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STANDARD ARTICLE

Anatomy, baseline characteristics, and procedural outcome of patent ductus arteriosus in German Shepherd dogs

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Background: German Shepherd dogs (GSD) are predisposed to developing patent ductus arteriosus (PDA) and are reportedly prone to type III (tubular) PDA anatomy. Dogs with type III anatomy are not considered favorable candidates for device-based intervention.

Objective: To describe the PDA anatomy, baseline characteristics, and procedural outcome of GSD with PDA.

Animals: Twenty-eight client-owned GSD.

Methods: Retrospective review of medical records of 28 GSD diagnosed with PDA that underwent surgical ligation or transcatheter device closure between 2007 and 2017.

Results: German Shepherd dogs with PDA often presented with clinical signs (50%), concurrent congenital heart disease (35.7%), and arrhythmias (29%). Dogs were typically mature at presentation (median age, 12.1 months) and evenly distributed by sex (57% female). The PDA anatomy was classified in 24 of 28 GSD, with type II anatomy being most common (21/24). Three dogs had unusual anatomy (type IV in 1, type V in 2). Median minimal ductal diameter (MDD) in this population was larger than previously reported in a mixed population and ranged between 4.4 and 4.9 mm depending upon imaging modality. Successful closure was achieved using an Amplatz canine duct occluder (ACDO) in 22 dogs or by surgical ligation in 6 dogs. No cases of type III anatomy were confirmed.

Conclusions and Clinical Importance: The majority of GSD in this population had type II PDA anatomy that was amenable to ACDO deployment. Predisposition for large MDD and occasional, unusual PDA anatomy suggests that transesophageal echocardiography may be beneficial for optimal procedural planning in this breed.

KEYWORDS

canine, congenital, ligation, minimal ductal diameter, morphology

1 | INTRODUCTION

Patent ductus arteriosus (PDA) is a common congenital heart defect in the dog.[1,2] Considerable variability in PDA anatomy occurs among affected dogs, with a previous angiographic classification scheme consisting of 4 distinct phenotypes: type I, type IIa, type IIb, and type III.[3] Recent work suggests that type IV (multiple narrowings of the ampulla) and type V (other configurations) phenotypes should be added to the previous classification scheme,[4] with type IV being the same as the previously reported type D.[5,6] Of these phenotypes, a type III PDA is described as having a tubular appearance with minimal to no tapering in ductal diameter before insertion into the pulmonary artery.[3] Identification of a type III PDA impacts treatment options, because this anatomy is not considered amenable to interventional procedures with devices such as the Amplatz canine duct occluder (ACDO; Infiniti Medical, LLC Haverford, Pennsylvania). In many dogs with type III PDA anatomy, surgical ligation is

Abbreviations: ACDO, Amplatz canine duct occluder; Amp-A, ampulla diameter measured by angiography; Amp-TEE, ampulla diameter measured by transesophageal echocardiography; CHF, congestive heart failure; GSD, German Shepherd dogs; MDD, minimal ductal diameter; MDD-A, MDD measured by angiography; MDD-TEE, MDD measured by transesophageal echocardiography; MDD-TTE, MDD measured by transthoracic echocardiography; PDA, patent ductus arteriosus; PLCVC, persistent left cranial vena cava; SAS, subaortic stenosis; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography.

performed to avoid device-related complications arising from inadequate device stability.

German Shepherd dogs (GSD) are predisposed to PDA, with a reported odds ratio of 5.2.[2] Among published studies of dogs with PDA where breed frequency is reported, GSD often are the most commonly represented purebred population.[5,7–10] In addition, this breed also is reportedly predisposed to type III PDA anatomy. The published evidence for this proposed predisposition ranges from comments regarding author experience[3] to individual cases of procedural abandonment in GSD because of type III PDA anatomy that resulted in easy dislodgement of an ACDO with gentle push/pull manipulation (n = 2).[11] Detailed description of the frequency of type III PDA anatomy in the GSD breed is lacking. In 3 previously published studies of dogs and 2 case reports that included angiographic descriptions of PDA anatomy, no type III morphology was identified despite 32 reported GSD included in these populations.[5,9,12–14]

Our objective was to describe PDA anatomy and procedural outcome of all GSD that were diagnosed with PDA and received definitive treatment, either surgical ligation or device-based intervention, over a 10-year period. Additional information about minimal ductal diameter (MDD), ampulla diameter, reason for surgical ligation, baseline characteristics at presentation, and long-term follow-up was also evaluated.

