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Unusual right ventricle outflow tract obstruction in Dacron valved conduits

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ABSTRACT

Various biological valved conduits have been used to treat the obstructions of the right ventricle. These conduits have different rates and etiologies for conduit stenosis during early- and long-term follow-up. We report on three cases of unusual intra-luminal peeling of a Dacron conduit leading to various types of conduit obstruction.

1. Introduction

Surgical implantation of a valved conduit to connect the right ventricle to the pulmonary artery is required in various Congenital Heart Diseases (CHD). The various prosthetic and biological valved conduits have been shown to demonstrate different early-and long-term results [1]. One of the most common etiologies of conduit failure is severe obstruction requiring either surgical replacement or percutaneous treatment [2,3]. Various causes of conduit stenosis have been described including valve degeneration, conduit-patient mis-match, proximal/distal anastomosis stenosis, conduit calcification, external compression, pulmonary artery stenosis, and endocarditis. Here we report on three rare cases of intraluminal peeling of a Dacron conduit leading to severe intra-procedural right ventricular outflow tract (RVOT) obstruction requiring intervention.

2. Case series

2.1. Case 1

A 34-year-old female with L-loop transposition of great arteries (L-TGA), ventricular septal defect (VSD) and pulmonary atresia status-post repair presented with exercise intolerance and systemic oxygen desaturations to 85%. Her surgical history consisted of a neonatal classic Blalock-Taussig shunt followed by a physiologic biventricular repair at 6 years of age with an atrial septal defect (ASD) closure, a VSD closure and the placement of an 18 mm homograft conduit from the left ventricle to the pulmonary artery (LV-PA). At 11 years of age, she developed conduit stenosis and underwent a surgical conduit replacement with a 22 mm Hancock porcine-valved Dacron conduit (Medtronic, Dublin, Ireland). Transthoracic echocardiogram demonstrated a small residual atrial septal defect and mild narrowing of the distal conduit. Cardiac computed tomography scan revealed a small peel within the proximal conduit (Fig. 1A). The patient was brought to the cardiac catheterization laboratory where a peak systolic gradient of 32 mmHg was measured across the LV-PA conduit. Intracardiac echocardiographic imaging and angiography revealed a mobile peel within the proximal end of the conduit that was not obstructive (Fig. 1B). The conduit valve was heavily calcified with immobile leaflets leading to severe conduit regurgitation. Given the sub-pulmonic left ventricle, the plan was made to restore conduit valve function while maintaining some degree of obstruction to optimize inter-ventricular interaction. After evaluating for coronary artery compression, a 3110 Palmaz XL stent (Cordis, Santa Clara, CA) was implanted within the mid conduit, distal to the conduit peel, on a 20 mm BiB balloon (NuMed, Cross Roads, TX) (Fig. 1C). This was followed by implantation of a Melody TPV on a 20 mm Ensemble delivery system (Medtronic, Dublin, Ireland). The procedure resulted in a residual conduit peak systolic gradient of 16 mmHg. Intracardiac echocardiography revealed no Melody valvar regurgitation with stable appearance of the mobile proximal conduit peel. In over 2 years since the procedure, transthoracic echocardiogram has shown stable gradients across the conduit.

