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Case Report

Coronary arteriovenous malformation in a dog with a complex arrhythmia and hypothyroidism

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Abstract A four-year-old Japanese Akita was referred for investigation of lethargy, exercise intolerance, and an irregular heart rhythm. He was diagnosed with atrial fibrillation, a complex ventricular arrhythmia, and hypothyroidism. Echocardiography identified a nest of anomalous vessels surrounding the heart and shunting into the pulmonary artery. Computed tomography confirmed a coronary arteriovenous malformation consisting of a coronary-to-pulmonary arterial communication and an associated complex nest of tortuous vessels, which was thought to be an incidental finding. Clinical signs improved with levothyroxine and antiarrhythmic treatment. Describing an unusual coronary artery anomaly as well, this case serves as a reminder to critically evaluate the hemodynamic significance of structural cardiac disease and to screen for systemic disease in patients with arrhythmias.

Abbreviation: AF, atrial fibrillation; CAF, coronary artery fistula; CAVM, coronary arteriovenous malformation; CT, computed tomography; TTE, transthoracic echocardiography.

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A four-year-old, 45-kg, male intact Japanese Akita was referred to the Royal (Dick) School of Veterinary Studies for investigation of lethargy, exercise intolerance, and an irregular heart rhythm of approximately 3 weeks' duration. Partial clinical improvement was reported after initiation of oral pimobendan (0.1 mg/kg twice daily) a week prior. On presentation, the patient was bright and alert with an irregular heart rhythm, pulse deficits, and a heart rate of 104 beats/minute. No murmur was heard, but auscultation was made difficult by panting and thick haircoat. The remainder of the physical examination was unremarkable.

Six-lead electrocardiography performed in right lateral recumbency showed atrial fibrillation (AF) with a ventricular response rate of 150 beats/minute and occasional, single, monomorphic ventricular premature complexes with a left bundle branch block morphology occurring at fixed coupling intervals of 325 msec from the preceding QRS complex. Transthoracic echocardiography (TTE) was performed by a board-certified cardiologist (Y.M.P.) and supervised cardiology residents (R.B. and G.S.) under sedation with intramuscular acepromazine (0.01 mg/kg) and butorphanol (0.3 mg/kg). This revealed mild left ventricular dilation (M-mode left ventricular internal dimension at end-diastole 6.06 cm; reference range 3.89–5.67 cm [1], 2D left apical 4-chamber Simpsons method of discs-derived end-diastolic volume index 65.23 ml/m^2; reference range 49–93 ml/m^2 [2]) with mildly reduced systolic function (M-mode left ventricular internal dimension at end-systole 4.4 cm; reference 2.35–4.18 cm [1], 2D left apical 4-chamber Simpsons method of discs-derived end-systolic volume index 57.9 ml/m^2; reference range 22–50 ml/m^2 [2], left ventricular ratio of pre-ejection period to ejection time 0.53; reference range 0.377 $\pm$ 0.075 [3], ejection fraction 35.79%; reference range 49 $\pm$ 7% [2], fractional shortening 33.33%; reference range 23–47% [1]). Left atrial size was normal (2D short axis left atrial to aortic ratio 1.34; reference range 0.86–1.57 [4]). Diastolic function was difficult to assess due to the presence of AF, but isovolumic relaxation time (53.42 ms; reference range 43–63 ms), deceleration time of early diastolic transmitral flow (94.55 ms; reference range 54–110 ms), and septal E/E' (10.5; reference range 4–15.1) taken from an average of five cardiac cycles were all within normal limits [5], suggesting normal diastolic function. The origin of the right coronary artery was dilated and adjacent to this; on a right parasternal short-axis view, between the base of the aorta and the pulmonary artery, an additional blood vessel was identified containing turbulent blood flow on color Doppler examination (Fig. 1A). A nest of tortuous vessels was imaged adjacent to the right ventricular outflow tract on the right parasternal short-axis view (Fig. 1B) and adjacent to the left atrium on a right-parasternal long-axis view. The origin of these anomalous vessels could not be determined. On assessment of the main pulmonary artery, a jet of continuous blood flow, entering just distal to the pulmonic valve, was identified traveling in an antegrade direction within the lumen of the main pulmonary artery along its cranial wall (Fig. 1C). The jet had a velocity of about 1.25 m/s in diastole and 0.75 m/s in systole. Routine blood work, urinalysis, and oscillometric blood pressure measurement (134/89 mmHg) were unremarkable. Cardiac troponin I concentration, measured on fresh heparinized blood using a commercially available point-of-care analyzer,[c] was mildly elevated at 0.08 ng/ml (reference range <0.05 ng/ml).

