J Vet Intern Med 2002;16:558–564

Cardiac Troponin I in Feline Hypertrophic Cardiomyopathy

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Measurement of plasma cardiac troponin I concentration ([cTnI]) is a sensitive and specific means for detecting myocardial damage in many mammalian species. Studies have shown that [cTnI] increases rapidly after cardiomyocyte injury. The molecular structure of cTnI is highly conserved across species, and current assays developed for its detection in humans have been validated in many species. In this study, [cTnI] was quantified using a 2-site sandwich assay in plasma of healthy control cats (n = 33) and cats with moderate to severe hypertrophic cardiomyopathy (HCM) (n = 20). [cTnI] was significantly higher in cats with HCM (median, 0.66 ng/mL; range, 0.05–10.93 ng/mL) as compared with normal cats (median, <0.03 ng/mL; range, <0.03–0.16 ng/mL) ($P <$ .0001). An increase in [cTnI] was also highly sensitive (sensitivity = 85%) and specific (specificity = 97%) for differentiating cats with moderate to severe HCM from normal cats. [cTnI] was weakly correlated with diastolic thickness of the left ventricular free wall ($r^2 = .354$; $P = .009$) but not with the diastolic thickness of the interventricular septum ($P = .8467$) or the left atrium : aorta ratio ($P = .0652$). Furthermore, cats with congestive heart failure at the time of cTnI analysis had a significantly higher [cTnI] than did cats that had never had heart failure and those whose heart failure was controlled at the time of analysis ($P = .0095$ and $P = .0201$, respectively). These data indicate that cats with HCM have ongoing myocardial damage. Although the origin of this damage is unknown, it most likely explains the replacement fibrosis that is consistently identified in cats with moderate to severe HCM.

Key words: Congestive heart failure; Ischemia; Protein.

Hypertrophic cardiomyopathy (HCM) is the most commonly diagnosed cardiac disease in cats.[1] It is characterized by a concentrically hypertrophied left ventricular wall and a normal-size to small left ventricular chamber in the absence of other cardiac or systemic disease associated with hypertrophy.[2] In humans, HCM is most commonly inherited as an autosomal dominant trait and is caused by mutations in one of several genes that encode sarcomeric proteins. In humans, the disease exists in several morphologic varieties, the most common of which is hypertrophy of the interventricular septum.[3] In the domestic cat, HCM most commonly involves diffuse hypertrophy of the entire left ventricle, but segmental hypertrophy also is recognized.[4,5] The clinical manifestations of HCM in domestic cats result from impaired diastolic function, which causes an increase in left ventricular filling pressure and the development of congestive heart failure (ie, pulmonary venous enlargement, pulmonary edema, pleural effusion, dyspnea, lethargy, tachypnea, and tachycardia).[1,6–8] Furthermore, cats with HCM frequently suffer from systemic arterial thromboembolism, which is difficult to prevent, difficult to treat, and associated with a poor survival rate.[7–9]

In human medicine, the need for more sensitive and noninvasive methods to identify early myocardial injury has led to increased interest in the use of circulating biochemical cardiac markers. Two such markers, cardiac troponin T (cTnT) and cTnI, have come to the forefront for detecting myocardial cell damage.[10–12] Troponin T is a sarcomeric protein that functions to bind the troponin complex to tropomyosin. Troponin I inhibits the structural interaction of the myosin heads with the actin-binding sites. This inhibition is released in the presence of a sufficient cytosolic calcium concentration. The troponin proteins in the cardiomyocyte exist in 2 major populations. Most of cTnT and cTnI occurs as structurally bound proteins, whereas approximately 6–8% of cTnT and 2–4% cTnI occupy a cytosolic pool.[13] Increased circulating concentrations of both troponins are sensitive and specific markers for myocardial damage.[10–12,14–16] Measurement of circulating cTnI may be a more sensitive means of detecting myocardial injury in humans than measurement of circulating cTnT.[17–19]

The molecular structure of troponin proteins is highly conserved across species, and current assays developed for their detection in humans have been validated in several other species.[15,16,20–22] In veterinary medicine, reports evaluating cardiac troponins relevant to clinical medicine are limited to a few studies.[23–30] Measurement of circulating cTnI concentration ([cTnI]) is a more sensitive means of detecting myocardial cell injury than measurement of either plasma [cTnT] or serum creatine kinase isoenzyme MB (CK-MB) concentration in dogs and cats after blunt chest trauma.[23,24] Clinical research on circulating cardiac troponins in humans has been performed primarily in an attempt to detect myocardial infarction, but evaluation of plasma [cTnI] in patients in congestive heart failure due to myocardial failure also has been investigated.[31–35] An association between cardiomyocyte death and an abnormally high plasma [cTnI] in advanced heart failure was first reported by Missov et al.[31] Investigators have since shown a correlation between the severity of congestive heart failure and circulating [cTnI].[31,33,34] Recent episodes of decompensated congestive heart failure in humans with dilated cardiomyopathy have been associated with an increase in plasma [cTnI], and the [cTnI] has returned to normal following resolution of such episodes.[32]

The primary purpose of this study was to determine the plasma [cTnI] in cats with moderate to severe HCM, with

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Submitted September 4, 2001; Revised March 14, 2002; Accepted May 6, 2002.

0891-6640/02/1605-0010/$3.00/0

and without heart failure, and to compare these values with a plasma reference range for [cTnI] in healthy cats.

Materials and Methods

Patient Population

Thirty-three clinically normal cats judged to be free of heart disease based on complete physical examination were used as the control population. Echocardiograms were performed in approximately 16% of the cats and were normal. Normal cats used in the study were owned by veterinarians and veterinary students at the Veterinary Hospital of the University of Pennsylvania, Philadelphia (VHUP). All animals were followed for at least 6 months after collection of blood for the study. If a cat was determined to be abnormal during follow-up, it was removed from the normal group. The [cTnI] of 21 of these cats has been reported previously.[28] The addition of 12 more normal cats did not change the reference interval for [cTnI] (median, <0.03 ng/mL; range, <0.03–0.16 ng/mL).

