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Echocardiographic Evidence of Left Ventricular Hypertrophy in Obese Cats

Obez Kedilerde Sol Ventrikül Hipertrofisinin Ekokardiyografik Bulguları

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ABSTRACT

The primary objective of this study was to assess how obesity impacts the occurrence of left ventricular hypertrophy in healthy cats. The researchers evaluated the body condition score of all participating cats before conducting the study. Additionally, they performed various echocardiographic examinations, including transthoracic 2D, M-mode, and Doppler, as well as measuring systolic blood pressure. The results indicated a notable distinction in body condition scores between the non-obese and obese groups ($P < .001$). Moreover, significant differences were observed in terms of systolic blood pressure (SBP) and heart rate between the 2 groups ($P < .001$, $P = .002$). Echocardiographic measurements revealed statistically significant variations in left atrium and left ventricular free wall thickness between non-obese and obese cats ($P < .001$ and $P = .003$). To address obesity, the obese group underwent a low-calorie diet, leading to improvements in echocardiographic parameters and SBP. However, these improvements did not achieve statistical significance. The study findings indicate that obesity can lead to substantial myocardial changes, resulting in left ventricular hypertrophy in cats, which has been previously reported in humans and dogs. Additionally, the study suggests that a diet intervention for obese cats might help prevent diseases like hypertrophic cardiomyopathy.

Keywords: Echocardiography, blood pressure, cardiomyopathy, obesity, cat

ÖZ

Bu çalışmanın temel amacı, sağlıklı kedilerde obezitenin sol ventrikül hipertrofisi oluşumunu nasıl etkilediğini değerlendirmektir. Araştırmacılar, çalışmayı gerçekleştirmeden önce tüm katılımcı kedilerin vücut kondisyon skorunu değerlendirdiler. Ayrıca, transtorasik 2D, M-mode ve Doppler dahil çeşitli ekokardiyografik muayeneler ve sistolik kan basıncı ölçümleri gerçekleştirdiler. Sonuçlar, obez ve obez olmayan gruplar arasında vücut kondisyon skorları arasında önemli bir farklılık olduğunu gösterdi ($P < .001$). Ayrıca, 2 grup arasında sistolik kan basıncı (SKB) ve kalp hızı açısından önemli farklar gözlendi ($P < .001$, $P = .002$). Ekokardiyografik ölçümler, obez ve obez olmayan kediler arasında sol atriyum ve sol ventrikül serbest duvar kalınlığı arasında istatistiksel olarak anlamlı farklılıklar ortaya koydu ($P < .001$ ve $P = .003$). Obeziteyi ele almak için obez grupa, ekokardiyografik parametrelerde ve SKB'de iyileşmelere yol açan düşük kalorili bir diyet uyguladı. Ancak, bu iyileştirmeler istatistiksel bir anlamlılığa ulaşmadı. Çalışma bulguları, obezitenin kedi popülasyonunda sol ventrikül hipertrofisine neden olabilecek önemli miyokard değişikliklerine yol açabileceğini gösteriyor, ki bu daha önce insanlarda ve köpeklerde rapor edilmiştir. Ayrıca, çalışma, bir diyet müdahalesinin obez kedilerde hipertrofik kardiyomiyopati gibi hastalıkları önlemeye yardımcı olabileceğini öne sürmektedir.

Anahtar Kelimeler: Ekokardiyografi, kan basıncı, kardiyomiyopati, obezite, kedi

INTRODUCTION

Obesity stands as the most common nutritional disorder affecting both dogs and cats, leading to significant metabolic and hormonal changes within their bodies.[1,2] In cats, obesity is generally recognized when a cat's weight exceeds 20% of its ideal body weight.[3] Studies estimate that obesity affects around 52% of cats, with some reports indicating even higher rates.[4] Certain risk factors for obesity in cats include male gender, neutering, cross-breeding, middle age, inactivity, confinement indoors, and unrestricted food access.[5,6]

