RESEARCH ARTICLE

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Investigation of Dilated Cardiomyopathy Tendency by Echocardiography in Kangal Dogs

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Abstract

Dilated cardiomyopathy (DCM) is defined as a cardiac disease characterized by dilatation of the cardiac chamber, decreased myocardial contractility, and decreased left ventricular ejection fraction. In this study, we aimed to determine whether there is a tendency to age-related dilated cardiomyopathy in Kangal Dogs, a breed of Anatolia, echocardiographically. The experimental groups of the study consisted of a total of 20 dogs, 10 of which were 1-3 years old, young group (Group I) and the other 10 were 4-7 years old, middle-aged group (Group II), who had no clinical problems at rest. By echocardiographic examination, the end-diastolic thickness of interventricular septum, the end-systolic thickness of interventricular septum, left atrial diastolic diameter, aortic root diastolic inner diameter, the ratio of left atrium diameter to aortic diameter, left ventricular end-diastolic inner diameter, left ventricular end-systolic inner diameter end-systolic thickness of the posterior wall, the end-diastolic thickness of the posterior wall of the left ventricle, heart rate, left ventricular ejection fraction, left ventricular contraction force, stroke volume, cardiac output, end-diastolic volume and end-systolic volume parameters were measured. The data collected from the two groups were analysed using independent samples T-test. In conclusion, considering that the age of the Kangal dogs constituting the middle-aged group (G II) of the study was within the age limits specified in the studies (6-7 years), a myocardial defect indicating echocardiographic DCM was not detected. Thus, it was concluded that there is no tendency for DCM among the age groups included in the study. However, it can be said that echocardiographic evaluation of more coiled dogs in the given age group may give a more reliable result considering that the rate of DCM is 6% in different breed dogs.

Keywords: Dilated cardiomyopathy, tendency, Kangal, Dog.

INTRODUCTION

Cardiac muscle disorders are referred to as cardiomyopathies (CMPs). CMPs lead to cardiac dysfunction resulting in decreased strength of heart muscle contraction (systolic failure) or decreased myocardial relaxation (diastolic failure). Apart from these, damaged heart muscle cells generate impulses as an ectopic focus and cause supraventricular or ventricular arrhythmias (O'Grady and Sullivan, 2004; Gökçe, 2014; Colakoglu and Sahal 2015; Turgut, 2017).

CMPs can emanate primarily or secondarily. The primary group of CMPs includes; dilated, hypertrophic, restrictive and intermediate cardiomyopathies (O'Grady et al., 2008; Gökçe, 2014). In the secondary group of CMPs, there are; toxic, inflammatory, nutritional, metabolic, infectious and infiltrative cardiomyopathies (Mansilla et al., 2019).

Dilated cardiomyopathy (DCM) is characterized by dilatation of the cardiac chamber, decreased myocardial contractility, and decreased left ventricular ejection fraction (Bonagura and Lehmkuhl, 2000). Primarily, dilated cardiomyopathies are known to be a vital cause of heart failure in large dog breeds. DCM is a disorder that progresses insidiously, reduces the contractility of the heart and eventually, results in congestive heart failure (Colakoglu et al., 2022). Heart failures due to secondary and infectious causes are seldomly seen (Grady and Sullivan, 2004; Turgut, 2017).

Although etiologically, primary DCM in dogs may occur due to genetic, familial, age-related, hormonal (hypothyroidism) and metabolic (carnitine, taurine, selenium deficiency) causes, it is mostly expressed as idiopathic DCM (Ettinger and Feldman, 1995; Bonagura and Lehmkuhl, 2000; Turgut, 2017). It is stated that genetic and familial factors play an essential role in most DCM cases and breeds such as; Doberman Pinscher, Scottish Deerhound, Irish Wolfhound, Great Dane, Saint Bernard, Afghan Hound, Newfoundland, Old English Sheepdog, Cocker Spaniels are included (Osterziel et al., 2005; O'Grady et al., 2008; Wiersma et al., 2008; Stephenson et al., 2012; Turgut, 2017). This disease is more common in middle (4-6 years old) and older dogs (Onmaz et al., 2011; Gökçe, 2014). In addition, it is emphasized that factors affecting the functions of myocytes, myocarditis with myocyte necrosis (parvovirus infections), global myocardial ischemia, and toxic destruction of myocytes (doxorubicin and monensin) may also cause dilated cardiomyopathy secondarily (Bonagura and Lehmkuhl, 2000).

