The Prevalence of Cardiomyopathy in the Irish Wolfhound: A Clinical Study of 500 Dogs

The prevalence of cardiomyopathy in Irish wolfhounds was evaluated by retrospective review of the results of cardiovascular examinations carried out in 500 dogs presented for veterinary services at the author's practice.

Abnormalities were found in 209 (41.8%) of the dogs examined. Dilated cardiomyopathy (DCM) was diagnosed in 121 (24.2%) of the dogs and was accompanied by atrial fibrillation in 106 dogs. Seventeen dogs were suffering from advanced congestive heart failure (CHF), and 55 dogs were suffering from mild to moderate CHF as a result of DCM. Congestive heart failure was most commonly characterized by mild to severe pleural effusion due to right-sided heart failure in addition to pulmonary edema. Rhythm disturbances without evidence of DCM were detected in 48 dogs. Forty dogs had echocardiographic abnormalities without signs of DCM. Soft to moderate mitral regurgitations were diagnosed in 13 (2.6%) of these 40 dogs examined. In 39 dogs that died as a result of DCM, the median survival time from the time of diagnosis was 5.1 months, and in 59 dogs with DCM that are still alive, the median survival time is 15.7 months. **J Am Anim Hosp Assoc 2000;36:125–32.**

Andrea C. Vollmar, DVM, Diplomate ECVIM

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From the Small Animal Veterinary Hospital of Wissen, Heisterstr. 5, D- 57537 Wissen, Germany.

Introduction

Cardiomyopathy is a common problem in the Irish wolfhound. Within the breed, there is a high prevalence of dilated cardiomyopathy (DCM) as well as a variety of ventricular and supraventricular arrhythmias and conduction disturbances, with atrial fibrillation representing the most common arrhythmic abnormality.[1–4] The purposes of this study were to detect the prevalence of cardiomyopathy in 500 Irish wolfhounds and to evaluate and describe the abnormal findings from cardiovascular examination.

Materials and Methods

Every Irish wolfhound older than 12 months of age, admitted for veterinary services to the small animal veterinary clinic of Wissen, was given a thorough cardiovascular examination. From February 1993 through July 1999, data of 500 Irish wolfhounds (201 males and 299 females) was available for retrospective review. The dogs were 12 months to 11 years (mean, 4.2±standard deviation [SD] 1.8 yrs) of age, with body weights between 48 and 93 kg (mean, 64.4±SD 8.8 kg). After general physical examination, a standard six-lead electrocardiogram (EKG)[a] was performed. Echocardiography was carried out by means of a color-flow Doppler machine.[b] All animals (n=500) were auscultated and had EKGs and echocardiograms recorded. Thoracic radiographs were taken of all dogs with echocardiographic findings of DCM or with marked increases in right atrial and ventricular dimensions, in order to determine if there was evidence of congestive heart failure (CHF; i.e., pulmonary venous distension, pulmonary edema, and pleural effusion) or of primary pulmonary disease. Echocardiography was performed using a 5- or a 3.5-MHz transducer with the dogs in a standing position. Because of restlessness,

some dogs were placed in right- and left-lateral recumbency to facilitate the examination. To determine if the position used would influence the echocardiographic measurements, 20 Irish wolfhounds were examined standing and in right- and left-lateral recumbency. Changes in heart position that were related to posture did not affect the results of the examinations.

Two-dimensional (2D) and M-mode echocardiograms were recorded and analyzed according to the recommendations of the American Society of Echocardiography and the Echocardiography Committee of the Specialty of Cardiology, American College of Veterinary Internal Medicine.[5,6] For 2D and M-mode interrogation of the heart, the standard right-parasternal long-axis and short-axis views were used. At least three to five cardiac cycles were measured. In addition, 2D measurements of end-diastolic right ventricular dimensions were made in the right-parasternal long-axis four-chamber view below the tricuspid valve. In the same view, widest end-systolic diameters of both atria were measured parallel to a line through each atrioventricular valve annulus. Doppler and color-flow imaging were used to detect any congenital or acquired abnormal flow within the heart and blood vessels. Taking body weight into account for the calculation of myocardial function, end-systolic volume indices (ESVI) were determined. Based on the left ventricular end-systolic measurements in cm at M-mode, ESVI was calculated according to the cube formula of Teichholz:[7]

$$\text{ESVI (ml/m}^2\text{)} = \frac{7\,(\text{LVIDs})^3}{2.4 + \text{LVIDs}} \times \frac{1}{\text{BSA}}$$

where BSA=body surface area and LVIDs=left ventricular internal dimension at end-systole

