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Phenotypes and echocardiographic characteristics of a European population of domestic shorthair cats with idiopathic hypertrophic cardiomyopathy

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Summary

111 Domestic Shorthair cats with idiopathic hypertrophic cardiomyopathy were reviewed retrospectively. Two-dimensional echocardiography was used to classify cases in 6 established phenotypes. Hypertrophy was diffuse in 61 % of cats and involved major portions of the ventricular septum and the left ventricular free wall (phenotype D). In the remaining cats, distribution of hypertrophy was more segmental and was identified on the papillary muscles exclusively (phenotype A, 6 %), on the anterior and basal portion of the ventricular septum (phenotype B, 12 %), on the entire septum (phenotype C, 14 %), or on the left ventricular free wall (phenotype E, 7 %). Echocardiographic characteristics and clinical findings were determined for each phenotype to study the correlation between distribution of hypertrophy and clinical implications. 31 cats demonstrated systolic anterior motion of the mitral valve, 75 % of them belonged to phenotype C of hypertrophy. Left ventricular-outflow turbulences were identified more frequently with patterns of hypertrophy involving the ventricular septum (65.5 %), while prevalence of mitral regurgitation was higher when hypertrophy included the papillary muscles (phenotypes A and E, 85 % and 87 %, respectively). Left atrial dilatation occurred more frequently when hypertrophy was diffuse or confined to the left ventricular free wall (61 % of cats with phenotype D or E) rather than to the ventricular septum (31 % of cats with phenotype B or C).

Keywords: Domestic Shorthair cat, hypertrophic cardiomyopathy, phenotype, echocardiography

Phänotypen und echokardiographische Besonderheiten einer Population von Europäischen Kurzhaarkatzen mit idiopathischer hypertrophischer Kardiomyopathiel

Die Daten von 111 Europäischen Kurzhaarkatzen mit idiopathischer hypertrophischer Kardiomyopathie wurden retrospektiv untersucht. Zweidimensionale Echokardiographie-Merkmale dienten der Einteilung der Katzen in 6 etablierte phänotypische Klassen. Die Hypertrophie war global in 61 % der Katzen und betraf grössere Anteile des ventrikulären Septums und der freien Wand des linken Ventrikels (Phänotyp D). Bei den anderen Katzen war die Hypertrophie mehr segmental und betraf nur die Papillarmuskeln (Phänotyp A, 6 %), die basalen Anteile des Septums im Bereich des linksventrikulären Ausflusstrakts (Phänotyp B, 12 %), oder das ganze Septum (Phänotyp C, 14 %), oder die ganze freie linksventrikuläre Wand (Phänotyp E, 7 %). Klinische Befunde und echokardiographische Merkmale wurden aus den Krankengeschichten herausgelesen und miteinander verglichen, um einen Zusammenhang zwischen Verteilung der Hypertrophie und Klinik herzustellen. 31 Katzen zeigten eine systolische Vorwärtsbewegung der Mitralklappe, 75 % dieser Katzen gehörten zum Phänotyp C. Strömungsturbulenzen im linksventrikulären Ausflusstrakt wurden öfters mit Hypertrophien des Septums gesehen (65.5 % der Katzen), während die Prävalenz von Mitralregurgitation bei Hypertrophie der Papillarmuskeln höher war (Phänotyp A, 85 %; Phänotyp E, 87 %). Dilatation des linken Vorhofs wurde häufiger festgestellt, wenn die LV-Hypertrophie global oder auf die freie linksventrikuläre Wand beschränkt war (61 % der Katzen mit Phänotyp D und E), und lag seltener vor, wenn das Septum hypertrophiert war (31 % der Katzen mit Phänotyp B und C).

Schlüsselwörter: Europäische Kurzhaar Katze, hypertrophische Kardiomyopathie, Phänotyp, Echokardiographie

Introduction

Hypertrophic cardiomyopathy (HCM) is the most common feline myocardial disease. Idiopathic (primary) HCM is characterized by concentric hypertrophy of the left ventricle (LV), without dilation, occurring in the absence of systemic disorders. Secondary enlargement of the left atrium (LA) and hyperdynamic ventricular function are frequently observed. Echocardiography is the most reliable diagnostic instrument to observe structural changes and evaluate pathological dysfunctions in cardiomyopathies. Different echographic phenotype patterns, with respect to location and severity of the hypertrophy, have been described in previous studies (Peterson et al., 1993; Fox et al., 1995). Global LV wall thickening was frequently observed. Asymmetric hypertrophy with isolated thickened portions of septum and/or free wall was observed in one study (Fox et al., 1995), while others found a high prevalence of symmetric hypertrophy between septum and free wall (Peterson et al., 1993).

