND METHODS

The retrospective study includes 60 cases of patent ductus arteriosus admitted for a period of 5 years.

RESULTS

The PDA is more common in females with female-to-male ratio of 2.75:1. Premature infants with PDA become symptomatic earlier as compared to full-term infants. Because of the late presentation, majority of the patients were symptomatic in the present study, and in symptomatic patients, symptoms of breathlessness of varying NYHA-class and recurrent respiratory tract infections were the commonest symptoms. Accentuated pulmonary sound, hyperactive precordium, murmur were the most signs. Chest x-ray and ECG are the important investigation in the diagnosis of PDA. Echocardiogram was the most commonly used diagnostic modality and was diagnostic in all cases. Echocardiogram does give an accurate assessment of PDA, but ultimately intraoperative assessment of PDA is the most important to decide about the surgical technique of closure. Chylothorax was seen in 3 patients. The overall mortality in the present study was 3%.

CONCLUSION

Surgical closure of PDA can be accomplished with low morbidity and mortality.

KEYWORDS

Patent Ductus Arteriosus, Ductus Arteriosus, Mortality.

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BACKGROUND

PDA is an abnormal persistence of a patent lumen in the foetal ductus arteriosus that usually connects the upper descending thoracic aorta with the proximal portion of the left pulmonary artery. When the aortic arch is right-sided, the ductus usually connects to the proximal right pulmonary artery. Ductus may at times connect to the adjacent subclavian or innominate artery rather than to the upper descending thoracic aorta. Thus, PDA is the anomaly that initiated not only the surgical treatment of congenital heart disease, but also the percutaneous treatment of congenital heart disease. The ductus arteriosus is a foetal vascular structure derived from the sixth aortic arch and normally extends from the main or left pulmonary artery to the descending aortic arch distal to the origin of the subclavian artery.

Rarely, the ductus may be right-sided, bilateral or absent. The ductus is usually 5 to 10 mm long and varies from a few millimetres to 1 to 2 cm in diameter. The aortic orifice is usually larger than the pulmonary orifice. The wall of the ductus arteriosus differs from that of other major vascular structures. The media is deficient in elastic fibers and is instead composed a primarily of smooth muscle cells. The smooth muscle in the wall of the ductus arteriosus is specifically sensitive to environmental factors particularly prostaglandins (PGE₂ and PGI₂) that promotes relaxation, and PO₂ that causes constriction.

Postnatal closure occurs in two stages, the first stage is complete within 10-15 hours after birth in full-term infants and is due to contraction of the smooth muscle in the media of the ductal wall, which produces shortening and an increase in wall thickness.¹ This functional closure is assisted by approximation of the intimal cushions. The second stage of closure is usually completed by 2 to 3 weeks. It is the result of diffuse fibrous proliferation of the intimal, sometimes associated with necrosis of the inner layer of the media and haemorrhage into the wall. The latter maybe due to intimal tears producing a limited dissection of the ductus wall; there may also be small thrombi within the lumen, but gross luminal thrombosis is rare.

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These changes result in permanent sealing of the lumen and produce the fibrous ligamentum arteriosum.² Closure usually begins at the pulmonary end and may remain incomplete at the aortic end leaving an aortic ampulla from which the ligamentum arteriosum arises. The ductus arteriosus is completely closed by 8 weeks of age in 88% of infants with a normal cardiovascular system. When the process is delayed, the term "prolonged patency" of the ductus arteriosus is appropriate; when the process ultimately fails, persistent patency of the ductus arteriosus is the appropriate term. Ductus closure is mediated by release of vasoactive substances." Oxygen tension and prostaglandins act in opposite directions with an increasing PO₂ constricting the ductus and prostaglandins relaxing it. The complex interplay of these factors is the reason that prolonged patency of the ductus is more common in premature than term infants. The pathophysiologic effects of an isolated patent ductus arteriosus are related to the associated left-to-right shunt and these effects vary greatly depending on the size of the shunt.

A PDA is in essence a portal between the systemic and the pulmonary circulations. Because the ductus creates a shunt beyond the aortic and pulmonary valves, shunt flows may occur during both systole and diastole. The amount of blood flow diverted by this "left-to-right" shunt depends primarily on two factors. One, size of the PDA, i.e., its diameter and length; second, difference between the pulmonary and systemic vascular resistances. Thus, if the ductus is small and/or the pulmonary resistance is high, the resulting shunt flow will be small. If the ductus is large and the pulmonary resistance low, a very large shunt can be the consequence.³ To study the clinical profile of patients with patent ductus arteriosus, role of various investigation modalities, various surgical treatment options available and overall morbidity and mortality.