A 24-h ambulatory electrocardiographic recording showed sustained AF with a mean ventricular response rate of 117 beats/minute. Approximately 27,000 ventricular premature complexes comprising couplets, triplets, trigeminy, and short runs of monomorphic ventricular tachycardia at a rate between 230 and 280 beats/minute were identified. Ventricular premature complexes had both right and left bundle branch block morphology. Given the ventricular arrhythmia with signs of malignancy and the presence of AF, treatment with amiodarone was recommended at a starting dose of 12.5 mg/kg twice daily. A thyroid function panel was submitted due to concern for amiodarone causing hypothyroidism. Pimobendan, which was already at a low dose, was discontinued as no clear indication for its continuation was identified.

The patient developed intolerable diarrhea and inappetence after the first four doses of amiodarone, which prompted withdrawal of the drug. After resolution of the gastrointestinal signs, sotalol was instituted at 0.45 mg/kg twice daily and increased to 0.9 mg/kg twice daily after five days.

Several days after amiodarone discontinuation, thyroid function tests returned consistent with hypothyroidism (total T4 <5 nmol/l; reference range 15–48 nmol/l, thyroid stimulating hormone 2.3 ng/ml; reference range 0–0.5 ng/ml, free T4 measurement by equilibrium dialysis 2.5 pmol/l; reference range 7–40 pmol/l). Levothyroxine was

[c] VetScan i-STAT® 1 Handheld Analyzer and VetScan i-STAT® Cardiac Troponin I (cTnI) cartridge; Abaxis, Union City, CA.

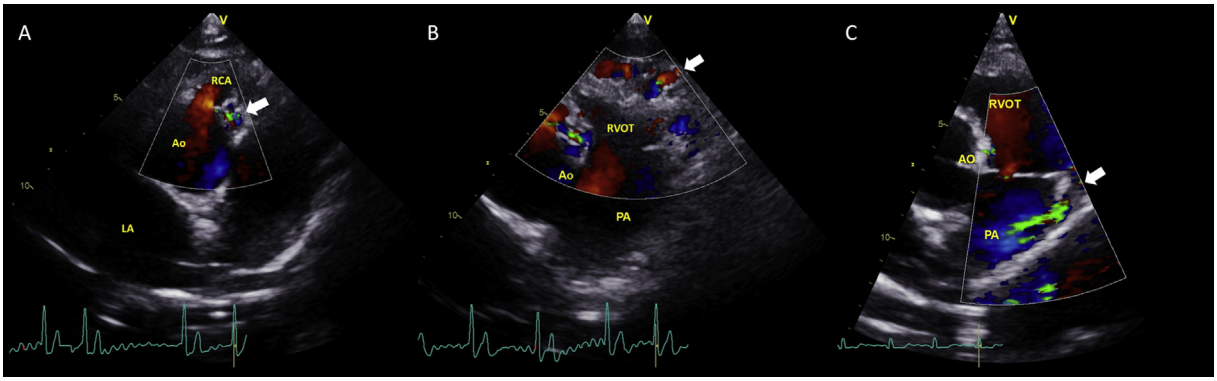

Fig. 1 Color-flow Doppler images from a right parasternal short-axis view showing A) turbulent flow within an aberrant vessel between the aorta and pulmonary artery, B) a nest of tortuous vessels adjacent to the right ventricular outflow tract, and C) a jet of antegrade flow entering the pulmonary artery distal to the pulmonic valve. Abbreviations: Ao, aorta; LA, left atrium; RCA, right coronary artery; PA, pulmonary artery; RVOT, right ventricular outflow tract.

prescribed at a low starting dose (9 mcg/kg once daily) and increased after a week to 18 mcg/kg once daily, as recommended for patients with concurrent cardiac disease [6].