Broad phenotypical variations have been observed among feline breeds. Maine Coon and Ragdoll cats show a more malignant form of disease compared to American Shorthair cats that have a more benign progression (Fox et al., 2001). Familial HCM is responsible for more than 90 % of human cases (Baty et al., 1998) and is inherited as an autosomal dominant tract in humans and Maine Coon cats (Kittleson, 1999; Maron, 2002) and American Shorthair (Meurs et al., 1997). Inheritance is highly suspected in numerous other breeds such as Domestic Shorthair (Baty et al., 2001), Rex, British Shorthair, Ragdoll (Meurs et al., 2001) and mixed-breed cats (Baty et al., 2001; Fuji et al., 2001; Nakagawa et al., 2002). Maine Coon familial HCM is caused by a specific mutation of a sarcomeric protein (Meurs et al., 2005).

Recent studies have described 2-D echocardiography, M-mode parameters, Doppler parameters, and clinical characteristics in cats with HCM (Atkins et al., 1992; Bright et al., 1992, Peterson et al., 1993; Fox et al., 1995; Sisson et al., 1995; Rush et al., 2002). Different breeds were included in these studies but the breed influence on presentation was not examined. The purpose of this study was to determine echocardiographic parameters in a European population of Domestic Shorthair cats with HCM, to examine the morphological variations of hypertrophy and its clinical consequences.

Animals, Material and Methods

Animals

Cats with HCM were examined by the same board certified cardiologist at the Veterinary Hospital Amberger-Philip between 2000 and 2006 in standardized clinical procedures. All cats in this study had been referred to the centre because of a heart murmur and/or suspicion of feline HCM. One or several of the following signs were present initially: Heart murmur and/or respiratory distress, distal paresis or syncope. Complete evaluation included echocardiography and screening for renal insufficiency, systemic hypertension and hyperthyroidism. T4 serum levels were analyzed in cats older than 7 years or if heart rate, behaviour and thyroid palpation were abnormal. Idiopathic HCM was diagnosed if the left ventricular free wall and/or the interventricular septum were ≥ 5.5 mm in diastole (Sisson et al., 1991). 111 Domestic Shorthair cats meeting these criteria were included in the study. Cats with hypertension, hyperthyroidism and other congenital cardiac diseases as well as cats with aortic flow velocity exceeding 2 m/sec were excluded from the investigation, the latter due to the possibility of fixed aortic stenosis or other congenital heart defects. 30 Domestic Shorthair cats from Geneva area (age range from 1.3 to 8.6 years, 12 males and 18 females) served as controls (phenotype F) and underwent identical examination procedures.

Echocardiographic equipment and techniques

Complete 2-D and M mode study was performed following recommended standards (Sahn et al., 1978; Thomas et al., 1984), using an ultrasound system (Esaote Megas GP) with a phased-array transducer of 7.5 MHz. Measurements of the aorta and left atrium were made on the 2-D short axis view of the aortic valve (Rishniw et al., 2000). Several consecutive measurements of myocardial thickness were taken from the long-axis 4-chamber view and verified on the short axis view: left ventricular free wall in diastole (LVFWd) at the chordinae tendinae level, between the papillary muscle and the mitral valve, and interventricular ventricular septum in diastole (IVSd) at the same level. Maximal thickness of isolated hypertrophied segments were also registered. Asymmetric hypertrophy was diagnosed if IVSd to LVFWd ratio was > 1.1 (Liu et al., 1981).

Spectral and colour flow Doppler examinations were also performed using a 7.5–10 MHz probe. Aortic flow velocities were obtained from the right parasternal long axis view (Riesen et al., 2007). Mitral inflow was assessed on the left apical 5-chamber view: Early filling (E-wave), atrial contraction (A-wave) and E/A ratios were obtained and diastolic function evaluated. The presence and magnitude of mitral regurgitation as well as turbulences in the left ventricular outflow tract were also recorded by Color Flow Doppler.

Heart rate and arterial blood pressure were measured with a VET BP 6000 (SDI Sensor Device Inc. Waukesha, WI 53188 USA). Cats with systolic pressure of less than 80 mm Hg or/either mean systemic arterial blood pressure less than 60 mm Hg were classified as hypotensive (Macintire, 2005). Cats with more than 180 mm Hg in systole and 100 mmHg in diastole were considered hypertensive and excluded from the study.