The diagnosis of DCM in this report was based on the results of the echocardiographic examinations. In earlier studies, echocardiographic reference values were determined for the breed, and the echocardiographic features of Irish wolfhounds with occult DCM were compared to dogs with CHF and to normal dogs.[8,9] Based on these earlier studies, the echocardiographic criteria used to diagnose DCM in Irish wolfhounds were: LVIDs wider than 41 mm and wider than 61.2 mm at end-diastole (LVIDd), fractional shortening (FS) below 25%, E-point to septal separation (EPSS) greater than 10.0 mm, and ESVI greater than 41 ml/m^2. Right ventricular dilatation was diagnosed when right ventricular internal dimensions, measured during end-diastole (RVIDd), were wider than 36.8 mm. Left or right atrial enlargement was present when the systolic internal diameter of the atrium being examined was greater than 56 mm.

The diagnosis of DCM as the cause of CHF in the dogs of this report was based on echocardiographic evidence of advanced myocardial failure with the radiographic diagnosis of pulmonary edema and pleural effusion. The presence of ascites was determined by physical examination (i.e., palpation of an abdominal fluid wave) and confirmed by ultrasonography and radiography or abdominocentesis. According to the recommendations of the International Small Animal Cardiac Health Council, CHF due to DCM was classified depending on radiographic and physical examination abnormalities.[10] Congestive heart failure was termed mild to moderate when radiography revealed cardiomegaly with left atrial enlargement, pulmonary venous distension, increased pulmonary densities typical for pulmonary edema, a small amount of pleural effusion, hepatomegaly, or mild ascites. Commonly, these dogs were exhibiting exercise intolerance and increased respiratory rates and effort. Typical findings on auscultation were cardiac arrhythmias, a gallop sound or a left apical systolic murmur, and in some dogs end-inspiratory pulmonary crackles. Advanced CHF was defined in dogs with radiographic changes including cardiomegaly, moderate to severe pleural effusion, alveolar lung infiltrates of pulmonary edema, hepatomegaly, or ascites. Dogs generally presented weak, often cachectic and anorexic, with dyspnea at rest or profound exercise intolerance, and, on auscultation, sinus tachycardia or tachyarrhythmia due to atrial fibrillation or premature ventricular complexes and sytolic murmurs. The term occult DCM was used to describe clinically asymptomatic dogs with echocardiographic evidence of DCM. In these dogs, arrhythmias were commonly found during auscultation, but no visible clinical signs of disease were present. Thoracic radiographs were generally normal, or only mild cardiomegaly with some degree of pulmonary venous distension was identified.

This study was not prospective, and it was not intended to assess the efficacy of any particular drug. Medical therapy consisted variably of angiotensin-converting enzyme (ACE) inhibitors (e.g., benazepril, enalapril, captopril), positive inotropic drugs (e.g., metildigoxin, digoxin, pimobendan [a calcium sensitizer], dobutamine), diuretics (e.g., furosemide, hydrochlorothiazide, spironolactone), calcium-channel antagonists (e.g., diltiazem, verapamil), beta blockers (e.g., atenolol), and other antiarrhythmic drugs (e.g., class IB drug: lidocaine; class IC drug: propafenone; and class III drug: sotatol). Some dogs with CHF already were on medical treatment at the time of first presentation.

