What Is Your Diagnosis?

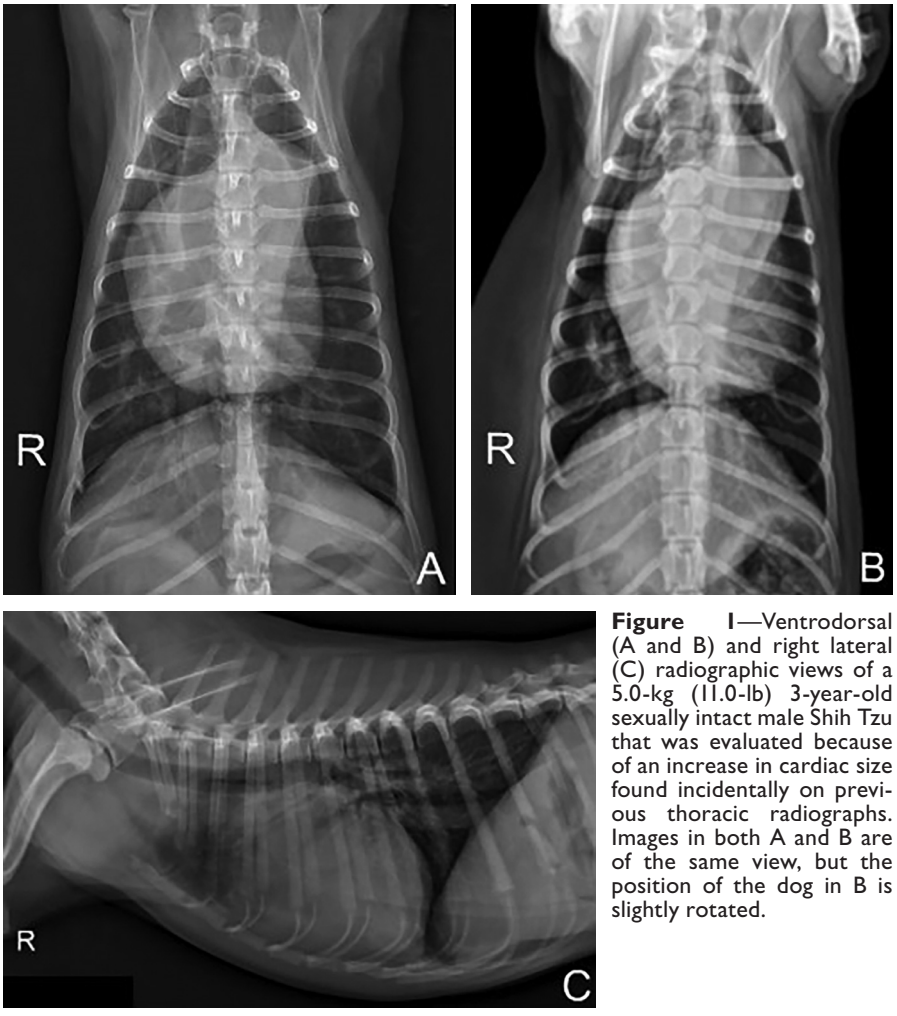

Figure 1—Ventrodorsal (A and B) and right lateral (C) radiographic views of a 5.0-kg (11.0-lb) 3-year-old sexually intact male Shih Tzu that was evaluated because of an increase in cardiac size found incidentally on previous thoracic radiographs. Images in both A and B are of the same view, but the position of the dog in B is slightly rotated.

History

A 5.0-kg (11.0-lb) 3-year-old sexually intact male Shih Tzu was referred for investigation of an enlarged heart that had been observed on routine thoracic radiographs by the referring veterinarian. When the patient was a puppy, medical records from the referring veterinarian indicated that a loud left-sided heart murmur was auscultated (murmur timing was not documented). This heart murmur decreased in intensity and was no longer present by 6 months of age. The patient had a recent history of gastrointestinal upset, but had been otherwise apparently healthy.

