Transpulmonary Stent Implantation for Dysplastic Pulmonary Valve Stenosis with a Single Left Coronary Ostium and Anomalous Prepulmonary Right Coronary Artery in an English Bulldog

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INTRODUCTION

Pulmonary valve stenosis (PS) occurs in isolation in 8% to 10% of cases of congenital heart disease in humans and is one of the most common causes of congenital heart disease in dogs.[1,2] Concurrent congenital heart defects may be detected with PS, including coronary anomalies. The prepulmonary course of a coronary artery is a congenital coronary artery anomaly that has been reported in both animals and humans.[3,4] This coronary anomaly increases clinical concern when associated with PS in dogs, as this coronary anomaly has previously limited opportunities for successful transcatheter intervention.[3] Coronary anatomy is an important consideration in cases of PS before transcatheter or surgical intervention to prevent fatal consequences that could result from damage to an anomalous coronary artery.[5] Although conservative balloon pulmonary valvuloplasty (BPV) for treatment of PS in animals with anomalous coronary arteries has been reported,[6] transcatheter stent intervention for PS with concurrent coronary anomalies has yet to be described.

Novel transcatheter therapeutics for congenital heart disease have emerged, with significant advancement over the past few decades.[7-9] Although the cost of transcatheter pulmonary valve implantation is a major factor for its limited use in dogs,[10] rare cases of nonvalved stent implantation have been reported as palliative therapy for dogs with PS.[11-13] This report describes the process of transpulmonary stent implantation in a dog with severe PS and a coronary anomaly.

CASE PRESENTATION

A 3-year-old male intact English bulldog weighing 22.4 kg was presented for evaluation of a heart murmur before general anesthesia and neutering. He was adopted 3 months before presentation and was reported to have increased respiratory effort and exercise intolerance. Physical examination revealed a grade IV/VI left basilar systolic heart murmur, a normal heart rate of 80 beats/min, and a regular rhythm. His lung sounds were normal, and femoral pulses were strong and synchronous. Systolic blood pressure was considered normal at 140 mm Hg. Transthoracic two-dimensional and Doppler echocardiography (iE33, Philips Medical Systems, Andover, MA) was performed and revealed a severely dysplastic pulmonary valve in addition to a discrete ridge of tissue immediately subvalvar to the pulmonary annulus (Figure 1A, Video 1). Color flow Doppler investigation identified flow acceleration beginning at the subvalvar ridge, as well as moderate pulmonary valve insufficiency. The peak transpulmonary systolic pressure gradient was 113 mm Hg, consistent with severe pulmonary valve and subvalvar dysplasia (Figure 1B). Subjectively severe right ventricular concentric and moderate eccentric hypertrophy were present, with prominent septal flattening throughout the cardiac cycle. A right-to-left shunting patent foramen ovale was noted. Evaluation of the aortic root demonstrated a prominent left coronary ostium arising off of the left coronary cusp (Video 2). A right coronary ostium could not be definitively identified. Concurrent electrocardiography during echocardiography revealed sinus rhythm with occasional ventricular premature complexes, likely of right-sided origin. Atenolol was prescribed, titrating to a maintenance dose of 25 mg orally every 12 hours.

Right heart catheterization, cardiac angiography, and high-pressure balloon valvuloplasty were initially recommended, and informed consent was obtained from the owner. The following month, the dog was premedicated with methadone and alfaxalone intramuscularly and preoxygenated, and anesthesia was induced with midazolam and propofol intravenously. Anesthesia was maintained with isoflurane in 100% oxygen, with constant-rate infusions of fentanyl and lidocaine. Right heart catheterization was performed after placing a 6-Fr, 6-cm introducer (Introducer set, Terumo Medical, Somerset, NJ) and upsizing to an 11-F, 10-cm introducer (Introducer set) in the right femoral vein. A pressure pull-back (Berman angiographic catheter; Arrow International, Morrisville, NC) from the pulmonary trunk to the right ventricle revealed a pressure gradient of 53 mm Hg. Right ventriculography was performed (Berman angiographic catheter; Optiray [ioversol] 741 mg I/mL [Guerbet, Raleigh, NC]), demonstrating severe pulmonary valve and subvalvar dysplasia (Figure 2A, Video 3). Severe post-stenotic dilatation of the pulmonary trunk and branch pulmonary arteries, severe concentric and eccentric right ventricular hypertrophy, and subjective right ventricular systolic dysfunction were present. The levo-phase raised suspicion for a single left coronary

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VIDEO HIGHLIGHTS

Video 1: Transthoracic echocardiographic cine acquired from right parasternal short axis at the base of the heart demonstrating pulmonary valve and subvalvar dysplasia.

