Recurrence of Patency of the Ductus Arteriosus After Surgical Ligation in Premature Infants

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ABSTRACT. Two premature infants who had surgical ligation of their patent ductus arteriosus are described. These infants initially did well postoperatively but then developed congestive heart failure. Both infants had echocardiographic evidence of recurrence of their patent ductus arteriosus. One of the infants required a repeat ligation procedure. It is important to continue to monitor premature infants for the return of clinical signs of a patent ductus arteriosus after surgical ligation.

The patent ductus arteriosus (PDA) is a common problem for premature infants. It has been reported that the incidence of PDA is 77% for infants of 28 to 30 weeks' gestation, 44% for infants of 31 to 32 weeks' gestation, and 21% for those of 34 to 36 weeks' gestation. When the PDA is large enough to be hemodynamically significant, there is a large left-to-right shunt which may lead to congestive heart failure with pulmonary edema. Whereas the ductus arteriosus will close spontaneously in many infants, persistent patency, with evidence of congestive heart failure, is an indication for either pharmacologic or surgical closure. We describe two newborns who had surgical closure of their PDA and subsequently developed signs of congestive heart failure. These infants were found to have recurrence of their PDA.

CASE REPORTS

Case 1

L.A. was the first of female twins born to a 28-year-old primigravida. The infant's gestational age was 29 weeks and birth weight was 970 g. Apgar score was 5 at one minute and 8 at five minutes. Within the first hour of life, the infant's respiratory effort was felt to be inadequate so she was intubated, ventilated, and transferred to the Children's Hospital Medical Center. On the second day of life, she developed the physical signs of a PDA. Echocardiography performed on days 3, 4, and 5 of life showed a large left atrial dimension (LAD = 0.9 cm, Ao = 0.6 cm) and left ventricular dimension (LVED = 1.3 cm); decreased left ventricular systolic time interval ratio (LVSTI = .28), and increased shortening fraction (SF = 42%), indicating a hemodynamically significant PDA. Because of increasing heart failure, despite medical treatment with fluid restriction and diuretics, surgical ligation of the ductus arteriosus was performed on the sixth day of life. The chest was entered by an anterolateral thoracotomy. The pleura was sharply incised over the aorta and reflected medially to uncover the PDA. The vagus nerve and recurrent laryngeal nerve were identified and dissected from the ductus arteriosus and protected. Using blunt dissection, the ductus arteriosus was encircled and doubly ligated with two sutures of 2-0 silk. A chest tube was then placed in the left pleural space through a separate stab wound and the incision was closed.

The infant improved clinically after the ligation. She was progressively weaned from the respirator and fluid administration was liberalized. On day 14 of life, a repeat echocardiogram was performed which showed improvement (LAD = 0.7 cm, Ao = 0.5 cm, LVED = 1.0 cm, LVSTI = 0.33, SF = 30%). On the 26th day of life, the infant again developed the clinical signs of PDA with increased pulse volume and an intermittent systolic murmur at the left upper sternal border. On the 27th day of life, clinical signs of congestive heart failure developed with increased liver size and decreasing urine output. On chest radiograph, the heart size was increased. On day 28 of life, an echocardiogram again showed large left atrial and left ventricular dimensions (LAD = 0.9 cm, LVED = 1.3 cm, Ao = 0.6 cm) and decreased LVSTI ratio (0.26). Two-dimensional echocardiography with Doppler showed a continuous murmur of ductal flow in the pulmonary artery in the area of the ductus. Despite medical treatment with fluid restriction and diuretics, the baby con-
Continued to have congestive heart failure. On the 32nd day of life, the infant was taken back to the operating room for repeat ligation of the PDA. A skin incision was made over the old scar through the skin, subcutaneous tissues, and lateral chest wall muscles. The area of the ductus was immediately identified. The sutures, which had been placed approximately 2 mm apart on the ductus, had loosened and the ductus appeared to be patent. Another 2-0 silk suture was placed around the patent ductus and was tied down. A silver clip was placed between the old sutures and the new suture on the ductus to obliterate the ductal lumen. After the second operation, the baby required no further diuretics and had no further signs of a patent ductus or congestive heart failure. Unfortunately, about 1 week after the second operation, the infant’s chronic lung disease worsened with a steady increase in PCO₂. She then began having episodes of bradycardia and hypotension with the development of metabolic as well as respiratory acidosis. On the 52nd day of life, there was a gradual worsening of her condition. She developed severe acidosis and then suffered a hypotensive episode from which she could not be resuscitated.

Case 2

J.H. was the second of twins. She was an 1,150-g female infant born after 28 weeks of gestation to a 24-year-old primigravida. The infant was delivered vaginally in the obstetrician’s office and no Apgar score was assigned. She was noted to have immediate respiratory distress but was pink in 50% oxygen. Both infants were referred to a community hospital where their condition deteriorated, necessitating intubation and ventilation. The infants were referred to Children’s Hospital Medical Center for further evaluation and therapy.