Results

All Irish wolfhounds (n=500) were auscultated and had EKGs and echocardiograms recorded. Of these, 209 (41.8%) dogs had abnormalities noted in one or more of these tests [Table 1]. In 121 (24.2%) of the 500 dogs, echocardiographic findings were consistent with DCM, 40 (8%) dogs had echocardiographic abnormalities without evidence of DCM, and 48 (9.6%) dogs had EKG abnormalities without evidence of DCM.

In the DCM group, males were affected relatively more often than females (65 [32.3%] of all males versus

Table 1

Abnormal Cardiovascular Findings in 209 Irish Wolfhounds (41.8%; 104 Males, 105 Females) Out of 500 Irish Wolfhounds (201 Males, 299 Females), One to 11 Years of Age

	Male	Female	Percentage of 209 Dogs With Abnormal Cardiovascular Findings
Dilated cardiomyopathy (n=121)			
DCM,* occult	26	23	23.4
DCM with mild to moderate CHF†	29	26	26.3
DCM with advanced CHF	10	7	8.1
DCM - combined with atrial fibrillation in 106 (87.6%) of DCM cases - combined with pericardial effusion in 17 (14.0%) of DCM cases - combined with ventricular premature beats in 13 (10.7%) of DCM cases			
Echocardiographic abnormalities without DCM (n=40)			
Dilated right ventricle and atrium	12	9	10.0
Left ventricular abnormal wall motion	1	5	2.9
Mitral regurgitation	4	9	6.2
Electrocardiographic abnormalities without evidence of DCM (n=48)			
Atrial fibrillation	8	3	5.3
Supraventricular premature beats	4	4	3.8
Ventricular premature beats	7	6	6.2
Ventricular tachycardia		1	0.5
Left anterior fascicular block	2	9	5.3
First-degree atrioventricular block	1	3	1.9

* DCM=dilated cardiomyopathy
† CHF=congestive heart failure

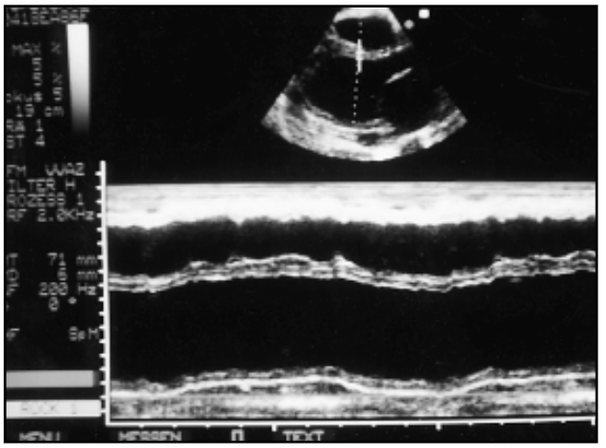

Figure 1—M-mode echocardiogram of a 7.5-month-old, female Irish wolfhound with DCM and atrial fibrillation, obtained from the right parasternal long-axis view. The left ventricular end-systolic diameter is markedly increased, indicating severe myocardial failure. Left and right end-diastolic diameters are increased to compensate for the severely decreased myocardial contractility. (Fractional shortening averaged 6%, but in some locations of the left ventricle no visible contractions were present.)

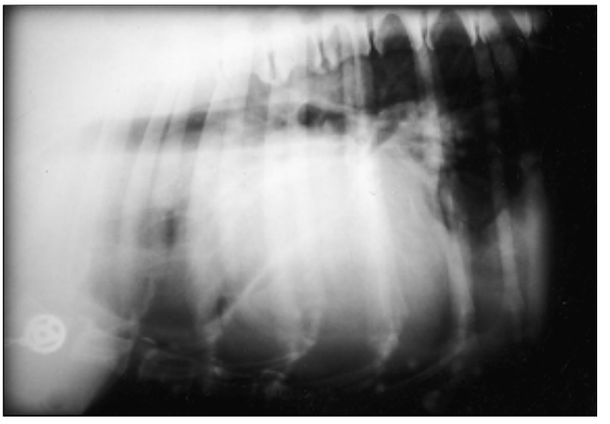

Figure 2—Lateral thoracic radiograph from an Irish wolfhound with dilated cardiomyopathy (DCM), atrial fibrillation, and congestive heart failure (CHF). Note the marked, generalized cardiomegaly and left atrial enlargement. Moderate pleural effusion due to bilateral CHF is present in this dog.

