

COMPARATIVE M-MODE ECHOCARDIOGRAPHIC STUDY OF MALE AND FEMALE DOGS WITH DEGENERATIVE MITRAL VALVE DISEASE (DMVD)

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ABSTRACT

Via the M-mode echocardiographic study of dogs, without and with pulmonary edema, various statistically significant differences were obtained in dogs separated by gender and showed the different response of the myocardium to the volume loading of the male and female dogs with MVD.

Key words: dogs, male, female, myxomatous, pulmonary, edema.

Introduction

In 2005 researchers examined a sample of 317 dogs with radiologically diagnosed heart disease, and provided their percentage distribution as follows: 225 dogs (70.98%) had mitral insufficiency, 54 (17.03%) had dilated cardiomyopathy, 28 (8.83%) had cor pulmonale, 5 (1.58%) had pericarditis, 3 (0.95%) had arterial stenosis and 2 (0.63%) had pulmonary artery stenosis. Based on these data, they confirm the assertion by many other authors that mitral insufficiency is the most common heart disorder among dogs. A strong predisposition to this disease is seen among small and medium-sized dog breeds. Mitral insufficiency was diagnosed in 211 out of a total of 238 dogs belonging to these breeds (88.66%) and only in 14 of 79 large breed dogs (17.72%). In the group of small and medium breed dogs the incidence of the disease was higher among male animals (58.77% male, respectively 41.23% female) (10). In addition, males have a tendency for a faster progression of heart failure (4).

Just recently, one study found a significant relationship between MR and gender in terms of mortality ($P = 0.035$) (6). The study showed that male dogs with moderate to severe MR had a higher mortality rate than males without MR. The researchers do not establish such a relationship in females. The severity of this cardiac disorder is also related to many other factors, for example, a study had shown that weight gain in dogs with myxomatous valve degeneration is associated with the survival rate of the animals where the longer surviving dogs are those with increased body mass (9). These facts show that the development of heart failure in case of MVD is a multifactor process.

Materials and methods

In order to achieve this goal, the dogs with left apical systolic murmur were divided by gender, forming two groups – male and female dogs. Based on the results obtained after the radiographic examination, the patients were further divided into two subgroups. First subgroup – dogs with MVD without pulmonary edema and second subgroup – dogs with MVD with pulmonary edema.

The ultrasound was done using the My Lab 70 vet XV apparatus manufactured by the Italian company Esaote. The patients were examined with a specialized cardiac phase matrix transducers suitable for cardiac patients from the small dog breeds in the right parasternal position. The chest radiographs were performed with a direct X-ray system in LLR (left lateral) and VD (ventrodorsal) projections.

To be able to compare echocardiographic measurements in patients of varying sizes, the parameters were indexed, as suggested by Brown et al. (2003), relative to the linear aortic size (Aom). With this way of indexation, the value of each index can be visually interpreted (3).

All indicators representing the echocardiographic M-mode and radiographic dimensions in patients with pulmonary edema and those without pulmonary edema were processed with a computer program (Statistica®, v. 6.0), the nonparametric method (Mann-Whitney U test) and a parametric method (One-way ANOVA) and represented as mean $\pm$ its standard deviation (SD). Differences were considered statistically reliable at $p < 0.05$.

Results

All of the obtained results are presented in Tables 1 and 2.

Table 1: M-mode echocardiographic transverse dimensions of male dogs with MVD, indexed to Aom ($\bar{x} \pm SD$)

Index	Without pulmonary edema (n = 6)		With pulmonary edema (n = 8)		Reliability
	$\bar{x}$	SD	$\bar{x}$	SD	
IVSd/Aom	0.484	0.077	0.502	0.144	
LVIDd/Aom	1.962	0.516	2.493	0.499	*
LVFWd/Aom	0.524	0.102	0.556	0.092	
IVSs/ Aom	0.694	0.126	0.826	0.085	*
LVIDs/Aom	1.109	0.341	1.278	0.321	
LVFWs/Aom	0.783	0.136	0.785	0.140	
aΔA	2.745	1.285	4.712	1.666	* ♦
LA/Aom	1.453	0.356	2.168	0.385	** ♦♦

Statistically significant difference determined by the nonparametric method Mann-Whitney U-test: * – $p < 0.05$; ** – $p < 0.01$.

Statistically significant difference determined by the parametric method one way ANOVA: ♦ – $p < 0.05$; ♦♦ – $p < 0.01$; ♦♦♦ – $p < 0.001$.

The results for the M-mode echocardiographic transverse dimensions of male dogs with MVD, indexed to the linear aortic size (Aom), are shown in Table 1 which shows that the size of the linearly indexed end-diastolic interventricular septum (IVSd / Aom) in both groups was very close, and in dogs without pulmonary edema it was statistically insignificantly lower (0.484 ± 0.077) compared to this indicator in the group of patients with pulmonary edema (0.502 ± 0.144).

The size of the linearly indexed LDH (LVIDd / Aom) was statistically significantly (determined by the nonparametric Mann-Whitney method) smaller in the non-pulmonary edema group ($1,962 \pm 0,516$) compared to the group with pulmonary edema ($2,493 \pm 0.499$).

