Received: 7 March 2019 | Accepted: 10 November 2019

DOI: 10.1111/jvim.15666

CASE REPORT

Journal of Veterinary Internal Medicine Open Access

ACVIM American College of Veterinary Internal Medicine

Graded balloon atrial septostomy for palliation of congenital pulmonary hypertension in a dog: A case report

Justin Allen | Nathan Peterson | Kirstie Barrett | Anthony Llamas

VCA West Los Angeles, Los Angeles, California

Correspondence
Justin Allen, VCA West Los Angeles, 1900 S Sepulveda Blvd, Los Angeles, CA 90025.
Email: justin.allen@vca.com

Abstract

Case Description: A 6-month-old intact female Maltese dog was presented for acute onset of syncope.

Clinical Findings: The dog was presented for collapse upon excitement and exercise. It collapsed at discharge and suffered cardiopulmonary arrest. Echocardiography after resuscitation indicated severe pulmonary hypertension without evidence of intracardiac or extracardiac shunting. A presumptive diagnosis of congenital pulmonary hypertension was made.

Treatment and Outcome: Initial treatment with sildenafil was effective at relieving syncope, but the extent of pulmonary hypertension as determined by serial echocardiography was unchanged. Graded balloon atrial septostomy was performed as a palliative procedure. Follow-up echocardiography identified a patent interatrial communication with bidirectional shunting. The dog remained asymptomatic 18 months after treatment.

Clinical Relevance: To the best of our knowledge, this study is the first report in the veterinary literature of graded balloon atrial septostomy performed for therapeutic purposes. Further studies are required to determine if this palliative procedure is a beneficial treatment option for dogs with congenital or severe refractory pulmonary hypertension.

KEYWORDS

cardiology, hemodynamics, interventional cardiology, right ventricular, syncope

1 | CASE

A 6-month-old, 1.68 kg intact female Maltese dog was evaluated by the emergency service at VCA West Los Angeles animal hospital for collapse episodes witnessed by the owners. The first 2 episodes occurred while the dog was running upstairs and the 3rd occurred during excitement at a pet store. Physical examination was performed at the time of presentation and was normal apart from a II-III/VI right apical systolic murmur and mildly increased respiratory rate that was attributed to excitement. The dog was hospitalized for observation overnight without recurrent episodes; the owner declined all diagnostic testing at that time. During discharge, the dog became excited and collapsed. A brief period of cardiopulmonary arrest followed and cardiopulmonary resuscitation was initiated. The dog was intubated and manually ventilated, and sinus bradycardia resumed. Spontaneous ventilation returned within 3 minutes, and the dog was extubated and provided supplemental oxygen. Echocardiography was performed upon recovery and disclosed moderate right ventricular (RV) and right atrial (RA) enlargement; moderate concentric RV hypertrophy and late-diastolic septal flattening were observed (Supplementary Content S1).

Abbreviations: AS, atrial septostomy; iASD, iatrogenic atrial septal defect; LA, left atrium; LVEDP, left ventricular end-diastolic pressure; PA, pulmonary artery; PDA, patent ductus arteriosus; PH, pulmonary hypertension; PTE, pulmonary thromboembolism; RA, right atrium; RV, right ventricle; TEE, transesophageal echocardiography; TEG, thromboelastography; TR, tricuspid regurgitation.

Right ventricular outflow tract velocity was normal (0.8 m/s; reference range, 0.64-1.51). Mild tricuspid regurgitation (TR) was observed with severely increased velocity (5.49 m/s; reference range, <2.8). Findings were consistent with severe pulmonary hypertension (PH) and cor pulmonale. An agitated saline contrast study (Supplementary Content S2) indicated no evidence of right to left shunting. Further diagnostic and treatment options were provided to the owners, who declined and considered humane euthanasia. After consideration, the owners decided to relinquish the dog to a foster home. The dog was started on sildenafil at a dosage of 3 mg/kg PO q12h. Heartworm antigen testing was negative and packed cell volume was 44% (reference range, 35-57). The pulmonary parenchyma was normal on thoracic radiographs. Thromboelastography (TEG) was performed and considered normal. A presumptive diagnosis of congenital pulmonary arteriopathy was made based on clinical signs, signalment, physical examination findings, and diagnostic test results.

