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Case Report

Case report — azygos vein drainage into the left atrium in a dog with cor triatriatum dexter and a patent foramen ovale☆

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Abstract A 2-year-old Airedale terrier was presented with exercise intolerance since birth and newly developed chylous pleural effusion. Imaging procedures including echocardiography, cardiac magnetic resonance imaging, computed

☆ A unique aspect of the Journal of Veterinary Cardiology is the emphasis of additional web-based images permitting the detailing of procedures and diagnostics. These images can be viewed (by those readers with subscription access) by going to http://www.sciencedirect.com/science/journal/17602734. The issue to be viewed is clicked and the available PDF and image downloading is available via the Summary Plus link. The supplementary material for a given article appears at the end of the page. Downloading the videos may take several minutes. Readers will require at least Quicktime 7 (available free at http://www.apple.com/quicktime/download/) to enjoy the content. Another means to view the material is to go to http://www.doi.org and enter the doi number unique to this paper which is indicated at the end of the manuscript.

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echocardiography;
Cardiac CT;
Giant Eustachian valve

tomography, and selective angiography revealed an aberrant connection of the azygos vein and the left atrium, a membrane in the right atrium consistent with cor triatriatum dexter, and a patent foramen ovale with right-to-left shunt. Balloon dilation of the membrane in the right atrium seemed to result in transient improvement of exercise tolerance compared with the previous 2 years. When chylothorax relapsed after three months, the dog was euthanized. Necropsy confirmed the azygos vein to left atrial connection, the patent foramen ovale, and the cor triatriatum dexter.

A 2-year-old, female intact Airedale terrier, weighing 19.5 kg, was presented to the Division of Cardiology of the Vetsuisse Faculty University of Zurich. The dog had been exercise intolerant since puppyhood, and a right-to-left shunting patent foramen ovale associated with a cor triatriatum dexter had been diagnosed at a prior cardiologic examination at 7 months of age. The reason for the new presentation was acute onset of tachypnea and more severe exercise intolerance for several days.

Abnormal findings of the clinical examination included a respiratory rate of 60 breaths/minute with increased respiratory effort and paradoxical breathing. Thoracic auscultation revealed ventrally muffled lung and heart sounds. Color of mucous membranes and capillary refilling time were normal, and no heart murmur, no jugular vein distension, and no abdominal effusion were present. The respiratory pattern and thoracic auscultation findings were suggestive of pleural effusion, which was confirmed on thoracic ultrasound.[f] Thoracocentesis yielded 1000 mL of a chylous fluid (2.6 mmol/L cholesterol and 7.2 mmol/L triglycerides in the free pleural fluid compared with 3.6 mmol/L [3.5—8.6 mmol/L] cholesterol and 0.3 mmol/L triglyceride [0.4—1.5 mmol/L] in the serum). The only serum biochemical abnormality was mild hypoproteinemia (52 g/L; 56—71 g/L) with normal albumin (34 g/L; 29—37 g/L). A complete blood count was unremarkable with a hematocrit concentration of 48%.

An echocardiographic examination[f] was conducted to evaluate if the chylothorax was caused by a primary cardiac condition. The left atrium and ventricle were structurally and the left ventricle functionally normal (left ventricular M-mode measurements were as follows: interventricular septum thickness at end-diastole: 0.9 cm; interventricular septum thickness at end-systole: 1.1 cm; left ventricular internal dimensions at end-diastole: 3.5 cm; left ventricular internal dimensions at end-systole: 2.7 cm; left ventricular free-wall thickness at end-diastole: 0.9 cm; left ventricular free-wall thickness at end-systole: 1.2 cm; fractional shortening: 24%). A vessel was visible entering the left atrium dorsally, which did not seem to be a normal pulmonary vein based on size and location; contrast echography subsequently confirmed this to be an abnormal vessel (see below). The right ventricle and right ventricular outflow tract were considered unremarkable qualitatively, and pulmonic transvalvular flow was laminar and symmetrical with normal flow velocity (peak velocity, 1 m/s). Color and pulsed-wave Doppler echocardiography revealed mild pulmonic insufficiency with a peak velocity of 0.6 m/s. Color Doppler interrogation of a slit-like passageway in the interatrial septum, suggestive for a patent foramen ovale, showed right to left flow. In addition, a thin, immobile, perforated membrane separated the systemic venous sinus from the body of the right atrium with the right auricle and the vestibule with the tricuspid valve. A faint, mobile, echoic, vertical line was visualized in the systemic venous sinus (Video 1 and Video 2). From the cranial vena cava, laminar flow bypassed this right atrial membrane (Video 3). The coronary sinus was subjectively normal.

