Selah Mae's Ductal Dilemma

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Introduction

Congenital heart disease is uncommon, but not rare in canine species. Congenital heart disease comprises numerous structural or functional cardiac abnormalities that are present at birth. Animals may be affected by one defect or less commonly multiple defects at once. The estimated prevalence of congenital heart disease in dogs presenting to veterinary hospitals is between 0.46% and 0.85%[1]. The most common congenital heart defects reported in the canine species are pulmonic stenosis (PS), patent ductus arteriosus (PDA), and subaortic stenosis (SAS)[1,2]. A PDA results when the ductus arteriosus does not close appropriately after birth. The ductus arteriosus is a normal fetal vascular structure that extends from the bifurcation of the pulmonary artery to the ventral portion of the descending aorta after the left subclavian branches off. The ductus arteriosus allows deoxygenated blood to bypass the lungs. The deoxygenated blood is instead diverted from the main pulmonary artery to the descending aorta and eventually to the placenta where it becomes oxygenated[1]. PDA has historically been the most commonly diagnosed congenital heart defect in dogs although more recent studies have suggested that pulmonic stenosis is more common[2]. In a study compiling 4,694 cases of canine congenital heart disease from ten studies found in veterinary literature, PDA was the most common malformation with a prevalence of 25.7%[3]. Although more common in dogs, PDA has also been described in cats, horses, cattle, humans, and experimentally in mice[2].

A hereditary basis for the development of PDA is well-documented. Toy breeds including Yorkshire terriers, toy or miniature poodles, Maltese, and Pomeranians as well as other breeds such as German shepherds, Shetland sheepdogs, and English springer spaniels are predisposed to developing a PDA[4]. Sex appears to play a role in the heritability of PDA with

females being almost twice as likely to be affected as males[2]. The exact mode of inheritance remains unclear, but a polygenic mode of inheritance is suspected[4].

The most common clinical finding with a PDA is a continuous murmur auscultated at the left heart base. The peripheral arterial pulses are often bounding. A precordial thrill can often be palpated in the same region as the murmur[1]. Diagnosis is often made based on history and murmur characteristics in the absence of clinical signs. When clinical signs are present, they are generally related to the development of left-sided congestive heart failure. Clinical signs found with pulmonary edema include coughing, dyspnea, and tachypnea. Imaging modalities such radiology and echocardiography can be used to confirm the diagnosis of a PDA. Radiographic changes present include left-sided enlargement of the heart, protrusion of the descending aorta and main pulmonary artery, and enlarged pulmonary lobar vessels. Visualization of continuous turbulence in the main pulmonary artery with the use of color flow Doppler echocardiography further supports diagnosis of a PDA.[4]

History and Presentation

The patient that will be discussed in this case report is Selah Mae, a 6-month-old, intact female Toy Poodle who presented to MSU-CVM Small Animal Surgery department on June 18th, 2019. Selah Mae was referred from her primary veterinarian for surgical correction of a suspected PDA after repeated auscultations during routine examinations revealed a continuous heart murmur. In early April 2019, Selah Mae presented to her referring veterinarian for vaccination boosters. Upon physical exam, a grade IV/VI holosystolic murmur was auscultated. During this visit, an echocardiogram was performed revealing a mosaic of turbulent blood flow in the pulmonary trunk. Based on these results, a PDA was presumptively diagnosed. Selah Mae returned to her primary veterinarian for vaccination boosters again in late April and May 2019,

and a severe continuous murmur was auscultated. At this time, Selah Mae was referred to MSU-CVM Small Animal Surgery department for further evaluation of her heart murmur and surgical correction if indicated.

Upon presentation, Selah Mae was bright and alert. She weighed approximately 1.85 kilograms (4.1 pounds) with a body condition score of 4/9. Her vital parameters were within normal limits with temperature of 102.6 degrees Fahrenheit and a pulse of 124 beats per minute. Her respiratory rate was slightly elevated with panting at 48 breaths per minute. Her mucous membranes were pink and moist, and her capillary refill time (CRT) was less than two seconds. Upon auscultation of the lungs, bronchovesicular sounds were normal with no crackles or wheezes auscultated. Upon cardiopulmonary auscultation, a grade VI/VI continuous murmur was auscultated, and a palpable thrill was appreciated on the left wall of the chest. The murmur was loudest on the left side of the chest over the base of the heart. No pulse deficits were observed, and her toes were pink. The rest of the physical exam was unremarkable. Rectal and neurological exams were not performed.

