Dynamic Obstruction of an Anomalous Cavoazygos Vessel Associated with Interrupted Caudal Vena Cava in a Boxer Dog

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INTRODUCTION

Interrupted caudal (inferior) vena cava (CVC), also referred to as segmental aplasia of the CVC or azygos continuation of the CVC, is a rare congenital defect in the dog. It is often diagnosed incidentally either ante- or postmortem for unrelated conditions in veterinary medicine. Here we describe a case of a boxer dog with symptomatic interrupted CVC associated with intermittent dynamic obstruction of the anomalous cavoazygos vessel.

CASE PRESENTATION

An 11-month-old, intact male, boxer dog was presented to a veterinary teaching hospital for evaluation of a heart murmur and intermittent exercise intolerance. The dog had originally been presented to his primary care veterinarian 1 month prior for exhaustion and labored breathing after short walks. The patient had a concurrent medical history of polydipsia and intermittent vomiting. Two-view thoracic radiographs were performed by the primary care veterinarian (Figure 1) and revealed a prominent vascular structure within the mediastinum caudal to the aortic arch and an otherwise normal canine thorax. Lab work performed at that visit showed mildly elevated serum creatinine (1.4 mg/dL, [0.3−1.2 mg/dL]), mildly decreased alkaline phosphatase (44 U/L [46-337 U/L]), and elevated amylase (1,599 U/L [300−1,300 U/L]). A urine analysis showed a urine specific gravity of >1.050, supportive of mild prerenal azotemia. There was also mild proteinuria (30 mg/dL), pyuria (19 white blood cells per high-power field, 2 red blood cells per high-power field), and suspected bacteriuria with rods. A urine culture was not performed, and no therapeutic intervention was initiated prior to referral.

Upon presentation to the veterinary teaching hospital, salient physical exam findings included cryptorchidism and a grade 2/6 left basilar, systolic heart murmur with a regular rhythm and strong femoral pulses that were synchronous with the heart rate. The patient was placed on a padded mat into right lateral recumbency with gentle restraint by

trained veterinary technicians for baseline blood pressure and electrocardiogram (ECG; GE Healthcare Diagnostic ECG MAC 5500 HD, Chicago) measurement as routinely performed in new patients visiting the cardiology service of the teaching hospital. After a brief acclimation period, systolic Doppler blood pressure measurement of the left hind limb was 40 mm Hg, while in the left forelimb it was 125 mm Hg. A 10-lead ECG performed in right lateral recumbency following Doppler blood pressure measurement revealed a normal sinus rhythm with an average heart rate of 95 beats per minute and borderline prolonged PQ interval (130 msec [60−130 msec][1]) without criteria for chamber enlargement. After blood pressure measurement and ECG acquisition, the patient was allowed to stand and the physical exam was repeated due to the discrepancy in systemic blood pressures between limbs. At that time, it was found that the patient no longer had a heart murmur and that his femoral pulse quality had deteriorated to weak pulses that remained synchronous with a tachycardic heart rate.

The patient was subsequently placed into right lateral recumbency on a padded table for an echocardiographic exam. An echocardiogram (Philips iE33 Ultrasound System, Bothell, WA) was performed using multiple sector array transducers with frequencies ranging from 1 to 8 MHz and with standard, previously described imaging planes for dogs.[2] Images were stored digitally and analyzed remotely using commercially available software (Image Arena, TOMTEC, Chicago). Evaluation of cardiac chamber size and function was performed by subjective assessment as well as with the use of previously established canine reference intervals.[3] Both the right parasternal long-axis and short-axis views revealed small left and right ventricular chambers (Table 1, study 1) with suspected ventricular pseudohypertrophy as well as diminutive right and left atria (Figure 2A−C). There was global systolic dysfunction consisting of hypokinesis of the interventricular septum and dyskinesis of the left ventricle free wall (Video 1, left panel; Video 2, left panel). There was spontaneous echocontrast present in all cardiac chambers. The valvular structures appeared normal, and there was no significant valvular regurgitation present. Pulmonic valve outflow velocity was decreased (pulmonic valve Vmax 0.4 m/sec, [0.5-1.5][3]; Figure 2D). Prior to performing left parasternal echocardiographic views, abdominal structures were briefly evaluated with the same echocardiographic probe, which revealed a large, tortuous vascular structure cranial to the left kidney that exhibited spontaneous echocontrast and trivial pulsatile flow (Figure 3A). No free abdominal effusion was present. The patient became increasingly agitated at this time and was subsequently removed from the echocardiography table. Once standing, he began compulsively circling to the left with focus on his abdomen showing apparent discomfort and was visibly weak in the rear limbs. This episode resolved without intervention over the next 5 minutes, and the patient's demeanor completely normalized.