Video 2: Transthoracic echocardiographic cine acquired from right parasternal short axis at the base of the heart demonstrating a prominent left coronary ostium arising off of the left coronary cusp and traversing cranially across the level of the pulmonary valve. A right coronary ostium is not identified. A single ventricular premature complex is present.

Video 3: Right ventriculogram showing the catheter tip positioned at the level of the RVOT. There is evidence of severe pulmonary valve and subvalvar dysplasia. There is severe poststenotic dilatation of the pulmonary trunk and left and right branch pulmonary arteries. There is severe concentric and eccentric right ventricular hypertrophy and mild tricuspid regurgitation. The esophageal marker catheter is present for image calibration.

Video 4: Aortic root angiography in the ventrodorsal projection demonstrating a single left ostium arising off the aorta and no evidence of a right coronary ostium. The left coronary ostium gives rise to the left main coronary artery, which branches into the paraconal interventricular branch coronary artery and the circumflex branch coronary artery. The anomalous prepulmonary course of the RCA appears to arise off the paraconal interventricular branch. A forme fruste of patent ductus arteriosus is present, as well as trivial aortic insufficiency due to pigtail catheter positioning.

Video 5: Aortic root angiography in the lateral projection demonstrating a single left ostium arising off the aorta and no evidence of a right coronary ostium. The left coronary ostium gives rise to the left main coronary artery, which branches into the paraconal interventricular branch coronary artery and the circumflex branch coronary artery. The anomalous prepulmonary course of the RCA appears to arise off the paraconal interventricular branch. A forme fruste of patent ductus arteriosus is present, as well as trivial aortic insufficiency due to pigtail catheter positioning.

Video 6: Simultaneous aortic root angiogram and right ventriculogram confirm a single left coronary ostium with anomalous prepulmonary course of the RCA.

Video 7: Fluoroscopic imaging demonstrating simultaneous balloon dilatation at the level of the pulmonary valve and aortic root angiography during coronary compression testing. Highlighted during balloon inflation, the anomalous prepulmonary RCA is seen wrapping around the RVOT, with no evidence of coronary compression. There is mild aortic insufficiency due to location of the pigtail catheter as well as mild mitral regurgitation.

Video 8: Deployment of a Palmaz Genesis stent previously hand-crimped onto a BIB dilatation catheter. The video starts after the inner balloon of the BIB is inflated. During deployment, the stent is centered across the stenotic waist, with disappearance of the waist after complete inflation of the outer balloon.

Video 9: Transpulmonary stent deployment is complete, and hand angiography through a long sheath is shown. The stent is appropriately positioned across the level of the pulmonary valve spanning the stenosis. The stent is apposed to the pulmonary annulus, and the distal aspect of the stent hangs freely within the dilated pulmonary trunk.

Video 10: Transthoracic echocardiographic cine acquired from right parasternal short axis at the base of the heart 1 day after transpulmonary stent implantation. The stent is shown centered across the pulmonary valve, spanning the previous valvar and subvalvar obstructions.

Video 11: Transthoracic echocardiographic image acquired from right parasternal short axis at the base of the heart 1 day after stent implantation demonstrating free pulmonary insufficiency.

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ostium. Vascular access to the right femoral artery was performed using a 5-F, 10-cm introducer (Introducer set). An aortic root injection using a 5-F, 100-cm pigtail catheter (Merit Medical, South Jordan, UT) confirmed a single left coronary ostium with an anomalous right coronary artery (RCA) arising from the left paraconal branch (Figures 2B and 2C, Videos 4 and 5). The anomalous RCA displayed a prepulmonary course. A forme fruste of patent ductus arteriosus was also present incidentally. Simultaneous aortic root and right ventricular injections further confirmed the single left coronary ostium and anomalous prepulmonary course of the RCA (Video 6). BPV was not performed, because of risk for damage to the anomalous coronary artery. The animal recovered from anesthesia uneventfully. Pimobendan was started at 5 mg orally every 12 hours because of right ventricular systolic dysfunction, and atenolol was decreased to 12.5 mg orally every 12 hours.

