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KEYWORDS
Congenital; Feline; Pulmonary hypertension; Surgery; Shunt

**Abstract**  
**Objectives:** To describe signalment, clinical characteristics, diagnostic, treatment, and outcome data in a large case series of cats with patent ductus arteriosus (PDA).

**Animals:** Fifty cats with confirmed PDA.

**Methods:** Retrospective review of medical records from five referral veterinary hospitals for cats with PDA between 2000 and 2015. Cats were included if a PDA was visualized echocardiographically, during surgery, or on post-mortem examination.

**Results:** Median age at presentation was 6 months (range: 36 days–9.7 years; n = 50), and sex distribution was approximately equal (27 male, 23 female). Most cats did not have clinical signs (70.2%; 33/47) at the time of presentation. Murmurs
were classified as continuous (55%; 22/40) or systolic (45%; 18/40). Echocardiography confirmed left-to-right shunting in 33 cats (82.5%; 33/40) and right-to-left shunting in 7 (17.5%; 7/40). Concurrent cardiac anomalies were identified in 54.5% (18/33) and pulmonary hypertension in 45.7% (16/35). Closure was pursued in 68% (34/50), and complications associated with the procedure occurred in 14.7% (5/34) of cats, including one intraoperative mortality. Long-term follow up was available in 80% (40/50) of cats.

Conclusions: Cats with PDA often do not display clinical signs and may not have the characteristic physical examination findings typical of PDA in dogs. An increased prevalence of concurrent cardiac anomalies and pulmonary hypertension were found relative to previous reports. Thoracic radiographs and echocardiogram may provide the most comprehensive information for making a diagnosis and treatment recommendations. PDA closure was associated with a favorable long-term outcome in cats included in this study.

Introduction

Patent ductus arteriosus (PDA) is a congenital anomaly that occurs when the ductus arteriosus, a fetal vessel allowing blood to shunt away from the lungs and into systemic circulation before birth, fails to close after birth. Closure of the ductus arteriosus normally occurs in response to changes in pulmonary vascular resistance, oxygen tension, and prostaglandin inhibition [1]. The resulting defect causes shunting of blood from the aorta into the main pulmonary artery (left-to-right shunting) due to differences in pressure between the two vessels. As the lungs are chronically overcirculated and pulmonary vascular resistance increases, a reversal of the shunt direction can occur (right-to-left shunting) [2].

Patent ductus arteriosus is one of the most common congenital cardiovascular defects in dogs [3,4]. Patent ductus arteriosus is reported infrequently in cats but is still an important congenital cardiac disease in this species [5–9]. Regardless, fewer cats than dogs with PDAs are represented in the veterinary literature [7,10–16]. Previously published case series describe high morbidity and mortality rates associated with the diagnosis and closure of PDA in cats [10–12]. More recent published case series highlight the diagnostic approach, surgical and anesthetic techniques, and medical management of cats with PDA [7]. Still, the largest case series on cats with PDA includes just 28 cats from three institutions [7].

Animals, materials and methods

Medical records from five academic, referral veterinary hospitals were searched to identify cats diagnosed with PDA between January 1, 2000 and September 30, 2015. Cats were included in the study if PDA was confirmed via echocardiogram, direct visualization during surgery, or on post-mortem examination.

Signalment, clinical signs at initial presentation, and physical exam findings were recorded for each case as available in the medical records. Murmurs were considered quiet if graded I–III out of VI and loud if graded IV–VI out of VI [2].

Electrocardiogram findings were recorded and thoracic radiographs (when available) were reviewed and interpreted by a radiologist (CR). Vertebral heart size was determined on right (preferred) or left lateral radiographs as available [17].

Echocardiogram findings including direction of shunting, presence of concurrent congenital anomalies, pulmonary hypertension, mitral regurgitation, heart chamber enlargement, tricuspid
regurgitation velocity, left atrial to aortic root ratio, end diastolic left ventricular diameter, end systolic left ventricular diameter, and fractional shortening were recorded. Pulmonary hypertension was diagnosed in cats with tricuspid regurgitant jet velocity >3 m/s and secondary right sided heart changes (right ventricular remodeling including enlargement and hypertrophy, and septal flattening or abnormal septal movement). No cats underwent right heart catheterization for measurement of direct pulmonary pressures.

