Transcatheter Closure of a Huge Ductus Arteriosus in a Severely Ill Neonate

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Summary

Hemodynamically significant patent ductus arteriosus (PDA) in preterm infants increases morbidity and mortality. Here we describe a 12-day-old neonate with a huge PDA who developed pulmonary hemorrhage following disseminated intravascular clotting and multiple organ failure. Medical treatment or surgical ligation could not be performed because of the patient’s poor condition. Transcatheter closure using a commercially available device (Amplatzer Vascular Plug II) successfully treated the huge PDA without major complications. The Amplatzer Vascular Plug II approach might become a new option for PDA closure in small infants, including those who are critically ill.

Key words: Catheter intervention, Disseminated intravascular clotting, Heart failure, Vascular plug

Case Report

A 12-day-old male neonate was referred to our hospital for the treatment of pulmonary hemorrhage, DIC, and MOF. He was born in a state of asphyxia at 37 weeks of gestation. His birth weight was 2,860 g. At birth, severe pulmonary hypertension, right-to-left directional shunt of patent foramen ovale and PDA, severe thrombocytopenia, and an elevated level of D-dimer were noted. Hence, he was diagnosed with persistent pulmonary hypertension of the newborn and DIC. He received artificial respiration, nitric oxide inhalation, exogenous surfactant, catecholamine, fresh frozen plasma, and recombinant thrombomodulin, but clinical relief could not be achieved. The PDA did not show progress toward closure, and its direction turned to the left-to-right shunt. On the 12th day after birth, pulmonary hemorrhage suddenly occurred and DIC worsened, and the patient was transferred to our hospital on the same day. On admission, he showed forced breathing and peripheral coldness. His vital signs were as follows: blood pressure, 72/38 mmHg; pulse rate, 189 beats/minute; respiratory rate, 60 breaths/minute; and body temperature, 39.0℃. Hematological parameters were as follows: platelet count, 53 x 10^9/L; international normalized ratio of prothrombin time, 3.70; activated partial thromboplastin time, 64.6 seconds (control: 30.0 seconds); fibrinogen level, 54 mg/dL; and fibrin degradation product level, 61.5 μg/mL. These findings satisfied the diagnostic criteria of DIC. Cardiac incompetence and increased serum levels of deviation enzymes (aminotransferase, 2,772...
IU/L; alanine aminotransferase, 188 IU/L; and lactate dehydrogenase, 9,151 IU/L) suggested MOF. Chest radiography revealed cardiomegaly, pulmonary congestion, and hemorrhage (Figure 1A). Echocardiography revealed a huge PDA (the smallest PDA diameter, aortic ampulla size, and PDA length were 4.6 mm, 7.8 mm, and 13.2 mm, respectively) with a left-to-right shunt. An increase in right ventricular pressure of 55 mmHg was estimated on the basis of a Doppler measurement (Figure 2). Although there was no doubt that the pulmonary hemorrhage due to the huge PDA triggered the exacerbation of his clinical condition, patient’s serious susceptibility to bleeding excluded surgical treatment. Hence, we attempted a transcatheter PDA closure. This was done using AVP II because the existing ADO device might damage the descending aorta. The desirable ADO size was estimated to be at least 10/8, and its retention skirt would be larger than 16 mm. Written informed consent to transcatheter PDA closure using AVP II was obtained from the patient’s parents. Immediately before the procedure, a prophylactic antibiotic (cefazolin, 20 mg/kg) was administered to prevent infectious endocarditis. A 5-Fr introducer sheath was placed in the right femoral vein. Intravenous heparin was administered to maintain an activated clotting time.

Figure 1. Chest radiographs obtained before and after PDA closure. A: Before treatment. Remarkable cardiomegaly (cardiothoracic ratio 0.67), pulmonary congestion, and pulmonary hemorrhage are observed. B: The day after PDA occlusion. Pulmonary congestion and hemorrhage show dramatic improvement (cardiothoracic ratio 0.55). PDA indicates patent ductus arteriosus.