Diagnostic Approach

Initial diagnostic tests included a small animal anesthesia profile (SAAP), thoracic radiographs, and an echocardiogram. The SAAP showed a mild hypoalbuminemia (5.4 g/dl) with all other values within normal limits. Thoracic radiographs revealed cardiomegaly characterized by dorsal deviation of the trachea, rounding of the left ventricular region, and the cardiac silhouette spanning > 50% of the width of the thorax on the ventrodorsal projection. A bulge was present in the region of the main pulmonary artery, the left auricle, and the proximal descending aorta. The pulmonary lobar vasculature and pulmonary parenchyma were within normal limits. The echocardiogram showed a jet of insufficiency at the pulmonic valve, and the main

pulmonary artery was dilated. Within the main pulmonary artery, there was a large jet of bidirectional turbulent flow extending into an aberrant vessel found adjacent to the left branch of the main pulmonary artery. Due to the physical exam findings, radiographic abnormalities, and echocardiogram results, a left-to-right shunting PDA was definitively diagnosed.

Pathophysiology

Persistent patency of the ductus arteriosus after the early neonatal period is diagnosed as a PDA. Distinct histological abnormalities in the ductal wall are responsible for the persistent patency of the ductus[1]. The primary abnormality seen with both sporadic and hereditary PDA development is ductal muscle hypoplasia at the aortic end of the ductus arteriosus. Elastic tissue resembling the aortic tissue replaces the muscle tissue. Inability of the elastic tissue to contract prevents closure in the affected areas adjacent to the aorta while the pulmonic end shows varying amounts of closure[2]. Abnormalities are classified into six grades based on ductal hypoplasia and degree of elastic tissue. Elastic tissue extends across less than half of the ductal wall in Grade 1 lesions. Grade 2 lesions have elastic tissue extending more than one half but not the entire diameter of the wall. Grades 1 and 2 allow for complete closure of the pulmonic end of the ductus. With grades 3-6, elastic tissue extends through increasing proportions of the ductal wall circumference. Grade 6 results in little to no ductal closure and a PDA that is congruent with the size of the aorta and pulmonary artery[8].

The shunting of blood with a PDA can be left-to-right or right-to-left. A left-to-right shunting PDA occurs when blood from systemic circulation is shunted to the pulmonary circulation. The higher pressure of the left heart relative to the right heart causes oxygenated blood to flow from the aorta to the pulmonary artery. Oxygenated blood is initially lost from systemic circulation and enters the pulmonary circulation. Due to the shunt, volume is increased

in the main pulmonary artery, the pulmonary vasculature, the left atrium and ventricle, and the aorta leading to volume overload and eccentric hypertrophy of the left ventricle.[4] A right-to-left shunting PDA, or a reverse PDA, involves admixture of venous blood into systemic circulation. Right-to-left shunting is possible because the pulmonary and aortic pressures become similar, and blood can flow in the opposite direction. Pulmonary hypertension or presence of a large left-to-right shunting PDA can lead to the development of a reverse PDA. A grade 6 PDA can lead to a right-to-left shunting PDA due to the lack of resistance at the pulmonic end of the ductus. The pulmonary artery is exposed to the full pressures of the aorta, and the pressures across the ductus eventually equilibrates[9]. Clinical signs in dogs with a right-to-left shunting PDA include a differential cyanosis, hindlimb weakness, and polycythemia. Closure of a right-to-left shunting PDA is contraindicated due to exacerbation of pulmonary hypertension[10].

Treatment and Management

The prognosis in dogs presenting with a left-to-right shunting PDA that undergo ductal closure is excellent compared to medical management. A study of 14 dogs diagnosed with PDA that did not undergo ductal closure found that 65% of the dogs died within a year of being diagnosed[5]. In contrast, canine patients with an uncomplicated PDA that was surgically repaired have been shown to live more than 11.5 years after their procedure[6]. Closure of the PDA is curative and leads to loss of the murmur and eventually reversal of the eccentric left ventricular hypertrophy in most cases[11].

