HEART SCREENING RESULTS OF MORE THAN 10000 IRISH WOLFHOUNDS:
PREVALENCE OF DILATED CARDIOMYOPATHY, SURVIVAL CHARACTERISTICS, WHOLE BLOOD TAURINE & DCM INHERITANCE

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Background: Within the Irish wolfhound (IW) breed, there is a high prevalence of dilated cardiomyopathy (DCM) as well as a variety of arrhythmias and conduction disturbances, with atrial fibrillation (AF) representing the most common arrhythmic abnormality.1-8 Several investigators have reported the incidence of arrhythmias in IW dogs. In a study of 496 IWs in Great Britain, arrhythmias were detected in 22.2%. Atrial fibrillation was the most common rhythm abnormality, recorded in 10.5% of the dogs which were 10 months to 9.25 years of age.1 Atrial fibrillation was also reported in 25 IWs from New England, USA, (mean age 4.5 years, median age, 5 years) with and without overt DCM.2 In a heart screening study evaluating sequential echocardiographic and EKG examinations of 232 IWs from northwestern Europe,7 AF was diagnosed in 30 dogs (12.9%) affected with DCM as well as in 19 dogs (8.2%) with relatively normal echocardiograms. Nine additional dogs (3.9%) with DCM were in sinus rhythm, and 12 dogs (5.2%) had sinus rhythm with intraventricular conduction abnormalities. In another study evaluating all cardiovascular examinations performed in 500 IWs from northwestern Europe, the prevalence of dogs with AF associated with DCM was 21.2%, while 3% of dogs had DCM and sinus rhythm, and 8 dogs (1.6%) had AF without evidence of DCM.6 While in one study5 conducted in a relatively small number of dogs, the author concluded that in the majority of IWs atrial fibrillation is well tolerated and not responsible for the development of progressive cardiovascular dysfunction, other authors6 reported progressive atrial and ventricular dilatation resulting in development of DCM and congestive heart failure in a significant number of Irish wolfhounds that initially presented with AF as the only abnormality: out of 115 IWs with AF, 42 dogs were lost to follow up, 13 died of unrelated illness, six died suddenly, and 39 dogs developed clinical signs of DCM and CHF after a mean time of 27 months in males and 24 months in females. Breed specific reference values for 2D- and M-mode echocardiographic measurements and characterization of the echocardiographic appearance of different stages of DCM in Irish wolfhounds (IW) have been established in earlier studies.9,10 In one genetic investigation of a line of IWs, there was evidence of genetic involvement in DCM, with a suspected autosomal dominant mode of inheritance, but due to in-breeding, large litter size and late onset in some cases, other modes could not completely be ruled out.11 The objective of this longitudinal study was not only to determine the prevalence of DCM and characterize the time course and inheritance of the disease in a large cohort of dogs, but also to gain and evaluate informations about the life expectancy of IWs, the overall effect of DCM on that expectation, and find out if whole blood taurin might be too low in a significant number of these giant breed dogs.
Methods
More than 1000 Irish wolfhounds in the Netherlands, Belgium, and Germany were screened for the presence of DCM by physical examination, electrocardiography, and 2D and M-mode echocardiography by one examiner (AV) as part of a prospective, longitudinal study. Screening criteria for DCM included the following echocardiographic parameters: LVIDs > 41 mm, LVIDd > 60 mm, and FS < 25%; additional measurements recorded from 2D 4-chamber view for detection of left atrial and right ventricular dilatation included LAd > 56 mm, RVIDd > 35 mm and the presence of atrial fibrillation (AF) or other arrhythmias from ECG recordings. In dogs with advanced stages of DCM, presence and degree of congestive heart failure (CHF) was evaluated from thoracic radiographs. Following initial examination, dogs were reexamined every 6 to 12 months or when possible. Breeders and owners were asked to report the date and circumstances of death when it occurred.

Prevalence and Characterization of DCM
Based upon longitudinal evaluations of a large population of Irish wolfhounds (more than one-thousand dogs) during a 12 year period, DCM and/or atrial fibrillation were diagnosed in almost one third of dogs. Males were overrepresented. The mean age of dogs at the time of diagnosis with DCM was approximately 4 yrs. Animals with decompensated heart failure were most often characterized by pleural effusion (=accumulation of fluid within the thorax outside of the lungs) with some degree of pulmonary edema (=accumulation of fluid within the lungs). Less commonly, mild pericardial effusions or ascites (=accumulation of fluid within the abdomen) were observed. Although some IWs were detected with AF but without echocardiographic evidence of DCM initially, these animals tended to develop overt DCM later on.