The following month, there was a perceived mild increase in activity attributed to starting pimobendan or the onset of colder weather, but the dog's clinical signs were otherwise unchanged. A recheck echocardiographic examination revealed static structural disease. The ventricular ectopy was more frequent, and paroxysms of ventricular bigeminy were noted during echocardiography, which prompted an increase in atenolol dose. After additional discussion with the owners regarding the potential benefit of the procedure, transpulmonary stent implantation was advised.

Eight weeks following the initial catheterization, transpulmonary stent implantation was performed. With the exception of pimobendan and a lower dose of atenolol being administered the morning of the procedure, the general anesthetic protocol was similar to the first cardiac catheterization. Percutaneous access to the left femoral vein and artery was achieved under ultrasound guidance. A 9-F,

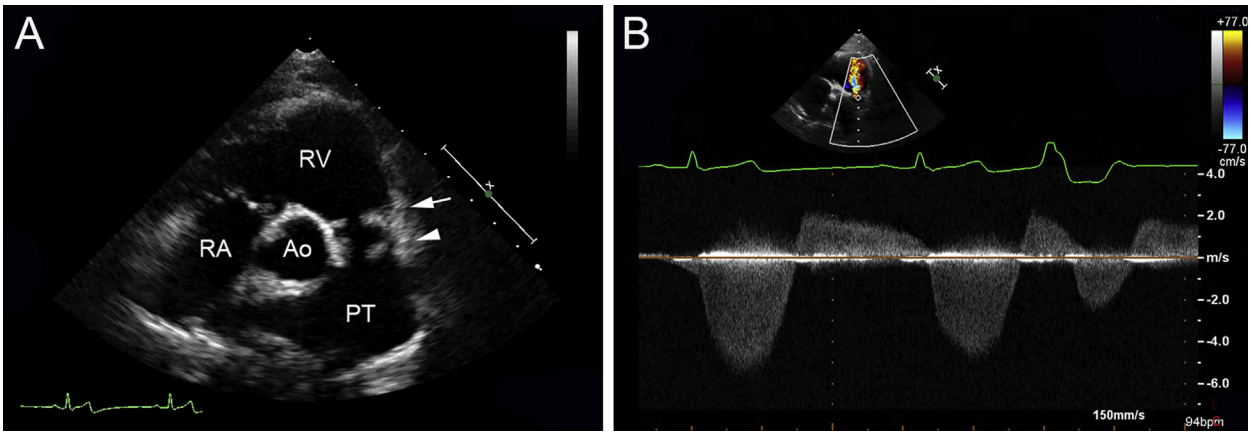

Figure 1 Transthoracic echocardiographic images acquired from right parasternal short axis at the base of the heart. **(A)** Pulmonary valve and subvalvar dysplasia as depicted by the *arrowhead* and *arrow*, respectively. **(B)** Severe transpulmonary Doppler pressure gradient is shown with a single ventricular premature complex. *Ao*, Aorta; *PT*, pulmonary trunk; *RA*, right atrium; *RV*, right ventricle.

6 cm introducer (Introducer set) was placed in the femoral vein, and a 4-F, 10-cm micropuncture introducer (Silhouette Transitionless Micropuncture introducer set; Cook Medical, Bloomington, IN) exchanged for a 5-F, 10-cm introducer (Introducer set) in the femoral artery.

A 6-F, 90-cm Berman angiographic catheter was advanced via the femoral vein into the right ventricular outflow tract (RVOT) and could not be passed across the subvalvar obstruction. Hemodynamics using this catheter revealed right ventricular systolic pressure of 106 mm Hg and mean right atrial pressure of 11 mm Hg. Right ventriculography was then performed (Berman angiographic catheter; Optiray) at the level of the RVOT, with findings similar to the first cardiac catheterization.