Treatment data were recorded when performed and included medical management, method of closure, and procedural complications.

Descriptive statistics (median, range, frequency, and mean ± standard deviation) were calculated for each parameter as appropriate. In the event that recorded data were missing for a particular parameter, the sample size was calculated using only those with available data for that parameter.

Results

Fifty cats were identified that met the study criteria. Of the 50 cases, 40 were confirmed with echocardiography. The remaining 10 cases were confirmed during surgical ligation (four cases) or on post-mortem examination (six cases).

Signalment

The median age at presentation was 6 months (mean: 15.2 ± 22.4 months, range: 36 days–9.8 years; n = 50). A majority (70%; 35/50) of cats were aged one year or younger when presented to the study institution for evaluation. Nine cats (18%; 9/50) were over 2 years of age at presentation. Represented breeds included domestic shorthair (72%; 36/50), domestic longhair (6%; 3/50), Persian (4%; 2/50), Siamese (4%; 2/50), and one each of domestic medium hair, Tonkinese, Oriental Shorthair, Exotic Shorthair, Ragdoll, Maine Coon, and mixed breed. The sex distribution was approximately equal with 27 males (12 intact, 15 castrated) and 23 females (14 intact, 9 spayed). The majority of cats did not have clinical signs of heart disease at presentation but were referred for evaluation of murmur (70.2%, 33/47). Fourteen cats presented with one or more clinical signs attributed to heart disease, the most common being increased respiratory effort (see Table 1).

Table 1  Clinical signs attributed to heart disease at presentation in cats with PDA.

<table>
<thead>
<tr>
<th>Clinical sign</th>
<th>Sample Size</th>
<th>Number observed (percent observed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>47</td>
<td>33 (70.2%)</td>
</tr>
<tr>
<td>Increased respiratory effort</td>
<td>47</td>
<td>10 (21.2%)</td>
</tr>
<tr>
<td>Exercise intolerance</td>
<td>47</td>
<td>4 (8.5%)</td>
</tr>
<tr>
<td>Respiratory distress</td>
<td>47</td>
<td>3 (6.4%)</td>
</tr>
<tr>
<td>Cough</td>
<td>47</td>
<td>2 (4.3%)</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>47</td>
<td>1 (2.1%)</td>
</tr>
</tbody>
</table>

Abbreviation: PDA, patent ductus arteriosus.

Physical examination

The median body weight of the cats was 2.9 kg (range: 0.2–6.02 kg; n = 44). A majority of the cats (75%; 15/20) were in good body condition, with a body condition score of 4 (40%; 8/20) or 5 (35%; 7/20) out of 9. Normal pulse quality was reported in 66.7% (24/36) of cats. Three cats (8.5%; 3/36) had decreased pulse quality and nine (25%; 9/36) had hyperdynamic pulses. Tachypnea (>38 breaths per minute) [18] was noted in 65.7% of cats (23/35). Three cats (10%; 3/30) were polycythemic with packed cell volumes of 48, 49, and 55% (reference range: 25–45% [19]). One cat with polycythemia had concurrent cyanosis. Cyanosis was noted in 4.9% of cats (2/41), with one cat (not polycythemic) described as having differential cyanosis. Additional physical exam findings are summarized in Table 2.

Murmur

Murmur characteristics were reported in 41/50 cats. A loud murmur (grade IV–VI) was reported in 92.7% (38/41) of cats at the time of presentation to the study institution. Murmur description beyond grade was not available for one cat. In 55% (22/40) of cats, a continuous murmur was reported, whereas in 45% (18/40), a systolic murmur was reported. Additional murmur characteristics are provided in Table 2.

