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Right-sided congestive heart failure secondary to supraventricular tachycardia in a dog with a right atrial mass

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TITLE OF CASE ***Do not include "a case report"***

Right-sided congestive heart failure secondary to supraventricular tachycardia in a dog with a right atrial mass

SUMMARY ***Up to 150 words summarising the case presentation and outcome (this will be freely available online) - 149***

A dog was referred to the authors' hospital for further investigations of pelvic limb collapsing episodes. Physical examination revealed a positive hepatojugular reflux, positive fluid thrill on abdominal palpation and an irregular heart rhythm (144 bpm) with pulse deficits. A 6-lead ECG showed focal atrial tachycardia (FAT). Doppler echocardiography revealed systolic dysfunction, dilated cardiomyopathy (DCM) phenotype and the presence of a heterogeneous mass in the right atrium; this was confirmed by a CT study. Free abdominal fluid was detected, sampled and analysed: this was consistent with modified transudate secondary to right-sided congestive heart failure (R-CHF). The dog responded well to heart failure and anti-arrhythmic medications. He was presented 8 weeks later after development of respiratory signs (cough). Investigations revealed stable cardiac disease but several radiopaque nodules within the lung parenchyma compatible with metastatic disease. Ten weeks after presentation the dog was euthanized due to worsening of the respiratory signs.

BACKGROUND ***Why you think this case is important – why did you write it up?***

This case report discusses investigations of a frequent and non-specific clinical sign (pelvic limb collapse) in an elderly dog with severe and complicated underlying cardiac disease. This highlights the importance of performing a thorough clinical examination, in this case focusing on auscultation and femoral pulse palpation, and the importance of further investigations including electrocardiography and echocardiography. Moreover, it provides insight into investigations, causes and treatment of supraventricular tachycardias (SVT) since these can be a challenge for many practitioners. It is also an example of how co-

morbidities can occur (i.e. laryngeal paralysis) making confirming a final diagnosis more challenging.

CASE PRESENTATION ***Presenting features, clinical and environmental history***

A 12-year-old male neutered Labrador Retriever was referred for investigations of collapsing episodes which started 2-3 days before presentation. These episodes were characterized by episodic hind limb weakness without loss of consciousness; they were not related to exercise although the dog was reported to be lethargic. The primary veterinarian's assessment reported a heart rate of 180 bpm with an irregular rhythm and a pulse rate of 70 ppm with variable pulse quality. There was no history of toxin ingestion, the patient was up to date with vaccinations, ecto- and endo-parasite prevention and he had never travelled abroad.

At presentation, the dog was quiet, alert and responsive, mucous membranes were pale and moist with a delayed capillary refill time (CRT) of 3 seconds. The heart rate was 144bpm with an irregular rhythm; femoral pulses were of variable quality and pulse deficits were evident. No heart murmur was detected, and pulmonary auscultation was unremarkable, although the dog was panting incessantly. There was minor upper respiratory tract noise that was more obvious during excitement. The abdomen was distended with a fluid thrill on ballottement, but no other abnormalities were detected on abdominal palpation. The jugular veins were not obviously distended, but a positive hepatojugular reflux (HJR) was elicited. He weighed 34.2 kg, body condition score 3/9. The rest of the physical examination was unremarkable.

INVESTIGATIONS ***If relevant***

The systolic arterial blood pressure (Doppler method) was 140 mmHg. A six-lead electrocardiogram (ECG) was recorded and showed the presence of an irregular tachyarrhythmia of supraventricular origin (supraventricular tachycardia; SVT) interrupted by occasional sinus complexes (figure 1, figure 2). The heart rate was 220-250 bpm. P' waves could be observed with varying P'Q intervals, which were positive in leads I, II and aVF, and negative in lead aVR, consistent with dorsal right atrial origin. Electrical alternans was also present. The RP'/P'R interval ratio was approximately 2, although there was considerable variation in this over the trace. This was consistent with a focal atrial tachycardia (FAT). The ladder diagram (figure 2) shows a trend to increasing P' rate prior to the AV block, and the variable slope showing conduction across the AV node indicates progressive slowing prior to the AV block, reflecting the physiological decremental conduction properties of the AV node at high atrial rates. When increasingly rapid atrial impulses are generated and reach the atrioventricular node, the conduction through this is decreased resulting in a slower ventricular rate.

Echocardiography revealed the presence of a right atrial mass at the dorsal aspect of the right atrium (figure 3); this was not resulting in reduced flow from the venae cavae demonstrated by colour Doppler. It was heterogeneous and measured 2.13 x 1.62cm. There was subjective dilation of the right ventricle and moderate right atrial enlargement. Left ventricular (LV) systolic function was mildly impaired (ejection fraction calculated with Simpson's method of discs: 39%; ref >50%; LV end-systolic volume index 42.2ml/m^2, ref <30ml/m^2) (1) with a rounded but not significantly dilated left ventricle and mildly increased left atrial size. These findings were compatible with myocardial dysfunction and, given the SVT, tachycardia-induced cardiomyopathy (TICM).

Abdominocentesis was performed and analysis confirmed a protein-rich modified transudate, consistent with ascites associated with right-sided CHF (appendix 1). Cardiac Troponin I was markedly elevated at 3.07ng/mL (ref <0.15ng/mL) indicating current or recent cardiomyocyte injury. The thyroxine hormone was normal at 38.9nmol/L (ref. 5-44) consistent with euthyroidism (2). A venous blood gas analysis revealed mild increase in creatinine (appendix 2).

Thoracic radiographs were also performed and did not show any significant abnormality (figure 4, figure 5).

The following day following treatment (see later), the patient was stable and further investigations were performed. An ECG was repeated and it showed a regular rhythm at a rate of 70 bpm and a negative, prolonged and notched P' wave (0.06s; ref. <0.04) in leads II, III and aVF and tall T waves (figure 6). The QRS complexes were within normal limits. These findings were compatible with an idio-junctional rhythm with retrograde concentric atrial activation.
CT scan with contrast (Ioversol, Optiray, Guerbet; arterial phase triggered by presence of contrast in the ascending aorta) of the thorax and abdomen was performed under sedation and confirmed the presence of a nodule in the right atrium (2.1x1.9 x 1.7cm); it also detected a larger mass (2.9x2.1x2cm), more cranial, adjacent to the ascending aorta at the base of the heart (figure 7; figure 8), which had not been detected during echocardiography.
A venous blood gas analysis was repeated (appendix 2) showing hypernatraemia and high bicarbonate and creatinine. Hypokalaemia was likely to be associated with furosemide administration and creatinine levels can increase due to mild azotaemia following diuretic therapy.

DIFFERENTIAL DIAGNOSIS *If relevant*

In an elderly dog presenting with episodic pelvic limb collapse which is not associated with tonic-clonic movements or loss of consciousness there are a significant number of differential diagnoses that need to be considered.

The presence of pale mucous membranes with delayed CRT, a tachyarrhythmia with pulse deficits, positive hepatojugular reflux and abdominal fluid thrill would suggest that a cardiac cause is likely; it is indeed well known that both bradyarrhythmias and tachyarrhythmias can induce collapse. Amongst tachyarrhythmias, supraventricular tachycardias (SVTs), atrial fibrillation or flutter and ventricular tachycardias, should be taken into consideration. These may be associated with transient loss of consciousness, which may also result in possible urination / defaecation. The recovery time is usually quick. The underlying mechanism is cerebral hypoxia secondary to decreased cardiac output. If persistent, tachyarrhythmias can lead to a DCM phenotype called TICM characterized by chamber dilation and systolic dysfunction.
Structural heart disease with impaired systolic function would lead to a decreased cardiac output and subsequent cerebral hypoxia. Amongst these, in a large breed dog we could consider DCM and valvular defects (i.e. aortic stenosis, pulmonic stenosis). The latter are less likely in this case considering the absence of an audible murmur. Cardiac output would be reduced in case of pericardial effusion; this is easily excluded on echocardiography. Pulmonary hypertension can also be responsible for similar episodes and echocardiography can be used to rule this out.
A delayed CRT and pale mucous membranes would also support a cardiovascular problem (i.e. shock, systolic failure, impaired cardiac output). Anaemia can mimic this sign and should be excluded.
Abdominal distension, a positive fluid thrill and positive hepatojugular reflux are signs compatible with increased central venous pressure that can be secondary to right-sided CHF or cardiac tamponade; the presence of the hepatojugular reflux indicates that the caudal vena cava and cranial vena cava are connected via the right atrium, so these signs are unlikely to be the consequence of obstruction to one or the other (e.g. Budd-Chiari causes of ascites, or evidence of cranial caval syndrome). Tricuspid dysplasia is a heritable disorder in Labrador Retrievers and can lead to right-sided CHF. Other causes of increased central venous pressure can be related to cardiac tamponade, TICM or DCM.

In cases of hypoalbuminaemia, ascites can be detected (a biochemistry panel can rule this out) and internal bleeding (i.e. haemoabdomen) can give both abdominal distension and collapse, if associated with acute blood loss (which would also increase the heart rate). These conditions are not associated with a concurrent positive hepatojugular reflux; the combination of these findings indicates R-CHF.

Orthopaedic or neurological issues such as osteoarthritis, intervertebral disc disease, other spinal disease, cruciate disease or neoplastic process can lead to pain and pelvic limbs weakness with episodic collapse. Usually, these are associated with orthopaedic pain or abnormalities on neurological examination; these were not present in this case.

Atypical seizures could not be ruled-out although are less likely considering the other physical examination findings and the rapid recovery with no post-ictal signs.

TREATMENT *If relevant*

The patient was hospitalized and intravenous furosemide therapy was initiated at a rate of 2 mg/kg IV every 8 hours (Dimazon, Intervet) for 48 hours to control the right-sided heart failure; afterwards, oral administration at the same dosage was commenced. To address the SVT, diltiazem (modified release) was started (2 mg/kg every 8 hours, PO; Crescent Pharma Ltd). Pimobendan was also administered intravenously initially (0.15 mg/kg) and then orally after 12 hours (0.23 mg/kg every 12 hours; Boehringer Ingelheim GmbH). ECG telemetry was used to monitor the patient overnight. After 12 hours, the heart rate was not yet controlled, therefore, the diltiazem dose was increased to 3 mg/kg PO every 8 hours and good rate control was achieved (120-140 bpm).
The patient was discharged on oral medications 48 hours after admission (diltiazem dose: 3 mg/kg PO q8h). Abdominal distension had resolved, the heart rate was 120 bpm with regular rhythm and he had pink mucous membranes with a CRT <2 seconds. The body weight was 30kg with a 4kg weight loss since admission, associated with loss of his abdominal effusion.

