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Case Report

A case of systemic pulmonary collaterals in a Doberman Pinscher with dilated cardiomyopathy phenotype

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Abstract
Dilated cardiomyopathy (DCM) in dogs is a common hereditary primary myocardial disease affecting large and giant breeds, predominantly Doberman Pinschers. This pathology is associated with mechanical and electrophysiological dysfunction of the myocardium, which is manifested by inadequate ventricular eccentric hypertrophy. Our report focuses on the case of unusual systemic pulmonary collaterals in a Doberman Pinscher with DCM phenotype. Cardiologic data have shown that myocardial histologic changes may be secondary to the presence of pulmonary collaterals and their potential influence on the development of left ventricular volume overload. Thus, we had every reason to believe that the volume overload that caused the development of congestive heart failure in this dog and possibly sudden cardiac death was caused by the presence of a combined arterial malformation. This case illustrates that atypical bypass manifestations detected during echocardiography can be further evaluated using computed tomography and angiography to further characterize and identify concomitant vascular abnormalities that might otherwise be masked or overlooked, especially when LV volume overload occurs in breeds predisposed to DCM. The research team considers it very important to publish such cases, as this will help the entire veterinary community quickly accumulate knowledge and experience for diagnostic and treatment of similar and other cases.

Keywords: dogs, dilated cardiomyopathy, systemic pulmonary collaterals, Doberman Pinscher.

Introduction

In dogs, the most prevalent congenital heart condition linked to a continuous murmur is the patent ductus arteriosus (PDA). Significant blood discharge through the PDA leads to the LV volume overload, and an increase in LVEDV, LVESV, LVIDd, LVIDs, EPSS. These changes can be falsely taken for markers of DCM in Doberman dogs, since they remain the main criteria for its detection during screening (18). Other less frequent causes of such murmurs include aortopulmonary windows, ruptured Valsalva sinus aneurysms, coronary artery fistulas, arteriovenous connections between the systemic and pulmonary circulations, bronchial collateral arteries, and pulmonary vessels that stem from truncus arteriosus. Alternatively, a continuous murmur can result from turbulent blood flow within major arteries (9).

Congenital communication between the ascending aorta and the main pulmonary artery is a rare but important lesion, as it can mimic the clinical picture of a PDA. In this case, a significant blood discharge from systemic to pulmonary circulation will also lead to an increase in the LV volume and dimensions (2). The specific structural defects that

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create aortopulmonary communication are varied, including an aortopulmonary window, an aberrant vessel joining the great arteries, truncus arteriosus, the anomalous origin of a pulmonary artery from the aorta, an aorticopulmonary fistula, an arteriovenous shunt, an aortopulmonary vascular malformation, and a persistent 5th aortic arch resulting in systemic-to-pulmonary blood flow (12).

Abnormal vascular connections between the systemic circulation (especially the aorta) and the pulmonary artery can cause pathophysiological clinical conditions very similar to those found in PDA and must always be considered among the differential diagnoses of a PDA. In dogs, the literature reports of these vascular malformations are still anecdotal, and studies on the pathogenesis of these malformations are lacking (14). They have been called a variety of names: broncho-esophageal aberrant arteries, systemic pulmonary collaterals, major aortopulmonary collateral arteries (MAPCAs), and systemic-pulmonary arteriovenous fistulas (SPAVF) (2). MAPCAs arise from the embryonic splanchnic vascular plexus, which, under normal conditions, regress with the formation of the normal pulmonary arterial system (2). In humans, congenital MAPCAs can be associated with or without heart defects. Those with heart defects tend to have cyanotic defects. MAPCAs without cyanotic congenital heart defects are rare and associated with premature birth, lung tissue infection, and pulmonary dysplasia (22).

Abnormal connections called systemic-to-pulmonary collateral arteries often form in children due to factors like congenital heart disease, prematurity, or chronic lung infections. The prevailing theory is that these connections develop from the embryonic vascular tissue that normally differentiates into the pulmonary and bronchial arteries. Intriguingly, one investigation suggested that the prevalence of such undetected collateral arteries is 1.57% in the general population of healthy infants aged one month (22). Therefore, it was estimated that systemic-pulmonary collateral arteries may be present normally after birth and then gradually disappear. However, if there are certain predisposing factors, they may persist in order to augment pulmonary flow (22). In dogs, the most frequent clinical and anatomical presentation resembles the MAPCAs known in humans. However, in most cases in dogs, these are not associated with any congenital or acquired cardiac disease causing a reduction in the pulmonary flow (2). The reason these blood vessels persist in some animals is unclear; it could be speculated that a genetic predisposition exists or that these blood vessels develop in the perinatal period in animals that are underdeveloped at birth, as is observed in very low birth weight premature infants. According to a study, small or large collateral vessels are observed in over 50% of premature infants needing prolonged ventilatory support (2).

One study described clinical, radiological, echocardiographic, and angiographic data in dogs with SPAVF (3). In veterinary medicine, the etiology of SPAVF is unknown, and the hypothesis of a component related to respiratory disease is not supported by our study because none of the dogs included in this study had clinical and/or radiographic signs of respiratory disease. The cases of SPAVF seen in the study (3) all arose from the descending aorta distal to the location of the ductus arteriosus.

In veterinary publications, the term arteriovenous fistula is often used to designate an abnormal connection of vessels of the systemic and pulmonary circulation (3, 14), which rather corresponds to MAPCas and other pathologies with a left-to-right shunt (11). The most common type of arteriovenous pulmonary fistula is the connection of the lobar pulmonary artery with the lobar pulmonary vein with the possible formation of a lobar sequestrum. In this case, a right-to-left shunt is formed. A similar abnormal structure of the shunt is described in a medical article (10). It is a left-to-left shunt; the authors call it a systemic arteriovenous fistula (15).

Definitions of shunt flows such as “right-to-left” and “left-to-left” appeared in the medical literature describing vascular malformations quite a long time ago (15), even in the era of the absence of echocardiography. Apparently, they were based on the degree of blood oxygenation, which was determined using cardiac catheterization. So, blood oxygenation in the aorta decreases when blood is discharged through the right-to-left shunt, which leads to the appearance of cyanosis. Blood oxygenation in the pulmonary artery will increase with left-to-right shunting. At the same time, with left-to-left shunting, blood oxygenation in the aorta, pulmonary artery and right ventricle (RV) will not change. However, the LV will enlarge due to volume overload during left-to-left shunting (15).