Electrocardiogram and radiographic findings

Sinus rhythm was reported in 95% (19/20) of the cats. One cat had ventricular premature complexes. Twenty-nine cats underwent thoracic radiography. Radiographs were obtained and reviewed by a single radiologist for 20 cats (17 complete, orthogonal view sets and 3 lateral views only), whereas the radiograph report alone was available for nine cats. Only cats with radiographs available for review were
included in analysis for consistency. Interpretation of the thoracic radiographs revealed cardiomegaly in all of the cats. Specific areas of heart enlargement recorded from radiographic interpretation included the left atrium (35.0%; 7/20), left ventricle (35.0%; 7/20), right atrium (10.0%; 2/20), right ventricle (10.0%; 2/20), aortic arch, or descending aorta (75.0%; 15/20) and main pulmonary artery (60.0%; 12/20). Median vertebral heart size was 10.1 v (range: 8.3–12.8 v; n = 20). Pulmonary overcirculation was reported in 80.0% (16/20) of cats, including 13 cats with left-to-right shunting and three cats with right-to-left shunting PDA. An interstitial pattern was present in eight cats (40.0%; 8/20), and pleural effusion was present in four cats (20%; 4/20). Two cats with an interstitial pattern had increased respiratory effort on initial presentation; the remaining six cats had no respiratory abnormalities.

### Echocardiographic findings

Echocardiographic examination confirmed left-to-right shunting in 33 cats (82.5%; 33/40) and right-to-left shunting in seven cats (17.5%; 7/40). Concurrent cardiac anomalies were reported in 54.5% (18/33) of cats that received echocardiographic imaging. Pulmonary hypertension was reported in 45.7% (16/35) of cats. Information regarding pulmonary hypertension was not available in five cats that received echocardiographic imaging. Echocardiographic evidence of pulmonary hypertension included peak tricuspid regurgitation velocity > 3 m/s (seven cats), right ventricular remodeling (14 cats), and septal flattening or abnormal septal movement (six cats). Median peak tricuspid regurgitant jet velocity was 4.0 m/s (range: 2.1–5.5 m/s; n = 11), including cats with shunting in either direction and one cat that died before shunt direction could be determined. In two cats that did not receive complete echocardiographic evaluation before death, diagnosis of pulmonary hypertension was made on histologic and gross changes at post-mortem examination. Echocardiographic measurements are provided in Table 3.

### Cats with left-to-right shunting PDA

The median age at presentation for cats with confirmed left-to-right shunting PDA was 5.4

<table>
<thead>
<tr>
<th>Exam finding</th>
<th>Sample Size</th>
<th>Median (range) or number observed (percent observed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (bpm)</td>
<td>41</td>
<td>180 (92–272)</td>
</tr>
<tr>
<td>Respiratory rate (rpm)</td>
<td>35</td>
<td>48 (20–108)</td>
</tr>
<tr>
<td>Tachypnea (&gt;38 rpm) [18]</td>
<td>35</td>
<td>23 (65.7%)</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>42</td>
<td>5 (11.9%)</td>
</tr>
<tr>
<td>Cracksles</td>
<td>42</td>
<td>2 (4.8%)</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>41</td>
<td>2 (4.9%)</td>
</tr>
<tr>
<td>PCV</td>
<td>30</td>
<td>37 (29–55)</td>
</tr>
<tr>
<td>Polycythemia (PCV &gt; 45) [19]</td>
<td>30</td>
<td>3 (10%)</td>
</tr>
<tr>
<td>Ascites</td>
<td>41</td>
<td>2 (4.9%)</td>
</tr>
<tr>
<td>Murmur quiet (grades I–III)</td>
<td>41</td>
<td>3 (7.3%)</td>
</tr>
<tr>
<td>Murmur loud (grades IV–VI)</td>
<td>41</td>
<td>38 (92.7%)</td>
</tr>
<tr>
<td>Murmur character Left basilar</td>
<td>39</td>
<td>23 (59%)</td>
</tr>
<tr>
<td>Parasternal</td>
<td>39</td>
<td>11 (28.2%)</td>
</tr>
<tr>
<td>Right sternal</td>
<td>39</td>
<td>3 (7.7%)</td>
</tr>
<tr>
<td>Left apex</td>
<td>39</td>
<td>1 (2.6%)</td>
</tr>
<tr>
<td>Right and left base</td>
<td>39</td>
<td>1 (2.6%)</td>
</tr>
</tbody>
</table>

Abbreviation: PDA, patent ductus arteriosus.