Assessment of left ventricular hypertrophy and morphological patterns

Distribution of hypertrophy was described using two cross-sectional planes following the nomenclature for humans (Lang et al., 2005) and adapting it to the feline cardiac anatomy. This approach was chosen to consider the distribution of coronary blood flow to the myocardium. The first plane divided the heart in a short axis and myocardial segments are named with reference to their circumferential location. The interventricular septum was divided into 3 segments: lateral, anterior and inferior (Fig. 1). The LV-free wall was divided into 3 other segments: lateral, posterior and inferior. The parasternal long axis image was divided into a basal portion from the aorta to the inferior margins of mitral leaflets and an apical portion from below the mitral leaflets.
Based upon previous studies (Maron et al., 1981; Peterson et al., 1993; Adin et al., 2007) and the authors' clinical experience, six phenotypic categories were defined as follows (Fig. 2):
Hyperechogenicity/ hypertrophy of papillary muscles (Phenotype A), hypertrophy confined to basal and anterior portions of septum (Phenotype B), hypertrophy involved entire septum but not the ventricular free wall (Phenotype C), hypertrophy involving important portions of septum and ventricular free wall in equal proportions (Phenotype D), hypertrophy of only the ventricular free wall (Phenotype E). Phenotype F is the normal heart defined according to our control population standards: Left ventricular free wall and septum don't exceed 5.5 mm, left ventricular internal dimensions are maintained between 12 and 18 mm and the LA/AO-ratio (2D) is inferior to 1.2. These values are consistent with reports from others institutions (Fox et al. 1995; Chetboul et al. 2006). Each case was classified according to closest fitting to one of the 6 established phenotypic categories.

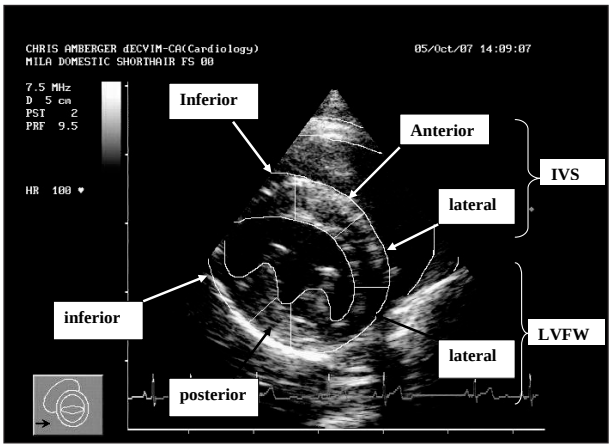


Figure 1: Segments of left ventricular wall visualized by two dimensional echocardiography in the short axis (anteroposterior) plane.

Identification of systolic anterior motion of the mitral valve and left atrial dilatation

The anterior mitral leaflet was observed carefully on 2-D images to determine if systolic anterior motion (SAM) was present and a definitive diagnosis was assessed on M-mode. Turbulence blood flow in the left ventricular outflow tract and across the mitral valve were assessed by Clour and Continuous Wave Doppler (CWD). Presence of mosaic patterns in relation to flow velocities higher than the Nyquist limit (set between 0.65 and 0.83 m/s) indicated turbulences and were confirmed with Pulsed Wave Doppler (PWD). Cats were then classified into 3 categories: presence of aortic turbulences, presence of mitral turbulences, or both. Left atrial dilation (LA/AO > 1.2 on 2-D) was also registered.

Accuracy

All echocardiographic measurements and calculations were made by the same board-certified cardiologist. The intra-observer variability was calculated with standardized methods (Chetboul et al., 2003). The coefficient of variation was between 1 and 8 %.

Statistical analyses

Data are reported as means and standard deviations (for normally distributed data) or medians and ranges (for skewed data) and proportions for categorical data. Descriptive statistics were used to define the population characteristics of the control and the HCM-cats.
Clinical characteristics and echocardiographic measurements of 32 variables were obtained from the 30 control and 111 affected cats. The two populations were then compared for each variable using the t-test or the Wilcoxon-test. The 32 variables were also compared with phenotypes using the Chi-square test for categorical data, and the Kruskal-Wallis test for continuous data. When these comparisons revealed significant differences ($p < 0.05$), sub comparisons were made between individual phenotypes using the Dunn's test with the Bonferroni correction to determine which phenotypes contributed to the significant association. Comparison between 2 variables of small frequencies was made with the Fisher exact test.