At least two types of vascular anomalies were identified in our clinical case. The first type is arterial aberrant vessels extending from the descending part of the thoracic aorta and flowing into the pulmonary artery or left-to-right shunts. They are also called major aortopulmonary collateral arteries (MAPCAs). In our case, the pattern was similar to most clinical cases described in the literature (3, 4, 5).

The second type of anomaly was associated with the presence of two bilateral arteries arising from the abdominal aorta, anatomically corresponding to the phrenicoabdominal arteries. These arteries flowed into the veins of the pulmonary lobes of the left and right lungs, passing through the diaphragm along the mediastinum. In the medical literature, such shunts are called arteriovenous fistulas (1, 11).

In the case we are describing, the definition of arteriovenous pulmonary fistula in the classical sense of the right-to-left type is unsuitable, but it reflects the essence of the anomaly in the connection between the artery and vein. In our case, the fistula connects the systemic and pulmonary circles and extends beyond the lung lobe and the chest cavity. It is a left-to-left shunt (10). We suggest using the abbreviation AAPVF – abdominal artery-to-pulmonary vein fistula. The classification proposed in medicine does not reflect the characteristics of such a connection despite isolated described cases (11).

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There are limited reports in veterinary medicine of communication abnormalities between dogs' aorta and pulmonary circulation. Systemic-pulmonary arteriovenous shunts originating in the abdominal aorta are especially rare; even in humans, these are isolated cases that have been described (10, 12, 14, 15). Thus, our report of unusual systemic pulmonary collaterals in a Doberman Pinscher with a DCM phenotype is of great interest to the veterinary community, which is the objective of our publication.

Case description

A two-year-old Doberman, who was previously diagnosed with DCM at the age of 7 months in another clinic, was admitted to the clinic for the heart check-up. Initially the dog was presented to another clinic, because the owner was informed about the problem of DCM in Doberman. The dog had been administered pimobendan 0.3 mg/kg/day and spironolactone (volumetric overload and increased troponin were noted, there were no complaints about well-being). The dog had been vaccinated and according to the owner didn't show any complaints about health condition: the dog was active, without deviations in height, build or body weight. Upon physical examination, a grade 2 systolic-diastolic murmur was detected in the area of the base of the heart on the left, and minor inspiratory dyspnea, a hard pulse, and no arrhythmias were detected. During auscultation of the lung, hard breathing was determined. During transthoracic echocardiography (TTE), there was no B-line, LV volume overload was noted (Fig. 1A, 1B), Systolic-diastolic flow was also detected in the area of the left branch of the pulmonary artery after bifurcation (Fig. 1C). The systolic velocity still did not exceed 2 m/s (Fig. 1D), which did not correspond to the expected gradient between the aorta and the pulmonary artery in PDA. Since this flow could be the cause of the appearance or aggravation of volumetric LV overload, it was decided to conduct additional diagnostics, and the dog was directed to the computed tomography (CT). The ECG results revealed left axis deviation. Single VPCs were less than 170 bpm.