DCM develops insidiously in three phases. In the first phase, the heart is morphologically and electrically normal and there is an asymptomatic picture. In the second phase, there is morphological cardiac enlargement and/or electrically ventricular ectopia. However, there is no clinically striking finding (latent course). The third phase is the period in which congestive heart failure (CHF) develops with clinical symptoms (O'Grady et al., 2008).

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Regarding the pathogenesis of the disease, two prominent histopathological disorders are known (Turgut, 2017). First and foremost, myocardial cells are deteriorated giving them a thin-wavy appearance during the latent period, that is, in the preclinical period, which is also considered as a predisposition to DCM (Wiersma, 2008). Secondly there are pathological disorders characterized by myocyte atrophy, lysis, fat infiltration and fibrosis in cardiac muscle cells. In the preclinical stage, dilatation begins to form in the heart cavities as the continuation of the cardiac muscle disorders. Parallel to this, while cardiac index (CI) decreases, sympathetic, hormonal and renal compensatory mechanisms are activated, causing an increase in heart rate, peripheral vascular resistance, and volume retention (Gökçe, 2014; Turgut, 2017). Systolic function deteriorates progressively and all cardiac chambers dilate and the diameter of the cardiac chamber increases, most notably in the left ventricle and left atrium. With the progression of dilatation, the ratio of left ventricular wall thickness to lumen volume decreases. This leads to decreased systolic function, dysfunction in papillary muscles, and regulations due to atrioventricular valve insufficiency (Bonagura and Lehmkuhl, 2000).

As a result of the progressive decrease in systolic function, an increase in left atrial and left ventricular pressure occurs at the end of diastole. Therefore, diastolic insufficiency develops in the dilated ventricle. Owing to that, loss of function in both ventricles and all the symptoms of congestive heart failure (CHF) manifest (Bonagura and Lehmkuhl, 2000; Gökçe, 2014, Turgut, 2017). Acute and severe ventricular arrhythmias may develop in such sick dogs and ultimately, may result in sudden death.

Patients in the latent period and the preclinical stage are asymptomatic (Bonagura and Lehmkuhl, 2000; O'Grady et al., 2008; Turgut, 2017). With the onset of left ventricular dysfunction, tachyarrhythmia emerges as an important symptom. In symptomatic dogs, cardiac arrhythmia, exercise intolerance, laziness, and fatigue predominate as signs of CHF. Aside from that, right and/or left CHF symptoms are seen (O'Grady et al., 2008; Turgut, 2017).

DCM can be diagnosed by evaluating radiographic, echocardiographic, electrocardiographic and laboratory findings (Bonagura, 1994; Dukes-McEwan et al., 2003; Wess et al., 2010; Stephenson et al., 2012; Turgut, 2017). Vollmar (1999), stated that Echocardiography (ECHO) is an important diagnostic method in the evaluation of heart diseases and that it can be used as an effective method in the early diagnosis of DCM in the preclinical stage. Hence, echocardiography is the "gold standard" method used in the diagnosis of DCM (Bonagura and Lehmkuhl, 2000; Wess et al., 2010; Jeyaraja et al., 2015). Also, the European Society of Veterinary Cardiology (ESVC) recommended that the diagnosis of DCM should be based on the results of 2D and M-mode echocardiographic examinations (Dukes-McEwan et al., 2003). For echocardiographic confirmation of DCM, left ventricular dilatation, decreased systolic myocardial function, and increased left ventricular sphericity should be detected (Dukes-McEwan et al., 2003). Generally, these disorders in the left heart are accompanied by right heart dilatation (Bonagura and Lehmkuhl, 2000).