56 [18.7%] of all females). The mean age of Irish wolfhounds with DCM was 4.2±SD 2.1 years. A female of 7.5 months of age was the youngest dog suffering from end-stage DCM (FS, 6%) [Figure 1]. Occult DCM was diagnosed in 49 dogs, while 72 dogs had evidence of CHF confirmed by radiography [Figure 2]. In 27 dogs with occult DCM or with DCM producing CHF, right

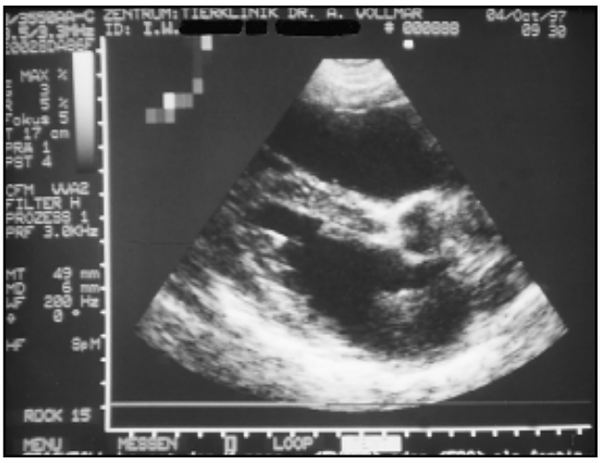

Figure 3—Two-dimensional, right parasternal, long-axis four-chamber view of a two-year-old male Irish wolfhound with atrial fibrillation. In this dog, right ventricular dilatation was more obvious than left ventricular changes.

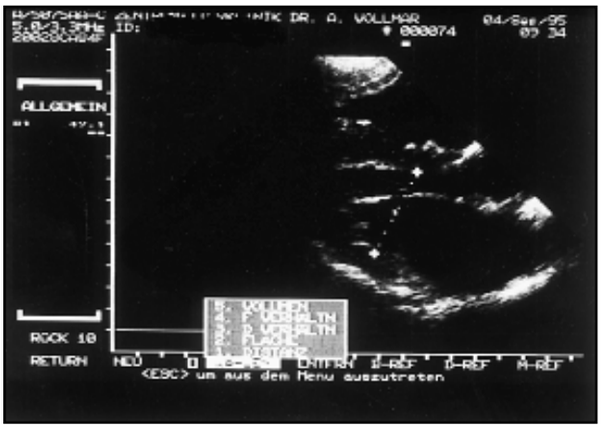

Figure 4—Two-dimensional, right parasternal, long-axis view showing left atrial enlargement in a two-year-old bitch. Abnormal left ventricular and septal wall motions without increases in wall thickness were present in this dog in addition to the normal systolic contractions with wall thickening. Three months later, the dog developed atrial fibrillation; six months later, there was echocardiographic evidence of DCM.

ventricular dilatation was much more pronounced than left ventricular dilatation [Figure 3].

In 21 (4.2%) dogs, there were marked increases of right atrial and right ventricular dimensions, without any detectable left ventricular changes during echocardiography or evidence of pulmonary disease on auscultation and radiography. Five of these latter 21 dogs had ventricular premature contractions that appeared to originate in the right ventricle, as the depolarization assumed a left bundle-branch block pattern in Lead II of the EKG, and four dogs progressed to DCM and CHF within two years.

In six dogs, abnormal left ventricular wall motions were found during echocardiography. In these dogs, permanent and rapid left ventricular free wall and septal motions toward the lumen, without increases in wall thickness, were present in addition to the normal systolic wall motions with marked increases in wall thickness. Ventricular dimensions and systolic indices were normal, but all six dogs had concurrent left atrial enlargement [Figure 4]. Three of these dogs were reexamined three to six months later; two of them had developed atrial fibrillation and the other dog showed typical changes of DCM at that time.

