NOTE *Surgery*

Patent Ductus Arteriosus Ligation in Two Young Cats with Pulmonary Hypertension

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ABSTRACT. We report two feline cases of patent ductus arteriosus (PDA) with pulmonary hypertension (PH). The subjects were both intact domestic shorthair cats, a 4-month-old, 2.5 kg male (case 1) and an 8-month-old, 2.12 kg female (case 2). At the first presentation, left-sided congestive heart failure was diagnosed in case 1 and severe aortic stenosis (AS) in case 2. Following surgical ligation of the ductus arteriosus (DA), furosemide therapy was no longer required in case 1, and the severe AS improved to mild status in case 2 perhaps because of reduced volume overload. In case 2, severe hypoxemia was revealed after surgery; however, this normalized within 96 days after surgery.

KEY WORDS: feline, patent ductus arteriosus, pulmonary hypertension.

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Patent ductus arteriosus (PDA) is less common in cats than in dogs [4, 16, 17]. As it is in dogs, left-sided congestive heart failure and/or pulmonary hypertension (PH) will develop unless the ductus arteriosus (DA) is closed [2, 4]. DA closure has been achieved in cats with surgical ligation or coil embolization [16, 17]. To the best of the authors' knowledge, this is the first report noting hypoxemia, perhaps due to hyperkinetic PH following PDA [11] and the course of hypoxemia after DA ligation in cats.

Case 1, a male domestic shorthair cat, was presented at the age of 4 months, because of respiratory effort after excitement and exercise intolerance. Body weight was 2.5 kg. At the hospital, heart rate, respiratory rate, and body temperature were 180 beats/min, 72 breaths/min, and 37.5°C, respectively. A systolic murmur was auscultated with its maximal intensity over the left parasternal region (grade 4/6). An electrocardiogram was not performed due to his severe clinical status. Global heart enlargement and focal pulmonary edema (interstitial/alveolar pattern) were revealed on thoracic radiography (Fig. 1). In addition, the pulmonary arteries of the cranial and caudal lobes were enlarged and tortuous (Fig. 1). Echocardiography showed a left ventricular diastolic diameter above the reference range (left ventricular internal dimension in diastole [LVIDd], 31.9 mm; reference range, 11.0–19.0 mm [16]), and left atrial enlargement was revealed with 2-dimensional echocardiographic assessment (left atrial/aortic root proportion [LA/Ao], 2.67; reference range, <1.5 [1]). Color-flow Doppler showed continuous flow from the descending aorta into the main pulmonary artery (Fig. 2; maximal flow rate, 2.69 m/sec, which indicates an aortic-pulmonary [Ao-PA] systolic pressure gradient of 28.9 mmHg). Pulmonary edema was immediately treated with furosemide (1 mg/kg intramuscular injection [IM]), and he was managed in an oxygen cage. On the same day, surgical correction of the PDA was performed. General anesthesia was induced using fentanyl (5 μg/kg intravenous injection [IV]), midazolam (0.2 mg/kg IV), and propofol (3 mg/kg IV) and maintained using fentanyl (5–10 μg/kg/hr, continuous rate injection [CRI]), dobutamine (2 μg/kg/min, CRI) and isoflurane (1.5–2.0% in 100% oxygen, inhalation). Thoracotomy was first performed via an incision in the left fourth intercostal space as previously described [10]; however, the DA could not be visualized well via this incision, because it was displaced caudally because of severe cardiomegaly, and a new incision in the left fifth intercostal space was implemented. The tissues surrounding the DA were carefully separated using a right-angle forceps. Two 1–0 silk ligatures were passed around the DA, and the aortic and pulmonary artery ends of the DA were ligated [3, 10]. The thoracotomy incision was closed in a routine manner. The cat recovered uneventfully from general anesthesia and was managed in an oxygen cage for 6 days after surgery, because of slightly increased respiratory effort. At 6 days after surgery, his respiratory status had improved and he was maintained in room air, although his blood oxygen level was close to lower limit (partial pressure of oxygen in arterial blood [PaO_2], 81.4 mmHg; partial pressure of carbon dioxide in arterial blood [$PaCO_2$], 30.6 mmHg; alveolar–arterial oxygen difference [A-aDO_2], 30.3 mmHg; reference ranges [9]: PaO_2, >80 mmHg; $PaCO_2$, 36–40 mmHg; A-aDO_2, 5–15 mmHg). Postoperative care consisted of antibiotic therapy with cefazolin (22 mg/kg, IV or per os [PO], q 12 hr for 7 days) and positive inotropic therapy for heart failure with pimobendan (0.25 mg/kg, PO, q 12 hr). The cat was discharged 10 days after surgery. At 23 days after surgery, he had no clinical symptoms. A systolic heart murmur was auscultated with maximal intensity over the right parasternal region (grade 2/6). On echocardiography, the left ventricle and left atrium were markedly