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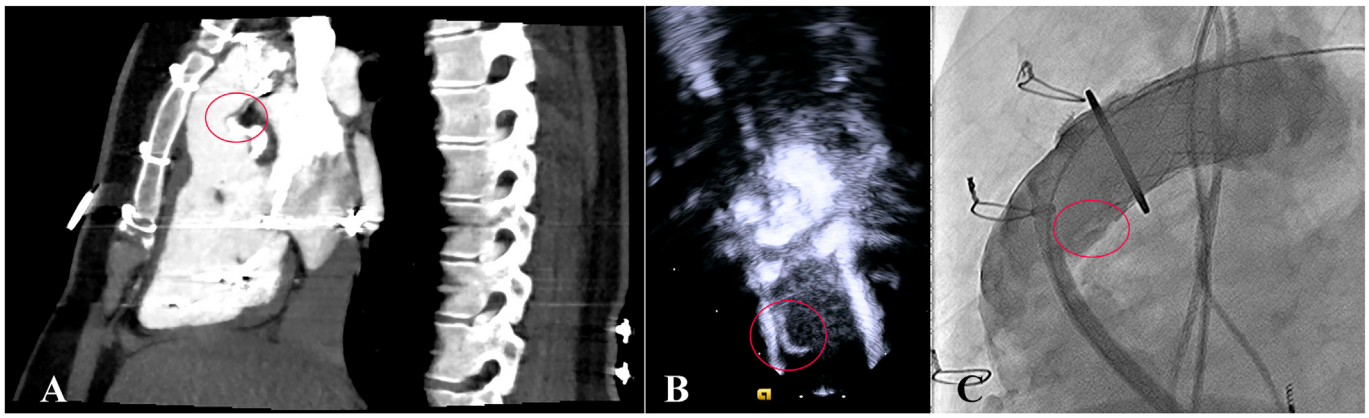

Fig. 1. Case 1: Small peel (red circles) within the inferior Dacron conduit seen on pre-procedural computed tomography (A), intra-procedural intracardiac echocardiography (B), and angiography following implantation of a 3110 Palmaz XL stent dilated to 20 mm (C).

2.2. Case 2

A 26-year-old female with L-TGA, VSD, and severe pulmonary stenosis status-post repair presented with progressive shortness of breath over the past 6 months. The surgical history included a neonatal systemic to pulmonary shunt followed by a Senning-Rastelli operation at 6 months of age. The patient then underwent replacement of the right ventricle to pulmonary arterial (RV-PA) conduit with a 20 mm Hancock porcine-valved Dacron conduit (Medtronic, Dublin, Ireland). Transthoracic echocardiography showed significant obstruction of the RV-PA conduit with a peak instantaneous pressure gradient of 70 mmHg and a high right ventricular (RV) systolic pressure (90 mmHg). Cardiac magnetic resonance imaging revealed severe sub-valvar conduit obstruction. The patient was referred to the catheterization laboratory for intervention on the RV-PA conduit. Hemodynamic evaluation revealed a peak systolic gradient of 50 mmHg across the conduit and the RV pressure was 100%

