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Left main coronary artery stenting in a 3.6 kg infant after arterial switch operation for transposition of the great arteries

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ABSTRACT

The patient presented with flash pulmonary edema related to severe left ventricular diastolic dysfunction several weeks after arterial switch operation (ASO) for D-transposition of the great arteries. Long segment, critical left main coronary artery stenosis in this 3.6 kilogram infant was successfully stented and resulted in resolution of the clinical findings. At 15-month follow-up, the patient remains asymptomatic and thriving.

Keywords: Arterial switch operation, coronary artery stent in infant, flash pulmonary edema in infant, percutaneous coronary intervention infants, transposition great arteries

INTRODUCTION

Percutaneous coronary intervention (PCI) though widely employed in adults, is rare in small children and infants.[1] We report successful left main coronary artery (LMCA) stenting in a 3.6 kilogram, 10-week-old infant with an unusual clinical presentation caused by critical, long-segment LMCA stenosis after arterial switch operation.

CASE REPORT

A 3.25 kg male infant with double outlet right ventricle, ventricular septal defect (VSD) with side-by-side great vessels and D-malposed great vessels (Taussig–Bing), underwent an arterial switch operation (ASO) and VSD closure at 4 days of age. The right coronary artery (RCA) was normal, but the left main coronary artery (LMCA) arose anomalously superior to the commissure between the posterior and left lateral sinuses of valsalva and through the wall of the aorta to the anterior interventricular groove (intramural). Coronaries were implanted using the medially based trapdoor approach. Due to a kink in the reimplanted LMCA, an anterior gusset in the pulmonary artery (PA) was placed, allowing the left PA to roll backwards and superiorly away from the coronary. However the kink persisted and a new anastomosis had to be carried, after which the patient was successfully weaned from bypass with normal ventricular function on intraoperative transesophageal echocardiogram. He had a long postoperative course with persistent chylous effusions and was discharged at 5 weeks of age. Two weeks after discharge (7 weeks postoperative), the patient had an episode of crying, followed by coughing, gasping, and struggling for breath. He was apneic for approximately 20 s, became unresponsive. and blood was noted in his mouth when his mother initiated cardiopulmonary resuscitation (CPR). Upon intubation, blood-tinged frothy secretions were noted in the endotracheal tube (ETT) and he required high-pressure ventilator support to maintain adequate oxygen saturation. Upon arrival at the cardiac intensive unit, his initial pH was 6.9. Chest X-ray showed bilateral pulmonary edema. Over the next 48 h he had numerous episodes of hypertension with systolic blood pressures in the 160s, agitation, desaturation, and bradycardia. He frequently appeared mottled and pale and blood was occasionally suctioned from the ETT. The episodes resolved with sedation, paralysis, and increased positive end expiratory pressure (PEEP). During these episodes, his electrocardiogram (EKG) and echocardiogram were normal with a troponin leak up to 1.17 ng/ml. At 10 weeks of age and 3.6kg, the patient was taken to the