To further investigate the etiology of the ventricular arrhythmia and to provide additional imaging of the anomalous vessels identified by TTE, an ECG-gated cardiac computed tomography (CT) scan of the thorax and late post-contrast phase CT of the abdomen were performed under general anesthesia a week after initial presentation. A multidetector 64-row CT scanner[d] was used in helical mode for prospective ECG-gated CT angiography. The region of interest was positioned in the ascending aorta. Images were acquired at 20% and 80% of the R—R interval. Scan settings included a spiral pitch factor of 0.2, collimation width of 0.6, tube potential of 80 kVp, reference tube current of 190 mA, slice thickness of 1 mm, matrix 512 × 512, and a low frequency reconstruction algorithm. Images were triggered to achieve optimally enhanced levophase after intravenous injection of contrast medium[e] in the right cephalic vein with a concentration of 800 mg iodine/kg (2 ml/kg) at a rate of 4 ml/s via a power injector system.[f] The examination revealed moderate distention of the left circumflex and right coronary artery (Fig. 2A), both of which had a markedly tortuous peripheral path. The left paraconal artery was mildly distended with a normal path. Multiple, small tortuous vessels were visible arising from the left circumflex coronary artery and extending adjacent to the wall of the left ventricle. Additional small, tortuous vessels were also visible originating from the right coronary artery and extending along the surface of the right atrium and left ventricle, as well as between the right coronary artery and the left auricle (Fig. 2B). Connections between these vessels and the adjacent structures were not clearly identified. However, at the level of the main pulmonary artery, just above the pulmonic valve, a 0.7-cm connection was visualized between the left circumflex coronary artery and the pulmonary artery, consistent with the jet identified on echocardiography (Figs. 2C and 3). A nest of vessels adjacent to the left auricle was also visible, but a clear origin or termination of these vessels was not identified. The coronary venous system did not appear dilated.

After 2 weeks of levothyroxine treatment, a further improvement in the patient's demeanor and activity level was reported, although total T4 remained low so the levothyroxine dose was increased to 27 mcg/kg once daily. Eight weeks after initial presentation, TTE was repeated, with minimal change noted. A 24-h ambulatory ECG recording confirmed persistence of AF with an average ventricular response rate of 120 beats/minute and a marked reduction in the frequency and complexity of the ventricular arrhythmia, with only 172 ventricular premature complexes identified.

Six months after initial presentation, the patient was symptom free and total T4 was within the normal range at a levothyroxine dose of 36 mcg/kg once daily. Atrial fibrillation was persistent with an average 24-h rate of 136 beats/minute, and the ventricular arrhythmia remained well controlled. Echocardiographic parameters remained unchanged.

[d] Somatom© Definition AS Siemens, Erlangen, Germany.
[e] Iopamir® Iopamidol; Bracco, Italy.
[f] Mark V Plus®; Medrad, UK.

