What´s your Cardiology Diagnosis? – Exercise intolerance and a low-grade heart murmur in a young dog – surgical treatment

What´s your Diagnosis?

Adriana França[1] DVM, Geoff Culshaw[1] BVMS PhD DVC, Nicolas Israeliantz[1] LdoVet, Kelly Blacklock[1] BVMS DipECVS PhD FRCVS, Maria Ines Oliveira[1] DVM MSc DipECVIM-CA

1. Hospital for Small Animals, Royal (Dick) School of Veterinary Studies, University of Edinburgh.

Corresponding author - Adriana Franca (adrianafranca99@gmail.com)

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History - A 7-month-old, 20 kg male Labrador Retriever was referred for investigation of a low-grade heart murmur in association with exercise intolerance.

On physical examination, the dog's body score condition was 4/9, rectal temperature was 37.9ºC (reference range, 37.2° to 39.2°C). The heart rate was 110 beats per minute (bpm) (reference range, 80 to 120 beats/min) with regular rhythm and normodynamic, symmetrical and synchronous pulses. There was a grade II/VI systolic heart murmur over the left base. Oscillometric systolic blood pressure was 164 mmHg (reference range, 120-130 mmHg). On pulse oximeter, his oxygen saturation in room air was 100% and there was no B-lines on point-of-care ultrasound that would suggest the presence of pulmonary oedema. Haematologic evaluation revealed mild neutropaenia ($3.460*10^9$/L, reference range 3.60 to 12.0 10^9/L) and mild anaemia (PCV 0.35L/L, reference range 0.39-0.55 L/L). On serum biochemistry, there was mild hypoproteinaemia (49.6g/L, reference range 58.00-73.00 g/L), mildly increased alkaline phosphatase (133 U/L reference range 20.0 - 60.00 U/L), mildly decreased triglycerides (0.47 mmol/L reference range 0.57-1.14 mmol/L), increased creatinine kinase (348 U/L, reference range 50.00 - 200.00 U/L), increased phosphate inorganic (2.3 mmol/L, reference range 0.90-2.00 mmol/L) and mildly decreased magnesium (0.65 mmol/L, 0.69-1.18 mmol/L). Prothrombin and partial thromboplastin times were within reference ranges.

Echocardiography was performed (Figure 1).

"Formulate differential diagnoses, then continue reading."

Diagnostic imaging findings and Interpretation – echocardiography identified severe left atrial dilation (LA:Ao ratio 2.19, reference <1.6:1), eccentric left ventricular and moderate mitral valve regurgitation. Systolic function was subjectively poor, and measurements of systolic function were compatible with systolic dysfunction. Interrogation of the right atrium, right ventricle and tricuspid valve was unremarkable. There was a small jet of pulmonic insufficiency that was below the threshold for pulmonary hypertension (velocity 0.65m/s, reference <2.2m/s). Cardiac masses or pleural/pericardial effusions were not observed. There was a continuous, retrograde turbulent flow within the main pulmonary artery. This appeared to consist of two divergent jets emanating from a common source. One jet was directed towards the pulmonic valve at 2.74m/s (systole) decreasing to 1.81m/s (diastole). The second jet entered the left pulmonary artery directly. An aortopulmonic vascular malformation with left-to-right-shunting was suspected. Because of the relatively low velocity, differentials included patent ductus arteriosus (PDA) with pulmonary hypertension, coronary artery fistula and aortopulmonary window [1,2,3,4]. The divergent turbulent jets also raised the possibility of an additional main pulmonary artery dissection [1,2,3,4]. Causes of mitral insufficiency included mitral valve dysplasia and mitral incompetence secondary to annular dilation caused by volume overload (Figure 2 – A, B, C).

A computed tomography multiphase contrast angiography (angio-CT) under general anaesthesia was performed for identification and characterisation of the shunting vessel (Figure 3 – panel D, E). Arising from the right side of the descending aorta at the level of the mid aspect of the seventh vertebral body, an abnormally enlarged dorsal intercostal artery was identified. It branched

immediately into an equally enlarged left bronchoesophageal artery that ran cranially and tortuously along the dorsal aspect of the oesophagus to form an abnormal network of small tortuous vessels surrounding the oesophagus and the trachea. Arising from this network, a prominent and aberrant vessel was visible entering the left pulmonary artery at its dorsomedial wall at the level of the caudal aspect of the fourth thoracic vertebra, through an ostium of approximately 3.7mm. On the dextro-phase post-contrast series, a jet of non-enhanced blood was present entering the contrast enhanced left pulmonary artery, confirming a left-to-right (systemic to pulmonary) direction of the shunt.

Treatment and outcome - The patient was managed with medical and surgical treatment. Oral pimobendan was initiated preoperatively for inotropic support at 0.25mg/kg twice daily, three weeks prior surgical ligation of the shunt. A left sided lateral thoracotomy was performed at the level of the fifth intercostal space. The lateral aspect of the branch originating from a perioesophageal network of tortuous vessels was identified at the entry of the pulmonary artery and a palpable thrill was felt when palpating the main branch as it joined the pulmonary artery. It was dissected free from the surrounding tissue and ligated. As it was occluded, the thrill was immediately dissipated, and the murmur no longer audible with oesophageal stethoscope. There was a momentary reflex bradycardia (105 bpm to 90 bpm) and a mild increase in arterial blood pressure (SP from 120mmHg to 130 mmHg) during the occlusion (possible Branham sign), that resolved, without the need for any medical intervention. Post-operative recovery was uneventful, and the patient was discharged two days later.