2 | MATERIALS AND METHODS

A search of the Texas A&M University Veterinary Medical Teaching Hospital's veterinary medical information system identified 28 GSD that were diagnosed with PDA and underwent definitive treatment between May 2007 and May 2017. The body weight, age, sex, angiographic PDA anatomy,[3,4] MDD measured by angiography (MDD-A), MDD measured by transesophageal echocardiography (MDD-TEE), MDD measured by transthoracic echocardiography (MDD-TTE), ampulla diameter measured by angiography (Amp-A), ampulla diameter measured by transesophageal echocardiography (Amp-TEE), ACDO size deployed, concurrent congenital heart defects, echocardiographic data, documented arrhythmias (noted on 6-lead ECG or on contemporaneous ECG monitoring during echocardiographic examination), presence or absence of clinical signs, and congestive heart failure (CHF) status were recorded for each dog as available. Angiograms were performed as previously described.[3] Sizing for ACDO devices was based on previously published MDD and ampulla oversize recommendations,[12] with the best available imaging modality, or a combination of modalities, for any given patient utilized to determine the MDD felt to be most accurate. Transesophageal echocardiography (TEE) commonly is performed preoperatively, perioperatively, or at both time points in all dogs undergoing interventional PDA correction that are large enough to accommodate the transesophageal probe used at our institution; however, this approach was not standard toward the beginning of the study period. Transesophageal echocardiography was performed in accordance with previously published guidelines.[15] Follow-up data was sought using internal records search as well as contact with referring veterinarians, clients, or both.

All MDD-A and MDD-TEE measurements were obtained by a single operator (Ashley B. Saunders), either at the time images were acquired or upon subsequent review. All MDD-TTE, Amp-A, and Amp-TEE measurements were obtained by a single operator (Sonya Wesselowski) by review of stored images. The single best image was identified and used to measure MDD-TTE after reviewing all stored right-sided and left-sided images for each dog. All measurements of ampulla diameter were made at a level 3 mm above the MDD. Angiographic PDA anatomy was determined by review of stored right lateral angiograms by a single operator (Sonya Wesselowski) and morphology classification was based on a recently updated description.[4]

Descriptive statistics are reported as median (range) where applicable. A Kaplan-Meier analysis was done using a statistical analysis package (XLSTAT 2017: Data Analysis and Statistical Solution for Microsoft Excel; Addinsoft, Paris, France) in all dogs that had documented follow-up after PDA closure (n = 27) to estimate the median and interquartile range for survival in years after PDA closure as well as the overall median survival time. As of May 2018, 8 dogs were known to be dead. The remaining 19 were censored at the time they were last known to be alive.

3 | RESULTS

Twenty-eight GSD had definitive treatment for PDA during the study period. Twenty-two had PDA occlusion using an ACDO, whereas 6 had surgical ligation of their PDA. There were 16 females (57%), 8 neutered, and 12 males (43%), 4 neutered. Median age at the time of presentation was 12.1 months (range, 2-109 months) and median body weight was 9.9 kg (range, 5.7-37.2 kg).

Fourteen of the 28 GSD (50%) were asymptomatic with no history of clinical signs at the time of presentation. Of the 14 dogs with reported clinical signs, in 8 dogs there were either on-going clinical signs consistent with CHF (eg, lethargy, exercise intolerance, cough, tachypnea) or previously reported clinical signs of CHF that were currently controlled with medical treatment that included furosemide. The diagnosis of CHF was made after identifying radiographic evidence of pulmonary edema in 4 dogs, ascites in 2 dogs presented with atrial fibrillation, and resolution of cough and lethargy after furosemide administration in 2 dogs. Five dogs had exercise intolerance, lethargy, or both but no radiographic evidence of CHF, although 1 of these dogs also had a previously reported cough that resolved after treatment with pimobendan before referral. The final dog with clinical signs was reported to have excessive panting by the owner but had no radiographic evidence of CHF at the time of evaluation and was not receiving diuretic treatment.

Arrhythmias were recognized in 8 dogs (29%), 5 of which had CHF. In 3 of these dogs, atrial fibrillation was documented, and in 5 dogs, infrequent to rare single ventricular premature contractions were identified. Two of the dogs with atrial fibrillation also had infrequent, wide QRS complexes that either represented single ventricular premature contractions or supraventricular beats conducted with aberrancy.