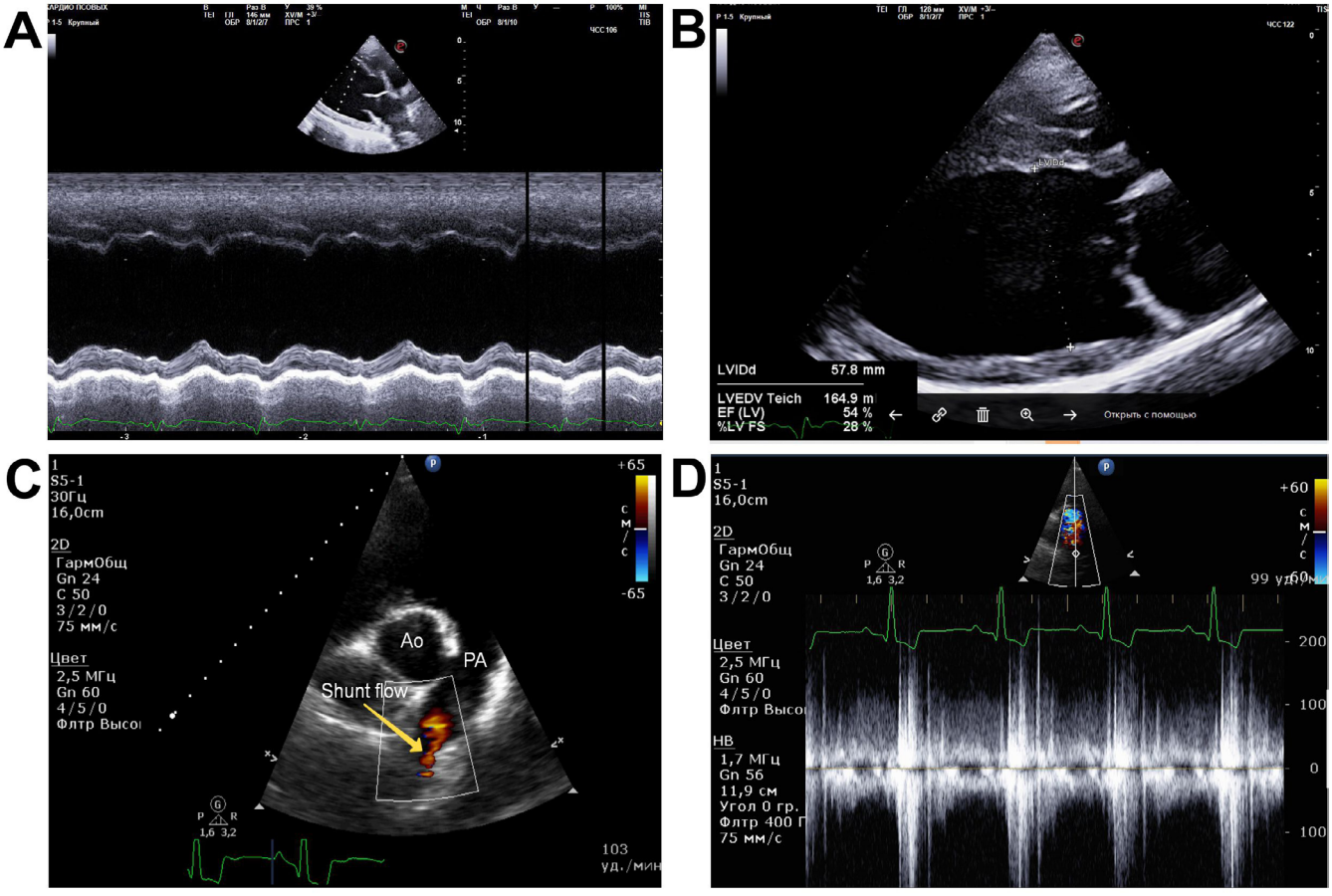

Figure 1. TTE examination. (A) M-mode, parasternal position, sings of the volumetric LV overload. (B) B-mode, the parasternal position long axis, LV volume overload, significantly increased CDR and slightly increased DAC, FS is normal, which indicates satisfactory contractility. (C) B-mode, parasternal position short axis, the diastolic flow in the trunk of the pulmonary artery (arrow). (D) CW-mode, the shunt flow profile is systolic-diastolic with a maximum velocity of up to 2.10 m/s.

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The dose of pimobendan was increased to 0.5 mg/kg/day, and ramipril, spironolactone, and torasemide were also prescribed. It was also recommended that the respiratory rate be counted in deep sleep. Two weeks later, shortness of breath was not noted (the sleeping respiratory rate decreased from 29 to 18 per minute).

As the result of the CT examination, the following was discovered. Pronounced dynamic heart rate artifacts – it is impossible to overtake multiple pathological long curved crimped arterial vessels coming from descending aorta in level 4-6 thoracic vertebras lies in mediastinum around and dorsally to the esophagus, apparently flows into vein of caudal lobe of right lung (Fig. 2A). From brachiocephalic artery departs curved pathologic artery that forms a loop on the right in the cranial mediastinum dorsally and goes to the caudal mediastinum under the trachea. It is not possible to determine the location of its confluence; it cannot be ruled out that it drains into the cranial vena cava or the vein of the cranial lobe of the right lung (Fig. 2B). Left and right phrenicoabdominal artery extends cranially from abdominal aorta, right one comes in the thorax together with vena cava, forms multiple loops lies lateroventrally (Fig. 2C). Left phrenicoabdominal artery lies in caudal mediastinal reflection forms multiple lops tortuous in mediastinum, connected (looks like that) to the vein of caudal lobe of left lung. The vein of the middle lobe of the right lung is dilated distally; we cannot exclude vessel anomaly in this area (presumably, the right phrenicoabdominal artery may flow into the vein of the middle lobe). In addition, the high-density structure was visualized connecting the aorta and pulmonary artery trunk; most likely, it was an open aortic duct. The pattern was closer to the type 1 according to Miller W.M. (Fig. 2D). Based on the received information, it was decided to carry out a surgical correction of the PDA.

The patient underwent an angiographic examination of the aortic arch and its descending part to identify and occlude the PDA.

