

Peripheral pulmonary artery stenosis in three cats

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ABSTRACT. Case 1 involved a 4-month-old intact male Somali cat in which peripheral pulmonary artery stenosis (PPS) was recognized after a cardiac murmur remained following patent ductus arteriosus ligation. Case 2, which involved a 1-year-old neutered male Norwegian Forest cat, and Case 3, which involved a 6-month-old intact female American Curl cat, were referred, because of cardiac murmurs. Grades III to IV/VI systolic heart murmurs were auscultated at the left heart base in all 3 cats. All cases showed bilateral pulmonary artery stenosis, although there were no associated clinical signs. In Cases 1 and 2, the pressure gradient through the stenosis decreased after treatment with atenolol.

KEY WORDS: atenolol, congenital heart disease, feline, patent ductus arteriosus, peripheral pulmonary artery stenosis

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Peripheral pulmonary artery stenosis (PPS) is a rare congenital heart disease in which one or more narrowed segments are formed in the main or peripheral pulmonary arteries [15, 16, 27]. Although pulmonary stenosis has been previously reported in cats [14], PPS is extremely rare [16, 26, 27]. We are aware of only 9 reported cases in cats [16, 26, 27], of which one was successfully treated by balloon angioplasty [16]; however, to our knowledge, medical treatment of cats with PPS has not previously been reported. In this report, we describe the clinical courses of 3 cats with PPS and our observation of a reduced pressure gradient (PG) through the stenosis in two of the cats medicated with atenolol.

Case 1: A 4-month-old intact male Somali cat, weighing 2.04 kg, was referred to our hospital for further evaluation of a heart murmur. He had no clinical signs and had not been medicated. A continuous grade V/VI murmur was auscultated, with maximal intensity heard over the left heart base. A bounding pulse was palpated in both femoral arteries. Global heart enlargement and enlarged pulmonary vessels, indicating pulmonary overcirculation, were revealed on the thoracic radiograph.

Echocardiography showed an increased left ventricular diastolic diameter and a left ventricular end-diastolic diameter (LVDd) of 28.0 mm (reference range, 11.0–19.0 mm [23]). Two-dimensional echocardiography revealed left atrium en-

largement with a left atrial/aortic root ratio (LA/Ao) of 2.14 (reference range, <1.5 [1]). Color-flow Doppler revealed a left-to-right patent ductus arteriosus (PDA) (Fig. 1A). Color flow and spectral Doppler echocardiography also revealed a turbulent flow from the main pulmonary artery (MPA) to the right pulmonary artery (RPA), with a velocity of 4.40 m/sec and a pressure gradient (PG) through the stenosis of 77.4 mmHg. However, this could not be differentiated from the retrograde flow of the PDA (Fig. 1A). Enalapril (0.5 mg/kg per os (PO) every (q) 24 hr) was prescribed, because of left ventricular volume overload.

At 6 days after first presentation, we attempted to close the ductus arteriosus using coil embolization; however, it was too wide for the coil device (Type III [18]), so we elected to perform a surgical correction. At this time, peripheral pulmonary artery stenosis was suspected based on angiography, although the image and pressure data of the MPA were lacking. After completing the angiography study, a thoracotomy was performed via the left fourth intercostal space under general anesthesia, and the ductus arteriosus was routinely ligated with silk sutures. A systolic grade III/IV heart murmur remained after surgery, with maximal intensity heard over the left heart base.

At 28 days after first presentation, the LVDd had decreased to 16.7 mm on M-mode echocardiography. However, a turbulent flow with a left pulmonary artery (LPA) velocity of 4.64 m/sec (estimated PG, 86.1 mmHg) and an RPA velocity of 4.03 m/sec (estimated PG, 65.0 mmHg) were revealed on color flow and spectral Doppler echocardiography (Fig. 1B). In addition, a slight flattening of the intraventricular septum (IVS) following a pressure-loaded right ventricle (RV) was subjectively observed in end-systole on two-dimensional echocardiography (Fig. 2A) [9].