Angiography images were available for review in 21 of the 22 dogs that underwent ACDO placement but for none of the 6 dogs that underwent surgical ligation. Seventeen dogs had type IIA

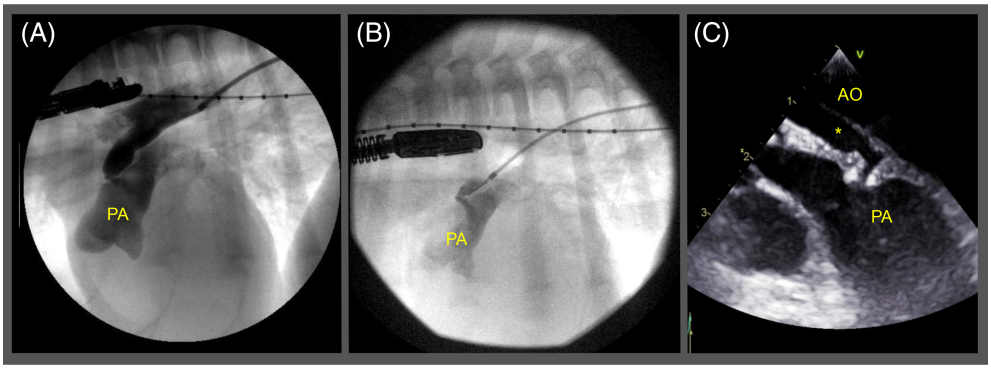

FIGURE 1 A and B, Angiograms from 2 German Shepherd dogs displaying type V (other configurations) patent ductus arteriosus (PDA) anatomy. Both dogs have a narrow section (longer in the dog in panels [B] and [C]) as the ampulla approaches the pulmonary ostium and an acute angle into the pulmonary artery resulting in a caudally directed contrast jet entering into the main pulmonary artery. Dog A also has a second narrowing of the mid-ampulla region consistent with type IV PDA anatomy. C, Transesophageal echocardiographic image corresponding to the angiogram labeled B, in which the PDA ampulla (*) substantially narrows, forms a brief tunnel, then turns acutely toward the pulmonary artery, with excessive tissue on the pulmonary artery side of the ostium. Images of this dog have previously been published.[4] AO, aorta; PA, pulmonary artery

anatomy, 1 had type IIB anatomy, 1 had type IV anatomy, and 2 had type V anatomy (1 of which also had a second narrowing of the ampulla consistent with type IV anatomy[4]). Both dogs with type V (other configurations) anatomy had a narrow section of tissue that formed a tunnel as the ampulla approached the pulmonary ostium and then turned acutely into the pulmonary artery resulting in contrast entering into the pulmonary artery in a caudal direction (Figure 1A and B). Transesophageal echocardiographic images in 1 of these cases further characterized the unusual anatomy (Figure 1C).

The MDD-TTE measurement was available in 16 of 22 GSD that underwent ACDO placement and in 4 of 6 GSD that underwent surgical ligation. The MDD-A measurement was available in 21 of 22 GSD that underwent ACDO placement and in none of the 6 dogs that underwent surgical ligation. The MDD-TEE measurement was available in 19 of 22 GSD that underwent ACDO placement and in 3 of 6 GSD that underwent surgery. The median and range for MDD-TTE, MDD-A, and MDD-TEE measurements are presented in Table 1.

The Amp-A measurement was available in 20 of 22 GSD that underwent ACDO placement and in none of the dogs that underwent surgical ligation. The Amp-TEE measurement was available in 14 of 22 dogs that underwent ACDO placement and in 3 of the 6 dogs that underwent surgical ligation. The median and range for Amp-A and Amp-TEE measurements are presented in Table 1.

Amplatz canine duct occluder placement was successful in all 22 dogs that underwent this method of PDA correction, including those with type V anatomy. No perioperative or postoperative complications were reported. In 19 of 22 dogs, the ACDO size was recorded, with the median ACDO size deployed being size 8 (range, 3-14). The originally chosen ACDO size was the size deployed in 18 of 19 dogs for which records were available. In the final dog, the first ACDO deployed was not felt to provide adequate ductal occlusion because of a pronounced, dynamic change in MDD size throughout the cardiac cycle that was appreciated with TEE. The ACDO then was retracted and an ACDO 1 size smaller was purposefully deployed entirely within the ductal ampulla. No residual ductal flow was detected 1 day postoperatively, and the device remained in position at all subsequent follow-up examinations.