On physical examination, the patient was bright, alert, and responsive, with a heart rate of 144 beats/min and femoral pulses that were synchronous with heartbeats and mildly reduced in strength. The patient had a body temperature of 38°C (101°F), pink oral mucous membranes, and a capillary refill time of < 2 seconds. The patient was mildly tachypneic (50 breaths/min) but had normal respiratory effort. Cardiac auscultation revealed a grade 4/6 right midheart systolic murmur and splitting of the second heart sound. Additionally, a grade 2/6 diastolic murmur was auscultated at the right heart base. A prominent cardiac impulse was palpated on the right hemithorax, equal in intensity to the cardiac impulse palpated on the left hemithorax. Hematologic evaluation revealed an Hct of 52%. The remainder of the physical examination findings were unremarkable. Thoracic radiography was performed for further evaluation of the heart murmur and previously identified cardiomegaly **(Figure 1)**.

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page →

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Diagnostic Imaging Findings and Interpretation

Severe right-sided cardiomegaly is observed, with rounding of the right ventricular margin. A lack of tapering of the right side of the heart on the ventrodorsal projection and an increase in sternal contact of the heart on the lateral projection are appreciable. On the ventrodorsal projection, a prominent soft tissue bulge associated with the cardiac silhouette that represents an enlarged main pulmonary artery is evident. On the ventrodorsal projection that is slightly rotated, a distinct bulge can be observed at the proximal portion of the descending aorta, and this is consistent with a ductal bump. The intrathoracic trachea is dorsally deviated cranial to the carina of the trachea, and this change is a mass effect caused by the enlarged main pulmonary artery. Mild peripheral pulmonary arterial enlargement is evident, but otherwise all other pulmonary vessels are normal in size. Pulmonary parenchyma and all other cardiac structures are unremarkable in all views **(Figure 2)**.

Echocardiography was performed to investigate the cause of the cardiomegaly. Severe concentric hypertrophy of the right ventricle and mild to moderate right atrial enlargement were observed. The main pulmonary artery and right pulmonary artery were severely enlarged, and a tubular, tapering structure was observed at the bifurcation of the main pulmonary artery **(Figure 3)**. The shape and location of this structure were consistent with a patent ductus arteriosus (PDA). Color Doppler echocardiography was used to identify blood flow through the PDA, which was primarily moving from the pulmonary artery into the PDA and was consistent with a right-to-left shunting PDA. Agitated saline (0.9% NaCl) solution was injected IV to pro-