OUTCOME AND FOLLOW-UP

The patient was re-checked 2 weeks later. No exercise intolerance or collapsing episodes were reported. His weight was 31.2 kg with a BCS of 4/9. The heart rate was 100 bpm with a regular rhythm and synchronous femoral pulses of good quality. No fluid thrill was detected. Serum biochemistry showed mild elevation of urea (13.1 mmol/L; ref. 2.5-9.6). Creatinine and electrolytes were within normal limits. Cardiac Troponin I was 0.3 ng/mL (ref <0.15ng/mL). A 6-lead ECG showed sinus rhythm with a rate of 88 bpm and evidence of P mitrale (P duration 0.05 s (ref. <0.04), suggestive of left atrial enlargement (figure 9). Echocardiography was repeated showing marked improvement in systolic function with an ejection fraction of 64.7% (prev. 38%; ref. <50%) (1) and end-systolic volume index of 17 ml/m^2 (prev. 42.2; ref. <30ml/m^2) (1) and normal right and left atrial size; the right ventricle was still dilated, similarly to the previous echocardiography. These findings, showing reverse remodelling and improved systolic function with management of the SVT, were consistent with previous diagnosis of TICM.
A 24-hour ambulatory ECG recording (Holter monitoring) showed predominantly sinus rhythm and sinus arrhythmia with occasional paroxysms of SVT (associated with stress or excitement).
Two months after presentation, the dog was re-assessed due to the onset of a dry cough of two week's duration, which was worse during exercise. He was quiet but alert and the heart rate was 100 bpm with regular rhythm and strong, synchronous femoral pulses. Pulmonary auscultation was unremarkable although loud upper respiratory stridor was noted. Lymph nodes were normal in size. Tracheal pinch test was negative. Rectal temperature was 38.5° C.
The dog was admitted for further investigations. Haematology was unremarkable. Renal biochemistry showed mildly elevated urea and normal creatinine (appendix 3). Echocardiography did not show significant changes, the right atrial mass and the systolic function were similar to last time. Thoracic radiographs were repeated and showed several radiopaque nodules (0.4-1.5cm) within the lung parenchyma (figure 10; figure 11). Sampling of the nodules was not possible as they were not accessible via the thoracic wall. Laryngeal function was also assessed under deep sedation, which showed bilateral laryngeal paralysis. The owner declined further investigations.
Ten weeks after initial presentation the dog was euthanized at the referring practice due to deterioration of the cough and respiratory distress. Post-mortem examination was declined.

DISCUSSION *Include a very brief review of similar published cases*

The ECG at presentation showed a variable P'R (120-160ms) and a long RP' (±240ms) segment (RP'/P'R=2), a positive P' in lead II and aVF, compatible with FAT with occasional 2nd degree AV block. Focal atrial tachycardia is characterized by narrow QRS complexes,

fast ventricular rates (210-330 bpm) and a mostly regular RR interval although 2nd degree AV block and cycle length irregularity are common. A long RP' interval suggests the P' wave is triggering the following QRS complex (atrial origin of the tachycardia), whereas a short RP' is typical of retrograde activation most commonly seen in atrioventricular reciprocating tachycardia (AVRT) and junctional rhythms. AVRT share some electrocardiographic features with FAT (narrow QRS complexes, regular RR interval) but are associated with the presence of muscle bundles (accessory pathways) that create a direct connection between atria and ventricles, bypassing the AV node. Although Labradors are predisposed to AVRT triggered by accessory pathways (3), the presence of AV block confirms this was an atrial tachycardia, independent of the AV node. In the case of AVRT an AV block (with retrograde atrial activation and a short R-P') will interrupt the re-entry circuit and end the tachycardia. Cycle length irregularity, as showed in the ECG at presentation, is also common in FAT (4). Labrador retrievers are reported to be predisposed to FAT and often the ectopic focus is distributed within the right atrium (5). However, there were some characteristics of re-entrant tachycardia such as electrical alternans that can be attributed to nonspecific intraventricular conduction abnormalities (6). Labrador Retrievers have also been reported to be affected by isorhythmic atrioventricular dissociation (IAVD) and accelerated idiojunctional rhythm; IAVD is characterized by independent atrial and ventricular foci that generate impulses at a similar rate. We consider this less likely since the P' and QRS complexes remained associated in this patient (7) and the rate is excessive and rhythm too irregular for both the atrial and ventricular complexes.
However, a definitive diagnosis of FAT would require an electrophysiological study that was not performed in this case.
After initiation of antiarrhythmic therapy and successful rate control, the patient had reduced heart chamber size and improved systolic function, resolution of the ascites and a marked clinical improvement. Dogs paced at 180 bpm for 3 weeks have been shown to develop a TICM (8); 73% of people with FAT develop TICM (9). Remodelling after rate control has also been described with substantial regain of systolic function (10). Primary DCM was unlikely considering the absence of marked dilation and the reverse remodelling noted after appropriate rhythm control. Diltiazem is a calcium channel blocker which slows conduction in nodal tissue leading to its safe and effective use in supraventricular arrhythmias (11,12). It is possible that the pimobendan therapy is also responsible for some of the reverse remodelling observed at the recheck; the inodilator effect of this drug will improve contractility and reduce ventricular size in dogs with mitral valve disease and dilated cardiomyopathy (13,14).

The patient showed a different rhythm after 24 hours of diltiazem therapy with a junctional rhythm and concentric retrograde atrial activation preceding the QRS complexes. It is possible that diltiazem suppressed both the ectopic focus and sinus node, leading to the junctional rhythm and retrograde activation of the atria from the junctional focus; atrial depolarization was earlier than ventricular depolarization suggesting the junctional focus was atrionodal and ventricular activation followed conduction through the compact AV node. It is also possible that the sino-atrial node had been suppressed by the ectopic focus during the long period of supraventricular tachycardia; this would have allowed the junctional rhythm to manifest (15).
It is possible the tachyarrhythmia was triggered directly by the presence of a mass in the right atrium; although this could not be confirmed, it is reported in people (16).
Based on location and appearance, the right atrial mass was speculated to be a hemangiosarcoma, whereas the heart base mass might be more consistent with a chemodectoma; histological confirmation was not possible since sampling was considered dangerous and the client declined necropsy. Haemangiosarcomas are the most common cardiac tumour identified in dogs and aortic body tumours are also frequently reported, especially in brachycephalic breeds. (17) Dogs with right atrial haemangiosarcomas have been reported to suffer from right-sided CHF. (18) It is unclear if the pulmonary nodules noted on the subsequent radiographs were associated with metastatic spread of the right atrial mass, but it is reported that haemangiosarcomas can metastasize to the lungs (19). Given the location in the thorax and the likelihood of bleeding during sampling, aspiration of the pulmonary nodules for cytology samples was not performed.

The arrhythmia did not induce loss of consciousness but hindlimb weakness that are more commonly attributed to neurological or orthopaedic issues. The most likely explanation for this clinical sign is a near-fainting episode also described in people suffering from severe paroxysmal arrhythmias; these usually precede full collapses.

The cough could be related to the lung nodules, considering the extent of the lung disease. Laryngeal paralysis surgical approaches (i.e. laryngeal tie-back) and associated risks were discussed but declined by the client (20, 21) given the poor prognosis with the other co-morbidities.

LEARNING POINTS/TAKE HOME MESSAGES ***3 to 5 bullet points – this is a required field***

1) Tachyarrhythmias, if persistent, can cause a dilated cardiomyopathy phenotype with systolic dysfunction called tachycardia-induced cardiomyopathy (TICM), which may be reversible after good rate control. TICM can result in congestive heart failure.
2) Tachyarrhythmias can be caused by cardiac or systemic disease. Labradors are frequently affected by AVRT and FAT with or without underlying disease. Cardiac neoplasia could also potentially trigger arrhythmias.
3) R-P' and P'-R intervals are useful to distinguish FAT from AVRT. These conditions have different treatment options therefore recognizing them is important.
4) Cardiac collapse and syncope can sometimes be confused with those caused by orthopaedic (i.e. osteoarthritis) or neurological (i.e. idiopathic epilepsy) conditions. Cardiac auscultation and femoral pulses palpation are useful and inexpensive tools helpful in guiding further investigations.

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FIGURE/VIDEO CAPTIONS ***figures should NOT be embedded in this document***

Figure 1: ECG trace at presentation showing irregular supraventricular tachycardia at a rate of 220bpm with occasional sinus complexes.
P' waves can be observed during the periods of SVT with varying P'Q intervals. The P' waves are positive in lead I, II, III and aVF (superior-to-inferior axis) indicating their origin is likely from the right atrial roof. Electrical alternans and cycle length variability are also present.
The P'R is variable (120-160ms) and R-P' long (240ms) that makes an atrial tachycardia more likely (R-P'/P'-R=2). The summation of P' on T waves increases their amplitude.
The ECG is compatible with focal atrial tachycardia with occasional 2nd degree AV block (see figure 2) and the presence of increased P wave duration (0.06s; ref. <0.04) (due to P mitrale or interatrial conduction disturbance).

50mm/s; 10mm/mV. The 50 mm/s six lead trace (main ECG; upper panel) is represented in the first half of the bottom trace (lead II rhythm strip; 25 mm/s).

Figure 2: Ladder diagram of the ECG trace at presentation. Close up of figure 1.
The black vertical lines indicate the conduction within the atria (top line), the conduction through the AV node (mid line) and the conduction through the ventricles (bottom line). If there is an AV block the bottom line will not be present since it will not reach the ventricles (i.e. blue lines).
There is a trend to increasing P' rate prior to the AV block, and the slope showing conduction across the AV node (between the first and second horizontal line in the ladder diagram) indicates progressive slowing prior to the AV block, reflecting the physiological decremental conduction properties of the AV node at high atrial rates.
In black the complexes originating from the ectopic focus (P'). In blue the P' waves that have been blocked (2nd degree atrioventricular block). In red the sinus complex. Lead I, II, III. 50mm/s; 10mm/mV.