Clinical signs improved with administration of sildenafil, with nearly complete resolution of the syncopal episodes. Echocardiographic RA and RV dimensions also improved (Supplementary Content and Table 1), but TR velocity was not measurable because of a decrease in TR. Long-term prognosis, however, was thought to be poor based on experience with young dogs with PH of unknown etiology. Therefore, graded balloon atrial septostomy (AS) was recommended.

The dog was sedated with methadone (0.4 mg/kg IV). General anesthesia was induced with alfaxalone (2.5 mg/kg IV) and intubation and ventilation were performed. Anesthesia was maintained with alfaxalone (1-6 mg/kg/h) and fentanyl continuous rate infusion (5-30 μg/kg/h). Electrocardiography, indirect blood pressure, pulse oximetry, end-tidal CO_2, and temperature were monitored throughout the procedure. Transesophageal echocardiography (TEE; GE Vivid *i*, GE Healthcare, Milwaukee, Wisconsin) was performed during the procedure. The dog was placed in left lateral recumbency and the right external jugular vein and carotid artery were isolated using a cutdown procedure. A 6 F introducer (Infiniti vascular sheath, Infiniti medical, Redwood City, California) was placed in the right jugular vein and a 5 F introducer (Check-Flo, Cook Medical, Bloomington Indiana) was placed in the right carotid artery. A 4 F Berman angiographic catheter (Teleflex, Morrisville, North Carolina) was advanced into the aortic root and 2 mL of a 1:1 solution of iohexol (Omni-paque 300, GE Healthcare) and saline was administered to define the aortic root. The catheter was left in place to monitor aortic pressure and obtain samples for oximetry. A 5 F Berman catheter was placed into the RA via the right jugular vein and a bolus of 2 mL iohexol was administered; dextro- and levo- angiographic phases were obtained. Still frames were utilized to identify the borders of the left and right atria, as well as the region of the fossa ovalis. The Berman catheter was advanced into the right pulmonary artery (PA); pressure tracings and serial blood oximetry were performed by pullback (Supplementary Content and Table 2). Pulmonary arterial pressure was increased (65/40 mm Hg; mean, 48 mm Hg; reference range, 30/19 mm Hg; mean, 14 mm Hg) without evidence of intracardiac or extracardiac shunting; postvalvular PA stenosis was ruled out. Right atrial pressure was 9 mm Hg, which was increased (normal, <5 mm Hg). This pressure was under the predetermined limit of 20 mm Hg based on recommendations when this procedure is performed in people,[1] and the procedure was continued. The Berman catheter was removed, and a 180-cm 0.035″ J-tip guidewire (Safe-T J, Cook Medical) was placed into the caudal vena cava. The 6 F introducer was exchanged for a 45-cm 6 F vascular sheath (Flexor Ansel 3, Cook Medical). The sheath was advanced with the dilator in place into the RA and the guidewire was removed. A 21 g, 56 cm pediatric Brockenbrough transseptal needle (Medtronic, Minneapolis, Minnesota) was advanced into the sheath to the tip of the dilator, taking care to not extrude the needle. The dilator was advanced to tent the septum. The needle was advanced across the septum; a small bolus (0.2 mL) of agitated saline was administered to confirm placement in the left atrium (LA) by TEE. A 0.014″ guidewire (Roadrunner extra-support wire guide, Cook Medical) was placed through the needle and advanced into the left caudal pulmonary vein; the needle was held in place while the dilator was advanced into the LA over the guidewire. The needle was withdrawn and the sheath was advanced into the LA body; the dilator then was removed. Heparin (150 U/kg) was administered. Left atrial pressure was obtained before the initial balloon inflation, and after each subsequent balloon inflation. Goals of the procedure were to create a right-to-left atrial shunt that decreased SaO_2 by 5%-10% and did not