With echocardiographic examination, cardiac functions can be interpreted based on quantitative values such as; fractional contraction of the heart muscle, ejection fraction, atrial and ventricular cavity dimensions, and qualitative evaluations such as; hypokinesia and hyperkinesia (Ettinger, 1991).

In this study, it was aimed to investigate whether DCM, which is progressive heart disease, causes CHF in patients in a short time, and causes death by affecting mostly large dog breeds, has a tendency to develop age-relatedly in Kangal dogs.

MATERIALS AND METHODS

The experimental groups of the study consisted of a total of 20 dogs, 10 female and 10 male Kangal dogs, aged between 1-7, which were domesticated by indigenes, did not have any clinical conditions at the time of rest. These dogs were divided into two groups, 10 (5 females, 5 males) 1-3 years old, young group (Group I=G1), 10 (5 females, 5 males) 4-7 years old, middle-aged groups (Group II).

Before the ECHO examination of the dogs used in the study, pulse and respiratory frequency, body temperature and mucosal examinations were performed. Then, in a calm and stress-free environment, M-mode echocardiographic examinations for DCM were performed from the right parasternal region with a Z6-Vet echocardiography device following the technique's protocols (Bonagura et al., 1985; Dukes-McEwan et al., 2003; Wess et al., 2010; Stephenson et al., 2012; Jeyaraj et al., 2015). The right parasternal window shaved with razor.

The right parasternal window was used for measurements on the left ventricle. Measurements were performed on the frozen M-mode image using the M-mode cursor under the guidance of 2-D echocardiography on the long axis from the right parasternal window (Lombard, 1984; Sahn et al., 1978).

In the case of diastole of the heart, all measurements on the long axis were made when the anterior movement of the posterior wall of the left ventricle was in the posterior position, and in the case of systole, all measurements were made when the anterior movement of the posterior wall of the left ventricle was in the most anterior position (Kayar, 2001).

First, in the echocardiographic examination, the heart of all dogs was subjectively evaluated on the right parasternal long axis four and five chamber view (RPS LAx-4C and RPS LAx-5C, respectively). Secondly, the right parasternal short axis (RPS SAx) papillary muscle level view was used for measuring the end-diastolic thickness of the interventricular septum (IVSd), the end-systolic thickness of the interventricular septum (IVSs), left ventricular end-diastolic inside diameter (LVIDd), left ventricle end-systolic inside diameter (LVIDs), the end-systolic thickness of the left ventricular posterior wall (LVPWs) and the end-diastolic thickness of the left ventricular posterior wall (LVPWd); RPS SAx aortic valve level was used for measuring the left atrial diastolic diameter (LA), the diastolic inner diameter of the aortic root (Ao) and the ratio of the left atrium diameter to the aortic diameter (LA/ Ao). Also, for measuring mitral valve leaflets, RPS Sax mitral valve level was used. Moreover, heart rate (HR), left ventricular ejection fraction (%EF), LV fractional shortening (%FS), stroke volume (SV), cardiac output (CO), the end-diastolic volume (EDV) and the end-systolic volume (ESV) parameters were determined.

IVSd is the distance between the endocardium facing the left ventricular cavity of the interventricular septum at the end of diastole and the endocardium facing the right ventricular cavity, and IVSs is the distance between the

interventricular septum facing the left ventricular cavity and the endocardium facing the left ventricular cavity at the end of systole (Boon, 1998).

LA was measured as the distance from the outer end of the posterior aortic wall to the endocardial border of the left atrial wall at the end of diastole, and Ao was measured as the distance from the anterior wall of the aorta to the posterior wall (Lombard and Spencer 1985; Vollmar 1999).

The ratio of the diameter of the left atrium to the diameter of the aorta (LA/AO) was calculated by dividing the diameter of the left atrium by the diameter of the aorta (Lombard and Spencer, 1985; Boon, 1998).

LVIDd is the distance from the lower point of the intraventricular septum at the level of the chorda tedinea at the end of diastole to the upper point of the posterior wall of the left ventricle, and LVIDs is from the lower point of the upper intraventricular septum posterior wall of the left ventricle at the end of systole. Measured as the distance to the point (Kittenson et al., 1984; Gooding et al., 1986; Haggostrom et al., 1996).

