Clinical presentation, echocardiographic findings, treatment strategies, and prognosis of dogs with myxomatous mitral valve disease presented with pericardial effusion due to suspected left atrial tear: a retrospective case–control study

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Clinical presentation, echocardiographic findings, treatment strategies, and prognosis of dogs with myxomatous mitral valve disease presented with pericardial effusion due to suspected left atrial tear: a retrospective case—control study☆

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Abstract *Introduction/objectives:* Left atrial tear (LAT) is a life-threatening complication in dogs with myxomatous mitral valve disease (MMVD). The study objective was to describe clinical presentation, echocardiographic findings, treatment

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Atrial splitting;
Atrial rupture

strategies, and survival in dogs with LAT compared to a control group of dogs with a similar stage of MMVD but no LAT.
Animals and materials and methods: two-center retrospective case—controlled study including 15 dogs with and 15 dogs without LAT was conducted. Clinical and echocardiographic data were reviewed, and survival information were collected.
Results: Nine dogs in each group were in stage C of MMVD, while the remaining were in stage B2. No differences between groups were found regarding age, body weight, sex, kidney values, and echocardiography-derived cardiac dimensions. Most reported clinical signs associated with LAT included weakness, respiratory signs, and syncope. Treatment varied and was mainly focused on the management of congestive heart failure. Three dogs with LAT received a pericardiocentesis. All 15 dogs with LAT had died of cardiac causes, 5 dogs during the first 7 days after admission. The median survival time for all 15 dogs with LAT was 52 days compared to 336 days in the control group (P=0.103). When excluding 5 dogs with LAT that died during the first 7 days, the median survival increased to 407 days, not different compared to the control group (P=0.549).
Conclusions: Dogs with MMVD and LAT have a high short-term mortality; however, when surviving the acute phase, the long-term prognosis may not differ from dogs with a similarly advanced degree of MMVD but without LAT.

Abbreviations

CHF	congestive heart failure
LA	left atrium
LAT	left atrial tear
MMVD	myxomatous mitral valve disease

Introduction

Left atrial tear (LAT) in dogs is a rare complication mainly associated with advanced myxomatous mitral valve disease (MMVD) [1,2]. The pathophysiology of the condition is not completely understood, but a multifactorial etiology is suspected. Several features and mechanisms have been suggested, such as severe left atrial (LA) enlargement, increase in atrial pressure causing wall over-stretching, genetic predisposition to collagen deposition alterations, and mitral regurgitation jets affecting the atrial wall (jet lesions) [2—4]. Once LAT occurs, it typically leads to rapidly accumulating hemorrhagic pericardial effusion with cardiac tamponade and signs of cardiogenic shock and/or congestive heart failure (CHF) [1,4,5]. The majority of published cases report an acute onset of weakness, collapse, respiratory distress, or cough [1,5—7]. The diagnosis of LAT is usually obtained with echocardiography by visualizing typical features of MMVD in addition to pericardial effusion and intrapericardial organized echogenic material suggestive of a thrombus [1,8]. A standardized therapeutic approach to patients with LAT has never been established, and in the current literature, it varies depending on clinical presenting signs and their severity [6—11]. Apart from guidelines for the management of CHF associated with MMVD [12], treatment including pericardiocentesis in case of cardiac tamponade, fluid therapy or dobutamine infusion in shock, the use of vasodilators or sedation after stabilization, as well as surgical repair or pericardiectomy have been described in dogs with LAT with various success [6,7,9,11,13,14]. Existing data show that prognosis of dogs experiencing LAT is poor [1], with the majority of them dying or being euthanized in the hospital [1,4,9,15] within the first three months [6,7,11]. A study from 2014 describing 11 dogs presented a more favorable outcome after the LAT event with survival reaching over a year, if no previous CHF was documented, or up to six months, if the patient had previously experienced CHF [8]. With this background, the aim of this study was to describe the clinical presentation, echocardiographic findings, short- and long-term treatment, as well as survival of dogs that experienced LAT due to advanced MMVD and to compare the findings with a matched control group of dogs that suffered from MMVD of similar severity but never experienced LAT.

