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Two cases of new-born puppies with transient pulmonary edema

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Abstract

Acute pulmonary edema in puppies generally occurs due to congenital left-right shunts such as patent ductus arteriosus or large ventricular septal defects. Herein, we presented two cases of puppies with no apparent congenital cardiovascular disease. Case 1: A 12-day-old male Labrador Retriever, weighing 1.15 kg, was unable to suckle sufficiently from its dam and exhibited laboured breathing. Pulmonary edema was identified in all lung lobes by radiography, furthermore, echocardiography revealed significant enlargement of the left side of the heart. Pulmonary edema secondary to volume overload was suspected and furosemide was administered. The respiratory status was improved on the following day. Pimobendan was administered orally in addition to furosemide and both were withdrawn 6 weeks later when the heart size was normalized. Case 2: A 15-day-old female Standard Poodle, weighing 0.68 kg, was less active than other littermates and exhibited laboured breathing. Radiography revealed pulmonary edema in the right posterior lobe, dilatation of the caudal vena cava and ascites. Echocardiography revealed significant enlargement of the left atrium and ventricle perhaps owing to decreased left ventricular contractility. Furosemide and pimobendan were administered. One week later, appetite was improved and supraventricular tachycardia of 375 bpm was observed. Therefore, tachycardia-induced dilated cardiomyopathy was suspected which returned to sinus rhythm with diltiazem treatment, however, it was recurred. Upon sotalol monotherapy, a normal heart size was observed seven months later. In conclusion, we encountered two new-born puppies with transient pulmonary edema that were temporarily treated with pimobendan and furosemide.

Introduction

In puppies, congestive heart failure (CHF), particularly pulmonary edema, generally occurs due to congenital left-right shunts such as patent ductus arteriosus or large ventricular septal defects. Myocardial insufficiency due to dilated cardiomyopathy or myocarditis may cause pulmonary edema in puppies with a poor prognosis.[1-3] Herein, we presented two cases of new-born puppies with transient pulmonary edema that were treated with temporary administration of pimobendan and furosemide.

Case Description

Case 1. An intact 12-day-old male Labrador Retriever, weighing 1.15 kg, was unable to sufficiently suckle from its dam due to coughing for 3 days before the visit. At the hospital, no heart murmur was detected, however, the puppy was in apparent respiratory distress, hence, oxygen was immediately administered. A radiographic examination within 10 min after oxygen administration to confirm the lung status revealed a diffuse alveolar pattern in all lung lobes (Fig. 1A). It was difficult to accurately evaluate the heart and great vessels due to the diffuse alveolar pattern. Focused point-of-care echocardiography (Xario 200GV; Canon Medical Supply, Tokyo, Japan) revealed a markedly enlarged left atrium. The left atrial-to-aortic ratio (LA/Ao) was 2.21 (Fig. 2A), left ventricular internal diameter in diastole (LVIDd) 1.58 cm, and left ventricular internal diameter in systole (LVIDs) 1.01 cm. During echocardiography, the mean recorded heart rate was 200 bpm. Fractional shortening (FS%) was 36.10%.

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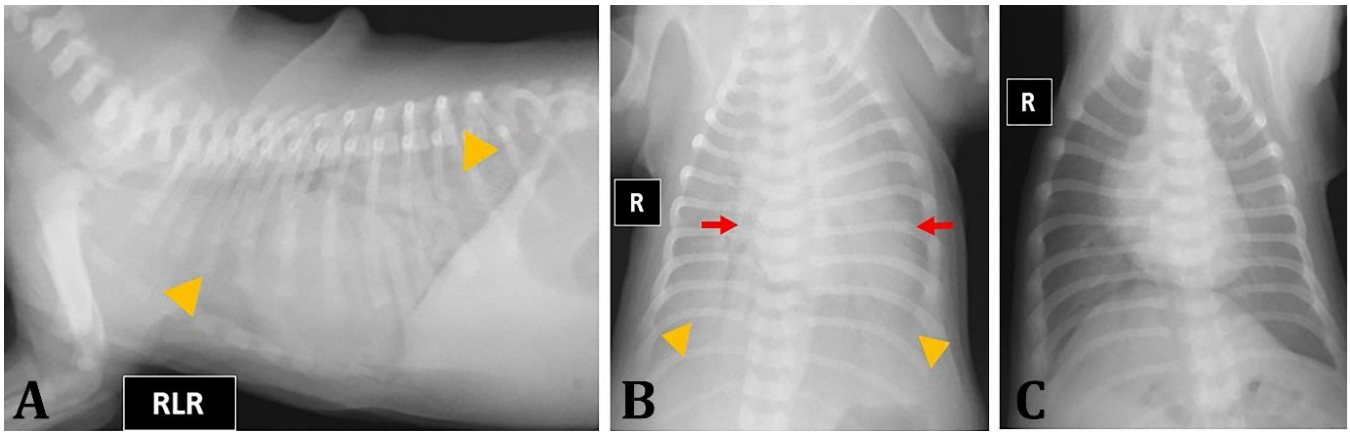

