Large Secundum Atrial Septal Defect Managed Conservatively in an Adult Dog

Carl Toborowsky, VMD, and Lauren E. Markovic, DVM, *Athens, Georgia*

INTRODUCTION

Atrial septal defect (ASD) is a common congenital heart defect (CHD) in people and represents the most common CHD diagnosed in adulthood.[1] The prevalence of ASD in dogs is much less common, generally reported to be around 2%.[2-4] We present findings of an ostium secundum ASD and subsequent right heart failure (RHF) in a 1.5-year-old Doberman pinscher, using echocardiography and computed tomography angiography (CTA) to assess the degree of cardiac remodeling and create three-dimensional (3D) reconstructed models to assess the feasibility of transcatheter intervention or surgical repair. We describe how conservative management of ASD and heart failure is possible in the dog when interventional and surgical options are not viable.

CASE PRESENTATION

An otherwise healthy 1.5-year-old male Doberman pinscher weighing 31.8 kg was presented to our teaching hospital for an acute history of nonproductive vomiting. On presentation, the patient was laterally recumbent and had signs of obstructive shock with tachycardia (heart rate of 160 beats per minute), weak femoral arterial pulses, and pain elicited from the cranial abdomen upon manual palpation. Oral mucous membranes were cyanotic with a prolonged capillary refill time. Cardiothoracic auscultation did not reveal any auscultable murmurs, gallop sounds, arrhythmias, or abnormal lung sounds. Point-of-care bloodwork revealed hyperlactatemia (3.3 mmol/L; normal, <2.5 mmol/L). Abdominal radiographs revealed gastric dilatation and volvulus (GDV), and the patient was taken to surgery for a gastric derotation and incisional gastropexy procedure. The patient recovered without immediate complication and was maintained on intravenous (IV) fluid therapy and pain control.

Two days postoperatively, the patient exhibited signs of increased respiratory rate and effort and developed a rectal temperature of 105.3 °F. Thoracic radiographs revealed a nonspecific bronchointerstitial caudal pulmonary pattern, with equivocal cardiomegaly, mild pulmonary arterial and venous distension, right heart enlargement, and a mild amount of pleural effusion. An obvious alveolar pulmonary pattern to suggest pneumonia was not apparent, but given the clinical signs, recent anesthesia event, and elevated rectal temperature, the patient was administered IV antibiotics and supplemental oxygen therapy and treated for suspected aspiration pneumonia. Point-of-care ultrasound confirmed mild pleural effusion, cytologic analysis of which revealed neutrophilic inflammation with a lymphoid component (fluid protein 3.4 g/dL; 53% nondegenerate neutrophils, 32% small lymphocytes).

Two-dimensional (2D) transthoracic echocardiography (TTE) including Doppler was performed. A timing lead electrocardiogram showed underlying sinus arrhythmia, with no ectopy or atrioventricular block. The TTE showed severe dilation of the right ventricle (RV) (RV area at end diastole from left parasternal apical image 3,354 mm^2 [reference interval, 440.4–1,327.0 mm^2], RV area at end systole from left parasternal apical image 2,564 mm^2 [reference interval, 230.2-870.5 mm^2])[5] and severe dilation of the right atrium (RA; RA area at end systole from left parasternal apical image 2,564 mm^2 [reference interval, 449.7-980.7 mm^2]),[5] diastolic septal flattening, and subjectively normal RV function. The left ventricular (LV) chamber was small (LV internal diameter in diastole normalized for body weight = 0.96; 95% reference interval, 1.14-1.61),[6] with normal systolic function (2D LV ejection fraction = 71%). Left atrial (LA) size was normal (right parasternal short-axis LA to aorta diameter ratio = 1.43). Color-flow Doppler revealed mild tricuspid regurgitation (TR), suspected to be secondary to annular stretch, but a degree of tricuspid valve dysplasia could not be ruled out.