MATERIALS AND METHODS

The retrospective study includes the 60 cases of patent ductus arteriosus admitted in Gandhi Hospital from January 2008 to October 2013. The study was carried out under the following heads. Data pertaining to the patient's age, sex, etc., history, chief complaints and the origin, duration and progress of the complaints, prematurity/respiratory distress at the time of birth, H/o consanguineous marriage, family history of congenital heart disease, physical examinations findings, investigations and morbidity and mortality. The investigations consisted of routine blood tests, chest x-ray, electrocardiogram and echocardiography. The usual operative techniques used for the closure of PDA at our institute are:

Triple Ligation

Was performed through standard left posterolateral thoracotomy through fourth intercostal space. The pleura over the descending aorta was incised vertically over the aorta close to the ductus. The plural flap was raised and cleared off the ductus. PDA was dissected all around.

A right-angled forceps was passed beneath the ductus and No. 20 Barbour's thread was fed. First ligature was tied on the aortic side followed by pulmonary ligatures and lastly middle one.

Division and Suturing

In the technique of division and suturing, aorta was cleared proximally and distally to the ductus and control was established by passing umbilical tape. Ductus was cleared all around and ductus clamps were applied. Ductus was divided in between the clamps and the cut ends were sutured with 4-0 polypropylene. The decision whether to do triple ligation or do division and suturing was mostly taken intraoperatively after assessing the size and length of the ductus. PDA closure, either triple ligation or division and suturing were done under hypotensive anaesthesia. Systolic pressures were brought down to between 60 and 70 mmHg. Except in few patients, blood pressures were monitored by NIBP. Patient who required invasive BP monitoring were the one with large PDA's and in whom division and suturing were contemplated before surgery. Halothane and intravenous nitroglycerine were used intraoperatively to reduce the blood pressure.

RESULTS

Age Group	No. of Patients
0-1	5
1-5	29
5-10	17
10-15	8
>15	1
Age at Onset of Symptoms	
<1 week	4
<1 Month	2
>1 Month	4
1 month - 1 year	19
<1 year	16

Table 1. Demographic Distribution in Study

Majority of the patients belonged to age group of 1-5 years. There was only one patient who was more than 15 years. Incidence of PDA was found to be more common in females with male-to-female ratio of 1:2.75. More than 80% of the patients were born full-term. There was only patients born prematurely. Majority of the patients born prematurely became symptomatic within 1 month of life. Parents of the 10 patients gave history of consanguineous marriage. More than 50% became symptomatic between one month and one year of age whereas 16 patients became symptomatic at an age more than 1 year.

Three patients were diagnosed to have PDA at the time of birth. 80% of the total patients were diagnosed to have PDA at an age more than one month. Majority of them were diagnosed at an age more than 1 year. Forty nine (81.7%) patients were symptomatic while 11 patients were asymptomatic.

Symptoms	No. of Patients
SOB	31
RRTI	33
FTT	24
CHF	6
Clinical Signs	
Precordial bulge	18
Hyperactive Precordium	38
Bounding Peripheral Pulses	26
Wide Pulse Pressure	26
Systolic Thrill	31
Loud P2	44
Continuous Murmur	32
Systolic Murmur	20
Diastolic Murmur	22
CHF	6
Table 2. Presentation of Symptoms and Clinical Signs	

Commonest symptoms was recurrent respiratory tract infections followed by symptoms of breathlessness, which was present in 31% of patients. Six patients gave history of congestive cardiac. Majority of the patients were in NYHA class II of class III. Most common clinical signs was accentuated P2. Hyperactive precordium was present in 63.4% patients. Continuous murmur classical of PDA was present in 32 patients, systolic murmur was present in 20 patients, whereas 8 patients did not have any murmur on auscultation.