LVPWd was measured as the thickness from the inside of the ventricular posterior wall to the intra-wall pericardium at the end of diastole, and LVPWs was measured as the thickness from the inside of the ventricular posterior wall to the intra-wall pericardium at the end of systole (Boon, 1998; Kayar, 2001).

Heart rate (HR) was measured as the distance between two systoles in the direction of the left ventricular posterior wall facing the left ventricular cavity (Boon, 1998).

Using the values obtained from the left ventricular Ejection Fraction (EF %), EF % was calculated with the formula; EF % = $[(LVIDd)^3 – (LVIDs)^3] /(LVIDd)^3$ x 100 (Koch et al., 1996).

Left ventricular Fractional Shortening (FS%), using the obtained values, was calculated with the formula FS% = [(LVIDd–LVIDs) / LVIDd] x 100 (Bayon et al., 1994).

Using the Teicholz method, stroke volume (SV) is; flow velocity x cross-sectional area. Cardiac output (CO) is the volume of blood ejected from each ventricle per unit time and is calculated with the formula; HR x SV. Body surface area (BSA) was used to calculate the cardiac index. Cardiac index (CI) was calculated with the formula; CO/BSA. End-diastolic volume (EDV) and end-systolic volume (ESV) parameters were also determined by the Teicholz method (Turgut, 2017).

The obtained data were analysed using independent samples T-test to determine the difference and significance between the groups at $p<0.05$ significance level (Akgül, 2005).

RESULTS

The clinical findings and echocardiographic measurement results of the Kangal dogs, which formed the animal material of the study and were divided into two groups (Group I; young group 1-3 years old, Group II; middle-aged group 4-7 years old) according to age ranges, are summarized in Table 1 and 2. In addition, some of the echocardiographic images obtained are presented in figures 1, 2 and 3.

Table 1. Clinical examination results of the dogs used in the study.

Parameter	Group I (n=10)	Group II (n=10)
	Mean ± Standard Deviation	Mean ± Standard Deviation
Pulse Frequency (number of beats /minute)	90.90 ± 16.99	83.44 ± 13.41
Breathing Frequency (r/minute)	20	16
Body Temperature (^{0}C)	38.7	38.5
Capillary Refill Time (seconds)	2	2
Mucous Membranes (rose pink)	+	+
Body Weight (kg)	49.50 ± 6.43	59.37 ± 3.20

Table 2. Echocardiographic findings of young and middle-aged groups.

Parameter	Group 1 (n=10)	Group 2 (n=10)	P
	Mean ± Standard Deviation	Mean ± Standard Deviation	value*
LA (cm)	3.27 ± 0.31	4.07 ± 1.23	0.06
AO (cm)	3.00 ± 0.28	3.29 ± 0.20	0.01
LA/AO	1.10 ± 0.16	1.22 ± 0.31	0.27
IVSd (cm)	1.06 ± 0.11	1.04 ± 0.06	0.64
LVPWd (cm)	1.15 ± 0.17	1.24 ± 0.18	0.27
LVIDd (cm)	4.59 ± 0.50	5.17 ± 1.06	0.14
IVSs (cm)	1.296 ± 0.20	1.30 ± 0.14	0.94
LVPWs (cm)	1.48 ± 0.31	1.48 ± 0.12	0.97
LVIDs (cm)	3.09 ± 0.46	3.54 ± 1.11	0.25
HR (Beat /minute)	90.90 ± 16.99	83.44 ± 13.41	0.30
ESV (ml)	39.52 ± 16.11	60.44 ± 56.20	0.27
EDV (ml)	98.26 ± 25.13	133.26 ± 78.64	0.19
EF (%)	59.93 ± 8.22	57.06 ± 10.43	0.50
SV (ml)	58.73 ± 15.58	72.96 ± 24.13	0.13
CO (L/minute)	5.43 ± 1.97	5.49 ± 1.66	0.94
SI (ml/m^2)	38.52 ± 10.41	38.78 ± 6.84	0.95
FS (%)	32.36 ± 5.89	31.11 ± 6.85	0.66
CI (ml/min/m^2)	3.59 ± 1.42	3.14 ± 1.17	0.48
Body Weight (BW) (kg)	49.50 ± 6.43	59.37 ± 3.20	0.001
Body Surface Area (BSA) (m^2)	1.53 ± 0.15	1.72 ± 0.64	0.001