Case 1 was diagnosed as bilateral PPS. Because of the flattened IVS (Fig. 2A), a high-pressure gradient through the

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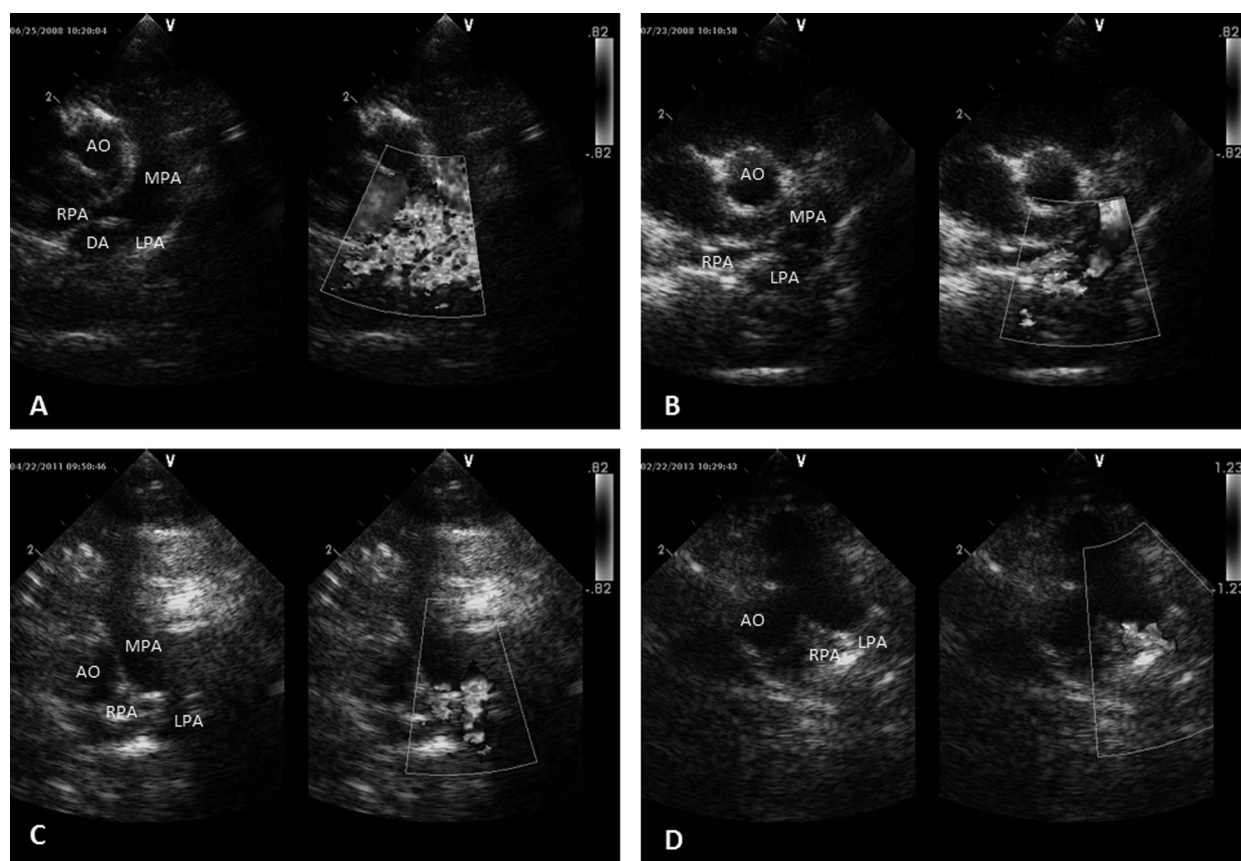


Fig. 1. Two-dimensional and color-flow Doppler echocardiographic images in 3 cats with PPS. (A) Right parasternal short-axis view at the level of the aorta in Case 1 at first presentation. PPS was suspected, but not identified due to retrograde flow from the PDA. (B) Right parasternal short-axis view at the level of the aorta depicting bilateral pulmonary artery stenosis after PDA ligation in Case 1. (C) Left cranial parasternal short-axis view depicting bilateral pulmonary artery stenosis at first presentation in Case 2. (D) Left cranial parasternal short-axis view depicting bilateral pulmonary artery stenosis at first presentation in Case 3. PPS: peripheral pulmonary artery stenosis; PDA: patent ductus arteriosus; AO: aorta; MPA: main pulmonary artery; RPA: right pulmonary artery; LPA: left pulmonary artery; DA: ductus arteriosus.

stenosis was suspected [9], and the cat was treated with atenolol (1 mg/kg PO q 24 hr). At 86 days after the first presentation, the owner complained that the cat exhibited exercise intolerance after atenolol treatment, although at this time, the velocity of the RPA had decreased to 3.35 m/sec (estimated PG, 44.9 mmHg) on spectral Doppler echocardiography (LPA velocity was not available). The dose of atenolol was decreased to 0.5 mg/kg q 24 hr, because of the exercise intolerance complaint. The owner reported resolution of the exercise intolerance problem after the atenolol dose was reduced, although the relationship to atenolol was unclear. Enalapril was withdrawn, because of reverse-remodeling of the left ventricle.

