

## COMPARATIVE ECHOCARDIOGRAPHIC EXAMINATIONS IN DOGS WITH MYXOMATOUS DEGENERATION OF THE MITRAL VALVE DEPENDING ON THE PRESENCE OR ABSENCE OF PULMONARY EDEMA

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### ABSTRACT

In the article echocardiographic studies of 12 dogs with myxomatous degeneration of the mitral valve were compared. Patients were divided into two groups according to the presence or absence of pulmonary edema. The echocardiographic measurements were indexed to Ao - size. We received significant difference ( $p = 0.0077$ ) only for LA / Ao, but this ratio can not be a predictor for the development of heart failure.

**Key words:** dogs, myxomatosis, mitral valve, pulmonary edema.

### Introduction

Mitral valve disease (MVD) is characterized by myxomatous valve degeneration and is the most common cardiac disease in the small dog breeds [3, 5, 7]. The first sign detectable by routine clinical examination is the left apical systolic murmur, which results from the abnormally positioned mitral valve flaps in the systolic phase of the cardiac cycle. MVD progression is manifested by left atrial and ventricular dilation and supraventricular arrhythmias; at a later stage, pulmonary edema might occur as a result of the left ventricular heart failure. The prevalence of MVD increases significantly with age. It has been established that the incidence and progression of the disease was higher and more rapid in male than in female dogs [6, 7].

Conventional ultrasound has a major role in the initial evaluation and monitoring of disease progression over time by performing periodic examinations to provide information about the anatomy of the mitral valve, left ventricular size, presence and severity of regurgitation. Several standard echocardiographic variables have been shown to be associated with the clinical outcome. One of these markers is left atrial/aortic root ratio, which might aid the identification of asymptomatic MVD dogs with a higher risk of early decompensation, which is a major issue in practice. The first effect of the mitral regurgitation on cardiac morphology is left atrial dilation, which may be assessed non-invasively using LA/Ao ratio via 2-D echocardiography. In this study, [9], the authors have established a significant correlation between RF and LA/Ao coefficient.

A study in a large number of animals conducted by Borgarelli and all has shown that only LA/Ao ratio had maintained statistical significance to predict survival in heart-related fatal outcomes in dogs with MVD. Left atrial dilation reflects the severity and chronic progression of mitral regurgitation [4].

Despite the advantages offered by echocardiography, variability in preload and afterload associated with the disease appears to be a limiting factor for ultrasonography in terms of accurate assessment of myocardial function [8]. The disease is characterized by slow clinical progression, sometimes over several years. Many of the affected dogs due to the late age onset and slow progression of the disease might not manifest any clinical signs throughout their lives. Although mitral insufficiency is the most common cause of heart failure, there are very few studies to evaluate the

natural history and potential risk factors for developing the disease in breeds other than Cavalier King Charles and dachshund [4].

The objective of our study is to compare ultrasound parameters between dogs with mitral valve degeneration in terms of the presence or absence of pulmonary effusion, regardless of breed and sex; in relation to the age, we selected patients over seven years.

### **Materials and methods**

For the purpose of fulfilling the study objective, the dogs with left apical systolic murmur underwent X-ray and ultrasound examination. Based on the results, they were divided into two groups. First group included MVD dogs with pulmonary edema while the second group included dogs with MVD without pulmonary edema.

The first group consisted of 6 patients aged 7 to 13 years, of body weight 4.3 to 12.5 kg, and included 4 males and 2 females. Breeds were as follows: 1 Shih Tzu, 1 Bichon frise, 1 Pinscher, 1 Bolognese, 1 Dachshund and 1 mixed. 2 of the dogs had mitral prolapse and 4 dogs had no mitral prolapse. Flail leaflet – with 4 patients, without 2 patients. One of the dogs had evidence of ruptured secondary chords.

The second group was formed by six dogs aged 8 to 14 years, of body weight 4.8 kg to 10 kg, and included 4 males and 2 females. Breeds were as follows: 1 Cavalier King Charles, 1 Pekingese and 4 mixed. All dogs in the second group had mitral prolapse, Flail leaflet and ruptured secondary chords.

Ultra-sonographic examination was performed with apparatus My Lab 70 vet XV (most modern veterinary Doppler apparatus by the Italian company Esaote – a leader in the design and manufacture of ultrasonic systems and software intended for veterinary medicine in Europe).

Patients were evaluated with specialized cardiac matrix phase-array transducers PA023E (frequency of 4–11 MHz) and PA122E (frequency of 3–7 MHz) suitable for small dog breed patients at right parasternal position. Patients have been studied at right parasternal sections – sagittal (long axis) and transverse (short axis).

Chest X-ray have been performed by direct digital radiography (DR X-ray system) in LLR (left lateral) and VD (ventrodorsal) projections.

For the purpose of comparing the echocardiographic measurements in patients of different sizes, the parameters were indexed to aortic size (AO). The use of this ratio was more beneficial as compared to the use of sizes normalized to body weight or body surface area, for AO size did not alter as a result of most cardiac disorders (1) and did not depend on the sex and nutrition of the patients. The value of each index had a visual interpretation (e.g. "How is the left ventricular diastolic size measured in aortic widths?") [2].

Statistical analysis has been performed with the program for statistical processing Statmost.

### **Results and Discussion**

Tabular listing of all results is available in Tables 1, 2 and 3.

Standardized aggregate statistical data from Group 1 - MVD dogs with pulmonary edema have been compared to Group 2 dogs with MVD without pulmonary edema in Table 1 which indicated the average and standard deviation of the investigated ratios.