Animals and materials and methods

Population characteristics

In this two-center case—control retrospective study, clinical databases from two veterinary teaching hospitals were reviewed, and 15 dogs with an echocardiographic diagnosis of LAT due to MMVD were selected in the time period between February 2012 and September 2016 (LAT group). Data collection included signalment, history, clinical examination, stage of MMVD [12], serum urea nitrogen and creatinine concentrations, treatments during and after hospitalization, survival time, and cause of death. Echocardiographic variables were collected from two different time points: at admission (T0) and before discharge (T1) after being successfully stabilized. This, based on clinical judgment, meant resolution of a clinically relevant amount of pericardial effusion, being alert, having normal mentation, and respiration without a need for oxygen supplementation for at least 12 h. Presence of left-sided CHF was defined as clinical signs of tachypnea or dyspnea and a radiographic diagnosis of perihilar or more diffuse pulmonary infiltrates, with resolution or amelioration after diuretic therapy. Right-sided CHF was diagnosed based on ultrasonographic evidence of abdominal effusion, together with subjective distension of the caudal vena cava.

A group of 15 dogs with MMVD that did not experience LAT served as controls. These animals were selected from the database of the same universities during the same time period. Control dogs were selected based on similar body weight, age, stage of MMVD, and left ventricular and LA dimensions compared to dogs with LAT. Ideally, the control dogs had to match (in the aforementioned criteria) the LAT dogs at T1, when the cardiac dimensions were considered the true dimensions reflecting appropriately the stage of the disease, i.e. unaffected by the pericardial effusion. If no echocardiographic data were available for LAT dogs at T1, control dogs were matched based on LAT measurement at T0.

Echocardiography

Echocardiographic studies obtained with different machines[c—f] were retrieved from storing systems and were reviewed by a board-certified cardiologist (MBT). The diagnosis of LAT was confirmed by the presence of typical signs for MMVD (mitral valve thickening and mitral valve regurgitant jet on color Doppler examination), together with pericardial effusion and the presence of an echogenic structure within the pericardium consistent with a thrombus (Fig. 1 and Video 1) [8,12] and exclusion of any neoplastic changes. Cardiac tamponade was diagnosed by identifying presence of pericardial effusion together with right atrial and/or right ventricular collapse [16]. All echocardiographic measurements were repeated offline by one operator (MBT). In particular, left ventricular internal diameter at end diastole normalized for body weight [17] and the ratio between the LA and aortic diameters were recorded for each patient. The left ventricular diameter was obtained from M-Mode at right parasternal short-axis view at the level of the papillary muscles, while LA and aortic diameters were measured in two-dimensional mode from the same view at the basilar level on the frame just after aortic valve closure [17—19].

Survival analysis

Survival data were obtained by reviewing the internal database or by telephone communication with the owners. Date of death, reason of death (natural or euthanasia), and cause of death (cardiac or non-cardiac) were annotated. Cardiac death was defined as death for CHF, euthanasia for refractory CHF or worsening of the general conditions related to the underlying cardiac disease, or due to sudden death.

Statistical analysis

Data distribution was assessed by visual inspection of the plots and by applying a Shapiro—Wilk normality test. Normally distributed data are presented as mean ± standard deviation and non-normally distributed data as median and range. Categorical variables are presented as fractional values of the whole sample and as percentage. Continuous variables were compared between groups with an unpaired *t*-test or a Mann—Whitney test, while within the LAT group, with a paired *t*-test or a Wilcoxon matched-pairs signed rank test. Categorical variables were compared with a Fischer exact test.