Chest X-Ray Findings	No. of Patients
Normal	15
Cardiomegaly	27
Plethoric Lung F	43
Dilated PA	11
Dilated A	6
ECG Findings	
Normal	26
LVH	31
BVH	6
LAE	16
Echocardiography PDA Size in MM	
Small (<6 mm)	29
Moderate (6-8 mm)	18
Large (>8 mm)	13
ECHO: Pulmonary Arterial Hypertension	
Mild	26
Moderate	14
Severe	4
Table 3. Investigatory Findings in Study	

Most common finding on the CXR was plethoric lung fields, cardiomegaly was present in 45% of patients and CXR was normal in 15 patients. Left ventricular

hypertrophy/volume overload was present in 52% of the patients.

Echocardiographically, twenty nine patients were diagnosed to have small PDA, eighteen patients had moderate-sized PDA and thirteen patients had large-sized PDA. Most of the patients had only mild PAH (pulmonary artery hypertension), fourteen patients had moderate PAH, only 1 patient had severe PAH.

Echo: Shunt Quantification

Forty patients (70%) had left to right shunt >2:1.

Intraoperative Findings: Size of PDA

Intraoperatively, 31 patients had small PDA, 16 patients had moderate-sized PDA whereas 13 patients had large-sized PDA.

Other Intraoperative Findings

One patient had coarctation of aorta, thirty patients had dilated PA and dilated aorta was present in patients.

Size of PDA	Intraop	Echo
Small	31	29
Moderate	16	18
Large	13	13
Table 4. PDA Size: Intraoperative and ECHO Comparison		

Surgical Techniques Used

Triple ligation of PDA was the most commonly used procedure and was used in 73% of patients.

Complications

1. One patient was diagnosed to have residual shunt postoperatively.
2. Chylothorax was seen in 3 patients.
3. One patient had tear in PDA while dissecting it.

Outcomes

1. The overall mortality in the present study was 3% (patients).
2. In one patient, cause of death was not known while other patient died as a sequelae to tear in the PDA while dissecting it.

DISCUSSION

PDA is not a benign entity, although prolonged survival is possible. The mortality in infants with untreated PDA maybe as high as 30%.

Clinical Profile

Isolated PDA in term infants occurs in approximately 1 in 2000 live births and accounts for 5% to 10% of all types of congenital heart diseases. The incidence increases with prematurity and with decreasing birth weights and maybe more than 80% in infants weighing less than 1000 gm. This fact was also found to be very important in our study. Out of 60 patients with PDA, 10 patients (16.7%) were born prematurely and 14 (23.4) patients had low birth weight.

The increased incidence is related to several factors including decreased smooth muscle in the ductal wall, diminished responsiveness of the ductal smooth muscle to oxygen and possibly elevated levels of circulating vasodilatory prostaglandins.

It is particularly common when the mother contracts rubella during the first trimester of pregnancy and may then be associated with multiple peripheral pulmonary artery stenoses and renal artery stenosis. In the present series of patients, none of the mothers had any history of exanthematous fever. It is twice as common in girls of 2-3 years old and may occur in siblings suggesting a genetic factor. In the present study, the M:F ratio was found to be 1:2.75. Out of 60 patients who were studied, there was history of siblings being affected by similar disease in 2 patients. Out of these two siblings, one was operated at our institute. The history of consanguineous marriage between parents of patient was present in 8 patients (13.4%). Symptoms and sign of a PDA are the consequence of left-to-right shunting with the magnitude of shunt depending on size of the communication and the relationship between the systemic and pulmonary vascular resistances. It also depends on the age at presentation and associated anomalies.⁴ Full-term infants usually do not become symptomatic until the PVR decreases at 6 to 8 weeks of age producing a significant left to right shunt. Because premature infants have less smooth muscle in the pulmonary arterioles, PVR decreases earlier and symptoms may develop during the first week of life.

In the present study, out of 10 patients who were born prematurely, 4 patients became symptomatic within 2-3 weeks of life, another 2 patients became symptomatic within 1 month of life and rest 4 patients became symptomatic after more than a month. Up to 60% of very low birth weight infants (<1000 gm) may show ductal shunting echocardiographically at 2 to 3 days of life without evidence of a murmur or other clinical signs of PDA. Approximately, 40% of these infants eventually develop a haemodynamically significant left to right shunt. Infants with a birth weight greater than 1000 gm have a much lower risk of developing a clinically significant shunt even if a murmur is present. A large haemodynamically significant PDA usually presents in infancy with CHF. Afflicted infants are irritable, tachycardiac and tachypnoeic and tolerate feeding very poorly. Physical examination often reveals a hyperdynamic precordium and bounding peripheral pulses. The systolic blood pressure is usually elevated, but diastolic pressures lowered. Auscultation reveals a systolic or continuous murmur, often called a machinery murmur (Gibson's murmur), which is heard best in the pulmonic area and radiates towards the middle third of the clavicle. A mid diastolic apical murmur maybe present secondary to increased flow across the mitral valve. Absence of these findings, however, does not exclude the presence of a significant PDA, especially in premature infants.