Fig. 1. Radiographic findings in case 1. **A)** Lateral, and **B)** dorsoventral views at the first presentation showing alveolar patterns in all lobes (yellow arrowheads). The diffuse alveolar pattern obscured the cardiac silhouette, however, it appeared large compared to the thorax (red arrows). **C)** The alveolar pattern was improved on the following day after management with furosemide. The cardiothoracic ratio was 59.10% and the heart size was normal.

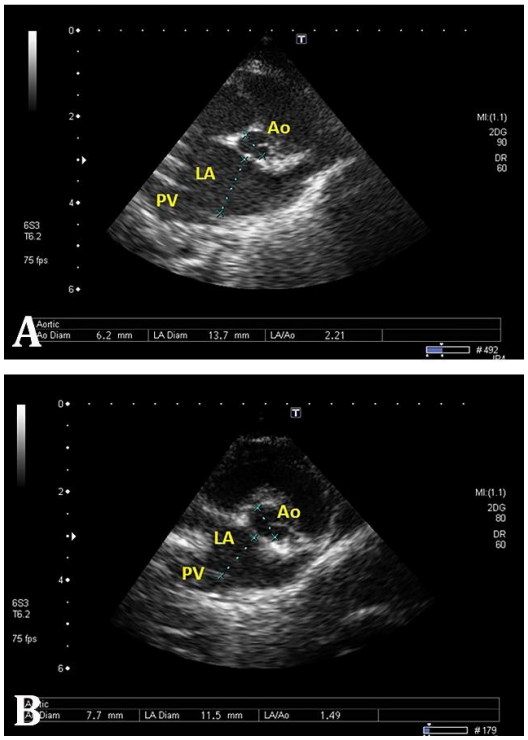

Fig. 2. Echocardiographic findings in case 1. **A)** Marked left atrial enlargement at the initial visit, **B)** which normalized 2 weeks after management with furosemide and pimobendan. LA: Left atrium, Ao: Aorta, and PV: Pulmonary vein.

Since regurgitation was found in the center of the mitral and tricuspid valves, we considered valve regurgitation to be secondary to volume overload. The puppy was placed in a chamber with 40.00% oxygen. Furosemide (Sanofi K.K., Tokyo, Japan) was administered subcutaneously at 0.50 mg kg^{-1} followed by the same dose 1 hr later. The puppy was force-fed on a commercial canine formula every 2 to 3 hr. The respiratory status was improved and radiographic examination revealed improvement in the radiolucency of the lung with heart size reduction (cardiothoracic ratio [CTR]: 59.10%) on the following day (Fig. 1B). The puppy was orally administered pimobendan (0.27 mg kg^{-1} twice daily; Boehringer Ingelheim, Ingelheim am Rhein, Germany) and furosemide (1.09 mg kg^{-1} once daily) and was discharged on the following day. Two weeks later, the respiration was restored and the appetite was improved with a 2.20 kg body weight. Radiography revealed a reduction in the heart size (Vertebral heart scale: 9.00 v; CTR: 57.80%) and lung normalization. On echocardiography, LA/Ao was 1.49 (Fig. 2B), LVIDd 1.86 cm, LVIDs 1.36 cm, and FS% 26.90%. Since the heart size seemed normal (Fig. 1C), furosemide was withdrawn and pimobendan was administered at a dose of 0.28 mg kg^{-1} twice daily. Six weeks later, the dog showed no clinical symptoms and pimobendan was discontinued. After eight weeks, no heart murmur was detected. The body weight was 6.15 kg. On echocardiography, LA/Ao was 1.23, LVIDd 2.63 cm, LVIDs 1.79 cm, and FS% 31.90%. A telephone follows up 22 months after the initial visit confirmed that the puppy survived without any symptoms.