Contrast echocardiographic studies using agitated saline were performed in an oblique left parasternal apical view, and serial contrast injections were made from both cephalic (Video 4) and saphenous (Video 5) veins to better characterize the flow across the detected abnormalities. Injection into each cephalic vein resulted in the same contrast enhancement pattern (Fig. I available in Supplemental Material online). There was contrast accumulation on both sides of the intra-atrial membrane. After a short delay, the contrast medium in the chamber of the right atrium being closer to the interatrial septum was partially

[f] Vivid 7, GE Healthcare; 8152 Glattbrugg, Switzerland.

cleared by anechoic blood and it did not cross the patent foramen ovale into the left atrium. There was a large amount of contrast flowing into the left atrium through the anomalous vessel in the left atrial roof. No contrast accumulated in the coronary sinus after cephalic vein injection. Injection in the saphenous vein resulted in enhancement of both compartments of the right atrium with subsequent flow of contrast medium through the patent foramen ovale into the left atrium. Anechoic blood replaced the contrast medium close to the membrane. The aberrant vessel in the roof of the left atrium did not enhance. Cardiac magnetic resonance imaging[g] was performed with retrospective vectorcardiography gating under general anesthesia[h] to better characterize the origin of the anomalous left atrial vessel. Maintenance of general anesthesia with Sevoflurane in a mixture of oxygen (95%), and air was supported with a continuous rate infusion of fentanyl and medetomidine. During anesthesia, it was detected that S_pO_2[i] was inversely related to heart rate. The saturation of 86% at a heart rate of 140 beats per minute rose to 96% when the heart rate dropped to 85 beats per minute, and saturation fell again when the heart rate increased. The cardiac magnetic resonance imaging study visualized a perforated membrane separating the systemic venous sinus from the right atrial body with the appendage and normal drainage through the tricuspid valves. The study reproduced the findings from echocardiography concerning laminar flow across the intra-atrial membrane into the right atrial body and turbulent right-to-left shunt through the patent foramen ovale (Video 6). In addition, cardiac magnetic resonance imaging visualized the moderately dilated azygos vein, identified by its origin, the entrance of multiple dorsal intercostal veins, its course along the trachea and esophagus, and the drainage into a single cranial vena cava. The azygos vein abruptly increased in diameter at the level of the origin of an aberrant vessel heading ventrally to drain into the roof of the left atrium (Video 7). A flow-sensitive sequence showed low-velocity, laminar flow, most rapid in diastole in the dilated portion of the azygos in the same direction as the descending aorta indicating retrograde azygos blood flow. A moderate amount of pleural effusion of high T1-weighted signal intensity was evident in the left thoracic cavity, resulting in atelectasis of the left lung lobes and a mild shift of the mediastinum and the heart. The dog recovered uneventfully from anesthesia.

Based on the imaging findings, a diagnosis of a perforated right atrial membrane partially separating the systemic venous sinus from the right atrial body, an azygos vein drainage into the left atrium, and a patent foramen ovale with right-to-left shunt was made.

Invasive pressure measurements[i] and fluoroscopic angiography[j] were performed to better define the degree of obstruction caused by the intra-atrial membrane. Through percutaneous access into the right jugular and left femoral veins, catheters[kl] were advanced into the cranial and caudal vena cava (Fig. 1).