A study of infants with symptomatic PDA found that the most sensitive clinical sign was a hyperdynamic precordium, which was present in 95% of patients. Bounding pulses and

a murmur were absent in 15% and 20% of the patients, respectively. During this time, at GH, we came across only one preterm infant who presented to us with signs and symptoms of congestive heart failure, but did not undergo surgery at our institution. Left-to-right shunt in moderate-sized PDA is regulated by the size of the ductus arteriosus.

In this setting, pulmonary artery pressure is only moderately elevated. As neonatal pulmonary vascular resistance declines, the shunt increases and heart failure may occur by the second or third month of life, however, compensatory left ventricular hypertrophy is usually associated with clinical improvement and stabilisation of symptoms. Physical development maybe somewhat retarded and breathlessness and fatigue may occur, but many individuals with moderate-sized PDA remain essentially asymptomatic until the second decade of life or later.

Whereas, in cases of small PDA, left-to-right shunt is small in early life and pulmonary vascular resistance decreases rapidly to normal after birth. Left ventricular failure does not occur and symptoms are absent in infancy and childhood. They may appear much later in life, but usually attention is drawn to the condition by a murmur detected on routine physical examination. In the present study, 11 patients (18.4%) remained asymptomatic and were diagnosed to have PDA incidentally during routine physical examination. All these patients had small PDA.

These patients were diagnosed to have PDA as early as within 1 month of their birth and as late as 20 yrs. of age. Patients who had small PDA, 65% of them became symptomatic late in their childhood. Patients who became symptomatic in this group had either symptoms of breathlessness or history of recurrent lower respiratory tract infections predominantly. Patients who had symptoms of breathlessness, 90% of them belonged to NYHA-I or CL-II. None of them had history of congestive heart failure. Three patients (5%) had history of infective endocarditis, which resolved with antibiotics. All the patient who had moderate-sized PDA were symptomatic, 56% of those patients developed symptoms between one to two months of life.

Symptoms of breathlessness and history of recurrent respiratory tract infections were the most common symptoms. Nine patients (56%) were in NYHA Class III failure to thrive was present in 40% of patients. More than 75% of patients who had large PDA developed symptoms within 1 month of age. These patients were diagnosed to have PDA much earlier as compared to patients with small and moderate-sized PDA. All the patient in this group had symptoms of breathlessness and history of recurrent respiratory tract infections. Out of 13 patients, 10 patients were in NYHA Class III.

Five patient gave history of congestive cardiac failure at an average age of 3.5 months which was treated medically and patient improved symptomatically. When compared to the published literature, even though the age at the onset of symptoms is same, the diagnosis of PDA in Indian population is made very late. One of the important factors for this delay in diagnosis is socioeconomic. Out of 60 patient studied, 26 patients (43.4%) were diagnosed at an age more

than 1 year. Another 22 patients (36.6%) were diagnosed between the age of 1 month and 1 year. Only 12 patients (20%) were diagnosed within 1 month of age. Absence of routine neonatal medical examination also accounts for this delay in diagnosis.

In patients with large PDA, there is an overactive precordium, sometimes with a systolic thrill and evidence of cardiac enlargement with a thrusting left ventricular apical impulse. The pulse is jerky or frankly collapsing and the pulse pressure is correspondingly wide. These features become more obvious when heart failure is medically controlled. On auscultation, there is a systolic murmur maximal in the pulmonary area with late systolic accentuation and minimal spill over into diastole. Occasionally, the murmur is continuous, but sometimes with severe heart failure, no murmur is heard. The first and second heart sounds are accentuated and there is a third heart sound at the apex or a prominent mid diastolic mitral flow murmur. Patients with moderate-sized ductus will have a jerky pulse, precordium is mildly overactive and the left ventricle is palpable at the apex in association with some cardiac enlargement. Left ventricular volume overload and congestive heart failure can usually be overcome by left ventricular hypertrophy and other compensatory mechanisms.

