Journal of Veterinary Cardiology (2019) **23**, 112—121

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Journal of Veterinary Cardiology ESVC

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Case Report

Treatment of caudal cavoatrial junction obstruction in a dog with a balloon-expandable biliary stent☆

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Received 9 October 2018; received in revised form 5 February 2019; accepted 21 February 2019

KEYWORDS
Cor triatriatum dexter;
Congenital heart disease;
Angiography;
Canine

Abstract A 2-year-old intact female mixed breed dog was presented for ascites. Echocardiography demonstrated severe obstruction at the level of the caudal right atrium. Initially, a variant of cor triatriatum dexter was diagnosed, and balloon catheter dilation was performed. However, ascites recurred within a week. Further imaging revealed an obstruction at the entrance of the caudal vena cava into the right atrium rather than a dividing membrane in the right atrium. The diagnosis was revised to suprahepatic obstruction of the caudal vena cava because of remnant Eustachian valve tissue. Deployment of a balloon-expandable biliary stent

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https://doi.org/10.1016/j.jvc.2019.02.004
1760-2734/

was performed relieving the obstruction. Fifteen months after stent deployment, the patient is doing well without reaccumulation of ascitic fluid.

Abbreviations

BIB® Balloon-in-Balloon
CauVC caudal vena cava
CTA computed tomography angiography
CTD cor triatriatum dexter;
EV Eustachian valve
RA right atrium
RBP rated burst pressure
TV Thebesian valve

A 2-year-old female, intact mixed breed dog was referred to the Ohio State University Veterinary Medical Center for evaluation of severe ascites and suspected cor triatriatum dexter (CTD). The dog was recently acquired through a rescue organization; thus, a complete patient history was unknown. On admission, the dog was in poor body condition, was flea-infested, and had severe ascites. The dog was seen initially at a local specialty hospital where seven liters of transudate was removed from the abdomen. Hematologic tests were unremarkable, and a heartworm test and full panel for tick-borne diseases were both negative. Thoracic radiographs revealed marked distention of the caudal vena cava (CauVC) and severe ascites, without other significant cardiopulmonary changes. Echocardiography was suggestive of the diagnosis of CTD. The dog was treated with furosemide and spironolactone and referred for further evaluation and potential catheter-based intervention.

Two weeks later, at presentation to The Ohio State University Veterinary Medical Center, the dog was bright, alert, and responsive with a body weight of 15.1 kg (body condition score of 1/9 with severe generalized muscle atrophy). Thoracic auscultation was unremarkable, mucous membranes were pink, and femoral pulses were strong and synchronous. There was marked abdominal distension with a palpable fluid wave. Under light sedation, 3.3 L of serosanguinous fluid was removed from the peritoneal cavity (cytological diagnosis of modified transudate with a total protein of 3.8 g/dL). The peripheral packed cell volume was 39% with a total protein of 5.6 g/dL. The systolic blood pressure was 125 mmHg. Echocardiography[c] using standard right and left parasternal imaging views revealed a four-chambered heart with unremarkable chamber size and global function (Video 1). Cardiac valves appeared normal with trivial to mild tricuspid regurgitation observed. In the right parasternal four-chamber view, turbulent blood flow starting just cranial to the oval fossa was identified, without a proximal isovelocity shell visible at the left side of the atrial septum (Fig. 1, Video 2). This flow was believed to be originating at the entrance of the CauVC into the right atrium (RA). From a tilted right parasternal short-axis view optimized for the right ventricular inflow tract, a valve-like structure with a thickened, poorly moving, obstructive, perforate membrane located near the entrance of the CauVC was identified (Videos 3 and 4). The maximal dimension of the cavoatrial junction was 13.9 mm, whereas the dimension of the stenotic orifice was 2.4 mm (17% of the cavoatrial junction). Doppler echocardiography revealed turbulent, continuous flow across the perforate membrane, from the CauVC to the RA, with velocities ranging from 1.7 to 2.3 m/s (estimated mean pressure gradient across the lesion around 16 mmHg; Fig. 2). The vena cava caudal to the obstruction was dilated but could not be further visualized.