Obesity in cats has been linked to various diseases, such as feline urinary tract disease, diabetes mellitus, hepatic lipidosis, lameness, and skin disorders.[7] In human studies, a correlation has been established between obesity and left ventricular hypertrophy (LVH) with or without left ventricular chamber dilation.[8] Moreover, LVH in obese humans is associated with an increased risk of heart failure.[9]

Left ventricular free wall (LVFW) hypertrophy in cats, characterized by thickening of the left ventricular free wall, is of growing interest in veterinary cardiology, with specific implications for cardiac function. Linked to factors such as obesity, this condition remains a subject of ongoing research. Understanding its prevalence and the potential influence of weight loss interventions on echocardiographic parameters is crucial, offering insights into preventive cardiac care for obese cats.[10]

Obesity-related cardiovascular diseases arise from a complex, multifactorial origin.[11] Human obesity may result in increased cardiac output, putting extra strain on the heart, or an increase in epicardial fat pads, leading to systolic and diastolic dysfunctions.[12,13]

Research on obese rabbits indicates that they have a higher heart rate (HR), increased cardiac output, and diastolic cardiac dysfunction. Additionally, obese rabbits show mild systolic dysfunction.[14] In dogs, "healthy" obesity (without hypertension or prior heart disease) has been linked to increased left ventricular free wall thickness at end-diastole and prolonged isovolumic relaxation time.[15] Obesity in dogs has also been associated with increased left atrial pressure and decreased exercise tolerance.[16]

Given the impact of obesity on cardiomyopathy in humans and dogs, and the rising prevalence of obesity in cats,[17] it becomes crucial to understand the effects of obesity on feline heart health. Therefore, it is hypothesized that obese cats, similar to dogs and humans, will exhibit left ventricular hypertrophy, and dietary interventions might help improve this condition.

The aim of this study is to compare echocardiographic parameters in both obese and non-obese cats. Additionally, we aim to assess the potential positive effects of weight loss interventions on the echocardiographic parameters within the obese cat group.

MATERIALS AND METHODS

The experiment was conducted under the supervision of the Iranian Society for Prevention of Cruelty to Animals, following the ethical codes for studies on laboratory animals in Iran. Approval for the study was granted by the Ethical Review Committee at the Islamic Azad University of Science and Research Branch, Tehran, Iran, (Date: 06.03.2023, Number: IR.IAU.SRB.REC.1401.553).

Forty client-owned adult domestic shorthair cats with no history of diseases were selected from xxx. These cats were on various diets, including commercial, homemade, or combination diets. The inclusion criteria involved a comprehensive assessment, including a standard full physical examination, a within-range complete blood count, and a thorough biochemistry profile that covered parameters such as symmetric dimethylarginine, electrolytes, and triglycerides. Additionally, specific hormone levels, including total thyroxine and insulin-like growth factor 1 (IGF-1), were considered. Urinalysis was conducted, with a particular focus on urine specific gravity. Cats with occult chronic kidney disease were appropriately excluded. Body condition score (BCS) was determined for all animals. In this study, the evaluation of obesity relied on the widely recognized BCS, a 9-point scale with well-documented criteria.[18] Cats with a BCS of 7 or higher were categorized as obese. The BCS was measured by the veterinary doctor during the clinical examination using the BCS scale. This method, known for its precision, provided a clear and accurate classification of obesity among the study's feline subjects.[19]

Exclusion criteria included systolic blood pressure (SBP) greater than or equal to 150 mmHg, chronic kidney disease, abnormal electrocardiography, aggressive behavior requiring strict restraint, ongoing medication, and a cardiac murmur grade of 2/6 or higher with evidence of systolic anterior motion of the mitral valve.[20]

Blood pressure measurements were conducted in a quiet room. The cats were gently and compassionately restrained by their owners, ensuring a comfortable and stress-free examination environment, all without the use of tranquilizer drugs. A Doppler flow detector with a 2.5-cm-wide inflatable cuff was used for the measurements. The cuff was placed around the right antebrachium below the elbow, and the Doppler probe with ultrasound gel was placed over the clipped area. Five readings were obtained from each cat, and the mean of these measurements was used for the analysis.[21]