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catheterization laboratory and noted to have severe stenosis of the LMCA [Figure 1 and Videos 1-3]. The left ventricular end diastolic pressure (LVEDP) was 14 mmHg at baseline. However, when the depth of the anesthesia was reduced, his systolic blood pressure and LVEDP increased to 120 and 24 mmHg, respectively, suggesting severe left ventricular diastolic dysfunction which was attributed to the compromised LMCA. After consultation with the patient's surgeon and informed consent from the patient's parent, we proceeded to stent the LMCA as a rescue procedure. A three-dimensional (3D) angiogram was performed through a 4F pigtail catheter positioned in the ascending aorta to define the length of the stenosis of the LMCA and its relationship to the aorta [Figure 2]. Thirty milliliter (60% contrast and 40% saline; 5 cc/kg of contrast) was given at 5cc/s times 6 s. Transesophageal pacing at a rate of 240/min was performed to optimize the images. The 3D rotational angiography was used to obtain the best working angle. Measurements of the LMCA were performed on 2D angiogram in that angle [Figure 3]. Clopidogrel (2 mg/kg) and heparin were administered. A 4F angled glide (Terumo, Japan) was placed retrograde in the ascending aorta and exchanged over a 0.035wire (Cook Inc, Bloomington, IN) for a4F long sheath (Cook Inc, Bloomington, IN) was used and its tip was positioned near the origin of the LMCA. A 4F JR1 catheter was passed through the long sheath and a 0.014 Balance Middle Weight (BMW) wire (Abbott, IL) was placed distally into the left anterior descending artery [Figure 1b]. After injecting 10 mcg of nitroglycerin (3 mcg/kg) into the LMCA, a 2 × 8 mm Multilink Mini-Vision Coronary Stent (Abbott, IL) was introduced over the wire into position. Multiple angiograms were performed to position the stent so as not to cross the bifurcation, and at the same time not protrude too much in the aorta [Figure 1c]. The stent was deployed with inflation to 16 atm [Video 4]. An angiogram was performed that showed a patent stent in good position [Figure 1d]. A week later following a decrease in clopidogrel dose to 0.5 mg/kg/day and having general anesthesia for minor eye surgery; the patient decompensated with acute left ventricular dilation and severe dysfunction. He was taken urgently to the catheterization laboratory and selective left coronary angiography showed partial obstruction of the proximal portion of the LMCA stent (40%), probably due to platelet aggregation [Figure 4]. Balloon angioplasty was performed using a 2 mm × 6 mm Sprinter balloon (Medtronic, MN) followed by intracoronary infusion of nitroglycerin and integrilin. There was marked improvement of ventricular function over a 24 h period and he was discharged home. The patient underwent elective cardiac catheterization 7 months later. The stent appeared patent with mild narrowing at the origin of left anterior descending distal to the stent. This may have been caused by intimal build up or a kink from vessel growth [Figure 5]. A dobutamine stress echo was performed (1 year after stent placement)

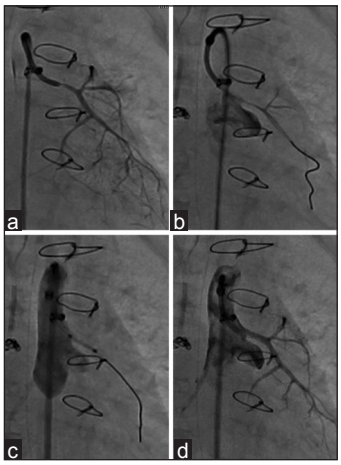

Figure 1: (a) Selective coronary angiograph of the left main coronary artery with evidence of severe stenosis at the origin. (b) 0.014 BMW wire placed distally into the left anterior descending artery. (c) Stent positioning so that it does not cross the bifurcation, and at the same time not to protrude too much into the aorta. (d) Stent deployed in the proximal left main coronary artery. Stent appears patent and in good position. BMW = Balance Middle Weight

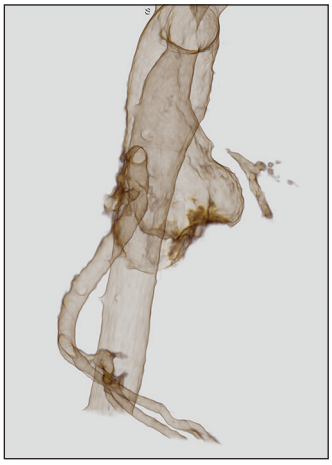

Figure 2: Three-dimensional (3D) rotational angiogram with evidence of severe narrowing of the proximal portion of the left main coronary artery

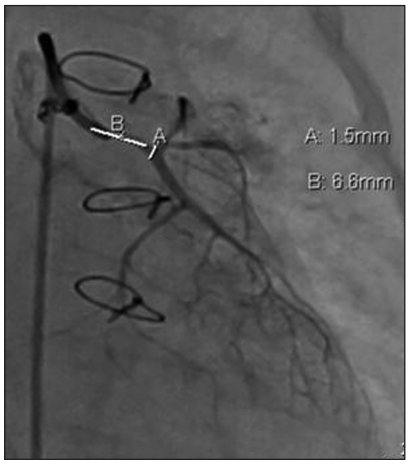

Figure 3: Measurements of selective left main coronary artery angiogram. The vessel diameter distal to the stenosis measured 1.5 mm and the length of the stenosis measured around 6.6 mm

Table 1: Demographic data, diagnosis, and affected vessel of the only two children found in the literature with coronary stenting that are less than 3 months in age