Initially, the diagnosis of CTD was maintained, although the anatomical appearance on echocardiography was not typical of a classic CTD [1]. Further diagnostic tests including cineangiography with hemodynamic evaluation were recommended, and if amenable, relief of the obstruction with balloon dilation was considered. Angiography was performed with fluoroscopic guidance under general anesthesia via a right femoral vein approach. Cineangiography revealed a dilated, tortuous CauVC with a perforate membrane dividing the RA and the CauVC. Fluoroscopy and transesophageal echocardiography were used to guide balloon dilation of the stenotic orifice. A 5 mm × 2 cm (rated burst pressure [RBP] 10 atm) cutting balloon[d] was used followed by a

[c] Vivid E9, GE Medical Systems, Waukesha, WI, USA.
[d] 5 mm × 2 cm Cutting Balloon Catheter, Boston Scientific Corporation, Marlborough, MA, USA.

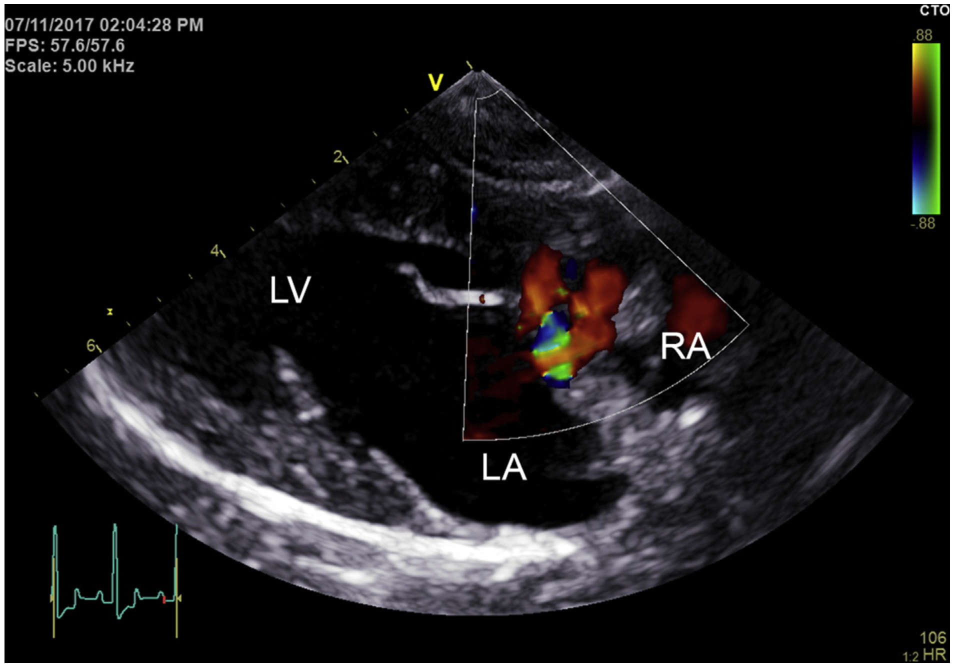

Fig. 1 Right parasternal long-axis view displaying the LA, LV, and RA. Turbulent blood flow can be seen entering the RA, but no proximal isovelocity shell is visible on the left atrial surface of the interatrial septum. LA: left atrium; LV: left ventricle; RA: right atrium.

12 mm × 4 cm (RBP 10 atm) balloon dilation catheter[e] and a 15 mm × 4 cm (RBP 8 atm) balloon dilation catheter[f], using previously described techniques [2]. A pullback pressure was recorded after balloon dilation, which revealed a remaining pressure gradient of 1 mmHg between the RA and dilated CauVC.

Recovery was uneventful, and the following morning, repeat echocardiography was performed, which showed a pressure gradient of 6 mmHg (16 mmHg pressure gradient before procedure), estimated by continuous wave Doppler across the membrane. The dog was discharged with instructions to discontinue diuretics.

One week after the procedure, it was reported that ascites had returned. The dog was otherwise doing well with a good energy level and appetite. Physical examination, 5 weeks after balloon dilation, was unremarkable with the exception of severe ascites. Abdominocentesis was performed, and 1.7 L of serosanguinous fluid was removed. At this time, it was elected to pursue computed tomography with angiography (CTA) to allow for better characterization of the obstructive lesion.

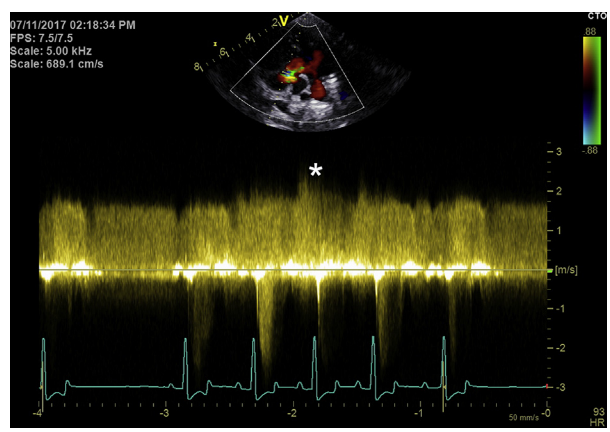

Fig. 2 Continuous wave Doppler flow across the obstructive orifice from a right parasternal short-axis view optimized for the right ventricular inflow tract. A continuous flow signal is seen with velocities ranging from 1.7 m/s to a peak of 2.3 m/s (*).