Figure 3: Echocardiographic images of the right atrial mass (arrows) from a non-standard left apical 4 chamber view optimized for the right heart (moved cranially with the left heart on the left of the image). The mass is showed here in the dorsal aspect of the right atrium.

Figure 4: Dorsoventral radiograph at presentation, not showing obvious signs of pulmonary oedema or masses. The cardiac silhouette is within normal limits as well as the lobar vessels.

Figure 5: Right lateral radiograph at presentation, not showing obvious signs of pulmonary oedema or masses. The cardiac silhouette is within normal limits as well as the lobar vessels.

Figure 6: ECG trace 24 hours after admission and therapy with diltiazem, initially at 2 mg/kg PO, then at 3 mg/kg every 8 hours. Note the regular rhythm at a rate of 70 bpm with negative, prolonged (0.06s; ref. <0.04) and notched P' wave and tall T waves (similar height of R wave). Considering the P' waves are negative in lead II, III and aVF, similar amplitude positive P' in leads aVR and aVL with isoelectric P' in lead I, this ECG is compatible with a retrograde concentric atrial activation. This would be consistent with a junctional rhythm with retrograde activation of the atria and later normal His-Purkinje activation of the ventricles. There is mild increase in QT interval (0.28s; ref 0.15-0.25). Note: electrical interference affecting baseline of leads I, III and aVL (likely poor contact for left fore electrode attachment). However, this does not affect interpretation of the ECG.
50mm/s; 10mm/mV. The 50 mm/s six lead trace is represented as the first half of the bottom trace (lead II; 25 mm/s).

Figure 7: CT image (post-contrast, arterial phase, soft tissue window) of the right atrial mass (arrows). RA: right atrium; RV: right ventricle; LA: left atrium; LV: left ventricle.

Figure 8: CT image (post-contrast, arterial phase, soft tissue window) of the heart base mass (arrows). A: aorta.

Figure 9: ECG trace 2 weeks after presentation showing sinus rhythm at a rate of 88bpm with P mitrale (0.05s; ref. <0.04) and notched QRS.
50mm/s; 10mm/mV. Baseline artefact associated with respiratory movement.

Figure 10: Dorsoventral radiograph at the 2 month recheck. Note the multifocal nodules in the lung parenchyma suspected to be metastatic disease.

Figure 11: Right lateral radiograph at the 2 month recheck. Note the multifocal nodules in the lung parenchyma suspected to be metastatic disease.

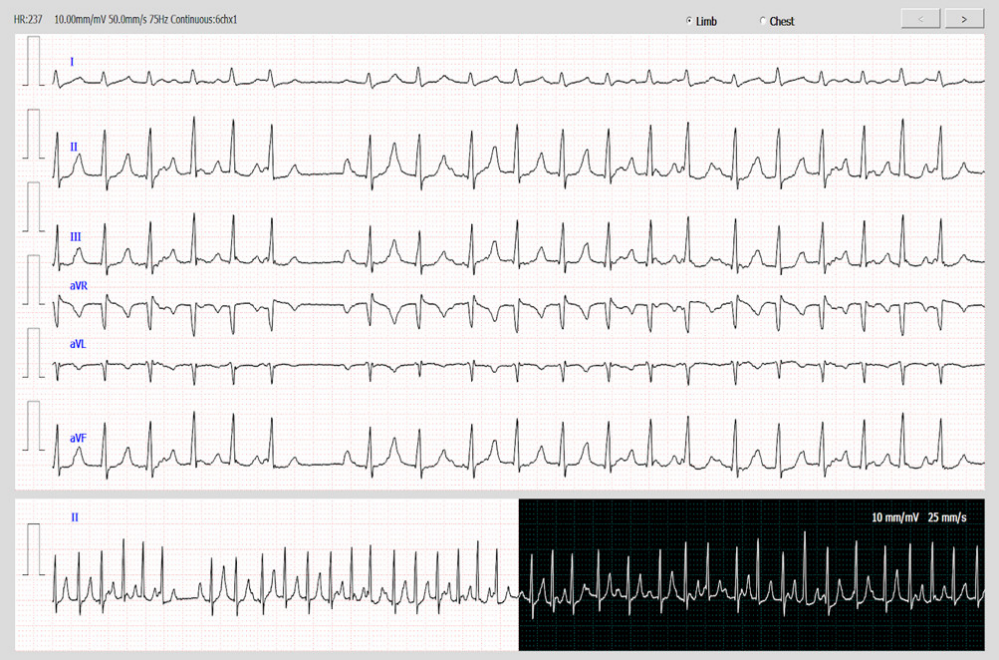

Figure 1 ECG trace at presentation showing irregular supraventricular tachycardia at a rate of 220bpm with occasional sinus complexes.P' waves can be observed during the periods of SVT with varying P'Q intervals. The P' waves are positive in lead I, II, III and aVF (superior-to-inferior axis) indicating their origin is likely from the right atrial roof. Electrical alternans and cycle length variability are also present. The P'R is variable (120-160ms) and R-P' long (240ms) that makes an atrial tachycardia more likely (R-P'/P'-R=2). The summation of P' on T waves increased their amplitude.The ECG is compatible with focal atrial tachycardia with occasional 2nd degree AV block (see figure 2) and the presence of increased P wave duration (0.06s; ref. <0.04) (due to P mitrale or interatrial conduction disturbance).

50mm/s; 10mm/mV. The 50 mm/s six lead trace (main ECG; upper panel) is represented in the first half of the bottom trace (lead II rhythm strip; 25 mm/s).

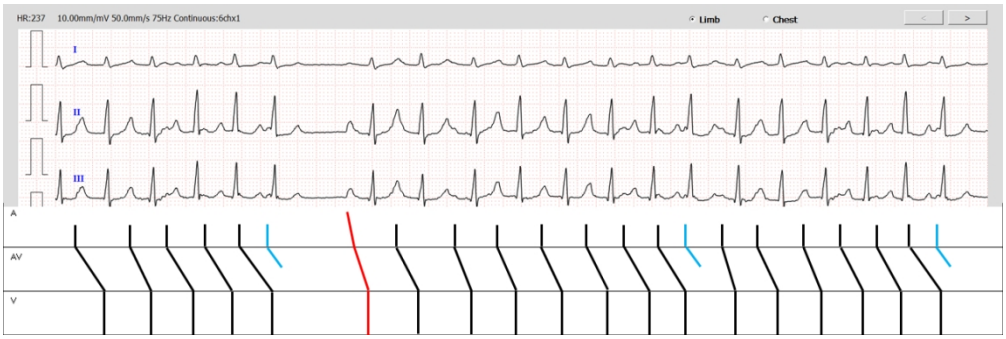

Figure 2 Ladder diagram of the ECG trace at presentation. Close up of figure 1. The black vertical lines indicate the conduction within the atria (top line), the conduction through the AV node (mid line) and the conduction through the ventricles (bottom line). If there is an AV block the bottom line will not be present since it will not reach the ventricles (i.e. blue lines). There is a trend to increasing P' rate prior to the AV block, and the slope showing conduction across the AV node (between the first and second horizontal line in the ladder diagram) indicates progressive slowing prior to the AV block, reflecting the physiological decremental conduction properties of the AV node at high atrial rates.In black the complexes originating from the ectopic focus (P'). In blue the P' waves that have been blocked (2nd degree atrioventricular block). In red the sinus complex. Lead I, II, III. 50mm/s; 10mm/mV.

54x17mm (600 x 600 DPI)

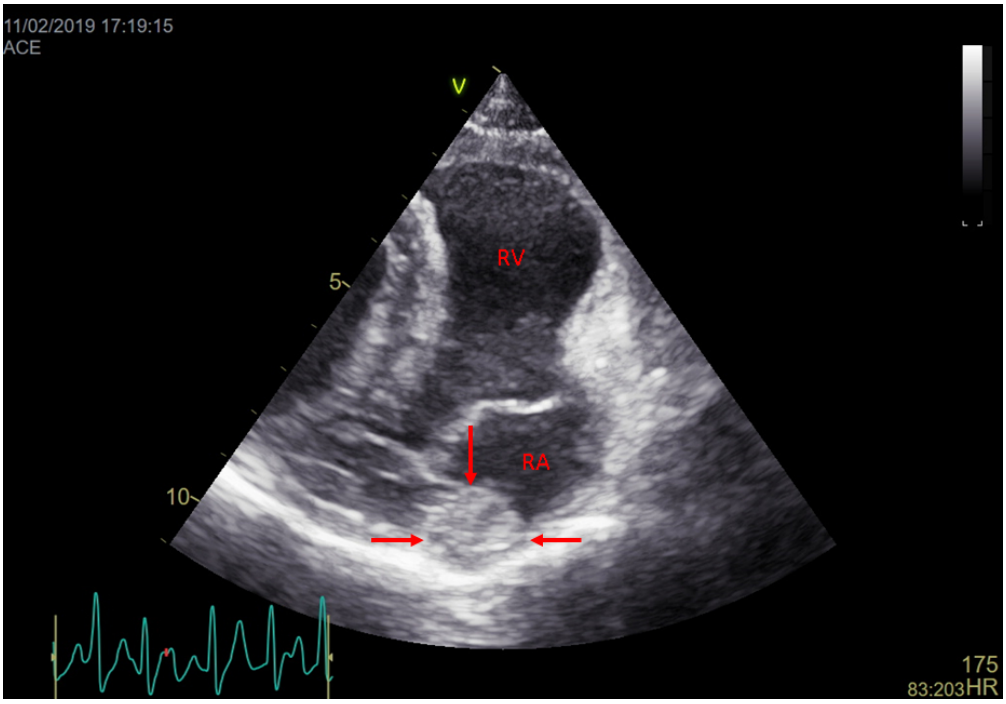

Figure 3 Echocardiographic images of the right atrial mass (arrows) from a non-standard left apical 4 chamber view optimized for the right heart (moved cranially with the left heart on the left of the image). The mass is showed here in the dorsal aspect of the right atrium.