Case 2. An intact 15-day-old female Standard Poodle, weighing 0.68 kg developed rapid breathing and appeared to be smaller in size and less active than its 10 littermates. The puppy was unable to suckle sufficiently to maintain its nutritional requirements and was force-fed on commercial dog milk by the owner. At the hospital, a heart murmur could not be detected or excluded owing to a rapid heart rate. Radiography revealed an alveolar pattern mainly in the right posterior lobe region and caudal vena cava dilation (Fig. 3A). Echocardiography (ViVid E9; GE Healthcare, Tokyo, Japan) revealed biatrial and biventricular enlargement with valve regurgitation (Fig. 4). The LA/Ao was 2.26, LVIDd was 1.18 cm and LVIDs was 1.01 cm. The FS% was 14.40%, suggesting systolic dysfunction. Mild mitral and tricuspid regurgitation were observed, respiratory variation in the caudal vena cava was absent and ascites was present (Fig. 5).

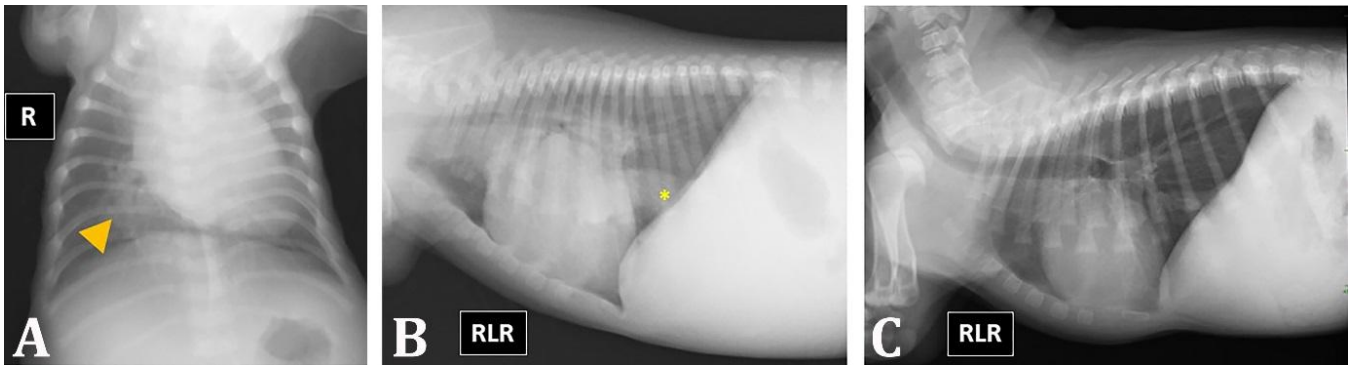

Fig. 3. Radiographic findings in case 2. **A)** A mixed interstitial-alveolar pattern in the right lung field on the dorsoventral view (yellow arrowhead) was found. **B)** Marked cardiomegaly and enlargement of the caudal vena cava (*) in lateral view were observed. **C)** The cardiac size and caudal vena cava were improved after two weeks of management with pimobendan, furosemide, and diltiazem.

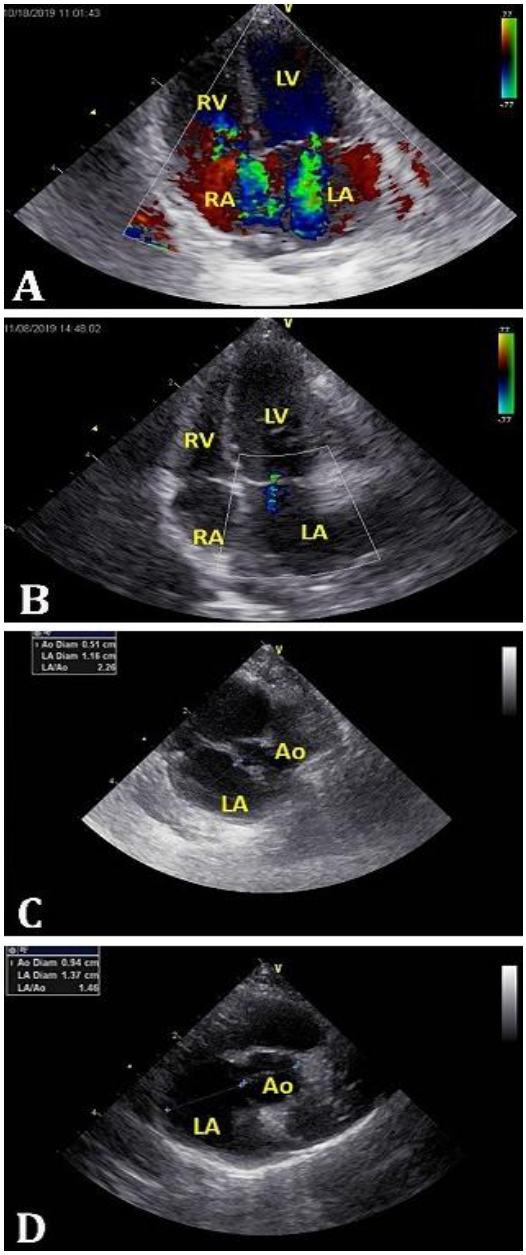

Fig. 4. Echocardiographic findings in Case 2. **A)** The biatrial and biventricular enlargement with valve regurgitation improved to **B)** Only mild mitral regurgitation 2 weeks after management with furosemide, pimobendan, and diltiazem. **C)** The left atrium was also markedly dilated; **D)** It was normalized after the same treatment. LA: Left atrium, LV: Left ventricle, RA: Right atrium, RV: Right ventricle, and Ao: Aorta.