For the survival analysis, Kaplan—Meier curves were generated, and differences between the groups were analyzed by the log-rank

[c] Philips iU22, Philips Medical Systems, Monza, Italy.
[d] Philips iE33, Philips Medical Systems, Monza, Italy.
[e] Philips EPIQ 7, Philips Health Systems, Hamburg, Germany.
[f] GE Vivid 7 dimension, GE Medical Systems, Munich, Germany.

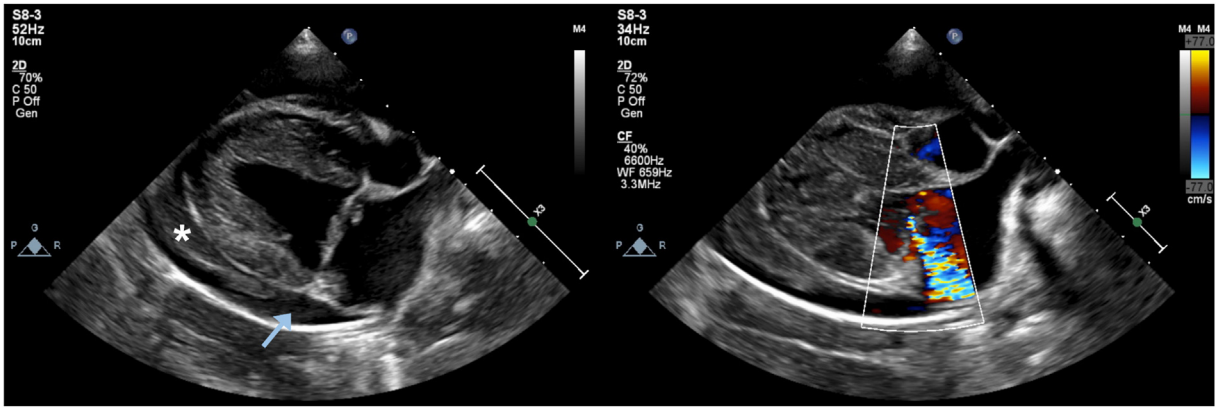

Figure 1 Echocardiographic right parasternal long-axis oblique views from a dog with left atrial tear included in the study. Typical features of myxomatous mitral valve disease with mitral valve thickening and prolapse (left panel) and eccentric mitral regurgitation jet visible on color Doppler examination (right panel). Anechoic pericardial effusion (arrow) and an echogenic structure (clot) partially occupying the pericardial space (asterisk) are also evident.

(Mantel—Cox) test. Non-cardiac deaths (with survival included up to the point of death) and dogs still alive at the time of analysis were right-censored. Data analysis was performed with statistical software packages[g,h]. P values$<$0.05 were considered significant.

Results

Population characteristics and clinical presentation

A total number of 30 dogs were enrolled in the study, 15 for each group. Eight different breeds were represented in the LAT group: Chihuahua (n = 5), dachshund (n = 3), mixed breed (n = 2), cotón de Tulear (n = 1), Medium Poodle (n = 1), spitz (n = 1), Pinscher (n = 1), and beagle (n = 1). In the control group, 8 different breeds were represented: mixed breed (n = 5), Yorkshire terrier (n = 3), Poodle (n = 2), Maltese (n = 1), Cavalier King Charles spaniel (n = 1), Chihuahua (n = 1), shar pei (n = 1), and Pomeranian (n = 1). There was no difference in sex and breed distribution, age, body weight, and MMVD stage between the groups (Table 1).

Presenting complaints and clinical signs observed in dogs with LAT are listed in Table 2. The most common complaints included weakness and respiratory signs, while syncope occurred in 6 of 15 dogs. Neurological signs included seizures, stupor, ataxia, strabismus, head tilt, and circling toward one side. Gastrointestinal signs included vomiting and hematochezia. One dog was referred due to tachypnea, dyspnea, and hematochezia, had cardiopulmonary arrest upon presentation, and was successfully resuscitated. Blood pressure was measured in 11 of 15 dogs using either a Doppler or an oscillometric method, with a mean systolic value of 122 $\pm$ 13 mmHg. The lowest systolic blood pressure values were between 100 and 110 mmHg and were recorded in three dogs. Isolated left-sided CHF was observed in 7 of 15 dogs, and in combination with right-sided CHF in 2 of 15 cases, while isolated right-sided CHF was present in 1 of 15 cases, and 5 of 15 dogs did not have CHF.

