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Clinical significance of ventricular ectopy and development of dilated cardiomyopathy in Irish wolfhound dogs

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Abstract *Introduction/Objectives:* Cardiac disease is the second leading cause of death in Irish wolfhound (IW) populations (15.27%). Dilated cardiomyopathy (DCM) is a common hereditary disease in IWs. No correlation between ventricular premature complexes (VPCs) and DCM has been noted in IW populations. The authors hypothesized that VPCs on a baseline electrocardiogram (ECG) do not predict the development of DCM.
Animals, Material and Methods: Twenty-four IWs of breeding age were prospectively recruited in the study. Irish wolfhounds underwent a cardiac examination, echocardiogram, laboratory testing, and 24-h Holter monitor (HM). Enrolled dogs had a normal echocardiogram, at least one VPC on a 1-min ECG, and HM placed within 90 days of echocardiogram. Irish wolfhounds were followed up for a minimum of 1 year and underwent a follow-up HM within 12 months.
Results: Three of 24 IWs (16.67%) developed structural changes and/or atrial fibrillation consistent with DCM during the study period. Ten of 24 dogs (41.6%) developed arrhythmias necessitating medical therapy. There was no significant

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association between complexity of VPCs and the development of DCM.
Study Limitations: Limitations include the small number of dogs and lack of standardized recheck interval and treatments.
Conclusions: Three of 24 IWs ultimately developed DCM at least three years after enrollment. No correlation can be made between VPCs on screening ECG and development of DCM within 12 months. Ten of 24 dogs were started on anti-arrhythmic medication, as indicated by arrhythmia complexity. An HM is recommended for all IWs with VPCs noted on surface ECG.

Abbreviations

AF	atrial fibrillation
DCM	dilated cardiomyopathy
ECG	electrocardiogram
IW	Irish wolfhound
HM	Holter monitor
VPC	ventricular premature complex
VT	ventricular tachycardia

Introduction/objectives

Dilated cardiomyopathy (DCM) is a common hereditary disease in Irish wolfhound (IW) populations that carries high morbidity and mortality risk [1,2]. Dilated cardiomyopathy in a North American population of IWs has been previously characterized by the development of progressive left atrial dilation disproportionate to the degree of left ventricular dilation with or without concurrent atrial fibrillation (AF) [1]. Contrary to DCM in other breeds, systolic function is often maintained in IW[f] [1,2]. Multiple genetic loci have been associated with DCM in the IW; however, no definitive individual gene mutations have been specifically identified as causing DCM in the breed [3,4]. The prevalence of DCM has been extensively reviewed in both North American and European populations [1,2]. Cardiac disease is the second leading cause of death in North American IW populations, including sudden cardiac death (15.27%). A survey study of 500 IWs presenting to a veterinary clinic in Germany found a prevalence for DCM of 24.2% and that for AF of 23.4% [5]. In this same population of IWs, only 13 dogs (2.6%) had a diagnosis of ventricular premature complexes (VPCs) without evidence of cardiac disease [5]. Another study reported that 37 of 151 European IWs diagnosed with preclinical or clinical DCM had electrocardiogram (ECG) abnormalities other than AF, predominantly VPCs [6]. This study also showed that 89.6% of IWs with clinical DCM and AF experienced cardiac-related death, with an average survival of 7.3 months from the time of diagnosis [6]. In contrast, IWs with preclinical DCM, with or without AF, experienced longer survival times of 21.9 months and 29.1 months, respectively [6]. This demonstrates the importance of early diagnosis and treatment of DCM in the preclinical stage.

Ventricular premature complexes can be seen secondary to primary cardiac disease or systemic illness. Many non-cardiac causes of VPCs in dogs have been documented, including gastric dilation-volvulus, splenic tumors or torsion, and pancreatitis [7]. While it is well documented that AF is correlated with the development of DCM in IWs, it is not yet clear if VPCs have a heritable basis that may influence breeding recommendations.