TABLE 2 Hemodynamic data

	Pre-iASD	Post-iASD	6 months post-iASD
RA pressure (mm Hg)	9	10	5
RV pressure (mm Hg)	63/0	35/0	31/0
PA pressure (mm Hg)	65/40 (48)	36/12 (20)	30/12 (18)
PCWP (mm Hg)	14/7 (9)	13/8 (10)	15/7 (10)
AO pressure (mm Hg)	130/72 (91)	115/70 (85)	115/68 (84)
LV pressure (mm Hg)	122/7	NP	NP

Abbreviations: AO, aortic; iASD, iatrogenic atrial septal defect; LV, left ventricle; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; RA, right atrium; RV, right ventricle.

TABLE 1 Echocardiographic data

	15 Jun 2017	20 Jun 2017	27 Jun 2017	11 Oct 2017
RAA index (cm^2/m^2)	NP	13.4	NP	6.25
RVAWd (mm)	5	4	4	2.5
RVIDd (mm)	13.4	12.2	11	7.1

Abbreviations: RAA, right atrial appendage; RVAWd, right ventricle anterior wall thickness in diastole; RVIDd, right ventricular internal dimension in diastole.

result in an increase in LA pressure >18 mm Hg, based on guidelines used in humans to optimize outcome and safety.[1-4] A 4-mm cutting balloon (small peripheral cutting balloon, Boston Scientific, Marlborough, Massachusetts) was advanced over the guidewire and centered across the atrial septum. The balloon was inflated 3 times, with an inflation time of 30 seconds each. The sheath was readvanced into the LA, and the balloon and guidewire were removed. Aortic blood samples were obtained for oximetry. Oximetric data, SpO_2, and TEE did not show clinically relevant right-to-left shunting, thus a 0.025″ guidewire (Safe-T J, Cook Medical) was advanced into the pulmonary vein and a 6 mm balloon (Tyshak, B. Braun, Bethlehem, Pennsylvania) was passed across the atrial septum. Three inflations of 30 seconds duration were performed and the balloon was removed (Supplementary Content S3). Oximetric and hemodynamic data did not show a clinically relevant right-to-left shunting, and TEE indicated bidirectional shunting across the iatrogenic atrial septal defect (iASD). Central venous pressure remained unchanged (10 mm Hg). An 8 mm balloon (Tyshak, B. Braun) then was advanced over the guidewire across the atrial septum; an inflation was performed and held for 30 seconds. Then, the balloon was removed. Oximetric and hemodynamic data were obtained and indicated no clinically relevant differences from baseline, except for a decrease in PA pressure. No further balloon inflations were performed, because 8 mm was the prespecified limit based on dog size. The prespecified limit was based on the intent to create a restrictive ASD, which has been described as an ASD resulting in a pulmonary-to-systemic blood flow ratio (Qp:Qs) of 1.4 or less.[5,6] This is thought to be true of atrial septal defects that are >25% of the total atrial septal length, regardless of the shunt direction.[6-9] Estimates of total septal length in this dog ranged from 19.8 to 27.2 mm based on angiography and TEE measurements, corresponding to a maximal balloon diameter of 6.8 mm. The final balloon was upsized to 8 mm to account for expected recoil and decrease in ASD size postprocedure; the higher balloon size also was selected to minimize the likelihood of spontaneous closure.[10-13] The sheath was retracted into the RA while leaving the guidewire in place within the LA, and the dog was monitored for hemodynamic decompensation. The dog remained stable and the guidewire was removed. The introducers were removed and the carotid and external jugular vein ligated. Closure was routine and recovery was uneventful. The dog was started on enoxaparin (1 mg/kg SC q12h), cefazolin (22 mg/kg IV q8h), sildenafil (3 mg/kg PO q12h), and buprenorphine (0.02 mg/kg IV q8h).