A 0.035-inch, 180-cm, angled hydrophilic guidewire (Merit Laureate; Merit Medical) was placed in a 6-F, 110-cm pressure wedge catheter (Balloon Wedge-Pressure Catheter; Arrow International), which allowed advancement of the catheter into the left branch pulmonary artery and a pulmonary artery pressure was obtained (26/9 [19] mm Hg). The right ventricular–pulmonary artery transpulmonary systolic pressure gradient was 80 mm Hg. In preparation for coronary artery compression testing before stent implantation, a 0.035-inch, 260-cm guidewire (Super Stiff Amplatzer Guidewire; AGA Medical, Plymouth, MN) was placed through the catheter (Balloon Wedge-Pressure Catheter) and into the left pulmonary artery, and the catheter was removed over the wire. Additionally, a 5-F, 100-cm pigtail angiographic catheter (Merit Medical) was advanced through a 5-F, 5.5-cm femoral artery introducer (Galt Micro-Access Elite HV; Galt Medical, Garland, TX), which had been placed via an exchange due to a kink in the initial femoral artery introducer, to the level of the aortic root.

On the basis of the catheter-derived pulmonary valve annulus of 15 mm, an 18 × 20 mm Atlas Gold catheter (C.R. Bard, Tempe, AZ) was selected and advanced into the femoral vein and centered across the pulmonary valve. The balloon was partially inflated (PRESTO Inflation Device; C.R. Bard), and aortic root angiography was performed simultaneously during the duration of the balloon inflation to document any risk for coronary compression (Figure 2D, Video 7). The inflated balloon mimicked the positioning of the transpulmonary stent, allowing evaluation of the anomalous course of the prepulmonary RCA in relation to the inflated balloon, assessing for changes in the coronary size, shape, and/or positioning (coronary geometry) and for any changes on electrocardiography. Angiography revealed normal coronary contrast flow, with no changes in coronary geometry, while continuous electrocardiographic monitoring revealed no evidence of ST-segment changes. The anomalous coronary artery was found to be free of compression and unaffected by balloon inflation and stent placement was deemed possible.

The balloon was removed, and the initial 9-F femoral vein introducer (Introducer set) was exchanged for a 10-F, 65-cm introducer sheath (Super Arrow-Flex Percutaneous Sheath; Arrow International). A 16mm × 3.5 cm outer balloon and an 8mm × 2.5 cm inner Balloon in Balloon (BIB) catheter (NuMed, Hopkinton, NY) was attached to two inflation devices (PRESTO Inflation Device). A 21mm × 10 mm diameter Palmaz Genesis stent (Palmaz Genesis Transhepatic Biliary Stent XD; Cordis, Santa Clara, CA) was hand-crimped onto the BIB, advanced through the sheath, centered across the valve, and deployed (Figure 2E, Video 8). Transpulmonary stent positioning was confirmed on fluoroscopy and angiography, showing the stent centered across the region of the stenosis (Figure 2F, Video 9).

Hemodynamics were repeated with a 5-F, 100-cm Judkins right catheter (Torcan NB Advantage Catheter, JR3; Cook Medical), revealing a right ventricular–to–pulmonary artery systolic pressure gradient of 34 mm Hg, representing a 58% reduction from the initial pressure gradient. A single Z-stitch with manual compression was used for vascular closure for both the femoral artery and vein.

Acepromazine was administered before extubation, and the dog recovered uneventfully from anesthesia. Overnight, the dog was sedated as needed with acepromazine and trazodone. Aspirin 81 mg orally every 24 hours was initiated to prevent device thrombus formation.

Echocardiography performed the day after the procedure confirmed appropriate placement of the transpulmonary stent (Figure 3A, Video 10). A 64% reduction in transpulmonary pressure gradient was observed (41 mm Hg, decreased from initial 113 mm Hg; Figure 3B). Free pulmonary insufficiency was noted, given the absence of effective valve function (Figure 3C, Video 11).

One month following the procedure, energy levels had dramatically increased according to the owner. Repeat echocardiography revealed evidence of reverse remodeling of the right ventricle on the basis of approximately 20% reduction in right ventricular wall thickness and a mild subjective decrease in right atrial size. The transpulmonary pressure gradient was 56 mm Hg, representing a 50% reduction