### Table 3 Echocardiographic findings and measurements in cats with PDA.

<table>
<thead>
<tr>
<th>Echocardiographic finding</th>
<th>Sample Size</th>
<th>Median (range) or number observed (percent observed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak flow through PDA (m/s)</td>
<td>26</td>
<td>4.3 (1.9–5.5)</td>
</tr>
<tr>
<td>Tricuspid regurgitation velocity (m/s)</td>
<td>11</td>
<td>4.0 (2.1–5.5)</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>35</td>
<td>16 (45.7%)</td>
</tr>
<tr>
<td>Tricuspid regurgitation velocity &gt; 3 m/s</td>
<td>16</td>
<td>8 (50.0%)</td>
</tr>
<tr>
<td>Right ventricular remodeling</td>
<td>16</td>
<td>14 (87.5%)</td>
</tr>
<tr>
<td>Interventricular septal flattening</td>
<td>16</td>
<td>6 (37.5%)</td>
</tr>
<tr>
<td>Histologic changes on necropsy</td>
<td>16</td>
<td>2 (12.5%)</td>
</tr>
<tr>
<td>Left atrial enlargement</td>
<td>34</td>
<td>31 (91.2%)</td>
</tr>
<tr>
<td>Right atrial enlargement</td>
<td>22</td>
<td>4 (18.2%)</td>
</tr>
<tr>
<td>End diastolic left ventricular diameter (cm)</td>
<td>24</td>
<td>2.26 (0.22–3.1)</td>
</tr>
<tr>
<td>End systolic left ventricular diameter (cm)</td>
<td>24</td>
<td>1.33 (0.36–2.34)</td>
</tr>
<tr>
<td>Fractional shortening (%)</td>
<td>25</td>
<td>41.8 (20.81–70.77)</td>
</tr>
<tr>
<td>Left atrial to aortic root ratio</td>
<td>21</td>
<td>1.73 (1.42–3.2)</td>
</tr>
<tr>
<td>Mitral regurgitation</td>
<td>34</td>
<td>21 (61.8%)</td>
</tr>
</tbody>
</table>

Abbreviation: PDA, patent ductus arteriosus.
months (range: 2.5 months—9.8 years; n = 33). Seven of the 33 cats (21.2%; 7/33) with left-to-right shunting PDA presented with one or more clinical sign of heart disease including increased respiratory effort (three cats), cough (two cats), respiratory distress (two cats), and exercise intolerance (one cat). One cat with left-to-right shunting PDA was polycythemic (PCV 55%). The prevalence of concurrent cardiac anomalies within this group was 48.5% (16/33) including: ventricular septal defect (nine cats), mitral dysplasia (four cats), subaortic stenosis (four cats), pulmonic stenosis (two cats), and persistent foramen ovale (one cat). Ventricular septal defect size was available in five of the nine cats and measured a median of 2.3 mm (range: 1.0–10.0 mm; n = 5). Two additional ventricular septal defects were described as 'small', but specific measurement was not available. Ventricular septal defect flow velocity was available in seven of the nine cases, with a median of 4.0 m/s (range: 1.8–5.9 m/s; n = 7). Subaortic stenosis was diagnosed if a distinct ridge of tissue could be observed in the left ventricular outflow tract (three cats) or if flow velocity remained elevated following PDA closure (one cat).

