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Evaluation of Plasma D-dimer Concentration in Cats with Hypertrophic Cardiomyopathy

Tae-Young Kim, Suk-Hee Han, Ran Choi and Changbaig Hyun[1]

Section of Small Animal Internal Medicine, College of Veterinary Medicine, Kangwon National University, Chuncheon 201-100, Korea

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Abstract : Arterial thromboembolism (ATE) is a common and fatal complication of hypertrophic cardiomyopathy (HCM) in cats. Therefore in this study, we evaluated the hypercoagulability (using plasma concentration of D-dimer) in HCM cats with different stage of heart failure and left atrial enlargement and also investigated the any correlation with echocardiographic indices (including left free wall thickness at diastole, interventricular septal thickness at diastole, LA to Ao ratio, heart failure stage, existence of systolic anterior motion of mitral valve). The median plasma D-dimer concentration in this study population was 0.51 ± 0.70 (range 0 to 2.50) ug/mL in the control group, 1.47 ± 1.29 (range 0.3 to 5.79) ug/mL in the HCM group, 1.48 ± 1.65 (range 0.3 to 5.79) ug/mL in the ISACHC I group, 1.62 ± 0.4 (range 1.31 to 2.07) ug/mL in the ISACHC II group, 1.36 ± 0.91 (range 0.3 to 2.31) ug/mL in the ISACHC III group, 1.90 ± 1.60 (range 0.3 to 5.79) ug/mL in the cat with LA dilation, 1.72 ± 0.72 (range 0.6 to 2.31) ug/mL in cats with SEC-T, 1.19 ± 0.70 (range 0.3 to 2.31) ug/mL in the cats with SAM, and 1.63 ± 0.80 (range 0.6 to 2.31) ug/mL in the cats with ATE. Our study found the median and mean concentration of plasma D-dimer was higher in cat with HCM, ATE, SECT and SAM and clearly provides evidence of hypercoagulability in cats with HCM, although the severity was not correlated to the dilation of LA and the presence of heart failure. This is the first study evaluating the hypercoagulability in cats with HCM in Korea.

Key words **:** arterial thromboembolism, hypertrophic cardiomyopathy, hypercoagulability, D-dimer, heart failure.

Introduction

Feline hypertrophic cardiomyopathy (HCM) is characterized by an abnormally thickened ventricle causing marked diastolic dysfunction, left atrial dilation, blood stasis, and eventual heart failure. Human study found that congestive heart failure could be a major cause of hypercoagulable state predisposing to thrombosis and thromboembolic events (9). Arterial thromboembolism (ATE) is a common and fatal complication of HCM in cats (8,12,16). The blood clots (Thrombi) are usually lodged in the descending aortic bifurcation causing partially or completely restricting blood flow to the hind limbs and inducing signs of severe pain, cold extremities, and caudal paresis. Furthermore, the thrombi also can be lodged in important blood vessels including the mesentery, kidneys, brain, and lungs and can induce very fatal problems (e.g., acute renal failure; 10,11,16). Therefore, the fatality is very high ranging 61-100% and many survivors of the initial episode frequently suffer rethrombosis (8,16). The cause of ATE may be fragmentation or dislodging of thrombi from left atrial (LA) or left atrial appendage (LAA). Formation of such thrombi is contributed by three factors of Virchow's Triad (endothelial injury, blood flow abnormalities, and hypercoagulability) in cats with HCM (13).

In human, the LA diameter in HCM was significantly correlated with concentrations of thrombin-antithrombin complex (TAT; 6). In addition, two veterinary studies found correlation with laboratory test results indicating hypercoagulable state in cats with HCM (2,18). Although the LA size is considered to be an indicator of risk of arterial thrombosis (8), no significant correlation between coagulation markers and LA size was found in two studies (2,18). However, one recent study found that plasma D-dimer concentration in cats with LA enlargement found to be higher, especially after signs of heart failure were developed. Therefore, there is a conflict finding in correlation of plasma d-dimer concentration with severity of HCM in cats. D-dimer and fibrin degradation product (FDP) are indicators of active fibrinolysis and have been used as markers of hypercoagulability (e.g. disseminated intravascular coagulation, ATE). D-dimer is a product of the terminal digestion of cross-linked fibrin by plasmin and is considered to be a sensitive marker of thrombosis (2). Therefore in this study, we evaluated the plasma concentration of d-dimer in HCM cats with different stage of heart failure and LA diameter and also investigated the any correlation with echocardiographic indices (including left free wall thickness at diastole, interventricular septal thickness at diastole, LA to Ao ratio, heart failure stage, existence of systolic anterior motion of mitral valve[SAM]).