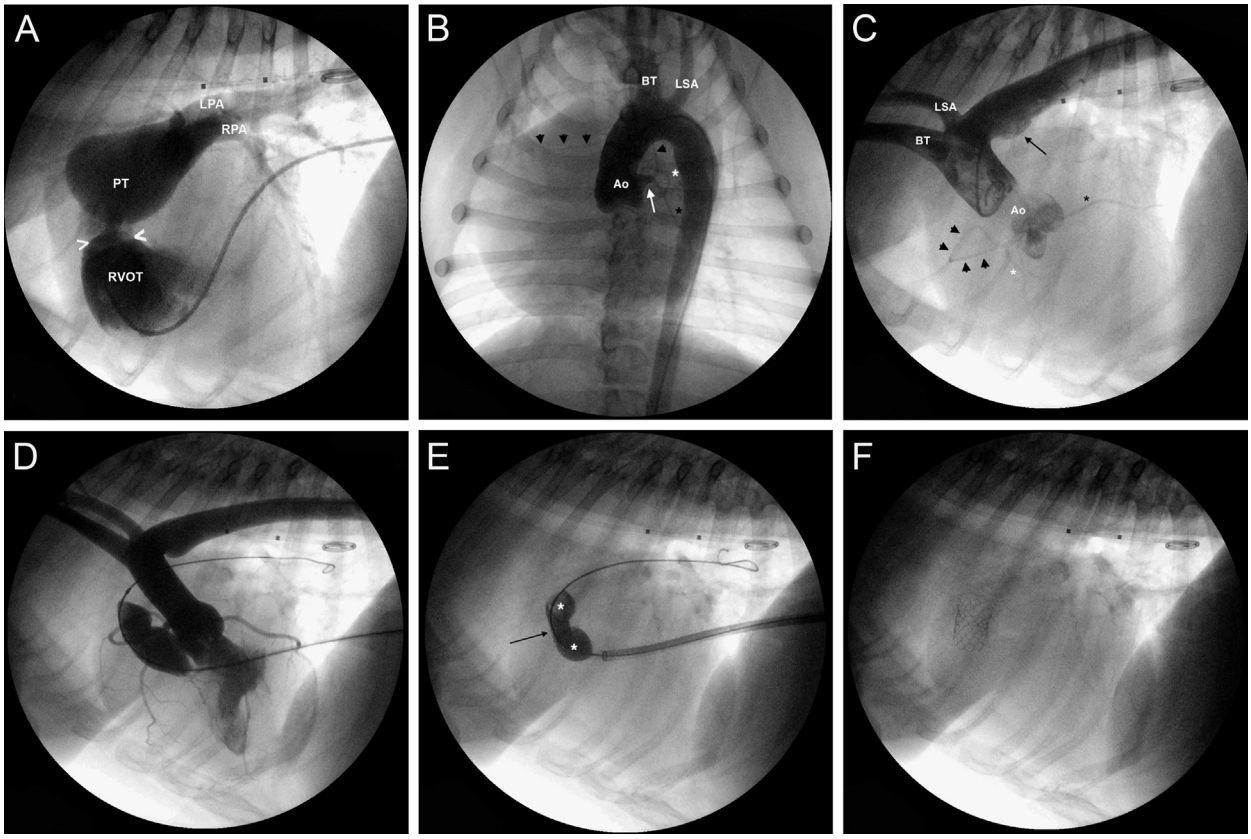

Figure 2 Fluoroscopic and angiographic imaging from initial cardiac catheterization **(A-C)** and during stent implantation **(D-F)**. **(A)** Right ventriculogram demonstrating a filling defect at the level of the pulmonary valve and subvalvar regions consistent with severe pulmonary valve and subvalvar dysplasia. There is severe post-stenotic dilation of the pulmonary trunk and left and right branch pulmonary arteries and secondary mixed right ventricular hypertrophy, which is partially shown on this right ventriculogram. The pulmonary valve annulus measures 15 mm (measured at points of the caret symbol), and the widest diameter of the post-stenotic pulmonary trunk measures 36 mm. **(B, C)** Aortic root angiography in the ventrodorsal **(B)** and lateral **(C)** projection demonstrating a single left ostium arising off the aorta and no evidence of a right coronary ostium. The left coronary ostium gives rise to the left main coronary artery (*white arrow*), which branches into the paraconal interventricular branch coronary artery (*white asterisk*) and the circumflex branch coronary artery (*black asterisk*). The anomalous prepulmonary course of the RCA (*black arrowheads*) appears to arise off the paraconal interventricular branch (*white asterisk*). A forme fruste patent ductus arteriosus is present (*black arrow*). **(D)** Simultaneous balloon dilatation at the level of the pulmonary valve and aortic root angiography for coronary compression testing. The image is taken midinflation, demonstrating a stenotic waist and the anomalous prepulmonary RCA wrapping around the RVOT, with no evidence of coronary compression. A diastolic frame is shown highlighting the coronary arteries, as well as aortic insufficiency due to location of the pigtail catheter. **(E)** Mid-deployment of a Palmaz Genesis stent previously hand-crimped onto a BIB dilatation catheter. The stent is centered across the stenotic waist (*black arrow*), with the inner balloon fully inflated. Midinflation of the outer balloon of the BIB (*white asterisks* depict the proximal and distal aspects of the outer balloon) is shown. **(F)** Stent position after deployment. An esophageal marker catheter is present for image calibration. *Ao*, Aorta; *BT*, bracheocephalic trunk; *LPA*, left branch pulmonary artery; *LSA*, left subclavian artery; *RCA*, right coronary artery; *RPA*, right branch pulmonary artery.