The patient was repositioned on the echocardiography table in left lateral recumbency. A repeat abdominal scan revealed reduction in the size of the large vascular structure with almost complete

disappearance over several minutes (Figure 3B and C). No free abdominal fluid was identified despite the disappearance of the abnormal structure. Upon obtaining left apical echocardiographic images, the dog had intermittent accelerated idioventricular rhythm with an average ventricular rate of 108 bpm for approximately the next 5 minutes of the exam (Figure 4A). The left apical views also revealed dramatic improvement in cardiac size and function compared to the previously obtained right parasternal views in right lateral recumbency (Figure 4B). High-normal transaortic valve flow velocity (Aortic Valve Vmax 1.7 m/sec)[3] was noted, as well as mild mitral regurgitation. A repeat study of the right parasternal views performed in right lateral recumbency confirmed improved cardiac chamber size and function (Figure 2E–G; Video 1, right panel; Video 2, right panel; Table 1, study 2) as well as mild mitral regurgitation from the right parasternal window that was not previously noted (Figure 2H). The Doppler blood pressure was repeated using the left hind limb prior to removing the patient from the echocardiography table, which indicated normalization of systemic blood pressure at 120 mm Hg (systolic). Simultaneously noted with the normalized blood pressure were strong femoral pulses and return of the soft left basilar systolic heart murmur.

To further investigate the dynamic echocardiographic findings and the abnormal vascular structure in the abdomen, a contrast-enhanced computed tomography (CT) study of the thorax and abdomen was performed using a 64-slice multidetector CT scanner (Toshiba Aquilion 64, Toshiba America Medical Systems, Tustin, CA) under sedation (butorphanol 0.3 mg/kg and acepromazine 0.01 mg/kg). The study was performed with the patient in sternal recumbency. The CT revealed that the CVC extended from the right atrium to the liver with normal hepatic and portal venous anatomy; however, immediately caudal to the liver, a discrete CVC was not detected. The right azygos vein was moderately dilated within the thorax and extended caudally through the right crus of the diaphragm lateral to the aortic hiatus where it connected to the large anomalous vessel. This cavoazygos vessel appeared constricted at the level of the diaphragm where it crossed into the abdomen (Figure 5; Videos 3–5). Caudally to the constriction, the anomalous vessel had an aneurysmal dilation at its cranial portion. The distended cavoazygos vessel extended caudally with connections to both the renal and iliac veins, which were also subjectively distended. No arteriovenous shunting was identified. Cryptorchidism was confirmed with the left testicle retained within the left inguinal region. A three-dimensional reconstruction of the anomalous cavoazygos vessel was generated using available software (Vitrea Advanced Visualization, Vital Images, Minnetonka, MS; Figure 6).

The patient's diagnosis was interruption of the prehepatic segment of the CVC with an azygos continuation. Given the aneurysmal dilation of the anomalous vessel at the level of the muscular diaphragm and profound change in vessel size during the echocardiogram, it was hypothesized that there was dynamic compression of the vessel due to positioning that led to intermittent obstruction of the anomalous vessel. This hypothesis would explain the intermittent decreased systemic venous return to the heart and subsequent decreased cardiac output, with attendant exercise intolerance or weakness during periods of exertion or when in recumbent positions. The patient had a routine recovery from sedation and was discharged with clopidogrel for prophylactic antithrombotic therapy. A repeat urine analysis and culture were performed at a follow-up visit with his primary care veterinarian, which confirmed a urinary tract infection (UTI), and a course of antibiotic therapy was instituted at that time. Various therapeutic options were discussed, which included diaphragmatic crus release or vascular stent to help maintain lumen patency; however, the patient's owners ultimately elected to continue monitoring him at home. At the time of writing, 11 months after diagnosis, the patient is alive and overall doing relatively well at home while receiving antiplatelet therapy with only intermittent exercise intolerance.