The infant’s course was complicated by moderately severe respiratory distress, necessitating increase in ventilatory support during the first 48 hours of life. On the third day of life, the infant developed the clinical signs of a PDA with an active precordial impulse, continuous murmur at the upper left sternal border, and bounding pulses. An echocardiogram demonstrated enlarged left atrial and left ventricular dimensions (LAD = 0.7 cm, Ao = 0.5 cm, LVED = 1.2 cm). The left ventricular systolic time interval ratio was decreased (LVSTI = 0.25), consistent with ductal runoff, and the shortening fraction (SF = 33%) was normal. The patient was maintained in negative fluid balance and was 12% below birth weight on the third hospital day. Her clinical status gradually improved with the slow weaning of ventilatory support. However, on day 7 of life, the patient’s condition deteriorated. A repeat echocardiogram showed further increases in left atrial and left ventricular dimensions (LAD = 1.0 cm, Ao = 0.5 cm, LVED = 1.5 cm). Her physical examination remained unchanged. On the ninth day of life, when there was no improvement in the patient’s condition, she was taken to the operating room for a ductal ligation. The patent ductus was exposed and was noted to have an external dimension about the same size as the aorta. Two 2-0 silk sutures were placed around the ductus and tied down. A chest tube was then placed in the pleural space and the incision was closed.

Following ligation, the patient tolerated weaning of the ventilator and was extubated 1 week later. However, on the eighth postoperative day, the patient developed increased respiratory distress, necessitating reintubation. Further attempts at weaning from the respirator were unsuccessful. Approximately 3 weeks after the PDA ligation, a grade II/VI long systolic murmur was heard at the upper left sternal edge. Precordial activity and pulses were again noted to be increased. Concomitantly, the patient was noted to have a 200-g weight gain for a three-day period of time. The clinical impression of congestive heart failure was supported by a chest radiograph, which showed increased heart size and a diffuse pulmonary edema pattern of lung markings. An echocardiogram done at that time demonstrated an enlarged left atrial dimension (LAD = 1.5 cm, Ao = 0.6 cm, LVED = 1.1 cm). The LVSTI ratio was decreased (0.23), and the SF (32%) was normal. A two-dimensional echocardiographic study with Doppler revealed evidence of diastolic flow in the pulmonary artery compatible with flow through a PDA. This study was repeated three days later with similar findings. A regimen of vigorous fluid restriction and diuretic therapy was begun. The infant’s hematocrit was also noted to be 30% so transfusion was performed to a hematocrit of 46%. For the next 2 weeks, the patient’s clinical course gradually improved and she tolerated weaning from the respirator. At the same time the cardiac murmurs and other physical findings of ductal runoff resolved. The remainder of her hospital course was complicated by hydrocephalus which required placement of a ventriculoperitoneal shunt. She was discharged from the hospital at 4 months of age with nasal oxygen supplementation. A follow-up echocardiogram done prior to discharge showed resolution of the left atrial dilation (LAD = 1.0, Ao = 0.7) and return of the LVSTI ratio to normal (0.31). There was no evidence of ductal flow by Doppler.

DISCUSSION

The first successful surgical closure of a patent ductus was performed in 1938. A simple ligation was used in this procedure. When a number of instances of recurrent patency were observed, some surgeons changed the technique to include division of the PDA. The operation in premature infants differs from that performed in older children and adults. The ductal tissue is friable in premature infants, and this makes the division of the PDA technically more difficult and inadvisable in these sick patients. For this reason the ductus is usually ligated with one or two ligatures in premature infants.

Recurrence of the PDA after medical treatment with indomethacin is well known, and this may lead to several courses of indomethacin therapy. To our knowledge this problem has not been previously reported after surgical ligation in premature infants. Lam described six patients with recurrent PDA after simple ligations. The youngest patient in his series was 6 months old at the time of surgery.
Eyster et al described a series of 140 consecutive PDA ligations in dogs over a 10-year period. In this series, three animals (2%) had recurrent PDA after surgery. One animal had another recurrence after a second ligation procedure.

We present these cases to alert physicians to the possibility that reappearance of heart failure after surgical ligation of a PDA may be due to recurrence of the PDA. It is important to continue to monitor premature infants for the return of clinical signs of PDA and congestive heart failure after surgical ligation of the PDA. If recurrence is suspected, then two-dimensional echocardiography with Doppler may be helpful in establishing the diagnosis of recurrence of the PDA. Once the diagnosis of recurrent PDA is made vigorous medical treatment should be instituted. If medical management fails, then repeat ligation should be considered.

REFERENCES

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