Results

Characteristics of patients (Table 1)

Median age of the affected cats was 7 years (range 1–18) and body weight (mean ± SD) was 4.53 ± 0.86 kg. Males were predominant (65 %) in the population. All 111 cats presented with a murmur, 28 with a gallop. 7 % of cats had a first grade murmur, 59 % a second grade and 34 %

a high-grade. Cardiac dyspnoea was observed in 18 of 111 cats and respiratory wheezes heard in 4 cats. We didn't find any significant differences between affected cats and the control population regarding Doppler variables and laboratory findings.

Echocardiographic characteristics of hypertrophy and diastolic function (Table 2)

7 cats had hypertrophy of papillary muscles without other modification (Type A, 6.3 %), 13 had hypertrophy of the

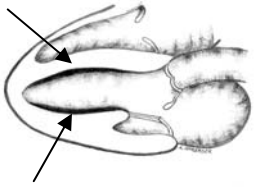
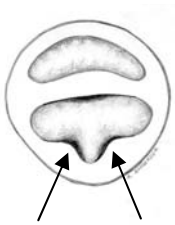
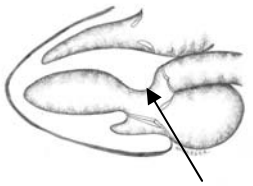
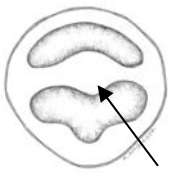
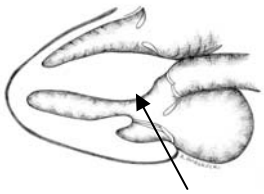
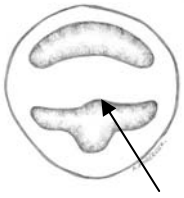
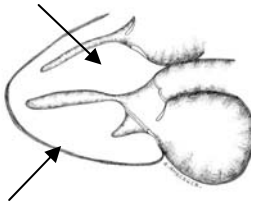
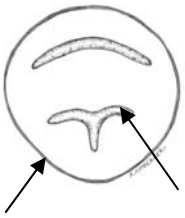
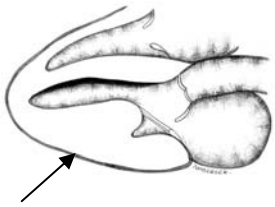
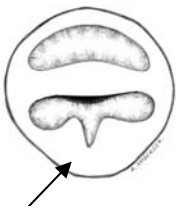
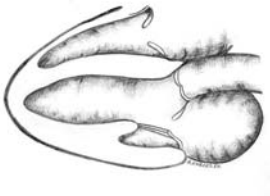
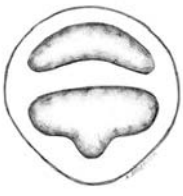
Classification from Amberger et al. (2009)	Long axis	Short axis	Moise et al. (1993)	Maron et al. (1981)
A. Hyperechogenicity/hypertrophy of papillary muscles without others modifications				
B. Hypertrophy of anterior and basal portion of septum			II	I
C. Hypertrophy of hole septum without modification of free wall			II	II
D. Widespread hypertrophy involving septum and free wall			I	III
E. Hypertrophy of the free wall in the basal or apical part.			III	IV
F. Normal heart				

Figure 2: Feline Hypertrophic Cardiomyopathy classification from Moise (1993) and Maron (1981).

Table 1: Physical examination and echocardiographic measurements of 111 Domestic Shorthair cats compared to 30 control cats. Values are given as mean ± SD or median and range in parentheses.

Variables	HCM cats n = 111		Control cats n = 30		P values
Characteristics of population					
Age	7	(1–18)	4	(1–16)	<0.05
Sex (%male)	64.86 %		40 %	<0.05	<0.05
Weight (kg)	4.53	±0.86	3,97	±0.98	<0.05
Clinical characteristics					
Mean arterial blood pressure (mmHg)	90.6	(69–140)	99	(74–119)	<0.05
Systolic blood pressure (mmHg) n = 88	122	(87–197)	131,8	(100–166)	<0.05
HR during examination (bpm)	188	(140–268)	149	128–198	<0.001
Morphologic characteristics (mm)					
Diameter of left atrium (2D)	15.4	(9.6–41)	11.7	(9.1–15.9)	<0.001
Diameter of aorta (2D)	9.93	±0.75	9.79	±0.74	NS
LA /Ao ratio (2D)	1.53	(1–4.1)	1.19	(0.93–1.41)	<0.001
IVSDd (Mmode)	6.2	(7.8–22)	4	(2–5.3)	<0.001
LV FW Dd (Mmode)	6.2	(4.2–16.2)	4.2	(2.8–4.6)	<0.001
LV IDd (Mmode)	14.48	(7.8–22)	14.96	(11.9–19.8)	NS
LV IDs (Mmode)	6.1	(2.1–16.4)	7.6	(5.5–11.6)	<0.001
IVSd/LVFW Dd ratio > 1,1 n = 24	0.98	(0.4–1.88)	0.96	(0.55–1.65)	
Systolic function					
Fractional shortening	0.57	±11	0.47	±6.08	<0.001