The presence of VPCs can be a precursor of structural myocardial disease in both Doberman pinschers and boxers [8,9]. Although AF has been shown to be correlated with DCM in IWs, there is limited knowledge of the relationship between ventricular ectopy and DCM in the breed [9]. This study examines the correlation between single VPCs on a baseline ECG in IWs with no history of AF and the development of DCM. The authors hypothesized that VPCs on a baseline ECG do not predict the development of DCM in the IW.

Animals/methods and materials

In this retrospective cohort study, apparently healthy IWs were recruited from breed-specific cardiac screening clinics in North America between

[f] Tyrrell WD, Abbott J, Green H, Rosenthal S, Dentino M, Abrams F. An Update on Cardiac Disease in the Irish Wolfhound: The North American Experience. J Vet Intern Med, 2015;29:1257—1283.

April 2007 and May 2022. The Irish Wolfhound Club of America provides incentives to owners by subsidizing the cost of screening echocardiograms and ECGs and assists in advertising and organizing these screening clinics. Informed consent was obtained from all owners prior to screening. At the time of enrollment, owners were asked to complete a survey with information regarding current medical history, medications, and any known history of familial heart disease. Each dog underwent a focused cardiac examination, standard 6-lead ECG of at least one-minute duration, echocardiogram performed by 1 of 2 authors who are both board-certified veterinary cardiologists (W. D. T. or S. L. R.), laboratory testing when possible (including complete blood count, serum chemistry, thyroid panel, and ± infectious disease panel), and 24-h Holter monitor (HM). Echocardiography was performed using standard techniques with the dogs standing, and measurements were made on 3 consecutive cardiac cycles on 2-dimensional and M-mode echocardiograms, as previously described [8—10]. Left ventricular systolic myocardial dysfunction was identified when left ventricular internal dimension (LVIDs) exceeded the upper limit of the 95% reference interval, as previously reported [1]. Dogs were eligible for the study if they had a normal initial echocardiogram, the presence of at least one VPC on baseline ECG, and HM placement within 90 days of the echocardiogram. Included IWs underwent at least one follow-up HM and echocardiogram after 12 months from the initial VPC diagnosis. Ventricular premature complexes—noted on HM—were graded on a scale from 0 to 4, as previously described; 0 for no VPCs, one for single VPCs, two for bigeminy or trigeminy, three for couplets or triplets, and four for R-on-T phenomenon or ventricular tachycardia (VT) [11].

Dogs were excluded from the study if there was AF on the baseline ECG, a previous or concurrent history of antiarrhythmic treatment, known systemic disease, or a 12-month HM was not performed. Dogs were followed up for a minimum of one year.

Statistical analysis was performed using commercially available software.[g] Fisher's exact test was used to assess associations between VPC complexity (0—4) and the development of DCM. P values <0.05 were considered statistically significant. Sample sizes were insufficient to assess associations between VPC complexity and the development of DCM while adjusting for time.

[g] SAS (version 9.4), Cary, NC, USA.