[1]Corresponding author.
E-mail : hyun5188@kangwon.ac.kr

Materials and Methods

Case selection

Eighteen hypertrophic cardiomyopathy (HCM) cats with various stage of heart failure and 18 clinically healthy cats were enrolled in this study. Selection was based on results from physical examination, diagnostic imaging studies (including echocardiographic evaluation), serum biochemistry and complete cell blood counts. Any cats showing abnormalities in these diagnostic studies were not included in healthy control group. The status and severity of cardiomyopathy in HCM cats were confirmed by echocardiography. Some cats had received or were currently being treated with drugs (e.g., diuretics, beta-blockers) prior to their enrollment in the study. Hyperthyroid cats with secondary myocardial hypertrophy were excluded. Total serum T4 concentration was measured in cats older than 6 years of age in order to exclude possible cases of occult hyperthyroidism. Animals were cared for according to guidelines provided by the Kangwon National University Council on Animal Welfare.

Echocardiographic evaluation

Echocardiographic examinations were conducted in accordance with recommended standards for cats. M-mode, Doppler, and 2-dimensional echocardiography were performed in left and right lateral recumbency. Cats were determined to have HCM based on measurements taken from standard echocardiographic views as described in elsewhere (15). M-mode echocardiography was used to measure LA diameter and proximal aortic (Ao) diameter. These measurements were used to determine the LA to proximal Ao diameter ratio (LA:Ao). The thickness of interventricular septum and left ventricular free wall at diastole were measured at the image plane from the right parasternal short axis of left ventricular papillary muscle.

Blood collection and D-dimer assay

Blood samples were obtained by jugular venipuncture using a 23-gauge butterfly needle, attached to a connecting tube with a multiple sample Luer adapter. Precautions were taken to avoid excessive tissue trauma and coagulation activation during blood collection. Blood was directly transferred into appropriate evacuated collection tubes containing 3.2% buffered sodium citrate in a ratio of 9 parts blood to 1 part anticoagulant. Citrated plasma was then separated within 45 minutes of collection by centrifugation at 3000 g for 10 minutes and frozen at –80ºC in 500uL aliquots until analyzed. All assays were performed on citrated plasma, in duplicate and according to the manufacturer's recommendations. D-dimer concentration was measured with an automated coagulation analyzer using an immunoturbidimetric method (STA-Liatest D-DI, Diagnostica Stago). Briefly, this method uses a change in light absorption to detect antigen-antibody reactions resulting in particle agglutination when plasma containing d-dimer is added to a suspension of latex beads coated with mouse monoclonal antibodies against human d-dimer. D-dimer plasma concentrations were reported as ug/mL of fibrinogen equivalent.

Data analysis

A 1-stage nested design for variation partition was used to estimate intra-assay variability of d-dimer in healthy cats and cats with HCM. Nonparametric tests were used after data distribution was assessed for normality using a Kolmogorov-Smirnov test. A Mann-Whitney U test was used to compare d-dimer results between healthy cats and cats with HCM and to compare LA and LA:Ao between healthy cats and cats with HCM. Relationships between echocardiographic (LA and LA:Ao) and d-dimer findings were tested with a Spearman rank correlation test. $P < 0.05$ was considered significant.

Results

The breeds of control group and HCM cats enrolled in this study is summarised in Table 1 and 2, and listed as follows: Cats in control group were Domestic short-haired (61%), Turkish Angora (17%), Persian (17%), Bengal(5%), while that of HCM cats were Scottish fold (66%), Persian (17%), Domestic short-haired (17%). The mean body weight was 5.07 ± 0.82 kg in control group whereas 4.23 ± 1.10 kg in HCM group. The mean age was 3.3 ± 1.58 years in control group whereas 6.17 ± 3.26 years in HCM group (Table 1, 2).