Computed tomography with angiography[g] was performed under general anesthesia, and 26 mL Iohexol[h] was used for angiography. The imaging revealed a severely distended and tortuous CauVC with abrupt and discrete obstruction at the level of

[e] 12 mm × 4 cm Z-Med Balloon Dilation Catheter, NuMED Incorporated, Hopkinton, NY, USA.

[f] 15 mm × 4 cm Z-Med Balloon Dilation Catheter, NuMED Incorporated, Hopkinton, NY, USA.

[g] Revolution™ Evo GE CT unit, GE Medical Systems, Waukesha, WI, USA.

[h] Omnipaque 240 mg iodine/mL, GE Healthcare Inc., Marlborough, MA, USA.

its convergence with the caudal RA wall. An ill-defined filling defect was present within the narrowing. Incidentally, a single left coronary ostium with an anomalous right (postpulmonary) coronary artery was identified. Thus, CTA confirmed echocardiographic findings of an atypical suprahepatic obstruction of the CauVC at the level of the cavoatrial confluence and rejected the previous diagnosis of CTD. As balloon dilation had failed to permanently relieve the obstruction, endovascular stent placement was considered a viable option in the treatment of this dog. Alternatively, surgery performed under inflow occlusion was discussed.

On presentation, 10 days later, physical examination was unremarkable with the exception of marked abdominal effusion. Echocardiography confirmed persistent obstruction with a mean pressure gradient around 16 mmHg. An intravenous contrast study using agitated saline ruled out presence of any intracardiac communication.

Acepromazine maleate (0.05 mg/kg, intramuscular) and morphine sulfate (0.2 mg/kg, intramuscular) were used for sedation, and anesthesia was induced and maintained with etomidate (2 mg/kg bolus, intravenous) and 1—2 vol% of isoflurane in oxygen. Cefazolin (22 mg/kg) was administered at induction and redosed every 90 min. Systemic blood pressure was monitored via the right dorsal pedal artery. Vascular access was obtained via the left femoral vein cut down with a single-wall modified Seldinger approach, and a 9 Fr × 6 cm vascular access sheath[i] was placed. The CauVC was then catheterized with a 6 Fr, 110 cm balloon wedge catheter[j] for pressure measurements. The catheter was passed through the stenotic lesion easily, and pressures during halted respiration were obtained from the RA (mean, 6 mmHg) followed by pressures within the CauVC (mean, 8 mmHg), indicating a mean pressure gradient of 2 mmHg. The balloon wedge catheter[j] was replaced by a 6 Fr Berman catheter.[k] Administration of contrast agent[h] via hand injection[l] into the CauVC revealed severe dilation of the vein with antegrade flow of contrast through the obstructive lesion present at the level of the RA entrance (maximum CauVC dimension: 15 mm and dimension of the orifice: 4 mm; Fig. 3 and Video 5). Angiography in the orthogonal plane confirmed the measurements. The Berman catheter[k] was replaced by the 7 Fr × 100 cm multipurpose angiographic catheter[m] over a 0.035″ 1.5 mm J-tipped 260 cm super stiff Amplatzer guidewire[n], which was anchored in the cranial vena cava. The multipurpose angiographic catheter was replaced with a 18 mm × 4 cm Tyshak balloon[o] (RBP 2 atm) which was slowly inflated to obtain size measurements at the cavoatrial junction (Video 6). Balloon selection was based on a measured CauVC dimension of 16 mm. The balloon dilation catheter was advanced into the RA, centered at the level of the stenosis, and inflated twice to 1 atm and once to 2 atm. The waist of the stenosis seemed to disappear after the first inflation but recoiled immediately thereafter. The balloon was removed, and the initial introducer was replaced with a 12 Fr 30 cm RCFW Check-Flo® II introducer guiding sheath[p]. Based on the measurements obtained via angiography, a 9—12 mm width × 36 mm length open cell bare metallic biliary stent[q] was selected