39x27mm (600 x 600 DPI)

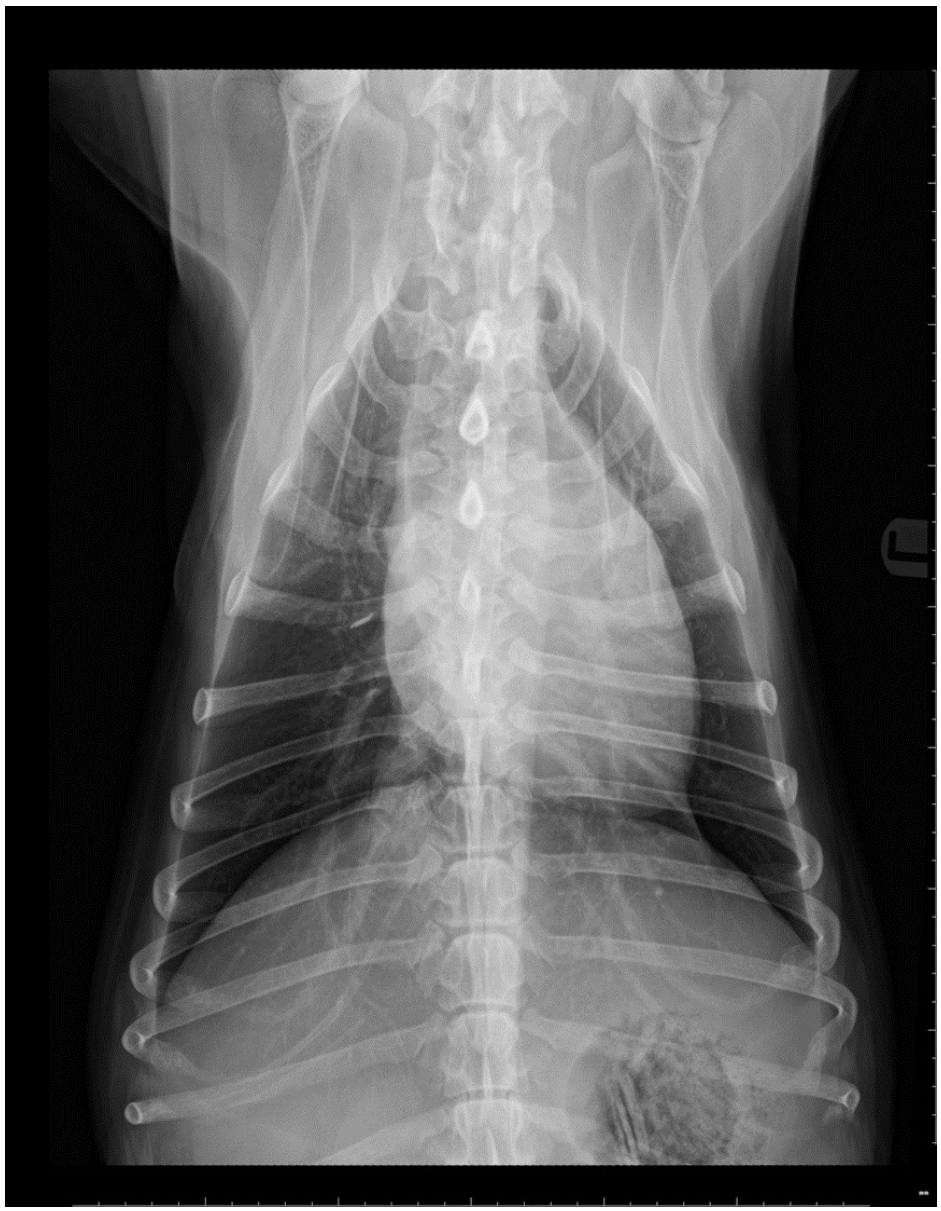

Figure 4 Dorsoventral radiograph at presentation, not showing obvious signs of pulmonary oedema or masses. The cardiac silhouette is within normal limits as well as the lobar vessels.

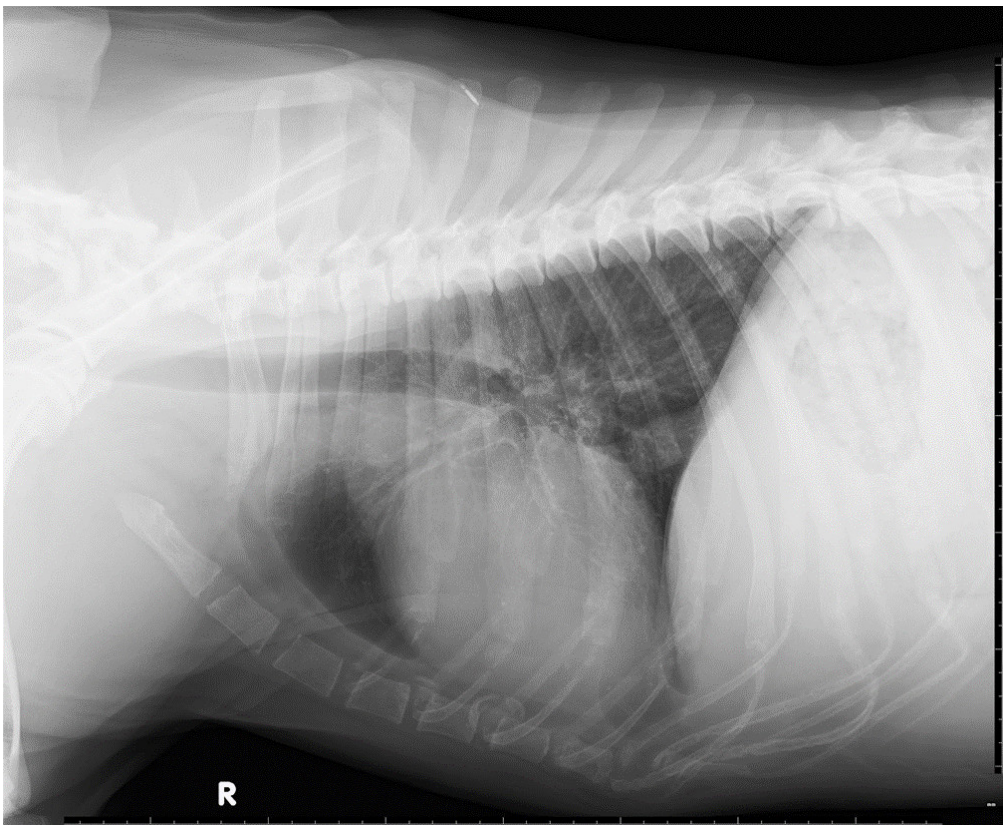

Figure 5 Right lateral radiograph at presentation, not showing obvious signs of pulmonary oedema or masses. The cardiac silhouette is within normal limits as well as the lobar vessels.

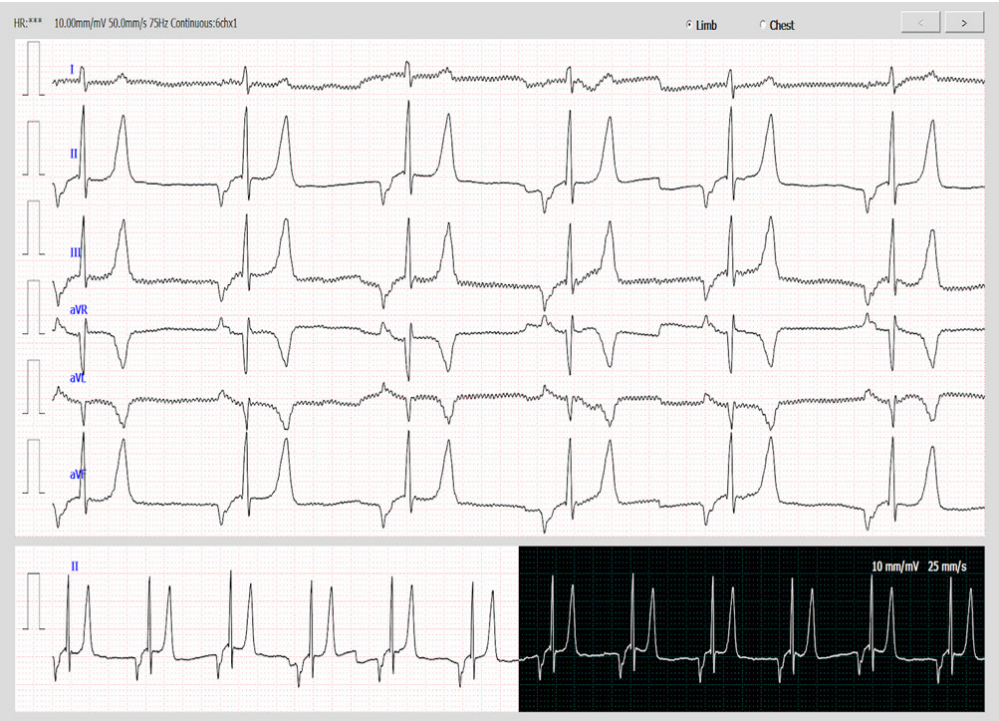

Figure 6 ECG trace 24 hours after admission and therapy with diltiazem, initially at 2 mg/kg PO, then at 3 mg/kg every 8 hours. Note the regular rhythm at a rate of 70 bpm with negative, prolonged (0.06s; ref. <0.04) and notched P' wave and tall T waves (similar height of R wave). Considering the P' waves are negative in lead II, III and aVF, similar amplitude positive P' in leads aVR and aVL with isoelectric P' in lead I, this ECG is compatible with a retrograde concentric atrial activation. This would be consistent with a junctional rhythm with retrograde activation of the atria and later normal His-Purkinje activation of the ventricles. There is mild increase in QT interval (0.28s; ref 0.15-0.25). Note: electrical interference affecting baseline of leads I, III and aVL (likely poor contact for left fore electrode attachment). However, this does not affect interpretation of the ECG. 50mm/s; 10mm/mV. The 50 mm/s six lead trace is represented as the first half of the bottom trace (lead II; 25 mm/s).