DISCUSSION

Anomalies of the CVC are rarely diagnosed in veterinary medicine and are often incidentally found during investigations for concurrent disease, in routine laparotomy, or at necropsy.[4-9] In human medicine, interrupted inferior vena cava has a relatively rare prevalence of 0.6%-1.3%.[10] Similar to veterinary patients, diagnosis of this condition is most commonly incidental; however, it can be associated with systemic venous thrombosis.[11] Interruption of the CVC in dogs has been associated with concomitant congenital cardiovascular abnormalities such as portal venous anomalies.[5,7,8] Portosystemic shunts are one of the most frequently encountered systemic venous anomalies in the dog, and unlike interrupted CVC, are frequently accompanied by clinical signs.[12] Therefore, it is not surprising that interrupted CVC or other venous malformations may be incidentally discovered when evaluating patients for suspected portosystemic shunts. No portal vein abnormalities were evident in this patient; however, the authors did consider a possibly increased risk of thrombosis given the aneurysmal dilation and significant spontaneous echocontrast noted on echocardiogram during periods of cardiovascular dysfunction.
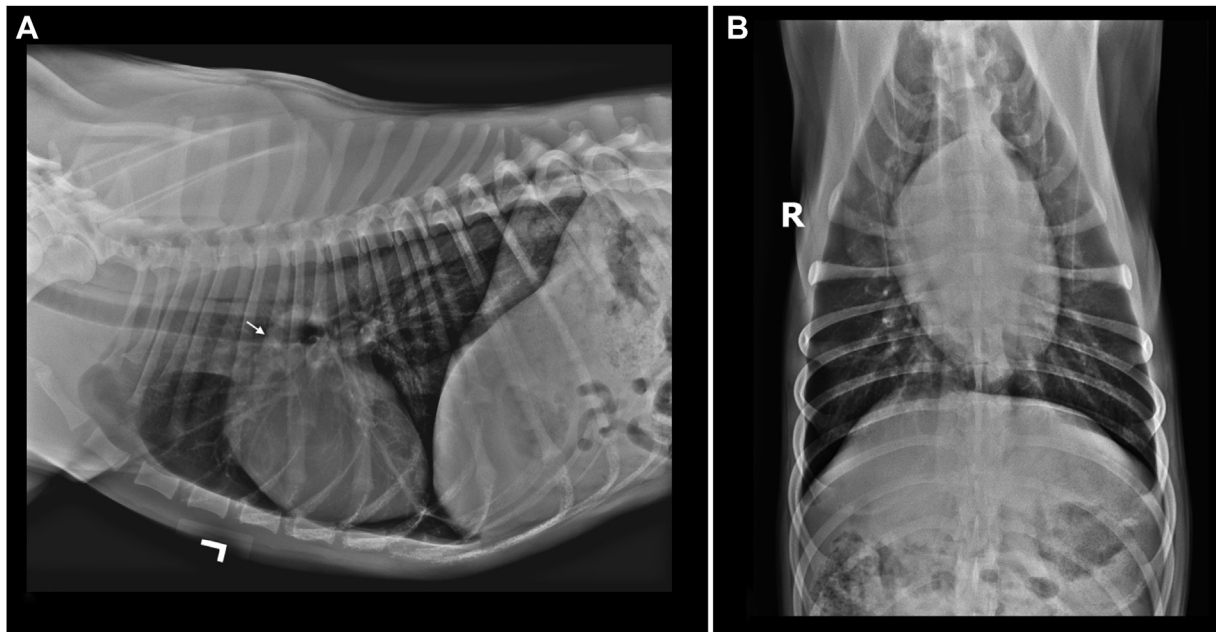
Figure 1 Thoracic radiographs. **(A)** Left lateral and **(B)** dorsoventral views. The *arrow* denotes a prominent azygos vein. *L*, Left; *R*, right.