Pulmonary hypertension was reported in 24.2% (8/33) of cats with left-to-right shunting PDA, with echocardiographic evidence including peak tricuspid regurgitation velocity >3 m/s (four cats), right ventricular remodeling (six cats), and septal flattening or abnormal septal movement (two cats). Median peak flow velocity through the PDA in cats with pulmonary hypertension was 2.6 m/s (range: 1.9–4.5 m/s; n = 8). Median peak flow velocity through the PDA in cats without pulmonary hypertension was 4.6 m/s (range: 2.1–5.5 m/s; n = 18). Median peak tricuspid regurgitant jet velocity in cats with left-to-right shunting PDA and pulmonary hypertension was 4.3 m/s (range: 3.9–4.5 m/s; n = 4). Median peak tricuspid regurgitant jet velocity in cats with left-to-right shunting PDA without pulmonary hypertension was 2.5 m/s (range: 2.1–2.95 m/s; n = 4).

Cats with right-to-left shunting PDA

Seven cats (17.5%; 7/40) were diagnosed with right-to-left shunting PDA on echocardiogram, confirmed with intravenous injection of agitated saline and visualization of bubbles in the abdominal aorta (agitated saline contrast study). Median age at presentation of these cats was 11.3 months (range: 5.6 months—1.5 years; n = 7). Five of these cats (71.4%; 5/7) presented with one or more clinical sign of heart disease including increased respiratory effort (four cats), exercise intolerance (two cats), dyspnea (one cat), and respiratory distress (one cat). Two cats with right-to-left shunting PDA were polycythemic (PCV 48% and 49%), one with concurrent cyanosis. Pulmonary hypertension was demonstrated in all of these cats, with a median peak tricuspid regurgitant jet velocity of 4.75 m/s (4.0 and 5.5 m/s; n = 2). Cats with right-to-left shunting PDA had loud murmurs (83.3%; 5/6) or no auscultable murmur (16.7%; 1/6). Murmur description was not recorded for one cat with right-to-left shunting PDA. The murmurs were characterized as systolic (100%; 4/4) and localized to the parasternal (40%; 2/5), right sternal (40%; 2/5), and left basilar regions (20%; 1/5).

One or more concurrent cardiac anomalies were identified in 28.6% (2/7) of these cats and included one cat with tricuspid dysplasia and one cat with mitral dysplasia, atrial septal defect, and persistent left cranial vena cava. Persistent left cranial vena cava was diagnosed based on echocardiographic findings and was not confirmed with angiography. None of the seven cats with right-to-left shunting PDA confirmed on echocardiography underwent PDA closure. Medical management was recorded for five of these cats and consisted of furosemide (40%; 2/5), pimobendan (20%; 1/5), beta-blocker (20%; 1/5), sildenafil (60%; 3/5), ACE inhibitor (60%; 3/5). One cat with right-to-left shunting PDA (20%; 1/5) was managed without any medications.

PDA closure

Closure of the PDA was attempted in 34/50 cats (68%). Thirty of these cats had a left-to-right shunting PDA confirmed on echocardiogram before surgery. The remaining four cats had PDA confirmed during surgery based on strong suspicion of PDA (continuous, loud murmur). While direction of shunting could not be definitively determined in these four cases, left-to-right shunting was presumed due to the presence of a continuous, loud murmur, and survival following acute closure. Pulmonary hypertension was diagnosed in 20.6% (7/34) and concurrent cardiac anomalies were present in 38.2% (13/34) of cats undergoing occlusion.

Medical management for 44.1% (15/34) of cats before attempted PDA closure included furosemide (32.4%; 11/34), ACE inhibitor (17.6%; 6/34), pimobendan (2.9%; 1/34), and dalteparin (2.9%; 1/34). Medical management was instituted
in cats with respiratory abnormalities (six cats), radiographic changes (four cats), and for unrecorded reasons (five cats). Following closure, 32.2% (10/31) of the cats received continued medical management consisting of an ACE inhibitor (19.4%; 6/31), furosemide (12.9%; 4/31), and antibiotic therapy (9.7%; 3/31).