Echocardiographic Doppler measurements were performed with the cats in lateral recumbency using a 7 MHz phased array transducer and an ultrasound machine (DP-A6, Wuhan Darppon Medical Technology Co., Ltd., China). Hair between the right and left third and sixth intercostal spaces was clipped, and ultrasound gel was applied. Measurements were taken using 2D-guided M-mode and Doppler echocardiography from left parasternal long- and short-axis views. Various dimensions and indices, such as left atrial (LA) and aortic diameter (AO), left ventricular end-systolic (LVIDs) and end-diastolic (LVIDd) dimensions, left ventricular free wall thickness in systole (LVFWs) and diastole (LVFWd), and interventricular septal thickness in systole (IVSs) and diastole (IVSd), were recorded. Fractional shortening (FS) was calculated as an index of systolic function using the given formula. The left atrial to aortic ratio (LA : AO) was also calculated to estimate LV filling pressure. Each measurement used for statistical analysis was the mean of 3 measures from 3 different cardiac cycles. The reference values for echocardiographic parameters in feline subjects were derived from the established guidelines set by Boon.[22]

In the radiographic evaluation, a fixed x-ray system (VIEWORK, Flat Panel Detector for Veterinary, South Korea) was employed to obtain chest x-rays immediately after the echocardiogram, capturing 2 perspectives (lateral and ventrodorsal). The animals were gently positioned without the need for sedatives, adopting a right lateral decubitus posture with extended thoracic limbs to ensure clear imaging and symmetry in the thoracic cavity, and care was taken to slightly extend the animals' heads to prevent tracheal displacement. Vertebral heart size (VHS) was determined by measuring the sum of the apex-base length and maximum craniocaudal width of the heart, perpendicular to each other, and compared to the length of vertebral bodies from the cranial side of the fourth thoracic vertebra. The specific technique used varied depending on the animal's body size and condition, with measurements starting at 60 kV and 4.5 mAs for the lateral view.[23]

After analyzing the data with the owners' assistance, the obese group of cats underwent a 3-month intervention in which they were placed on a low-calorie diet using "Royal Canine—Feline Weight Control Dry Cat Food." This diet is formulated to contain 250 kilocalories of metabolizable energy per cup when fed as recommended on an as-fed basis. Each cat's dietary intake was individually calculated based on their body weight using the product's feeding guidelines.[7] Following the dietary intervention, a follow-up assessment was conducted, including echocardiographic examinations, evaluations of the VHS, and measurements of SBP.

Statistical Analysis

Statistical analysis was performed using the Statistical Package for Social Science Statistics software, version 20.0 (IBM Corp.; Armonk, NY, USA). Data were presented as mean ± SD. The 1-sample Kolmogorov–Smirnov test was used to test the normality of the data, and the independent-samples *t*-test was utilized for data analysis. *P*-values less than .05 were considered statistically significant.

RESULTS

In this study, a total of 40 cats were included, with 20 cats classified as obese and 20 as non-obese. Among the included cats, there were 23 females and 17 males, consisting of 19 intact cats and 21 neutered cats. The mean BCS for cats in both the obese and non-obese groups was determined as 7.35 ± 0.58 (with a weight range between 7.5 kg and 9.8 kg) and 5.00 ± 0.00 (with a weight range between 3.9 kg and 4.8 kg), respectively. Additionally, the average age of cats in the obese group was calculated as 2.82 ± 1.15 years, with an age range of 3 to 4.5 years. In the non-obese group, the mean age was found to be 2.68 ± 1.12 years, with an age range between 2.5 and 4 years. A significant difference in body condition scores between the non-obese and obese groups was observed ($P < .001$).

Echocardiographic data obtained from the cats were found to have a normal distribution based on the 1-sample Kolmogorov–Smirnov test ($P > .24$). Systolic blood pressure was measured as 119.48 ± 8.67 mmHg2 among non-obese cats and 135.60 ± 9.78 mmHg2 among obese cats. The HR was recorded as 180.25 ± 22.1 bpm for non-obese cats and 206.40 ± 27.04 bpm for obese cats. There were significant differences in SBP and HR between the non-obese and obese groups ($P < .001$ and $P = .002$).