The classical continuous murmur of PDA was present in 32 patients (53.4%) in the present study and was the third most common clinical findings. The most common clinical findings was accentuated P2 and was present in 44 (73.4%) patients. The most obvious reason for this was the delayed diagnosis when pulmonary arterial hypertension has already set in. The second commonest clinical findings in the present study was hyperactive precordium and was present in 38 (63.4%) patients. A study conducted by Kupferschmid C.H. et al⁵ to study the sensitivity, specificity and predictive value of clinical findings on symptomatic patients with PDA found that most sensitive clinical sign was a hyperdynamic precordium, which was present in 95% of patients. Bounding pulses and a murmur were absent in 15% and 20% of the patients, respectively. In the present study, murmur was absent in 8 patients (13.4%), bounding pulses were present in (43.4%) of patients. A diastolic flow murmur across mitral valve was present in 22 patients (36.7%). Campbell M et al also reported presence of diastolic flow murmur in 42% of patients with PDA.

Investigations

The diagnosis of PDA can often be made noninvasively and physical examination maybe almost diagnostic.

Electrocardiogram

In patients with small PDA, ECG are normal or nearly so. In patients with moderate-sized PDA, ECG may be relatively normal during infancy, but some degree of left ventricular hypertrophy develops in older children. Whereas in patients with large PDA, ECG shows left ventricular enlargement with deep Q and tall R waves in left ventricular leads. There may be evidence of right ventricular hypertrophy with upright T

waves in the right precordial leads and evidence of left atrial enlargement with widened P waves. In the present study, 26 patients (43.4%) had normal ECG. ECG was normal in all the patients who had small PDA whereas only 3 patients with moderate-sized PDA had normal ECG.

Commonest ECG abnormality seen in the present study was left ventricular volume overload and left ventricular hypertrophy, which was present in 31 (51.7%) patients. Twelve patient (92%) out of 13 who had large PDA had above ECG findings whereas 12 patients (75%) out of 16 with moderate-sized PDA has left ventricular volume overload and left ventricular hypertrophy. Sixteen (26.7%) patients had left atrial enlargement on ECG. Six patients (10%) had biventricular hypertrophy on ECG.

Chest X-Ray

Chest x-ray findings associated with PDA include varying degrees of cardiac enlargement, plethoric lung fields, dilated pulmonary artery, dilated aorta, interstitial oedema, left atrial enlargement and left ventricular enlargement. Chest x-ray usually appears normal in patients with small PDA. Whereas moderate-to-large PDA's shows evidence of cardiomegaly, plethoric lung fields, dilated pulmonary artery and dilated ascending aorta. In the present study, chest x-ray was normal in all patients with small PDA, whereas 70% of the patients with moderate PDA and 85% of patient with large PDA showed evidence of cardiomegaly. Most common finding on the CXR in the present study was plethoric lung fields, which was present in 40% of the patients. Chest x-ray was normal in 13 (21.7%) patients. In a series, presented by Higgins et al. 22% of patients with a symptomatic PDA showed no increase in radiologic heart size.

Echocardiography

Echocardiography is the diagnostic method of choice for evaluating the patient with a patent ductus arteriosus. Two dimensional echocardiography and color Doppler can accurately define the anatomy of the PDA, characterise the associated shunt flow, left atrial enlargement, ventricular function and ventricular hypertrophy maybe determined. The presence or absence of associated cardiac malformations may also be assessed. Pulsed wave Doppler reveals additional important information. Examination of the shunt flow through the PDA will reveal the pressure gradient between the aorta and pulmonary arteries. Doppler evaluation velocity of tricuspid regurgitation will often determine further the level of increase in right ventricular and pulmonary artery pressures. The sensitivity of these techniques has made it possible to recognise a subgroup of patients who have a small PDA with normal pulmonary pressures who cannot be identified clinically. In the present study, echocardiogram was diagnostic in all the cases. Echocardiogram diagnosed 29 patients to have small PDA, which correlated very well with intraoperative findings, except 2 patients in whom it was found to be of moderate size. Twenty six patients had mild PAH, 14 patients had

moderate PAH while 4 patients were diagnosed to have severe PAH.