Thirty-three cats underwent PDA ligation through a left lateral thoracotomy and one cat underwent occlusion using embolization coils. The cat that underwent occlusion via embolization coils was diagnosed with pulmonary hypertension preoperatively. Perioperative or intraoperative complications were reported in five cats undergoing surgical PDA closure (15.1%; 5/33), including one cat with pulmonary hypertension and two cats with concurrent cardiac anomalies. Two cats (6.1%) experienced non-fatal hemorrhage, one cat (3%) developed pulmonary edema, one cat (3%) developed hypoxemia, and one cat with pulmonary hypertension (3%) died during surgery following entry into the thorax but before complete dissection of the PDA. No hemorrhage was reported in the cat that died. In the two cats that developed non-fatal hemorrhage, one went on to become pyrexic and the other developed generalized megaesophagus, aspiration pneumonia, and increased respiratory effort. No complications occurred in the cat that underwent occlusion with embolization coils.

**PDA closure not pursued**

Sixteen cats (32%; 16/50) did not undergo PDA closure. Seven of these cats (43.8%; 7/16) were confirmed to have right-to-left shunting PDA on echocardiogram, as discussed above. Three cats (18.8%; 3/16) had left-to-right shunting PDA confirmed on echocardiogram but closure was not pursued. Two of these three cats had concurrent cardiac anomalies (ventricular septal defect, subaortic stenosis) documented on echocardiogram. Six cats (37.5%; 6/16) were diagnosed with PDA on post-mortem examination. Four of these cats were presented by an animal shelter for increased respiratory effort (two cats), femoral fracture (one cat), or unrecorded reasons (one cat). Two of the six cats diagnosed on post-mortem exam were client-owned animals. One was euthanized before echocardiogram for unrecorded reasons. The second cat died during the echocardiogram before injection of agitated saline to assess shunt direction, and the PDA with pulmonary hypertension was confirmed on post-mortem examination.

**Outcome of PDA closure**

Following PDA closure and before discharge from the hospital, murmur characteristics were recorded for 20 of the 34 cats (58.8%) that underwent closure. Twelve of the 20 cats (60.0%) had a persistent heart murmur following attempted PDA closure. Six of the 12 cats (50.0%) with persistent murmurs had one or more concurrent cardiac anomalies present that were not addressed at the time of closure. These anomalies included ventricular septal defect (four cats), subaortic stenosis (two cats), pulmonic stenosis (one cat), and mitral dysplasia (one cat). Of the 12 cats with persistent murmurs, eight had echocardiograms performed postoperatively and none of the eight had residual flow through the PDA. Four cats did not have results recorded from a post-operative echocardiogram.

**Survival**

At the time of writing, 50% (25/50) of the cats included in the study are known to be alive, 30% (15/50) are known to be deceased (including the seven cats that did not survive to discharge), and 20% (10/50) are of unknown status. Cats that did not survive to discharge (n = 7) were not included in survival analyses. Of the deceased cats, median age at death was 4.4 years (range: 6 months–11.4 years; n = 7). Age at death was not available for one of the eight deceased cats.

Forty-three cats survived to discharge, including 33 that underwent PDA closure and 10 that did not. One cat that underwent closure (3%; 1/33) and seven that did not (70%; 7/10) are now deceased. Of the seven cats with clinical signs of heart disease before PDA closure, six cats (85.7% 6/7) survived to discharge, and five cats (71.4%; 5/7) are alive at the time of writing. One cat (14.3%; 1/7) with clinical signs that survived to discharge is of unknown status. Median follow up duration on cats that underwent PDA closure was 7.4 years (range 4 months–13.5 years, n = 23). One cat that underwent PDA closure was diagnosed with hypertrophic cardiomyopathy 1.1 years after diagnosis of PDA.

Eight cats were reported to have an interstitial pattern on pre-operative radiographs; six (75%; 6/8) are alive, one (12.5%; 1/8) is deceased, and one (12.5%; 1/8) is of unknown status. Seven cats with pulmonary hypertension and left-to-right shunting underwent attempted PDA closure, one of which died intraoperatively, as discussed above. Of the remaining 6 cats, 50.0% (3/6) are alive and 50.0% (3/6) are of unknown status.
Of the seven cats with right-to-left shunting PDA, five are deceased and two are alive (currently 4 and 6.5 years old) at the time of writing. Median age at death of these cats was 1.7 years (range: 6 months – 9.4 years, n = 5). Three were euthanized; one due to heart failure and two for unrecorded reasons. Two cats did not have a known cause of death, although cardiac-related disease was presumed in one cat that died suddenly two weeks after diagnosis.