LA = Left atrium, LVIDs = Left ventricular internal diameter at end systole, LVIDd = Left ventricular internal diameter at end diastole, LVFWd = Left ventricular free wall thickness at end diastole, IVSd = interventricular septum thickness at end diastole, NS = Non Significant.

basal and anterior part of the septum (Type B, 11.8 %), 16 had generalized septal hypertrophy (Type C, 13.6 %), 67 had diffuse hypertrophy involving the septum and free wall (Type D, 60.9 %) and 8 had hypertrophy confined to the free wall (Type E, 7.2 %). Asymmetric septal hypertrophy was present in 24 cats (22 %) belonging to phenotype B and C. Diastolic dysfunction was present in 30 % of our population of affected cats. Impaired relaxation pattern of left ventricular filling (E/A ratio < 1) was present in 26 cats, most of these belonged to phenotypes D and E. Inversely a restrictive pattern (E/A ratio > 2) was observed in 4 cats with phenotype D and in 2 cats with phenotype E.

Correlation of clinical variables with the distribution of hypertrophy

Significant differences among cats in the 5 phenotypes of hypertrophy were found with regard to age, the ISACHC (international small animal cardiac health council) class, heart rate, packed cell volume and total proteins.

Cats with phenotype A were much younger, and those from phenotype B tended to be the oldest. A majority of phenotype A-cats was asymptomatic (class ISACHC 1), whereas those from the other phenotypes were mostly symptomatic (class ISACHC 2 and 3) and did not differ statistically with regard to severity of cardiac failure. Phenotype B-cats had a significantly lower heart rate than phenotypes C, D and E. No significant differences were found among phenotypes regarding murmur intensity. Total proteins were higher in phenotype D-cats.

The Echo-Doppler findings are shown in Table 3. Aortic turbulences without other concomitant turbulences occurred more frequently in cats with hypertrophy confined to the septum (19 of 29 cats with types B and C of hypertrophy, 65.5 %) than in any other phenotype. Cats with hypertrophy involving both septum and free wall had the highest prevalence of aortic turbulences associated with mitral regurgitation (24 of 67 cats with type D, 36 %). Isolated mitral regurgitation was more frequent when hypertrophy did not involve the septum (6 of 7 cats with type A and 7 of 8 cats with type E). Mitral regurgitation resulted in LA dilation in all cats with phenotype E and in 58 %of cats with phenotype D, but did not occur in cats with phenotype A.

Systolic anterior motion of the mitral valve was detected in 31/111 (28 %) cats. Most of these cats (62 %) had prevalent septal hypertrophy (phenotypes B and C).

Table 2: Physical examination and echocardiographic measurements in 111 Domestic Shorthair cats with Hypertrophic Cardiomyopathy grouped on the basis of phenotypic distribution of hypertrophy. Values are given as mean ± SD or median and range in parentheses. In each row, values with different subscript letters are significantly ($P < 0.05$) different from each others. See legend Table 1.