At 854 days after first presentation, the velocity of the LPA was 2.42 m/sec (estimated PG, 23.4 mmHg) and the flattening of the IVS disappeared on echocardiography, although the owner had stopped the administration of atenolol 10 months prior (Fig. 2A). Thereafter, annual follow-up was continued without atenolol treatment. At the time of manuscript preparation (2,225 days after first presentation), the cat's clinical status was good, and there were no clinical

signs associated with PPS.

Case 2: A 1-year-old neutered male Norwegian Forest cat, weighing 4.52 kg, was referred to our hospital for further evaluation of a heart murmur. He had no clinical signs and had not been medicated. A grade IV/VI systolic heart murmur was auscultated with maximal intensity heard over the left heart base. An electrocardiogram revealed a sinus rhythm with a heart rate of 174 beats/min. The QRS complex, which was negative in lead I and positive in both leads aVR and aVL, indicated right axis deviation (mean electrical axis, -150° ; reference range, 0° to 160°). Thoracic radiography showed no abnormal findings.

Color flow and spectral Doppler echocardiography revealed turbulent flow from the MPA to both the RPA (3.66 m/sec; estimated PG, 53.6 mmHg) and LPA (the velocity was not available). The maximum right atrial diameter and left atrial diameter ratio (RAD/LAD) was 1.02 (reference range <1.0 [26]). However, the RAD/LAD was 1.46 at 35 days after first presentation. In addition, flattening of the IVS was observed in end-systole on the right parasternal short-axis view at the level of the chordae tendineae (Fig. 2B).

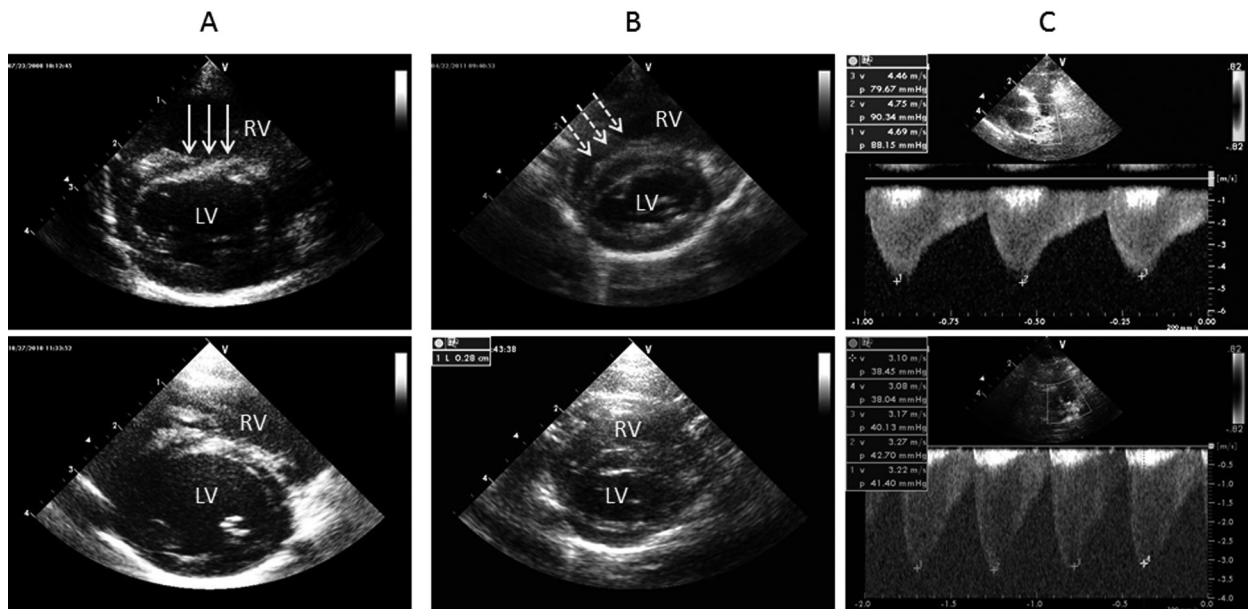


Fig. 2. Echocardiographic images before and after atenolol treatment in Case 1 (A) and Case 2 (B, C). In Case 1, flattening of the IVS was observed before atenolol treatment on two-dimensional echocardiography (A top, arrows, at 28 days after first presentation). The flattening of the IVS was resolved at 854 days after first presentation (A bottom), although, the atenolol treatment had been discontinued by the owner for 10 months. In Case 2, IVS flattening was observed at 35 days after first presentation (B top, dotted arrow). The velocity of the LPA decreased from 4.75 m/sec (C top, estimated PG, 90.3 mmHg; at 35 days after first presentation) to 3.27 m/sec (C bottom, estimated PG, 42.8 mmHg; at 70 days after first presentation) according to atenolol treatment, though the flattening of the IVS remained at 70 days after first presentation (B bottom). RV: right ventricle; LV: left ventricle.