Several PDA closure techniques have been described. The closure of an uncomplicated PDA can be accomplished via surgical ligation or via an interventional procedure. Surgical ligation is one of the most commonly performed closure methods. However, perioperative complications, such as ductal hemorrhage, and post-operative pain due to the thoracotomy are a

concern. Complications and mortality rates are significantly higher for surgical ligation when compared to interventional procedures, with mortality rates up to 7%[12]. The traditional surgical ligation involves circumferential ligation after dissection of the ductus cranially and caudally and blind dissection medially. Blind dissection must be done carefully to avoid ductal hemorrhage. An alternative surgical technique called the Jackson and Henderson technique avoids blind dissection of the ductus[7]. An intrapericardial technique has also been described to allow better visualization of the ductus and to decrease risk of ductal hemorrhage[13]. Long-term prognosis with surgical ligation is excellent with two-year survival rates as high as 87%[14]. Interventional procedures include trans-catheter procedures in which an occlusion device such as the Amplatz Canine Duct Occluder or a thrombogenic coil is placed within the PDA[1]. Benefits of interventional procedures include a lower complication rate and speedier recovery. With trans-catheter coil occlusion, the rate of major complications has been shown to be significantly decreased compared to surgical ligation[15]. Although the complication rate is lower compared to the traditional surgical ligation, not all animals are candidates for a minimally invasive closure. Patient size and morphology of the PDA can affect the feasibility of interventional procedures. The Amplatz Canine Duct Occluder cannot be used in dogs that weigh under 3 kilograms, and coil embolization should only be used in patients with a minimal ductal diameter less than 2 mm[7]. Success of interventional procedures is comparable to that of surgical procedures in most studies except for coil occlusion. One study found that surgery had a 94% success rate while coil occlusion had an 84% success rate[15].

Selah Mae underwent ductal closure by surgical ligation on June 19, 2019. She was given cefazolin pre-operatively and intra-operatively. Once under general anesthesia, Selah Mae was placed in right lateral recumbency. Her left thorax was clipped and surgically prepped with a 4%

chlorhexidine solution. A left lateral thoracotomy was performed between the 4th and 5th ribs (4th intercostal space). Using a #10 scalpel blade, a 10 cm skin incision was made through the skin from the transverse processes of the vertebrae and extended ventrally to 3 cm dorsal to the sternum. The subcutaneous tissues were bluntly and sharply dissected using Metzenbaum scissors down to the level of the latissimus dorsi, and the latissimus dorsi was retracted dorsally. Monopolar and bipolar electrocautery were used for hemostasis throughout the procedure. The serratus ventralis, and the external and internal intercostal muscles were transected to allow visualization of the parietal pleura. Metzenbaum scissors were used to penetrate the pleural space and to extend the length of the incision. The lung lobes were retracted with moistened 4 x 4 pieces of gauze followed by the use of Finochietto retractors to aid in visualization of the thoracic cavity. The vagus nerve was isolated near the ductus and was retracted ventrally using umbilical tape. A window into the mediastinum was made, and angled forceps were used to bluntly dissect the ductus arteriosus. Two ligatures were placed around the ductus arteriosus with 2-0 silk. The ligature closest to the aorta was tightened first followed by the ventral suture. A 14 FR MILA chest tube was placed caudal to the incision site and tunneled to the 7^{th} intercostal space where it traversed the thoracic wall. The chest tube was secured with a finger trap suture pattern using 3-0 Ethilon. Stay sutures of 4-0 PDS were placed around the 4th and 5th ribs adjacent to the incision. The stay sutures were used to oppose the ribs and close the musculature. A simple continuous pattern using 3-0 PDS followed by an intradermal pattern of 4-0 Monocryl was used to close the subcutaneous tissue. The chest was evacuated via the chest tube until negative pressure was achieved. Lastly, the chest was bandaged with Telfa pads and a Suresite followed by a stockinette. Selah Mae recovered from anesthesia uneventfully. Cardiac auscultation revealed no murmurs or arrhythmias. Post-operative management included a

fentanyl constant rate infusion followed by Tylenol #3 after discontinuation of the fentanyl. The chest tube was evacuated every 8 hours until it was removed on June 20, 2019.

Case Outcome

Selah Mae was discharged on June 21, 2019, with Tylenol 3 for pain control. She had a 10 cm incision on the lateral aspect of her left thorax that was slightly painful when palpated. Selah Mae's owners were instructed to restrict her activity level for the next two weeks to allow time for the incision to heal. They were also instructed to place cold packs on the incision three times a day for the next three days then to switch to warm packing the incision for 3-5 days. The owners were directed to have Selah Mae's primary veterinarian check the incision in approximately two weeks. On July 1, 2019, the owner reported that Selah Mae's incision was healing appropriately, and she was doing excellent at home. As of April 15, 2020, Selah Mae had made a full recovery and has had no setbacks since her surgery. Her owner reports that she is living her life to the fullest with her other canine companions.