The TTE at the level of the interatrial septum (IAS) was consistent with a large secundum ASD, and color-flow Doppler evaluation of the IAS revealed predominantly left-to-right flow across the ASD (Figure 1A, Video 1). The pulmonary valve was subjectively normal in appearance, with mild pulmonary regurgitation. Peak systolic transpulmonary velocity was increased (2.3 m/sec), thought to be secondary to increased flow from the left-to-right shunt. The mitral and aortic valves were normal in appearance with trivial regurgitation. The remainder of the study was unremarkable. A shunt ratio (Qp:Qs) was estimated to be 2.76 (normal reference interval, 0.71-1.29).[7]

An agitated saline study demonstrated an anechoic filling defect in the saline contrast within the RA at the level of the IAS, consistent with left-to-right shunting across the ASD (Figure 1B, Video 1). Mild pleural effusion was suggestive of early right-sided congestive heart failure caused by or exacerbated by a component of iatrogenic fluid overload. No cardiac medications were initiated at the time of initial TTE, but IV fluids were discontinued. Overall, the study provided a diagnosis of a left-to-right shunting secundum ASD and early decompensated RHF.

The patient was discharged with oral antibiotics (amoxicillin/clavulanic acid), gabapentin, trazodone, and an appetite stimulant (capromorelin). Repeat TTE 2 weeks later was largely static, with progressive mild to moderate pleural effusion. Given the persistence of cavitary effusion in the absence of IV fluid therapy, the patient

From the University of Georgia, College of Veterinary Medicine, Athens, Georgia.

Keywords: Canine, Congenital heart disease, ASD, Three-dimensional modeling, Structural cardiac disease

Correspondence: Lauren E. Markovic, DVM, Department of Small Animal Medicine and Surgery, University of Georgia, College of Veterinary Medicine, 2200 College Station Drive, Athens, GA 30602. (E-mail: *lauren.markovic@uga.edu*).

2468-6441
https://doi.org/10.1016/j.case.2023.12.022

VIDEO HIGHLIGHTS

Video 1: Two-dimensional TTE, right parasternal long-axis view with color-flow Doppler (*left*), demonstrates the left-to-right signal across the secundum ASD and with agitated saline (*right*) demonstrates the anechoic filling defect within the RA at the level of the IAS, consistent with a predominantly left-to-right shunting ASD.

Video 2: Two-dimensional TTE, right parasternal long-axis 4-chamber view at initial diagnosis (*left*) and after medical therapy for RHF was instituted (*right*), demonstrates improved, yet persistent, enlargement of the right heart from the ASD and improved preload to the left heart.

Video 3: Two-dimensional TTE, right parasternal transverse view at initial diagnosis (*left*) and after medical therapy for RHF was instituted (*right*), demonstrates slight improvement of the previous septal flattening and persistent right heart dilation.

View the video content online at www.cvcasejournal.com.

was started on outpatient oral heart failure medications (pimobendan 7.5 mg orally every 12 hours, furosemide 30 mg orally every 12 hours, enalapril 7.5 mg orally every 12 hours, and spironolactone 37.5 mg orally every 12 hours).

The patient was reevaluated 4 months postoperatively and had no clinical signs of heart failure and complete resolution of cavitary effusion. He had been diagnosed with hypothyroidism since the previous visit, and medical management with levothyroxine had been initiated. A CTA without cardiac gating was performed under anesthesia in sternal recumbency to evaluate the morphology of the ASD for potential interventional planning (Figure 2). Images were acquired in 0.6 mm isotropic and reconstructed in both 0.6 mm and 2-mm-thick slices, pitch of 0.8, 120 kVp, and 300 mAs. Ioxehol 350 mgI/mL was delivered intravenously via power injector at 600 mgI/kg. Bolus tracking was utilized with the trigger set to 80 HU on the aortic arch. Multiplanar reconstructions were created in 0.6 mm isotropic voxels. The Digital Image Communication in Medicine data sets were uploaded into cardiac 3D modeling software, and 3D virtual models of the heart were generated to assess the morphology of the ASD (Figure 3). The maximum and minimum ASD diameters were approximately 3.8 and 2.1 cm, respectively, and blood flow was demonstrated between the left atrium and RA. Due to the morphology of the defect and an insufficient rim of surrounding tissue, transcatheter closure of the ASD was not pursued. Open heart surgery was discussed with the owner but ultimately declined, and medical management was continued. The dog was presented for TTE rechecks at 3, 8, 15, and 20 months after the CTA. Two-dimensional TTE revealed improvement of the RV dilation and no progression of right heart disease (from initial study to most recent recheck: sequential long-axis mid RV cavity diameter in end diastole of 5.1, 4.4, 3.5, and 3.6 cm; short-axis RV, 5, 5.0, 3.9, 3.6, and 4.5 cm; Figure 4, Videos 2 and 3). The RA area index at diagnosis was 26.9 cm^2/m^2, and at subsequent visits it was 27.3, 18.1, and 16.8 cm^2/m^2, measuring 15.8 cm^2/m^2 at the most recent visit. The TR velocity ranged from equivocal to mildly increased through visits, with the most recent TR Vmax being 3.03 m/sec. The most recent recheck Qp:Qs was estimated to be 1.82. At the time of writing, the dog has been successfully medically managed for more than 2 years since the initial diagnosis of ASD and RHF, without relapse of active heart failure or echocardiographic progression of heart disease.