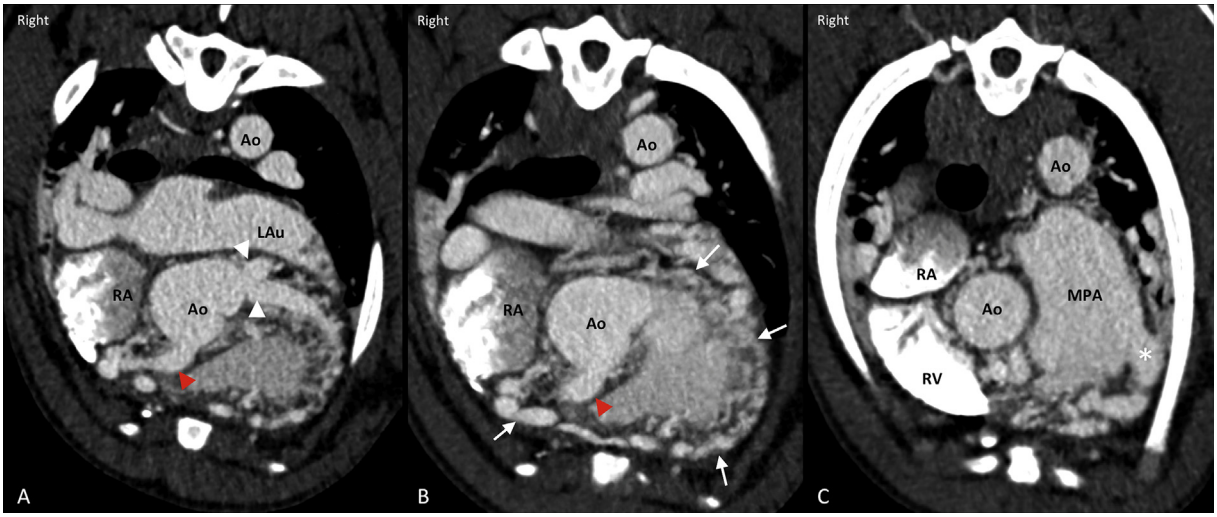

Fig. 2 Transverse CT images from caudal to cranial (A—C) at the level of the heart base during levophase. A) Arising from the aortic sinuses, the right (red arrowhead) and left (white arrowheads) coronary arteries connect to B) a multitude of tortuous vessels (arrows) which in turn C) form a single vessel feeding into the main pulmonary artery (asterisk). Images were acquired in diastole, and streamlining artefact is visible in the right atrium. Abbreviations: Ao, aorta; LAu, left auricle; RA, right atrium; RV, right ventricle; MPA, main pulmonary artery; CT, computed tomography.