The reason for surgical ligation in the 6 identified cases varied. In 2 cases, concurrent pneumonia and perceived risk of bacteremia were documented as a primary reason for avoidance of device placement. No transthoracic or transesophageal images were available for review in these 2 cases, although 1 dog had a reported MDD-TTE of 7.7 mm. In 1 dog, procedural error during catheterization necessitated an emergency thoracotomy before device deployment, but perioperative TEE confirmed tapering ductal anatomy consistent with type II morphology (MDD-TEE of 5.8 mm, Amp-TEE of 12.4 mm). In 1 dog, client preference dictated surgical ligation over additional anesthesia time for TEE to determine whether ACDO deployment was feasible. This dog had an estimated MDD-TTE of 8 mm, suggesting a device large enough to successfully close the PDA would not be available. In another case, clinician preference dictated surgical ligation over ACDO deployment despite tapering anatomy confirmed on TEE (MDD-TEE of 4.6 mm, Amp-TEE of 11.1 mm). In the final case, the MDD-TTE was estimated to be too large (9.2 mm) for the largest ACDO device. Subsequently, tapering ductal anatomy was confirmed with TEE during surgical ligation in this dog (MDD-TEE of 7.3 mm, Amp-TEE of 14.9 mm) although the MDD-TEE indicated that the largest ACDO might not be adequate for occlusion. Overall, tapering ductal anatomy consistent with type II phenotype was confirmed by TEE in 3 of 6 dogs that were sent to surgery, with no angiography

TABLE 1 Minimal ductal diameter and ampulla diameter measurements obtained with various imaging modalities

	Angiography		Transesophageal echocardiography		Transthoracic echocardiography	
	N	Median (range)	N	Median (range)	N	Median (range)
Minimal ductal diameter (mm)	21	4.5 (1.6-6.4)	22	4.4 (2.0-7.3)	20	4.9 (3.0-9.2)
Ampulla diameter (mm)	20	6.9 (3.3-16.1)	17	10.4 (4.4-14.9)	Not available	

N indicates number of dogs available out of 28 dogs.