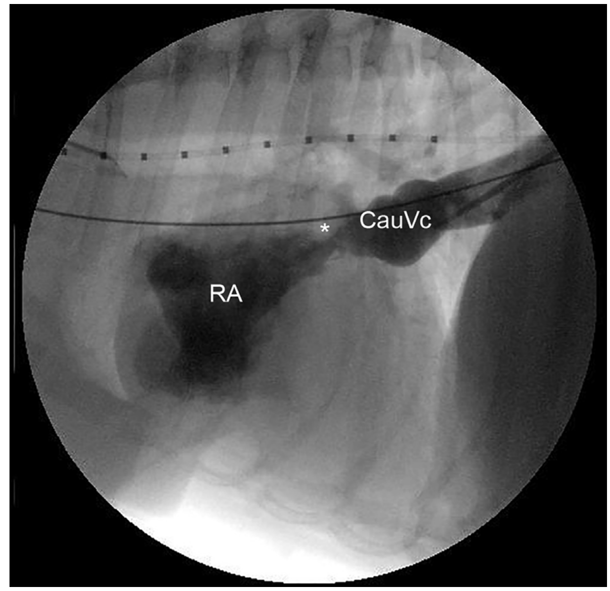

Fig. 3 Lateral angiographic image after contrast injection in the CauVC demonstrating the stenosis (*) at the junction of the CauVC and the RA. A wire has been anchored in the cranial vena cava in preparation for stent deployment. CauVC: caudal vena cava; RA: right atrium.

[i] 9 Fr 6 cm Terumo Pinnacle R/O II introducer, Terumo Medical Corporation, Elkton, MD, USA.
[j] 6 Fr 110 cm balloon wedge catheter, Teleflex Inc, Morrisville, NC, USA.
[k] 6 Fr 90 cm Berman angiographic balloon catheter, Teleflex Inc, Morrisville, NC, USA.
[l] Digital inflation device, B. Braun, Bethlehem, PA, USA.
[m] Torcon NB® Advantage catheter, 7 Fr, 100 cm multipurpose angiographic catheter, Cook Medical, Bloomington, IN, USA.
[n] 0.035″ 1.5 J-tipped 260 cm Amplatzer® super stiff guidewire, AGA Medical Corporation, Plymouth, MN, USA.
[o] 18 mm × 4 cm Balloon Dilation Catheter, NuMED Incorporated, Hopkinton, NY, USA.
[p] 12 Fr 30 cm RCFW Check-Flo® II Introducer, Cook Incorporated, Bloomington, IN, USA.
[q] 9—12 mm (width) × 36 mm (length) biliary stent, Endovascular Company (ev3), Plymouth, MN, USA.

with the plan to balloon dilate the stent to a maximum diameter of 16 mm. The maximum diameter of 16 mm was selected considering the maximum dimension of the CauVC on angiography. Based on prior experience of one of the authors in pediatrics, a balloon size-to-stenosis ratio of approximately 4:1 seemed sufficient to adequately anchor the stent. The stent was gently crimped onto the outer balloon of a Balloon-in-Balloon (BIB®) delivery dilation catheter[r] (inner balloon size, 8 mm × 3.5 cm, and outer balloon size, 16 mm × 4.5 cm) by using finger pressure and a 'rolling action.' A small amount of undiluted contrast[h] was used to 'coat' the stent and improve adherence to the balloon. The balloon-mounted stent was covered with a protective plastic covering (6-cm length of a cut vascular introducer[i]) while introducing through the back-bleed valve of the delivery sheath. Before stent delivery, to prevent potential thrombogenesis related to stent deployment, 100 U/kg of heparin was administered IV [3]. The balloon-mounted stent was then placed over the guidewire[n] and centered across the stenosis while the delivery sheath was slightly retracted to expose the stent. Before inflation, contrast[h] was administered through a 6 Fr Berman catheter,[k] which was passed through the introducer,[p] adjacent to the balloon, to confirm proper stent position (Video 7). The inner balloon[r] (RBP 5 atm) was first fully inflated, using an inflation device.[l] This position still allowed for repositioning of the stent by moving the BIB® catheter.[r] While holding the inflated inner balloon in position, the larger outer balloon was slowly inflated, using a second inflation device.[l] The balloon developed a 'dog bone'—shaped appearance, with the cranial and caudal borders of the stent flared, before fully engaging the balloon (Video 8). This helped prevent asymmetric balloon/stent dilation to avoid 'milking' off the stent during the dilation procedure. Once the stent was fully engaged, both balloons were deflated completely and light, gentle pressure was used on the balloon catheter to ensure the stent was free before withdrawing the balloon to avoid disrupting the stent. The 6 Fr balloon wedge angiographic catheter[j] was then advanced over the wire, past the stent, to aid in safely removing the J-tipped wire.[n] A final angiographic study was performed revealing no residual stenosis and no significant tricuspid regurgitation (Fig. 4, Video 9). Postprocedural pressure measurements were not obtained because of malfunction of the equipment. Instrumentation was removed, the femoral vein was ligated, and the skin was closed in routine fashion.