Effect of DCM on survival
Survival information was available from more than 400 dogs with reported causes of death. Of these, about half had been affected with cardiomyopathy; in addition, 350 dogs were confirmed alive at time of this writing, of these, slightly less than one-fifth had been diagnosed with DCM. Kaplan-Meier survival curves were constructed from the date of birth until the date of death for dogs that had died as a result of DCM, and for dogs that had died of other causes with no evidence of DCM. The survival curves were compared by the Log Rank test, and were found to be significantly different between groups. Median life expectancy for Irish wolfhounds that have died to date was about 6 years. DCM had a clear and statistical significant adverse effect on survival, but due to predisposition of Irish wolfhounds to a variety of other disease complexes with negative influence on longevity, the magnitude of the DCM effect on survival was not as great in this breed as it is generally considered to be in other breeds. The median survival advantage of dogs that were not affected by DCM was about 5 months compared to dogs which died from DCM.

Whole blood taurine in Irish wolfhounds
Taurine is a sulfur containing amino acid essential for normal cardiac function. Low blood taurine has recently been documented in dogs of various breeds with DCM, and in a substantial portion of Newfoundland dogs, another representative of a giant breed dog. Taurine supplementation or changing dietary protein source resulted in general improvement in left ventricular morphology and function.
During heart-screening examinations blood samples for determination of whole blood taurine were collected from more than 100 Irish wolfhounds, almost half of these were affected with DCM. According to diet history, the majority of dogs were fed a number of different complete and balanced commercial diets. Approximately half of the unaffected and the DCM dogs had whole blood taurine < 200 nmol/ml which is reported as the low normal borderline. But in both groups, the percentage of dogs with very low whole blood taurine (<130 nmol/ml) was low. Preliminary observations on the dietary history of the dogs does not indicate a clear and constant association between the diet and taurine status. Some commercial diets resulted consistently in whole blood taurine > 200 nmol/mL while other diets resulted in wide blood taurine variations from very low to the high normal range.

Results indicate that as reported in Newfoundlands, also Irish wolfhounds have a significant prevalence of mild to moderate taurine deficiency in dogs on commercial complete and balanced diets. Preliminary observations don’t indicate that in IWs taurine deficiency is an underlying cause for development of DCM, but in a breed with a genetic predisposition to develop DCM, systemic taurine deficiency should be avoided as it might contribute to a more rapid disease progression.

**DCM Inheritance in Irish wolfhounds, complex segregation analysis**

We analysed mode of DCM inheritance using regressive logistic models by testing for different mechanisms of genetic transmission. Up to 15 generations were available for some dogs. DCM is genetically transmitted in Irish wolfhounds. Mode of inheritance is complex and involves major genes as well as modifying genes. About half of the offspring from an affected IW with DCM can be expected to develop DCM.

Facing the high prevalence of DCM in IWs, it is in the best interest to improve breeding health and reduce the incidence of this condition. A procedure for the simultaneous prediction of breeding values and the estimation of genotype probabilities for DCM in individual dogs can be expected to markedly improve breeding programmes.

Genetic research will be continued to identify the mutation of the major gene responsible for DCM.

Currently, the best way to diagnose DCM in the dogs are standardized heart examinations including ECG and echo. In some European states breeders already attempted to reduce the prevalence of the disease by selective breeding. A major problem is that even if ECG and ultrasound examinations are carried out on a regular basis in breeding stock, some dogs may already have produced progeny by the time the disease is diagnosed.

Suggestions from the author:

1) All breeding dogs should have heart examinations performed on an annual basis not only as long as they are used for breeding but also later in life to make sure they did not develop DCM. Proven healthy (> 8 yrs old) parents and grandparents are the most valuable information we can get concerning the likelihood for development of DCM in an individual dog used for breeding today.

2) Heart examinations must be performed in a standardized fashion and all results must be centrally registered and become available for official publication.

3) The number of litters produced by an individual dog preliminarily must be restricted to a maximum (suggestion: 5 litters).
Today there are possibilities available to preserve semen from sires which can be used later again, after it was proven that the dog did not develop DCM.
More litters can be permitted for dogs with proven normal hearts of both parents or all 4 grandparents (i.e. dogs were reexamined and still normal at/after 8 yrs. of age).

References:


Curriculum vitae

Married to a veterinarian, Hans Vollmar, 2 children, 15 and 13 years of age.

After studies of veterinary medicine at Hannover vet-school and DVM, postgraduate education as a specialist of small animal vet. medicine (Fachtierarzt für Kleintiere), 1987, and Ophthalmology, 1993.

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