In 17 (14%) of the dogs with DCM, advanced CHF was diagnosed which was characterized by severe pleural effusion in combination with pulmonary edema. Additionally, in some of these dogs, mild (n=7) or moderate (n=2) ascites was present. Thoracentesis was carried out as an emergency treatment in 13 dogs and had to be repeated in four dogs for the management of CHF in addition to medical treatment. In all cases, 4 to 5 L of fluid were removed during each centesis; fluid analysis revealed chylothorax, characterized by a white to slightly pink color and remaining opaque when centrifuged, and a triglyceride content much higher and a cholesterol content lower than what was present in the serum.[11,12] In these chylous effusions, the content of total protein and the specific gravity measured between 2.5 and 4.6 g/dl and between 1.020 and 1.032, respectively; however, measurements were performed with a refractometer and may be inaccurate due to interference secondary to the high lipid refractive index of the fluid. The total nucleated cell count was 1,000 to 7,000/µl, with lymphocytes or neutrophils representing the predominant cell type and a lesser number of mesothelial cells and macrophages. Mild to moderate pericardial effusion was noted during echocardiography in 17 dogs with moderate or advanced CHF.

Concurrent, mild to severe, mitral or tricuspid regurgitation was observed in most DCM patients with marked left or right ventricular enlargement. Commonly, valve leaflets appeared to be mildly or moderately thickened, with an increased echogenicity. Mitral insufficiency without signs of DCM was diagnosed in 13 (2.6%) dogs.

Atrial fibrillation was diagnosed in 70 (97.2%) dogs with CHF [Figure 5] and in 36 (73.5%) dogs with occult DCM. In eight males and three females, atrial fibrillation was the only abnormality found during the initial examination and was combined with marked left atrial or biatrial enlargement in eight of these dogs. In two dogs, immediate antiarrhythmic therapy was required because of poor hemodynamics due to tachyarrhythmia [Figure 6]. Three dogs were reexamined between one and two years later and had developed echocardiographic evidence of DCM. Another dog was reported by the owner to have died because of sudden death six months after presentation.

Supraventricular or ventricular premature depolarizations were the only abnormalities found during cardiovascular examinations in seven and 13 dogs, respectively. In eight dogs with CHF, ventricular premature depolarizations were present at a rate between six per minute and bigeminus rhythm.

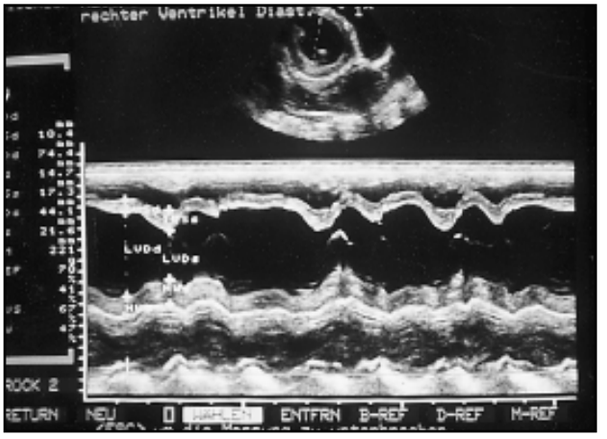

***Figure 5*—**M-mode obtained from the right parasternal, transverse image of the left ventricle in a 2.5-year-old male with DCM and CHF. Due to atrial fibrillation, the contractions of the left ventricular free wall and interventricular septum are irregular. Below the left ventricle, a severe pleural effusion is present.