Results

Between 2007 and 2022, 978 IWs had ECGs performed and submitted to the Irish Wolfhound Foundation. Of these, forty-three (4.4%) dogs had unifocal VPCs as the only abnormality and were eligible for inclusion in the study. Nineteen IWs were excluded. Twenty-four dogs successfully met the inclusion criteria: thirteen females (54.2%)—6 spayed, 5 intact, and 2 with unknown altered status—and 11 males (45.8%)—1 neutered, 6 intact, and 4 with unknown altered status. Ages ranged from 1.8 to 9.5 years, with a mean age of 5.6 years and median age of 5.3 years at the time of onset of VPCs. Of the 24 dogs deemed eligible for this study, 3 of 24 IWs (16.67%) developed structural cardiac changes and AF consistent with DCM. One additional dog (intact female) developed AF but at the time of writing did not have structural changes consistent with DCM. All three dogs that developed DCM were intact males. None of the enrolled dogs developed structural heart disease within 1 year of VPC diagnosis. Of the three dogs that developed DCM, the diagnosis was made 4, 6, and 7 years from the initial VPC diagnosis (mean: 5.6 years of age). Of the four dogs that developed AF, the mean age was 7 years (range: 5—9 years). Two dogs diagnosed with DCM had AF diagnosed concurrently with DCM, while the remaining dog was diagnosed with AF approximately 1 year before diagnosis of DCM. One IW was in congestive heart failure based on the presence of pleural and peritoneal effusion at the time of initial DCM diagnosis. This dog acutely died 3 months later of suspected cardiac causes based on necropsy reports. Six of 24 IWs (25%) succumbed to neoplasia within 1 year of VPC diagnosis. Of the three dogs with DCM, the mean age of diagnosis was 7.3 years (range: 6.5—9).

Association for VPC complexity and development of DCM ($P=0.170$) was compared via Monte Carlo estimates for exact test. No statistical significance was found.

Anti-arrhythmic therapy was initiated in 10 of 24 IWs (41.6%) after HM based on the recommendations of a board-certified veterinary cardiologist. The most used anti-arrhythmic agent was sotalol, with 9 of 10 initially starting sotalol for ventricular and/or supraventricular arrhythmias. The mean number of VPCs was 1650 (range: 0—8755), with 8 of 24 dogs having short runs of non-sustained VT (beats in run ranged from 4 to 42 beats). An additional two dogs had cardiologist recommendations to start antiarrhythmic medication, but these recommendations were not followed by the owner.

Table 1 Cause of death.

Cause of death	Frequency
Osteosarcoma	8
Splenic hemangiosarcoma	1
Pneumonia complications	3
Sudden death	4
Surgery/anesthesia complications	2
Acute hemorrhage of unknown etiology	1

The mean age of death was 7.5 years (range: 4.5—10 years), with 5 of 24 dogs still alive at time of publication (see Table 1). Four of 24 dogs experienced sudden death, one of which was previously diagnosed with DCM and had a suspected cardiac cause of death via necropsy report. Two of the four were not receiving anti-arrhythmic medications at the time of death. One of the four dogs that experienced sudden death was suspected to have suffered snake envenomation. Other causes of death include surgery complications, general anesthesia complications, and acute oral/nasal hemorrhage of unknown etiology.

Discussion

The present study explored the relationship between VPCs on surface ECG and the development of DCM. No relationship was found. Three dogs in this study developed DCM. All three dogs were diagnosed with AF either before or at the time of diagnosis of structural disease, and diagnosis of DCM did not occur for four or more years after the initial finding of ventricular ectopy on a screening surface ECG.

There are two classical forms of DCM noted on postmortem examination, including a fibrofatty version and an 'attenuated wavy myofiber' type [12]. Previous studies have described the histopathologic changes observed in IWs with DCM as mild to moderate multifocal myocardial fibrosis with variable myocardial adipocyte infiltration in the right and left ventricular myocardium [13]. In that study, however, a third (5/15) of the hearts evaluated had no ventricular myocardial changes noted on histopathology [13]. The variation and sometimes the absence of ventricular abnormalities on histopathology may explain the lack of association of clinically relevant ventricular arrhythmias and relatively preserved ventricular systolic function observed in the IW. In contrast, ventricular arrhythmias are commonly seen in Doberman pinschers, often preceding the development of structural disease [8,9,12]. Ventricular premature complexes are a cardinal finding in the occult phase of DCM in Doberman pinschers, with 25—30% of dogs experiencing sudden cardiac death as the only clinical sign [12]. Given the lack of consistent histopathological ventricular changes and preserved ventricular systolic function observed in IWs with DCM, it is unlikely that VPCs would be indicative of occult DCM when compared to DCM in other breeds. This is supported by the findings of this retrospective study, which showed no significant correlation between VPCs based on complexity and development of DCM within 12 months of VPC diagnosis in the IW.