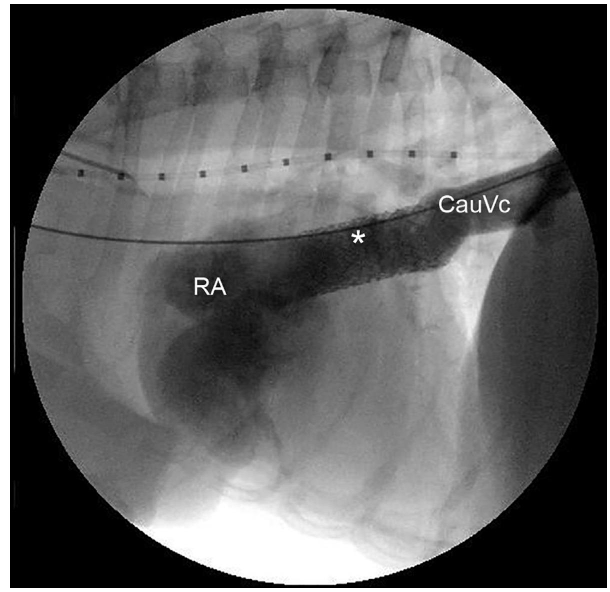

Fig. 4 Lateral angiographic image after stent deployment. The wire used for stent deployment is seen spanning from the cranial vena cava to the CauVC. The stent has been expanded over the previous region of stenosis (*) allowing unobstructed flow from the CauVC to the RA. CauVC: caudal vena cava; RA: right atrium.

The patient recovered uneventfully in the intensive care unit overnight. Maintenance intravenous crystalloid fluids, cefazolin (22 mg/kg intravenous q 8 h), heparin (50 U/kg subcutaneous q 8 h), and clopidogrel (37.5 mg per os q 24 h) were administered. The following morning, repeat echocardiography and thoracic radiography (Figs. 5 and 6 and Videos 10 and 11) were performed. Echocardiography revealed stable stent position and laminar flow across the stent at 1 m/s (mean pressure gradient of 4 mmHg). Minor flow turbulence was visualized immediately proximal to the stent, but peak velocity was similar to intraluminal stent flow. The stent was not impinging on the tricuspid valve. The electrocardiogram documented normal sinus arrhythmia without ectopy. Heparin was discontinued that morning, and the patient was discharged on cephalexin and instructed to continue clopidogrel for the next 6 months.

The dog returned 3, 6, and 15 months later for recheck evaluations and was reported to be doing well with no evidence of ascites. On physical examination, the dog's body condition was markedly improved (body condition score, 5/9; body weight, 19.2 kg). The remainder of the physical examination was unremarkable. Thoracic

[r] BIB® Balloon-in-Balloon dilation catheter, NuMED Incorporated, Hopkinton, NY, USA.