DISCUSSION

Similar to people born with initially hemodynamically insignificant CHD, veterinary patients may not be diagnosed with CHD until well into adulthood. Due to the lack of clinical signs of disease, the dog presented in this case report was not diagnosed with the ASD until almost 2 years of age. In people, a late diagnosis of ASD is attributed to the slow clinical disease progression, and ASD is the most commonly diagnosed congenital heart disease in adulthood (25%-30% of all newly diagnosed CHD).[1,8] The prevalence of ASD in adults is estimated to be 0.88 per 1,000 patients.[9] Those afflicted are typically asymptomatic until the second to fourth decades of life, at which time chronic pulmonary vascular overcirculation can lead to increased pulmonary vascular resistance, development of pulmonary hypertension (PH), and ultimately shunt reversal from right to left consistent with Eisenmenger physiology.[9,10]

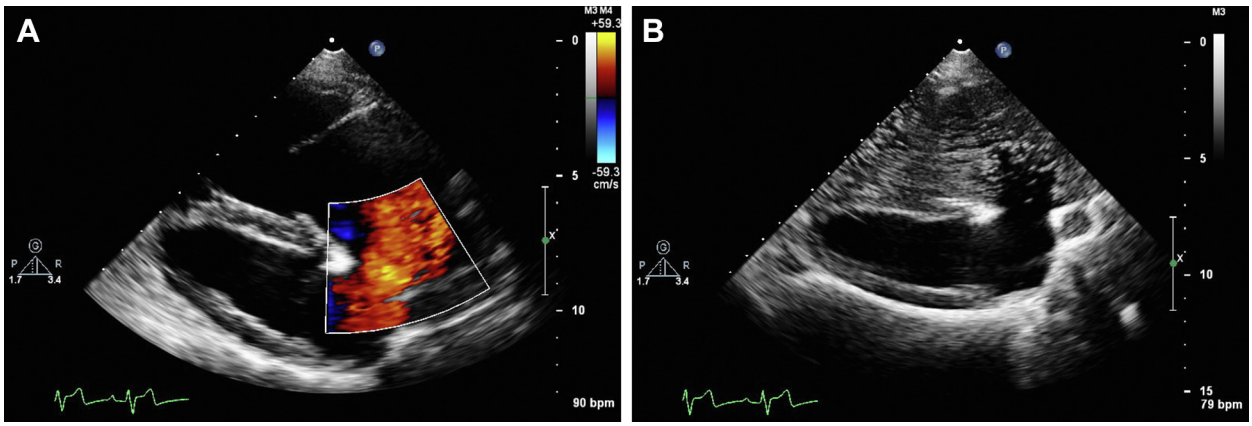

Figure 1 Two-dimensional TTE, right parasternal long-axis view with color-flow Doppler **(A)**, demonstrates the left-to-right signal across the secundum ASD, and with agitated saline **(B)** demonstrates the anechoic filling defect within the RA at the level of the IAS, consistent with left-to-right shunting.

