

PATENT DUCTUS ARTERIOSUS: SURGICAL COMPLICATIONS, MORBIDITY AND MORTALITY IN 510 PATIENTS

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ABSTRACT

In this retrospective study, 510 infants and children with patent ductus arteriosus (PDA) who underwent surgery at the Shahid Rajai Heart Hospital in Tehran were evaluated to determine the rate of surgical complications, morbidity and mortality.

30 patients (6%) developed minor to major complications postoperatively. Four patients who had undergone PDA ligation without division required reoperation due to recanalization of the ductus. In one patient, the left pulmonary artery was mistakenly ligated instead of the ductus, requiring reoperation and correction. There were two mortalities (0.4%), both in patients with pulmonary hypertension. Four patients (0.8%) developed bacterial endocarditis with positive blood cultures.

The frequency of complications in our group of patients is in accordance with that of the literature. We conclude that patent ductus arteriosus, when diagnosed and treated early, can yield excellent results with very little complications. Delay in diagnosis and treatment, especially when associated lesions coexist, significantly increases the rate of complications, morbidity and mortality.

MJIRI, Vol. 12, No. 3, 211-215, 1998

Keywords: Ductus arteriosus, patent; surgical complications; morbidity; mortality.

INTRODUCTION

The ductus arteriosus is a large channel found normally in all mammalian fetuses and connects the main pulmonary trunk to the descending aorta about 5 to 10 millimeters distal to the origin of the left subclavian artery. Its physiologic function is to divert blood flow away from the high-resistance pulmonary circulation to the descending aorta and particularly to the low-resistance placental circulation.

Ductus closure is mediated by release of vasoactive substances, variation in pH, and particularly oxygen tension¹ and prostaglandins.² The latter two act in opposite directions,

a rising pO₂ constricting the ductus and prostaglandins relaxing it. The ductus arteriosus is completely closed by 8 weeks of age in 88% of people with an otherwise normal cardiovascular system.³ When the process is delayed, prolonged patency, and when closure ultimately fails, persisting patency of the ductus is said to exist.

PDA is a relative common congenital cardiac anomaly which if diagnosed and treated early yields excellent surgical results.⁴⁻⁸ If diagnosis and treatment are delayed, and especially if associated cardiac anomalies such as left-to-right shunt, the most common of which is ventricular septal defect, or inflow and outflow left heart lesion coexist,

complications such as growth failure, recurrent pulmonary infections, heart failure, bacterial endocarditis and pulmonary hypertension may ensue, which significantly increase surgical risks. Furthermore, delay in treatment, especially when the ductus is large, may lead to pulmonary vascular disease and render the patient inoperable (Eisenmenger syndrome).⁹⁻¹¹

This report presents our experience with surgical results, complications, morbidity and mortality of PDA patients in Iran, and compares them with that in the literature.

MATERIALS AND METHODS

This study was performed retrospectively over a 10-year period from January 1984 to January 1994 on over 2000 patients who referred to the Pediatric Cardiology Department of the Shahid Rajai Heart Hospital in Tehran with patent ductus arteriosus. Diagnosis was made by echocardiography as well as angiography in most cases. All cases underwent surgical correction. 510 cases were randomly chosen from the total number of patients for this study. Patients with complex cardiac lesions and whose PDA had a right-to-left shunt and was necessary to maintain life were excluded.

RESULTS

510 patients underwent surgery for patent ductus arteriosus. Age ranged from 4 months, to 15 years, and 80% were between 2 to 10 years old. 361 patients were female and 149 were male (F:M= 2.4:1). 180 patients (39%) had pulmonary hypertension (PH) (PA pressure > 45 mmHg) and 65 patients (12%) had severe PH (PA pressure near or equal to systemic). 14 patients (3%) had congestive heart failure (CHF). Patients were followed up from 6 months to 9.5 years; 18 patients did not return for follow-up.