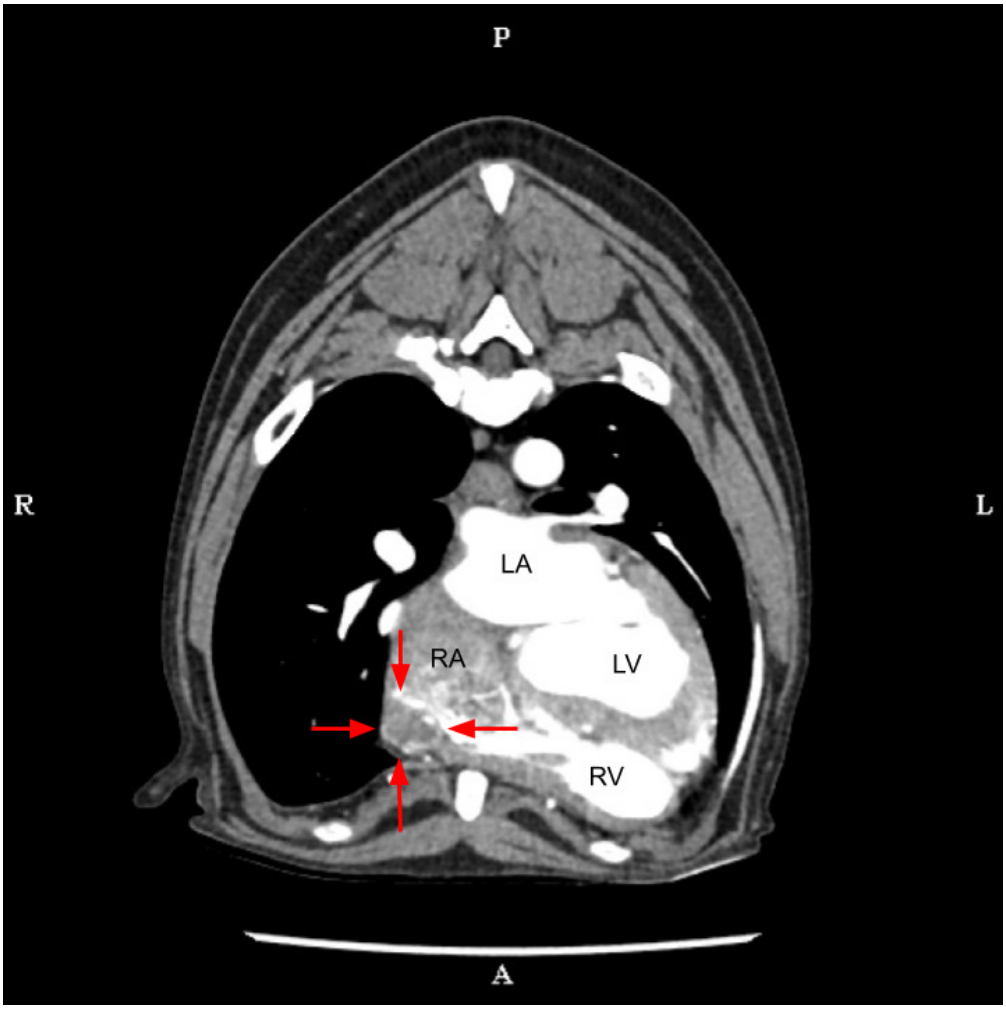

Figure 7 CT image (post-contrast, arterial phase, soft tissue window) of the right atrial mass (arrows). RA: right atrium; RV: right ventricle; LA: left atrium; LV: left ventricle.

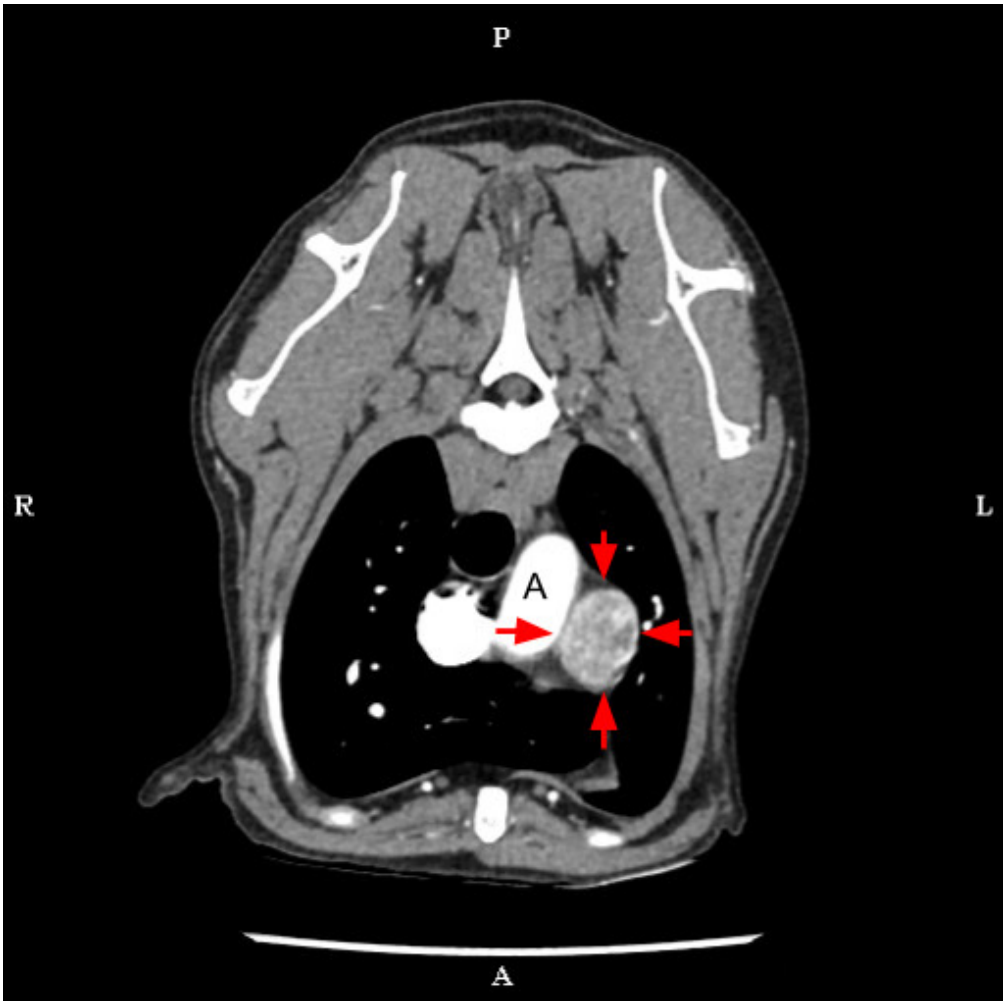

Figure 8 CT image (post-contrast, arterial phase, soft tissue window) of the heart base mass (arrows). A: aorta.

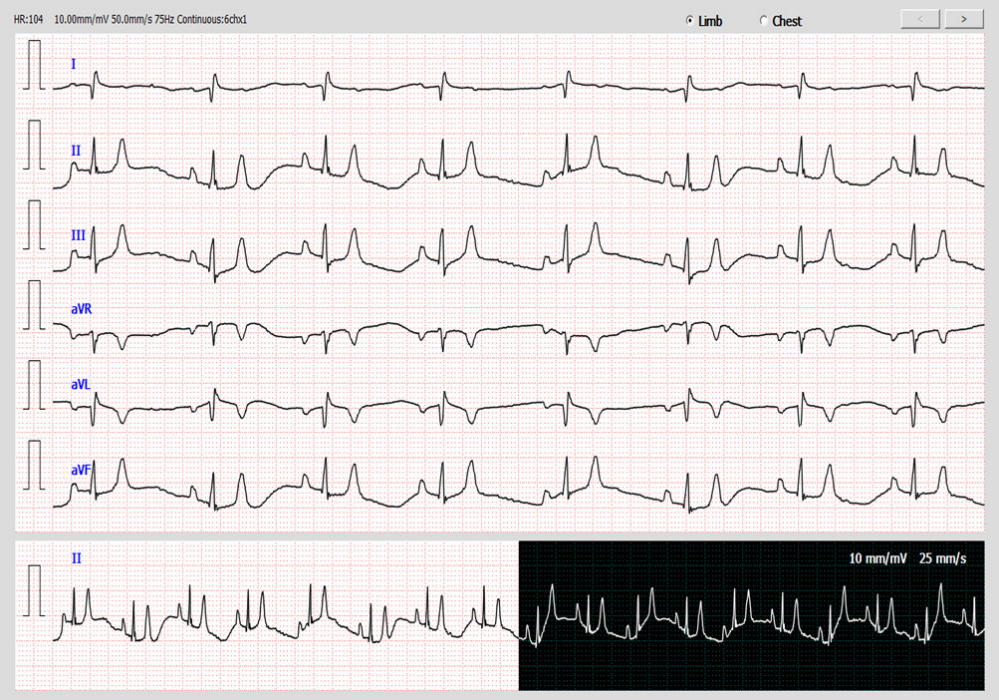

Figure 9 ECG trace 2 weeks after presentation showing sinus rhythm at a rate of 88bpm with P mitrale (0.05s; ref. <0.04) and notched QRS.
50mm/s; 10mm/mV. Baseline artefact associated with respiratory movement.