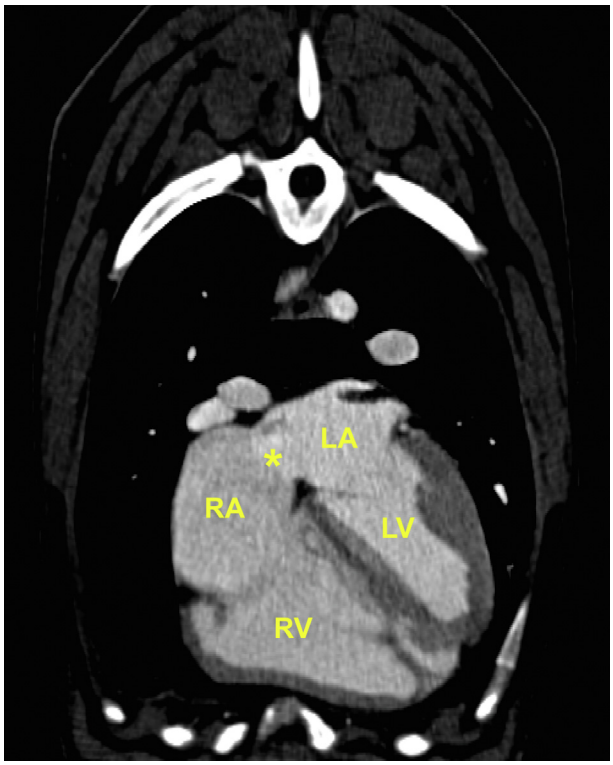

Figure 2 Computed tomography angiography multiplanar reconstruction, 4-chamber display, demonstrates the secundum ASD (*) with severe right heart dilation. *LA*, Left atrium; *LV*, left ventricle.

The prevalence of congenital ASD in dogs and cats is unknown, but as a percentage of dogs with diagnosed CHD, reported figures generally range from 1.1% to 3.1%.[2-4] Some dogs with an ASD might present without an obvious heart murmur, without chamber remodeling, or with a defect too small to see on standard TTE, and the defect may go unrecognized without an agitated saline study or without scrutinizing the IAS with proper Nyquist limit settings to detect low-velocity flow.[11]

Management of ASD is largely dependent on the location, size, and magnitude of the shunt, as well as presence or absence of clinical signs of disease.[1] Significant left-to-right shunting may result in volume overload of the RA and RV, increased pulmonary flow, and eventually RHF, all of which were noted in the dog in this case report. Over time, there is a risk of severe PH and shunt reversal, with up to 16% of adults with unrepaired ASD having concomitant PH and a 5% to 10% prevalence of Eisenmenger physiology; however, more recent studies report much lower prevalence of PH in patients with a repaired ASD (3%), likely due to earlier diagnosis and closure.[12] In people, the estimated mortality rate for an unclosed ASD is 25% by year 27 and 90% by year 60; however, ASD closure prior to age 25 in people is associated with excellent long-term prognosis and survival.[8] Similar prognostic data are not available in veterinary patients due to the paucity of prospective longitudinal studies, coupled with a high level of variability in disease progression.

Preprocedural assessment of congenital heart disease can be challenging due to complex 3D anatomy, which demands comprehensive understanding to determine pathophysiologic implications and to guide therapeutic intervention. Advancements in cross-sectional imaging in human cardiology have heralded a transformative shift in the diagnosis and treatment of CHD in veterinary medicine, progressively becoming more integral to its management. Cardiac computed tomography and cardiovascular magnetic resonance imaging, along with innovative 3D