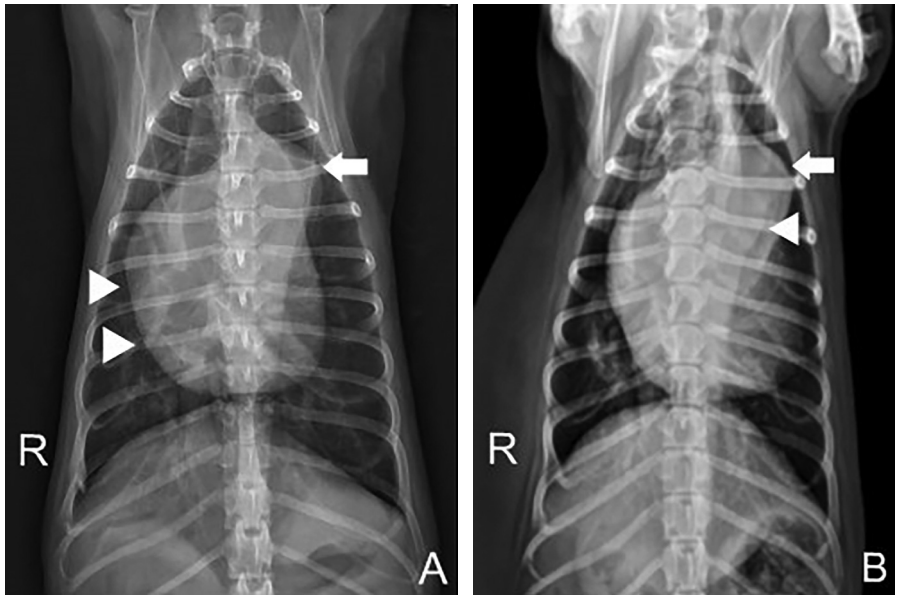

Figure 2—Same radiographic images as in Figure 1. On the first ventrodorsal view (A), a soft tissue opacity bulges at the 1 to 2 o'clock position on the cardiac silhouette, which corresponds with an enlarged main pulmonary artery (arrow). The right side of the heart is increased in size (arrowheads) resulting in cardiomegaly, present in all views. In the slightly rotated second ventrodorsal view (B), the enlarged main pulmonary artery is again observed (arrow) but a distinct bulge is observed at the descending aorta (arrowhead) corresponding to a ductal bump. On the lateral projection, the trachea is deviated cranial to the level of the carina of the trachea (arrowhead) corresponding to a mass effect caused by the main pulmonary artery enlargement.

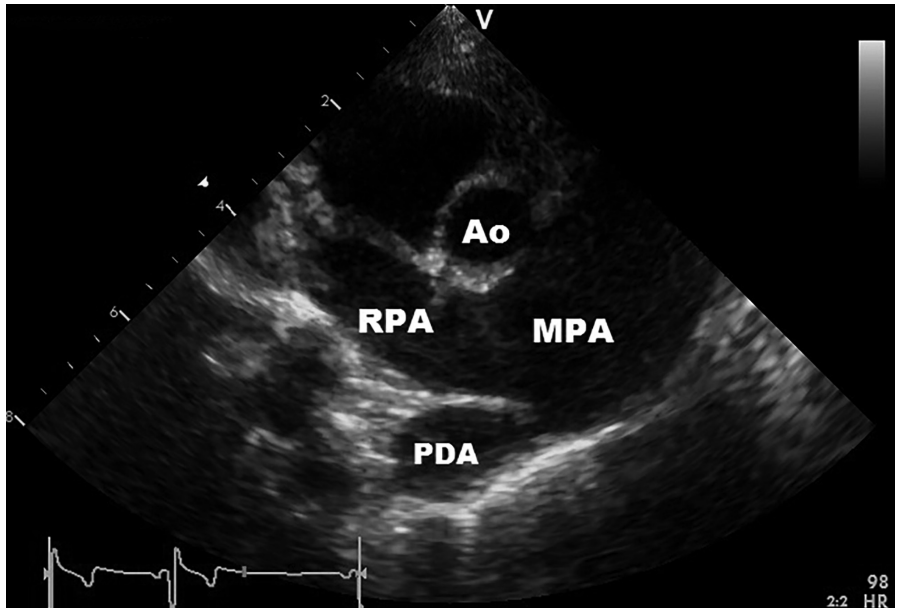

Figure 3—Transthoracic echocardiographic image of the dog in Figure 1. On the right parasternal short-axis view, a severely enlarged main pulmonary artery (MPA) and right pulmonary artery (RPA) are observed, both of which are larger than the aorta (Ao). Additionally, a tubular, tapering patent ductus arteriosus (PDA) can be observed.

vide microbubbles for contrast echocardiography, during which the lack of an intracardiac shunt and the presence of a right-to-left shunting PDA were evident. There was a moderate amount of pulmonic insufficiency and a mild amount of tricuspid regurgitation, the velocities of which were consistent with severe pulmonary hypertension. The changes to the right ventricle and right atrium were also consistent with severe pulmonary hypertension. No further abnormalities were found on the remainder of the echocardiographic examination.

Treatment and Outcome

The patient was prescribed sildenafil citrate (2 mg/kg [0.91 mg/lb], PO, q 8 h), a phosphodiesterase type 5 inhibitor, for treatment of pulmonary hypertension, and benazepril (0.35 mg/kg [0.16 mg/lb], PO, q 12 h), an angiotensin-converting enzyme inhibitor, to prevent further right ventricular myocardial remodeling associated with heart disease. Given the patient's Hct and apparent lack of clinical signs, the owners were instructed to have the dog's Hct rechecked in 4 to 6 months to monitor for development of polycythemia, which would be treated as necessary with phlebotomy. Six months after initial referral and evaluation, the dog was continuing to do well with no apparent clinical signs.