* P<0,05: Level of Significance

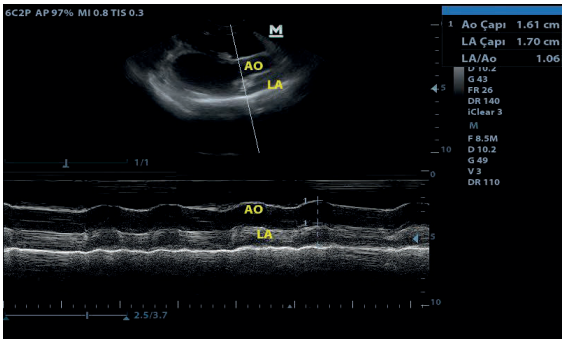

Figure 1. M-mode imaging of the left atrium and aorta.

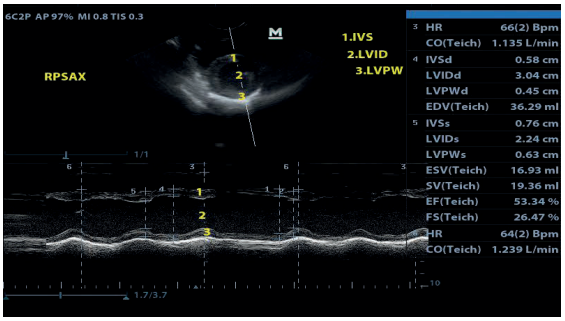

Figure 2. Measured image of intraventricular septum thickness, left ventricular diastolic inner diameter and left ventricular posterior wall thickness.

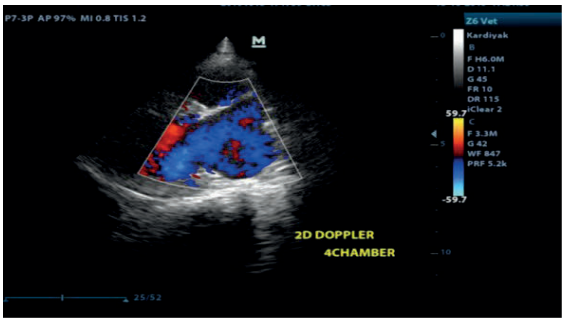

Figure 3. Right Parasternal Long axis 4 Chamber Color Doppler image (normal blood flow).

From the results of the study, although there were differences between groups in left atrium diastolic diameter (LAd), left atrium diameter to aorta diameter ratio (LAd/AOd), the diastolic thickness of the interventricular septum (IVSd), left ventricular end-diastolic posterior wall thickness (LVPWd), left ventricular end-diastolic inner diameter (LVIDd), the end-systolic thickness of the interventricular septum (IVSs), left ventricular end-systolic posterior wall thickness (LVPWs), left ventricular end-systolic inner diameter (LVIDs), pulse frequency (HR), end-systolic volume (ESV), end-diastolic volume (EDV) ejection fraction (%EF), stroke volume (SV), cardiac efficiency, cardiac output (CO), stroke volume index (SI), left ventricular facial contraction force (%FS), and cardiac index (CI) measurements, these differences were not statistically significant (Table 2).

When the mean AOd value of the young group (G I) and that of the old group (G II) was compared, that of G II (3.29 ± 0.20) was higher than G I (3.00 ± 0.28) and was found to be significant at $p< 0.01$.

It was determined that the mean body weights (BW) of the young group (G I) among the Kangal dogs included in the study (49.50 ± 6.43) were lower than those of the middle-aged group (G II), and this was statistically significant at $p<0.01$.

It was found that the mean body surface area (BSA) (1.53 ± 0.159) of the young group (G I) was lower than the mean body surface area (1.72 ± 0.64) of the middle-aged group (G II) and this was statistically significant at $p<0.01$.