from the preprocedural gradient. There was no evidence of device thrombus formation. An occasional single ventricular premature complex, likely right-sided in origin, was noted during echocardiography. No changes in the dog's medication schedule were planned at this visit. On long-term follow-up 233 days since implantation, the dog continues to do well, with no clinical signs, and antithrombotic therapy was discontinued 9 months after implantation.

DISCUSSION

Procedural risk factors during BPV in dogs with PS and associated coronary anomalies could preclude operators from moving forward with the therapeutic intervention, but in this case we describe transpulmonary stent implantation as a novel intervention for a canine after taking precaution to assess for coronary artery compression.

The proximity of the prepulmonary coronary artery to the implant may place the coronary artery at risk for compression during implantation of a stent at the level of the pulmonary valve. Coronary artery compression from a stent in the RVOT, with or without a valve, was first recognized in 2006 in humans.[14] A more recent study assessed coronary artery compression as a factor in whether to proceed with intended transpulmonary Melody valve implantation in patients with obstructed RVOT conduits.[15] Coronary artery compression was diagnosed in 19 of 404 patients (5%) during test balloon angioplasty as

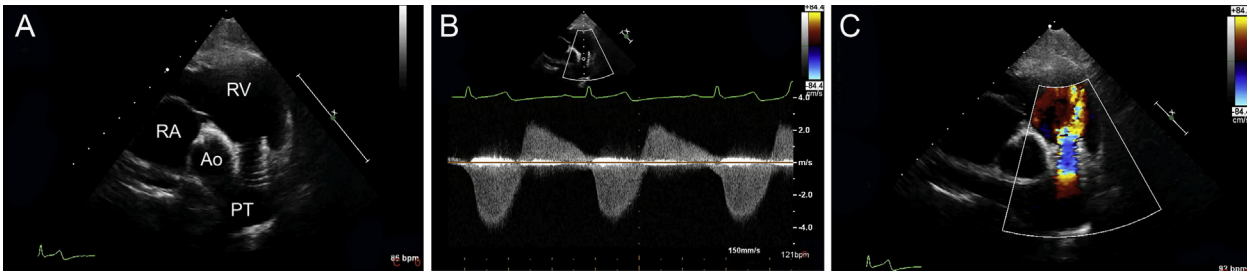

Figure 3 Transthoracic echocardiographic images acquired from right parasternal short axis at the base of the heart 1 day after transpulmonary stent implantation. **(A)** The stent is shown centered across the pulmonary valve, spanning the previous valvar and subvalvar obstructions. **(B)** A significant reduction of the transpulmonary Doppler pressure gradient. **(C)** Free pulmonary insufficiency is noted, with flow acceleration at the distal aspect of the stent. *Ao*, Aorta; *PT*, pulmonary trunk; *RA*, atrium; *RV*, right ventricle.

an indication not to proceed with the intended transpulmonary Melody valve implantation.[15] Using angiography alone or during simultaneous balloon dilation of the RVOT to characterize coronary artery anatomy is a crucial step during intervention to ensure that there is no evidence of coronary compression.[14] This potential fatal complication highlights the importance of evaluating the coronary arteries before transpulmonary stent implantation, especially in those with coronary anomalies as in this case. Those dogs with coronary compression are more likely to be excluded from the intended procedure because of elevated risk, as is the case in humans.[15]