Furthermore, statistically significant differences were found in LA and LVFWs between the non-obese and obese cats ($P < .001$, $P = .003$) when assessed from the left parasternal long- and short-axis views.

After the obese group underwent weight loss, improvements were observed in echocardiographic parameters, body condition scores, and SBP compared to the parameters before weight loss (obese group); however, these improvements did not reach statistical significance.

The summarized results of the echocardiographic parameters are presented in Table 1.

DISCUSSION

Obesity in domestic cats is a serious health issue that can increase the risk of developing heart problems such as cardiomyopathy, hypertension, and thromboembolism.[24] Clinical signs of feline obesity encompass weight gain, increased body fat, reduced physical activity, and behavioral changes like decreased grooming and agility. Notably, obesity can contribute to heart problems such as hypertension and heart murmurs. Addressing these signs is crucial for overall well-being and heart health management in obese cats. The effects of obesity on the feline cardiovascular system, as evaluated through echocardiographic assessments, remain inadequately explored, with limited studies on this subject.[19]

Table 1. Echocardiographic Parameters, Heart Rate, Systolic Blood Pressure and Heart Rate of Females, Males, Intact Cats, Neutered Cats, Obese Cats, Non-Obese Cats, and Obese Cats After Weight Loss in the Study Population

Parameters	Females (n = 23)	Males (n = 17)	Intact Cats (n = 19)	Neutered Cats (n = 21)	Obese Cats (n = 20)	Non-Obese Cats (n = 20)	Obese Cats After Weight Loss (n = 20)
LVFWd	4.04 ± 0.92	4.07 ± 0.93	3.86 ± 1.07	4.20 ± 0.75	4.27 ± 0.76[‡]	3.84 ± 1.01	4.01 ± 0.23
LVFWs	7.33 ± 0.91	7.08 ± 1.32	6.91 ± 0.92	7.48 ± 1.18	7.84 ± 0.84[‡]	6.61 ± 0.98[‡]	6.98 ± 0.36
IVSs	6.84 ± 0.99	6.72 ± 1.06	6.61 ± 1.13	6.93 ± 0.91	7.02 ± 0.94	6.56 ± 1.05	6.89 ± 0.85
IVSd	4.00 ± 0.68	4.30 ± 0.70	4.05 ± 0.78	4.20 ± 0.63	4.02 ± 0.71	4.32 ± 0.68	4.019 ± 0.18
LVIDd	15.46 ± 1.93	15.26 ± 1.93	16.11 ± 1.69	14.77 ± 1.90	15.18 ± 2.05	15.57 ± 1.78	15.41 ± 1.55
LVIDs	8.11 ± 2.00	8.21 ± 1.38	8.30 ± 1.89	8.04 ± 1.65	8.30 ± 1.75	8.01 ± 1.77	8.18 ± 1.32
FS	46.80 ± 7.70	45.94 ± 7.27	47.43 ± 6.76	45.62 ± 8.01	47.32 ± 8.32	47.55 ± 6.46	47.38 ± 5.69
LA:AO	1.27 ± 0.13	1.28 ± 0.14	1.29 ± 0.17	1.27 ± 0.10	1.28 ± 0.14	1.27 ± 0.13	1.27 ± 0.15
AO	9.33 ± 1.21	8.89 ± 0.97	9.04 ± 1.33	9.22 ± 0.95	9.41 ± 1.05	8.87 ± 1.15	9.09 ± 1.1
LA	11.83 ± 0.82	11.36 ± 1.05	11.54 ± 0.83	11.70 ± 1.04	12.06 ± 0.86[‡]	11.19 ± 0.83[‡]	12.79 ± 0.74
VHS	7.88 ± 0.34	7.60 ± 0.11	7.62 ± 0.21	7.77 ± 0.42	7.92 ± 0.43	7.58 ± 0.63	7.83 ± 0.39
BCS	6.65 ± 1.22[*]	5.52 ± 1.00[*]	5.50 ± 0.98[†]	6.72 ± 1.20[†]	7.35 ± 0.58[‡]	5.00 ± 0.00[‡]	6.19 ± 0.25
BW	7.31 ± 0.14	6.12 ± 0.21	6.35 ± 0.36	7.49 ± 0.22	8.73 ± 0.71	4.42 ± 0.32	6.98 ± 0.13
SBP	131.23 ± 11.39[*]	122.54 ± 11.86[*]	122.21 ± 11.26[†]	131.90 ± 11.47[†]	135.60 ± 9.78[‡]	119.48 ± 8.67[‡]	126 ± 7.79
HR	201.43 ± 28.91[*]	182.35 ± 22.52[*]	184.94 ± 20.11	200.18 ± 31.53	206.40 ± 27.04[‡]	180.25 ± 22.16[‡]	188.36 ± 18.17