Cardiac Catheterisation

Cardiac catheterisation provides the most accurate physiologic and anatomic evaluation of PDA. However, because most patients can be adequately evaluated by noninvasive methods, the role of cardiac catheterisation in the present day is reserved primarily for the evaluation of patients suspected of having irreversible pulmonary vascular changes (Eisenmenger's Syndrome). In the present study, none of the patients underwent cardiac catheterisation.

Management

The presence of a persistent PDA in a child or adult is a sufficient indication for surgical closure because of increased risk of endocarditis. In symptomatic patients, closure should be performed when the diagnosis is made. In asymptomatic, can be postponed if desired, but should be operated in pre-children. Similar protocol was followed in our institution. The increasing use of echocardiography has revealed a group of patient with clinically silent PDA in which a very small amount of flow can be identified.

Thilen U, Astrom-Olsson K, et al⁶ from Sweden published their study on 270 patients of PDA with respect to infective endocarditis. They reported no incidence of infective endocarditis over an aggregate 1196 years at risk. They concluded at the end of the study that routine closure of a PDA for the sole purpose of eliminating risk of infective endocarditis is unnecessary. Out of 44 patients who underwent triple ligation at our institute, commonly used technique of closure was simple triple ligation without any commonest transfixation PDA was closed by this technique in 38 patients. Whereas in 6 patients, in between 2 simple ligatures, i.e., at PA (pulmonary artery) and aortic ends, a third transfixation suture was used. The more classic technique is the "clamp and divide" technique advocated by Gross." We have used Gross' technique for the division and suturing of PDA at our institution.

The superiority of one method over the other has not been established. There are no case controlled studies in the literature to pursue the superiority of division over ligation. The technique of ligation without division is usually quicker and easier to complete. Generally, less dissection, mobilisation and retraction are required and there is no concern for bleeding from the open ends of the transacted ductus as there is when the patent ductus is divided. For these reasons, the technique of ligation is often preferred. However, there is some concern and controversy as to whether ligation carries increased risks of residual patent ductus arteriosus and recanalisation. Ligation closes the PDA by diminishing the circumference and crumpling the wall into many irregular folds that obliterate the lumen. Inherently, this technique causes significant distortion of the PDA and its surrounding structures, and in fact, often increases the local tissue stresses. Thus, injury and disruption of the wall of the PDA during ligation under pressure are issues of concern.

With the associated pressures, stresses and brittle nature of the wall of the PDA, there is a tendency for a ligature to tear and/or cut into the PDA wall as it is tied down. This can sometimes result in trouble, some bleeding or create the potential for late recanalisation or pseudoaneurysm formation.

In the present series, one patient died as a sequelae of tear in PDA during dissection. Technique of division requires more time and effort. Greater exposure and control are usually needed and thus more dissection, mobilisation and retraction are necessary. Visualisation and separation of the transacted ends of the ducts, however, may provide greater assurance of complete closure and this technique has been advocated as having a lower incidence of residual patency and recanalisation. The open technique is used only rarely, usually inpatients of fifth or sixth decade in whom the PDA is short, heavily calcified and brittle, and in whom degenerative vascular disease makes pulmonary arteries through the left chest difficult. Transaxillary muscle sparing PDA closure performed as a same day procedure was described in 10 patients by Cetta F et al.⁷ They reported very good cosmetic results while obviating the need for thoracostomy tube placement. Closure of the ductus in neonates by applying surgical clips has been described.

There has been interest in video-assisted thoracoscopic interruption of PDA. Rashkind. W.J et al⁸ from Jamaica published their data of PDA closures done over 8 years period and concluded that the treatment options for PDA does not support a change in management strategy in favour of nonsurgical methods and surgery still remains the gold standard in the management of PDA. Surgical closure of PDA maybe complicated by haemorrhage, pneumothorax, chylothorax, left recurrent laryngeal nerve damage and infection. In the present study, only one patient had transient hoarseness of voice, which recovered spontaneously. Three patients (5%) had developed chylothorax, but all the three patients responded to conservative management and none of them required any surgical intervention. None of the patients had bleeding problems postoperatively. One patient had developed pneumothorax after removing ICD tube on postoperative day one and that patient required reinsertion of ICD tube.