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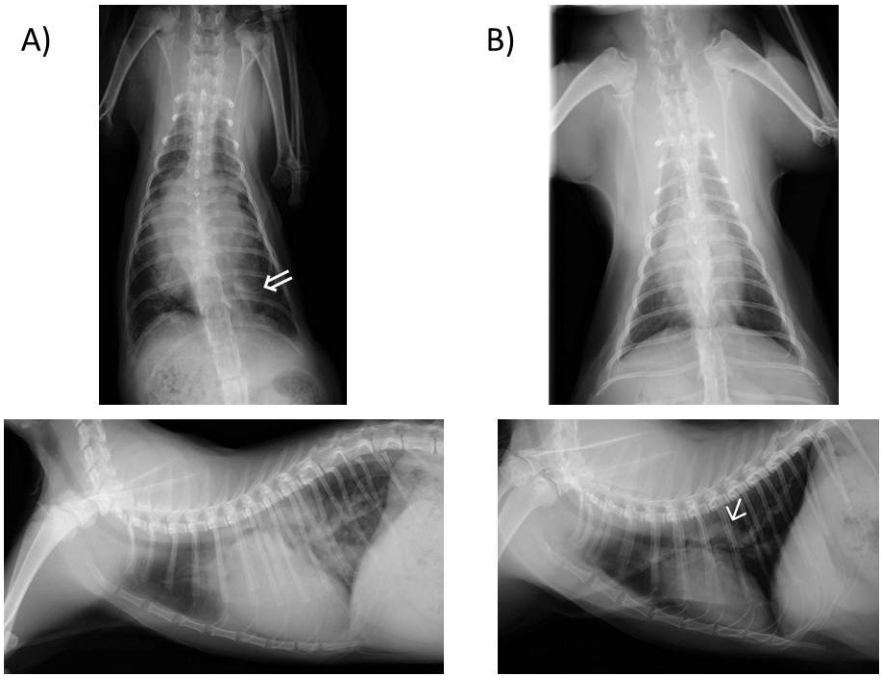

Fig. 1. Radiographic images in case 1. (A) Dorsoventral (top) and right lateral (bottom) radiographs at the first presentation. There are enlarged pulmonary arteries and increased lung opacity, which are suggestive of pulmonary overcirculation and edema. In the top image, there is an alveolar pattern in the left caudal lobe (open arrow). In the bottom image, there is a patchy alveolar pattern in the caudal lobe and enlarged and tortuous pulmonary arteries. (B) Dorsoventral (top) and right lateral (bottom) radiographs at 205 days after surgery. Heart size is markedly reduced, although enlarged and tortuous pulmonary arteries (closed arrow), suggestive of pulmonary hypertension, persist.