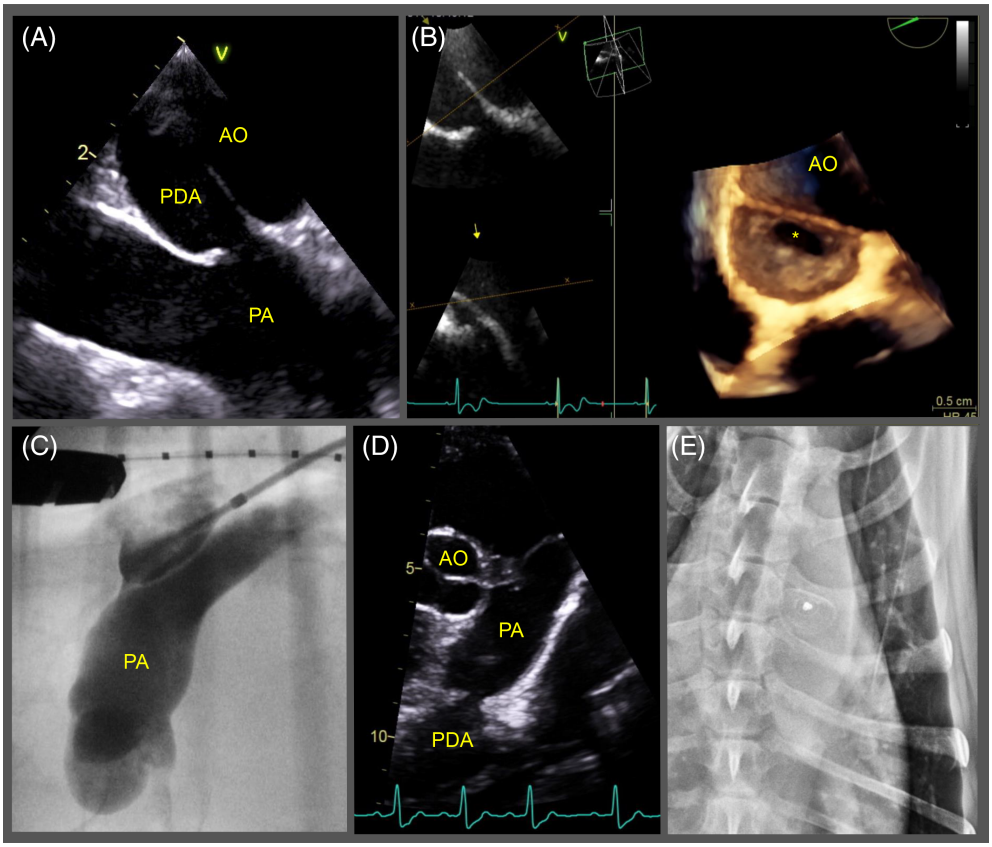

FIGURE 2 Images from a German Shepherd dog (GSD) with a type II patent ductus arteriosus (PDA) morphology. A, Transesophageal echocardiographic image demonstrating ductal anatomy that appears to taper only on 1 side, whereas flat on the other, consistent with the 2-dimensional appearance of an eccentric pulmonary ostium in association with type II PDA morphology. B, Three-dimensional transesophageal echocardiography image looking down the PDA ampulla depicting the eccentric (noncentral) location of the pulmonary ostium (*) relative to the PDA ampulla. Two-dimensional reference images for the 3-dimensional image appear to the left of the 3-dimensional image. The lateral angiogram (C) and transthoracic echocardiogram (D) failed to highlight this anatomic difference in this case. E, Zoomed ventrodorsal radiograph after ACDO deployment shows flattening of the proximal cup of the device on 1 side due to the eccentric pulmonary ostium. AO, aorta; PA, pulmonary artery

or TEE to better define anatomy in the other 3 dogs. Surgical ligation was successful in all 6 cases.

During TEE image review, 3 GSD with type II PDA anatomy also were noted to have an eccentric (not central) location of the pulmonary ostium relative to the ampulla of the PDA. On 2-dimensional TEE, this anatomy gives an appearance of the PDA ampulla being flat on 1 side and tapered on the other (Figure 2A). Three-dimensional TEE confirms the eccentric position of the ostium (Figure 2B). Two of these 3 dogs were occluded successfully using an ACDO, but the proximal cup of the device did not assume a completely native shape after release because of the eccentric location of the ostium (Figure 2E). The PDA in the third dog was surgically ligated for reasons unrelated to anatomy. An eccentric pulmonary ostium was not appreciable on angiographic (Figure 2C) or transthoracic (Figure 2D) images in the dog presented in Figure 2.

Ten of the GSD (35.7%) had concurrent congenital heart defects identified in addition to their PDA. These defects included subaortic stenosis (SAS, n = 9), persistent left cranial vena cava (PLCVC; n = 3), valvular pulmonic stenosis (n = 1), supravalvular pulmonic stenosis (n = 1), and tricuspid valve dysplasia (n = 1). Seven GSD had a single additional congenital lesion, 2 had 2 additional lesions, and 1 had 4 additional lesions. Diagnosis of SAS was made based upon documentation of increased transaortic velocity measured 1 day after successful PDA occlusion or ligation with a normal cutoff of <2.25 m/s.[16,17] A subvalvular ridge in the left ventricular outflow tract also was identified in 6 dogs. Dogs considered affected with SAS in this population had post-procedural aortic velocities ranging between 2.9 and 3.8 m/s. Pre-procedural aortic velocities in these dogs ranged between 3.6 and 6.6 m/s, whereas pre-procedural aortic velocities in dogs that went on to have aortic velocities <2.25 m/s after their procedure ranged between 1.2 and 4.7 m/s. Diagnosis of PLCVC was confirmed by direct observation during thoracotomy in 2 dogs and inferred based upon substantial dilatation of the coronary sinus on TTE in the absence of concurrent right-sided cardiac disease in 1 dog.