Color flow and spectral Doppler echocardiography revealed a turbulent flow from the MPA to the LPA, with a velocity of 4.75 m/sec (estimated PG, 90.3 mmHg; Figs. 1C and 2C), although it could not be measured at first presentation.

Case 2 was diagnosed with bilateral PPS. Because the IVS was flattened, a high-pressure gradient through the stenosis was suspected, and the cat was treated with atenolol (6.25 mg/head PO q 12 hr). The velocity of the LPA decreased to 3.27 m/sec (estimated PG, 42.8 mmHg; Fig. 2C) at 70 days after first presentation, although, flattening of the IVS remained. The RAD/LAD was 1.18. A serum biochemistry panel revealed elevated creatinine (2.2 mg/dl; reference range, 0.8–1.8 mg/dl) at 467 days after first presentation. The atenolol dose was decreased to 3.125 mg/head q 12 hr, because the blood atenolol level may have increased due to a decreased glomerular filtration rate [21].

At 672 days after first presentation, the velocities of both RPA and LPA were 3.07 m/sec (estimated PG, 37.7 mmHg) on spectral Doppler echocardiography. Serum creatinine level had decreased to 1.2 mg/dl, although the relationship with the dosage of atenolol was unclear. Semi-annual follow-up was continued. At the time of manuscript preparation (1,229 days after first presentation), the cat's clinical status was good, and there were no clinical signs associated with PPS.

Case 3: A 6-month-old intact female American Curl cat, weighing 2.06 kg, was referred to our hospital for further evaluation of a heart murmur, which was detected before sterilization at a referring veterinary hospital. She had no

clinical signs and had not been medicated. A grade IV/VI systolic heart murmur was auscultated with its maximal intensity heard over the left heart base. Radiography showed right atrium enlargement. An electrocardiogram revealed a sinus rhythm with a heart rate of 241 beats/min. The mean electrical axis was within the normal range (106°), although the QRS complex was negative in lead I.

Color and spectral Doppler echocardiography revealed turbulent flows from the MPA to both the RPA (3.40 m/sec; estimated PG, 46.2 mmHg) and LPA (2.90 m/sec; estimated PG, 33.6 mmHg) without right ventricular hypertrophy (Fig. 1D). Case 3 was diagnosed with bilateral PPS; only annual follow-up was planned. At the time of manuscript preparation (522 days after first presentation), the cat has remained in good clinical condition without clinical signs associated with PPS.

PPS is a rare congenital cardiovascular heart disease in veterinary as well as human medicine; it is found in only 2–3% of humans with congenital heart disease [4, 12, 15, 16, 26, 27]. There are few reports of congenital PPS in the veterinary literature. We are aware of 10 reported cases: 1 in a dog with bilateral pulmonary artery stenosis [15], 1 in a cat with right pulmonary branch stenosis who underwent successful balloon angioplasty [16], 7 in cats with isolated stenosis of the main pulmonary artery [26] and 1 in a cat concurrently affected with PDA and juxtaductal coarctation of both pulmonary arteries [27].

In Case 1, the following etiologies are possible causes of

PPS: 1) the LPA may have been compressed by a tubular aneurysm secondary to an enlarged ductus arteriosus, since acquired pulmonary stenosis has reportedly followed aortic aneurysms in humans [8, 10] and the ductus arteriosus was anatomically close to the LPA; 2) the LPA was inadvertently occluded at the time of PDA ligation [22] or compressed by a suture granuloma secondary to the silk used to ligate the PDA; 3) tissue from the ductus arteriosus migrated to the pulmonary artery, and stenosis occurred when the migrated tissue contracted after birth [27]; or 4) the proximal sixth left and right aortic arches, which connect to the ipsilateral pulmonary artery, were abnormally atrophied as an embryologic developmental abnormality [3, 16]. Inadvertent ligation of the LPA was ruled out, because hypovascularity was not recognized in the left lung field on thoracic radiography after the surgery [22]. Compression of the LPA due to a tubular aneurysm secondary to a large PDA appeared unlikely, because similar cases of large tubular aneurysms secondary to PDA concurrent with pulmonary hypertension did not have PPS before or after surgery [2]. A granuloma due to the silk materials used for PDA ligation is undeniable; however, aberrant involution of the proximal sixth aortic arch and/or ectopic ductus arteriosus tissue are possible causes of PPS in all 3 cats in the present report, because both pulmonary arteries were affected, as described in previous reports [15, 16, 27].