In the present study, it was recommended to start antiarrhythmic therapy in 41.6% (10/24) of the IW after their initial Holter monitor due to high supraventricular and/or ventricular arrhythmia burden. Eight dogs had short runs of non-sustained VT (beats in runs ranging from 4 to 42 beats). The most prescribed anti-arrhythmic medication was sotalol at an initial dose between 1 and 1.7 mg/kg twice daily for those dogs whose weight was recorded. In other breeds, a 5-min 12-lead ECG has been shown to be insensitive when compared with Holter monitoring for detecting ventricular arrhythmias [14], and Orthopedic Foundation for Animals (OFA) certification requires HM clearance within 90 days of the screening echocardiogram. Currently, OFA certification for the IW does not require HM testing and 6-lead ECGs are routinely performed at screening clinics. Recommendations to change OFA certification requirements cannot be definitively made based on this study; however, further investigation looking at routine HM screening utility in IWs for breeding soundness should be investigated.

Limitations

There are several limitations to the present study. Only 24 dogs were included in this study. The small sample size may not be a true representation of the breed. Given the broad enrollment window, advances in technology and techniques used today when compared to 2007 might result in changes that introduce minor discrepancies in measurements but are considered overall unlikely to cause changes to the findings of this study. Interobserver and intra-observer variability of the echocardiograms was not performed. The long enrollment window and a change in data storage platforms during the enrollment period precluded this evaluation. Variability between measurements

performed by experienced echocardiographers has been previously shown to be low [15—17], but lack of repeated measurement remains a drawback. Due to known systemic causes of VPCs, baseline blood work was recommended at enrollment of the study but was not considered an exclusion criteria if full diagnostic lab work including tick titers was unavailable for retrospective analysis. Recent reports show high-pulse diets fed to apparently healthy IWs led to a higher prevalence of VPCs than in IWs fed a low-pulse diet [18]. Diet was not considered in the present study. Some dogs were lost to follow-up or had inconsistent follow-up beyond the 1-year requirement for inclusion, and there were too few follow-up HMs for statistical evaluation. As with other breeds, cardiomyopathy in the IW has a long preclinical phase; therefore, some dogs included in this study may have developed structural cardiac changes consistent with DCM beyond the year-long period required for inclusion in this study. Without performing post-mortem examinations, the true phenotypes of the dogs included in this study are unknown. However, within a year of VPC diagnosis, none of the IWs included in this study developed changes consistent with DCM. Lastly, there was no standardization of treatment for dogs in this study. Treatment recommendations were made at the discretion of the attending cardiologist. Similarly, echocardiographic and ECG reports with measurements were available for review but the echocardiographic images and/or ECGs were not always available for present analysis. Therefore, pre-existing echocardiographic measurements obtained from board-certified cardiologists and reported presence of VPCs on baseline ECG during echocardiogram were used for determination of suitability for inclusion in this study.

Conclusions

The results of this study do not show any correlation between VPCs on screening ECG and the development of DCM when evaluated after 12 months. Three of the 24 included IWs ultimately developed DCM three or more years after the initial screening, indicating that future studies should focus on the longer-term consequence of observing initial VPCs in IWs. Importantly, 10 of the 24 (41.7%) dogs displayed clinically relevant arrhythmia complexity and were started on anti-arrhythmic medication, indicating that an HM is recommended for all IWs with VPCs noted on surface ECG to determine if the arrhythmias are severe enough to warrant treatment. Careful auscultation and yearly ECGs are ideally recommended for all IWs. If an arrhythmia is noted on physical examination or ECG, additional cardiac workup, including echocardiography and Holter monitoring, is recommended as DCM incidence does increase with age. An ECG or HM at a single point in time does not indicate an IW will be free from heart disease for their lifetime.

Declaration of Competing Interest

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

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