416 patients had isolated PDA (82%), and 94 patients (18%) had one or more associated congenital cardiac anomaly as depicted in Table I.

Surgical complications

From 510 patients who underwent surgical closure of their patent ductus arteriosus via standard left lateral thoracotomy, 30 patients (6%) developed various surgical complications (Tables II,III). Four patients who had undergone ligation alone without division required a second operation due to recanalization of the ductus. Three patients developed chylothorax following injury to the thoracic duct, and two patients were returned to the operating room due to excessive postoperative hemorrhage. Four patients developed hemothorax, two patients developed left lung atelectasis, one patient had intraoperative rupture of the



Fig. 1. Postoperative angiography depicting accidental ligation of the left pulmonary artery instead of the patent ductus arteriosus. No dye enters the LPA upon injection into the pulmonary trunk.

Table I. Cardiac anomalies associated with PDA.

Anomaly	No. of patients	Percent
VSD	37	7
Aortic valve anomaly	28	5.6
Mitral valve anomaly	17	3.4
Coarctation of aorta	16	3.2

aorta with hypotension, one patient had rupture of an aneurysmal ductus and one patient had erroneous ligation of the left pulmonary artery instead of the ductus (Fig. 1). There was one case of wound dehiscence and two cases of wound infection.

Morbidity and mortality

From the 510 patients in this study, 4 patients (<1%) developed infective endocarditis with positive blood culture. Isolated etiologic agents are shown in Table IV.

There were two mortalities in the group of 510 patients studied (0.4%). Both mortalities were patients with large PDAs and severe pulmonary hypertension who had presented for treatment very late.

Table II. Major surgical complications in 510 patients with PDA.

Complication	No.
Recurrence of PDA (recanalization)	4
Postoperative bleeding	2
Rupture of aneurysmal ductus	1
Rupture of aorta	1
LPA closure instead of PDA	1

Table III. Minor surgical complications in 510 patients with PDA.

Complication	No.
Hoarseness	5
Hemothorax	4
Chylothorax	3
Wound dehiscence	2
Pneumothorax	2
Left lung atelectasis	2
Postoperative hypertension	3

Table IV. Etiological agents of endocarditis in PDA patients.

Agent	No.
Salmonella	1
<i>Streptococcus viridans</i>	1
Non-hemolytic streptococcus	1
Enterobacter	1

DISCUSSION

The fetal ductus arteriosus is patent at birth, but postnatal closure begins promptly and occurs in two stages. The first stage consists of contraction of the smooth muscle in the media of the wall of the ductus and is complete within 10 to 15 hours in full-term infants. The second stage of closure is the result of fibrous proliferation of the intima and is usually completed by 2 to 3 weeks.¹²

Isolated PDA occurs in about 1 in 2000 term infants and is twice as common in girls. The symptoms and signs in patent ductus arteriosus are the result of left-to-right shunting,

similar to those of other high-pressure shunts. Large PDAs may rapidly lead to congestive heart failure with tachypnea, sweating and irritability, slow weight gain and poor feeding. About 30% of infants born with an isolated PDA die within the first year of life.¹³

Smaller PDAs may have mild or essentially no symptoms for some time after birth and may be discovered incidentally as a continuous murmur on routine physical examination.

Persisting patency of the PDA is an indication for its closure and the optimal time for operation is during the first year of life.¹⁴ Operation in the first few months of life is indicated when symptoms of heart failure are present, otherwise surgery may be delayed until the age of about 6 months if the patient has no symptoms. Life expectancy is normal after surgical closure of an uncomplicated PDA in infancy or childhood. When moderate or severe pulmonary vascular disease has developed preoperatively, late deaths may result from its progression. Severe pulmonary vascular disease is a contraindication to closure.

Most patients with PDA are now operated in infancy or childhood, and the probability of early postoperative death is very low and approaches zero. However, various complications have been reported following surgical closure.