Manual injection of a 10-mL bolus of iodinated, non-ionic contrast medium[m] into the cranial vena cava caused initial enhancement in the cranial aspect of the systemic venous sinus and the right atrium with subsequent flow to the right ventricle. The contrast medium did not cross the patent foramen ovale. A small amount of contrast medium was evident flowing in a retrograde direction into the azygos vein (Fig. 1A). A multipurpose catheter could be advanced from the cranial into the caudal vena cava without resistance. Contrast injection into the caudal vena cava resulted in enhancement of the caudal aspect of the systemic venous sinus, the right atrium, and the left atrium through the patent foramen ovale. Injection into the azygos vein highlighted the aberrant vessel between the azygos vein and the left atrium (Fig. 1B and C). Pressure in the cranial and caudal caval veins and different levels of the right atrium ranged between 4 and 8 mm Hg; however, a clear pressure drop from cranial vena cava to the right atrium could not be objectively documented. Systolic and diastolic right ventricular pressures were 17 and 5 mm Hg. Even

[g] Philips Ingenia 3T with dStream body coil Solution, Philips AG, 8027 Zurich, Switzerland.

[h] Premedication: acepromazine (Prequillan®, Arovet AG, 8953 Dietikon, Switzerland; 0.05 mg/kg) and methadone (Methadon®, Streuli Pharma AG, 8730 Uznach, Switzerland; 0.3 mg/kg) i.m. Induction: propofol (Propofol MCT®, Fresenius Kabi AG, 6370 Stans, Switzerland; 3 mg/kg). Maintenance: sevoflurane (Sevorane®, AbbVie AG, 6340 Baar, Switzerland). Constant rate infusion: fentanyl (Sintenyl®, Sintetica SA, 6850 Mendrisio) and medetomidine (Dorbene®, Graeub E. Dr. AG, 3018 Bern). Skeletal musculature relaxation: rocuronium (Esmeron®, Merck Sharp & Dohme AG, 6005 Luzern).

[i] Cardiocap MRI Monitor, GE Healthcare, Finland.

[j] Philips PCR, Corado, CR-IR 362; Philips AG, 8027 Zurich, Switzerland.

[k] 5 French pigtail catheter, Cook Medical, 6002 Luzern, Switzerland.

[l] 5 French multipurpose catheter, Cook Medical, 6002 Luzern, Switzerland.

[m] Accupaque 350®, Iohexol, GE Healthcare, 8152 Glattbrugg.