Echocardiography was performed the next day and disclosed a patent iASD with bidirectional shunting. Agitated saline contrast was administered and contrast was observed within the LA. Doppler echocardiography was performed at a 2-week reevaluation and identified left-to-right shunting across the iASD; no right-to-left shunting was evident. The RA area was similar to previous measurements. Antibiotics were discontinued, and treatment with clopidogrel (11 mg/kg PO q24h) and sildenafil (3 mg/kg PO q12h) were continued. At 2-month reevaluation, a marked decrease in RA volume was identified (RAA index from 13.4 to 7.14 cm^2/m^2); the RV wall thickness was also decreased (RV anterior wall thickness in diastole [RVAWd] from 4 to 2.5 mm). Hematocrit was 47% (reference range, 35-57). At 6 months, the dog underwent hysterectomy and PA catheterization. Pulmonary arterial pressures were in the high-normal range, decreased from initial results (30/12 mm Hg; mean, 18 mm Hg; Supplementary Content and Table 2). The iASD was patent on TEE and angiography, with no right-to-left shunt present. The dog was asymptomatic 18 months postoperatively and remained on sildenafil and clopidogrel.

2 | DISCUSSION

Pediatric PH is poorly characterized in small animal medicine. In practice, the condition is a diagnosis of exclusion in dogs presenting for syncope or respiratory signs at a young age; the prognosis largely depends on the severity of hypertension and the underlying etiology.[14-17] Pulmonary hypertension in dogs has been classified into groups, modeled after the World Health Organization classification scheme in adult humans.[14] Group I is comprised of diseases that cause pulmonary arterial pathology; including congenital pulmonary arteriopathies, heartworm disease, idiopathic pulmonary arterial hypertension, and congenital heart diseases resulting in pulmonary overcirculation (Eisenmenger's physiology), such as large ventricular or atrial septal defects, or patent ductus arteriosus (PDA). Most young dogs presenting for signs caused by PH will fall into this group. Group II represents PH caused by increased left atrial pressures, which is 1 of the most common causes of PH in small animal dogs overall,[14-16] and can occur in young dogs with left heart disease (such as mitral dysplasia or cor triatriatum sinister). Group III is because of chronic intermittent hypoxia (such as chronic lung disease or brachycephalic airway syndrome); young dogs would not be expected to fall into this group, because it requires chronicity. Group IV dogs have PH associated with pulmonary thromboembolism (PTE), which can be present in young dogs, but is thought to be uncommon. Finally, Group V includes miscellaneous causes such as postvalvular pulmonary arterial obstruction; this also is considered uncommon in young dogs and in small animal medicine in general.[15] In humans, pediatric PH most often is caused by congenital heart disease and idiopathic causes.[17,18]

In our case, PH was thought to be caused by congenital pulmonary arteriopathy. Heartworm disease was not likely because of the young age of the dog at initial presentation, negative heartworm antigen test results, and low geographical prevalence. Congenital cardiovascular diseases were ruled out by echocardiography and catheterization studies. No evidence of lung disease was present on physical examination and thoracic radiography in this dog before or after the procedure. Thromboembolic disease was not ruled out in this dog. It is considered unlikely, however, because of age, lack of an identifiable predisposing cause of thrombogenicity, normal TEG and D-dimers, and no evidence of filling defects on pulmonary angiography.