Among HCM cats, 5 cats had marked LA dilation (LA:Ao > 2:1), 5 cats had echocardiographic evidence of SEC-T (spontaneous echocardiographic contrast, atrial thrombi or both), 12 cats had SAM and 4 cats had ATE. Most cardiomyopathic cats had enlarged left atria (25% had severe enlargement [LA :Ao > 2.0]). Of the LA dilation cats, 100% (5/5) presented with CHF. All SEC-T and SAM cats were presented with CHF. All ATE cats presented with clinical signs of thromboembolism (Table 2).

The median plasma d-dimer concentration in this study population was 0.51 ± 0.70 (range 0 to 2.50) ug/mL in the control group, 1.47 ± 1.29 (range 0.3 to 5.79) ug/mL in the HCM group, 1.48 ± 1.65 (range 0.3 to 5.79) ug/mL in the ISACHC I group, 1.62 ± 0.4 (range 1.31 to 2.07) ug/mL in the ISACHC II group, 1.36 ± 0.91 (range 0.3 to 2.31) ug/mL in the ISACHC III group, 1.90 ± 1.60 (range 0.3 to 5.79) ug/mL in the cat with LA dilation, 1.72 ± 0.72 (range 0.6 to 2.31) ug/mL in cats with SEC-T, 1.19 ± 0.70 (range 0.3 to 2.31) ug/mL in the cats with SAM, and 1.63 ± 0.80 (range 0.6 to 2.31) ug/mL in the cats with ATE (Table 1, 2).

The median plasma d-dimer concentration in control dogs were approximately 2.88 times lower than dogs with HCM. A statistically significant difference was found between the control and the HCM groups ($P < 0.01$). Using a univariate analysis, plasma d-dimer concentration were not found to be directly correlated with LA:Ao ratio, IVSD, LVFWD and others. Plasma d-dimer concentrations were more closely related to the existence of SAM, ATE and SEC-T.

Table 1. Plasma d-dimer concentration and echocardiographic indices in healthy control cats

ID	Breed	Age	Sex	IVSd (mm)	LVFWd (mm)	LA:Ao	D-dimer (ug/mL)
1	DSH	5	SF	3.2	4.7	1.68	0.31
2	ASH	4	NM	5.3	5.6	1.35	1.77
3	DSH	3	SF	4.1	4.1	1.47	2.5
4	DSH	1.3	NM	4.0	4.3	1.50	0
5	DSH	1.3	NM	4.7	4.2	1.56	0.34
6	DSH	1.3	F	3.9	4.1	1.44	0.39
7	DSH	1.3	NM	3.9	4.0	1.49	0.22
8	DSH	4	F	3.4	3.6	1.61	0.29
9	DSH	0.7	NM	4.9	5.7	1.28	0
10	Turkish angora	3	M	4.3	4.5	1.42	0.21
11	Persian	3	M	4.1	5	1.38	0.24
12	Bengal	2.5	M	5	5.8	1.19	1.58
13	Turkish angora	5	SF	5.1	4.3	1.1	0.49
14	Persian	5	F	3.8	3.4	1.25	0.2
15	Turkish angora	5	NM	4.1	4	1.28	0.13
16	Persian	5	M	2.7	5.5	1.34	0.4
17	DSH	5.8	SF	4.2	4.0	1.63	0.37
18	DSH	5.5	NM	4	4.2	1.39	0

IVSd: interventricular septal thickness at diastole, LVFWd: left ventricular free wall thickness at diastole, LA:Ao: LA to Ao ratio, D-dimer: 0.00-0.30 ug/mL (reference range) NM: neutered male, SF: spayed female, F: female, M: male

Table 2. Plasma d-dimer concentration and echocardiographic indices in cats with hypertrophic cardiomyopathy