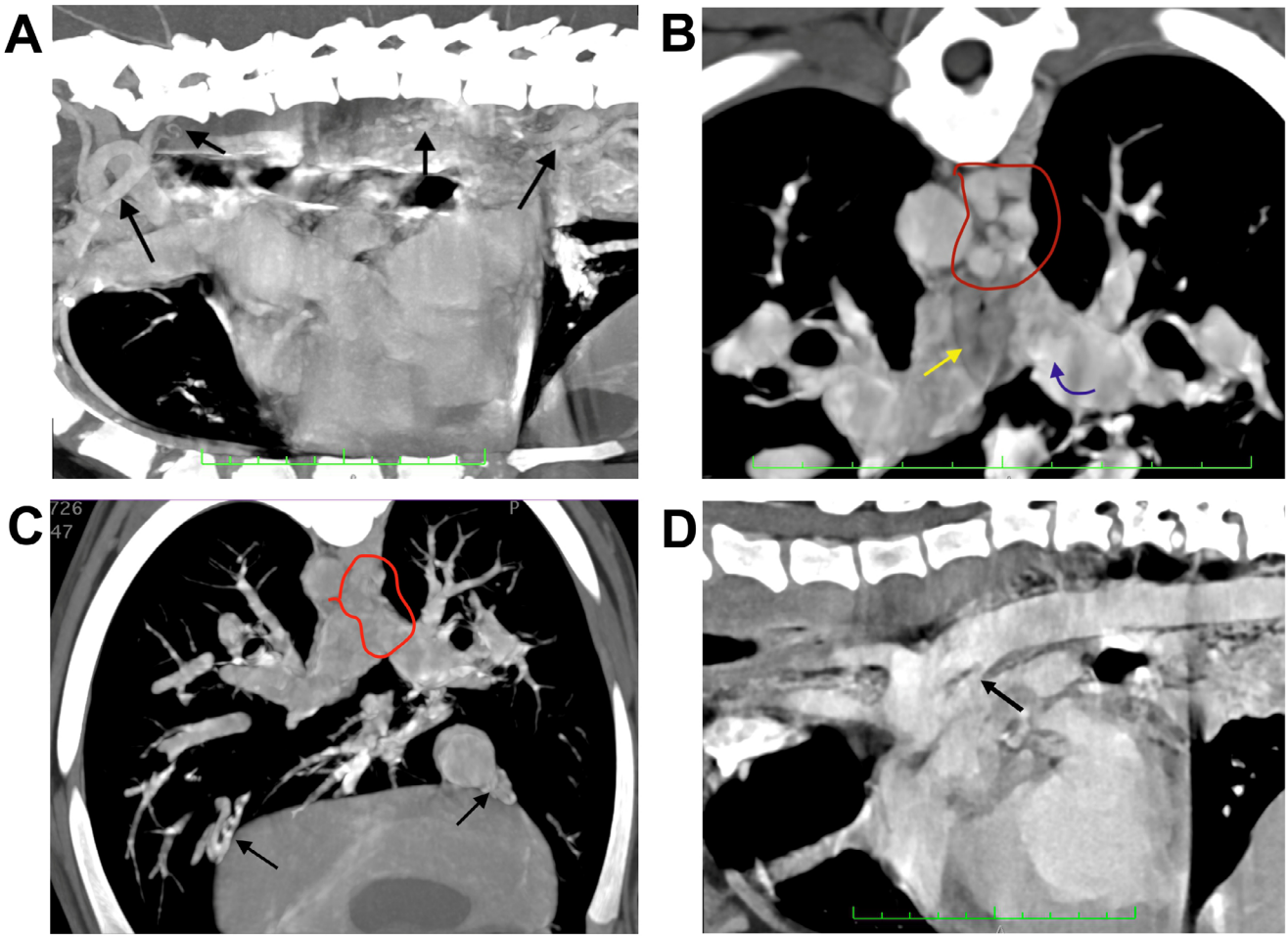

Figure 2. CT scan results. (A) Loops of pathological arteries in mediastinum (first arrow - artery arising from the brachycephalic trunk, forming a loop). (B) Pathological artery arising from the descending aorta (red circle), esophagus (yellow arrow), vein of the caudal lobe of the right lung at the level of the pathological vessel entering it. (C) Right and left phrenicoabdominal arteries (black arrows) and a tortuous arterial vessel arising from the thoracic descending aorta (red circle). (D) High density structure connecting aorta and pulmonary artery trunk (arrow), most likely this is open aortic duct.

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The study was conducted using the Philips Zinition 50 EOP. For the surgical access, a section of the right femoral artery was performed, the vessel was taken for turnstile sutures, and an introducer 7 FR was inserted into the lumen. An angiographic catheter with a size of 5 FR was inserted into the aortic arch for external angiography. Angiography was performed with a nonionic iodine solution at the rate of 1.5 ml per kg of body weight. After the surgery, the introducer was removed. The defect in the vessel was closed with a continuous seam with a polypropylene thread USP 7-0. The skin incision was closed with a Halsted suture.

Angiography did not reveal PDA. When a contrast agent was administrated into the aortic cavity, an increase in contrast was observed in the region of the esophagus, in the region dorsal to the descending aorta, and convoluted vessels were also revealed in the region of the caudal mediastinum, giving contrast to the pulmonary vessel (phrenicoabdominal artery) (Fig. 3B). These data correspond to the results of CT examination. In the same area, the left phrenicoabdominal artery was visualized in the caudal mediastinum (Fig. 4).

Three months later, repeated examination revealed a negative trend in relation to the degree of volumetric overload, and Holter monitoring was carried out. It revealed an increase in the average heart rate, obviously due to an increase in sympathetic tone. Single VPCs, without life-threatening coupling intervals (Fig. 5A). An increase in sympathetic tone is compensatory to a decrease in cardiac output or may also be a consequence of stress during Holter monitoring (Fig. 5B). Also, an increase in sympathetic tone may be associated with the progression of chronic heart failure against the background of volume overload.

Repeated echocardiography showed an increase in LV volume overload. During the therapy the dog's condition remained satisfactory, and the sleeping respiratory rate did not exceed 18 per minute. Activity persisted, there were no complaints of shortness of breath or decreased physical activity. The treatment plan included additional angiography to determine the possibility of surgical closure of the shunt.

A month later, the dog died as a result of sudden cardiac death on the background of satisfactory physical condition. Subsequently, a post-mortem autopsy

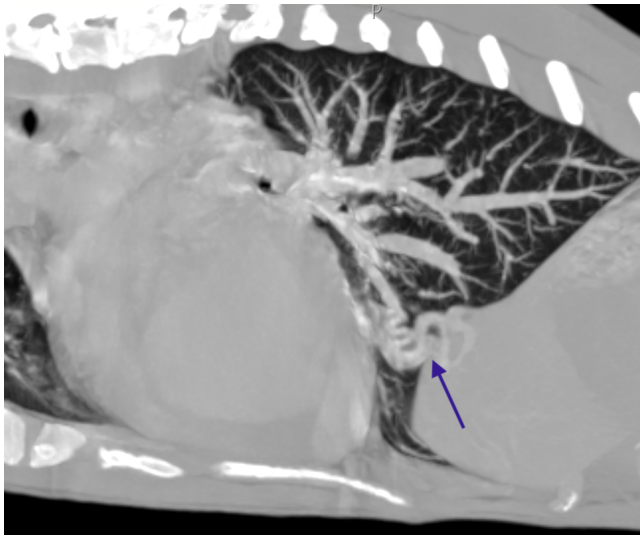

Figure 4. CT scan of the caudal mediastinum. Left phrenicoabdominal artery lies in caudal mediastinal reflection forms multiple lops tortuous in mediastinum (arrow).

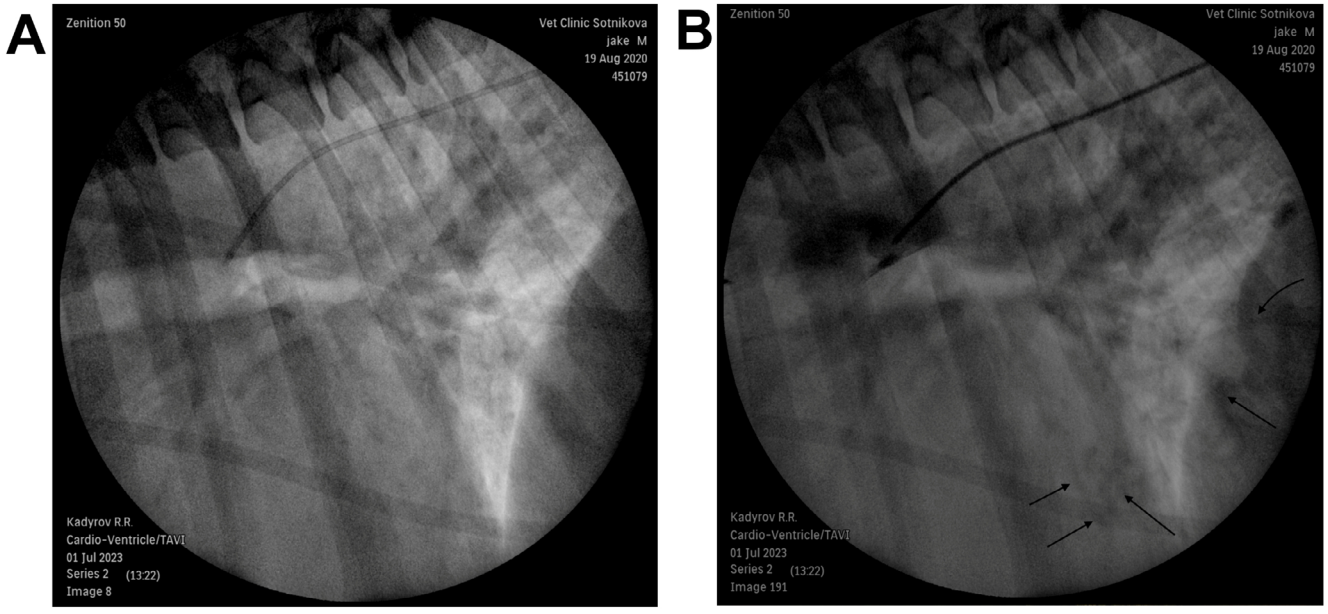

Figure 3. Angiographic examination. (A) Image before contrast medium injecting. (B) Image after contrast medium injecting, convoluted vessels in the region of the caudal mediastinum, contrasted phrenicoabdominal artery (arrows).