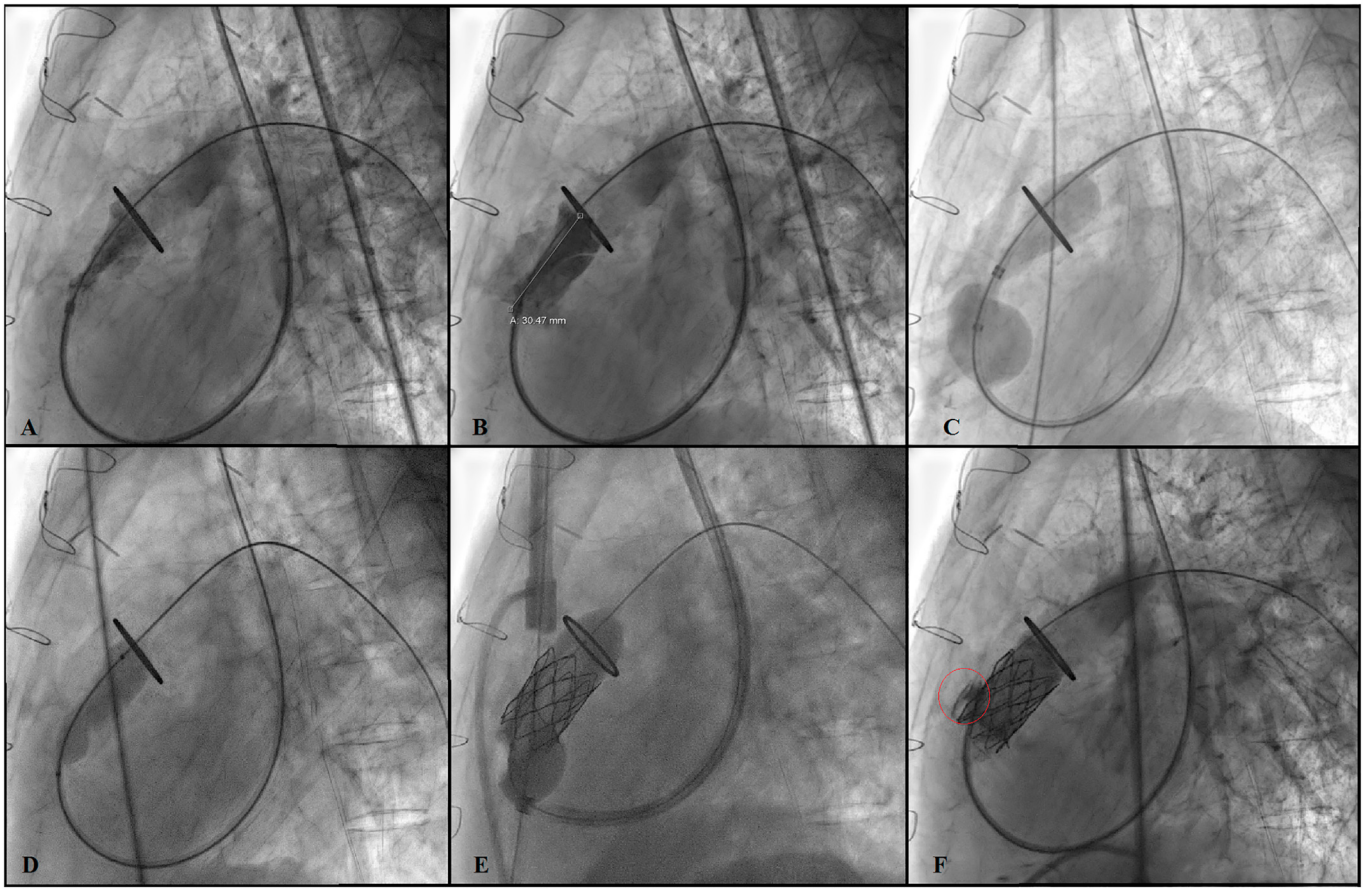

Fig. 2. Case 2: Interventional cardiac catheterization steps. Right ventricle angiography in lateral view showed a sub-valvular "funnel shape" obstruction (A) with the distance between the "tip of funnel" and the leaflets measured 30 mm (B). An AGA sizing balloon 34 mm unmasked the narrowest tract (C) and the inflation of a no-compliant BiB balloon 18 × 40 mm demonstrated the distension of lesion (D). A covered CP stent 28 mm crimped onto a BiB balloon 20 × 45 mm enlarged the stenosis (E.). The final angiography showed an adequate stent expansion without dissection signs and a characteristic enhancement (red circle) of the Dacron sub-valvular material smashed by the stent (F).

of systemic pressure. Angiography revealed severe sub-valvar stenosis of the RV-PA conduit secondary to significant intra-luminal peel. The lesion was unresponsive to balloon angioplasty using an 18 mm × 4 cm BiB balloon (NuMed, Cross Roads, TX), and was ultimately treated by implanting a 28 mm covered CP stent (NuMed, Cross Roads, TX) on a 20 mm × 4.5 cm BiB balloon (NuMed, Cross Roads, TX) proximal to the conduit leaflets. The intervention resulted in good expansion of the proximal conduit, though there was mild staining of the conduit peel which was compressed by the stent (Fig. 2). Hemodynamic data revealed a non-significant residual peak systolic gradient across the conduit (10 mmHg) with a RV pressure of 33% of systemic. The post-procedural transthoracic echocardiogram showed mild conduit stenosis (peak instantaneous pressure gradient 20 mmHg) and no conduit regurgitation. The patient's shortness of breath has resolved and transthoracic echocardiograms have remained stable over the past 3 years since the procedure.