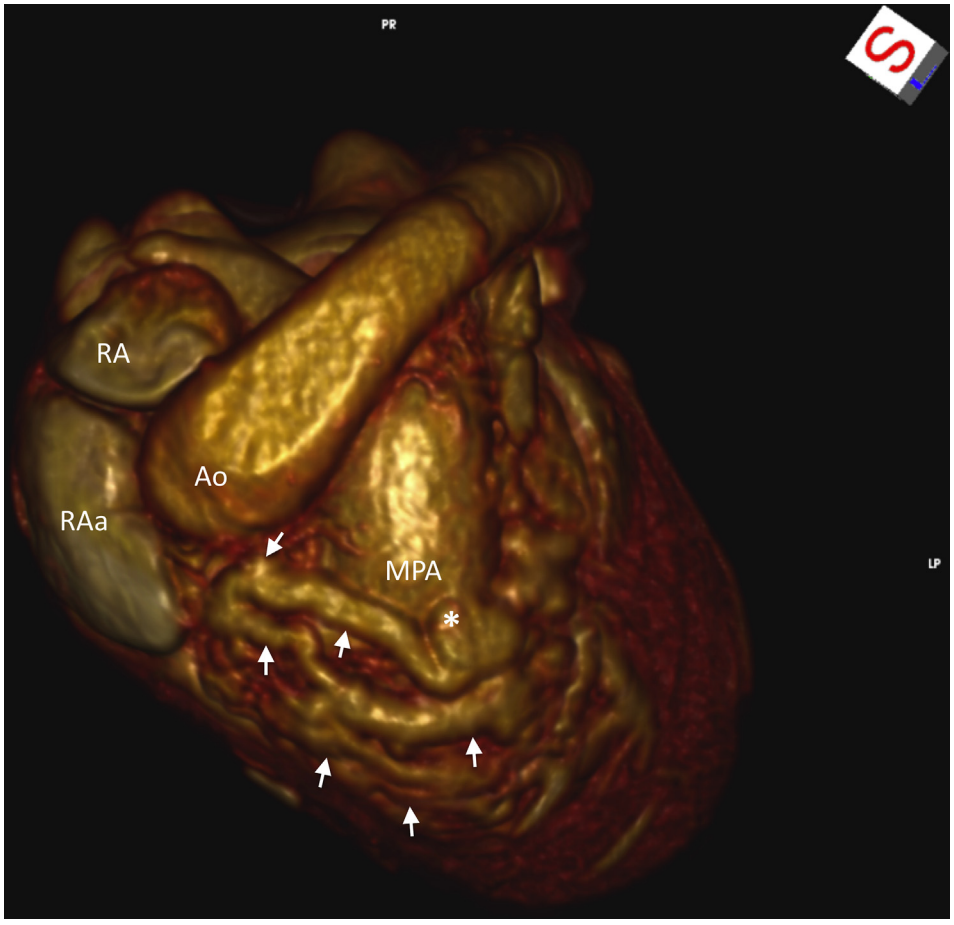

Fig. 3 Three-dimensional multiplanar reconstructed CT image of the heart from the cranial aspect showing the aberrant coronary vessel (arrows) feeding into the pulmonary artery (asterisk). Abbreviations: Ao, aorta; RA, right atrium; RAa, right auricular appendage; MPA, main pulmonary artery.

Discussion

This case report describes a dog with hypothyroidism and a complex arrhythmia, presenting with a coronary arteriovenous malformation consisting of a coronary-to-pulmonary arterial communication and an associated complex nest of tortuous vessels.

There are numerous classification systems in human medicine for describing coronary artery anomalies [7—10], and a distinction between coronary arteriovenous malformation (CAVM) and coronary artery fistula (CAF) is not made clear, with the terms often being used interchangeably [10—12]. When applied to hepatic, cerebral, and other systemic vessels, although definitions vary, the term fistula generally refers to a direct connection between an artery and vein and arteriovenous malformation refers to multiple connecting vessels forming a nest [13—15], but this does not currently appear to be widely applied to coronary artery anomalies. In humans, CAFs are most commonly single [16,17], although multiple CAFs have been reported [16,18], and there are some reports that are more consistent with CAVM in the true sense of the definition [11,19,20]. In dogs, reports consist of a single coronary to pulmonary fistula in a German shepherd dog [21] and multiple coronary to pulmonary fistulae in an English bulldog [22]. An observation was also made by Scansen [22] that coronary to pulmonary fistulas are more often seen in association with larger thoracic arteriovenous malformations. In the present case, the nest of tortuous vessels had a similar appearance to aortopulmonic vascular malformation described previously in the dog [23] and so a concurrent CAF and aortopulmonic vascular malformation were considered, but was not identified on echocardiography or CT.

Coronary angiography may have helped in further characterization of this lesion and is considered the gold standard in diagnosis of CAF in humans [24]. Transesophageal and 3D echocardiography, CT, and magnetic resonance imaging also prove useful, but TTE is rarely useful [17]. Additional imaging was not performed in this case as the coronary artery anomalies were thought to be incidental.

Coronary artery fistulas/CAVM in humans are rare and most often incidental and small, although medium- and large-sized malformations can occur and result in clinical signs [16]. Clinical signs can be related to the phenomenon called 'coronary steal' whereby coronary blood flow is directed through the low-resistance fistula instead of to the myocardium, resulting in fatigue, chest pain, and exertional dyspnea [16,17,24]. The clinical signs reported in this case were lethargy and exercise intolerance, which may have been related to the coronary steal phenomenon, but could also have been due to the development of AF, ventricular arrhythmias, or hypothyroidism. Given the improvement reported after levothyroxine treatment, the latter was thought more likely, although antiarrhythmic treatment may have also resulted in clinical improvement. The reason for the initial, partial improvement after pimobendan administration is unclear, but the dose was low and withdrawal did not result in deterioration, so the improvement is likely unrelated or may be explained by a placebo effect.

Thrombosis of these anomalous vessels in humans is uncommon but can also result in myocardial ischemia and therefore arrhythmias, angina, and systolic and diastolic dysfunction [17]. Recent thrombosis of the CAVM in this case was thought unlikely given the low cardiac troponin I levels and was also not identified on CT. However, the sensitivity of CT for this is low and coronary artery thrombosis cannot be completely ruled out. Alterations in myocardial perfusion due to coronary artery steal or thrombosis could be a cause for the arrhythmias and systolic dysfunction noted in this case.