Pre-procedural normalized left ventricular internal dimensions were >1.85[18] at end diastole in 25 of 28 dogs and >1.26[18] at end-systole in 18 of 28 dogs. Normalized left ventricular internal dimensions 24 hours after PDA closure were >1.85 at end-diastole in 11 of 28 dogs and >1.26 at end-systole in 16 of 28 dogs. Post-procedure echocardiograms indicated no evidence of residual flow in all 22 dogs that underwent ACDO deployment before discharge. Of dogs that underwent surgical ligation, 2 had moderate residual flow[19] and 1 had mild residual flow[19] postoperatively before discharge. Fifteen of 28 dogs had aortic insufficiency noted pre-procedurally, whereas

21 of 28 dogs had aortic insufficiency noted post-procedurally. Twenty-three of 28 dogs had mitral regurgitation noted pre-procedurally, with 19 of 28 dogs having persistent mitral regurgitation post-procedurally. The severity of post-procedural mitral regurgitation before discharge was trivial in 5, mild in 9, moderate in 4, and severe in 1 dog based upon color Doppler regurgitant jet area.[20]

Congestive heart failure resolved and furosemide was successfully discontinued in all 8 dogs after ACDO placement (n = 4) or surgical ligation (n = 4). In 7 cases, furosemide administration had been discontinued by the time of discharge from the hospital. In the 8th case, it was discontinued 48 hours after discharge from the hospital.

One-year survival data were available for 24 dogs, with 4 dogs lost to follow-up. Twenty-three dogs were alive (96%), and 1 dog (4%) had died 51 weeks postoperatively. The dog that died had presented at 6.5 years of age in CHF with atrial fibrillation and ventricular premature contractions and had persistent left-sided heart enlargement, systolic dysfunction, and severe magnetic resonance post-procedurally. Two-year survival data were available for 21 dogs, with 6 lost to follow-up and 1 dog not having 2 years of elapsed time since surgery. Of these 21 dogs, 17 were alive (81%) and 4 were dead (19%). Three-year survival data were available for 19 dogs, with 7 lost to follow-up and 2 dogs not having 3 years of elapsed time since surgery. Of these 19, 15 were alive (79%) and 4 were dead (21%). The estimated median survival time after PDA closure in this population was 8.3 years. The 25th percentile was estimated as 5.4 years and the 75th percentile could not be estimated. The estimated median overall survival time was 9.6 years with an interquartile range of 7.5-12.8 years.

4 | DISCUSSION

The data presented here suggest that GSD presenting with PDA may differ in several ways compared to previously published, all-breed data. Baseline characteristics in large cohorts of dogs with PDA consistently report a predisposition for females (73.1%-80%), a relatively young age at presentation (median, 5.1-7.0 months), infrequent arrhythmias (2.9%-15%), and an absence of clinical signs in the majority of cases (69%-73.8%).[8,21] In our population of GSD, sex distribution was more balanced, with 57% of dogs being female. German Shepherd dogs also were presented at an older age (median, 12.1 months) and were more likely to have reported clinical signs (50%) and arrhythmias (29%) at presentation.

In this population of GSD, concurrent congenital heart disease also was more prevalent (35.7%) than previous reports of only 8.8%-10%.[8,21] The most common concurrent defect was SAS, with 9 dogs having increased transaortic velocities ranging between 2.9 and 3.8 m/s after PDA closure. The cutoff for normal transoartic velocity remains controversial among veterinary cardiologists, with some considering 2.0 m/s a more appropriate upper normal limit. Breed-specific data for GSD suggest that in 95% of these dogs, normal transaortic velocities range between 0.7 and 1.3 m/s.[22] Had a cutoff of 2.0 m/s been used in this cohort of GSD, 5 more dogs would have been considered abnormal, potentially increasing the percentage of GSD affected by both PDA and SAS from 32% to 50%. Even this figure potentially could be an underestimation, given the persistence of left ventricular systolic dysfunction after PDA occlusion in many of these GSD (16/28 had increased normalized left ventricular end-systolic internal dimensions post-procedurally). In the absence of postmortem data, however, confirmation of mild forms of SAS remains difficult and open to interpretation.

The identification of 3 dogs in this population with concurrent PLCVC may suggest a possible predisposition for this infrequently reported congenital defect in the breed. Published case reports of PLCVC have included GSD in 1 prior instance.[23] Although this defect is considered clinically irrelevant, surgeons performing PDA ligation should be aware of this potential anatomic variant, because the PLCVC crosses over the base of the heart on the left, potentially interfering with visualization of a PDA during a standard left thoracotomy approach. This is particularly true if a right cranial vena cava is absent, causing the PLCVC to be of larger size.[23]