2.3. Case 3

A 7-year-old female with DiGeorge syndrome and late diagnosis of pulmonary atresia, VSD, confluent pulmonary arteries supplied a ductus arteriosus status-post repair was referred to the catheterization laboratory for intervention on the stenotic RV-PA conduit. Her procedural history consisted of a complete surgical repair at 11 months of age consisting of VSD closure and placement of a 14 mm aortic Homograft RV-PA conduit. At 18 months of age, the patient developed stenosis of the distal conduit and proximal left pulmonary artery (LPA) which was treated by implantation of a single 26 mm Mega LD stent (Medtronic, Dublin, Ireland) on an 8 mm balloon. The patient developed conduit stenosis and underwent replacement with a 16 mm Hancock porcine-valved Dacron conduit (Medtronic, Dublin, Ireland) with resection of the conduit stent at 3 years of age. The stent extending to the LPA could not be completely resected. Transthoracic echocardiogram demonstrated a peak instantaneous pressure gradient of 61 mmHg with mild regurgitation. On baseline hemodynamic evaluation, there was a peak systolic gradient of 47 mmHg across the RV-PA conduit and RV pressure was 85% of systemic. Angiography revealed severe stenosis of the distal conduit and proximal LPA stent (Fig. 3A). After performing angioplasty of these lesions with a 12 mm balloon, there were two large intimal peels which had become disrupted from the conduit leading to worsening proximal and distal conduit stenosis (Fig. 3B). This resulted in a peak systolic gradient of 62 mmHg across the conduit and RV pressure of 130% systemic. The proximal conduit peel was treated with implantation of a 26 mm Mega LD stent on a 14 mm balloon, and the distal conduit was covered by implantation of a second 26 mm Mega LD stent on two 10 mm balloons using the Flower-Blossom technique [4]. A 16 mm Z-Med II balloon (B. Braun Medical Inc. Bethlehem, PA) was used to dilate the stents. The procedure resulted in a complete covering of the conduit peel (Fig. 3C) with a residual conduit peak systolic gradient of 16 mmHg and RV pressure was 35% of systemic. The patient has done well with no concerns in the 12 months since the catheterization.

3. Discussion

Xenograft implants are widely used to surgically treat RVOT obstructions [5,6]. The Hancock conduit is a xenograft valved conduit, characterized by a porcine aortic valve sutured into the center of a Dacron conduit reinforced by an external ring useful to avoid a loss of leaflets coaptation, and is one of the most commonly used conduits. Previous studies have demonstrated high rates of freedom from reoperation (81.9%) and a low-risk of infective endocarditis (1.9%) over 5 years of follow-up in Hancock conduits [2]. The most common etiology for Hancock conduit dysfunction is degeneration or calcification of the valve with consequent valvular stenosis [7,8]. Other mechanisms of conduit failure are conduit-patient mis-match and stenosis of proximal/distal anastomosis. Our case series describes the spectrum of conduit obstruction caused by the progressive formation of neointimal peel adherent to the inner Dacron wall of the Hancock conduit. In the first case, the neointimal proliferation was apparent on pre-procedural imaging and did not require intervention. The neointimal proliferation leading to intervention was present on the second case and was successfully treated via stent implantation. During the third case, there was diffuse disruption of the neointima during balloon angioplasty requiring treatment with stent implantation.

The surgical literature first reported this type of stenosis in a patient with transposition of the great vessels, VSD and pulmonary stenosis who had undergone a Rastelli operation with a Hancock conduit [9]. One year later, the patient developed a late sub-valvular obstruction due to a prominent pannus formation requiring surgical replacement of Hancock. Histological analysis described pannus in contact with the Dacron conduit consisted mostly of collagen bundles, while the luminal portion of the pannus consisted of organized thrombus. Edwards and colleagues discovered 46% of patients treated with a Hancock RV-PA conduit developed significant neoinimal proliferation requiring surgical replacement [10]. The mechanism of obstruction involved formation of a thick obstructive fibrous lining and separation of the neointima from the conduit.