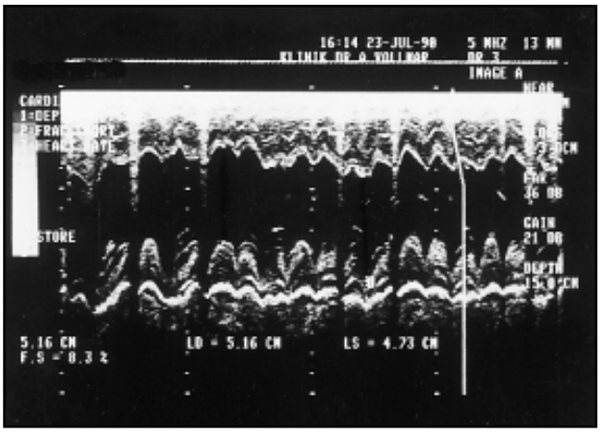

***Figure 6*—**M-mode obtained from the right parasternal image of the left ventricle, showing severe dyskinesia of the free wall and interventricular septum due to tachycardia (240 beats per minute) as a result of atrial fibrillation in a 1.5-year-old bitch.

The only abnormality represented on cardiovascular examination in 11 dogs was left anterior fascicular block (LAFB), characterized on the EKG by marked left-axis deviation in the frontal plane, normal QRS complex duration, QRS complexes in leads aVR and aVL each ending in an R wave, small q waves and tall R waves in leads I and aVL, and deep s waves which exceeded the R waves in leads II, III, and aVF.[13] Two other dogs with LAFB had echocardiographical evidence of DCM (one with occult DCM and one with moderate CHF); both of these dogs had concurrent atrial fibrillation. In all cases of LAFB, systolic and diastolic left ventricular and septal wall thicknesses were toward the upper limits of reference ranges. Most dogs showing LAFB were closely related to each other.

Survival Times in 98 Dogs With DCM

Survival time was counted from the day the case was presented to the clinic to the day the dog died or had to be euthanized as a direct result of the disease. Of the 121 dogs diagnosed with DCM in this study, 98 were available to be included in survival data [Table 2]. Twenty-three dogs had to be excluded; 14 of them died of unrelated diseases, and nine were lost to follow-up. One dog with moderate CHF and four dogs with occult DCM and concurrent atrial fibrillation died of sudden death. In 34 dogs, euthanasia had to be performed either because of failure of therapy to relieve symptoms associated with pulmonary edema and pleural effusion (i.e., failure to produce adequate stabilization to allow minimal exercise), or because of clinical deterioration or relapse of CHF with respiratory distress, anorexia, and weakness. Fifty-nine dogs are still alive, having a median survival time of 15.7 months at the time of manuscript preparation. The median survival time of all 59 dogs that died as a result of DCM was 5.1 months.

Discussion

Cardiomyopathy is a common problem in the Irish wolfhound. Eventually, two principal clinical manifestations develop: sudden cardiac death or congestive heart failure. In the author's experience, some affected dogs live to old age and die of unrelated problems, often without the presence of cardiomyopathy being detected until postmortem examination.[14] Dilated cardiomyopathy was identified in 38 Irish wolfhounds out of 696 new hospital admissions of this breed at Purdue University, based on a search of the Veterinary Medical Data Base (VMDB).[1] In the author's study, 121 out of 500 dogs were diagnosed with DCM. A higher percentage of males were affected (32.3%) and developed CHF (19.4%), while 18.7% of females were affected with DCM and 11% developed CHF.

In the dogs of this study, advanced CHF (n=17) most commonly was characterized by severe pleural effusion in addition to pulmonary edema. Thirteen of these dogs underwent thoracocentesis, with the results of fluid analysis consistent with chylothorax. According to the author's knowledge, chylothorax has not been reported as a feature of CHF in Irish wolfhounds before. Chylous effusion may be present in dogs suffering from bilateral heart failure. Left-sided heart failure, resulting in increased pulmonary venous and capillary pressures, induces increased transudation of protein-enriched fluid into the pulmonary parenchyma and bronchial mucosa. If capillary pressure increases above a critical level, the filtration rate will exceed the lymphatic drainage flow rate, and pulmonary edema will occur. In the presence of right-sided heart failure, an increased systemic venous pressure can result in obstruction of lymphatic drainage. From intact but dilated subpleural and intrapulmonary lymph channels, the chyle escapes into pleural and bronchial spaces, resulting in chylothorax.[11,12,15,16]