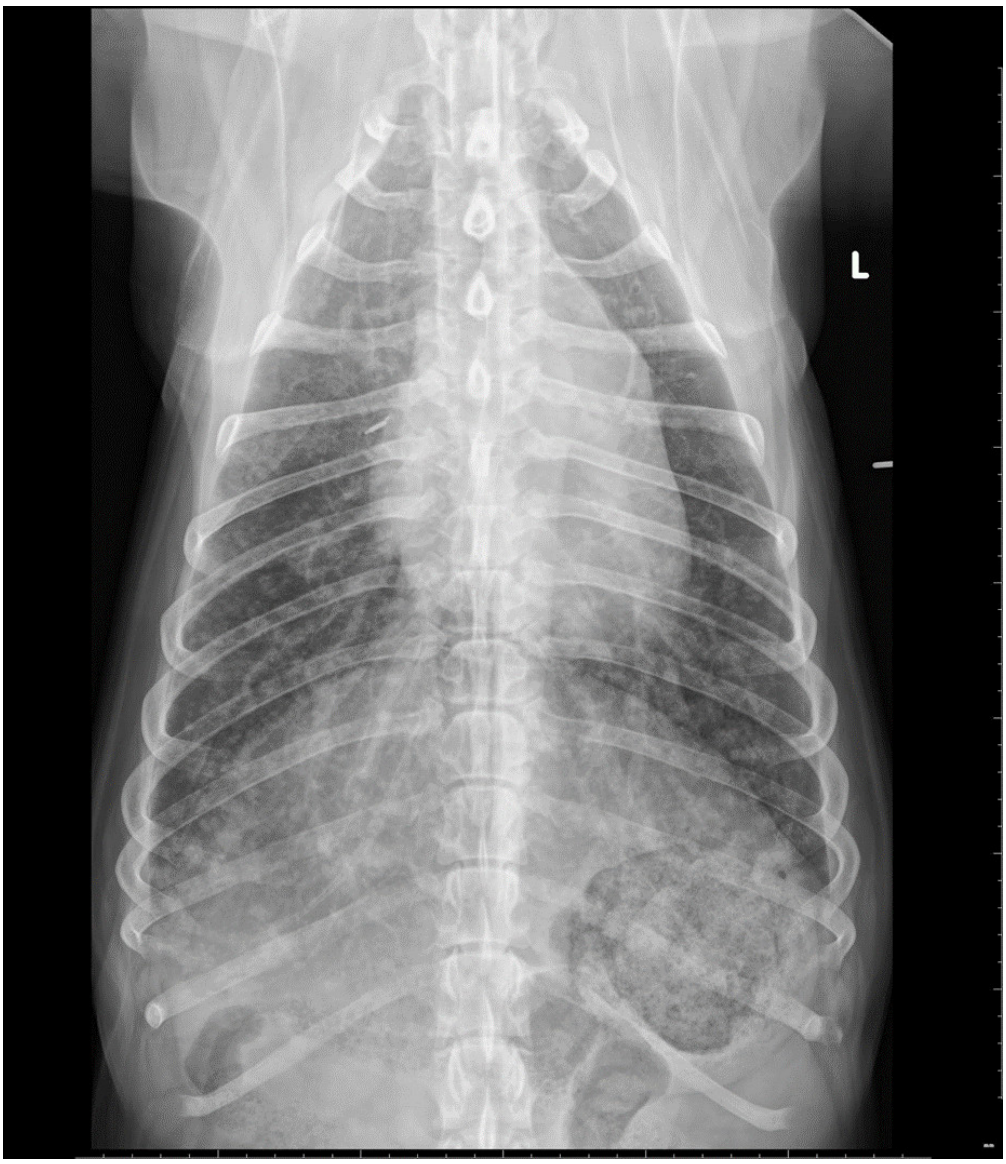

Figure 10 Dorsoventral radiograph at the 2 month recheck. Note the multifocal nodules in the lung parenchyma suspected to be metastatic disease.

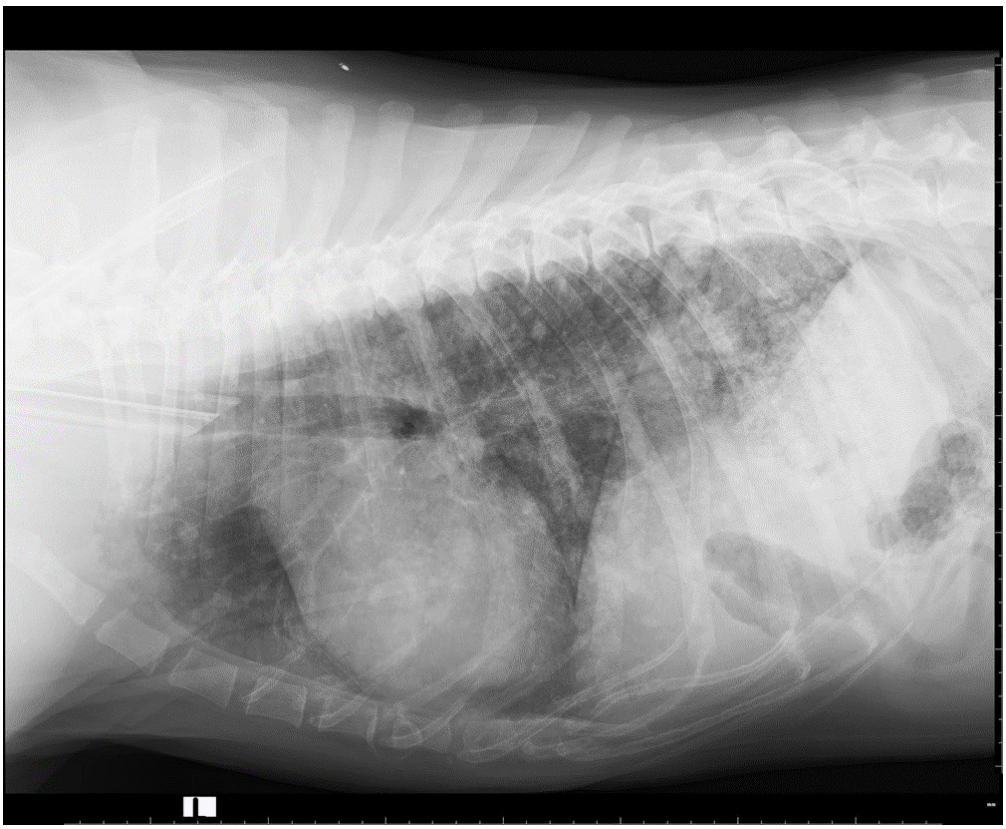

Figure 11 Right lateral radiograph at the 2 month recheck. Note the multifocal nodules in the lung parenchyma suspected to be metastatic disease.

Appendix 1: Peritoneal fluid cytology report

	Value
Fluid appearance	Slightly cloudy; pale orange
Fluid nucleated cell count (x 10^9/L)	0.59
Fluid red cell count (x10^{12}/L)	0.02
Hct fluid (%)	0
Fluid protein (g/L)	37
Fluid albumin (g/L)	18
Fluid globulin (g/L)	19

Appendix 2: Venous blood gas analysis at presentation and 24 hours after showing hypernatraemia (154 mmol/L; ref. 139-150) and bicarbonate was elevated at 32 mmol/L (ref. 15-23 mmol/L) as well as the creatinine at 148 µmol/L (ref. 44-115; prev. 104). Hypokalaemia is likely to be associated with furosemide administration and creatinine levels can increase due to mild azotaemia following diuretic therapy.
Na^+: sodium; K^+: potassium; Cl^-: chloride; Ca^{++}: calcium; Hct: haematocrit; Hgb: haemoglobin; HCO_3^-: bicarbonate.

Variable (units)	At presentation	24h after presentation	Reference Range
Na^+ (mmol/L)	149	154	139-150
K^+ (mmol/L)	4.4	3.3	3.4-4.9
Cl^- (mmol/L)	120.0	109.0	109-122
Ca^{++} (mmol/L)	1.3	1.29	1.26-1.39
Hct (%)	36	30	35-50
Hgb (g/dL)	12.3	10.3	12-17
Creatinine (µmol/L)	135	148	44-115
Lactate (mmol/L)	1.79	0.99	<2.5
Glucose (mmol/L)	5.2	5.2	4.1-5.5
HCO_3^- (mmol/L)	18.7	32.3	15-23
pH	7.354	7.431	7.35 – 7.45

Appendix 3: Renal biochemistry 2 weeks after presentation. Abbreviations in appendix 2.

Variable (units)		Reference Range
Na^+ (mmol/L)	156	144-160
K^+ (mmol/L)	4.0	3.5-5.8
Cl^- (mmol/L)	113.0	109-122
Urea (mmol/L)	13.1	2.5-9.6
Creatinine (µmol/L)	135	44-159

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TITLE OF CASE ***Do not include "a case report"***

Right-sided congestive heart failure secondary to supraventricular tachycardia in a dog with a right atrial mass

SUMMARY ***Up to 150 words summarising the case presentation and outcome (this will be freely available online) - 149***

A dog was referred to the authors' hospital for further investigations of pelvic limb collapsing episodes. Physical examination revealed a positive hepatojugular reflux, positive fluid thrill on abdominal palpation and an irregular heart rhythm (144 bpm) with pulse deficits. A 6-lead ECG showed focal atrial tachycardia (FAT). Doppler echocardiography revealed systolic dysfunction, dilated cardiomyopathy (DCM) phenotype and the presence of a heterogeneous mass in the right atrium; this was confirmed by a CT study. Free abdominal fluid was detected, sampled and analysed: this was consistent with modified transudate secondary to right-sided congestive heart failure (R-CHF). The dog responded well to heart failure and anti-arrhythmic medications. He was presented 8 weeks later after development of respiratory signs (cough). Investigations revealed stable cardiac disease but several radiopaque nodules within the lung parenchyma compatible with metastatic disease. Ten weeks after presentation the dog was euthanized due to worsening of the respiratory signs.

BACKGROUND ***Why you think this case is important – why did you write it up?***

This case report discusses investigations of a frequent and non-specific clinical sign (pelvic limb collapse) in an elderly dog with severe and complicated underlying cardiac disease. This highlights the importance of performing a thorough clinical examination, in this case focusing on auscultation and femoral pulse palpation, and the importance of further investigations including electrocardiography and echocardiography. Moreover, it provides insight into investigations, causes and treatment of supraventricular tachycardias (SVT) since these can be a challenge for many practitioners. It is also an example of how co-

morbidities can occur (i.e. laryngeal paralysis) making confirming a final diagnosis more challenging.

CASE PRESENTATION ***Presenting features, clinical and environmental history***

A 12-year-old male neutered Labrador Retriever was referred for investigations of collapsing episodes which started 2-3 days before presentation. These episodes were characterized by episodic hind limb weakness without loss of consciousness; they were not related to exercise although the dog was reported to be lethargic. The primary veterinarian's assessment reported a heart rate of 180 bpm with an irregular rhythm and a pulse rate of 70 ppm with variable pulse quality. There was no history of toxin ingestion, the patient was up to date with vaccinations, ecto- and endo-parasite prevention and he had never travelled abroad.