Pulmonary arteriopathy is poorly characterized in small animal medicine due because of relative rarity, need for lung histopathology to obtain a definitive diagnosis in some cases, and rapid progression in dogs with clinical signs. The causes, treatment, and prognosis are unclear because of the lack of available data. Pulmonary veno-occlusive disease, idiopathic, familial, and infectious etiologies have been reported, but the incidence and clinical courses remain poorly

understood.[19-23] Medical treatments for other causes of PH (such as sildenafil, pimobendan, antiplatelet and anticoagulant treatment, and oxygen) often are utilized. Clinical experience, however, suggests a poor overall prognosis regardless of initial response to treatment, with a high risk of mortality within a year for dogs <1 year old presenting for syncope or dyspnea associated with PH without an identifiable cardiovascular cause (such as a right-to-left shunting PDA).

Prognosis for dogs with congenital PH without a right-to-left shunt appears poor when compared to dogs with congenital PH caused by a right-to-left shunt (Eisenmenger's physiology) or adult-onset PH. This difference in prognosis was the impetus for recommending balloon AS in our dog.[24-26] The goal was to delay progression and extend therapeutic efficacy by mimicry of Eisenmenger's physiology; improvements in PA pressure and RV function over time were not anticipated. Decrease in PA pressures is not a consistent outcome of the procedure in humans, and AS is largely performed as a bridge to transplantation or as a palliative option to improve quality of life after failure of medical treatment.[1,4,27-29]

In our dog, clinical signs (syncope and collapse) were resolved by sildenafil treatment alone; right heart function and PA pressure, however, remained suboptimal. Pulmonary arterial pressures and RV function normalized after the procedure, but we are unable to determine how much, if any, of the improvement was associated with the creation of the iASD. Based on prior experience, however, we suspect that stability and improvement were at least in part a consequence of the iASD, as alternative explanations are considered unlikely. It is possible that the severe PH was thromboembolic in nature and that long-term clopidogrel administration resulted in resolution of PTE over time. Antiplatelet treatment alone, however, usually is insufficient to result in resolution of thromboembolic PH, and laboratory and angiographic findings did not support thromboembolic PH. Sildenafil may be primarily responsible for improvement or resolution of PH over time,[30] but this is considered less likely because it is uncommon for sildenafil to reverse pulmonary arterial changes, although it may delay progression of disease and minimize clinical signs.[31-33] The sildenafil dosage was atypical because of the size of the dog, but within the range that typically is utilized in dogs with severe PH.[14,33] Regression of RV pathology secondary to suspected pulmonary arteriopathy and hypertension has been reported in a dog that received sildenafil,[24] and similar effects may have occurred in our dog. The effects of the iASD in this case, therefore, remain speculative. At minimum, the procedure appeared to be well-tolerated and did not result in overt harm to the dog.

Atrial septostomy was first described in children as a palliative procedure to improve right heart blood flow in cases of transposition of the great vessels as a bridge to definitive correction.[34] Prognosis in human patients with PH because of Eisenmenger's physiology appears to be better than in patients with congenital or idiopathic PH.[25] This observation and experience in the pediatric population led to the application of AS to patients with severe PH and few alternative treatment options. The procedure was first reported as a treatment for refractory PH in 1983[35]; it has remained an infrequently utilized treatment since that time, and generally reserved for severe or refractory PH in humans or as a bridge to transplantation.[2-4,27,28,36-45] Some evidence, however, suggests that patients may benefit from the procedure at an earlier time.[3,28] Prognosis in patients with PH appears to be related largely to RV function and RA pressure.[24,25] Atrial septostomy can benefit each by providing a diastolic pop-off valve for the overloaded right heart, and in humans has been shown to decrease RA pressure and improve RV stroke index.[1] Left ventricular filling is improved by right-to-left shunting at the atrial level, which results in improved cardiac output and net tissue oxygen delivery, albeit at a decreased oxygen saturation.[4,27,41,43] These changes translate to benefits in quality of life, function, and prognosis in many patients. Perioperative mortality appears to be low with appropriate patient selection and increased operator experience.[1] Graded AS refers to progressive balloon upsizing after initial atrial septal puncture, which allows monitoring for hemodynamic complications and for more precise sizing of the defect.[4,37] Reported targets for AS in humans include a decrease of arterial oxygen saturation (SaO2) <10% and left ventricular end-diastolic pressure (LVEDP) <18 mm Hg.[44] Graded AS, as well as more strict adherence to reported contraindications (RA pressure >20 mm Hg, LVEDP >18 mm Hg, resting SpO_2 <90%)[1-4] are thought to optimize outcomes, and were employed in our case. Complications of the procedure in humans, when they occur, frequently are fatal. These include complications inherent to the procedure (cardiac perforation, arrhythmias, thrombosis, and vascular complications) and severe hypoxemia because of excessive right-to-left shunting. Spontaneous closure of the defect is reported in up to 25% of human patients undergoing graded AS, and this also is a concern in dogs given the small size of the iASD. These complications were not evident in this case, though monitoring for iASD closure is ongoing.