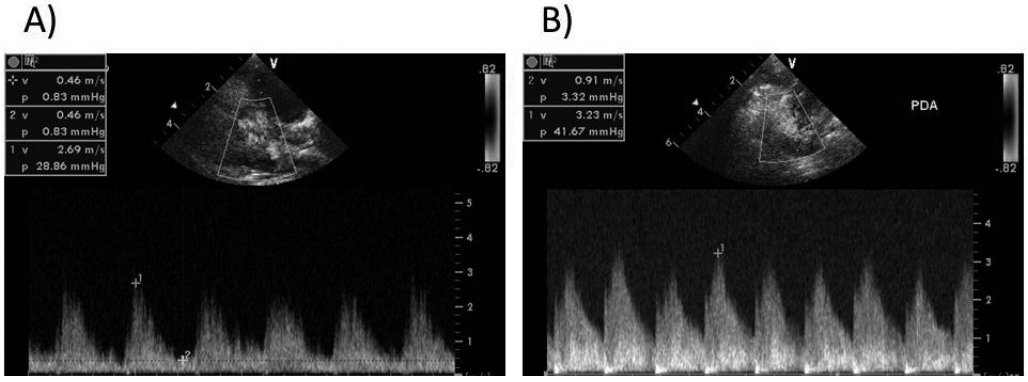

Fig. 2. Doppler spectrum images of 2 patent ductus arteriosus cats at the first presentation. In both cases, case 1 (A) and case 2 (B), the maximal velocity through the ductus arteriosus was low (2.69 and 3.23 m/sec, respectively), suggesting pulmonary hypertension.

reduced in size (LVIDd, 18.0 mm; LA/Ao, 1.54). On color-flow Doppler and spectral Doppler echocardiography, there was no retrograde flow from the descending aorta into the main pulmonary artery, although there was mild mitral valve regurgitation and PH (tricuspid valve regurgitation velocity [TRV], 3.60 m/sec, which indicates a right ventricular-right atrial [RV-RA] systolic pressure gradient of 51.8 mmHg). The PH persisted at 205 days after surgery. TRV was 3.53 m/sec, which indicates an RV-RA systolic pressure gradient of 49.8 mmHg, and enlarged and tortuous pulmonary arteries were revealed on radiography (Fig. 1); however, blood gases had markedly improved (PaO_2, 111.6 mmHg; $PaCO_2$, 31.6 mmHg; A-aDO_2, 0.0 mmHg). On follow-up echocardiography at 233 days after surgery, the DA was completely

occluded.

Case 2, a female domestic shorthair cat, was presented to a primary veterinary hospital for sterilization at 8 months of age. However, a cardiac murmur was detected, and she was diagnosed with PDA on the basis of various examinations. Thoracotomy was performed through the fifth intercostal space, but the DA could not be visualized well at the primary veterinary hospital. Therefore, the cat was presented to our veterinary hospital 19 days after surgery. Body weight was 2.12 kg. She had no clinical symptoms, and heart rate, respiratory rate and body temperature were 180 beats/min, 80 breaths/min, and 38.8°C, respectively. A continuous murmur was auscultated with its maximal intensity over the left heart base (grade 5/6). An electrocardiogram revealed a sinus rhythm with a heart rate of 192 beats/min. The R wave amplitude was increased (6.4 mV; reference range, <0.9 mV [7]), which was suggestive of left ventricular enlargement. Global heart enlargement without pulmonary edema was revealed on thoracic radiography. However, the pulmonary arteries of the caudal lobe were enlarged and slightly tortuous. Echocardiography showed that the LVIDd (25.9 mm) and LA/Ao (2.14) were above the reference ranges, and concentric ventricular hypertrophy (diastolic interventricular septal thickness [IVSd], 6.3 mm; reference range, <6.0 mm [6]) was also revealed. Color-flow and spectral Doppler showed continuous flow from the descending aorta into the main pulmonary artery (Fig. 2; maximal flow rate, 3.23 m/sec, which indicates an Ao-PA systolic pressure gradient of 41.7 mmHg). In addition to the PDA, valvular aortic stenosis (AS) was found. Color flow and spectral Doppler echocardiography from the left parasternal transducer location revealed turbulent flow from the left ventricle to the aorta with a velocity of 5.95 m/sec, which indicates a left ventricular-aortic (LV-Ao) systolic pressure gradient of 141.6 mmHg (continuous-wave Doppler expected range, 0.8–1.6 m/sec [16]). On the same day, a thoracotomy was performed through the left fourth intercostal space as previously described [10]. General anesthesia was induced by using fentanyl (10 μg/kg IV), midazolam (0.2 mg/kg IV), and propofol (2 mg/kg IV), and then maintained using fentanyl (5–10 μg/kg/hr, CRI) and isoflurane (1.5–2.0% in 100% oxygen, inhalation). As in case 1, two 1–0 silk ligatures were passed around the DA, and the aortic and pulmonary artery ends of the DA were ligated. The thoracotomy incision was closed in a routine manner. The cat recovered uneventfully from general anesthesia; however, she displayed respiratory effort after surgery, and blood gas tests revealed severe hypoxemia (PaO_2, 56.6 mmHg; $PaCO_2$, 33.6 mmHg; A-aDO_2, 51.2 mmHg). Therefore, she was managed in an oxygen cage for 7 days. The hypoxemia had improved slightly at 7 days after surgery (PaO_2, 73.5 mmHg; $PaCO_2$, 34.2 mmHg; A-aDO_2, 34.2 mmHg); she had no clinical symptoms and was discharged. Postoperative care consisted of antibiotic therapy with cefazolin (22 mg/kg, IV or PO, q 12 hr for 7 days) and negative inotropic therapy for AS with atenolol (3.125 mg/head, PO, q 24 hr). At 96 days after surgery, she had no clinical symptoms, although there was a systolic heart murmur with its maximal intensity over the left heart base (grade 4/6). Blood gases had markedly improved (PaO_2, 101.8 mmHg; $PaCO_2$, 28.6 mmHg; A-aDO_2, 12.1 mmHg). The enlarged and slightly tortuous pulmonary arteries were still visible on radiography. On echocardiography, the diameter of the left ventricle had markedly decreased (LVIDd, 15.9 mm), but the IVSd was 6.3 mm and the diastolic left ventricular posterior wall thickness was 6.3 mm, which were suggestive of pressure overload due to AS. On color-flow Doppler, there was no retrograde flow from the descending aorta into the main pulmonary artery, but AS and aortic regurgitation persisted. However, aortic flow velocity (3.56 m/sec, which indicates an LV-Ao systolic pressure gradient of 50.7 mmHg) was markedly reduced, perhaps because the volume overload caused by the PDA had resolved.