Three cats with left-to-right shunting PDA did not undergo closure, one of which is still alive (currently 12.2 years old) and two are now deceased (age at death 4.4 years and 11.4 years). The cat that remains alive has concurrent subaortic stenosis with no reported clinical signs of heart disease. Median length of follow up on these cats was 4.1 years (range: 1.6—8.4 years, n = 3).

Discussion

Most of the cats in the present study did not have clinical signs attributed to heart disease at initial diagnosis. This finding is similar to dogs with PDA, in which 73.8% have no clinical signs of heart disease [15]. The median age at presentation of cats in this study was 6 months, which is similar to dogs with PDA that present at a median age of 5.1 months [15]. Nine cats were over 2 years of age at presentation, suggesting that congenital heart disease in cats can go undetected into adulthood. This is in contrast to the recent case series of 28 cats with PDA, in which all of the cats were diagnosed before one year of age [7]. In the present study, an approximately equal number of male and female cats were diagnosed with PDA, similar to the recent report in 28 cats [7]. This is in contrast to dogs, where PDA is over represented in females (73.1%) [15]. In dogs with PDA, the majority (92.5%) of murmurs is continuous and left basilar and pulses are typically hyperdynamic [15]. In contrast, only 55% of cats in this study had continuous murmurs and only 25% had bounding pulses.

Pulmonary hypertension was documented with echocardiography in 45.7% (16/35) of cats in this study. This prevalence of pulmonary hypertension is higher than reported in a recent case series of 28 cats with PDA in which pulmonary hypertension was diagnosed in only 8% [7]. This difference could be related to concurrent disease or differences in genetic populations. Geography could have played a role, given that half of the cats with pulmonary hypertension from the current study were from Colorado (50%; 8/16), which is located at significantly higher elevation than the other institutions studied. Arterial hypoxemia that occurs when residing at high altitude has been reported to lead to pulmonary hypertension in a variety of veterinary species, including cats, although the altitudes studied were much higher than those experienced in Fort Collins, CO [20].

Another interesting finding was the prevalence of pulmonary hypertension in cats maintaining a left-to-right shunting PDA. Pulmonary arterial changes occur secondary to chronic overcirculation and result in pulmonary hypertension. When pulmonary vascular resistance exceeds systemic vascular resistance, reversal of shunting occurs [2]. The cats with pulmonary hypertension and a left-to-right shunting PDA may have had a lesser degree of pulmonary hypertension than those with right-to-left shunting PDA. None of the cats in the present study underwent right heart catheterization for direct measurement of pulmonary pressures and comparison of severity of pulmonary hypertension. In the recent case series of 28 cats with PDA, there was no report of reversal of flow through the ductus in any of the cats studied and only 8% had pulmonary hypertension [7].

Congenital cardiac defects were frequently diagnosed concurrently with PDA in cats in this study. This finding is not reflected in dogs, in which just 8.8% were reported to have concurrent congenital cardiac defects [15]. The prevalence of concurrent congenital defects in this study is higher than in both of the more recent case series, which reported concurrent cardiac anomalies in 26% [7] and 29% of the cats studied. The high prevalence of concurrent cardiac defects emphasizes the need for complete echocardiographic examination in cats with murmurs ausculted on physical exam and may impact treatment recommendations. Many cats in this series with concurrent congenital defects underwent PDA closure without complication. Although not evaluated in this study, the presence of concurrent cardiac defects has been reported to negatively impact survival time in dogs [15].