Therapeutic options for PH in small animal medicine are even fewer than those in humans, and largely confined to phosphodiesterase inhibition alone or in combination with treatment for congestive heart failure. Alternatives are limited by cost, availability, or both. The dearth of effective long-term options, along with the observation that dogs with PH associated with Eisenmenger's physiology tend to live longer,[46] led to intervention in our case. The appropriate timing of AS is unknown in humans. The argument has been made that earlier intervention may result in more long-term benefit, and this may be true for dogs as well.

In dogs, AS is appealing because of its relatively low cost compared to alternative treatments for severe or refractory PH, feasibility, and potential for therapeutic benefit for dogs with few alternative options. Further study and more experience with the technique will be necessary to determine which (if any) dogs will benefit from the procedure. Based on the outcome in this single case, AS is feasible and may represent a treatment option for refractory, severe, or congenital PH.

CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

ORCID

Justin Allen https://orcid.org/0000-0003-2881-0542

REFERENCES

1. Law MA, Grifka RG, Mullins CE, Nihill MR. Atrial septostomy improves survival in select patients with pulmonary hypertension. *Am Heart J*. 2007;153:779-784.
2. Rich S, Dodin E, McLaughlin VV. Usefulness of atrial septostomy as a treatment for primary pulmonary hypertension and guidelines for its application. *Am J Cardiol*. 1997;80:369-371.
3. Bhamra-Ariza P, Keogh AM, Muller DWM. Percutaneous interventional therapies for the treatment of patients with severe pulmonary hypertension. *J Am Coll Cardiol*. 2014;63:611-618.
4. Maluli HA, DeStephan CM, Alvarez RJ, Sandoval J. Atrial septostomy: a contemporary review. *Clin Cardiol*. 2015;38:395-400.
5. Kaye D, Shah SJ, Borlaug BA, et al. Effects of an interatrial shunt on rest and exercise hemodynamics: results of a computer simulation in heart failure. *J Card Fail*. 2014;20:212-221.
6. De Rosa R, Schranz D. Creation of a restrictive atrial left-to-right shunt: a novel treatment for heart failure. *Heart Fail Rev*. 2018;23:841-847.
7. Bauer A, Esmacili A, DeRosa R, et al. Restrictive atrial communication in right and left heart failure. *Transl Pediatr*. 2019;8:133-139.
8. Bauer A, Khalil M, Lüdemann M, et al. Creation of a restrictive atrial communication in heart failure with preserved and mid-range ejection fraction: effective palliation of left atrial hypertension and pulmonary congestion. *Clin Res Cardiol*. 2018;107:845-857.
9. Bauer A, Khalil M, Schmidt D, et al. Transcatheter left atrial decompression in patients with dilated cardiomyopathy: bridging to cardiac transplantation or recovery. *Cardiol Young*. 2019;29:355-362.
10. Mitchell SE, Anderson JH, Swindle MM, Strandberg JD, Kan J. Atrial septostomy: stationary angioplasty balloon technique—experimental work and preliminary clinical applications. *Pediatr Cardiol*. 1994;15:1-7.
11. Weldon CS. Hemodynamics in acute atrial septal defect. *Arch Surg*. 1966;93:724-729.
12. Hanslik A, Pospisil U, Salzer-Muhar U, Greber-Platzer S, Male C. Predictors of spontaneous closure of isolated secundum atrial septal defect in children: a longitudinal study. *Pediatrics*. 2006;118:1560-1565.
13. Radzik D, Davignon A, Van Doesburg N, et al. Predictive factors for spontaneous closure of atrial septal defects diagnosed in the first 3 months of life. *J Am Coll Cardiol*. 1993;22:851-853.
14. Williams JG. Pulmonary hypertension and pulmonary thromboembolism. In: Ettinger SJ, Feldman EC, Côté E, eds. *Textbook of Veterinary Internal Medicine*. 8th ed. St. Louis, MO: Elsevier; 2017:1131-1136.