Biochemistry and echocardiographic examination

Serum biochemistry data were available in 13 of the 15 dogs in the LAT group at T0 and in 12 of the 15 dogs in the control group. No differences between the two groups were observed for blood nitrogen urea (P=0.640) and creatinine (P=0.343) (Table 3). Echocardiographic examination was available in all dogs with LAT at T0, while a second echocardiogram repeated at T1 was available in 13 of 15 dogs as two dogs died during hospitalization. Time between T0 and T1 was 4 days (2—9 days) and was a direct reflection of hospitalization length. No differences in the ratio between the LA and aortic diameters and left ventricular internal diameter at end diastole normalized for body weight were present between the LAT group at T1 and the control group (P=0.119 and P=0.248, respectively). In the LAT group, the left ventricular internal diameter at end diastole normalized for body weight increased from T0 to T1 (P=0.001), while the ratio between the LA and aortic

[g] Microsoft Excel software, Microsoft Office version 16.35, Microsoft Corporation, Bellevue, WA.
[h] Prism 8.4.2. software, GraphPad Software LLC., San Diego, CA.

Table 1 Clinical characteristics of a population of dogs with myxomatous mitral valve disease that experienced left atrial tear (LAT group) or did not (control group).

Variable	LAT group (n = 15)	Control group (n = 15)	P value
M/F	8/7	5/10	0.462
Pedigree/mixed breed	13/2	11/4	0.651
Age (years)	10.1 (7.7–14)	10 (8–15)	0.942
Body weight (kg)	6.1 (2.3–22.2)	5.2 (2.2–18.7)	0.986
MMVD stage B2/C	6/9	6/9	1.000

Data are expressed as median and range. F: females; M: males; MMVD: myxomatous mitral valve disease; LAT: left atrial tear.

diameters remained stable (P=0.121) (Table 3). Echocardiographic signs of cardiac tamponade were identified in 7 of 15 (46.7%) dogs with LAT at T0, which included two dogs that subsequently received a pericardiocentesis but excluded one dog, which had a pericardiocentesis performed by the emergency service before echocardiogram.

Treatment

Seven of 15 dogs had no previous history of MMVD; therefore, they were not under treatment when LAT happened, while the remaining dogs were previously reported to be in stage B2 (4/15) or stage C (4/15) and were being treated with medications. Information about treatment during hospitalization was available for all dogs with LAT, with an owner of one dog declining treatment and requesting euthanasia. Pericardiocentesis was performed in 3 of 14 dogs. This procedure was mainly chosen based on clinician assessment in presence of cardiac tamponade and signs of shock/patient clinical status. Supportive treatments included oxygen (7/14) and furosemide (8/14). Other treatments included crystalloid infusion (4/14), pimobendan (6/14), benazepril (4/14), amlodipine (4/14), and butorphanol (2/14). Table 4 shows treatment in all groups, as well as LAT subgroups divided depending on survival ($\leq$7 days or $>$7 days after admission). One patient in the control group had no treatment information available.

Survival characteristics

Survival data were retrieved for all dogs. All 15 dogs in the LAT group died of a cardiac cause with a median survival time of 52 days (2–1699 days), whereas in the control group, 11 dogs died of a cardiac cause with a median survival time of 336 days (82–1147 days), while three dogs were still alive at the end of the study period, and one dog

Table 2 Presenting complaints and clinical findings retrieved from a population of 15 dogs diagnosed with left atrial tear. Numbers indicate the number of dogs that presented with a specific finding.