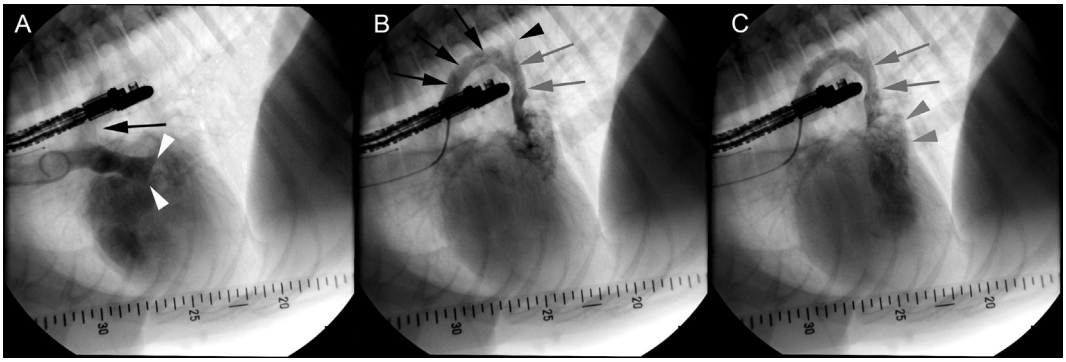

Fig. 1 A to C: Selective, fluoroscopic angiography in two different positions of the catheter: A: Manual administration of contrast medium into the cranial vena cava. There was mild, retrograde enhancement of the dilated azygos vein (black arrow). The contrast medium filled the right atrium and drained into the right ventricle. A well-defined transition (white arrow heads) occurred between the blood with contrast medium from the cranial vena cava and the blood from the caudal vena cava. B: A catheter was advanced from the right jugular vein through the cranial vena cava into the dilated azygos vein. The manually administered contrast filled the azygos vein (black arrows) in a retrograde direction. It formed a small beak sign in the normal part of the azygos vein (small black arrow head) and highlighted a shunt (gray arrows) connecting the azygos vein with the left atrium. C: The contrast medium injected in the azygos vein drained through the shunt into the left atrium (gray arrow heads) and ventricle.

though invasive pressure measurements did not show a pressure gradient across the venous blood flow, balloon dilation was still performed. A 15-mm low-pressure balloon[n] was advanced along the cranial vena cava to the right atrium over a stiff guiding wire, and a mild waist was observed during balloon inflation, which disappeared once the balloon was fully inflated. After recovery, the amount of chylous pleural effusion requiring drainage decreased rapidly and completely resolved after a few days. After discharge, the owner reported complete resolution of the congenital exercise intolerance; however, this improvement was only transient. Three months later, the chylous pleural effusion recurred and again caused mild dyspnea and exercise intolerance.

A thoracic computed tomography (CT)[o] was performed to visualize the thoracic duct and further investigate any intracardiac or extracardiac obstruction to venous return. Attempts to visualize the thoracic duct by CT of the thorax failed because ultrasound-guided contrast injection into both popliteal lymph nodes leaked through the needle track.

Angiographic cardiac CT[o] with prospective electrocardiographic triggering reproduced the aberrant connection between the azygos vein and the left atrium. Similar to cardiac magnetic resonance imaging, the perforated membrane was visualized separating the systemic venous sinus from the body of the right atrium in a dorsolateral to ventromedial plane. The CT study confirmed the previously observed selective enhancement pattern depending on cephalic or saphenous injection of contrast medium[m] (Fig. 2). A small discontinuity in the cranial aspect of the membrane allowed cephalic contrast medium to drain into the body of the right atrium. A central perforation allowed communication of the caudal part of the systemic venous sinus with the right atrial body, visible after saphenous injection. No mixture of cephalic and saphenous blood occurred in the systemic venous sinus.

Thoracoscopy,[p] performed through ports in the right 9th and 11th intercostal spaces by a one-lung-ventilation technique, visualized the aberrant vessel draining into the left atrium at its origin from the azygos vein (Fig. 3). Compression of the aberrant vessel caused no increase in the cranial vena cava pressure[i], measured at 8 mmHg by a central venous catheter.[q] The thoracic duct was ligated with a vessel sealing device[r], and the pericardium resected subtotally.

The pleural effusion resolved after thoracoscopic surgery. However, after three months, chylous pleural effusion relapsed again and euthanasia was elected. Necropsy confirmed the

[n] Veterinary Balloon Catheter 15 mm, NuMed Inc.; Hopkinton, NY, USA.
[o] Philips Brilliance CT 16 slice; Philips AG; 8027 Zurich, Switzerland.
[p] Storz, Tuttlingen, Germany.
[q] Multi-lumen central venous catheterization Set, 7 Fr, 3 Lumen, 20 cm; Teleflex Medical GmbH; 3123 Belp, Switzerland.
[r] Ligasure® 5 mm, Covidien, Boulder, CO, USA.