Comments

In the dog of the present report, findings on echocardiography and contrast echocardiography were integral in making the final diagnosis of a primarily right-to-left shunting PDA. Thoracic radiography revealed severe right-sided cardiac enlargement, an enlarged main pulmonary artery, and bulging of the cranial portion of the descending aorta, which are suggestive of a right-to-left PDA.[1] However, echocardiography was required for confirmation and determination of the direction of the shunt. Saline solution microbubble contrast studies confirmed the lack of intracardiac shunting and also the presence of microbubbles in the abdominal aorta after cephalic vein injection; this, in combination with the previous findings, confirmed a right-to-left shunting PDA. Other conditions, such as pulmonic stenosis, were also considered in the patient of the present report given the physical examination findings suggesting a right-sided congenital heart defect and increased pressure in the right side of the heart. However, the history of a left-sided heart murmur that went away was not consistent with pulmonic stenosis. Physical examination at hospital admission revealed the presence of a grade 4/6 right systolic murmur, a grade 2/6 right diastolic murmur, and mildly weak femoral pulses. The systolic murmur was caused by tricuspid regurgitation, and the diastolic murmur was caused by high-velocity pulmonic insufficiency. Typically, a mild amount of tricuspid regurgitation or pulmonic insufficiency would not be appreciated on auscultation, but the high pulmonary pressures accentuated these murmurs. The patient of the present report had no history of exercise intolerance, coughing, or loss of consciousness, which is somewhat unusual for a patient with severe pulmonary hypertension and a right-to-left shunting PDA.

Patent ductus arteriosus is one of the most common congenital heart defects in dogs, accounting for 25% to 30% of congenital cardiac defects.[2,3] Normally, the ductus arteriosus closes within hours after birth, but in some dogs, the ductus remains patent, allowing blood to shunt from the descending aorta to the pulmonary artery during both systole and diastole. In a smaller subset of cases (15% of dogs with PDA), the excess pulmonary flow induces pulmonary vascular changes, increased vascular resistance, and pulmonary hypertension.[4–6] If the pulmonary artery pressure exceeds the aortic pressure, then severe pulmonary hypertension is present and shunting occurs from the right side of the circulation toward the left side. This right-to-left flow of blood is sometimes referred to as a reversed PDA.

Clinical signs of a right-to-left shunting PDA may include exercise intolerance, shortness of breath, syncope, and seizures.[4,5] Commonly, there is no heart murmur in dogs with a right-to-left shunting PDA. None of these clinical signs were seen in this patient; therefore, imaging findings were integral to diagnosis. Although selective angiography and direct pressure measurements can be diagnostic for a reversed PDA, the increasing availability and noninvasive nature of echocardiography make this the diagnostic test of choice. In the case described in the present report, thoracic radiography suggested severe right heart enlargement and a substantial increase in right heart pressures. Additionally, a suspected ductus bump observed in combination with severe main pulmonary artery and right heart enlargement led to the presumptive diagnosis of a reversed PDA.[1,5]

Once the direction of blood flow through a PDA changes from a left-to-right to a right-to-left direction, it can no longer be surgically corrected without risking rapid development of life-threatening, acutely worsened pulmonary hypertension.[4,7] In the patient of the present report, sildenafil citrate was prescribed for management of pulmonary hypertension and will need to be continued on a lifelong basis. Sildenafil citrate has been shown to decrease the pulmonary artery pressure gradient and improve quality of life in dogs with clinically evident pulmonary hypertension.[8,9] Management of a patient with a right-to-left shunting PDA revolves around managing clinical signs associated with pulmonary hypertension as well as periodic phlebotomy for treatment of polycythemia.[4,7]

Findings described for the dog of the present report suggested that a PDA with clinically relevant pulmonary hypertension should be considered when a left-sided continuous heart murmur has a decrease in loudness or becomes undetectable over time. Additionally, young dogs with severe right-sided heart and

main pulmonary artery enlargement on diagnostic imaging without an obvious congenital lesion such as pulmonic stenosis should be evaluated for a right-to-left shunting PDA.

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