The systemic venous system of mammals is derived from a complex series of changes involving the formation, anastomosis, and regression of the embryonic venous system.[13] While there are minor differences in nomenclature between bipedal and quadrupedal mammals, the CVC can be subdivided into five segments, derived from various portions of the supracardinal, subcardinal, and vitelline veins. The most cranial CVC segments are formed by the vitelline venous system that develop into the posthepatic and hepatic portions of the CVC. The vitelline and umbilical venous systems also contribute to portal vascular development. Moving caudally, the right vitelline vein fuses with the right subcardinal vein to form the prehepatic segment of the CVC. The anastomosis between the right subcardinal and right supracardinal veins forms the renal segment of the CVC. The prerenal segment is formed by the caudal portion of the right supracardinal vein. The cranial portion of the right supracardinal vein forms the right azygos vein, and the middle portion ultimately regresses. Should the right subcardinal vein not fuse with the vitelline venous system, the CVC terminates at the prehepatic segment, and subsequently the middle portion of the right supracardinal vein persists to form a cavoazygos continuation of the caudal venous system. This prehepatic interruption of the CVC is the most commonly described presentation of interrupted CVC in the dog, and to the authors' knowledge, other variations of interrupted CVC in this species have not been reported.

Our patient presented for episodic weakness and exercise intolerance. While this is an uncommon finding in cases of interrupted CVC without other cardiovascular defects, similar signs have been occasionally reported in several dogs.[6,7] Even more rarely, dogs with interrupted CVC have been diagnosed with complicating thrombosis, with one dog reported as being treated with CVC thrombectomy, which ultimately resolved the exercise intolerance and ascites that were present.[5-7,9] Given the obstruction to blood flow and aneurysmal dilation seen in several of these cases, there is a risk of thromboembolism in affected dogs. Although no visible venous thrombus was noted on the echocardiogram or CT, we felt

it prudent to institute antithrombotic medication in this patient given the reported risk and intermittent spontaneous echocontrast present.

Table 1 Selected echocardiographic measurements of right parasternal views performed in right lateral recumbency during initial echocardiographic evaluation (study 1) and repeated after resolution of clinical signs (study 2)

Echo parameter	Study 1	Study 2
Aortic diastolic diameter, mm	19.6	19.6
LA diastolic diameter, mm	24.5	30
LA:aorta	1.25	1.53
LVEDV, mL	16.6	68.8
LVESV, mL	11.6	31.5
IVSd, mm	13.4	11.5
LVIDd, mm	28.5	41
LVFWd, mm	13.7	10.5
LVIDs, mm	25.9	27.5
Fractional shortening, %	9.1	33
Ejection fraction, %	30.1	54.2
LA volume, mL	8	28
LA volume/kg, mL/kg	0.32	1.1

LA, Left atrial, atrium; *IVSd*, interventricular septal end diastole; *LVEDV*, left ventricular end-diastolic volume; *LVESV*, left ventricular end-systolic volume; *LVFWd*, left ventricular free wall in diastole; *LVIDd*, Left ventricular internal diameter end diastole; *LVIDs*, left ventricular internal diameter end systole.