At presentation, the dog was quiet, alert and responsive, mucous membranes were pale and moist with a delayed capillary refill time (CRT) of 3 seconds. The heart rate was 144bpm with an irregular rhythm; femoral pulses were of variable quality and pulse deficits were evident. No heart murmur was detected, and pulmonary auscultation was unremarkable, although the dog was panting incessantly. There was minor upper respiratory tract noise that was more obvious during excitement. The abdomen was distended with a fluid thrill on ballottement, but no other abnormalities were detected on abdominal palpation. The jugular veins were not obviously distended, but a positive hepatojugular reflux (HJR) was elicited. He weighed 34.2 kg, body condition score 3/9. The rest of the physical examination was unremarkable.

INVESTIGATIONS ***If relevant***

The systolic arterial blood pressure (Doppler method) was 140 mmHg. A six-lead electrocardiogram (ECG) was recorded and showed the presence of an irregular tachyarrhythmia of supraventricular origin (supraventricular tachycardia; SVT) interrupted by occasional sinus complexes (figure 1, figure 2). The heart rate was 220-250 bpm. P' waves could be observed with varying P'Q intervals, which were positive in leads I, II and aVF, and negative in lead aVR, consistent with dorsal right atrial origin. Electrical alternans was also present. The RP'/P'R interval ratio was approximately 2, although there was considerable variation in this over the trace. This was consistent with a focal atrial tachycardia (FAT). The ladder diagram (figure 2) shows a trend to increasing P' rate prior to the AV block, and the variable slope showing conduction across the AV node indicates progressive slowing prior to the AV block, reflecting the physiological decremental conduction properties of the AV node at high atrial rates. When increasingly rapid atrial impulses are generated and reach the atrioventricular node, the conduction through this is decreased resulting in a slower ventricular rate.

Echocardiography revealed the presence of a right atrial mass at the dorsal aspect of the right atrium (figure 3); this was not resulting in reduced flow from the venae cavae demonstrated by colour Doppler. It was heterogeneous and measured 2.13 x 1.62cm. There was subjective dilation of the right ventricle and moderate right atrial enlargement. Left ventricular (LV) systolic function was mildly impaired (ejection fraction calculated with Simpson's method of discs: 39%; ref >50%; LV end-systolic volume index 42.2ml/m^2, ref <30ml/m^2) (1) with a rounded but not significantly dilated left ventricle and mildly increased left atrial size. These findings were compatible with myocardial dysfunction and, given the SVT, tachycardia-induced cardiomyopathy (TICM).

Abdominocentesis was performed and analysis confirmed a protein-rich modified transudate, consistent with ascites associated with right-sided CHF (appendix 1). Cardiac Troponin I was markedly elevated at 3.07ng/mL (ref <0.15ng/mL) indicating current or recent cardiomyocyte injury. The thyroxine hormone was normal at 38.9nmol/L (ref. 5-44) consistent with euthyroidism (2). A venous blood gas analysis revealed mild increase in creatinine (appendix 2).

Thoracic radiographs were also performed and did not show any significant abnormality (figure 4, figure 5).

The following day following treatment (see later), the patient was stable and further investigations were performed. An ECG was repeated and it showed a regular rhythm at a rate of 70 bpm and a negative, prolonged and notched P' wave (0.06s; ref. <0.04) in leads II, III and aVF and tall T waves (figure 6). The QRS complexes were within normal limits. These findings were compatible with an idio-junctional rhythm with retrograde concentric atrial activation.
CT scan with contrast (Ioversol, Optiray, Guerbet; arterial phase triggered by presence of contrast in the ascending aorta) of the thorax and abdomen was performed under sedation and confirmed the presence of a nodule in the right atrium (2.1x1.9 x 1.7cm); it also detected a larger mass (2.9x2.1x2cm), more cranial, adjacent to the ascending aorta at the base of the heart (figure 7; figure 8), which had not been detected during echocardiography.
A venous blood gas analysis was repeated (appendix 2) showing hypernatraemia and high bicarbonate and creatinine. Hypokalaemia was likely to be associated with furosemide administration and creatinine levels can increase due to mild azotaemia following diuretic therapy.

DIFFERENTIAL DIAGNOSIS *If relevant*

In an elderly dog presenting with episodic pelvic limb collapse which is not associated with tonic-clonic movements or loss of consciousness there are a significant number of differential diagnoses that need to be considered.

The presence of pale mucous membranes with delayed CRT, a tachyarrhythmia with pulse deficits, positive hepatojugular reflux and abdominal fluid thrill would suggest that a cardiac cause is likely; it is indeed well known that both bradyarrhythmias and tachyarrhythmias can induce collapse. Amongst tachyarrhythmias, supraventricular tachycardias (SVTs), atrial fibrillation or flutter and ventricular tachycardias, should be taken into consideration. These may be associated with transient loss of consciousness, which may also result in possible urination / defaecation. The recovery time is usually quick. The underlying mechanism is cerebral hypoxia secondary to decreased cardiac output. If persistent, tachyarrhythmias can lead to a DCM phenotype called TICM characterized by chamber dilation and systolic dysfunction.
Structural heart disease with impaired systolic function would lead to a decreased cardiac output and subsequent cerebral hypoxia. Amongst these, in a large breed dog we could consider DCM and valvular defects (i.e. aortic stenosis, pulmonic stenosis). The latter are less likely in this case considering the absence of an audible murmur. Cardiac output would be reduced in case of pericardial effusion; this is easily excluded on echocardiography. Pulmonary hypertension can also be responsible for similar episodes and echocardiography can be used to rule this out.
A delayed CRT and pale mucous membranes would also support a cardiovascular problem (i.e. shock, systolic failure, impaired cardiac output). Anaemia can mimic this sign and should be excluded.
Abdominal distension, a positive fluid thrill and positive hepatojugular reflux are signs compatible with increased central venous pressure that can be secondary to right-sided CHF or cardiac tamponade; the presence of the hepatojugular reflux indicates that the caudal vena cava and cranial vena cava are connected via the right atrium, so these signs are unlikely to be the consequence of obstruction to one or the other (e.g. Budd-Chiari causes of ascites, or evidence of cranial caval syndrome). Tricuspid dysplasia is a heritable disorder in Labrador Retrievers and can lead to right-sided CHF. Other causes of increased central venous pressure can be related to cardiac tamponade, TICM or DCM.

In cases of hypoalbuminaemia, ascites can be detected (a biochemistry panel can rule this out) and internal bleeding (i.e. haemoabdomen) can give both abdominal distension and collapse, if associated with acute blood loss (which would also increase the heart rate). These conditions are not associated with a concurrent positive hepatojugular reflux; the combination of these findings indicates R-CHF.

Orthopaedic or neurological issues such as osteoarthritis, intervertebral disc disease, other spinal disease, cruciate disease or neoplastic process can lead to pain and pelvic limbs weakness with episodic collapse. Usually, these are associated with orthopaedic pain or abnormalities on neurological examination; these were not present in this case.

Atypical seizures could not be ruled-out although are less likely considering the other physical examination findings and the rapid recovery with no post-ictal signs.

TREATMENT *If relevant*

The patient was hospitalized and intravenous furosemide therapy was initiated at a rate of 2 mg/kg IV every 8 hours (Dimazon, Intervet) for 48 hours to control the right-sided heart failure; afterwards, oral administration at the same dosage was commenced. To address the SVT, diltiazem (modified release) was started (2 mg/kg every 8 hours, PO; Crescent Pharma Ltd). Pimobendan was also administered intravenously initially (0.15 mg/kg) and then orally after 12 hours (0.23 mg/kg every 12 hours; Boehringer Ingelheim GmbH). ECG telemetry was used to monitor the patient overnight. After 12 hours, the heart rate was not yet controlled, therefore, the diltiazem dose was increased to 3 mg/kg PO every 8 hours and good rate control was achieved (120-140 bpm).
The patient was discharged on oral medications 48 hours after admission (diltiazem dose: 3 mg/kg PO q8h). Abdominal distension had resolved, the heart rate was 120 bpm with regular rhythm and he had pink mucous membranes with a CRT <2 seconds. The body weight was 30kg with a 4kg weight loss since admission, associated with loss of his abdominal effusion.

OUTCOME AND FOLLOW-UP

The patient was re-checked 2 weeks later. No exercise intolerance or collapsing episodes were reported. His weight was 31.2 kg with a BCS of 4/9. The heart rate was 100 bpm with a regular rhythm and synchronous femoral pulses of good quality. No fluid thrill was detected. Serum biochemistry showed mild elevation of urea (13.1 mmol/L; ref. 2.5-9.6). Creatinine and electrolytes were within normal limits. Cardiac Troponin I was 0.3 ng/mL (ref <0.15ng/mL). A 6-lead ECG showed sinus rhythm with a rate of 88 bpm and evidence of P mitrale (P duration 0.05 s (ref. <0.04), suggestive of left atrial enlargement (figure 9). Echocardiography was repeated showing marked improvement in systolic function with an ejection fraction of 64.7% (prev. 38%; ref. <50%) (1) and end-systolic volume index of 17 ml/m^2 (prev. 42.2; ref. <30ml/m^2) (1) and normal right and left atrial size; the right ventricle was still dilated, similarly to the previous echocardiography. These findings, showing reverse remodelling and improved systolic function with management of the SVT, were consistent with previous diagnosis of TICM.
A 24-hour ambulatory ECG recording (Holter monitoring) showed predominantly sinus rhythm and sinus arrhythmia with occasional paroxysms of SVT (associated with stress or excitement).
Two months after presentation, the dog was re-assessed due to the onset of a dry cough of two week's duration, which was worse during exercise. He was quiet but alert and the heart rate was 100 bpm with regular rhythm and strong, synchronous femoral pulses. Pulmonary auscultation was unremarkable although loud upper respiratory stridor was noted. Lymph nodes were normal in size. Tracheal pinch test was negative. Rectal temperature was 38.5° C.
The dog was admitted for further investigations. Haematology was unremarkable. Renal biochemistry showed mildly elevated urea and normal creatinine (appendix 3). Echocardiography did not show significant changes, the right atrial mass and the systolic function were similar to last time. Thoracic radiographs were repeated and showed several radiopaque nodules (0.4-1.5cm) within the lung parenchyma (figure 10; figure 11). Sampling of the nodules was not possible as they were not accessible via the thoracic wall. Laryngeal function was also assessed under deep sedation, which showed bilateral laryngeal paralysis. The owner declined further investigations.
Ten weeks after initial presentation the dog was euthanized at the referring practice due to deterioration of the cough and respiratory distress. Post-mortem examination was declined.