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was performed, which revealed the following: multiple extraorgan convoluted, dark blue, narrow, elastic pathological blood vessels are visualized, extending from the abdominal aorta of the epigastric region, gastric veins and common mesenteric veins, abdominal and thoracic esophagus, thoracic trachea, thoracic aorta, pericardium, cranial mediastinum, pericardial-diaphragmatic ligaments, which connect into one vessel with a diameter of about 0.4 cm (Fig. 6A, 6B, 6C, 6D). The latter flows into the left branch of the pulmonary artery before branching into branches to the three lobes of the left lung lob (Fig. 6E, 6F).

Macroscopic heart examination revealed moderate enlargement of the heart due to an increase in the LV (Fig. 7A). The heart sections showed moderate LV dilation with moderate thinning of its wall, and the thickness of the heart at the base of the papillary muscle was 1.8 cm (Fig. 7B). It was also discovered a moderate smoothness of the LV papillary muscles. Multiple foci of lightening of the myocardium (to an almost white color) of the LV up to 3.5 cm, with blurred borders (more pronounced in interventricular septum). Presumably, the expansion of the LV was partially due to the presence of the above-described systemic pulmonary vascular shunt (systemic pulmonary

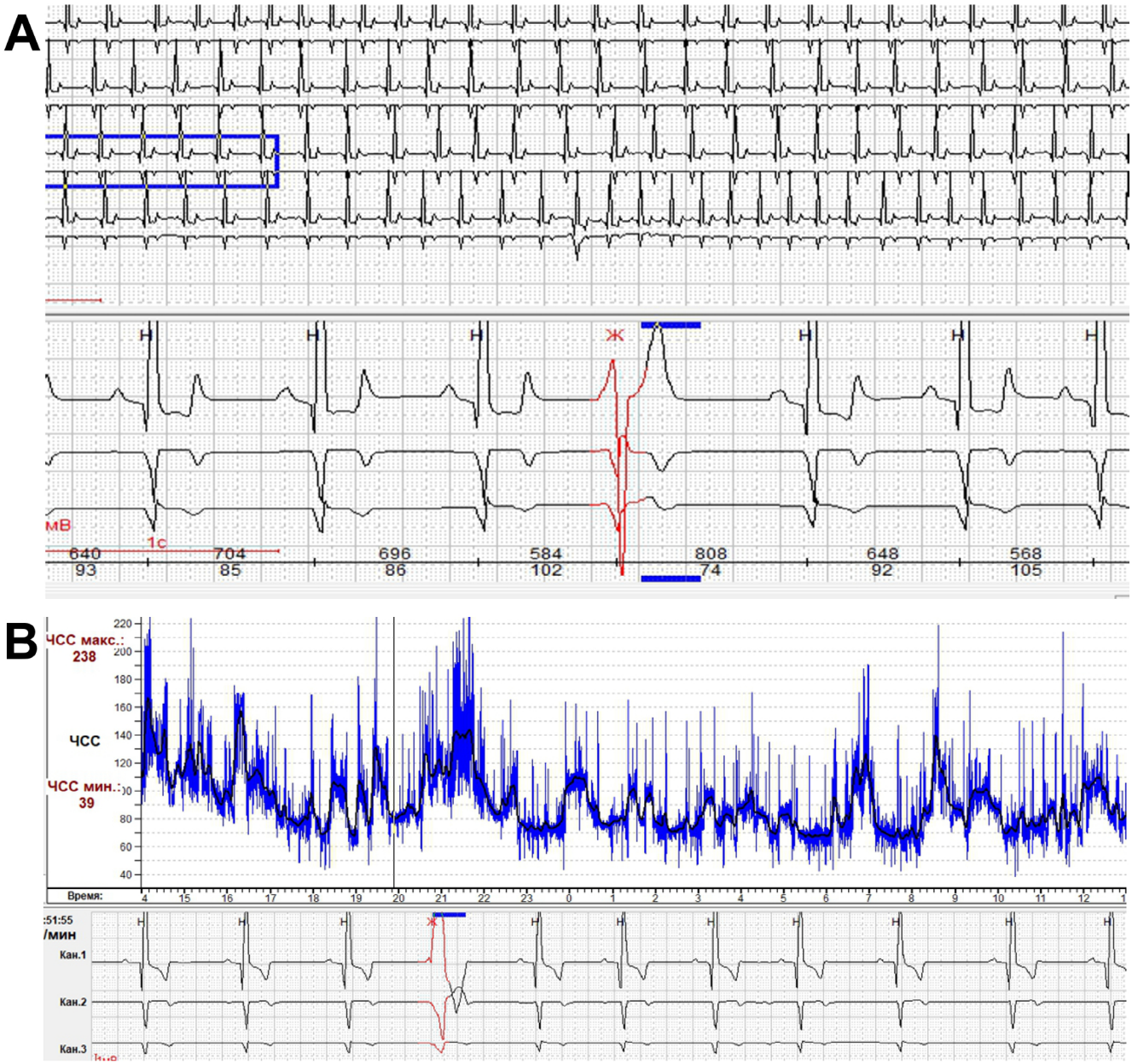

Figure 5. Holter monitoring. (A) The identified VPCs are not life-threatening, coupling intervals were less than 170 bpm. (B) Tachogram, there is a general increase in heart rate during the day, which may indicate an increase in sympathetic tone.

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collateral arteries) in the animal. Examination of the RV showed moderate hypertrophy with an area of moderate severity (thickness 1.4 cm). The myocardium of the RV on the incisions was without visible changes (Fig. 7C).