Presenting complaints	Number (%)	Clinical findings	Number (%)
Weakness/apathy	12 (80)	Heart murmur	10 (66.7)[a]
Dyspnea	8 (53.3)	Hypothermia (Temp. < 37 °C)	6 (40)
Tachypnea (RR > 35/min)	7 (46.7)	Tachycardia (HR > 160 bpm)	5[b] (33.3)
Syncope	6 (40)	Muffled cardiac sounds	5 (33.3)
Gastrointestinal signs	5 (33.3)	Weak pulse/pulsus paradoxus	4 (26.7)
Neurological signs	4 (26.7)	Crackles on lung auscultation	3 (20)
Inappetence	4 (26.7)	Left-sided CHF	7 (46.7)
Cough	3 (20)	No CHF	5 (33.3)
Exercise intolerance	3 (20)	Left- and right-sided CHF	2 (13.3)
Distended abdomen	2 (13.3)	Right-sided CHF	1 (6.7)
Cardiopulmonary arrest	1 (6.7)	Cardiac tamponade	7 (46.7)

bpm: beats per minute; CHF: congestive heart failure; HR: heart rate; RR: respiratory rate.
[a] Systolic heart murmur intensity grade V/VI (n = 1 + 1 dog with muffled tones), IV/VI (n = 6), III/VI (n = 1), II/VI (n = 1).
[b] HR data were retrievable in eight patients.

Table 3 Selective biochemical and echocardiographic variables in a population of dogs with myxomatous mitral valve disease that experienced left atrial tear (LAT group) or did not (control group).

	LAT group (n = 15)		P value	Control group (n = 15)	P value
Variable	T0	T1			
Serum creatinine (umol/L)	111 (73–355) n = 13	NA		91 (56–161) n = 12	0.343[a]
Blood nitrogen urea (mmol/L)	17.5 (4.9–53.9) n = 13	NA		20.1 (3.8–106.5) n = 12	0.640[a]
LA/Ao	2.2 ± 0.4	2.5 ± 0.4[a] n = 13	0.121	2.3 ± 0.4	0.119[b]
LVDDN	1.86 ± 0.32	2.24 ± 0.24[a] n = 13	0.001	2.11 ± 0.34	0.248[b]

LA/Ao: left atrium to aorta ratio; LVDDN: left ventricular internal diameter at end diastole normalized for the body weight; LAT: left atrial tear; n: number; NA: not applicable; T0: time 0, admission; T1: time 1, after resolution of pericardial effusion.
[a] Compared to LAT T0.
[b] Compared to LAT T1.

died of non-cardiac cause. When considering the entire two populations, no difference in survival was detected (P=0.103) (Fig. 2A). In the LAT group, mortality was high in the first seven days after diagnosis, with 5 of 15 (33.3%) dogs dying or being euthanized in this period (two during hospitalization and three shortly after discharge), whereas none of the control dogs died during the first seven days after inclusion in the study. The dog that developed cardiopulmonary arrest at presentation survived 171 days after resuscitation. When excluding dogs with LAT that died or were euthanized during the first seven days after diagnosis, median survival times were similar between the LAT (407 days [30–1699 days]) and control groups (P=0.549) (Fig. 2B). From the 10 dogs with LAT that survived more than seven days, 5 dogs died suddenly, 2 dogs were euthanized for refractory CHF, 2 died spontaneously from CHF, and 1 dog was euthanized for worsening of the general condition related to the underlying cardiac disease. In the control group, 6 of the 11 dogs died suddenly, 3 were euthanized for refractory CHF, 1 dog died spontaneously from CHF, and 1 was euthanized for

Table 4 Treatment strategies from a population of dogs with myxomatous mitral valve disease that experienced left atrial tear and lived less or more than 7 days after admission. The control group includes dogs that never had left atrial tear.