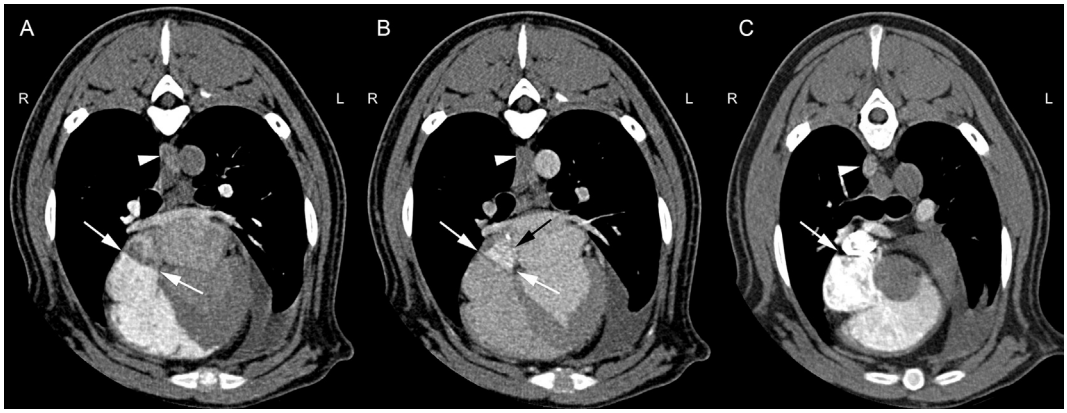

Fig. 2 A to C: Transverse slices of angiographic computed tomography studies of the heart in diastole after A: cephalic injection in a caudal location, B: saphenous injection at the same level as A, C: cephalic injection in a further cranial location. A: The right atrial membrane (white arrows) oriented in a dorsal-oblique plane separated the systemic venous sinus from the right atrial body identified by the auricle. The dilated azygos vein (white arrow head) and the left atrium enhanced faintly. B: Saphenous injection resulted in enhancement of the other side of the membrane (white arrows). The contrast medium escaped through the patent foramen ovale (black arrow) into the left atrium and simultaneously contributed to the enhancement of the main compartment of the right atrium. The azygos vein (white arrow head) did not enhance. C: The well-defined membrane (white arrow) allowed the contrast medium to reach the main right atrial compartment through a small opening at this cranial level. The dilated azygos vein (white arrow head) contained a small amount of contrast medium.

aberrant connection of the dilated azygos vein with the left atrium (Fig. 4). A large, thin membrane in an oblique dorsal position separated the systemic venous sinus from the body of the right atrium. Two large openings, separated by a well-defined tissue band, allowed blood from the systemic venous sinus to enter the body of the right atrium. The diameter for the smoothly marginated opening of the caudal vena cava exceeded the roughly defined opening for the cranial vena cava. The body of the right atrium, identified by the right auricle, had an unhindered connection to the vestibule of the tricuspid valve. An additional, thin soft-tissue band crossed the systemic venous sinus vertically. The shape of the membrane and this parenchymal band potentially guided blood from the caudal vena cava toward the patent foramen ovale and hindered drainage of the cranial vena cava which only had a small, ill-defined opening for blood drainage into the body of the right atrium. The additional vertical parenchymal band most likely hindered mixture from blood arriving from the cranial and caudal vena cava. As this multiperforated membrane appeared to have been of hemodynamic importance, the anomaly was classified as cor triatriatum dexter, as opposed to persistent Eustachian valve. A normally developed coronary sinus, characterized by a continuous separation from the left atrium and complete roofing, drained into the caudal aspect of the right atrium immediately ventral to the fixed membrane at the level of the opening for the caudal vena cava. No other aberrant vessels could be found. The thoracic duct was mildly distended but did not have abnormal connections to the venous system.