Case	Age	Weight (kg)	Diagnosis	Stent	Balloon size	Follow-up
1	3 months	4.5	ALCAPA after surgical repair with aortic wall flap obstruction	2.25 × 8 Sonic Hepacoat (Cordis)	3 mm	Died at 43 days from sepsis/ MOS failure. Good LV fX prior
2	3 days	4.3	TGAs/pASO	3 × 9 Integrity (Medtronic, Minneapolis, Minnesota)	2.25 mm	Stent patent 7 years later

Case 1 refers to reference[2] andcase 2 refers to reference,[3] ALCAPA: Anomalous origin of the left coronary artery from the pulmonary artery, TGAs: Transposition of the great arteries, pASO: Post arterial switch operation, MOS: Medical Outcomes Study, LV fX: Left ventricular functioning

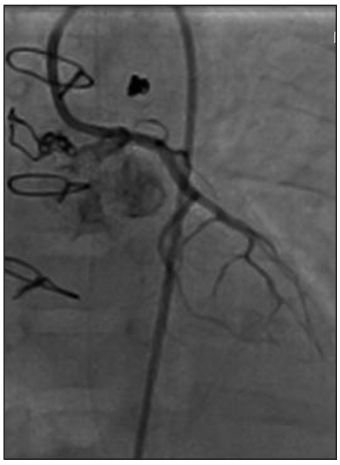

Figure 4: Selective left main coronary artery angiogram with evidence of partial obstruction of the proximal portion of the coronary stent (40%)

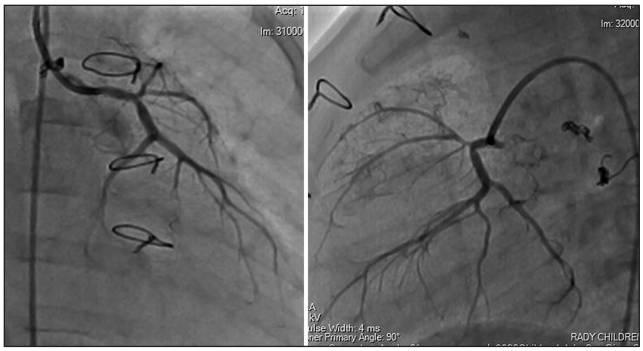

Figure 5: Follow-up repeat selective left coronary artery angiogram with evidence of stable mild narrowing at the proximal portion of the coronary stent. There is mild narrowing at the origin of the left anterior descending artery distal to the stent

and did not show regional wall abnormality. He remains asymptomatic, without shortness of breath or agitation and is growing and developing well.

DISCUSSION

Percutaneous coronary artery stent implantation has not been extensively employed in the pediatric population[1] because of lack of appropriate equipment for such procedures and future growth of the patient and coronary. There are only a few case reports in the literature in infants less than 3 month of age, summarized in Table 1.[2,3] This case represents an unusual presentation of coronary artery stenosis post ASO. Even though flash pulmonary edema is a well-known manifestation of coronary stenosis in the adult population, it has not been described in the pediatric population. The incidence of coronary stenosis post ASO is about 7%.[4] The lack of available stent sizes for this sized distal vessel and lack of equipment designed for this procedure made it more challenging. The distal vessel measured 1.5 mm and the length to the bifurcation was 6 mm, the smallest available stent is 2 mm × 8 mm. Precise placement so as not to cover the bifurcation and not to protrude too much into the aorta was achieved using 3Drotational angiogram to guide the exact camera angle needed to achieve this task. Seven months later, the stent appeared patent with mild narrowing at the origin of left anterior descending distal to the stent. This may have been caused by intimal build up or a kink from vessel growth. A drug-eluting stent could have been considered; however, the smallest was 2.25 mm and with some studies showing no clinically significant difference in the rates of the death or myocardial infarction between the two stents,[5] we elected to proceed with the smaller one. Also, the need for longer dual antiplatelet therapy in drug-eluting stents might be problematic. Medium term follow-up for this patient including a normal dobutamine stress echo is promising. Bench testing of the current stent suggests a future expandable diameter up to 6 mm.[6] The stent will continue to be dilated by angioplasty as the child and coronary artery grows. If angioplasty can no longer achieve goal diameter, surgical repair may be necessary.

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