Type of treatment	During hospitalization						After hospitalization			
	LAT group (n = 14)		LAT survival ≤7 days (n = 5)		LAT survival >7 days (n = 9)		LAT group (n = 13)		Control group (n = 14)	
	N	Dosages (mg/kg/day)	N	Dosages	N	Dosages	N	Dosages	N	Dosages
Pericardiosentesis	3		1		2		0		0	
Oxygen	7		3		4		0		0	
Furosemide	8	4 (1–9)	2	6 (4–9)	6	2.5 (1–8)	10	4 (1.4–6.4)	8	4.1 (2.8–6.4)
Crystalloids[a]	4	3.2 (2–10)	2	2.55 (2–3.1)	2	3.5 (2–5)	0		0	
Pimobendan	6	0.5 (0.4–1.5)	3	0.54 (0.5–0.8)	3	0.42 (0.2–1.5)	13	0.5 (0.3–0.8)	8	0.6 (0.5–0.8)
Benazepril	4	0.4 (0.2–0.5)	1	0.46	3	0.25 (0.2–0.5)	12	0.3 (0.2–0.5)	7	0.4 (0.3–0.8)
Amlodipine	4	0.4 (0.1–0.8)	1	0.2	3	0.5 (0.1–0.8)	7	0.2 (0.1–0.5)	1	0.2
Butorphanol	2	0.3 (0.2–0.5)	1	0.2	1	0.5	0		0	
No treatment	1		1		0		0		6	

LAT: left atrial tear; N: number of dogs.
[a] ml/kg/h.

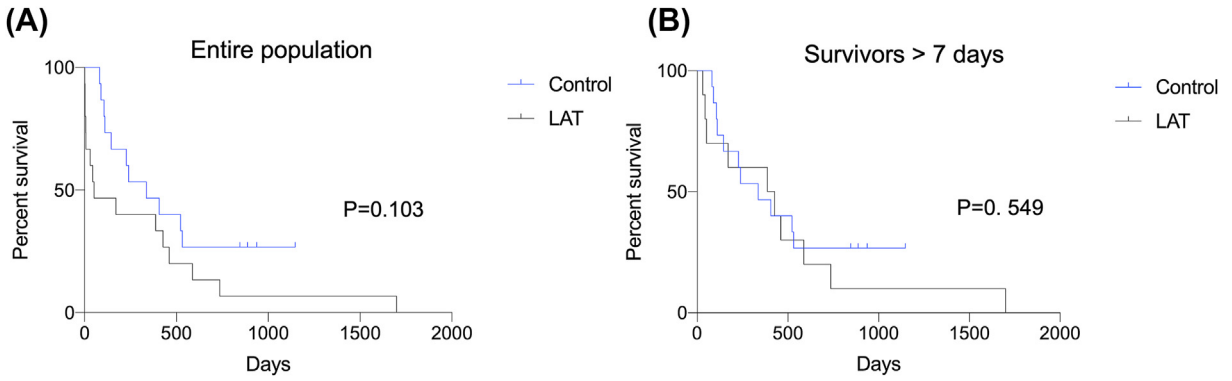

Figure 2 Kaplan—Meier curves showing survival in dogs with myxomatous mitral valve disease that experienced left atrial tear (LAT) or did not (control). Dogs experiencing non-cardiac death or still alive were right censored. (A) All the population of dogs (15 in the LAT group and 15 in the control group). Median survival time was not different between LAT (52 days [2—1699 days]) and control groups (336 days [82—1147 days]) (P=0.103). (B) Only dogs in the LAT group that survived longer than seven days were retained. The median survival of the remaining 10 dogs with LAT (407 days [30—1699 days]) was approximately the same as in the 15 dogs of the control group (P=0.549).

worsening of the general condition related to the underlying cardiac disease. Four of the 6 dogs with LAT in stage B2 survived longer than seven days with a median survival time of 387 days (30—1699 days), whereas among the six control dogs in stage B2, 3 were still alive (>887 days), 2 died because of cardiac causes (106 and 111 days, respectively), and 1 died of non-cardiac cause (846 days).

