Case Report

Flow Reduction of a Neonatal Stented Arterial Duct by Covered Coronary Stent

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The final flow rate through a stented duct is variable and depends on stent diameter, ductal length, and ductal tissue protrusion after ductal constriction. Typically after duct stenting, there is initially mild overperfusion which may require some antifailure treatment. Severe heart failure is uncommon, but in some cases flow reduction is required. We present a case of overperfusion after arterial duct stent implantation which was successfully managed with implantation of covered stents. © 2015 Wiley Periodicals, Inc.

Key words: arterial duct stent; overflow; flow reduction; covered coronary stent

INTRODUCTION

Stenting the arterial duct is an alternative to surgical palliation in newborn patients with duct-dependent pulmonary circulation [1,2]. However, the final flow rate through the stent is variable and depends on stent diameter, ductal length around the stent, and protrusion of ductal tissue through the struts after ductal constriction [3]. This article describes a case of successful pulmonary flow reduction using graft coronary stents in a patient with pulmonary overperfusion after arterial duct stent implantation.

CASE

A 2-day-old female weighing 3.2 kg presented with cyanosis since birth. Echocardiographic examination showed membranous pulmonary atresia (PA) with intact ventricular septum, hypoplastic and hypertensive tripartite right ventricle, and good-sized, confluent pulmonary arteries being filled exclusively from the arterial duct. The patient was taken to the catheterization laboratory for perforation of the pulmonary valve and stenting of the arterial duct (Fig. 1A and B). Following perforation and balloon dilatation of the pulmonary valve, the arterial duct was stented with a coronary stent (20 mm × 4 mm monorail system). Systemic arterial saturation stabilized at 91%. In the following days, the patient developed marked tachycardia and tachypnea due to pulmonary overflow. Fluid restriction was applied and diuretics were started. After three weeks of conservative management, the clinical status remained unchanged with pulmonary overflow requiring ventilator assistance (nasal CPAP). The patient was taken to the catheterization laboratory for flow reduction. There was minimal antegrade flow through the pulmonary valve. The duct was short (8 mm) which was accentuated by non-constriction of the ampulla at the aortic end. There was only minimal protrusion of ductal tissue into the stent (Fig. 1C and D). Through a long 4-Fr sheath (Flexor, COOK, Bjaeverskov, Denmark), a graft-covered coronary stent (3.5 mm ×12 mm) (BeGraft coronary stent, Bentley InnoMed GmbH, Hechingen, Germany) was inserted within the first stent to lengthen the tube. The BeGraft consists of a cobalt-chrome stent with a micro-porous expanded polytetrafluoroethylene membrane; the premounted

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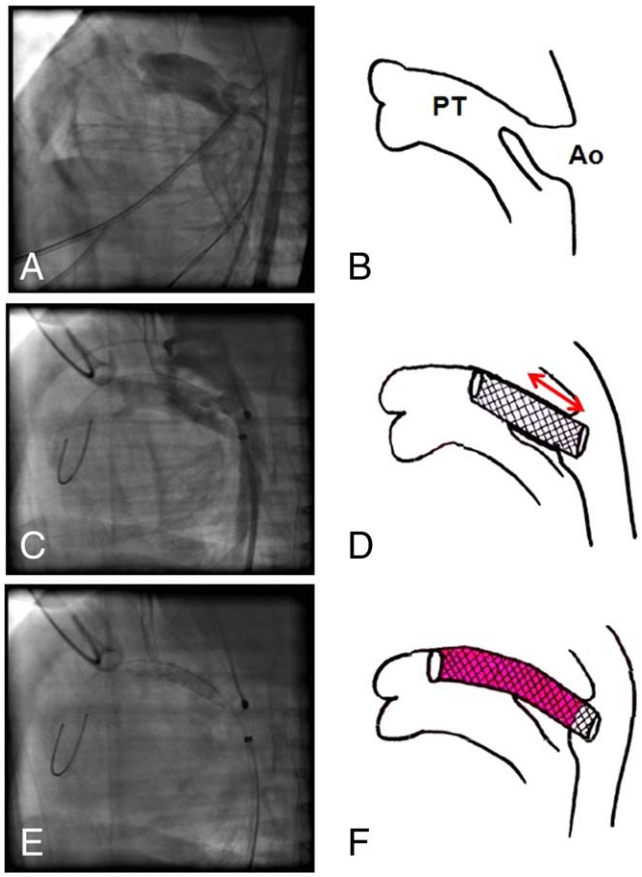