Medium to large CAFs can also result in volume overload and chamber remodeling, which can lead to congestive heart failure and arrhythmias, particularly AF if there is atrial enlargement [16]. In the case described, mild left ventricular dilation and systolic dysfunction were identified. This could have been due to sedation, although was persistent on repeat echo when the dog was not sedated. Also, a previous study showed no effect of a similar sedation protocol (0.02 mg/kg of acepromazine and 0.2 mg/kg of butorphanol) on echocardiographic parameters of systolic function [25]. Other differentials considered in this case included preclinical dilated cardiomyopathy, tachycardia-induced cardiomyopathy, systolic dysfunction secondary to hypothyroidism, or breed variation. Volume overload caused by the CAVM was also considered a possibility and may have led to myocardial failure resulting in systolic dysfunction. As the echocardiographic changes were very mild, Qp:Qs was estimated to be 1.5 on echocardiography, and a range of other possible causes were identified; the hemodynamic significance of the CAVM was considered to be minor. It is possible that the accuracy of the Qp:Qs measurement was negatively affected by the presence of AF and so our decision was not based on this ratio alone.

In humans, American Heart Association/American College of Cardiology Congenial Heart Disease guidelines 2008 [26] recommend closure of CAFs if they are large or symptomatic, in which they include the presence of arrhythmias and systolic or diastolic dysfunction. Closure is also recommended if Qp:Qs is $>$ 1.5—1.7. These guidelines recommend rechecking asymptomatic patients with small CAFs every 3—5 years for the development of symptoms, arrhythmias, or chamber dilation. Guidelines for management of CAVM/CAF in dogs are not available, but cases of left-to-right shunting arteriovenous malformations with minimal hemodynamic significance have been described that have achieved a good long-term outcome without closure [27]. As the CAVM in our patient was considered to be hemodynamically inconsequential and given the good clinical response to medical management of the arrhythmia and concurrent hypothyroidism, closure of the anomalous vessels was deemed to be unnecessary at this point. However, as this patient's anomaly is unusual, the patient will need to be monitored for evolution of clinicopathological changes and closure needs to be considered if these progress.

This case was complicated by the diagnosis of hypothyroidism, which has the potential to cause many of the clinical, echocardiographic, and electrocardiographic abnormalities displayed by our patient. Atrial fibrillation in association with hypothyroidism has previously been reported in dogs [28—32], in one case converting to sinus rhythm after levothyroxine supplementation [30]. Three of the reported cases that remained in AF despite treatment for hypothyroidism were also reported to have dilated cardiomyopathy [31,32], which may have been the reason for persistent AF. It is also possible that the dilated cardiomyopathy phenotype was secondary to hypothyroidism. Similarly, in our patient, it is difficult to know whether AF was secondary to hypothyroidism or early dilated cardiomyopathy or was a true 'lone' AF. Although he has failed to convert to sinus rhythm after six months of treatment with levothyroxine, hypothyroidism may have been the triggering event for AF. Atrial fibrillation promotes the perpetuation of AF through electrophysiological changes in the atria [33]. As we do not know the duration of AF in this dog before presentation, resolution of AF may not be possible due to these secondary changes, even if the triggering event has resolved.

An association between hypothyroidism and ventricular arrhythmias has also been reported in humans [34,35], although rare. To the contrary, a recent study in Doberman pinschers with dilated cardiomyopathy showed no relationship between hypothyroidism and the number of ventricular premature beats [36]. An increased risk of atherosclerosis has been reported in dogs with hypothyroidism [37], so this cannot be ruled out as a possible cause of the ventricular arrhythmia identified in our patient, particularly given the marked improvement after levothyroxine supplementation, although this could also be attributed to antiarrhythmic treatment.

Hypothyroidism was only diagnosed in this patient because of screening before treatment with amiodarone. We suspect that his initial presentation was most likely related to this diagnosis, although we cannot rule out arrhythmia or CAVM as the cause of lethargy and exercise intolerance. This report therefore highlights the importance of screening for systemic disease in patients with arrhythmias. The coronary artery anomaly described is unusual in dogs, and, although interesting, it was most likely incidental. Therefore, this report also serves as a reminder to critically evaluate the hemodynamic significance of structural cardiac disease.

Conflict of interest statement

The authors do not have any conflicts of interest to disclose.

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