Discussion

The main findings of this study are that LAT is responsible for a high cardiac mortality and that the first week after the event is crucial for their survival. However, in dogs with LAT, long-term survival did not differ from dogs with MMVD of similar severity that never had been diagnosed with LAT; therefore, prognosis appears fair beyond the critical first week period.

LAT in dogs is not a well-studied complication. This may be due to its rare occurrence with some publications describing a prevalence of only 2% of all canine pericardial effusion cases [1]. In dogs, LAT is mainly associated with MMVD, with rare other causes such as neoplasia [3,5]. Pathophysiology of the condition is, yet, only speculative. It is possible that the mitral regurgitation jet weakens the endocardial endothelium, predisposing to tears as well as other complications such as acquired septal defects [2,11,20]. Increased LA pressure and its enlargement together with underlying connective tissue disorders, potentially causing endocardial degeneration, have also been proposed as mechanisms leading to LAT [1,11].

In the current study, LAT due to MMVD was, as expected, predominantly observed in older small-size dogs, which corresponds to disease predilection and is in agreement with previous publications [1,2,4,9,21,22]. As opposed to humans, where men are more predisposed to develop LAT [23], in our study, no sex predisposition was noted. Chihuahuas were overrepresented in our study, which is in agreement with other publications [5,8], together with dachshunds, which have previously been observed to have a higher incidence of endocardial splitting, from hypothesized genetic collagen degeneration, than other breeds [2]. This is not surprising since these two breeds are highly predisposed to MMVD, and in the authors' institutions, they are also the two breeds that most often are admitted for cardiac consultation. If this breed prevalence is related to a particular predisposition to LAT due to any genetic factor, it cannot be confirmed based on our study and the present literature. It is worth mentioning that no Cavalier King Charles spaniel, the breed most affected by MMVD, was present in the study population. This should be

analyzed with caution as it may simply reflect demographic trends in certain counties. Indeed, for both Italy and Switzerland, Cavaliers were not the most popular breeds during the study period.

Most frequently presenting complaints were likely secondary to the cardiogenic and obstructive shock induced by the acute hemorrhage and cardiac tamponade, together with left-sided CHF. Muffled heart sounds were detected in only one-third of the patients. This is likely due to the relatively small amount of pericardial effusion seen in LAT as opposed to other causes of hemopericardium such as in case of neoplasia and because patients with LAT are usually small breed dogs with enlarged heart. Therefore, it is likely that cardiac sounds were not muffled at the degree that this could be appreciated with an auscultation. Moreover, most of dogs with LAT (9/15) had a loud systolic heart murmur of grade IV/VI or more, with a thoracic thrill being felt over muffled cardiac tones in one case. Therefore, a clinical presentation of patients with LAT is often heterogenous, as typical signs of cardiac tamponade are sometimes missing, a cardiac murmur can be absent, and presenting complaints are vague. A suspicion for LAT should anyway be raised in case of a dog with known history of MMVD, when acute signs of cardiogenic shock are detected, since these are rarely present in animals with typical uncomplicated MMVD. Interestingly, many dogs had left-sided CHF, alone or in combination with right-sided CHF. The reason for this high prevalence can be explained not only by the underlying cardiac disease with severe LA dilatation but also by the cardiac tamponade itself. Acute pericardial effusion can indeed induce a rise in LA pressures, which are already elevated in dogs with MMVD at an advanced stage, eventually leading to lung edema [24]. Potentially, it also explains why LA-to-aortic diameters for dogs with LAT were not different at T0 compared to T1, unlike the left ventricular size, which was significantly smaller at T0 than at T1, most likely secondary to an acute loss of blood volume and external compression of pericardial effusion that impaired its filling and reduced its output. Whereas, LA, being directly 'connected' by a tear to the pericardial space, most likely at the moment of a tear itself had reduced its size but then quickly equilibrated with the pericardial space and further increased its intracavitary pressure.