Material for histological examination was taken from the different parts of the heart: walls of the right and left ventricles and interventricular septum. Microscopically myocardial lesions were manifested in the form of lymphocytes and plasma cells, as well as fat vacuoles in the myocardial stroma (Fig. 8A). Focal leukocyte infiltrations and cardiomyocyte dystrophic changes were found in many myocardial fragments (Fig. 8B). In some cases, foci of myocardial fibrosis with cardiomyocyte atrophy were detected (Fig. 8C, 8D). There were no reliable signs of significant hypertrophy in the presented sections. These morphological changes may indicate DCM (6, 17). The following criteria of DCM were taken into account: atrophy and attenuated wavy fiber type of cardiomyocytes, edema, subendocardial fibrosis, dystrophic changes in cardiomyocytes (cardiomyocytolysis, degeneration, vacuolization), severe fibrosis, fatty infiltration (17). However, considering the results of echocardiography, CT, and angiography, histological changes in the heart may be secondary to the detection of pulmonary collaterals and their potential impact on the development of left ventricular volume overload.

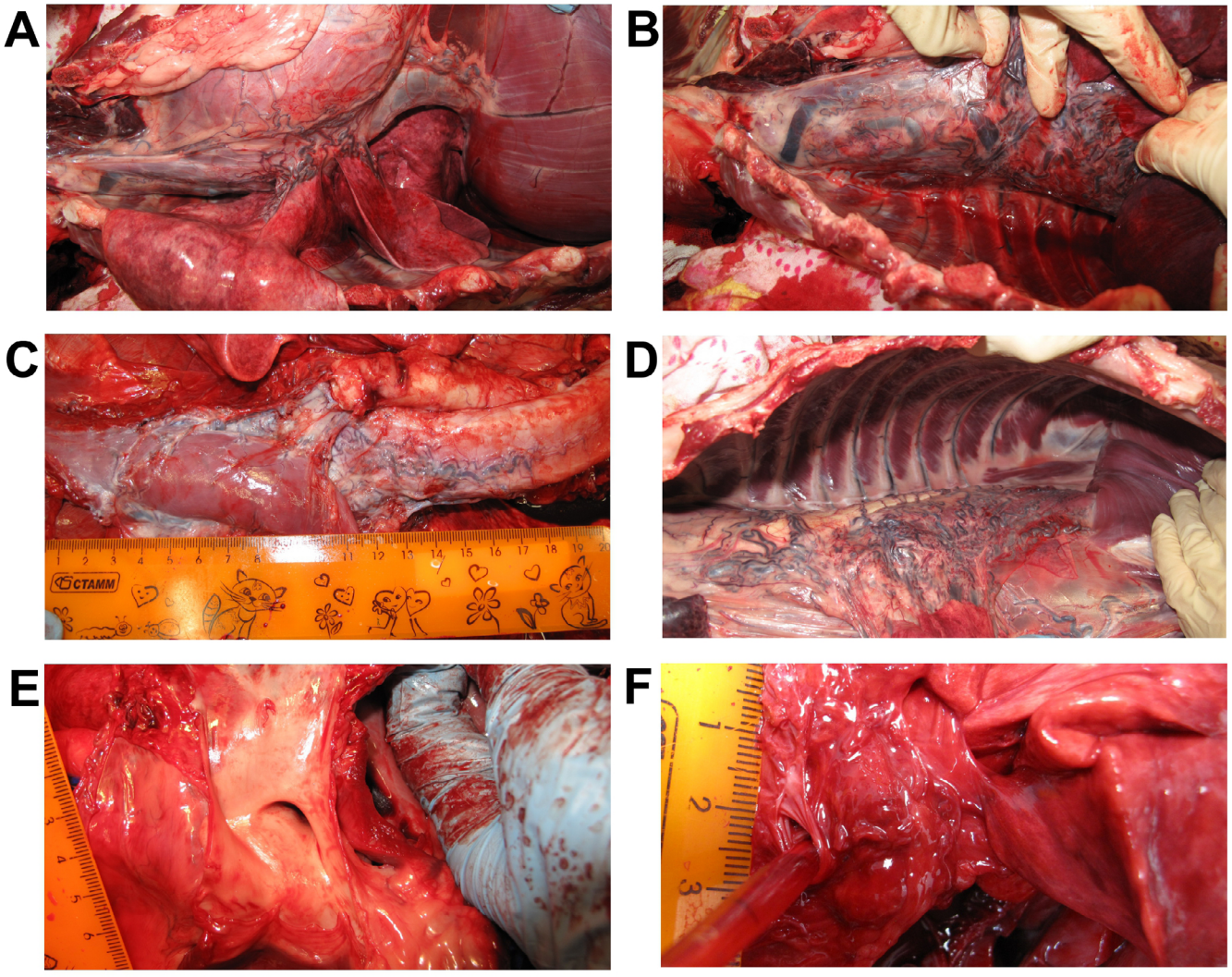

Figure 6. Macroscopic examination. (A) Right pleural cavity, multiple abnormal vessels in the pericardium, right pericardiodiaphragmatic ligament and on the surface of caudal vena cava. (B) Right pleural cavity, multiple abnormal vessels in the cranial, middle and caudal mediastinum. (C) Multiple abnormal vessels on the surface of the thoracic trachea and thoracic esophagus. (D) Left pleural cavity, multiple abnormal vessels on the surface of thoracic aorta, thoracic esophagus, left pericardiodiaphragmatic ligament and in the cranial mediastinum. (E) Pathological passage in the left pulmonary artery (before its division into lobar branches of the left lung). (F) Another view of the same pathological vessel as in Figure E, partially showing lumen and vascular endothelium.