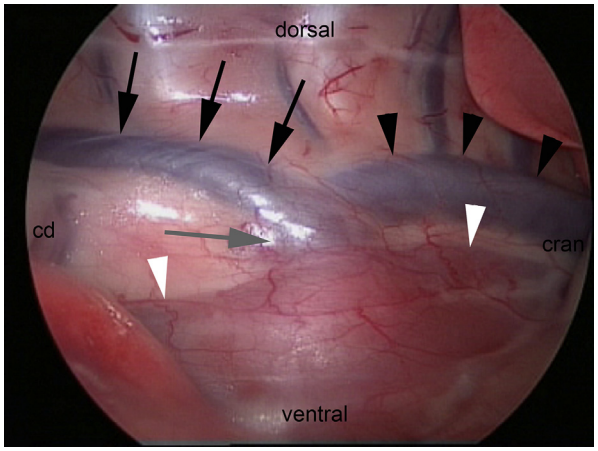

Fig. 3 Thoracoscopic image from the right thoracic cavity; orientation of the patient indicated with cd (caudal) and cran (cranial), respectively, in dorsal and ventral views. The azygos vein (black arrows) was identified by the dorsal intercostal veins (not labeled in the dorsal aspect of the image). It was connected with the left atrium through an aberrant vessel (gray arrow) which was mostly covered by the esophagus (dorsal margin indicated by white arrow heads). The cranial aspect of the azygos vein (black arrow heads) connected the cranial vena cava (not included in the image) with the left atrium over the shunting vessel.

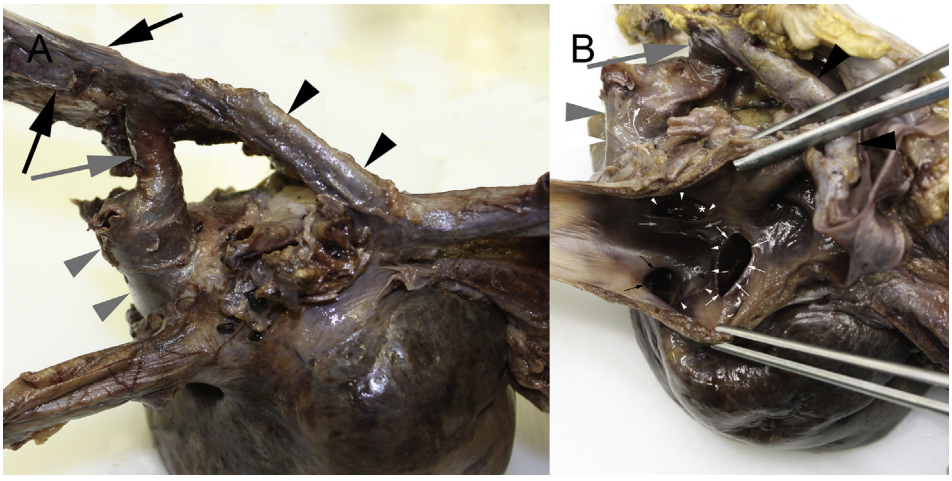

Fig. 4 A and B: Necropsy of the heart; A: focus on vascular connections and B: dissection of the caval system and right atrium. A: The trachea, bronchi, esophagus, and pericardium were removed. The azygos vein (black arrow heads cranial and large black arrows caudal to the shunt) gave rise to an aberrant vessel (large gray arrow) draining into the left atrium (gray arrow heads). B: A large membrane separated the systemic venous sinus from the right atrial body in a horizontal oblique plane. The cranial vena cava (between the forceps on the right side) only had a small, irregularly defined opening into the main chamber of the right atrium (white small arrows). The caudal vena cava had a large opening with smooth margins (white arrow heads). An additional, well-defined parenchymal band (small gray arrows) was located in a vertical orientation between the openings for cranial and caudal venous blood flow. The position of the membrane and the parenchymal band guided blood from the caudal vena cava to the patent foramen ovale (white asterisk), disturbed blood flow from the cranial vena cava, and separated cranial from caudal venous blood flow. The coronary venous sinus (small black arrows) emptied immediately ventral to the membrane at the level of the opening for the caudal vena cava into the body of the right atrium. The azygos vein (black arrow heads) was connected with the cranial vena cava and with the left atrium (gray arrow head) by an aberrant vessel (large gray arrows).