The width of the linearly indexed end-diastolic left ventricular free wall (LVFWd / Aom) was statistically significantly less in the non-pulmonary edema group (0.524 ± 0.102) compared to the patients with pulmonary edema (0.556 ± 0.092).

The size of the linearly indexed end-diastolic interventricular septum (IVSs / Aom), determined by the nonparametric Mann-Whitney method, was statistically significantly lower in dogs without pulmonary edema (0.694 ± 0.126) than in those with pulmonary edema (0.826 ± 0.085).

The size of the linearly indexed end-sistolic left ventricular inter diameter (LVIDs / Aom) is statistically insignificantly smaller in the group without pulmonary edema (1.109 ± 0.341) compared to the pulmonary edema group (1.278 ± 0.321).

The width of the linearly indexed left atrial diameter (LVFWs / Aom) is similar and close in the dogs in both groups – in those without pulmonary edema (0.783 ± 0.136); with pulmonary edema (0.785 ± 0.140).

The mean value of the linearly indexed left atrial diameter (LA / Aom) in patients without pulmonary edema was 1.453 ± 0.356 and was statistically significantly (determined by the two statistical methods) lower than that found in the pulmonary edema group (2.168 ± 0.385).

The size of the linearly indexed short-axis strike area (aΔA) in the non-pulmonary edema group ($2,745 \pm 1,285$) was statistically significantly less than that in the patients with pulmonary edema ($4,712 \pm 1,666$). This was also determined using both methods for calculating the statistically significant difference.

The mean value of the linearly indexed left atrial diameter (LA / Aom) in patients without pulmonary edema was 1.453 ± 0.356 and was statistically significantly (defined by the two statistical methods) lower than that found in the pulmonary edema group (2.168 ± 0.385).

Table 2: M-mode echocardiographic transverse dimensions of female dogs with MVD, indexed to Aom ($\bar{x} \pm SD$)

Index	Without pulmonary edema (n = 3)		With pulmonary edema (n = 3)		Reliability
	$\bar{x}$	SD	$\bar{x}$	SD	
IVSd/Aom	0.475	0.082	0.392	0.058	
LVIDd/Aom	2.112	0.185	2.453	0.190	
LVFWd/Aom	0.518	0.085	0.450	0.031	
IVSs/ Aom	0.741	0.230	0.633	0.110	
LVIDs/Aom	1.162	0.163	1.386	0.178	
LVFWs/Aom	0.790	0.074	0.705	0.055	
aΔA	3.113	0.472	4.099	0.617	
LA/Aom	1.357	0.241	1.957	0.262	* ♦

Statistically significant difference determined by the non-parametric method Mann-Whitney U-test: * – $p < 0.05$; ** – $p < 0.01$.

Statistically significant difference determined by the parametric method one way ANOVA: ♦ – $p < 0.05$; ♦♦ – $p < 0.01$; ♦♦♦ – $p < 0.001$.

The results of the M-mode echocardiographic transverse dimensions of female dogs with MVD, indexed to the linear aortic size (Aom), are shown in Table 2 which shows that no statistical differences were found between female dogs without and with pulmonary edema with respect to the following sizes: IVSd / Aom, LVIDd / Aom, LVFWd / Aom, IVSs / Aom, LVIDs / Aom, LVFWs / Aom and aΔA.

The mean value of the linearly indexed left atrial diameter (LA / Aom) determined by the two methods for calculating the statistically significant difference in the non-pulmonary edema group was (1.357 ± 0.241) which is statistically significantly lower than that found in the patients with pulmonary edema (1.957 ± 0.262).

Discussion

IVSd/Aom had no statistically significant difference in males and females. In contrast to male dogs, in which this echocardiographic size increases as the disease progresses, in female dogs it decreases. The same pattern was observed with IVSs/Aom, which was found to be statistically significantly greater in dogs with pulmonary edema. These discrepancies in IVS echocardiographic dimensions in males and females may be due to different myocardial response to volume overload, and this may explain the higher gradient of systolic apex noise in males (8) and the higher prevalence of the disease in them (10).

In males the remodeling is directed towards thickening of the heart walls and the development of hipercontractility as the heart disease progresses. We found the complete opposite in female dogs – the progression of MVD is associated with thinning of IVS and decreasing of its contractility. These differences between males and females in the left ventricular remodeling with the progression of MVD may require different treatment regimens.

The lack of significant difference in the sizes LVFWd/Aom and LVFWs/Aom in male and female dogs, creates a model for semi remodeling. In our opinion, this model of remodeling is a result of the lack of support of the free wall which is provided to the septum by the blood pressure in the right ventricle. Thus, the increased sympathetic stimulation in dogs with pulmonary edema exerts its influence more strongly on the interventricular septum with respect to its contractility. The lack of support for the free left ventricle wall causes the increased volume load to counteract to the sympathetic stimulation, and thus not allowing it to effect its contractility.

