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CASE REPORT

Partial anomalous pulmonary venous connection with suspected pulmonary hypertension in a cat☆

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Abstract Partial anomalous pulmonary venous connection has previously been reported in the dog, but never in a cat. A 14-month-old Devon Rex cat was presented for echocardiography to evaluate a heart murmur noticed during a routine examination. The pertinent finding was right-sided cardiomegaly in the absence of an atrial septal defect or tricuspid regurgitation; pulmonary hypertension was suspected. A thoracic computed tomographic angiography study identified a partial anomalous pulmonary venous connection with the lobar veins of the left caudal, right middle, right caudal and accessory lung lobes draining into the caudal vena cava. The resultant volume overload is an easily overlooked differential diagnosis for right-sided cardiac enlargement. This is the first such report of this anomaly in a cat.

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Abbreviations

ASD	atrial septal defect
BAL	bronchoalveolar lavage
CT	computed tomography
ECG	electrocardiogram
PAPVC	partial anomalous pulmonary venous connection
PH	pulmonary hypertension
Qp:Qs	quantification of pulmonary and systemic flows
2-D	two-dimensional

A 14-month-old, 3.5 kg, female, spayed, Devon Rex cat was referred to the cardiology service at the University of Sydney Veterinary Teaching Hospital for evaluation of a heart murmur detected 2 weeks previously. The cat had no abnormal clinical signs at the time of presentation, although the owner did report intermittent coughing and wheezy respiratory sounds since the cat was 12 weeks old. On physical examination the cat was bright, alert and responsive, with a heart rate of 200 beats/minute and a grade II/VI left parasternal systolic murmur. The remainder of the physical examination, including jugular venous examination, was unremarkable.

Transthoracic two-dimensional (2-D) echocardiography revealed subjective evidence of mild right atrial dilatation and moderate right ventricular eccentric and concentric hypertrophy (Fig. 1 and Fig. 2). The main pulmonary artery and right pulmonary artery were subjectively enlarged compared to the aorta (pulmonary artery: aorta ratio 1.3). No structural cardiac diseases that could result in right ventricular concentric (any form of right ventricular outflow tract obstruction, including: subvalvular, valvular and supravalvular pulmonic stenosis, and double chamber right ventricle) or eccentric hypertrophy (including: atrial septal defect, tricuspid valve insufficiency, and arrhythmogenic right ventricular cardiomyopathy) could be identified. There was a mild increase in right ventricular outflow tract velocity (1.81 m/s). Doppler-derived systolic time intervals of pulmonary artery flow were obtained (acceleration time 30 ms; acceleration time: ejection time ratio 0.30), which if extrapolated from dogs would be supportive evidence of pulmonary hypertension (PH) (acceleration time $<$58 ms and acceleration time: ejection time $<$0.31).[1] There was no tricuspid regurgitation to interrogate and, therefore, estimation of systolic pulmonary artery pressure could not be performed. Quantification of pulmonary and systemic flows (Qp:Qs) was estimated by combining 2-D echocardiography and pulsed-wave Doppler (the estimated Qp:Qs in this patient was 3.13). Contrast echocardiography using agitated saline was performed, and demonstrated trivial right-to-left shunting through a patent foramen ovale; no other abnormalities were identified. In addition to the

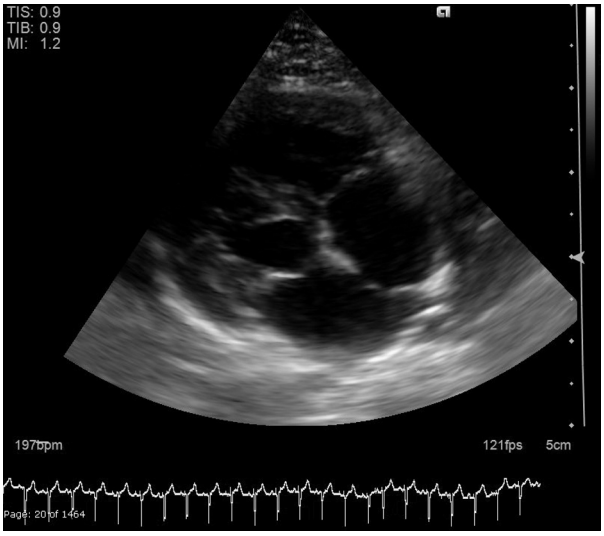

Figure 1 Right parasternal four-chamber long-axis view of the heart. Right ventricular eccentric and concentric hypertrophy is evident. The right atrium is subjectively mildly enlarged.

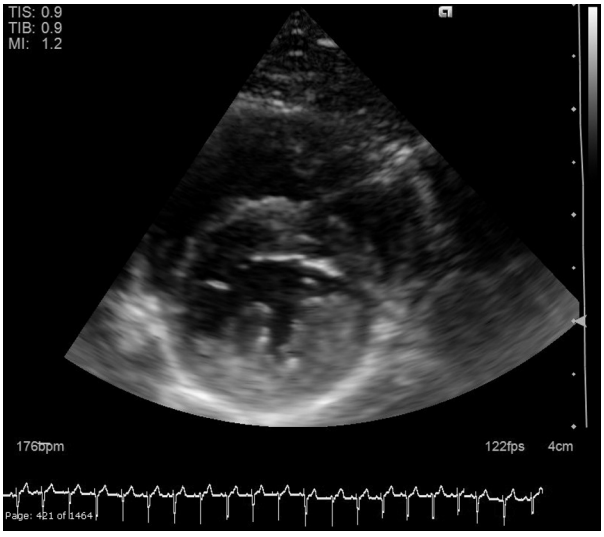

Figure 2 Right parasternal short-axis view at the level of the papillary muscles. Right ventricular eccentric and concentric hypertrophy is evident.