All measurements were obtained from right parasternal short-axis views (aortic diastolic diameter, LA diastolic diameter, IVSd, LVIDd, LVFWd, LVIDs) or right parasternal long-axis views (LVEDV, LVESV, LA volume) or were derived using the aforementioned values (LA:aorta, fractional shortening, ejection fraction, LA volume/kg).
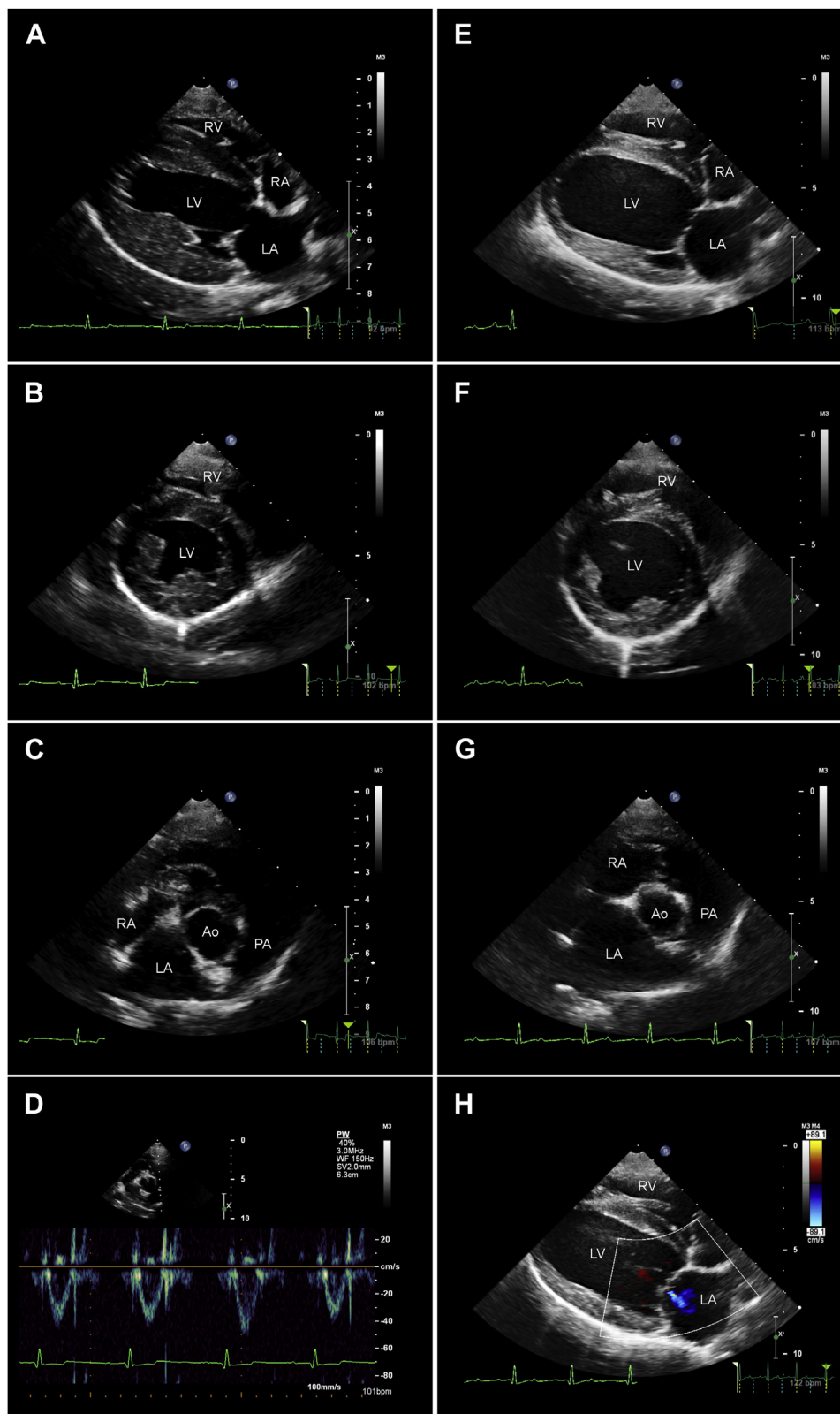
Figure 2 Selected right parasternal echocardiographic views performed in right lateral recumbency during initial echocardiographic evaluation **(A–D)** and repeated after resolution of clinical signs **(E–H)**. **(A)** Right parasternal long-axis four-chamber view. The left and right ventricles appear pseudohypertrophied. **(B)** Right parasternal short-axis left ventricular view. **(C)** Right parasternal short-axis left atrial view. **(D)** Pulsed Doppler study of right parasternal short-axis pulmonary arterial view. The sampling gate