Statistically, the significantly larger size of LVIDd / Aom in male dogs contrasts with the lack of statistical reliability in terms of the same size in female dogs. This fact, along with the established lack of a reliable difference in the size of LVIDs / Aom in male and female dogs, shows the hyperkinetic nature of the cardiac activity in male dogs with MVD. This may be associated with a larger mitral valve defects in male animals or a significantly stronger response of their myocardium to sympathetic stimulation, factors directly related to the increased volume overload and the enlargement of the left atrium. In principle, it is proven that the increase in LA / Ao is a risk factor indicating the progress of MMVD (1, 2, 5, 7). In dividing the patients by gender we found a higher statistically significant difference (between dogs without edema and pulmonary edema) in terms of the size LA/Aom in male dogs compared to the females. It is possible that the higher statistical accuracy (determined by both of the methods for calculation of the statistical significance – Mann-Whitney U-test and one way ANOVA) is associated with the more rapid progression of the MVD in male dogs. This is a strong argument supporting the thesis of the usefulness of the separation by gender in DMVD studies. We assume that the established higher statistically significant difference between LA / Aom, which is directly related to the increased volume loading in male dogs, is directly related to the faster progression of MVD in them as well.

The assumed by us stronger response to the sympathetic stimulation of the myocardium of the male dogs compared to the females and the probably higher grade damage of the mitral valve, leading to greater volume overload, was also confirmed by the statistically significantly greater change in the internal Δ AA space in male dogs with the progression of MVD and the lack of statistically significant reliability in females in terms of the same size between patients without pulmonary edema and those with pulmonary edema.

Conclusion

1. The echocardiographic M-mode study showed that when dividing the dogs by gender in DMVD studies, we obtained statistically significant differences for more left ventricular dimensions in male dogs as the disease progresses.
2. When separating the dogs with MVD by gender, we identified a different myocardial response of males and females to volume overload.
3. It is possible that such a different myocardial reactivity in this disease is associated with the choice of different treatment regimens in male and female dogs.

References

1. Borgarelli, M., P. Savarino, S. Crosara, R. A. Santilli, D. Chiavegato, M. Poggi, C. Bellino, G. La Rosa, R. Zanatta, J. Häggström, A. Tarducci. (2008). *Survival Characteristics and prognostic variables of dogs with mitral regurgitation attributable to myxomatous valve disease*. Journal of Veterinary Internal Medicine, 22, 1, 120–128.
2. Borgarelli, M., S. Crosara, K. Lamb, P. Savarino, G. La Rosa, A. Tarducci, J. Haggstrom. (2012). *Survival characteristics and prognostic variables of dogs with preclinical Chronic Degenerative Mitral Valve Disease attributable to myxomatous degeneration*. Journal of Veterinary Internal Medicine, 26, 1, 69–75.
3. Brown, D. J., J. E. Rush, J. MacGregor, J. N. Ross Jr, B. Brewer, W. M. Rand. (2003). *M-mode echocardiographic ratio indices in normal dogs, cats, and horses: A novel quantitative method*. Journal of Veterinary Internal Medicine, 17, 653–662.
4. Buchanan, J. W. (1977). *Chronic valvular disease (endocardiosis) in dogs*. Advances in Veterinary Science and Comparative Medicine, 21, 75–106.
5. Moonarmart W., A. Boswood, V. Luis Fuentes. (2010). *N-terminal Pro B-type natriuretic peptide and left ventricular diameter independently predict mortality in dogs with mitral valve disease*. Journal of Small Animal Practice, 51, 84–96.
6. Reimann, M. J., J. E. Møller, J. Häggström, T. Martinussen, S. S. C. Zatrzeni, L. Svanholm, L. B. M. Nielsen, H. D. Pedersen, L. H. Olsen. (2017). *Mitral regurgitation severity and left ventricular systolic dimension predict survival in young Cavalier King Charles Spaniels*. Journal of Veterinary Internal Medicine, 31, 4, 1008–1016.
7. Reynolds, C. A., D. C. Brown, J. E. Rush. (2012). *Prediction of first onset of congestive heart failure in dogs with degenerative mitral valve disease: The PREDICT cohort study*. Journal of Veterinary Cardiology, 14, 1, 193–202.
8. Serfass, P., V. Chetboul, C. C. Sampedrano, A. Nicolle, T. Benalloul, H. Laforge, C. Gau, C. Hebert, J. L. Pouchelon, R. Tissier. (2006). *Retrospective study of 942 small sized-dogs: prevalence of left apical systolic heart murmur and leftsided heart failure, critical effects of breed, and sex*. Journal of Veterinary Cardiology, 8, 11–18.
9. Slupe, J. L., L. M. Freeman, J. E. Rush. (2008). *Association of body weight and body condition with survival in dogs with heart failure*. Journal of Veterinary Internal Medicine, 22, 3, 561–565.
10. Stanin, D., B. Škrin, M. Ščić, B. Radišić. (2005). *Radiological analysis of mitral insufficiency among dogs in Croatia*. Veterinarski Arhiv, 75, 4, 329–337.