The most common method of PDA closure in cats was left lateral thoracotomy and ligation of the PDA. Most of the cats included in this study that underwent PDA closure had a good outcome, as reflected by the proportion that remains alive (at least 66.7%) with a median follow up length of 7.6 years. PDA closure in this study was associated with a single intraoperative mortality (2.9%), which is dramatically lower than previous reports [10,11]. An early case series of five cats with PDA reported peri/intraoperative mortality in 80% of
A different case report describes a kitten that died following PDA closure, presumably due to acute spike in pulmonary pressure [11]. Non-fatal hemorrhage was the most commonly reported complication in this case series (5.9%). The prevalence of complications in this study, and bleeding in particular, is also much lower than previously reported [10–14]. In cats that underwent PDA closure, clinical signs attributed to heart disease at initial presentation did not affect survival in this study; however, these data should be interpreted carefully given the small sample size (seven cats). In dogs that underwent PDA closure, the presence of clinical signs at presentation was reported to negatively affect the survival [15].

Seven cats were diagnosed with right-to-left shunting PDA, and medical management without PDA closure was pursued in all of these cats. One cat with right-to-left shunting PDA had multiple concurrent cardiac anomalies including an atrial septal defect, so it is possible that intracardiac shunting interfered with the agitated saline contrast study, although this cannot be confirmed retrospectively. Cats with right-to-left shunting PDA were more likely to have presented with clinical signs attributed to heart disease than cats with left-to-right shunting PDA (71.4% vs. 21.2%, respectively). Murmurs in these cats were described exclusively as systolic rather than continuous. In right-to-left shunting PDA, equalization of the pressure gradient between the main pulmonary artery and the ascending aorta reduces turbulence such that a murmur may not be appreciated. The presence of systolic murmurs in these cats may be related to tricuspid regurgitation (two cats), concurrent congenital defects (two cats), or suprasystemic pulmonary pressures (not evaluated in this study). In addition, it is possible that these cats were evaluated relatively early in the reversal process given that the diastolic component of the murmur is lost first during reversal, followed by loss of the systolic component [2]. Interestingly, three cats with right-to-left shunting were found to have evidence of pulmonary over-circulation on thoracic radiographs, which is unexpected in cats where pulmonary pressures meet or exceed systemic pressures. Possible causes of this unexpected finding include concurrent left-sided heart disease or variation in radiographic positioning resulting in the appearance of enlarged vasculature in the absence of increased flow. Although these data must be interpreted with caution due to the small sample size, cats with right-to-left shunting demonstrated a lower median age at death (1.7 years, n = 5) compared with cats with left-to-right shunting PDA that were treated medically without PDA closure (7.9 years, n = 2). It is important to note that four cats with right-to-left shunting PDA survived well into adulthood (range: 4–9.4 years, n = 4).

This study has limitations, many due to the retrospective nature. A major limitation was sparse or missing medical records, particularly for the six cases that were diagnosed on post-mortem examination. Diagnosis of pulmonary hypertension was based on echocardiographic surrogates for elevated pulmonary pressures rather than direct measurement of pulmonary pressures. In addition, morphologic description and size of the ductus in these 50 cats were not collected in this study. Ductus size and morphology are important considerations when planning PDA closure in the dog [21,22] and would have been an interesting comparison point. Further description of PDA morphology and size in cats may provide important information when planning device occlusion rather than surgical ligation.

Conclusions

In conclusion, many of the characteristic findings used to diagnose a PDA in dogs, such as continuous murmur, bounding pulses, and female gender are not consistent in the majority of cats with PDA. Instead, male and female cats are equally likely to have PDA, the murmur may or may not be continuous and pulses may be normal. In addition, concurrent congenital cardiac disease is not uncommon, and pulmonary hypertension may be more likely to occur in cats than in dogs with PDA. Therefore, when a murmur is detected in a cat, thoracic radiographs and an echocardiogram may provide the most comprehensive information for making a diagnosis and treatment recommendations. PDA closure in cats with left-to-right shunting was associated with a good long-term outcome, even in cats with concurrent cardiac defects or pulmonary hypertension.

Conflicts of Interest

The authors do have any conflicts of interest to disclose.

References


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