AO, aorta diameter; BCS, body condition scores; BW, body weight; FS, fractional shortening; HR, heart rate; IVSd, interventricular septum in diastole; IVSs, interventricular septum in systole; LA, left atrium diameter; LA : AO, left atrium to aorta ratio; LVIDd, left ventricular internal diameter in diastole; LVIDs, left ventricular internal diameter in systole; LVFWd, left ventricular free wall in diastole; LVFWs, left ventricular free wall in systole; SBP, systolic blood pressure; VHS, vertebral heart scale.
Data are mean ± SD.
*Significantly different between males and females ($P < .05$).
†Significantly different between intact and neutered cats ($P < .05$).
‡Significantly different between obese and non-obese cats ($P < .05$).

The current investigation included animals categorized as overweight and those in optimal body condition, as determined by the assessment of their BCS. These assessment methods, acknowledged for their practicality, simplicity, and reliability, are extensively employed in the evaluation of nutritional status for both cats and dogs, as outlined in the previous studies.[25] The inclusion of BCS in evaluation demonstrated associations with measurements, highlighting a significant distinction between the obese and non-obese groups ($P < .05$). The increase in food intake and obesity in neutered cats may be attributed to changes in their hormonal milieu, including a decrease in sex hormones and an increase in leptin, prolactin, and IGF-1.[26] This emphasizes the importance of conducting a nutritional assessment to gauge the effects of obesity on cardiovascular health. Despite differences in diet, insulin resistance, SBP, dyslipidemia, exercise, obesity, and hereditary conditions influencing cardiac morphology in dogs,[27] these factors did not show statistically significant differences between obese and non-obese cats in this study.[28]

In this study, the neutered animals were found to be older (with a mean age of 3.38 years compared to 1.95 years for intact cats) and had higher body weight (with a mean of 7.49 ± 0.22 kg compared to 6.35 ± 0.36 for intact cats), systolic blood pressure (with a mean of 131 mmHg^2 compared to 122 mmHg^2 for intact cats), BCS (with a mean of 6.72 compared to 5.50 for intact cats), and HR (with a mean of 200 bpm compared to 184 bpm for intact cats). These findings align with previous studies that have also investigated the impact of neutering on obesity in cats.[26] One such study reported that neutered cats gained approximately 30%-40% more weight than intact cats within 1-3 months after the surgery.[29] The study revealed that approximately 10% of cats in the obese group (2.82 ± 1.15) were of a comparable age to those in the non-obese group (2.68 ± 1.12), aligning with findings from the Lund et al[19] study indicating that middle-aged cats are more susceptible to obesity. However, this observed age difference did not reach statistical significance. In this study, gender differences were observed in BCS, HR, and SBP among feline subjects. The higher BCS in female cats (6.65 ± 1.22) compared to males (5.52 ± 1.00) suggested hormonal influences on fat distribution, similar to variations seen in estrogen levels in humans. This hormonal impact likely contributed to the observed BCS differences between male and female felines. The elevated HR in female cats (201.43 ± 28.91) compared to males (182.35 ± 22.52) indicated potential gender-specific disparities in cardiovascular regulation influenced by hormonal fluctuations, particularly estrogen.[30] Additionally, differences in physical activity levels or intrinsic cardiac function may have contributed to the observed variations in HR between male and female cats. The higher SBP in female cats (131.23 ± 11.39) compared to males (122.54 ± 11.86) could be influenced by hemodynamic and vascular factors, including estrogen's vasoconstrictive effects, indicating gender-specific considerations in feline cardiovascular health.[31] The increase in food intake and obesity in neutered cats may be attributed to changes in their hormonal milieu, including a decrease in sex hormones and an increase in leptin, prolactin, and IGF-1.[26]