Various studies demonstrated a high risk of developing a significant internal peeling in extracardiac Fontan pathways created from Dacron conduits in 68–75% of patients over a mean follow-up of 3.9–6.5 years [11,12]. The continuous and low-pressure blood-flow in the Fontan circulation, associated with an increased thrombo-embolic risk, might explain the increased rate of pannus formation in this physiology.

There are multiple lessons learned from this case series. First, the

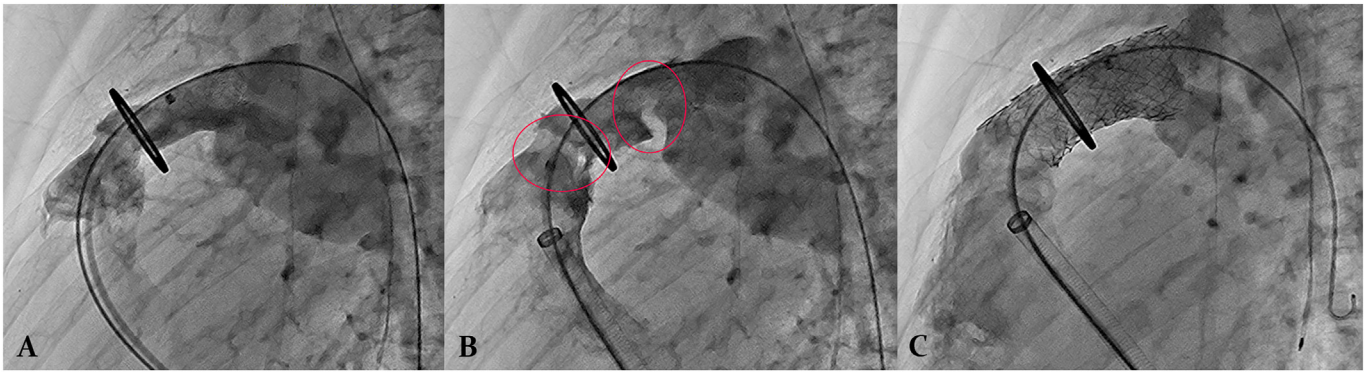

Fig. 3. Case 3: Lateral projection of the RV-PA conduit with severe stenosis (A). Following balloon angioplasty, two large intimal peels were seen in the proximal and distal conduit (red circles) (B). The lesions were successfully treated with implantation of two 26 mm Mega LD stents dilated to 16 mm along the RV-PA conduit (C). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

importance of pre-procedural imaging is demonstrated so that operators have the best understanding of the etiology of conduit stenosis. The neointimal peel was seen in the first two cases and allowed for pre-procedural planning. Second, proximal and distal Dacron conduit stenosis can be secondary to neointimal proliferation which can be disruptive during cardiac catheterization and requiring swift intervention. Lastly, care must be made to fully understand the lesion to appropriately follow and/or perform transcatheter intervention. In the first case, intracardiac echocardiography optimally showed the peel which may be mistaken for an infective vegetation in the setting of a febrile illness. Prior to elective intervention in the second case, a sizing balloon inflated across the proximal conduit to gain a better understanding of the substrate and obtain the inner shape of the obstruction. This was followed by inflation of a non-compliant balloon to test the compliance of the lesion. Based on the recoil of the neointimal peel, we elected to treat the lesion using a covered stent to reduce the risk of conduit injury and embolization of neointimal fragments. Given the acute severe obstruction in the last case, balloon testing of the lesion was not performed. Due to the distal location of the peel, and open-cell stent was implanted in a Flower-Blossom technique to prevent branch pulmonary arterial obstruction.

4. Conclusion

Neointimal intraluminal peel can form within Dacron conduits. This peeling may partially “dissect” and create significant conduit obstruction either during medical management or may be exacerbated during an interventional procedure. These lesions should be anticipated in these conduits and can be treated via transcatheter interventions.

Declaration of competing interest

Nothing to declare.

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