**Table 1: M-mode Echocardiographic Measurements indexed to the AO of dogs with MVD without pulmonary edema compared with dogs with MVD with pulmonary edema**

| M-mode      | Without pulmonary edema<br>n = 6 |         | With pulmonary edema<br>n = 6 |         | Authenticity<br>P |
|-------------|----------------------------------|---------|-------------------------------|---------|-------------------|
|             | Mean                             | S D     | Mean                          | SD      |                   |
| IVS- d /AO  | 0.4973                           | 0.0340  | 0.5233                        | 0.1692  | 0.7197            |
| LVID- d /AO | 2.1268                           | 0.3040  | 2.3150                        | 0.4669  | 0.4274            |
| LVW- d /AO  | 0.5565                           | 0.0804  | 0.5267                        | 0.1051  | 0.5929            |
| IVS- s / AO | 0.7440                           | 0.1578  | 0.7867                        | 0.1441  | 0.6354            |
| LVID- s /AO | 1.2107                           | 0.1965  | 1.2342                        | 0.3395  | 0.8862            |
| LVW – s /AO | 0.8212                           | 0.0877  | 0.7538                        | 0.1265  | 0.3092            |
| LA/AO       | 1.4397                           | 0.2709  | 1.9898                        | 0.3013  | 0.0077*           |
| Weight kg.  | 8.3333                           | 3.0885  | 6.1833                        | 2.2436  | 0.1978            |
| F S (%)     | 43.0833                          | 4.9829  | 38.8000                       | 0.3162  | 0.0619            |
| Heart rate  | 124.0000                         | 20.1990 | 151.8333                      | 28.3014 | 0.0783            |
| Age         | 10.0000                          | 2.0976  | 10.8333                       | 2.0412  | 0.5014            |

\*  $p < 0.05$ ; IVS – interventricular septum; LVID - left ventricular internal dimension; LVW- left ventricular wall; LA- left atrial dimension ; AO - aortic dimension; FS - Fractional shortening

**Table 2: Dogs with MVD without pulmonary edema**

| M-mode | Djeini | Maks | Qris   | Michael | Mery  | Fiodr  |
|--------|--------|------|--------|---------|-------|--------|
| LA/AO  | 1.176  | 1.16 | 1.686* | 1.587 * | 1.264 | 1.765* |

\* overlap results

**Table 3: Dogs with MVD with pulmonary edema**

| M-mode | Bari  | Roni  | Chiko  | Kuche | Kros   | Poli  |
|--------|-------|-------|--------|-------|--------|-------|
| LA/AO  | 2.212 | 2.268 | 1.541* | 2.075 | 1.688* | 2.155 |

\* overlap results

Significant difference ( $p = 0.0077$ ) has only been established for LA/AO ratio which was higher in dogs with pulmonary edema  $\bar{x} = 1.9898$ , SD 0.3013 against  $\bar{x} = 1.4397$ , SD 0.2709 in dogs without pulmonary edema. The detailed analysis of this ratio (Tables 2 and 3) indicated that three dogs of the first group and two dogs of the second group exhibited overlapping values from 1541 to 1765. Ppulmonary edema was manifested in patients above these values, while no pulmonary edema occurred below these values. The small number of test animals and characteristics of changes associated with myxomatous valve disease did not give us the right to determine the limiting values for this ratio as a predictor of heart failure. In regards to the remaining ratios in dogs with MVD with pulmonary effusion compared to dogs without pulmonary edema, a tendency can be noticed for higher systolic ( $p = 0.6354$ ) and diastolic ( $p = 0.7197$ ) size of the ventricular septum, larger internal transverse diastolic diameter of the left ventricle ( $p = 0.4274$ ), while systolic left ventricular diameter in both groups was almost identical ( $p = 0.8862$ ). The mean deviation of the transverse diameter of the left ventricle has raised an interest. Dogs with pulmonary edema had a significantly higher systolic diameter (SD = 0.3395) compared to dogs without edema (SD = 0.1965) and diastolic (SD = 0.4669) as compared to (SD = 0.3040) internal transverse left ventricular diameter. We presume that this is due to individual differences from disease onset to the manifestation of heart failure in individual patients. The left ventricular free wall had a smaller systolic ( $p = 0.3092$ ) and diastolic size ( $p = 0.5929$ ).

These results are not unexpected and are directly related on the characteristics of myxomatous degeneration of the valve, which is a prerequisite for a retrograde blood flow. The lack of a secure barrier between the left atrium and the left ventricle leads to an increase of the cavity with thinner and therefore more susceptible to stretch walls - left atrium. The latter resulted in significant differences in LA/AO ratio. The increased volume overload and sympathetic stimulation (our study included dogs with pulmonary effusion with an increased heart rate) trigger a left ventricular reaction, however these changes were less manifested due to the thicker and more refractory muscular layer. Hyperextension of the left atrium associated with progressive MVD resulted in a compensatory reduction of the left ventricular wall stress and may only be considered in terms of establishing trends, rather than significant differences in ventricular echocardiographic ratios.

## Conclusions

1. The LA/AO ratio in comparison with the other echocardiographic values in dogs of different breeds, gender, age and body weight is the most indicative parameter of MVD progression. Our team will perform a further research to establish the likelihood of this coefficient to be a prognostic factor for onset of early heart failure.
2. Echocardiographic left ventricular size as indexed towards AO size exhibit only trends where there are no significant differences between dogs without pulmonary edema and dogs with pulmonary effusion, which is related to the characteristics of the pathological changes associated with myxomatous valve degeneration.

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