The prevalence of PDA in cats is significantly less than in dogs [10], although PDA is reported as the most common congenital heart defect in cats [2]. Therefore, there are few case reports, perhaps because of the low number of patients. PDA occurs as an isolated cardiac defect or coexists with other congenital anomalies (e.g., subaortic stenosis and ventricular septal defects) [5, 16]. In case 2 with concurrent AS, the aortic flow velocity markedly reduced after surgery as described in a similar PDA cat with subaortic stenosis [16]. PDA should be closed as soon as possible because, if no treatment is performed, fatal heart failure develops. However, if left-sided congestive heart failure is present, it should be treated aggressively before surgery. In addition, a cat with right-to-left shunting PDA has been reported [4]. This condition is significantly rare in cats; however, this is another reason that left-to-right shunting PDA should be closed as soon as possible. In animals with PDA, contraindications to occlusion of the DA are right-to-left shunting, bidirectional shunting or concurrent cardiac conditions that rely on the PDA for survival (e.g., tetralogy of Fallot) [3]. In this report, both cases were diagnosed as PH with echocardiography. Although PH should be definitively diagnosed with a catheterization study [11], this could not be performed because both cases were in a critical state. On the other hand, the peak velocity of the PDA is reduced in humans and dogs with PDA and PH [3, 13–15], and PH was diagnosed in dogs with echocardiography when the DA velocity was <5 m/sec, particularly <3.5 m/sec [13]. Hypoxemia, apparently caused by hyperkinetic PH due to PDA [11], was still present immediately after surgery, but was completely resolved with DA occlusion. However, enlarged and tortuous pulmonary arteries visible on radiography remained after DA occlusion, unlike findings with feline heartworm treatment [12]. DA closure has been performed with surgical treatment [10, 17] or coil embolization [16] in cats. We performed surgical ligation in both cases, because the femoral artery, which we routinely use for coil embolization in dogs, appeared too small for coil embolization. However, the transvenous technique might allow coil embolization to be performed in cats with PDA, because the femoral vein is larger than the femoral artery [16]. There are several limitations in this report. First, blood gas tests were not performed before surgery because both cases were in a critical state, although the hypoxemia would have been present before surgery. Second, PH should

be diagnosed with a catheterization study, which is the gold standard for definitive diagnosis of PH [11]. Finally, pathological investigation of lung tissue should be performed to determine the cause of hypoxemia [8], although hyperkinetic PH due to PDA appeared to be the cause of hypoxemia [11].