is placed at the pulmonic valve. Right ventricular outflow tract flow velocities are decreased. **(E–G)** All cardiac chambers appear adequately filled in the right parasternal long-axis four-chamber view **(E)** and right parasternal short-axis views **(F, G)**. **(H)** Trace mitral regurgitation is noted in the right parasternal long-axis four-chamber view. *Ao*, Aorta; *LA*, left atrium; *LV*, left ventricle; *PA*, pulmonary artery; *RA*, right atrium; *RV*, right ventricle.
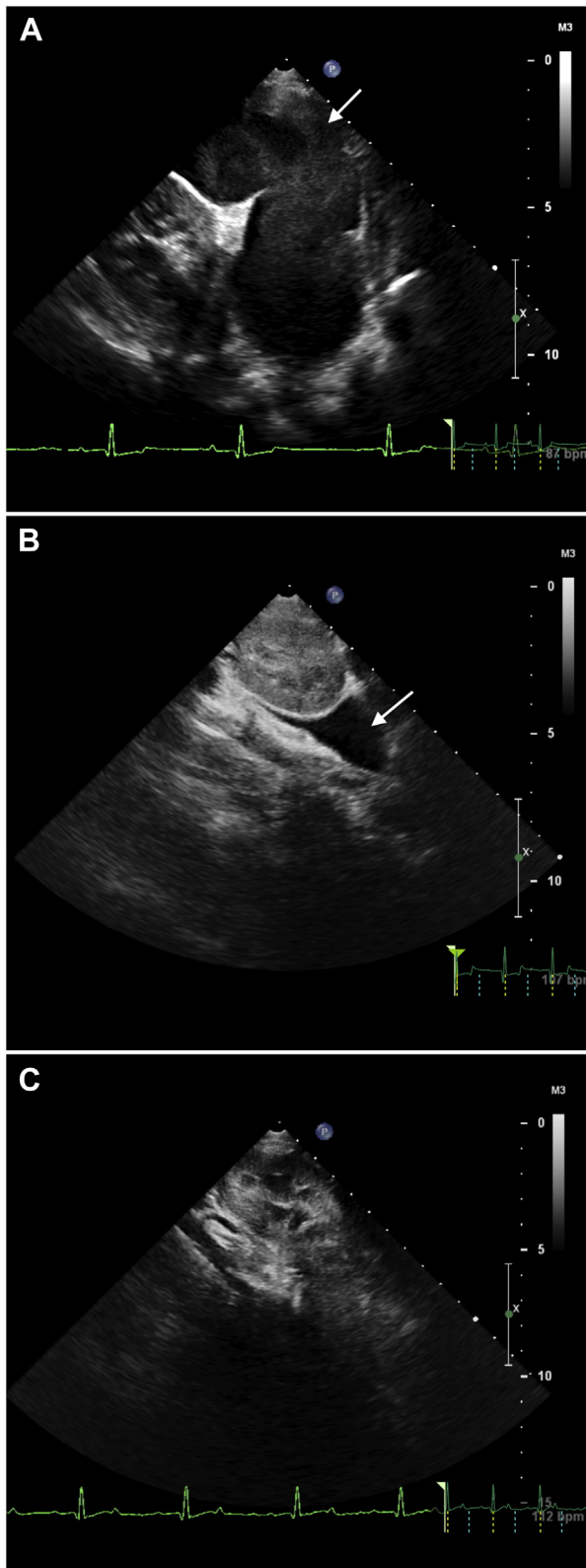
Figure 3 Anomalous abdominal vessel. **(A)** Right lateral recumbency, full distension of the anomalous vessel. **(B)** Left lateral recumbency, resolving distension. **(C)** Left lateral recumbency, 10 minutes later, resolved distension. The *white arrow* denotes the tortuous, anomalous vessel with the presence of spontaneous echocontrast.
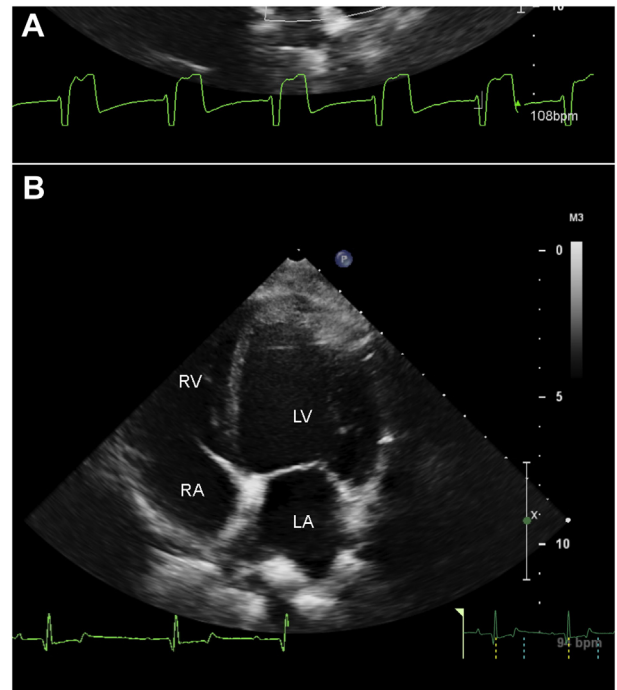