Treatment varied widely between patients in our series. Considering the variety of symptoms that we encountered, it is difficult to propose a unique approach. In the presence of left-sided CHF, diuretics and oxygen were always administered, taking the risk of further reducing the cardiac output, especially in patients with signs of cardiogenic shock [22]. Diuretics were continued for a short term in one patient being in stage B2 MMVD to further unload the LA. In other patients being in shock but without clear signs of lung edema, initial stabilization consisted of intravenous fluids administration. Pimobendan and amlodipine were individually administered based on clinician assessment. Even though our data were collected before the published EPIC-study recommendations [25], pimobendan was administrated to improve systolic function, reduce annulus diameter, reduce afterload (same rational for amlodipine), and reduce the regurgitant fraction [25—28]. While administration of pimobendan does not have any contraindication in unstable patients [28], amlodipine should be chosen with caution in dogs with unstable CHF, hypotension, or kidney disease [29].

Three dogs in the LAT group received pericardiocentesis, with stabilization of the cardiovascular function. In all these cases, pericardiocentesis had not directly led to patient deterioration. In dogs with LAT, the reported outcome of pericardiocentesis is variable, with some reports highly recommending it, whereas others, where the procedure led to sudden death, do not [7,10,15]. From one side, pericardiocentesis might improve cardiac filling and systemic output, while on the other side, it exposes cardiac structures to the risk of harm, or of further hemorrhage, once the intrapericardial pressure drops [30]. Therefore, we conclude that pericardiocentesis should be approached individually, based on hemodynamic stability of a patient and acknowledging the associated risks. From our experience, if pericardial effusion appears to be hemodynamically relevant, pericardiocentesis can be performed with low risk.

Mortality rate was high in our study, with a third of patients dying in the first days after admission. Similar findings were observed before with over 60%

of dogs dying during hospitalization [1]. However, many dogs in our study survived long after discharge, with a survival time not different from dogs without LAT. Better prognosis for dogs with LAT was also described in other publications [5,8]. Therefore, although LAT is a severe and possibly life-threatening complication of MMVD, dogs surviving the first week might experience a relatively fair prognosis. Especially those being in stage C had a survival matching with previous studies of dogs with decompensated MMVD without LAT [28]. However, it is worth mentioning that controls in stage B2 lived longer than dogs with LAT in the corresponding MMVD stage, which is in agreement with previous studies [25] and may reflect the differences in overall prognosis. When excluding two dogs with LAT in stage B2 that died during the first seven days after admission, though, the median survival time of these animals was notably longer, overcoming the survival time of two dogs in stage B2 without LAT. Sudden cardiac death was the most common cause of death in both groups, affecting about half of the population. The remaining animals in both groups died for causes related to the progression of CHF or worsening of general condition.

The current study bears some additional limitations. The main limitation is the small sample size, an unavoidable drawback of a disease with low prevalence. Then, the retrospective nature of the study accounted for not always retrievable data, no treatment standardization, and loss of some patients to follow-up. Furthermore, the cause of death, whether cardiogenic or not, was often based on owners' perception, thus it could have been misinterpreted. Finally, as mentioned before, due to missing histopathological analysis, LAT diagnosis was made purely echocardiographically, which holds a risk of a false patient inclusion in the study group.

Conclusion

In conclusion, dogs with MMVD-associated LAT bear a high cardiac mortality risk, especially in the first week after the event. They usually present in shock and with respiratory distress. The absence of heart murmur or muffled cardiac tones does not exclude LAT. Treatment should be tailored individually with a cautious decision on pericardiocentesis. When surviving the critical time of the first seven days after the event, LAT may not affect long-term survival of dogs with advanced MMVD.

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Conflict of Interest Statement

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

Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.jvc.2023.11.005.

Video	Title	Description
1	Echocardiographic image from a dog with myxomatous mitral valve disease and left atrial tear included in the study	Right parasternal long axis oblique view, showing typical features of myxomatous mitral valve disease, with valvular thickening, and evidence of pericardial effusion with a hyperechoic structure attached to the left atrial wall, consistent with a clot.

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