The phrenic nerve injury resulting into paralysis of the diaphragm was not seen in the present study. Late complication after closure of a PDA include recanalisation and false aneurysm formation. Recanalisation was considered a significant concern in earlier experiences and was thought to occur in about 3-5% of patients in whom the ductus was ligated. In more recent series, the incidence of recanalisation or residual patency approaches zero. In our series, only one (1.6%) patient had residual PDA, which was diagnosed postoperatively. Patient refused to undergo any procedure and was kept on followup, later lost to follow-up. Dani et al⁹ has described residual PDA in 2 of his patients in a series published in Paediatrics reported 2.7% incidence of residual PDA in their series. There were two deaths in the present study. One patient died on postoperative day 10. Cause of death was not known as postmortem study is not

performed. Another patient died of bleeding from the tear in the wall of PDA at the junction with aorta posteriorly, which could not be controlled.¹⁰

CONCLUSIONS

The PDA is more common in females with female-to-male ratio of 2.75:1. Premature infants with PDA become symptomatic earlier as compared to full-term infants. Because of the late presentation, majority of the patients were symptomatic in the present study and in symptomatic patients, symptoms of breathlessness of varying NYHA Class and recurrent respiratory tract infections were the commonest symptoms. Accentuated pulmonary sound, hyperactive precordium, murmur were the most signs. History and clinical examination does give the diagnosis of PDA, but ultimately needs confirmation by further investigations. Chest x-ray and ECG are the important investigation in the diagnosis of PDA. Echocardiogram was the most commonly used diagnostic modality and was diagnostic in all cases. Echocardiogram does give an accurate assessment of PDA, but ultimately intraoperative assessment of PDA is the most important to decide about the surgical technique of closure.

Presence of ductus itself is an indication for closure. The only contraindication is severe pulmonary vascular disease with or without Eisenmenger's syndrome. To know the reversibility of the pulmonary vascular disease, cardiac catheterisation should be performed in these patients. Even though, different types of techniques have been described for the closure of PDA in the literature, superiority of one technique over another has not been established. Commonly performed procedure for the closure of PDA is triple ligation. The technique of division and suturing is used for short and wide ductus. Type of surgical technique need to be chosen based on morphology of PDA, time of presentation PAH, presence or absence of calcification in PDA. Surgical closure of PDA can be accomplished with low morbidity and mortality.

REFERENCES

1. Evans N. Preterm patent ductus arteriosus: should we treat it? *J Paediatr Child Health* 2012;48(9):753-758.
2. Clyman RI, Couto J, Murphy GM. Patent ductus arteriosus: are current neonatal treatment options better or worse than no treatment at all? *Semin Perinatol* 2012;36(2):123-129.
3. Schena F, Ciarmoli E, Mosca F. Patent ductus arteriosus: wait and see? *Journal of Maternal-Fetal and Neonatal Medicine* 2011;24(Suppl 3):2-4.
4. Hammerman C, Shchors I, Schimmel MS, et al. N-terminal-pro-B-type natriuretic peptide in premature patent ductus arteriosus: a physiologic biomarker, but is it a clinical tool? *Pediatric Cardiology* 2010;31(1):62-65.
5. Kupferschmid CH, Lang D, Pohlandt F. Sensitivity, specificity and predictive value of clinical findings, m-mode echocardiography and continuous-wave doppler sonography in the diagnosis of symptomatic patent ductus arteriosus in preterm infants. *Eur J Pediatr* 1988;147(3):279-282.
6. Thilén U, Aström-Olsson K. Does the risk of infective endarteritis justify routine patent ductus arteriosus closure? *Eur Heart J* 1997;18(3):503-506.
7. Cetta F, Deleon SY, Roughneen PT, et al. Cost-effectiveness of transaxillary muscle-sparing same-day operative closure of patent ductus arteriosus. *Am J Cardiol* 1997;79(9):1281-1282.
8. Rashkind WJ, Mullins CE, Hellenbrand WE, et al. Nonsurgical closure of patent ductus arteriosus: clinical application of the Rashkind PDA occluder system. *Circulation* 1987;75(3):583-592.
9. Dani C, Bertini G, Pezzati M, et al. Prophylactic ibuprofen for the prevention of intraventricular hemorrhage among preterm infants: a multicenter, randomized study. *Pediatrics* 2005;115(6):1529-1535.
10. El-Khuffash A, McNamara PJ, Lapointe A, et al. Adrenal function in preterm infants undergoing patent ductus arteriosus ligation. *Neonatology* 2013;104(1):28-33.