Between 1986 and 1990, a clinical and EKG survey of the Irish wolfhound breed in Great Britain was carried out at breed shows, rallies, and during visits to large breeding facilities to investigate the prevalence of cardiac arrhythmias in asymptomatic dogs.[3] Included in the study was an investigation regarding whether or not the

Table 2

Survival Times of 98 Irish Wolfhounds With Dilated Cardiomyopathy

Parameters	n=	Median (mos)	Range (mos)
Mild to moderate CHF,* still alive	26	14	2.0–33
Mild to moderate CHF, dogs died	18	5.5	0.07–35
Advanced CHF, dogs died	17	2.75	0.2–14
Occult DCM,† still alive	33	17	0.25–68
Occult DCM, dogs died	4	13	10–28

* CHF=congestive heart failure
† DCM=dilated cardiomyopathy

largest individuals of the breed have a higher prevalence of cardiac arrhythmias. Measurements of height, length, and girth were obtained from 265 adult and asymptomatic Irish wolfhounds. Males with a larger girth and females with a larger girth, or greater height, or both, had a higher incidence of supraventricular arrhythmias. However, according to the authors' conclusion of that study, it could not be said that one is a direct result of the other, as both may be dependent on a third, as of yet unknown, factor.[17] During the complete British study, clinical and EKG examinations were carried out in 496 Irish wolfhounds. In 22.2% of the dogs, rhythm disturbances were detected with an atrial fibrillation prevalence of 10.5% in dogs from 10 to 111 months of age. Ventricular ectopic contractions, diagnosed in 4.8% of dogs, were mainly unifocal and infrequent, but bigeminal rhythm and paroxysmal tachycardia were seen. Supraventricular ectopics were present in 3.2% of dogs and developed into atrial fibrillation in two animals. Heart murmurs were found in 2.6% of the dogs.[3]

In the author's study, rhythm disturbances were present in 33.4% of the dogs, with atrial fibrillation showing a prevalence of 23.4%. Atrial fibrillation is reported to be present in 75% to 80% of giant-breed dogs with DCM.[1] When combined with DCM or chronic valvular heart disease, atrial fibrillation is considered a severe cardiac arrhythmia that carries a high mortality rate. Generally, atrial fibrillation represents a common cause of sudden death or of progressively deteriorating myocardial contractility.[14,18] In contrast, some individual giant-breed dogs exhibit atrial fibrillation with no evidence of underlying cardiac disease (so-called "lone atrial fibrillation"). In one study, the authors conclude that the Irish wolfhound is affected by atrial fibrillation far more commonly than other breeds of dogs, and that atrial fibrillation in this breed is neither a form of DCM, nor is it responsible for the development of progressive cardiac dysfunction.[4] In the present study, atrial fibrillation was most commonly found in Irish wolfhounds affected with DCM, as 106 of the 121 dogs with DCM showed atrial fibrillation. So-called "lone atrial fibrillation" was found in 11 dogs. Three of these dogs had evidence of DCM 1.5, two, and 3.2 years later, and another dog died from sudden death. Three dogs have been on medication to control the heart rate for two, five, and 29 months at the time of manuscript preparation and have not developed additional heart problems as of yet. Three dogs with near-normal heart rates (between 80 and 110 beats per minute at home, and up to a maximum of 140 beats per minute under clinical conditions) but with severe left and right atrial enlargement are receiving ACE inhibitors in an attempt to delay the onset of DCM, and one dog was lost to follow-up. It is possible that there are different etiologies for atrial fibrillation in the Irish wolfhound and that there may exist differences regarding atrial fibrillation between Irish wolfhounds bred in the USA and the German, Dutch, and Belgian breeding stocks, which are more closely related to the British dogs.