The symptomatic cat with PPS had been reported to have had recurrent episodes of dyspnea, which was purportedly due to right pulmonary branch stenosis and subtotal left pulmonary atresia [27]. In humans with PPS, exertional dyspnea caused by pulmonary hypoperfusion is one of the most common clinical symptoms [12]. However, clinical signs are uncommon in the cats with PPS [26]. Our 3 cases, even Case 1 with concurrent PDA, had no clinical signs secondary to heart disease.

In humans with PPS, transcatheter or surgical treatments are conducted with the following indications: 1) the right ventricular pressure is over two-thirds the systemic pressure; 2) pulmonary blood flow distribution is inadequate after other cardiac abnormalities are corrected; 3) the pulmonary artery is distorted in symptomatic patients with or without cardiopulmonary shunts; 4) pulmonary blood flow has marked asymmetry; or 5) segmental pulmonary artery hypertension is present, i.e., the mean distal pressure is over 25 mmHg [4]. Transcatheter treatments are the first line therapy in humans with PPS, because the results of surgical treatment have been unsatisfactory [4, 5]. Indeed, it has been previously reported that balloon angioplasty was helpful even in a cat with PPS [16]; however, to the authors' knowledge, there are few reports of medical treatment for PPS in the veterinary medicine.

Atenolol is a selective beta-1 receptor antagonist, which has anti-ischemic, negative chronotropic, negative inotropic and anti-arrhythmogenic properties, and is believed to reduce myocardial oxygen consumption [7, 28]. It is also empirically used for pulmonary artery stenosis in veterinary medicine [9, 16]. However, the clinical evidence for its efficacy in this situation is lacking. It has been reported that

the PG significantly decreased in humans with dynamic left ventricular outflow tract obstruction (DLVOTO) during treatment with atenolol [20]. In addition, regression of left ventricular hypertrophy was observed in young dogs with DLVOTO treated with atenolol [13], although DLVOTO resolved without atenolol as the dogs grew, i.e., there were anatomical changes within the heart as it grew into its adult form [13]. In the present report, PG through the stenosis decreased in 2 cats that were medicated with atenolol, although the PG remained low after atenolol was withdrawn in Case 1. In Case 1, hypercontraction of the left ventricle secondary to volume overload due to PDA might have affected the contraction of the right ventricle and the increasing PG [2]. In addition, maturation might have affected the degree of stenosis in Case 1, as described in humans with PPS [4]. Therefore, treatment with atenolol might not be required in all PPS cases. The indications for atenolol treatment in cats with PPS should be established.

A standard dosage of atenolol cannot be based on our report; however, a lower dosage of atenolol (0.5–0.69 mg/kg, q 12–24 hr) appeared to be sufficient to reduce the PG through the stenosis. However, atenolol has not prolonged survival time in obstructive heart disease cases, e.g. subaortic stenosis in dogs [6] and DLVOTO in cats with hypertrophic obstructive cardiomyopathy [24]. In addition, decreasing the PG secondary to surgical correction [19] or balloon valvuloplasty [11, 17] might not prolong survival time in severe subaortic stenosis. Therefore, atenolol might not prolong survival time in cats with PPS, although it might decrease the PG through the stenosis, and reducing the PG followed by balloon angioplasty has been shown to alleviate clinical symptoms in a cat with PPS [16] and was effective in cats with infundibular pulmonary stenosis without heart failure [25]. However, the prognosis for cats with PPS might be benign with or without medical treatments, as previously reported [26] and supported by the current cases in which all 3 cats survived without clinical signs over 1.5 to 7 years after diagnosis.

In conclusion, PPS can occur concurrently with PDA. The clinical signs with pulmonary hypoperfusion, e.g. exercise intolerance and exertional dyspnea, are not common in cats. The degrees of stenosis with PPS might improve as the cats mature. The prognosis appears to be good in cats with PPS. Although the PG through the stenosis might decrease after the atenolol treatment, further studies are required to determine the appropriate dosage of atenolol and elucidate the survival advantage of its use in cats with PPS.

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