With regard to PDA anatomy, 24 of 28 dogs in our study could be classified, with 21 classified based on angiography and 3 based on TEE. The majority of GSD were found to have type II anatomy (21 of 24 dogs), typically type IIA. Interestingly, the other 3 GSD were best characterized as having type IV and type V anatomy.[4] There were no confirmed cases of type III PDA anatomy in this cohort. Of the 4 GSD in which anatomy could not be confirmed, 1 dog did not have stored angiographic cine loops available for review. In this adult GSD, a size 3 ACDO was successfully used for PDA occlusion, suggesting that the PDA was very small. In the other 3 instances, type III anatomy cannot entirely be ruled out, but none of the records specifically noted suspected type III anatomy as the reason for surgical ligation. Overall, these results suggest that despite a purported predisposition for type III anatomy, this anatomic phenotype should not be assumed to be more common than others in GSD and actually may be uncommon or rare in the breed. Most GSD appear to have type II anatomy, with a small number having other phenotypes that appear just as likely to be type IV or type V as type III.

Comparison of MDD in GSD versus other dogs is difficult, given the retrospective nature of our study and the variability in imaging modalities used for MDD measurements in previous work. Despite these limitations, some useful comparisons can be drawn. In 1 population of 112 dogs with PDA, MDD was measured by TEE, TTE, or angiography, depending on the patient, and resulted in a median MDD of 2.2 mm (range, 1.0-8.0 mm).[24] In another population of 246 dogs, MDD was measured angiographically in all cases and resulted in a median MDD of 2.5 mm (range, 1.0-9.5 mm).[3] In the GSD reported here, median MDD was 4.4-4.9 mm depending upon the imaging modality used, with an overall range of 1.6-9.2 mm. Of the GSD with an available MDD-A, the median was 4.5 mm. These comparisons suggest that median MDD in GSD does seem to be larger than reported median MDD in all breed populations. This conclusion is further supported by 3 prior cases of very large MDD deemed too large for successful occlusion with the largest ACDO or vascular plug in GSD in the absence of type III anatomy.[11,24] Additionally, median ACDO size deployed in a mixed population cohort of 152 dogs from our institution was size 6,[10] whereas the median ACDO size deployed in the GSD of this study was size 8.

Accurate measurement of MDD is essential for adequate device sizing, appropriate procedural selection, and subsequent procedural success. The recommended method for ACDO sizing is to select a device

with a waist that is twice the diameter of the MDD.[25] Any dog with an MDD-TTE >7 mm on initial screening has the potential to require surgical backup, because the largest ACDO available (size 14) may not provide adequate oversize and device stability. Sole reliance on TTE to determine if the MDD is too large for an ACDO is likely insufficient, however, because MDD-TTE has been shown to overestimate MDD compared to angiography or TEE-derived measurements.[4,9,26] Additionally, TTE can be suboptimal in some GSD, such that tapering anatomy is difficult to definitively confirm without additional imaging modalities such as TEE. Type II anatomy with a large MDD occasionally could be mistaken for type III anatomy in a dog with suboptimal image quality and TTE imaging alone. Thus, in GSD suspected of having type III anatomy, or those with large MDD-TTE, it may be prudent to recommend additional pre-procedural imaging before determining the necessity of surgical ligation in an individual dog.

Limitations of our study are largely related to its retrospective nature. Missing data for different imaging modalities in the same patient limited the utility of statistical comparison for different measurements of MDD and also precluded our ability to definitively confirm PDA anatomy in 4 cases. In addition, the anatomy that was confirmed in this cohort reflects the GSD population and genetic lines within the referral region of our institution and may not reflect larger populations within the United States or in other parts of the world. A prospective, anatomic study comparing TEE, TTE, and angiographic imaging of a larger, more geographically diverse, GSD cohort would be useful.

In summary, GSD with PDA in this population were more likely to present older, have clinical signs at the time of diagnosis, and have more frequent concurrent congenital heart defects and arrhythmias than other breeds. Although a female predominance was noted, it was less pronounced than in previous reports for all-breed populations. German Shepherds in this cohort most commonly had type II PDA anatomy, with rare instances of type IV and type V anatomy. Despite a tendency for GSD to have a larger median MDD than previously reported with all-breed data, in the majority of cases, tapering ductal anatomy allowed for PDA occlusion by ACDO deployment. Occlusion or ligation of the PDA offered a clinically relevant survival benefit and resulted in an overall median survival time (9.6 years) consistent with a relatively normal life span for GSD. Additional imaging with TEE may be required for optimal procedural planning in this breed.

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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.

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