	Phenotypes					
	A	B	C	D	E	
	n = 7	n = 13	n = 16	n = 67	n = 8	
Variables Characteristics						**P values**
Age	1 (1–4)^{b,d,e}	10 (2–15)^{a}	5 (1–10)	7 (6–10)^{a}	9.5 (3–15)^{a}	<0.05
Sex (%male)	42.85	61.53	62.5	65.67	87.5	NS
Weight (kg)	3.8 (3.1–5.4)	4.4 (4–4.8)	4.25 (3.8–4.5)	4.6 (4.3–4.8)	4.85 (3.8–5.2)	NS
Clinical characteristics						
HR (bpm)	172 ± 27	164 ± 10.5^{c,d}	191 ± 30.62^{b}	193 ± 22.81^{b}	201 ± 31.14^{e}	<0.001
Murmur (%)						
low (grade 1–2)	71.4	69.2	62.5	64.2	75	NS
high (grade 3–4)	28.6	30.6	37.5	35.8	25	NS
MAP (mmhg)	93 (76–114)	90.6 (78–99.3)	90 (80–98)	91.3 (87.3–96.6)	80.6 (78.6–11.3)	NS
Gallop (%)	14.2	15.4	37.5	25.4	25	NS
Abnormal respiratory						NS
pattern (n)	1	1	3	15	2	
ISACHC graduation (%)						
1	57.14	15.4	18.75	1.49	12.5	<0.001
2	42.86	53.84	50	64.18	50	
3	0	30.8	31.2	35.8	37.5	
Morphologic characteristics (mm)						
LA (Mmode)	10.6 (9.2–18)^{d,e}	13.4 (11.3–15.2)	14.55 (10.2–18.7)	15.5 (14.6–17.1)^{a}	21.8 (10.8–26.6)^{a}	<0.001
LA (2D)	11.5 (9.6–17)^{d,e}	14.1 (12–16)	15.4 (10.7–18.2)	16 (15–17.7)^{a}	21.45 (11–29)^{a}	<0.001
LA/Ao (2D)	1.15 (1–1.9)^{d,e}	1.4 (1.27–1.67)	1.47 (1.12–1.74)	1.57 (1.5–1.74)^{a}	2.15 (1.2–2.72)^{a}	<0.05
IVSDd	5.5 (5.4–5.9)^{b,c,d}	6.2 (5.9–6.5)^{a,e}	6.4 (5.9–6.6)^{a,e}	6.2 (6.1–6.4)^{a,e}	5.05 (4.4–5.4)^{b,c,d}	<0.001
LVFW Dd	5.6 (5.2–5.8)^{d,e}	5.5 (5.2–6.2)^{d,e}	5.25 (5.2–5.6)^{d,e}	6.4 (6.2–6.8)^{a,b,c}	6.9 (6–12.4)^{a,b,c}	<0.001
IVSd / LVFW d ratio	1.018^{e}	1.156^{d,e}	1.204^{d,e}	0.949^{b,c,e}	0.71^{a,b,c,d}	<0.001
LVIDd	14.8 (7.8–18)	14.5 (10.8–18.3)	14 (11.6–15.1)	14.6 (13.4–15.2)	13.5 (8.6–17)	NS
LVIDs	6.1 (2.7–7)	6.3 (5.8–8.6)	5.4 (4.8–6.4)	6.1 (5.8–6.5)	5.75 (3.2–9)	NS

Table 3: Distribution of Echo-Doppler abnormalities and ISACHC heart failure classes among patterns of hypertrophy groups, in 111 Domestic Shorthair cats with Hypertrophic Cardiomyopathy.

Patterns of hypertrophy	N = 111 %	%Turbulences			% SAM Observed	Most frequent ISACHC class
		Ao	MR	AO + MR		
A	7	14	86	0	0	1
B	13	85	15	0	46	2
C	16	50	19	31	75	2
D	67	42	22	36	16	2
E	8	13	63	25	25	2

Ao = aortic turbulences, MR = mitral regurgitation, Ao + MR = aortic turbulences and mitral regurgitation together; SAM = systolic anterior motion of the mitral valve. ISACHC = international small animal cardiac health council.

However SAM was also present in phenotypes D and E. In these, mitral regurgitation was present in 7 (22 %), aortic turbulences occurred in 18 (58 %), and both together were present in 6 cats (20 %).