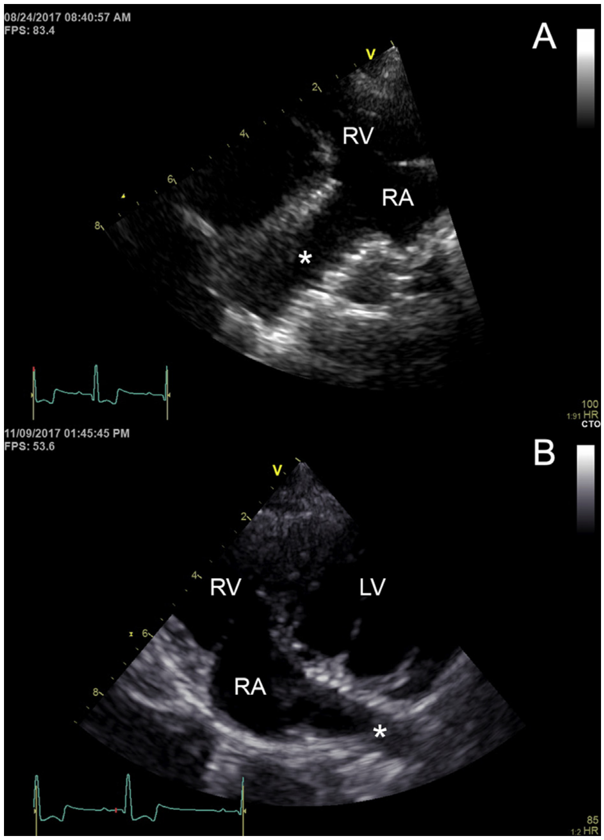

Fig. 5 Echocardiographic images. (A) Tilted right parasternal long-axis view optimized for the RA, RV, and stent (*). The stent is seen spanning from the entrance to the RA into the caudal vena cava. (B) Modified, left apical image optimized for the caudal RA. The stent (*) can be seen from the entrance of the RA into the caudal vena cava. In both the images, it can be appreciated that the stent does not significantly impinge on the tricuspid valve apparatus. LV: left ventricle; RA: right atrium; RV: right ventricle.

radiographs demonstrated unchanged position of the stent. Echocardiography revealed static stent position, persistent patency of the stent without obstruction, no evidence of thrombus formation, and no impingement on the tricuspid valve.

Discussion

Although CTD has been documented in at least 30 dogs in the veterinary literature, to our knowledge, congenital stenosis of the CauVC at the cavoatrial level has only been reported in a single case [4]. This report described a 4-month-old rottweiler puppy who presented for ascites and pleural effusion. Angiography revealed stenosis at the junction of the CauVC and RA. The lesion was resected surgically with complete resolution of clinical signs. The conclusion was that the obstruction resulted from an anomalous formation of the embryologic valve of the CauVC or a persistent Eustachian valve (EV). Acquired obstruction of the CauVC or near the CauVC—RA junction has also been described and is referred to as Budd—Chiari—like syndrome [5—7]. Reported etiologies of this syndrome include fibrosis [5] and neoplasia [6,7].

In early embryologic development, venous drainage of the left and right common cardinal veins reaches the primitive atrium through the sinus venosus. These two cavities are connected through the sinoatrial orifice, which is guarded by two well-developed valvular folds. Later, in development, the right valve grows, partially dividing the RA, and functions to direct oxygenated blood flow from the CauVC to the foramen ovale and thus the left atrium. As the heart develops, the sinus venosus valves separate into two venous orifices while retracting into the expanding RA and regress. The left sinus valve merges with the atrial septum secundum. The right valve of the sinus venosus is very large, at one time, nearly dividing the RA into two chambers, but later almost disappears, with the cranial portion remaining as the crista terminalis and the caudal portion being divided to form the valve of the CauVC (EV) and the valve of the coronary sinus (Thebesian valve, TV) [8—10]. It is interesting to note that, as early as 1897, Chiari [11] reported on the great anatomical variability of the remnants of the right sinus venosus valve in people and also mentioned animal observations relevant to this case. In particular, the EV is a heterogenous structure. Various descriptions have included a prominent ridge; cord-like anatomy; a thin-valved structure; flap-like, fenestrated, small strands of tissue with varying insertions; or total absence [11,12].

The human literature [8] describes four basic anatomic variants of right sinus venosus valve remnants based on morphology, insertion, and size: CTD, Chiari network, persistent EV, and persistent TV. Among these, CTD is the most severe form and occurs when the large obstructive membrane dividing the RA into two cavities persists. Six anatomic subtypes of CTD have been reported in people, including type 3 as 'a membrane covering only the opening of the CauVC' [8]. A Chiari network is described as a reticulated network of thin fibers with whip movement originating from the EV or TV connecting different parts of the RA. A persistent TV is a membranous or filiform remnant of the caudal part of the right valve of the sinus venosus located at the orifice of the coronary sinus. Finally, a persistent EV is the membranous crescent-like or web-like residuum of