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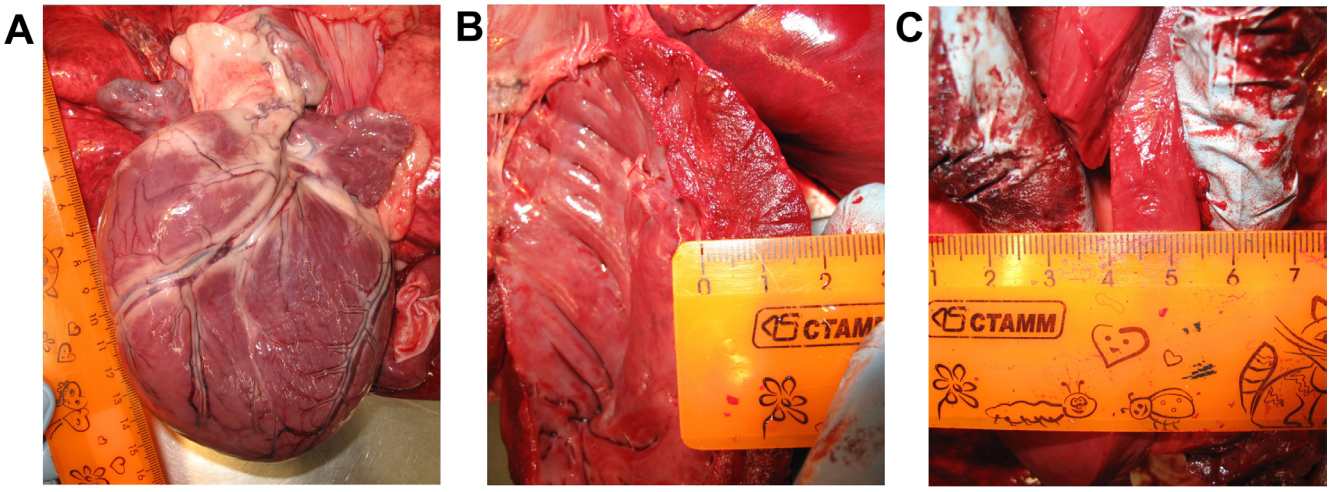

Figure 7. Heart macroscopy. (A) Heart on the external examination, enlargement of the LV cavity (based on the landmark in the form of displacement of the anterior interventricular groove to the right). (B) Thinning of the LV wall up to 1.8 cm (measurement was taken at the base of papillary muscle). (C) Thickening of the RV wall up to 1.4 cm.

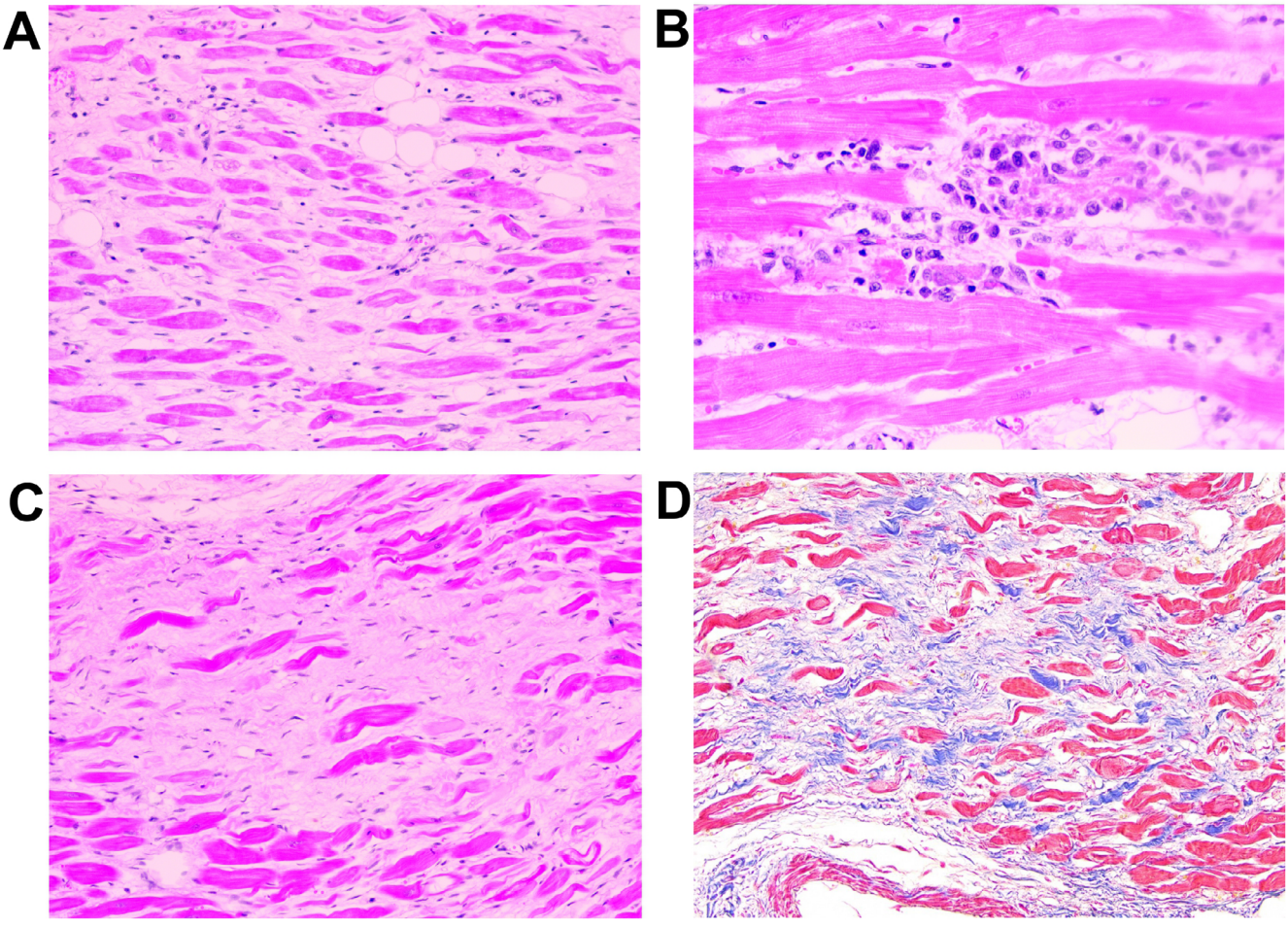

Figure 8. Myocardial microscopy. (A) Lymphocytes, plasma cells and fat vacuoles in the myocardial stroma (H-E staining, x200). (B) Fragment with leukocyte infiltration and dystrophic changes in cardiomyocytes (H-E staining, x400). (C) Focus of myocardial fibrosis with cardiomyocyte atrophy (H-E staining, x200). (D) Fibrous tissue in the myocardial stroma stained blue (Masson trichrome staining, x200).

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Discussion

Dilated cardiomyopathy in Doberman Pinschers is a common, inherited, primary myocardial disease that usually has a late onset. It is a disease of the myocardium associated with dilatation and impaired contraction of the ventricles (16). In dogs, it primarily affects large and giant breeds, with the Doberman Pinscher being one of the most frequently affected (20).