Discussion

Distribution of hypertrophy and breed variations

This investigation demonstrates that our Domestic Shorthair population with HCM presented 5 major patterns of hypertrophy. In 61 % of cats, hypertrophy was diffuse and involved extended portions of ventricular septum and free wall (phenotype D). In the remaining cats (39 %), hypertrophy affected some isolated segments (papillary muscles; basal and anterior part of the septum) or larger portions (entire septum or free wall). Our results are comparable to the findings of Fox et al. (1995) in American DSH: 33 % of cats with segmental hypertrophy and 67 % with diffuse thickening of the left ventricular septum and free wall.
Prevalence of asymmetric septal hypertrophy is highly variable depending upon which cut-off is used for the septal-to-free-wall ratio, increasing from 2.7 % of our cats with cut-off of 1.3, to 22 % of cats with cut-off of 1.1. In human studies, the calculation of septal-to-free-wall ratio from M-mode echocardiograms was found to give an accurate idea of overall LV morphology in only 20 % of patients (Maron, 1984). This is mostly due to the fact that segmental distribution of hypertrophy is seen in up to 48 % of affected human patients, and involved lateral regions only seen in 2D. Our feline cases did not reveal similar segmentation of the lateral regions. The most common isolated segment of hypertrophy, the anterior septum, is easy to measure using M-mode. Therefore, the septal-to-free-wall ratio can be a useful screening tool to distinguish asymmetric from symmetric septal hypertrophy in cats. The cut-off ratio of 1.1 was found to be reliable in our echocardiographic study. Why hypertrophy of lateral segments occurs more frequently in humans than in cats is difficult to explain. The human heart is located in a thorax with relatively large transversal dimensions, whereas the feline heart has more possibility to grow in the longitudinal axis.
Papillary muscle hypertrophy is a consistent finding in human HCM (Shapiro et al., 1983) and in Maine Coon cats (Kittleson et al., 1999). 6 % of our 111 DSH had papillary muscle hypertrophy without other involved regions, and these cats were significantly younger. This finding reinforces the hypothesis that papillary muscle hypertrophy could be an early indicator of HCM. Objective papillary muscle measurement tools for the detection of HCM were only recently proposed by others (Adin et al., 2007).
Unexpectedly, the cats with segmental hypertrophy of the septum were older than those with diffuse septal thickening. Therefore, these two phenotypes could be unrelated, distinct morphologic presentations of the disease. However, this phenotype B could also be mistaken with the normal geriatric morphology of the feline heart. Only histological analysis can differentiate a true HCM segment from hypertrophy secondary to chronic myocardial alterations. In Maine Coon cats, hypertrophy is more commonly confined to the LV free wall than to the septum (Kittleson et al., 1999). In our DSH cats, the interventricular septum was the most consistently hypertrophied region (96 of 111 cats).

Clinical implications

Cats with severe ventricular hypertrophy were found to have a bad prognosis in several previous studies (Spirito et al., 1990; Fox et al., 1995). In fact, cats with the more extensive distribution of LV hypertrophy (phenotype D) were almost all symptomatic (98 percent) at the time of clinical examination, even if belonging to a middle-age category. These cats reached a symptomatic phase relatively early in life. Moreover, LA dimensions tended to be higher in these cats and in cats with left free wall hypertrophy (phenotype E). LA enlargement has been associated with an unfavourable prognosis (Rush et al., 1990; Fox et al., 1995). Abnormal diastolic filling patterns were also more commonly seen in these latter two phenotypes. Total proteins were higher in group D too, which could be partially explained with the highest PCV registered for this phenotype, or could be due to secondary inflammation syndrome due to cardiac failure. Further studies are needed to correlate phenotypes D and E with a shorter survival time, however these patterns of hypertrophy seem to be associated with a poor prognosis.
A higher prevalence of heart failure was expected in cats with widespread hypertrophy as occurring in humans (Maron; 1981). However, no significant differences of heart failure incidence was found among phenotypes B through E, except for phenotype A-cats which were predominantly asymptomatic. This could be partially explained by the high percentage of cases referred because of clinical signs of heart failure. Secondly, our study was limited by the small number of cats existing in group A, B, C and E and could not reach statistical significance in many occasions.

Doppler findings, SAM and heart murmurs

All 111 cats had heart murmurs of different intensities. We observed aortic turbulences and/or mitral regurgitation in every one. The 31 cats with systolic anterior motion had mostly aortic turbulences, which could explain the murmur. In the 80 remaining cats, the murmur could have been generated by either the mitral regurgitation or the turbulences in the left ventricular outflow tract (LVOT).
The majority of cats with SAM had an asymmetric septal hypertrophy (phenotypes B and C). SAM was usually

thought to be due to hypertrophy of the basal part of the septum that, narrowing the outflow tract, creates a Venturi effect and a forward suction of the mitral leaflet towards the septum. This results in an obstruction of the LVOT. In our study, we didn't detect any LVOT velocity higher than 2 m/s. However, we believe that the right parasternal approach for the aortic flow velocity determination might have created an angle problem between the ultrasound beam and the direction of aortic flow and possible underestimation of peak velocities.
SAM was also present in others patterns of hypertrophy without septal involvement (phenotypes D and E). The geometrical alteration of the papillary-mitral apparatus with HCM changes the balance of forces acting on the mitral leaflets, and creates a SAM by dragging the displaced mitral leaflets into the LVOT (Jiang et al, 1987). Hypertrophy of the entire septum (phenotype C) actually combines the effects of outflow tract narrowing and papillary muscle distortion, resulting in a significantly higher prevalence of SAM than in cats with a septal bulge only (phenotype B). This mechanism also explains why phenotypes of hypertrophy involving the papillary muscles (phenotypes A, D and E) experienced more frequent mitral regurgitation by altering the effectiveness of cordal support.