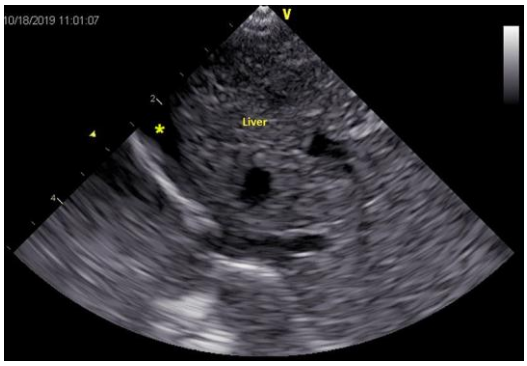

Fig. 5. Ultrasonographic findings in Case 2. Respiratory variation in the caudal vena cava was absent with an enlarged hepatic vein. The ascites (*) was present.

Based on these findings, we diagnosed left- and right-sided heart failure due to dilated cardiomyopathy. Pimobendan was orally administered twice a day at a dose of 0.23 mg kg^{-1} at home. Further, the owner subcutaneously administered furosemide at a dose of 0.50 mg kg^{-1} twice daily. After one week, the activity was improved, however, the shallow rapid breathing was still present during sleep. At that time, the body weight was 0.92 kg. Upon arrival at the hospital, auscultation revealed a heart rate of > 300 bpm. An electrocardiogram (ECG, Cardisuny D700; Fukuda M-E Kogyo, Tokyo, Japan) revealed tachycardia at 375 bpm, thus, supraventricular tachycardia was suspected (Figs. 6A and 6B). Proceeding with the ECG examination was difficult due to physical movement; thus, we monitored the tachycardia by echocardiography during slow intravenous administration of 0.10 mg kg^{-1} diltiazem (Mitsubishi Tanabe Pharma, Osaka, Japan). The tachycardia suddenly was resolved with a return to sinus rhythm (Fig. 6C).

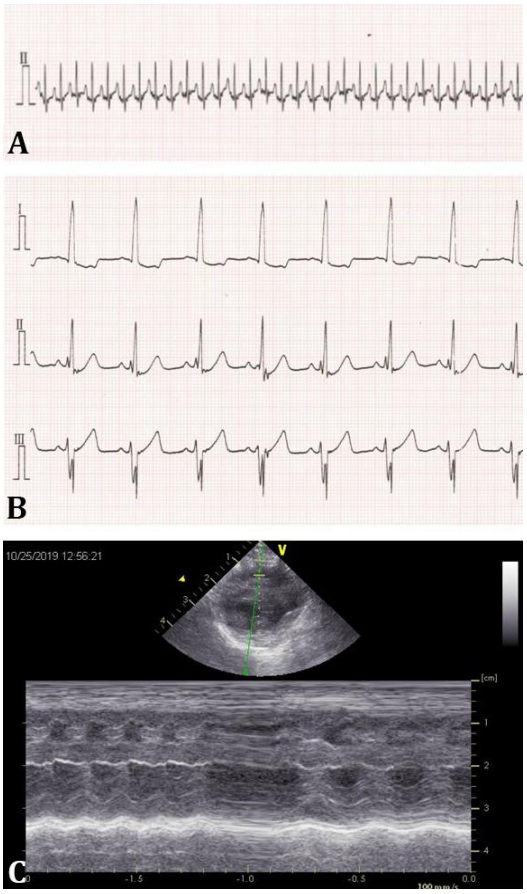

Fig. 6. A) Electrocardiographic and echocardiographic findings in case 2. Supraventricular tachycardia of 375 bpm was detected on electrocardiogram (1.00 mV cm^{-1}, 25.00 mm sec^{-1}). **B)** At sinus rhythm, electrocardiographic examination (1.00 mV cm^{-1}, 50.00 mm sec^{-1}) revealed splinted QRS complexes with R' morphology. **C)** Supraventricular tachycardia was present during echocardiography 8 days after the first presentation which suddenly was resolved with intravenous diltiazem.