Fig. 1. A: Aortogram showing the arterial duct originating from the descending aorta. The pulmonary end of the duct is constricted down to 3.5 mm; the aortic end is wide open. B: Schematic representation of (A). Ao: thoracic aorta; PT: pulmonary trunc. C: Aortogram at second procedure through the 4 Fr long sheath: the duct has closed around the stent except for the aortic ampulla, leaving bare stent at both ends, creating an effective tube of only 8 mm (arrow). There is minimal protrusion of ductal tissue through the struts. D: Schematic representation of (C). E: Lateral fluoroscopic image after implantation of two covered BeGraft stents: the tube has been lengthened from 8 mm to 17 mm, and the diameter decreased from 4.0 till 3.5 mm. F: Schematic representation of (E). Colored area represents two covered stents telescopically inserted. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

stent has a 4 Fr profile, wall thickness of 0.10 mm. Because of insufficient effect (saturation 90%), a second covered stent (3.0 mm × 12 mm) was inserted to achieve a decrease of duct diameter; while advancing the second covered stent the first was pushed slightly forward. The effective tube length increased from 8 up to 17 mm, while reducing the diameter down to 3.0 to 3.5 mm (Fig. 1E and F). The saturation dropped to 72%, the stents were then dilated with a 3.5 mm balloon resulting in a final saturation of 82%. Pulmonary overflow resolved and the patient was discharged in good condition five days later. The stented duct provided good lung perfusion with adequate growth of the pulmonary arteries; a Glenn shunt was performed 5 months later.

DISCUSSION

The final flow through the stented arterial duct is difficult to predict as it depends on many variables which change with a different time-course. After ductal stenting, flow depends on stent diameter and ductal length. The stent should cover the full length of the duct, however when placed the stent will be held typically only at the constricted area of the pulmonary end. In the following days, the remainder part of the duct will constrict around of the stent which then determines the stented restrictive ductal length. The stent lumen will further decrease due to ductal tissue protrusion through the stent struts. On the other hand, pulmonary vascular resistance decreases which will increase flow through the stented duct. Typically after duct stenting there is initially mild overperfusion requiring some anti-failure treatment; the patient typically outgrows his stented duct within weeks. Severe heart failure due to pulmonary overflow is uncommon [3,4], but in some cases the flow rate is too high and reduction is required.

There are three primary factors that determine the resistance to blood flow within a single vessel: vessel diameter (or radius), vessel length, and viscosity of the blood. Vessel resistance (R) is directly proportional to the length (L) of the vessel and the viscosity (η) of the blood, and inversely proportional to the radius to the fourth power (r^4). Therefore, doubling the length of a vessel will double the resistance to flow (Poiseuille's equation). In our case, the reason for pulmonary overperfusion was a short arterial duct and minimal protrusion of ductal tissue into the stent.

In this situation there are a few treatment options. Medical treatment which consists of fluid restriction, diuretics, angiotensin-converting enzyme inhibitors, and blood transfusion to increase viscosity had an insufficient clinical result. The other main options are to replace the shunt or decrease the ductal stent diameter by adding several stents (Russian Doll technique) or to increase the length of the tube. Because coronary stents are thin, multiple stents would be required to decrease the stent diameter. Increasing the length of the tube can be achieved by using a covered stent. In this way, reducing the diameter and lengthening the duct caused the resistance to increase and the flow rate to decrease. We lengthened the duct with two stents; in retrospect the same result might have been obtained with a single long stent (length available up to 24 mm). This technique may be complicated by excessive protrusion of the covered stent at either end, causing

aortic subobstruction or pulmonary valve dysfunction; as there is no real stenosis in the previous stent, the covered stent may slip and embolize; compression of the origin of either the right or left branch pulmonary artery is unlikely in this high flow situation.

Flow reduction with covered stent implantation has been described in transjugular intrahepatic porto-systemic shunts (TIPS) [5] and in aorto-pulmonary shunts [6,7].

CONCLUSION

Despite the use of an appropriately-sized arterial duct stent, overperfusion may occur due to insufficient ductal changes after stent implantation such as incomplete constriction of the arterial duct around the stent and minimal ductal tissue protrusion into the stent. If medical treatments fails, a graft-covered coronary stent can be used to safely reduce the flow rate.

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