Our present report of HCM in DSH-cats, emphasizes the diffuse distribution of hypertrophy involving most frequently the interventricular septum. Our data also highlight the heterogeneity of the disease and provide some useful tools for understanding its characteristics in this breed. Future studies of the natural progression of HCM are needed to provide better information about the clinical course and prognosis of each phenotype.

Particularités phénotypiques et échocardiographiques d'une population de chats européens à poil court souffrant de cardiomyopathie idiopathique hypertrophique

Les données de 111 chats européens à poil court souffrant de cardiomyopathie idiopathique hypertrophique ont été examinés rétrospectivement. Des critères échographiques bi-dimensionnels ont servi à classer les chats en 6 groupes phénotypiques établis. L'hypertrophie était globale chez 61 % des chats et concernait des parties importantes du septum ventriculaire et de la paroi libre du ventricule gauche (phénotype D). Chez les autres chats, l'hypertrophie était plus segmentaire et touchait uniquement les muscles papillaires (phénotype A, 6 %) la base du septum dans la région de la zone d'éjection du ventricule gauche (phénotype B, 12 %) , le septum en entier (phénotype C,14 %) ou la seule paroi ventriculaire gauche libre (phénotype E, 7 %). Les symptômes cliniques et les constatations échocardiographiques ont été extraits des dossiers et comparés entre eux pour rechercher une relation entre la répartition de l'hypertrophie et la clinique. 31 chats présentaient un prolapsus systolique de la valvule mitrale, 75 % d'entre eux appartenaient au phénotype C. Des turbulences circulatoires dans la zone d'éjection du ventricule gauche étaient relevés plus souvent dans les cas d'hypertrophie du septum (65.5 % des chats) alors que la prévalence d'une régurgitation mitrale était plus élevée lors d'hypertrophie des muscles papillaires (phénotype A 85 %, phénotype E 87 %). Une dilatation de l'oreillette gauche était

Particolarità del fenotipo e dell'ecocardiografia di una popolazione di gatti europei a pelo corto affetti da cardiomiopatia ipertrofica idiopatica

Sono stati analizzati in modo retrospettivo i dati raccolti su 111 gatti europei a pelo corto affetti da cardiomiopatia ipertrofica idiopatica. Le particolarità ecocardiografiche bidimensionali sono state utilizzate per la suddivisione dei gatti in 6 classi con fenotipo conosciuto. L'ipertrofia era totale nel 61 % dei gatti ed era situata in gran parte nel setto ventricolare e nella parete libera del ventricolo sinistro (fenotipo D). In altri gatti, l'ipertrofia era più segmentata e riguardava solo il muscolo papillare (fenotipo A, 6 %), la porzione basale del setto nell'ambito del tratto di efflusso del ventricolo sinistro (fenotipo B, 12 %), oppure tutto il setto (fenotipo C, 14 %), oppure tutta la parete libera del ventricolo sinistro (fenotipo E, 7 %). Le caratteristiche dei reperti clinici ed ecocardiografici sono state estratte dalla storia sanitaria dell'animale e paragonate mutualmente, alfine di trovare un legame tra distribuzione dell'ipertrofia e stato clinico. 31 gatti presentavano un movimento in avanti della valvola mitrale, il 75 % di questi gatti apparteneva al fenotipo C. Si sono rilevate più spesso turbolenze del flusso nel tratto di efflusso del ventricolo sinistro (65.5 % dei gatti) mentre era più alta la prevalenza della rigurgitazione mitrale nell'ipertrofia del muscolo papillare (fenotipo A, 85 %; fenotipo E, 87 %). Si è costatata spesso la dilatazione dell'atrio sinistro, nel caso vi fosse un'ipertrofia del ventricolo sinistro completa o limitata alla parte

constatée plus fréquemment lors d'une hypertrophie globale du ventricule gauche ou de la paroi libre de ce ventricule (61 % des chats avec le phénotype D et E) et elle était plus rare lorsque le septum était hypertrophié (31 % des chats avec les phénotypes B et C).

libera della parete ventricolare sinistra (61 % dei gatti con fenotipo D ed E) e più rara se il setto era ipertrofizzato (31 % dei gatti con fenotipo B e C).

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