In the author's opinion, the significance of ventricular premature depolarizations (VPDs) in Irish wolfhounds is different to that in Doberman pinschers[19–22] and boxers.[23] When associated with DCM and atrial fibrillation, VPDs especially were observed more frequently in severely affected Irish wolfhound hearts. In 13 (2.6%) dogs between one and six years of age, VPDs were diagnosed without evidence of DCM or any other cardiac pathology. Four of these dogs, which were reexamined repeatedly over a period of several years, lost the dysrhythmia completely without developing other cardiovascular abnormalities. Two dogs developed DCM and atrial fibrillation one and two years after the first examination.

There is evidence for genetic involvement in DCM in the Irish wolfhound. In Great Britain, a line of Irish wolfhounds was identified in which one or more individuals affected with DCM was present in a number of generations and for which reasonably complete records of the disease status of many related dogs were available. The incidence of the disease in siblings of affected individuals fit most closely with an autosomal-dominant mode of inheritance.[24] This result is consistent with the

author's observations that within affected breeding lines, DCM was diagnosed in closely related dogs of every generation. Up until present, the author has identified only two dogs, out of 121 dogs affected with DCM, where both parents are normal on cardiovascular examination; in the other 119 cases, at least one of the parents was affected as well. Concerning the recorded LAFB, there also seems to be a hereditary influence, as nine out of 13 dogs were closely related to each other (i.e., a brother and sister and their offspring).

Survival times for Irish wolfhounds with DCM in this study were much longer than times reported for Doberman pinschers with DCM[19,21,22] and also were longer than the times reported in a study on 189 dogs of different breeds suffering from DCM.[25] The relatively long survival time for Irish wolfhounds with DCM compared to some other breeds of dogs may be attributed to several factors. First, in Irish wolfhounds presented with CHF, pleural effusion is the prevailing symptom, while pulmonary edema often is not as pronounced as in other breeds of dogs.[19] Pleural effusion and pulmonary edema usually evolve over a subsequent period of several weeks, and only then do more severe clinical signs (i.e., complete anorexia and severe respiratory distress) develop. Second, only rarely does left ventricular function become as compromised as in other breeds of dogs diseased with DCM, and in the majority of cases, DCM in Irish wolfhounds demonstrates a good response to positive inotropic therapy. Third, as atrial fibrillation is found in most Irish wolfhounds with DCM, producing extreme tachycardia, reduction of the heart rate by means of digoxin and antiarrhythmic drugs often leads to a good clinical improvement of cardiac performance and reduces further myocardial deterioration due to tachycardia.

In the individual case of DCM, prognosis is difficult to predict at the first presentation. Under appropriate treatment, many Irish wolfhounds with DCM and atrial fibrillation can have an acceptable quality of life for many months or even years. But, from the author's experience, the time course of the disease can be significantly accelerated when abnormalities are present in young dogs less than 1.5 years of age.

An early diagnosis of cardiomyopathy and early initiation of treatment with ACE inhibitors and, if indicated, digoxin or antiarrhythmic drugs, seem to influence the course of the disease in a positive way. Further investigations will be needed to evaluate the time point in the development of the disease when medical intervention is indicated.

Conclusion

Cardiomyopathy is a common disease in the Irish wolfhound that impairs expectation and quality of life. In the present study, out of 500 dogs examined, DCM was diagnosed in 121 (24.2%) dogs and atrial fibrillation was present in 117 (23.4%) dogs. The total prevalence of cardiomyopathy within the breed must be calculated as even higher, because some of the younger dogs in the study (which were regarded as normal at the first time of examination) will likely develop cardiomyopathy over time. In addition, there are reports from dog owners about sudden death, which seems to occur most often in young adult dogs between one and two years of age. Those animals usually have not been examined cardiovascularly and, therefore, were never taken into account statistically. Because of the suspected autosomal-dominant inherited genetic cause of cardiomyopathy in Irish wolfhounds, the author recommends regular heart examinations of all breeding dogs.

[a] Cardiovit AT-10; Schiller AG, Switzerland

[b] SIM 7000 CFM Challenge; Esaote Biomedica, Italy

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