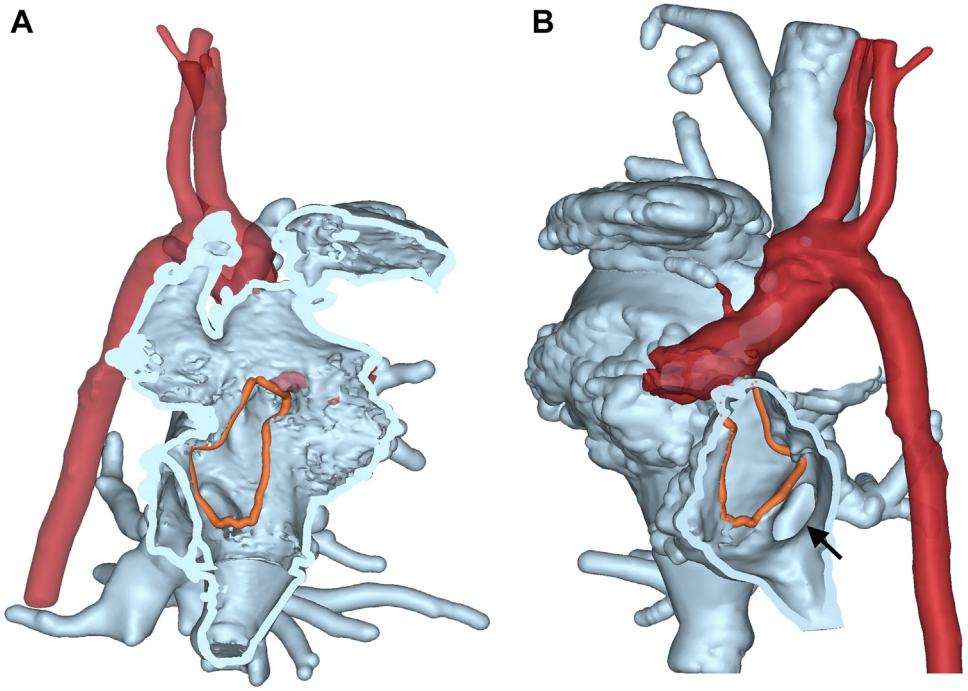

Figure 3 Three-dimensional CTA, volume-rendered displays with the right heart (*blue*) and aorta with great arteries (*red*) separately highlighted from the RA **(A)** and LA **(B)** perspective. The atria have been virtually opened by manipulating the slice thickness and cropping into the chambers. The ASD is traced (*orange line*) and emphasizes the proximity of the large ASD to the aortic root and pulmonary vein origins (*arrow*).