On follow-up echocardiography after one month, there was moderate residual mitral insufficiency, but systolic dysfunction had improved slightly after surgery. Residual left-to-right shunting could not be identified, indicating successful ligation of the shunt. The left atrial size and left ventricular (diastolic) diameters normalised after surgery, consistent with a reduction of the left-sided cardiac preload.

At ten months post-operatively, mitral insufficiency has reduced further. Systolic function was unchanged compared to the first post-operative scan. Left atrial and ventricular sizes were still within the normal reference ranges and residual shunting was still not apparent.

Comments –

Patent ductus arteriosus results in a highly characteristic continuous heart murmur over the left cranial thorax[3]. Far less common are aortopulmonic or arteriopulmonic vascular malformations that clinically resemble a PDA and which have previously reported in dogs[1,2]. Similar hemodynamic consequences such as left sided volume overload and reduced systolic function can make a challenge to differentiate from PDA solely by Doppler echocardiography [1].

A computed tomography scan with contrast angiography has been shown to play a determinant role in the characterisation of the shunting vessels[1,4]. Previous studies suggest that the volume overload seen in aortopulmonic vascular abnormalities can lead to left-sided congestive heart failure, even though these abnormalities often present as an incidental finding[1]. In this clinical case there was evidence, early in the patient's life, of volume overload. Surgical ligation should therefore be considered and was shown to be a valid method of occlusion

and successful in resolution of congestive heart failure in aberrant bronchoesophageal arteries[1]. Furthermore, the use of angio-CT to plan the surgical approach and a suitable site for ligation close to the main pulmonary artery entry point is validated by the lack of residual shunting and reduction in volume overload still present nearly one year later.

Two key features raised the suspicion that it was not a PDA causing the continuous murmur. First, the murmur intensity was relatively low, relating to a systolic shunt velocity within the main pulmonary artery <3m/s. Second, there was no evidence of pulmonary hypertension and bidirectional shunts, which would be expected to accompany low shunt velocities with PDA[1,5]. Nevertheless, an angio-CT computed tomography scan with contrast angiography was required in order to confirm the diagnosis and to characterise the nature of the shunting vessels, which emphasises the importance of advanced imaging and angiography prior implementation of treatment plans. [1,4]

The noteworthy feature of this case was the indirect and tortuous route of the shunt. Previous reports of aortopulmonic vascular malformations or aberrant bronchoesophageal arteries identify a single parent vessel following a relatively direct course and plexuses of tortuous vessels of unclear origins. In our case, there was a clear parent dorsal intercostal artery, an intermediate network of tortuous vessels and a final, single vessel entering the main pulmonary artery.

In the dog of the present report, the ten-month follow-up has shown successful surgical ligation of the aberrant bronchoesophageal based on absence of residual flow, improved mitral regurgitation, normalisation of cardiac dimensions and complete resolution of clinical signs.

In conclusion, angio-CT plays a vital role in the characterisation and successful surgical planning of haemodynamically significant arteriopulmonic vascular malformations that mimic PDA.

Conflicts of interest

The authors declare no conflict of interest.

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Figure Legends

Figure 1 – Two-dimensional echocardiographic views of a 7-month-old, male Labrador Retriever with a low-grade heart murmur and clinical signs of exercise intolerance and reduced energy levels (A-Parasternal short-axis view of the heart base, B – Short-axis view at the heart base, optimized for pulmonary artery, C- Right parasternal long-axis view),

Figure 2 – Two-dimensional echocardiographic views (A- Parasternal short-axis view of the heart base showing the left atrium, LA (asterisk) and the aorta, Ao (star). Increased LA:Ao ratio = 1,78. Electrocardiogram showing sinus rhythm with a heart rate of 116 beats per minute. B – Short-axis view at the heart base, optimized for pulmonary artery– demonstrating a turbulent blood flow into two jets, one into the main pulmonary artery (asterisk) and one into the left pulmonary artery – demonstrating a turbulent blood flow into two jets, one into the main pulmonary artery and one into the left pulmonary artery secondary to a left to right shunting structure (white arrow). Electrocardiogram showing sinus rhythm with a heart rate of 107 beats per minute.. C- Right parasternal long-axis view demonstrating mitral regurgitation with colour doppler. Electrocardiogram showing sinus rhythm with a heart rate of 116 beats per minute.

Figure 3 - Post contrast thoracic CT images with a 5 mm-thick slab Maximum Intensity Projection in transverse plane at the level of T7 (D) and in sagittal reconstruction (E). Note the abnormally engorged dorsal intercostal artery arising from the right side of the aorta (arrow), as well as the dense network of

paraoesophageal vessels (asterisk). Window width, 714 HU; window level 270 HU; 1-mm slice thickness. L = Left.