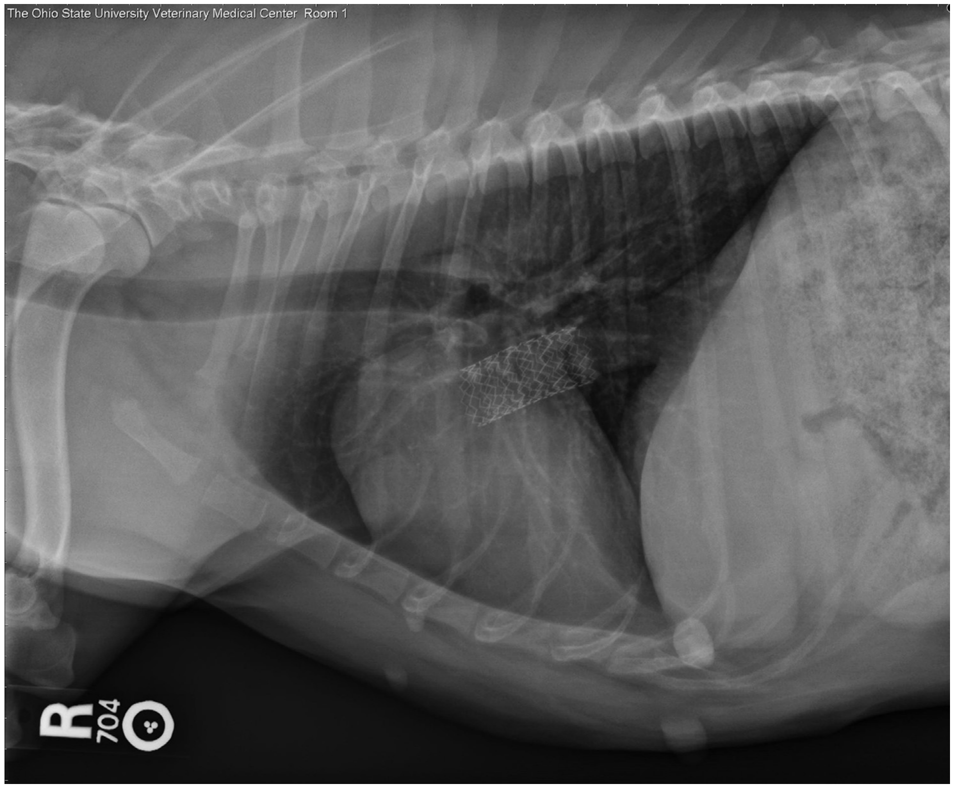

Fig. 6 Right lateral thoracic radiograph, obtained 1 day after stent deployment, demonstrating appropriate stent position.

the caudal part of the right sinus venosus valve inserting at the orifice of the CauVC [12,13]. The latter three defects are typically incidental findings and not obstructive in people [8]. However, they can act as sites for attachment of masses and thrombus formation and can be affected by infective endocarditis causing posthepatic CauVC obstruction [8].

As mentioned, a persistent EV tends to be an incidental finding in people with a reported prevalence of $>$50% at autopsy. To the authors' knowledge, comparative canine data do not exist. There are at least four case reports of this defect resulting in inflow obstruction in the human literature [14–17]. These cases describe suprahepatic obstruction of the inferior vena cava localized at the level of entry to the RA, secondary to a prominent EV [14–17]. One case report emphasizes that these lesions tend to not be obstructive; however, if the EV becomes inflamed and thrombosis develops, stenosis can develop as an acquired lesion [14].

In this dog, the initial diagnosis was CTD based on clinical signs and echocardiographic findings. However, it was appreciated that classic CTD could not be present because of the absent partition of the RA and failure to identify a typical dilated caudal chamber. Obstruction in the present case occurred at the cavoatrial junction, and a prominent ridge of immobile tissue was observed.

As there was a stenotic lesion that seemed amenable to a catheter-based intervention, it was elected to proceed with dilation using a two-phase approach of a cutting balloon followed by a larger transluminal angioplasty balloon catheter. These treatments for CTD have been previously described [2,18–22]. The procedure appeared successful initially after loss of the stenotic waist was observed on fluoroscopy. However, recoil of the lesion probably led to early restenosis, as clinically evidenced by relapse of ascites. Advanced imaging was pursued to gain a better understanding of the obstruction. The morphology of the lesion seen on review of the initial angiograms and CTA allowed refinement of the diagnosis and exclusion of accompanying malformations.

Treatment options for CTD and stenosis of the cavoatrial junction in dogs include surgery [1,4,23,24], balloon dilation either using high-pressure balloons or by cutting balloon dilation followed by high-pressure balloon dilation [2,18–21], or balloon dilation followed by balloon-expandable stent placement [22]. The use of endovascular stents has been reported for a variety of stenosing defects in animals, including palliation of supravalvular pulmonary stenosis [25], treatment of Budd-Chiari—like syndrome [6], treatment for severe pulmonary valve stenosis in dogs [26], and treatment for stenosis of the suprahepatic CauVC in cats [27,28]. The use of a balloon-expandable stent was then successfully pursued for this dog. As suggested by Barncord et al. [22], a BIB® dilation catheter was used for controlled expansion of the stent. The BIB® catheter contains an inner balloon that is one-half of the diameter of the outer balloon. When the inner balloon is inflated, expansion of the stent begins and allows the stent to be engaged with the stenotic region. This is followed by biphasic inflation of the outer balloon with the out-of-stent portion inflated first, preventing slipping of the stent from the edge. Finally, the central portion expands slowly and uniformly to its predetermined width.