15. Kellihan HB, Stepien RL. Pulmonary hypertension in dogs: diagnosis and therapy. *Vet Clin North Am Small Anim Pract*. 2010;40:623-641.
16. Ware W. Pulmonary hypertension. *Cardiovascular Disease in Small Animal Medicine*. London, UK: Manson Publishing Ltd; 2013:340-348.
17. Abman SH, Hansmann G, Archer SL, et al. Pediatric pulmonary hypertension: guidelines from the American Heart Association and American Thoracic Society. *Circulation*. 2015;132:2037-2099.
18. Hansmann G. Pulmonary hypertension in infants. *J Am Coll Cardiol*. 2017;69:2551-2569.
19. Glaus TM, Soldati G, Ehrensperger F, Maurer R. Clinical and pathological characterisation of primary pulmonary hypertension in a dog. *Vet Rec*. 2004;154:786-789.
20. Zabka TS, Campbell FE, Wilson DW. Pulmonary arteriopathy and idiopathic pulmonary hypertension in six dogs. *Vet Pathol*. 2006;43:510-522.
21. Williams K, Andrie K, Cartoceti A, et al. Pulmonary veno-occlusive disease: a newly recognized cause of severe pulmonary hypertension in dogs. *Vet Pathol*. 2016;53:813-822.
22. Borgeat K, Sudunagunta S, Kaye B, Stern J, Luis Fuentes V, Connolly DJ. Retrospective evaluation of moderate-to-severe pulmonary hypertension in dogs naturally infected with *Angiostrongylus vasorum*. *J Small Anim Pract*. 2015;56:196-202.
23. Calvert CA, Rawlings CA. Pulmonary manifestations of heartworm disease. *Vet Clin North Am Small Anim Pract*. 1985;15:991-1009.
24. D'Alonso GE, Barst RJ, Ayres SM, et al. Survival in patients with primary pulmonary hypertension: results from a national prospective registry. *Ann Intern Med*. 1991;115:343-349.
25. Hopkins WE, Ochoa LL, Richardson GW, Trulock EP. Comparison of the hemodynamics and survival of adults with severe primary pulmonary hypertension or Eisenmenger syndrome. *J Heart Lung Transplant*. 1996;15:100-105.
26. Hopkins WE. The remarkable right ventricle of patients with Eisenmenger syndrome. *Coron Artery Dis*. 2005;16:19-25.
27. Reichenberger F, Pepke-Zaba J, McNeil K, Parameshwar J, Shapiro LM. Atrial septostomy in the treatment of severe pulmonary arterial hypertension. *Thorax*. 2003;58:797-800.
28. Sandoval J, Gaspar J, Pena H, et al. Effect of atrial septostomy on the survival of patients with severe pulmonary arterial hypertension. *Eur Respir J*. 2011;38:1343-1348.
29. Sandoval J, Gomez-Arroyo J, Gaspar J, Pulido-Zamudio T. Interventional and surgical therapeutic strategies for pulmonary arterial hypertension: beyond palliative treatments. *J Cardiol*. 2015;66:304-314.
30. McMahon P, Saelinger C. Reversal of echocardiographic right-sided heart pathology in a dog with severe pulmonary hypertension: a case report. *Vet Med*. 2015;6:211-218.
31. Brown AJ, Davison E, Sleeper MM. Clinical efficacy of sildenafil in treatment of pulmonary arterial hypertension in dogs. *J Vet Intern Med*. 2010;24:850-854.
32. Kellum HB, Stepien RL. Sildenafil citrate therapy in 22 dogs with pulmonary hypertension. *J Vet Intern Med*. 2007;21:1258-1264.
33. Bach JF, Rozanski EA, MacGregor J, Betkowski JM, Rush JE. Retrospective evaluation of sildenafil citrate as a therapy for pulmonary hypertension in dogs. *J Vet Intern Med*. 2006;20:1132-1135.
34. Rashkind WJ, Miller WW. Creation of an atrial septal defect without thoracotomy: a palliative approach to complete transposition of the great arteries. *JAMA*. 1966;196:991-992.
35. Rich S, Lam W. Atrial septostomy as palliative therapy for refractory primary pulmonary hypertension. *Am J Cardiol*. 1983;51:1560-1561.
36. Klepetko W, Mayer E, Sandoval J, et al. Interventional and surgical modalities of treatment for pulmonary arterial hypertension. *J Am Coll Cardiol*. 2004;43:S73-S80.
37. Sandoval J, Gaspar J, Pulido T, et al. Graded balloon dilation atrial septostomy in severe primary pulmonary hypertension: a therapeutic alternative for patients nonresponsive to vasodilator treatment. *J Am Coll Cardiol*. 1998;32:297-304.