Chylothorax may rarely occur due to injury to the thoracic duct during dissection or manipulation of the ductus.¹⁵ Treatment usually consists of tube thoracostomy drainage and replacement of lost nutrients and surgical intervention is rarely required. Three of our patients developed this complication (<1%) which is in accord with reports in the literature. They recovered with conservative management.

Ductal aneurysm may occur as a distinct entity¹⁶ or rarely as a complication following surgery, usually in the form of a false aneurysm, especially in cases with recanalization following ligation or in the setting of infection or endocarditis or in patients with elevated pulmonary artery pressure.¹⁷ If following operation an opacity is seen in the vicinity of the aortic knob on x-ray, this complication should be suspected and requires urgent operation. In our series, one case of ductus aneurysm was seen which led to rupture and hemorrhage during surgery but was successfully controlled and the patient survived.

Transient left vocal cord paralysis and hoarseness may occur uncommonly following surgery and results from manipulation of the left recurrent laryngeal nerve. Five patients with this complication were seen in our series and all recovered spontaneously.

Phrenic nerve paralysis may uncommonly be seen following surgery, with resultant elevation of the hemidiaphragm and impaired ventilation. It usually occurs on the left side and nearly always in infants and may or may not be due to surgical injury. Most reported cases have regressed spontaneously. No cases were seen in our series.

Pneumothorax or pneumomediastinum is another rare

complication following surgery which if unrecognized can be fatal. Two cases occurred in our group of patients.

Pulmonary hypertension usually regresses following surgical correction of PDA, unless pulmonary vascular obstructive disease was present before operation or an undiagnosed left sided heart lesion coexists.¹⁸ In our group of patients, due to exclusion of cases who were considered inoperable due to severe pulmonary hypertension and cases with pulmonary vascular disease, no cases of late, non-regressing pulmonary hypertension were noted.

Recurrence of ductal patency currently approaches zero when appropriate techniques are used, but has been reported in earlier patients who underwent ligation of the ductus alone without division. This may be seen in patients with elevated pulmonary artery pressure who undergo ligation alone to decrease the risk of hemorrhage,¹⁹ or in ill infants to decrease operation time as much as possible. We had four cases of recurrence in our series, three of whom had undergone ligation alone due to severely elevated pulmonary artery pressure.

There was one case of erroneous ligation of the left pulmonary artery instead of the ductus in our series. In a 27 year study comprising 7 centers, 10 cases in whom the left pulmonary artery was mistakenly ligated were found. Most of these patients had abnormal anatomy of the ductus.²⁰

The two mortalities which occurred in our series were both cases with severe pulmonary hypertension and elevated surgical risk.^{4,11} This number is quite low considering that 94 of our 510 cases had associated cardiac lesions. The mortality rate for simple isolated PDA has been reported to be less than 1% which is in accordance with our results.²¹

The risk of bacterial endocarditis is always present in patients with PDA, but regresses about 6 months postoperatively and prophylaxis is no longer necessary unless there is a residual shunt. Four patients in our series developed endocarditis, each with a different etiological agent. The most common organism responsible for endocarditis was *Streptococcus viridans* followed by *Staphylococcus aureus* in one report.²²

Generally, surgery for patent ductus arteriosus is among the safest and most complete operations for congenital heart disease. In cases with isolated PDA the risks and complications are negligible, but with increased pulmonary artery pressure, abnormal anatomy, associated anomalies and complications such as endocarditis or calcification, prematurity or pulmonary disease, surgical risk is increased.¹⁹ In our series, complications were seen mainly in patients with associated anomalies or elevated pulmonary artery pressure. Despite this, the risk of each of the above complications for simple isolated PDA is less than 1%²³ and this is in accordance with our results.