The condition then moves into the occult or Stage II phase (19), where initial subclinical derangement is detectable. This derangement includes either a structural change — Left Ventricular (LV) enlargement in contraction, relaxation, or both — or an electrical change, specifically the presence of Ventricular Premature Contractions (VPCs). Finally, Stage III represents the overt stage, when definitive clinical signs of heart failure emerge. Patients often retain normal exercise tolerance until congestive heart failure and pulmonary edema have already developed (18, 19). Ultimately, DCM represents a diverse group of myocardial disorders that feature mechanical and/or electrical dysfunction, often accompanied by inappropriate ventricular hypertrophy (4).

Congenital or acquired cardiac diseases other than DCM might also cause volume overload, systolic dysfunction, or both, and need to be excluded accordingly. Screening for occult DCM in Dobermans should start at the age of three and include both Holter monitoring and echocardiography (18). The preferred echocardiographic method is measuring the left ventricular volume by Simpson's method of discs (4).

One big study on the distribution of age groups of Dobermans with DCM shows a very low percentage of stage 2 DCM (with detectable changes in the form of LV volume overload or arrhythmia). Only 3.3% of Dobermans fell into the DCM group between the ages of 1 and 2 years old (20). In our clinical case, LV volume overload was detected in the dog at the age of 7 months. Therefore, the cause of enlarged heart size cannot be ignored even if it is not found at present. MAPCAs may be a potential cause of heart enlargement. Due to technical limitations and pulmonary air interference, MAPCAs are often undiagnosed on echocardiography imaging. In our case, the detected vascular malformation could also cause LV volume overload.

In one clinical case (21), a 9-year-old boy with a history of a dilated LV was admitted to the cardiovascular center of Beijing Children's Hospital. The dilated LV was accidentally diagnosed due to a history cough and pneumonia at another medical center 5 years ago, and the underlying cause was not found. Several imaging modalities were performed. Initial evaluation via echocardiography at an outside facility noted moderate LV dilation alongside hyperdynamic wall motion. The Color Doppler signal confirmed significant systemic-to-pulmonary blood shunting on suprasternal long-axis imaging, which immediately raised suspicion for MAPCAs. This diagnosis was subsequently confirmed by enhanced cardiac CT. Selective angiography provided detailed anatomy, showing thickened, tortuous MAPCAs arising from both the thoracic and upper abdominal aortas to supply the left and right lungs. The collaterals were successfully embolized: the main bodies were closed using 6 mm and 3 mm diamond spring coils, resulting in the occlusion of vessels measuring 3.8 mm and 1.8 mm in diameter, respectively. At the one-month follow-up, the patient was doing well. The intervention achieved a significant reduction in cardiac chamber size, evidenced by the Left Ventricular End-Diastolic Diameter (LVEDD) decreasing from 49 mm to 40 mm (placing it within the normal range of 38.8–42.19 mm) (21).

This case described in medicine proves that even a relatively small diameter of the systemic pulmonary shunt into the trunk of the pulmonary artery can lead to a significant volumetric overload of the LV, which was eliminated after surgical closure of the blood discharge through this shunt.

In our case, the diameter of the vessels flowing into the left pulmonary artery was also relatively small (4 mm). However, the convoluted network of collaterals indicates the possibility of developing LV volumetric overload against this background. Indirect signs can also be considered hypertrophy of the RV, which developed as a result of overloading the volume of the pulmonic circle of blood. Also, the presence of AAPVF contributed to an increase in pulmonary circulation. As noted (10), the presence of such fistulas leads to volume overload of the LV due to left-to-left shunting (2, 3, 4).

Fetal lung development begins with the vascular plexus of the lung buds connecting to segmental arteries from the dorsal aortae. Around day 40, this plexus matures, and the lung is dual-perfused by the RV (via the sixth branchial arches) and these aortic segmental arteries. This dual-supply state is temporary, as the segmental arteries typically disappear about 50 days after ovulation. Normally, the lung transitions to being supplied solely by the central pulmonary arteries derived from the sixth branchial arches. The current findings — that all collateral arteries arose uniformly from the midthoracic aorta — are significant. Since these arteries originated above the normal descent zone of the segmental arteries (from the neck to the diaphragm), the observation strongly suggests that a failure or interruption of normal fetal vascular development occurred at approximately the same embryonic age in every case of the present series (8).

Aortopulmonary vascular malformations are complex, can cause clinically relevant volume overload, and can mimic PDA even on Doppler echocardiography, although certain clinical and echocardiographic features should prompt suspicion of their presence (7). Diagnosis can be confirmed on selective angiography and ANGIO-CT, which also determine an appropriate approach. Surgical ligation is a viable occlusion method and will decrease shunting through abnormal collateral vessels and volume loading of the heart. Resolution of congestive heart failure is possible (3).

In conclusion, the histological changes in the heart may be secondary to detecting pulmonary collaterals and their potential impact on the development of LV volume overload. Thus, we have every reason to believe that the volume overload that caused the development of congestive heart failure

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in this dog and possibly sudden cardiac death was caused by the presence of a combined arterial malformation.

This case illustrates that atypical bypass manifestations detected during TTE can be further evaluated using CT and/or angiography to further characterize and identify concomitant vascular abnormalities that might otherwise be masked or overlooked, especially when LV volume overload occurs in breeds predisposed to DCM. Since each case of anomaly development is unique, its detailed study in all possible ways is very important from the point of view of gaining experience and understanding the options for the formation of pathologies. In our case, it was shown that the cause of LV volume overload in a Doberman can be not only DCM or PDA, but it also can be caused by rarer and difficult to diagnose vascular malformations. An important diagnostic criterion was the dog's too young age for diagnosing DCM, since Wess G. (2010) showed that DCM in Dobermans can extremely rarely occur in dogs under two years of age (18). This fact forced us to approach the exclusion of a shunt more carefully during echocardiography, which was detected despite poor visualization. It was subsequently confirmed by CT, angiography and autopsy. The authors consider it very important to publish material when identifying such cases, as this will help the entire veterinary community quickly accumulate knowledge and experience for diagnosis and treatment of similar and other cases.

Conflict of Interest

The authors declare no competing interests.
The dog's owner approved the publication of the case.

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