38. Allcock RJ, O'Sullivan JJ, Corris PA. Atrial septostomy for pulmonary arterial hypertension. *Heart*. 2003;89:1344-1347.
39. Kurzyna M, Dabrowski M, Bielecki D, et al. Atrial septostomy in treatment of end-stage right heart failure in patients with pulmonary hypertension. *Chest*. 2007;131:977-983.
40. Nihill MR, O'Laughlin MP, Mullins CE. Effects of atrial septostomy in patients with terminal cor pulmonale due to pulmonary vascular disease. *Cathet Cardiovasc Diagn*. 1991;24:166-172.
41. Kerstein D, Levy PS, Hsu DT, Hordof AJ, Gersony WM, Barst RJ. Blade balloon atrial septostomy in patients with severe primary pulmonary hypertension. *Circulation*. 1995;91:2028-2035.
42. Micheletti A, Hislop AA, Lammers A, et al. Role of atrial septostomy in the treatment of children with pulmonary arterial hypertension. *Heart*. 2006;92:969-972.
43. Sandoval J, Rothman A, Pulido T. Atrial septostomy for pulmonary hypertension. *Clin Chest Med*. 2001;22:547-560.
44. Keogh AM, Mayer E, Benza RL, et al. Interventional and surgical modalities of treatment in pulmonary hypertension. *J Am Coll Cardiol*. 2009;54(Suppl 1):S67-S77.
45. Espinola-Zavaleta N, Vargas-Barron J, Tazar JI, et al. Echocardiographic evaluation of patients with primary pulmonary hypertension before and after atrial septostomy. *Echocardiography*. 1999;16:625-634.
46. Cote E, Ettinger SJ. Long-term clinical management of right-to-left ("reversed") patent ductus arteriosus in 3 dogs. *J Vet Intern Med*. 2001;15:39-42.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

How to cite this article: Allen J, Peterson N, Barrett K, Llamas A. Graded balloon atrial septostomy for palliation of congenital pulmonary hypertension in a dog: A case report. *J Vet Intern Med*. 2020;34:283–288. https://doi.org/10.1111/jvim.15666