An EF value below 55% was accepted as the limit for systolic dysfunction. While systolic dysfunction was detected in 2 dogs (20%) in GROUP 1, systolic dysfunction was observed in 4 dogs (40%) in group 2. One of the dogs in Group 2 with systolic dysfunction also had a high LA/Ao ratio (LA/Ao: 1,93).

DISCUSSION AND CONCLUSION

Primarily formed dilated cardiomyopathies (DCM) are an important cause of heart failure in large dog breeds and cause dilatation of the cardiac chamber, decrease in myocardial contractility and decrease in left ventricular ejection fraction (Bonagura and Lehmkuhl, 2000; O'Grady and Sullivan, 2004; Jeyaraja et al., 2015; Turgut, 2017; Colakoglu et al. 2022).

In our study, we aimed to determine whether there is a tendency to age-related dilated cardiomyopathy in 1-3 and 4-7 years old Kangal Dogs, a breed unique to our country, Turkey, by using 2-D and M-mode echocardiographic techniques.

O'Grady et al., (2008) state that the pre-clinical phase of DCM has a latent period, and at the end of the progressive state, congestive heart failure (CHF) develops with clinical symptoms. It was observed that there was no clinical finding indicating DCM in the Kangal dogs included in the study (Table 2). While no significant finding was found in this phase even with echocardiography, it is stated that during this phase only myocardial cells deteriorate and this can be revealed by histopathological examination (Wiersma et al., 2008).

It has been reported that a decrease in arterial blood pressure, deterioration in pulse quality, arrhythmia, prolongation of capillary filling time and pallor of the mucous membranes may develop in the phases after the preclinical, clinical phase and in very advanced cases of CHF (Bonagura and Lehmkuhl, 2000; Wess et all., 2017). However, none of the pathological findings mentioned was encountered in our study.

In the study conducted, in the examination of the ejection fraction values of the young (G I) and middle-aged groups (G II), it was found that the mean G I value was higher than the mean value of the G II, but this difference was not statistically significant ($p=0.50$) (Table 2). These values are consistent with the values obtained by Kayar, (2001) from the healthy Kangal dogs and the values obtained by Jeyaraja et al., (2015) from the healthy control, Labrador retriever dogs.

Some researchers state that left atrium diastolic diameter and aorta diastolic diameter are positively correlated with body weight and body surface area, and may increase physiologically with age (Lombard, 1984; Vollmar, 1999). In our study, the body weight, body surface area, and aorta inner diameter values of the middle-aged group were found to be higher than the values of the younger group (Table 2). However, since the LAd/AOd ratios in both groups were within normal physiological

limits, the above difference was not considered as a finding for DCM.

Increased left ventricular end-diastolic inside diameter (LVIDd) and left ventricular end-systolic inside diameter (LVIDs) were found in some echocardiographic studies in dogs with DCM (Vollmar, 1999; Stephenson et al., 2012; Jeyaraja et al., 2015). However, in our study, left ventricular end-diastolic inside diameter (LVIDd) and end-systolic inside diameter (LVIDs) values and IVSs values were close to each other in the two groups (GI = 1.296 ± 0.20; GII = 1.30 ± 0.14). Similarly, IVSd were found to be close to each other (GI = 1.06 ± 0.11; G II = 1.04 ± 0.06) and there was no statistical difference.

Although in some studies, low % FS, increased LA/AO ratio, increased ESV and EDV values were observed in dogs with DCM. In our study, despite the individual differences in the parameters of both groups, there was no statistically significant difference between the groups (FS% – p=0.66; LAD/AOD – p=0.27; ESV-p=0.27; EDV – p=0.19) (Stephenson et al., 2012; Jeyaraja et al., 2015, Vollmar, 1999). Nonetheless, our results were found to be consistent with the findings of Kayar's (2001) study on healthy Kangal dogs (Table 2).