In conclusion, this report describes a rare form of presumably congenital stenosis at the level of the junction of the CauVC and RA. This stenosis is hypothesized to be secondary to a persistent EV and could represent a subtype of CTD. The lesion was not successfully treated with balloon dilation alone, and endovascular stent placement was necessary to relieve the obstruction and alleviate clinical signs.

Conflict of interest statement

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

Acknowledgments

The authors thank Sarah Bell, DVM, MS, ACVIM (Cardiology) for referring this case and Emily Chapel, DVM, MS, ACVIM (Cardiology) and Samantha Kochie, DVM for their contributions with general patient care.

Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.jvc.2019.02.004.

Video table

Video 1	Two-dimensional echocardiographic images recorded at baseline demonstrating cardiac anatomy before intervention	Left upper panel: right parasternal four-chamber view. Right upper panel: right parasternal short-axis view. Lower left panel: right parasternal short-axis heart base view. Right lower panel: the left apical five-chamber view. All cardiac structures appear unremarkable. Ao: aorta; LA: left atrium; LV: left ventricle; PT: pulmonary trunk; RA: right atrium; RV: right ventricle
Video 2	Right parasternal long-axis echocardiographic view with color Doppler recorded at baseline	Turbulent flow can be seen entering the caudal RA just behind the atrial septum. The lack of an aliased proximal isovelocity shell on either side of the atrial septum indicates that flow is not across an interatrial communication. Minimal tricuspid regurgitation is also present. LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle
Video 3	Tilted right parasternal short-axis echocardiographic image optimized for the right ventricular inflow tract	A thickened, poorly mobile, obstructing membrane (*) can be seen at the junction of the CauVC and the RA. CauVC: caudal vena cava; RA: right atrium; RV: right ventricle
Video 4	Tilted right parasternal short-axis echocardiographic image optimized for the right ventricular inflow tract and superimposed with color flow Doppler	Continuous, turbulent flow can be seen starting at the level of the obstructing membrane (*) at the entrance of the RA. Ao: aorta; CauVC: caudal vena cava; PT: pulmonary trunk; RA: right atrium; RV: right ventricle

Video 5	Fluoroscopic right lateral thoracic angiographic image with CauVC contrast injection	A dilated, tortuous CauVC, the RA, and the location of the obstruction (*) at the entrance of the RA are demonstrated. Transesophageal echocardiography was used to help define cardiac anatomy and aid in the appropriate positioning of catheters and balloons. CauVC: caudal vena cava; RA: right atrium
Video 6	Fluoroscopic right lateral thoracic image with sizing balloon	Slow inflation of the Tyshak balloon to estimate defect size (*) used for selection of stent size. A vessel-sizing catheter, used for calibration of the measurement tool on the fluoroscopic unit, is visible in the esophagus. CauVC: caudal vena cava; RA: right atrium
Video 7	Fluoroscopic right lateral angiographic image depicting stent positioning before deployment	Visual stent 'fitting' was performed immediately before stent deployment across the stenosis (*) to confirm the stent position. CauVC: caudal vena cava; RA: right atrium
Video 8	Fluoroscopic right lateral image demonstrating deployment of the stent	Balloon deployment of the stent using the Balloon-in-Balloon (BIB®) catheter across the defect (*). CauVC: caudal vena cava; RA: right atrium
Video 9	Fluoroscopic right lateral thoracic angiographic image after stent deployment	Angiogram performed after stent (*) deployment demonstrating unobstructed flow across the previously stenotic opening at the junction of the CauVC and the RA. CauVC: caudal vena cava; RA: right atrium
Video 10	Left apical two-dimensional tilted right parasternal long-axis echocardiographic image	Echocardiographic imaging view optimized for the RA, RV, and stent (*). The stent can be identified in the distal portion of the caudal vena cava just at the entrance into the RA. RA: right atrium; RV: right ventricle
Video 11	Left apical two-dimensional four-chamber echocardiographic image optimized for the right heart	Left panel: The stent (*) can be seen in the distal caudal vena cava ending at the entrance into the RA. On color flow Doppler (right panel), laminar blood flow across the stent (*) is demonstrated. LV: left ventricle; RA: right atrium; RV: right ventricle

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