In conclusion, surgical ligation of the DA was performed in two cats with evidence of PH. Both cats became clinically normal after surgery, and the aortic flow velocity was markedly reduced in the cat with coexisting AS, perhaps because of reduced volume overload. The radiographic changes, i.e., enlarged and tortuous pulmonary arteries, appear to persist after surgery, although lung function normalizes, in cats.

REFERENCES

1. Abbott, J. A. and MacLean, H. N. 2006. Two-dimensional echocardiographic assessment of the feline left atrium. *J. Vet. Intern. Med.* **20**: 111–119. [Medline] [CrossRef]
2. Allen, D. G. 1982. Patent ductus arteriosus in a cat. *Can. Vet. J.* **23**: 22–23. [Medline]
3. Broaddus, K. and Tillson, M. 2010. Patent ductus arteriosus in dogs. *Compend. Contin. Educ. Vet.* **32**: E1–E14. [Medline]
4. Connolly, D. J., Lamb, C. R. and Boswood, A. 2003. Right-to-left shunting patent ductus arteriosus with pulmonary hypertension in a cat. *J. Small Anim. Pract.* **44**: 184–188. [Medline] [CrossRef]
5. Dear, M. G. 1970. An unusual combination of congenital cardiac anomalies in a cat. *J. Small Anim. Pract.* **11**: 37–43. [Medline] [CrossRef]
6. Fox, P. R., Liu, S. K. and Maron, B. J. 1995. Echocardiographic assessment of spontaneously occurring feline hypertrophic cardiomyopathy. An animal model of human disease. *Circulation* **92**: 2645–2651. [Medline] [CrossRef]
7. Goodwin, J. K. 2001. Electrocariography. pp. 43–70. *In:* Manual of Canine and Feline Cardiology, 3rd ed. (Tilley, L. P. and Goodwin, J.K. eds.), Saunders, Philadelphia.
8. Heath, D. and Edwards, J. E. 1958. The pathology of hypertensive pulmonary vascular disease; a description of six grades of structural changes in the pulmonary arteries with special reference to congenital cardiac septal defects. *Circulation* **18**: 533–547. [Medline] [CrossRef]
9. Irizarry, R. and Reiss, A. 2009. Arterial and venous blood gases. *Compend. Contin. Educ. Vet.* **31**: E1–E11. [Medline]
10. Jones, C. L. and Buchanan, J. W. 1981. Patent ductus arteriosus: anatomy and surgery in a cat. *J. Am. Vet. Med. Assoc.* **179**: 364–369. [Medline]
11. Kellihan, H. B. and Stepien, R. L. 2010. Pulmonary hypertension in dogs: diagnosis and therapy. *Vet. Clin. North Am. Small Anim. Pract.* **40**: 623–641. [Medline] [CrossRef]
12. Nelson, C. T., McCall, J. W., Rubin, S. B., Buzhardt, L. F., Dorion, D. W., Graham, W., Longhofer, S. L., Guerrero, J., Robertson-Plouch, C. and Paul, A. 2005. Guidelines for the diagnosis, prevention and management of heartworm (Dirofilaria immitis) infection in cats. *Vet. Parasitol.* **133**: 267–275. [Medline] [CrossRef]
13. Oyama, M. A. and Sisson, D. D. 2001. Evaluation of canine congenital heart disease using an echocardiographic algorithm. *J. Am. Anim. Hosp. Assoc.* **37**: 519–535. [Medline]
14. Sahn, D. J. and Allen, H. D. 1978. Real-time cross-sectional echocardiographic imaging and measurement of the patent ductus arteriosus in infants and children. *Circulation* **58**: 343–354. [Medline] [CrossRef]
15. Schneider, D. J. and Moore, J. W. 2006. Patent ductus arteriosus. *Circulation* **114**: 1873–1882. [Medline] [CrossRef]
16. Schneider, M. and Hildebrandt, N. 2003. Transvenous embolization of the patent ductus arteriosus with detachable coils in 2 cats. *J. Vet. Intern. Med.* **17**: 349–353. [Medline] [CrossRef]
17. Summerfield, N. J. and Holt, D. E. 2005. Patent ductus arteriosus ligation and pulmonary artery banding in a kitten. *J. Am. Anim. Hosp. Assoc.* **41**: 133–136. [Medline]