Figure 4 Left parasternal apical four-chamber view performed in left lateral recumbency. **(A)** Accelerated idioventricular rhythm noted immediately postresolution of clinical signs. **(B)** Improved cardiac filling. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

A previously published case series of dogs with similar clinical signs showed segmental collapse of the cavoazygos vessel that occurred during peak inspiration in one dog and was consistent with the findings of ventricular underfilling and tachycardia.[7] We postulate that our patient's weakness and exercise intolerance were due to either changes in position (recumbency) or respiratory-associated diaphragmatic compression and occlusion of the cavoazygos vessel during peak excursion leading to decreased venous return and subsequent poor cardiac output. The underfilled cardiac chambers and cavoazygos vessel dilation were most pronounced in right lateral recumbency in this patient, and the lateralized positioning of the anomalous vessel at aortic hiatus could feasibly be compressed by abdominal viscera. It is of uncertain clinical significance that the patient was reported to lie exclusively in sternal recumbency at home.

This patient's obstructive episodes were associated with disappearance of the heart murmur, palpably weak femoral pulses, depressed systolic function, poor caudal arterial blood pressure with hind limb weakness, and circling. It was suspected that circling was due to discomfort in the abdomen as the patient was highly attentive to and attempting to reach his left flank; however, transient neurological activity secondary to stroke, hypoxemia, or other metabolic derangements cannot be excluded. Based on the murmur localization and echocardiographic findings, the heart murmur was attributed to high-normal transaortic blood flow due to breed-related mild aortic hypoplasia and likely exacerbated by increased cardiac output following resolution of obstructive episodes. The presence of accelerated idioventricular rhythm noted immediately following resolution of venous obstruction likely suggests the occurrence of some degree of myocardial insult with consideration given to myocardial hypoxia or microthrombosis. It is suspected that the transient myocardial
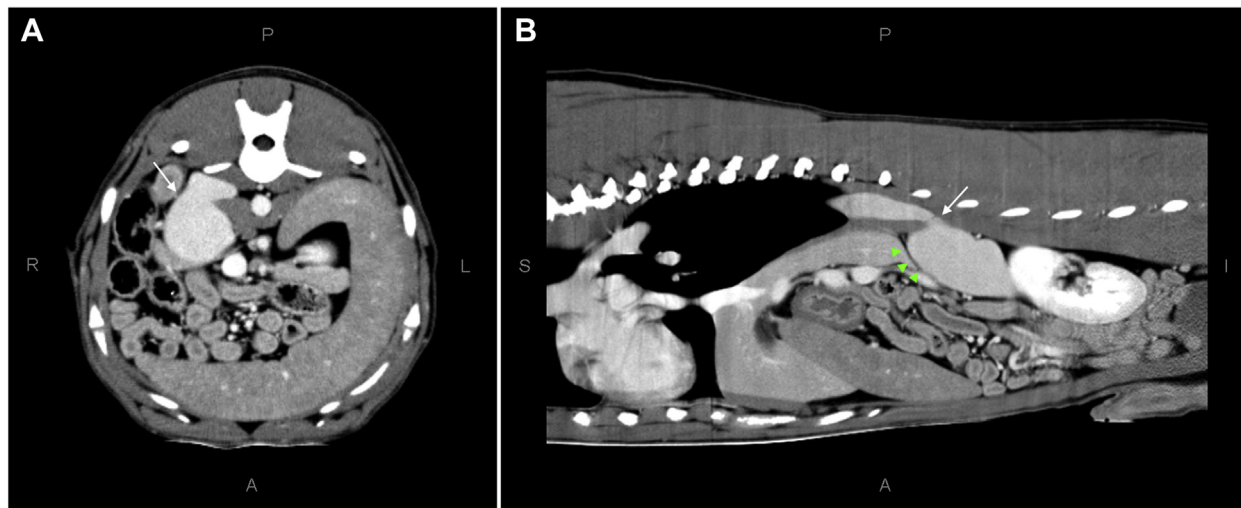
Figure 5 Computed tomography angiographic still images, horizontal **(A)** and sagittal **(B)** views. *White arrows* denote the diaphragmatic compression of the cavoazygos vessel. *Green arrowheads* denote the aneurysmal dilation of the cavoazygos junction.