The supraventricular tachycardia was recurred and diltiazem was administered four times at the aforementioned dosage. Based on this response, tachycardia-induced cardiomyopathy was diagnosed and 1.10 mg kg^{-1} diltiazem (Mitsubishi Tanabe Pharma) was orally administered three times a day. However, despite the resolution of the biventricular heart failure (Fig. 3B) and the normalization of the LA/Ao (seen as mild mitral regurgitation, Fig. 5B) in-hospital electrocardiography revealed supraventricular tachycardia two weeks later, therefore, the anti-arrhythmic therapy was changed to sotalol (1.00 mg kg^{-1}, PO; Aspen, Tokyo, Japan) twice daily. After five weeks, there was no obvious supraventricular tachycardia and only a splintered QRS complex with R' morphology was noted (Figs. 6A and 6B). The patient's appetite and activity were markedly improved following sotalol administration. The body weight was 3.48 kg.

The LA/Ao, LVIDd and LVIDs were 1.36, 1.99 cm and 1.54 cm, respectively, and FS% was improved to 22.60% (Fig. 3C). Therefore, pimobendan and furosemide were withdrawn. The puppy was 2 years old, apparently healthy, with normal cardiac function and receiving sotalol monotherapy.

Discussion

Acute pulmonary edema due to causes other than congenital heart disease has been reported in young dogs as shown below. A juvenile form of dilated cardiomyo-pathy has been described in Portuguese Water Dogs and Doberman Pinschers,[2,3] with cardiac death due to pulmonary edema occurring at 10 - 19 days of age in rapid cases or at 7 - 21 weeks of age. In addition, parvovirus-induced myocarditis reportedly resulted in the sudden death of 4 - 6 week-old puppies.[1] In the present cases, cardiac enlargement and pulmonary edema were transient and resolved with temporary treatment.

In case 1, cardiac contractility was normal suggesting transient volume overload. In human neonates and children, volume overload secondary to acute kidney injury is commonly associated with morbidity and mortality.[4] Since we did not perform blood tests in the case, the possibility of reversible acute kidney injury could not be ruled out. Similarly, volume overload due to anaemia could not be excluded.[5] Excessive water intake is one of the causes of volume overload, however, it was ruled out because the puppy could not suckle sufficiently and was not force-fed by the owner. Tachycardia also causes myocardial in-sufficiency and heart failure,[6] however, 200 bpm is within the normal range for new-born puppies.[7] Conversely, case 2 had tachycardia of 375 bpm indicating tachycardia-induced dilated cardiomyopathy. Also, after controlling the supraventricular tachycardia with diltiazem or sotalol, the myocardial insufficiency and CHF was resolved. Tachycardia-induced dilated cardiomyo-pathy due to pre-excitation syndromes such as Wolff-Parkinson-White syndrome is common in dogs and left ventricular dysfunction has been reported to improve following treatment for tachycardia.[6] In case 2, the PR interval was not shortened even during supra-ventricular tachycardia and there was no delta wave excluding pre-excitation syndrome. Supra-ventricular arrhythmias increase in proportion to left atrial diameter[8] and in this case it may have occurred secondary to cardiac enlargement.

The LVIDd normalized by body weight (LVIDdN) was 1.51 in case 1 and 1.32 in case 2.[9] The LVIDdN of Spanish Mastiffs puppies was reportedly 1.19[10] indicating that the left ventricle was enlarged in both studied cases.

The off-label use of pimobendan administered to 12- and 15-day-old puppies weighing less than 2.00 kg without apparent adverse effects has not been reported, however, case 1 perhaps should have been treated with diuretics alone.

There were several limitations to the present cases. In case 1, we could not rule out volume overload due to anaemia and acute kidney injury because we did not perform blood tests at the time of heart failure.[4,5] Blood tests were necessary to differentiate infectious or aspiration pneumonia from pulmonary edema, especially in case 2, where the patient was force-fed. In case 2, we could not confirm whether supraventricular tachycardia was adequately controlled by sotalol because we did not perform Holter ECG. Finally, the diagnosis of CHF was made based on imaging findings and response to treatment with diuretics, however, physical examination findings such as neck-vein distension and an extra heart sound which are diagnostic criteria were lacking.

In conclusion, we encountered transient pulmonary edema in new-born puppies one of which was due to unexplained volume overload and the other due to tachycardia-induced dilated cardiomyopathy. In new-born puppies, euthanasia should not be considered as the first-line option because severe pulmonary edema might be managed with appropriate treatment.

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Conflicts of interest

The authors declare no conflict of interest.

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