Standard angiography may not depict the changes of the shape of the RVOT and the effects intervention could have on the coronary arteries, so simultaneous balloon dilation of the RVOT and coronary arteriography is often needed to accurately characterize the risk for compression.[15]

In this case, before stent implantation, we performed aortic root angiography to highlight the coronary arteries with simultaneous test balloon dilation across the PS. This method was used to simulate changes that could occur during the transpulmonary stent implantation. An Atlas Gold balloon was used during coronary artery compression testing. A balloon/annulus ratio of 1.2 was chosen to mimic the effects of a transpulmonary stent implant, and not for BPV, for which the ratio used is typically 1.3 to 1.5.[8] The balloon diameter selected for compression testing should have a diameter of approximately the same size to slightly larger than the pulmonary annulus. Given that the Atlas Gold selected was 1.2 times the annulus, we did not inflate the balloon fully to rated burst pressure (Figure 2D). Simultaneous aortic root angiography was performed during the duration of the balloon inflation. Continuous electrocardiographic monitoring revealed no evidence of ST-segment changes, and angiography revealed normal coronary contrast flow with no changes in coronary geometry, indicating no evidence of coronary compression, after which the transpulmonary stent was successfully implanted. Given the severe post-stenotic dilation, apposition of the stent to the pulmonary trunk in this case was not possible. Therefore the stent was mounted on a BIB catheter, which was selected with an outer diameter of 16 mm, as the pulmonary annulus was holding the stent in place and the distal aspect of the stent positioned free within the pulmonary trunk without apposition (Video 9). The stent (NuMed) was selected because it could be hand-crimped on the BIB catheter and balloon-expanded to oppose the pulmonary annulus, with an appropriate length to span both the pulmonary valve and subvalvar dysplasia.

ST-segment changes are well studied in human cardiology because of the higher incidence of coronary artery disease in humans compared with the canine species, and the appearance of the ST-segment can be used during cardiac catheterization to monitor for coronary occlusion. It has been previously suggested that coronary compression can result in coronary insufficiency and myocardial ischemia.[16] As coronary flow occurs primarily during diastole, systolic compression may result in ischemia if a sufficient severity of compression persists into diastole.[17] Coronary artery compression can result in elliptical narrowing of the coronary artery, significantly reducing blood flow, which may lead to myocardial ischemia.[17] Depending on whether the zone of ischemia is subepicardial or subendocardial, ST-segment elevation or depression, respectively, can be observed on a surface electrocardiogram. We advise close monitoring for ST-segment changes during coronary compression testing and transpulmonary stent implantation in dogs with PS and concurrent coronary anomalies.

Aspirin was initiated in this dog to prevent device thrombus formation. To date, there are no published guidelines regarding the use of monotherapy or combination anticoagulant and/or antiplatelet therapy after canine transpulmonary stent implantation and for what duration. Dual-antiplatelet therapy with aspirin and clopidogrel has been described in a case report of stent angioplasty in four dogs with PS, on the basis of a similar dual-antiplatelet approach taken for coronary stent implantation in humans, with no evidence of thrombus formation in any of the four dogs.[12] Aspirin continues to be a widely used antiplatelet agent in pediatric cardiac practice, including intracardiac device or stent implantation.[18] The use of aspirin monotherapy for 6 months following pulmonary artery stenting has been reported in humans[19,20] and was selected in our case. We advise waiting ≥6 months before discontinuation of antiplatelet therapy to ensure endothelialization of the stent. Future randomized controlled studies are warranted to determine the optimal antithrombotic therapy and duration for dogs after transpulmonary stent implantation. In addition, pimobendan, which is typically contraindicated in the presence of an RVOT or left ventricular outflow tract obstructive lesion,[21] was started after the first cardiac catheterization because of concern for right ventricular systolic dysfunction in this dog. Need for ongoing medication will be reassessed according to the dog's progress and future diagnostic monitoring results.

CONCLUSION

Transpulmonary stent implantation may be considered as a therapeutic intervention in patients with dysplastic PS and a single left coronary ostium with a prepulmonary RCA, provided precaution is taken ahead of implantation. The interventionalist should evaluate for coronary compression ahead of deployment of a transpulmonary stent in patients with these types of coronary anomalies. Future studies with long-term follow-up are necessary to evaluate the efficacy of this technique in a larger cohort.

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SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2020.08.009.

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