Discussion

In the dog of this report with exercise intolerance since puppyhood and development of chylothorax at 2 years of age, several imaging modalities visualized an azygos vein branch aberrantly emptying non-oxygenated blood into the left atrium, a patent foramen ovale with right-to-left shunt, and a cor triatriatum dexter. These findings were considered the hemodynamic cause of the right-to-left shunts. Cor triatriatum dexter or division of the right atrial components is a congenital malformation in people and dogs in which case a membrane divides the right atrium into two chambers [1—6]. During fetal life, the right sinus venosus valve directs oxygen-rich blood from the caudal vena cava to the foramen ovale. Regression of the right sinus venosus valve leads to formation of the crista terminalis, the Thebesian valve, and the Eustachian valve. Cor triatriatum dexter develops owing to failure of the right sinus venosus valve to regress during embryogenesis. This intra-atrial membrane restricts atrial blood flow, causing a pressure gradient across the right atrium. It typically causes an obstruction to the caudal vena cava venous return [4,7]. In the present case, there seemed to be also obstruction from cranial because the aberrant azygos shunted from right to left, implying a high pressure in the cranial vena cava. This may occur in people with confined superior caval blood flow within the proximal right atrial chamber, restricted by the right venous valve [6]. Other right intra-atrial membranes have been described in humans, such as persistent Eustachian valve [8—12] or Chiari network, but these do not cause any significant hemodynamic obstruction to blood flow [13,14]. Based on preferential streaming of blood from the caval system to the left atrium through a patent foramen ovale, these membranes represent an 'anatomical theory' for the explanation of a right-to-left interatrial shunt with normal right atrial pressures [15]. The membrane and band in the presented case could have played an important anatomical role in 'guiding' flow toward the patent foramen ovale.

In the dog of this report, despite a right-to-left shunt in two locations, invasive pressure measurements could not demonstrate an elevated pressure in the systemic venous system, a gradient along the cranial vena cava or a gradient across the membrane in the right atrium. Nevertheless, right-to-left shunting through the aberrant azygos vein reflects a higher pressure in the

cranial vena cava. Hemodynamically significant right-to-left shunt can occur in people with normal right-sided pressures [15]. An additional remarkable finding was the change in oxygen saturation associated with changing heart rates. If it is accepted that the intermittent drop in oxygen saturation in this anesthetically completely stable and unremarkable dog was purely due to more right-to-left shunt, this finding implies that small changes in (venous) pressure have significant hemodynamic effects. It is unresolved if the chylous effusion was a result of the congenital defects or if this was due to an unexplained cause, i.e., idiopathic. Pathology confirmed the cor triatriatum dexter but failed to document an additional explanation for elevated cranial vena cava pressure.

The foramen ovale closes functionally and anatomically after birth because of the increase in left atrial pressure and drop in right atrial pressure. Patency may persist if the right atrial pressure remains higher than the left atrial pressure, resulting in a right-to-left shunt as documented in the presented case [16]. In cases with restricted venous drainage, the patent foramen ovale can act as a 'pop off' valve to prevent accumulation of effusion. In these cases, hypoxemia secondary to the right-to-left shunting explains the exercise intolerance [17]. In our dog, life-long exercise intolerance was thought to be caused by the right-to-left shunts, but this 'pop-off' valve did not prevent chylous effusion. As indicated, a cause-and-effect relationship between the congenital defects and the effusion in this dog is not proven.

Persistent left superior/cranial vena cava is a vascular anomaly in people [18] and dogs [19], and this typically drains into the coronary sinus in most cases. Development of unroofing of the coronary sinus varies between the cases, and entrance of the vein into the roof of the left atrium represents a more extreme form of this congenital malformation that occurs in 8% of the cases of persistent left superior vena cava in people. Persistent connection of the left superior vena cava with the left atrium is explained by failure of incorporation of the left common cardinal vein and the left sinus horn during the invagination of the sinus horns and the left atrium [20—22]. The embryologic anomaly of left superior vena cava draining into the left atrium can also occur with a normal coronary sinus [23]. A persistent left cranial vena cava draining into the left atrium has been described in a dog [24]. Nevertheless, the dog of the present report did not have a persistent left cranial vena cava, and so this does not seem to be the origin of the anomalous azygos to left atrium connection.