Studies have established a positive link between cats' body weight and cardiac measurements obtained through echocardiography.[32] In this study, the comparison of LVFWs and LVFWd in non-obese and obese cats suggested the presence of LVFW hypertrophy in obese cats without concurrent thickening of the septum.[33] This finding aligns with previous reports of LVFW hypertrophy in normotensive dogs and humans.[16,34] The FS was similar in both groups, indicating comparable systolic function. The LA : AO ratio was greater in non-obese cats, mainly due to larger AO measurements in obese cats.[15] Left ventricular internal diameter was similar in both groups, suggesting irregular concentric hypertrophy of the heart without chamber dilatation, as observed in obese humans and cats.[9] This phenomenon may be linked to the association with heightened blood pressure values, considering the heart as a target organ for systemic arterial hypertension. Additionally, systemic arterial hypertension can cause damage to target organs, and the persistent increase in blood pressure brings about cardiac changes such as left ventricular hypertrophy and heart failure.[35] Eccentric ventricular hypertrophy, leading to cardiomegaly, can be assessed through chest radiography and VHS measurement.[36] Hence, it can be deduced that feline obesity has the potential to result in cardiovascular dysfunction. In alignment with the results of this investigation, similar studies have also noted an increase in certain morphometric parameters during echocardiographic assessments of obese cats.[31]

While the obese group of cats exhibited higher VHS values in chest radiography compared to the non-obese group, surpassing the reference values (7.5 ± 0.3), this disparity did not reach statistical significance ($P > .05$). Excess pericardial and subcutaneous fat led to an enlarged cardiac silhouette, complicating accurate heart size identification and reducing radiographic image quality.[23] Other studies have reported varying results on the measurements of VHS in obese cats.[36]

In the study, obese cats exhibited higher SBP and HR, both of which fell within the normal reported reference range in healthy cats.[37] Systolic blood pressure was generally below 150 mmHg2, indicating minimal risk of target organ damage following high SBP. Only 1 cat with a BCS of 7 showed an SBP of 157 mmHg2 and was removed from the study due to a mild risk of target organ damage.

This study identified obesity and weight as key factors for LVH in cats, as in previous studies.[38,39] Unlike humans, where LVH is associated with various consequences, including cardiovascular mortality, no longitudinal studies in dogs or cats have observed the outcomes of LVH in obese animals. In contrast to human studies, our research found that the influence of sex on LVH in veterinary medicine, encompassing its effects on SBP, HR, or BCS, did not reach statistical significance. Notably, a study on human patients with hypertrophic cardiomyopathy achieving weight loss reported a decrease in affected LV mass and wall thickness, aligning with the findings in this study's obese cats.[40]

The study's limitations include the consistent BCS of 5 out of 9 for all non-obese cats, confinement to a single breed, and the requirement for future research involving diverse breeds with varying BCSs to enhance generalizability. Additional investigations are essential to delve into the specific roles of sex, age, and dietary composition in LVH among both obese and non-obese cats in a broader population. Moreover, forthcoming studies should examine hyperthyroidism and acromegaly as potential causes of LV hypertrophy in cats. The study also recognizes constraints in addressing factors such as salt sensitivity, blood volume regulation, and endothelial function, highlighting the necessity for further exploration in subsequent research endeavors.

In conclusion, this study revealed a significant association between obesity and LVH in healthy cats, with obese cats exhibiting LVFW hypertrophy. However, no concurrent thickening of the septum was observed in obese cats. The findings underscore the importance of weight management in promoting feline cardiovascular health and highlight the need for further research to explore potential influencing factors and the long-term consequences of LVH in obese cats. Understanding the impact of obesity on feline cardiac health can lead to targeted interventions to prevent and manage obesity-related heart conditions in these companion animals.

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