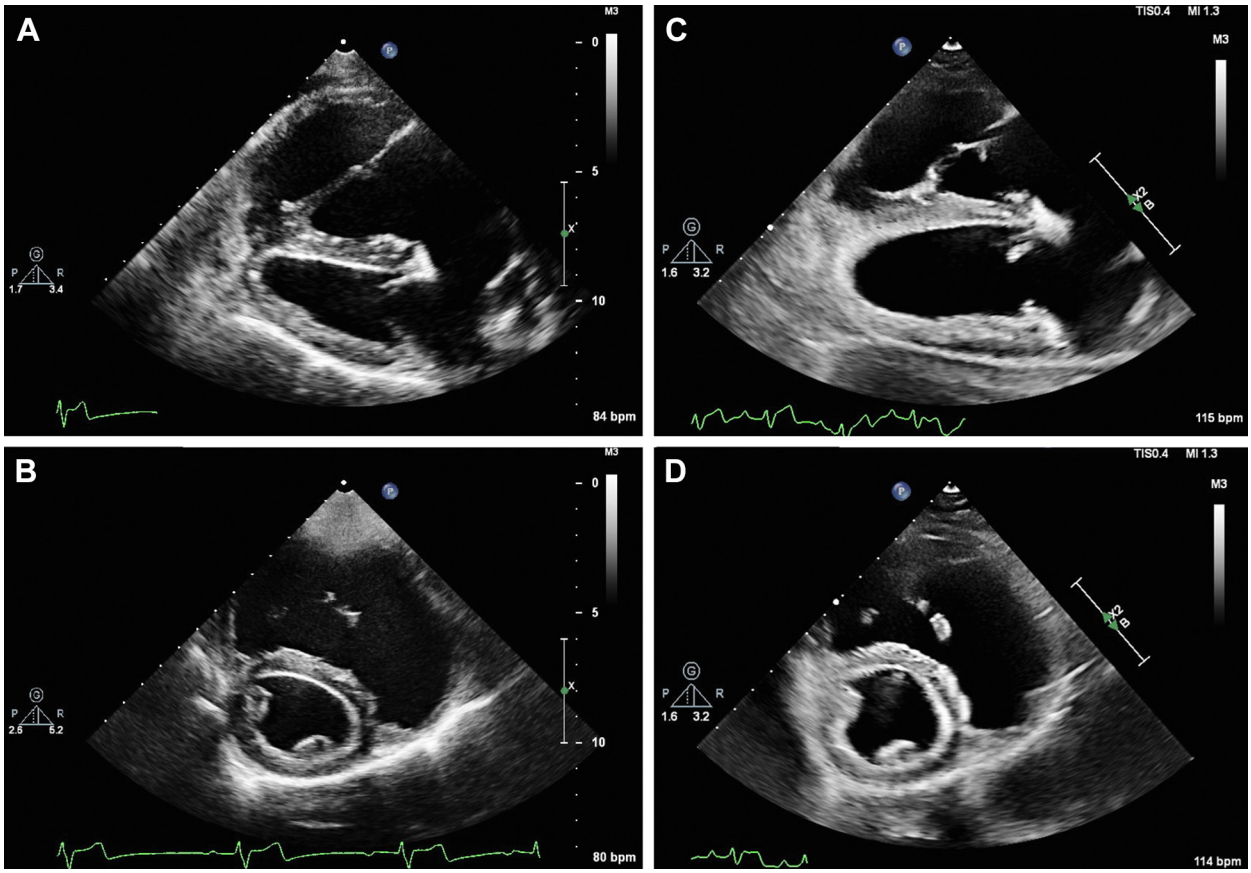

Figure 4 Two-dimensional TTE images at initial diagnosis **(A, B)** and after medical therapy for RHF was started **(C, D)**. **(A)** Right parasternal long-axis 4-chamber view demonstrates severe RA and RV dilation from the secundum ASD and volume underloading in the LA and LV. **(B)** Right parasternal transverse view demonstrates severe RV dilation and evidence of diastolic septal flattening. **(C)** Right parasternal long-axis 4-chamber view demonstrates improved, yet persistent, enlargement of the right heart from the ASD and improved preload to the left heart. **(D)** Right parasternal transverse view demonstrates slight improvement of the previous septal flattening and persistent right heart dilation.

modeling and printing, are complementary to standard 2D and 3D echocardiography for providing the most comprehensive data for interventional and surgical planning. Transcatheter, surgical, or hybrid interventions are available for ASD closure in both people and in animals.[8,13] The typical indication for shunt closure is a moderate or large ASD with significant left-to-right shunting or if there is a pulmonary to systemic shunt ratio/fraction (Qp:Qs) of >1.5.[10] While shunt fraction can be derived via TTE, there can be significant inconsistencies between echo-derived and cardiac catheterization shunt calculations; therefore the generally accepted sign of a hemodynamically significant ASD that warrants closure is RV dilation with normal or hyperdynamic function.[14] Closure of an ASD is contraindicated if there is significant right-to-left shunting, severe PH (defined as pulmonary vascular resistance >8 WU), or irreversible pulmonary vascular occlusive disease[10], in which case medical management with an endothelin receptor antagonist or phosphodiesterase 5 inhibitor is recommended.[1] Transcatheter closure with an occluding device is contraindicated in people if the defect is >36 mm, if there are inadequate rims or margins to seat the device, or if there is potential device interference with atrioventricular valves or venous drainage, multiple defects, or excessively bulging atrial septal aneurysm.[1]

Hemodynamically significant ASD in dogs and cats is not routinely closed due to cost of available occluding devices, suitability of ASD morphology for a device, need for additional training for transcatheter occlusion, and lack of availability of cardiopulmonary bypass at most veterinary centers. However, several centers have demonstrated success with transcatheter, surgical, and hybrid intervention in dogs and cats.[13,15] The dog in this case report was not considered a candidate for transcatheter ASD occlusion due to the size of the defect and the lack of suitable tissue margins around the defect for device stability.

While the dog presented here was not diagnosed until almost 2 years of age, the diagnosis occurred during the time of hospitalization for GDV. The volume loading related to therapy of the GDV likely caused an acute right heart decompensation of this dog's previously undiagnosed secundum ASD. As TTE was not performed in this dog prior to the GDV, to know the extent of the right heart overload, it is possible that this dog would have remained subclinical for 2 years or longer had the patient not developed GDV. Congestive heart failure medications were first started in the dog when, 2 weeks after being released from the hospital for GDV management, the dog developed progressive pleural effusion despite being at home and off any IV fluids. It is possible that the GDV unmasked an undiagnosed large secundum ASD, and medical management might not have been indicated for some time in this case. Nonetheless, the patient's CHD has now been medically managed for more than 2 years without recurrence of heart failure and with minimal progression of heart disease.

CONCLUSION

Atrial septal defect in dogs has a variable presentation and might not require closure depending on the degree of hemodynamic significance. The dog in this case report demonstrates that even large defects can be managed medically for years without the need for transcatheter or surgical intervention.

CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

ETHICS STATEMENT

The authors declare that the work described here has been carried out in accordance with the ethical policies and animal use approval by the University of Georgia College of Veterinary Medicine Clinical Research Committee.