ID	Breed	Age	Sex	IVSd (mm)	LVFWd (mm)	LA:Ao	SAM	SECT	ISACHC Stage	D-dimer (ug/mL)
1	Scottish Fold	11	NM	10.2	7.8	1.48	○	-	2	1.31
2	Scottish Fold	8	SF	5.4	6.0	1.15	○	-	1	0.69
3	Scottish Fold	8	SF	6.5	5.5	0.79	○	-	1	0.57
4	Scottish Fold	9	SF	3.5	3.3	1.18	-	-	1	1.2
5	Scottish Fold	8	F	6.9	6.5	1.1	-	-	1	0.79
6	Scottish Fold	7	SF	6.1	5.9	1.38	○	-	1	0.88
7	Scottish Fold	11	SF	7.5	7.6	1.12	○	-	1b	0.55
8	Scottish Fold	7	F	4.1	4.1	1.64	-	-	1	5.79
9	Scottish Fold	7	SF	6.8	4.5	1.5	-	-	1	2.57
10	DSH	7	NM	7.7	13.5	1.88	-	-	2	1.47
11	Scottish Fold	1.5	NM	8.3	9.3	3	○	○	3	1.4
12	Scottish Fold	1.5	NM	8.5	9.8	2	○	○	3	0.6
13	DSH	3.5	NM	7.2	11.6	2.75	-	-	3	0.3
14	Persian	2	F	8	6.1	1.55	○	-	1b	0.3
15	Persian	4.2	SF	9.8	8.3	1.58	○	○	2	2.07
16	Scottish Fold	1.6	NM	8.5	9.8	2.08	○	○	3	2.31
17	DSH	3.7	F	7.65	7.31	2.59	○	○	3	2.2
18	Persian	10	NM	9	4.9	1.4	○	-	1b	1.45

IVSd: interventricular septal thickness at diastole, LVFWd: left ventricular free wall thickness at diastole, LA:Ao: LA to Ao ratio, SAM: systolic anterior motion of mitral valve, SECT: spontaneous echocardiographic contrast, atrial thrombi or both, ISACHC: international small animal cardiac health council, D-dimer: 0.00-0.30 ug/mL (reference range), NM: neutered male, SF: spayed female, F: female, M: male
[○]: present, [-]: absent

Discussion

D-dimer is a byproduct from the pathway of thrombin-mediated cleavage of fibrinogen and degradation of fibrin clot and widely used for detecting active thrombosis process including pulmonary thromboembolism and disseminated intravascular coagulation (DIC) in dogs (20). Although two earlier feline studies (4,7) found only a few ATE cats had high plasma D-dimer concentration. However, recent feline study (18) found that D-dimer concentration was high in 50% of ATE cats. Although these differences could be responsible for the use of different reagents and assay methods, previous studies could not detect the actual elevation of d-dimer concentration in their study cohorts (19,20), because the cats could rapidly clear D-dimer. In addition, the thrombus lodged in the aorta may not be large enough to increase systemic D-dimer concentrations in the cats with ATE (3). However, our study found the median and mean concentration of plasma D-dimer was higher in cat with HCM, ATE, SECT and SAM. Our study clearly provides evidence of hypercoagulability in cats with HCM, although the severity was not correlated to the dilation of LA and the presence of heart failure. Our study results are quite close to the findings from Stokol *et al* (18). However one other study found that d-dimer was increased only in cats with CHF (7).

D-dimer measurement is increasingly available in veterinary medicine and is included in coagulation panels in numerous clinical pathology laboratories (1). Its clinical use in cats with HCM remains to be demonstrated. Various assays for D-dimer detection are commercially available, and only a few have been tested and validated for clinical use in veterinary medicine (5,17,19). Human D-dimer assays have been validated as reliable and accurate in dogs (5). Immunoturbidimetry D-dimer assay, the Liatest D-Di can be recommended for commercial veterinary laboratories using an automated coagulation analyzer (1). The sensitivity and specificity of the immunoturbidimetry D-dimer assay were 65%, 97% (5).

In our HCM cat population, hypercoagulable cats were no more likely to have severe LAE (LA:Ao > 2.0) than nonhypercoagulable cats. Even some cats with left atrial enlargement (LAE) had LA:Ao > 2.0, but not were hypercoagulable. Our finding indicated that the LAE alone may not be enough to induce hypercoagulable state in cats with HCM and other contributing factors may be also involved. Possible contributing factors are i) decreased atrial contractibility, ii) increased blood stasis/ decreased blood flow, iii) enhanced erythrocyte aggregability, and iv) hyperviscosity of blood (6,14,15,21).