DISCUSSION *Include a very brief review of similar published cases*

The ECG at presentation showed a variable P'R (120-160ms) and a long RP' (±240ms) segment (RP'/P'R=2), a positive P' in lead II and aVF, compatible with FAT with occasional 2nd degree AV block. Focal atrial tachycardia is characterized by narrow QRS complexes,

fast ventricular rates (210-330 bpm) and a mostly regular RR interval although 2nd degree AV block and cycle length irregularity are common. A long RP' interval suggests the P' wave is triggering the following QRS complex (atrial origin of the tachycardia), whereas a short RP' is typical of retrograde activation most commonly seen in atrioventricular reciprocating tachycardia (AVRT) and junctional rhythms. AVRT share some electrocardiographic features with FAT (narrow QRS complexes, regular RR interval) but are associated with the presence of muscle bundles (accessory pathways) that create a direct connection between atria and ventricles, bypassing the AV node. Although Labradors are predisposed to AVRT triggered by accessory pathways (3), the presence of AV block confirms this was an atrial tachycardia, independent of the AV node. In the case of AVRT an AV block (with retrograde atrial activation and a short R-P') will interrupt the re-entry circuit and end the tachycardia. Cycle length irregularity, as showed in the ECG at presentation, is also common in FAT (4). Labrador retrievers are reported to be predisposed to FAT and often the ectopic focus is distributed within the right atrium (5). However, there were some characteristics of re-entrant tachycardia such as electrical alternans that can be attributed to nonspecific intraventricular conduction abnormalities (6). Labrador Retrievers have also been reported to be affected by isorhythmic atrioventricular dissociation (IAVD) and accelerated idiojunctional rhythm; IAVD is characterized by independent atrial and ventricular foci that generate impulses at a similar rate. We consider this less likely since the P' and QRS complexes remained associated in this patient (7) and the rate is excessive and rhythm too irregular for both the atrial and ventricular complexes.
However, a definitive diagnosis of FAT would require an electrophysiological study that was not performed in this case.
After initiation of antiarrhythmic therapy and successful rate control, the patient had reduced heart chamber size and improved systolic function, resolution of the ascites and a marked clinical improvement. Dogs paced at 180 bpm for 3 weeks have been shown to develop a TICM (8); 73% of people with FAT develop TICM (9). Remodelling after rate control has also been described with substantial regain of systolic function (10). Primary DCM was unlikely considering the absence of marked dilation and the reverse remodelling noted after appropriate rhythm control. Diltiazem is a calcium channel blocker which slows conduction in nodal tissue leading to its safe and effective use in supraventricular arrhythmias (11,12). It is possible that the pimobendan therapy is also responsible for some of the reverse remodelling observed at the recheck; the inodilator effect of this drug will improve contractility and reduce ventricular size in dogs with mitral valve disease and dilated cardiomyopathy (13,14).

The patient showed a different rhythm after 24 hours of diltiazem therapy with a junctional rhythm and concentric retrograde atrial activation preceding the QRS complexes. It is possible that diltiazem suppressed both the ectopic focus and sinus node, leading to the junctional rhythm and retrograde activation of the atria from the junctional focus; atrial depolarization was earlier than ventricular depolarization suggesting the junctional focus was atrionodal and ventricular activation followed conduction through the compact AV node. It is also possible that the sino-atrial node had been suppressed by the ectopic focus during the long period of supraventricular tachycardia; this would have allowed the junctional rhythm to manifest (15).
It is possible the tachyarrhythmia was triggered directly by the presence of a mass in the right atrium; although this could not be confirmed, it is reported in people (16).
Based on location and appearance, the right atrial mass was speculated to be a hemangiosarcoma, whereas the heart base mass might be more consistent with a chemodectoma; histological confirmation was not possible since sampling was considered dangerous and the client declined necropsy. Haemangiosarcomas are the most common cardiac tumour identified in dogs and aortic body tumours are also frequently reported, especially in brachycephalic breeds. (17) Dogs with right atrial haemangiosarcomas have been reported to suffer from right-sided CHF. (18) It is unclear if the pulmonary nodules noted on the subsequent radiographs were associated with metastatic spread of the right atrial mass, but it is reported that haemangiosarcomas can metastasize to the lungs (19). Given the location in the thorax and the likelihood of bleeding during sampling, aspiration of the pulmonary nodules for cytology samples was not performed.

The arrhythmia did not induce loss of consciousness but hindlimb weakness that are more commonly attributed to neurological or orthopaedic issues. The most likely explanation for this clinical sign is a near-fainting episode also described in people suffering from severe paroxysmal arrhythmias; these usually precede full collapses.

The cough could be related to the lung nodules, considering the extent of the lung disease. Laryngeal paralysis surgical approaches (i.e. laryngeal tie-back) and associated risks were discussed but declined by the client (20, 21) given the poor prognosis with the other co-morbidities.

LEARNING POINTS/TAKE HOME MESSAGES ***3 to 5 bullet points – this is a required field***

1) Tachyarrhythmias, if persistent, can cause a dilated cardiomyopathy phenotype with systolic dysfunction called tachycardia-induced cardiomyopathy (TICM), which may be reversible after good rate control. TICM can result in congestive heart failure.
2) Tachyarrhythmias can be caused by cardiac or systemic disease. Labradors are frequently affected by AVRT and FAT with or without underlying disease. Cardiac neoplasia could also potentially trigger arrhythmias.
3) R-P' and P'-R intervals are useful to distinguish FAT from AVRT. These conditions have different treatment options therefore recognizing them is important.
4) Cardiac collapse and syncope can sometimes be confused with those caused by orthopaedic (i.e. osteoarthritis) or neurological (i.e. idiopathic epilepsy) conditions. Cardiac auscultation and femoral pulses palpation are useful and inexpensive tools helpful in guiding further investigations.

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FIGURE/VIDEO CAPTIONS ***figures should NOT be embedded in this document***

Figure 1: ECG trace at presentation showing irregular supraventricular tachycardia at a rate of 220bpm with occasional sinus complexes.
P' waves can be observed during the periods of SVT with varying P'Q intervals. The P' waves are positive in lead I, II, III and aVF (superior-to-inferior axis) indicating their origin is likely from the right atrial roof. Electrical alternans and cycle length variability are also present. The P'R is variable (120-160ms) and R-P' long (240ms) that makes an atrial tachycardia more likely (R-P'/P'-R=2). The summation of P' on T waves increases their amplitude.
The ECG is compatible with focal atrial tachycardia with occasional 2nd degree AV block (see figure 2) and the presence of increased P wave duration (0.06s; ref. <0.04) (due to P mitrale or interatrial conduction disturbance).

50mm/s; 10mm/mV. The 50 mm/s six lead trace (main ECG; upper panel) is represented in the first half of the bottom trace (lead II rhythm strip; 25 mm/s).

Figure 2: Ladder diagram of the ECG trace at presentation. Close up of figure 1.
The black vertical lines indicate the conduction within the atria (top line), the conduction through the AV node (mid line) and the conduction through the ventricles (bottom line). If there is an AV block the bottom line will not be present since it will not reach the ventricles (i.e. blue lines).
There is a trend to increasing P' rate prior to the AV block, and the slope showing conduction across the AV node (between the first and second horizontal line in the ladder diagram) indicates progressive slowing prior to the AV block, reflecting the physiological decremental conduction properties of the AV node at high atrial rates.
In black the complexes originating from the ectopic focus (P'). In blue the P' waves that have been blocked (2nd degree atrioventricular block). In red the sinus complex. Lead I, II, III. 50mm/s; 10mm/mV.

Figure 3: Echocardiographic images of the right atrial mass (arrows) from a non-standard left apical 4 chamber view optimized for the right heart (moved cranially with the left heart on the left of the image). The mass is showed here in the dorsal aspect of the right atrium.

Figure 4: Dorsoventral radiograph at presentation, not showing obvious signs of pulmonary oedema or masses. The cardiac silhouette is within normal limits as well as the lobar vessels.

Figure 5: Right lateral radiograph at presentation, not showing obvious signs of pulmonary oedema or masses. The cardiac silhouette is within normal limits as well as the lobar vessels.

Figure 6: ECG trace 24 hours after admission and therapy with diltiazem, initially at 2 mg/kg PO, then at 3 mg/kg every 8 hours. Note the regular rhythm at a rate of 70 bpm with negative, prolonged (0.06s; ref. <0.04) and notched P' wave and tall T waves (similar height of R wave). Considering the P' waves are negative in lead II, III and aVF, similar amplitude positive P' in leads aVR and aVL with isoelectric P' in lead I, this ECG is compatible with a retrograde concentric atrial activation. This would be consistent with a junctional rhythm with retrograde activation of the atria and later normal His-Purkinje activation of the ventricles. There is mild increase in QT interval (0.28s; ref 0.15-0.25). Note: electrical interference affecting baseline of leads I, III and aVL (likely poor contact for left fore electrode attachment). However, this does not affect interpretation of the ECG.
50mm/s; 10mm/mV. The 50 mm/s six lead trace is represented as the first half of the bottom trace (lead II; 25 mm/s).

Figure 7: CT image (post-contrast, arterial phase, soft tissue window) of the right atrial mass (arrows). RA: right atrium; RV: right ventricle; LA: left atrium; LV: left ventricle.

Figure 8: CT image (post-contrast, arterial phase, soft tissue window) of the heart base mass (arrows). A: aorta.

Figure 9: ECG trace 2 weeks after presentation showing sinus rhythm at a rate of 88bpm with P mitrale (0.05s; ref. <0.04) and notched QRS.
50mm/s; 10mm/mV. Baseline artefact associated with respiratory movement.

Figure 10: Dorsoventral radiograph at the 2 month recheck. Note the multifocal nodules in the lung parenchyma suspected to be metastatic disease.

Figure 11: Right lateral radiograph at the 2 month recheck. Note the multifocal nodules in the lung parenchyma suspected to be metastatic disease.