Bonagura and Lehmkuhl (2000), determined that with the progressive deterioration of cardiac muscle functions in dogs with DCM, all cardiac chambers, especially the left ventricle and left atrium, will become dilated. In this case, he states that the diameter of the heart circle will increase and the ratio of left ventricular wall thickness to lumen volume will decrease (wall stress) with the progression of dilatation. It was observed that the left ventricle diastolic and end-systolic wall thickness (LVPWd and LVPWs) values obtained in our study are within physiological limits as stated by Kayar, (2001) and there is no condition indicating wall stress in both GI and G II.

After the preclinical stage, during the progression of the cardiac muscle disorders, dilatation begins to form in the heart cavities and parallel, the cardiac index (CI) decreases (Gökçe, 2014; Turgut, 2017). When the cardiac index values of both groups obtained in the study were examined, the mean values of G I (3.59 ± 1.42) were higher than those of G II (3.14 ± 1.17) but statistically insignificant (p = 0.48) (Table 2). Two dogs in Group I had systolic dysfunction, compared to 4 in Group II. All dogs having systolic dysfunction had no CHF signs. It can be thought that this situation must be because of preclinic DCM (O'Grady et al. 2008; Colakoglu and Sahal, 2015).

It is known that the evaluation of left ventricular systolic functions provides information about DCM and thus, parameters such as pulse frequency (HR), stroke volume (SV), cardiac output (CO) are important (Turgut, 2017; Wess, 2021). In our study, the mean HR values measured by echocardiography were G I = 90.90 ± 16.99; G II 83.44 ± 13.41; p=0.30, mean SV values GI = 58.73 ± 15.58; G II 72.96 ± 24.13; p=0.13. These results indicate that no statistical difference was found between the groups. Naturally, there was no statistical difference between the groups (GI = 5.43 ± 1.97; G II = 5.49 ± 1.66) in the mean cardiac output (HR x SV = CO) values calculated with these two values (p=0.94) (Table 2).

The results obtained from the examination of the above parameters determined by echocardiography, it was observed that there was no finding indicating the tendency to DCM developing in the 1-3 years old Kangal dogs, which made up the young group (G I) and also, in the 4-7 years old Kangal dogs, which made up the middle-aged group (G II). However, Jeyaraja et al. (2015) reported that the mean age exposing Labrador retriever dogs to DCM was 6.68 ± 0.49 years. Also, Tidholm et al., (1997) found the mean age of 189 dogs with DCM to be 6.6, while Tidholm and Jonsson, (1996) found the mean age of Newfoundland dogs with DCM to be 5 years, and they stated that DCM was age-related. Moreover, in a study conducted by Wess et al., (2010) on Doberman pinchers, DCM was found to be 13,2% in dogs younger than 4 years of age and 44.1% in dogs older than 8 years of age. The author suggested that the probability of DCM in Doberman pinchers increases with age and that dogs should be checked for DCM annually from the age of 2 years. Similarly, Matli et al., (2021) reported that age indicates poor prognosis in dogs over 8 years of age.

In conclusion, considering that the age of the Kangal dogs which constituted the middle-aged group (G II) of the study were within the age limits stated in the studies above, no myocardiological disorder indicating DCM was detected by echocardiography. In other words, the conclusion that there was no age-related tendency to DCM in the Kangal breed dogs can be reached. However, considering the DCM rate of 6% in the study of Jeyaraja et al., (2015) in the Labrador retriever dogs, it can be said that the echocardiographic evaluation of a larger number of Kangal dogs in the specified age group may yield a more reliable result.

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Conflict of Interest

The authors declare that they have no competing interests.

Authorship contributions

Concept: G.T., S.Y.D., Design: G.T., S.Y.D., Data Collection or Processing: G.T., S.Y.D., Analysis or Interpretation: G.T., S.Y.D., Literature Search: G.T., S.Y.D., Writing: G.T., S.Y.D.

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Ethical Approval

All methods and procedures used in this study comply with the guidelines of the Turkey and EU directive (Directive 2010/63/EU) on the protection of animals used for scientific purposes. This study did not require approval from the authorities or the ethics committee of the institution. However, patient owners were informed and consent was obtained. This study was performed according to The Declaration of Helsinki, Ethical Principles.

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