Spontaneous echo contrast can be observed in the left atrium and the LA appendage of cats with HCM and is attributed to the formation of RBC aggregates because of low shear conditions and blood stasis. This phenomenon is considered to be a hallmark of prothrombotic hypercoagulable states in both human and feline patients. One study found that SEC-T cats were similarly hypercoagulable in the absence of systemic thromboembolism, suggesting systemic hypercoagulability is a contributing factor, although the systemic hypercoagulability is unlikely to be the primary force initiating thrombus formation (18). Similar to this study, we also found all SEC-T cats were hypercoagulable.

There are several limitations of our study. Firstly, relatively low numbers of cats in each disease group were enrolled so that decreases statistical power and increases risk of a type II error. Secondly, we could not exclude cats with medication, although none of the drugs used are known to impact the plasma D-dimer assay. Thirdly, we only evaluated D-dimer concentration for evaluating systemic hypercoagulability in cats. Future study should be included more markers indicating hypercoagulability such as fibrinogen, FVIII:C, antithrombin (AT), thrombin-antithrombin complex (TAT). Finally, there will be a chance of SEC or atrial thrombi in some cats with HCM and occult HCM in healthy control cats, even though we did thorough echocardiography for all cats enrolled in this study.

In conclusion, we found evidence of hypercoagulability in approximately 70% of the cats with more severe forms of cardiomyopathy (e.g., those with SEC or ATE). Prospective studies are needed to evaluate whether the echocardiographic findings of SEC or left atrial flow dynamics and laboratory evidence of hypercoagulability are indeed predictive of future ATE or are useful for guiding prophylactic therapies. As in previous studies, our data suggest that the pathogenesis of ATE is multifactorial and therefore treatment and prevention of this syndrome might involve drug combinations modulating hemostasis (e.g., platelets and coagulation factors) and inflammatory pathways.

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비대성 심근증이 있는 고양이에서 혈장 D-dimer 농도의 평가

김태영 · 한숙희 · 최란 · 현창백[1]

강원대학교 수의학과 소동물 내과교실

요 약 : 동맥 혈전색전증은 비대성 심근증이 있는 고양이에서의 흔하고 치명적인 합병증이다. 그러므로 금번 연구에서 비대성 심근증의 고양이에서 좌심방 확장 및 심부전의 중등도에 따른 응고 항진 (혈장 D-dimer의 농도 측정을 사용)을 평가하였고, 심장초음파 지표들과의 상관성을 조사 하였다 (이완기 좌심실 자유벽 두께, 이완기 중격의 두께, 좌심실 대 대동맥의 비율, 심부전 단계, 이첨판막의 수축기 전방운동의 존재). 이번 연구 집단에서 평균 혈장 D-dimer 농도는 대조군에서 0.51 ± 0.70 (range 0 to 2.50) ug/mL, HCM 고양이 집단에서 1.47 ± 1.29 (range 0.3 to 5.79) ug/mL, ISACHC I군에서 1.48 ± 1.65 (range 0.3 to 5.79) ug/mL, ISACHC II군에서 1.62 ± 0.4 (range 1.31 to 2.07) ug/mL, ISACHC III군에서 1.36 ± 0.91 (range 0.3 to 2.31) 였으며, 좌심방확장이 있는 고양이에서 1.90 ± 1.60 (range 0.3 to 5.79) ug/mL, SEC-T이 있는 고양이에서 1.72 ± 0.72 (range 0.6 to 2.31) ug/mL, SAM이 있는 고양이에서 1.19 ± 0.70 (range 0.3 to 2.31) ug/mL 그리고 ATE가 있는 고양이에서 1.63 ± 0.80 (range 0.6 to 2.31) ug/mL 였다. 본 연구에서 D-dimer의 농도치가 좌심방 확장과 심부전 유무와 절대적인 상관관계를 보이지는 않았으나, HCM, ATE, SECT 그리고 SAM이 있는 고양이군이 대조군에 비해 혈장 D-dimer의 중간값과 평균 농도치가 더 높다는 것을 발견했다. 이는 HCM이 있는 고양이에서 응고 항진이 나타난다는 명확한 증거이다. 이번 연구는 한국에서 HCM이 있는 고양이의 응고 항진을 평가한 첫 연구이다.

주요어 : 동맥 혈전색전증, 비대성 심근증, 응고항진, D-dimer, 심부전