References

[1] Beijerkink, Nick J. et al. Congenital Heart Disease. Ettinger SJ, Feldman EC, and Côté E, eds. Textbook of Veterinary Internal Medicine: Diseases of the Dog and the Cat. 8th ed. St. Louis: Elsevier, 2017. 1211-1218.

[2]Oliveira, P., Domenech, O., Silva, J., Vannini, S., Bussadori, R., & Bussadori, C. (2011). Retrospective Review of Congenital Heart Disease in 976 Dogs. Journal of Veterinary Internal Medicine, 25(3), 477–483.

[3] Scansen BA, Cober RE, Bonagura JD. Congenital Heart Disease. In: Bonagura JD, Twedt DC, eds. Kirk's Current Veterinary Therapy XV. 15th ed. St. Louis: Saunders Elsevier, 2014; 756-761.

[4]Kittleson, M. D. (2005). Chapter 12 PDA. Small Animal Cardiovascular Medicine Textbook. *VIN.Com*. Retrieved from https://www.vin.com/doc/?id=5496007

[5]Eyster, G. E., Eyster, J. T., & Cords, G. B. J. (1976). Patent ductus arteriosus in the dog: characteristics of occurrence and results of surgery in one hundred consecutive cases. *Journal of the American Veterinary Medical Association*, *168*(5), 435–438.

[6]Stauthammer CD, Tobias AH, Leeder DB, et al. Structural and functional cardiovascular changes and their consequences following interventional patent ductus arteriosus occlusion in dogs: 24 cases (2000–2006). *J Am Vet Med Assoc* 2013; 242:1722–1726.

[7]Tobias, A. H., & Stauthammer, C. D. (2010, July 1). Minimally invasive per-catheter occlusion and dilation procedures for congenital cardiovascular abnormalities in dogs. Veterinary Clinics of North America - Small Animal Practice, Vol. 40, pp. 581–603. https://doi.org/10.1016/j.cvsm.2010.03.009

[8]Buchanan, J. W., & Patterson, D. F. (2003). Etiology of Patent Ductus Arteriosus in Dogs.

Journal of Veterinary Internal Medicine, *17*(2), 167–171. https://doi.org/10.1111/j.1939-1676.2003.tb02429.x

[9]Buchanan, J. W. (2001). Patent Ductus Arteriousus Morphology, Pathogenesis, Types and Treatment. *Journal of Veterinary Cardiology*, *3*(1), 7–16. https://doi.org/10.1016/S1760-2734(06)70010-8

[10]Ferasin, L., Rizzo, F., & Darke, P. G. G. (2007). Original investigation of right-to-left shunting patent ductus arteriosus in an Irish setter puppy. *Veterinary Journal*, *173*(2), 443–448. https://doi.org/10.1016/j.tvjl.2005.11.015

[11]C.D. Stauthammer, T.P. Nguyenba, A.H. Tobias. Short and long term cardiac changes following complete occlusion of uncomplicated patent ductus arteriosus in dogs. J Vet Intern Med, 21 (2007), p. 609

[12]Birchard, S. J., Bonagura, J. D., & Fingland, R. B. (1990). Results of ligation of patent ductus arteriosus in dogs: 201 cases (1969-1988). *Journal of the American Veterinary Medical Association*, *196*(12), 2011–2013. http://www.ncbi.nlm.nih.gov/pubmed/2365630

[13]Selmic, L. E., Nelson, D. A., Saunders, A. B., Hobson, H. P., & Saunders, W. B. (2013). An intrapericardial technique for PDA ligation: Surgical description and clinical outcome in 35 dogs. *Journal of the American Animal Hospital Association*, *49*(1), 31–40. https://doi.org/10.5326/JAAHA-MS-5838

[14] Bureau, S., Monnet, E., & Orton, E. C. (2005, December 1). Evaluation of survival rate and prognostic indicators for surgical treatment of left-to-right patent ductus arteriosus in dogs: 52 Cases (1995-2003). Journal of the American Veterinary Medical Association, Vol. 227, pp. 1794–1799. https://doi.org/10.2460/javma.2005.227.1794

[15]Goodrich, K. R., Kyles, A. E., Kass, P. H., & Campbell, F. (2007). Retrospective Comparison

of Surgical Ligation and Transarterial Catheter Occlusion for Treatment of Patent Ductus Arteriosus in Two Hundred and Four Dogs (1993?2003). *Veterinary Surgery*, *36*(1), 43–49. https://doi.org/10.1111/j.1532-950X.2007.00233.x