The azygos vein contributes to canine congenital vascular malformations. It often bypasses caval anomalies or relieves pressure from an imperforated cor triatriatum dexter [4,7,25,26]. As an example, in a dog with absent fetal anastomosis of the caudal vena cava with its hepatic portion, persistence of the left azygos vein draining over the coronary sinus into the caudal part of the right atrium bypassed the majority of caudal venous blood [27]. This azygos shunt clearly brought venous unoxygenated blood to the left atrium and therefore was considered to contribute to the congenital exercise intolerance, together with the right-to-left shunting patent foramen ovale.

Lymphaticovenous connections potentially contribute to the chylous effusion in complex congenital defects [7]. Unfortunately, our attempt to visualize the thoracic duct with popliteal CT-lymphangiography of the thoracic duct failed owing to contrast leakage. In the dog of the presented case, no lymphaticovenous connection could be identified in the angiographic imaging studies, thoracoscopy, or necropsy.

To the authors' knowledge, a vascular malformation involving connection of the azygos vein with the left atrium has not been previously described. We describe in the present report a multimodality diagnostic approach for a complex congenital cardiac defect. This was a dog with cor triatriatum dexter with an intracardiac and an extracardiac right-to-left shunt. The hemodynamic relevance of the defects could not be proven by invasive pressure measurements but was evident based on the shunt directions. Balloon dilation of the cor triatriatum dexter seemed to be followed by clinical improvement, but this was only transient, and a true effect is questionable. Using a cutting balloon or placement of a stent might have resulted in a longer lasting effect [5,17].

Conflicts of interest statement

The authors have no conflicts of interest.

Acknowledgements

In memoriam Charlotte Marly.

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jvc.2019.09.002.

Video table

Video	Title	Description
1	Transthoracic two dimensional echocardiography	B-Mode echocardiographic examination in a right parasternal short axis view at the level of the right atrium. An incomplete crescent shaped membrane separated the systemic venous sinus from the right atrial body.
2	Transthoracic two dimensional echocardiography	B-Mode echocardiographic examination in a right parasternal long axis four chamber view. The perforated membrane was oriented in a dorsal oblique plane and an additional, well-defined soft tissue band could be visualized in a vertical orientation.
3	Transthoracic Doppler echocardiography	Color flow echocardiographic examination in a right parasternal short axis view at the level of the right atrium demonstrated a laminar flow pattern along the membrane from the systemic venous sinus into the body of the right atrium.
4	Contrast enhanced echocardiography: cephalic injection	Both portions of the right atrium separated by the intra-atrial membrane enhanced. The contrast did not pass through the patent foramen ovale and was replaced by anechoic blood in the area of the atrial septum. Bubbles exclusively arrived in the left atrium through the aberrant vessel in the roof of the left atrium.
5	Contrast enhanced echocardiography: saphenous injection	Both sides of the membrane enhanced but the contrast medium in the area of the membrane was replaced by anechoic blood. A large amount of the contrast medium escaped through the patent foramen ovale into the left atrium. The aberrant vessel in the roof of the left atrium did not enhance. Bubbles arrived only through the patent foramen ovale in the left atrium.
6	cardiac magnetic resonance imaging study	Short axis plane at the level of the right atrium. The comma shaped incomplete membrane in the atrium did not cause turbulent flow. A small turbulence resulted in complete loss of signal in the blood shunting through the patent foramen ovale.
7	Maximum Intensity Projection of a contrast enhanced magnetic resonance angiography; cephalic injection	Left side: in a caudocranial wiew and right side: in a laterolateral view. The contrast medium filled the right atrium incompletely and enhanced the pulmonary artery. Simultaneously, an aberrant vessel connecting the dilated azygos vein and the left atrium enhanced immediately after contrast injection and in the second pass.

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