REFERENCES

1. McMurphy DM, Heymann MA, Rudolph AM, Melmon KL: Developmental change in constriction of the ductus arteriosus: response to oxygen and vasoactive substances in the isolated ductus arteriosus of the fetal lamb. *Pediatr Res* 6: 231, 1976.
2. Clyman RI, Heymann MA: Pharmacology of the ductus arteriosus. *Pediatr Clin North Am* 28: 77, 1981.
3. Christie A: Normal closing time of the foramen ovale and the ductus arteriosus. *Am J Dis Child* 40: 323, 1930.
4. Satur CR: Day case ligation of PDA in preterm infants: a 10 year review. *Arch Dis Child* 66(4): 477-80, 1991.
5. Brook MM: Patent ductus arteriosus. In: Emmanouilides G, (ed). *Moss and Adams Heart Disease in Infants, Children and Adolescents*. Fifth edition, Baltimore: Williams and Wilkins, pp. 746-68, 1995.
6. Perloff J: *The Clinical Recognition of Congenital Heart Disease*. Fourth Edition, Philadelphia: W.B. Saunders Co., pp. 510-535, 1994.
7. Hang CL: Patent ductus arteriosus presenting in old age. *Cathet Cardiovasc Diag* 28(3): 228-30, 1993.
8. Barst RY: The pharmacological treatment of patent ductus arteriosus. A review of the evidence. *Drugs* 38(2): 249-66, 1989.
9. Mullins CE: PDA. In: Garson Jr A, (ed). *The Science and Practice of Pediatric Cardiology*. Boston: Lea & Febiger, pp. 1059-1065, 1990.
10. Yilmaz AT: Ligation in adults of persisting ductus arteriosus. *J Cardiovasc Surg Torino* 32(5): 575-80, 1991.
11. Nihill MR: Clinical management of patients with pulmonary hypertension. In: Emmanouilides G, (ed). *Moss and Adams Heart Disease in Infants, Children and Adolescents*. Fifth edition, Baltimore: Williams and Wilkins, p. 1695, 1995.
12. Cassels DE: *The Ductus Arteriosus*. Springfield, IL: Charles C. Thomas Publishers, p. 75, 1973.
13. Hay JD: Population and clinic studies of congenital heart disease in Liverpool. *Br Med J* 2: 661, 1966.
14. Ash R, Fischer D: Manifestations and results of treatment of patent ductus arteriosus in infancy and childhood. An analysis of 138 cases. *Pediatrics* 16: 695, 1995.
15. Kausel GW, et al: Anatomic and pathologic studies of the thoracic duct. *J Thoracic Surg* 34: 631, 1957.
16. Kalantar Motamedi MH: Huge aneurysm of the ductus arteriosus in childhood: case report and literature review. *Med J Islamic Republic Iran* 11(1): 49-52, 1997.
17. Rees RS, Feber FP, Spencer FC: Aneurysm of the previously ligated patent ductus arteriosus. *Circulation* 23: 350, 1961.
18. Morris MH, McNamara PG: Residue, sequelae and complications of heart surgery. In: Garson Jr A, (ed), *The Science and Practice of Pediatric Cardiology*. Boston: Lea & Febiger, pp. 2626-28, 1990.
19. Eagner HR, et al: Surgical closure of patent ductus arteriosus in 268 preterm infants. *J Thorac Cardiovasc Surg* 87: 870, 1984.

20. Robert G, Poatius MM, et al: Illusions leading to surgical closure of the distal left pulmonary artery instead of the ductus arteriosus. *Thorac Cardiovasc Surg* 82: 109-13, 1981.
21. Panagopoulos PG, et al: Patent ductus arteriosus in infants and children. *Thorax* 26: 137, 1971.
22. Johnson DH, Rosenthal A, Nadas A: A forty year review of bacterial endocarditis in infancy and childhood. *Circulation* 51(4): 581-88, 1975.
23. Nelson RJ, Thibeault DW, Emmanouilides GC, Lippmann M: Improving the results of ligation of patent ductus arteriosus in small preterm infants. *J Thorac Cardiovasc Surg* 71: 169, 1976.