dyskinesis seen in this patient was due to acutely decreased myocardial perfusion, which resolved with improved cardiac filling; furthermore, the increase in preload likely also contributed to improved systolic function as reflected by Frank-Starling's law. The relationship between this dog's UTI and venous malformation is uncertain and may be coincidental. However, there are reports of UTIs and abdominal diseases such as pyometras being diagnosed in several canine cases of interrupted CVC.[4,5] Retrocaval ureter is another abnormality that has been associated with vascular anomalies and UTI in humans; however, cases of retrocaval ureter are rarely reported in veterinary
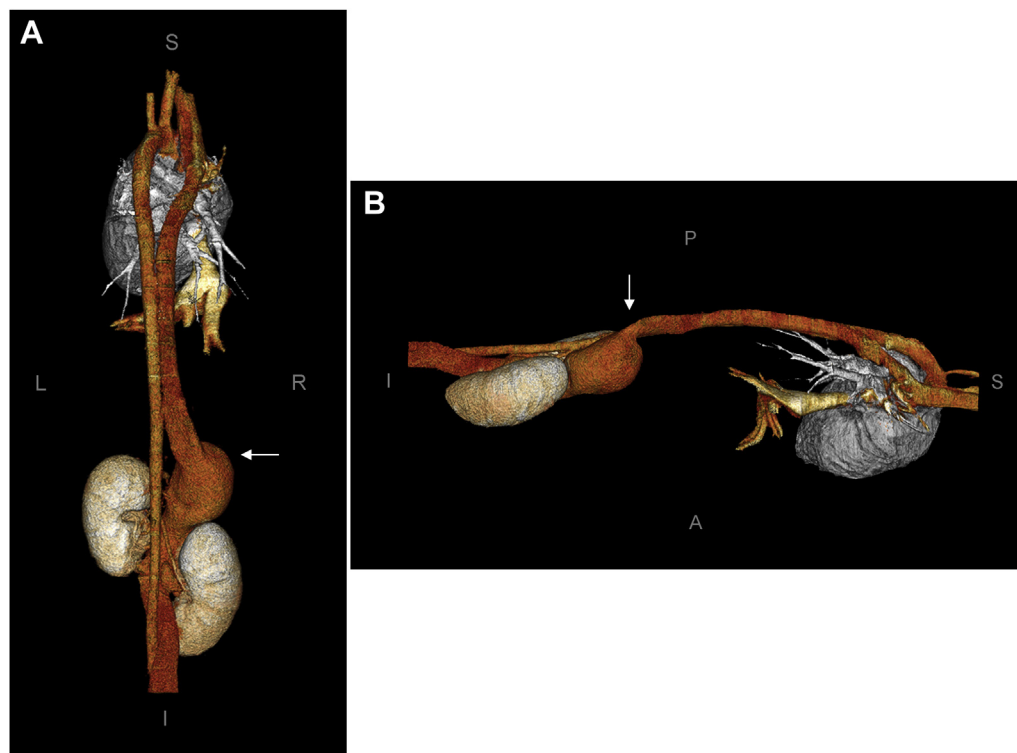

Figure 6 Three-dimensional CT reconstruction of the interrupted CVC. Coronal **(A)** and sagittal **(B)** views are presented. The *arrow* indicates the level at which the anomalous vessel traverses the right crus of the diaphragm. The diaphragm extends from the point of the arrow in a cranial (superior) and ventral (anterior) direction. *A*, Anterior (ventral); *I*, inferior (caudal); *L*, left; *P*, posterior (dorsal); *R*, right; *S*, superior (cranial).

literature. While no ureteral abnormalities were evident on the CT in this dog, UTIs in healthy young male dogs are relatively rare, so it plausible that increased caudal venous pressure leading to passive congestion of the urinary vasculature may have resulted in decreased barrier function and predisposed him to infection. Dogs diagnosed with interrupted CVC should be monitored at home for clinical signs of lower urinary or reproductive tract disease.

CONCLUSION

Clinical signs associated with vascular anomalies of the CVC are rarely encountered. Here we described one case of interrupted CVC being associated with dynamic cavoazygos obstruction and clinical signs during exertion and recumbency. Given our findings, interrupted CVC should be considered as a rare cause of exercise intolerance and intermittent weakness in dogs, and advanced imaging studies may be necessary to definitively diagnose this condition. Clients of affected animals should be advised regarding reported sequelae of thromboembolism or urogenital dysfunction.

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SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2021.11.005.

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