Figure 2. Two-dimensional echocardiogram of the patient at admission. The asterisk indicates PDA. The smallest diameter, aortic ampulla size, and PDA length are 4.6 mm, 7.8 mm, and 13.2 mm, respectively. Ao, aorta; DscAo, descending aorta; MPA, main pulmonary artery; PDA, patent ductus arteriosus; RPA, right pulmonary artery.
been noted since then (Figure 4). Stenosis nor aortic obstruction associated with A VP II has sequelae. Neither significant secondary left pulmonary discharged from the hospital on day 65 without serious tion dramatically improved (Figure 1B). The patient was catheter intervention, pulmonary hemorrhage and congestion was started to avoid renal failure. On the day after the hemodialysis catheter, and continuous hemodiafiltration sheath in the right femoral vein was exchanged for a 6-Fr C). After successful device closure of the PDA, the 5-Fr was completely occluded without adverse events (Figure 3 A). Hence, an 8 mm AVP II was placed as previously described. The ductus was completely occluded without adverse events (Figure 3 C). After successful device closure of the PDA, the 5-Fr sheath in the right femoral vein was exchanged for a 6-Fr hemodialysis catheter, and continuous hemodiafiltration was started to avoid renal failure. On the day after the catheter intervention, pulmonary hemorrhage and congestion dramatically improved (Figure 1B). The patient was discharged from the hospital on day 65 without serious sequelae. Neither significant secondary left pulmonary stenosis nor aortic obstruction associated with AVP II has been noted since then (Figure 4).

**Discussion**

Hemodynamically impaired neonates with PDA occasionally show gastrointestinal, cerebral, and pulmonary complications and morbidities. Surgical ligation of the ductus remains very risky in such neonates, although advances in perioperative intensive care have enabled cardiac surgery even in premature infants. Several studies demonstrated feasibility and efficacy of early cardiac catheterization and transcatheter interventions in severely ill neonates with congenital heart disease. In the present case, we successfully treated hemodynamically significant PDA using AVP II in a neonate with DIC and MOF. For transcatheter PDA closure in neonates, ADO II Additional Sizes (ADO II AS; St. Jude Medical Inc.) may be recommended, but this device cannot be used in some countries, such as Japan and the United States.

Recently, successful transcatheter PDA closure using a commercially available device has been reported. Zahn et al. first reported transcatheter PDA closure using AVP II in extremely premature neonates. In their study, 6 preterm infants underwent transcatheter PDA closure using AVP II. The median gestational age, procedure age, birth weight, and procedure weight were 26 weeks, 20 days, 953 g, and 1,180 g, respectively. Complete closure was achieved in all patients, and there were no major procedural complications. However, 2 correctable device-related incidents occurred; one was caused by oversize of the device and the other by device migration. Although other similar studies have reported relatively favorable results, there were several complications, such as left pulmonary stenosis, aortic obstruction, and femoral arterial injury. Backes, et al. reported that 6 of 48 infants (13%) could not undergo transcatheter PDA closure using AVP II because of left pulmonary artery or aortic obstruction. All these PDAs had Krichen type C morphology. Schwartz, et al. reported that the ratio of minimum PDA diameter/length was >0.5 in all unsuccessful cases and <0.4 in all successful cases. In the present case, we performed successful PDA closure using AVP II even though the PDA was of type C (a diameter/length ratio of <0.4). Further research is needed to validate the results of these previous studies. Nevertheless, these results are useful for planning and performing transcatheter PDA closure in low-weight infants.

In catheter procedures in small infants, one of the major concerns is renal function. In newborns, the use of contrast media increases the risk of worsening the immature renal function, especially when the patient is very ill. Several authors reported successful device implantation using fluoroscopy and echocardiography but not contrast angiography. However, we decided to perform AVP II implantation using contrast angiography because PDA closure under echocardiographic guidance was unfamiliar to us, and such an intervention would require the knowledge of precise PDA morphology and an exact device size.

In summary, we demonstrated that transcatheter PDA closure using AVP II is a feasible and safe treatment approach in neonates with DIC and/or MOF. It is particu-
Figure 4. Two-dimensional echocardiogram of the patient after discharge. The asterisk indicates the AVP II deployed in the patent ductus arteriosus. There is no significant secondary left pulmonary stenosis (A) or aortic obstruction (B) associated with AVP II. The flow velocities of the left pulmonary artery and descending thoracic aorta are 1.5 and 1.4 m/second, respectively (data not shown). Ao, aorta; AscAo, ascending aorta; AVP II, Amplatzer Vascular Plug II; LPA, left pulmonary artery; MPA, main pulmonary artery; RPA, right pulmonary artery.

particularly useful in small infants who have a risk of device-related aortic obstruction. The AVP II approach may become a new option for PDA closure in small infants, including those who are critically ill.

Disclosures

Conflicts of interest: The authors declare no conflict of interest.

Author contributions: S.O. and J.M. contributed to the conception and design of this study, drafted the manuscript, and carried out the whole study process. Y.N., C.Y, J.O., J.Y., M.W., C.I., and H.S. contributed to the conception and design of this study and critically reviewed the manuscript. Y.T. critically reviewed the manuscript and supervised the whole study process. All authors read and approved the final manuscript.
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