FUNDING STATEMENT

This work was supported by a grant from the Companion Animal Fund within the Small Animal Medicine and Surgery Department at the University of Georgia College of Veterinary Medicine in Athens, GA, USA.

DISCLOSURE STATEMENT

The authors report no conflicts of interest.

ACKNOWLEDGMENTS

We thank the Cardiology Service at the University of Georgia for their continued dedication to medical management of this case.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2023.12.022.

REFERENCES

1. Brida M, Chessa M, Celermajer D, Li W, Geva T, Khairy P, et al. Atrial septal defect in adulthood: a new paradigm for congenital heart disease. Eur Heart J 2022;43:2660-71.
2. Oliveira P, Domenech O, Silva J, Vannini S, Bussadori R, Bussadori C. Retrospective review of congenital heart disease in 976 dogs. J Vet Internal Med 2011;25:477-83.
3. Schrope DP. Prevalence of congenital heart disease in 76,301 mixed-breed dogs and 57,025 mixed-breed cats. J Vet Cardiol 2015;17:192-202.
4. Brambilla PG, Polli M, Pradelli D, Papa M, Rizzi R, Bagardi M, et al. Epidemiological study of congenital heart diseases in dogs: prevalence, popularity, and volatility throughout twenty years of clinical practice. PLoS ONE 2020;15:e0230160.
5. Gentile-Solomon JM, Abbott JA. Conventional echocardiographic assessment of the canine right heart: reference intervals and repeatability. J Vet Cardiol 2016;18:234-47.
6. Visser LC, Ciccozzi MM, Sintov DJ, Sharpe AN. Echocardiographic quantitation of left heart size and function in 122 healthy dogs: a prospective study proposing reference intervals and assessing repeatability. J Vet Intern Med 2019;33:1909-20.
7. Serres F, Chetboul V, Tissier R, Gouni V, Desmyter A, Sampedrano CC, et al. Quantification of pulmonary to systemic flow ratio by a Doppler echocardiographic method in the normal dog: repeatability, reproducibility, and reference ranges. J Vet Cardiol 2009;11:23-9.
8. Oster M, Ami BA, Zaragoza-Macias E, Dendukuri N, Marelli A. Interventional therapy versus medical therapy for secundum atrial septal defect: a systematic review (Part 2) for the 2018 AHA/ACC guideline for the management of adults with congenital heart disease A report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. Circulation 2019;139:E814-30.
9. Marelli AJ, Ionescu-Ittu R, Mackie AS, Guo L, Dendukuri N, et al. Lifetime prevalence of congenital heart disease in the general population from 2000 to 2010. Circulation 2014;130:749-56.
10. Bradley EA, Zaidi AN. Atrial septal defect. Cardiol Clin 2020;38:317-24.
11. Silvestry FE, Cohen MS, Armsby LB, Burkule NJ, Fleishman CE, Hijazi ZM, et al, American Society of Echocardiography, Society for Cardiac Angiography and Interventions. Guidelines for the echocardiographic assessment of atrial septal defect and patent Foramen Ovale: from the American Society of Echocardiography and Society for Cardiac Angiography and Interventions. J Am Soc Echocardiogr 2015;28:910-58.
12. Lammers AE, Bauer LJ, Diller GP, Helm PC, Abdul-Khaliq H, Bauer UMM, et al., German Competence Network for Congenital Heart Defects Investigators. Pulmonary hypertension after shunt closure in patients with simple congenital heart defects. Int J Cardiol 2020;308:28-32.
13. Gordon SG, Miller MW, Roland RM, Saunders AB, Achen SE, Drourr LT, et al. Transcatheter atrial septal defect closure with the Amplatzer® atrial septal occluder in 13 dogs: short- and mid-term outcome. J Vet Intern Med 2009;23:995-1002.
14. Baumgartner H, De Backer J, Babu-Narayan SV, Budts W, Chessa M, Diller GP, et al., ESC Scientific Document Group. 2020 ESC guidelines for the management of adult congenital heart disease. Eur Heart J 2021;42:563-645.
15. Gordon SG, Nelson DA, Achen SE, Miller MM, Roland RM, Saunders AB, et al. Open heart closure of an atrial septal defect by use of an atrial septal occluder in a dog. J Am Vet Med Assoc 2010;236:434-9.