Cats with HCM were obtained from the cardiology referral clinics of the VHUP (n = 12) and the University of California-Davis Veterinary Medical Teaching Hospital (VMTH) (n = 8). Cats consecutively diagnosed with moderate to severe HCM at the participating institutions were included in the study. The diagnosis of HCM was made by identifying an end-diastolic measurement of the interventricular septum or left ventricular posterior free wall thickness using 2-dimensional or M-mode echocardiography of ≥6 mm in the absence of systemic or cardiac diseases commonly associated with left ventricular myocardial hypertrophy (eg, systemic arterial hypertension, uremia, hyperthyroidism, aortic stenosis).[4,36–40] Cats with systemic arterial blood pressure of >180 mm Hg, blood urea nitrogen of >34 mg/dL, serum creatinine concentration of ≥2.3 mg/dL, or serum T4 concentration of >5.2 μg/dL were excluded from the study. Specific attempts to diagnose acromegaly by means of growth hormone analysis and computed tomography and to diagnose infiltrative disease by means of histopathology were not routinely conducted. The presence of congestive heart failure was determined with thoracic radiography (ie, evidence of pulmonary edema or pleural effusion).

Plasma Collection and cTnI Analysis

After obtaining owner consent, 1–3 mL of whole blood was collected by venipuncture and promptly placed into a vacutainer tube containing lithium heparin.[a] Plasma samples where obtained by anticoagulant tube centrifugation and supernatant extraction. All samples were analyzed within 2 hours of collection or were stored at −80°C for subsequent assay. Samples were assayed with a 2-site sandwich assay based on solid phase radial partition immunoassay technology by means of the Stratus® CS stat fluorometric analyzer.[b] The analytical sensitivity of this machine is 0.03 ng/mL.[41] Plasma samples from the VMTH were assayed consecutively in a single-blind fashion.

Statistical Analysis

The data from the normal cats and the cats with HCM were analyzed according to signalment (gender and reproductive status, age, and breed), disease status, and [cTnI]. The gender and reproductive status of each group were compared by means of the Pearson chi-square test, and breed comparison was performed with Fisher's exact test. [cTnI] and age in the control group and [cTnI] in the cats with HCM were not normally distributed and were compared by means of the Wilcoxon rank-sum test. Linear regression analysis was performed on the cats with HCM to determine whether the ratio of left atrial diameter to aortic diameter (La/Ao), diastolic interventricular septal thickness (IVSd), or diastolic left ventricular posterior wall thickness (LVPWd) measurements were correlated with [cTnI]. The cat in the HCM that had the highest [cTnI] was determined to be a statistical outlier and was removed from linear regression analysis for each of these comparisons. Data that were not normally distributed are reported as the median, with maximum and minimum values. Characteristics of the group of cats with HCM, including body weight, La/Ao, IVSd, and LVPWd, are reported as mean ± SD. A P value of <.05 was considered significant.

Table 1. Characteristics of the cats studied.

	Normal (n = 33)	HCM (n = 20)
Gender[a]		
Neutered female	8	3
Intact female	2	0
Neutered male	21	17
Intact male	2	0
Age (years)[b]	5.0 (10/0.4)	5.5 (19/1)
Body weight (kg)	NR	5.3 ± 1.7[c]
IVSd (mm)	NR	6.9 ± 1.7[c]
LVPWd (mm)	NR	7.2 ± 1.9[c]
La/Ao	NR	1.8 ± 0.42[c]
Breed[d]		
DSH	28	13
DLH	5	1
Persian	0	1
Scottish Fold	0	1
Maine Coon	0	1
Himalayan	0	1
Burmese	0	1
Persian/DSH	0	1

HCM, hypertrophic cardiomyopathy; NR, not routinely performed; IVSd, interventricular septum in diastole; LVPWd, left ventricular posterior wall in diastole; La/Ao, ratio of left atrium to aorta in diastole; DSH, domestic shorthair; DLH, domestic longhair.

[a] No significant difference between groups, $P = .29$.

[b] No significant difference between groups, $P = .24$. Value is median (maximum/minimum). Only 31 cats from the normal group were included in the age comparison.

[c] Values are mean ± SD.

[d] Significant difference between groups, $P = .04$.

Because the troponin data for the normal cats was not normally distributed, it was transformed using a natural log transformation to establish a cutoff concentration at 2 SD above the mean [cTnI] for normal cats. Normality of the transformed data set was assessed with the Shapiro-Wilk test for normality.[42] The mean and SD of the log-transformed data were calculated, and the upper and lower limits for the reference intervals were determined by the mean ± 2 SD. The antilogarithm of these points was calculated.

Sensitivity was determined by dividing the number of cats with HCM with [cTnI] ≥ 0.157 ng/mL by the total number of cats with HCM. Specificity was determined by dividing the number of normal cats with a [cTnI] < 0.157 ng/mL by the total number of normal cats.

Cats with HCM were grouped into categories as follows: no clinical signs (n = 9), history of congestive heart failure (n = 3), and current congestive heart failure (n = 6). The 2 cats with thromboembolic disease were excluded from HCM subgroup analysis. The Kruskal-Wallis test performed on these groups indicated that they were significantly different from one another. Therefore, the Wilcoxon rank-sum test was performed to determine significant differences in [cTnI] amoung groups.

Results

Patient characteristics for the 33 normal cats and 20 cats with HCM are listed in Table 1. There was no significant difference between the normal cats and those with HCM

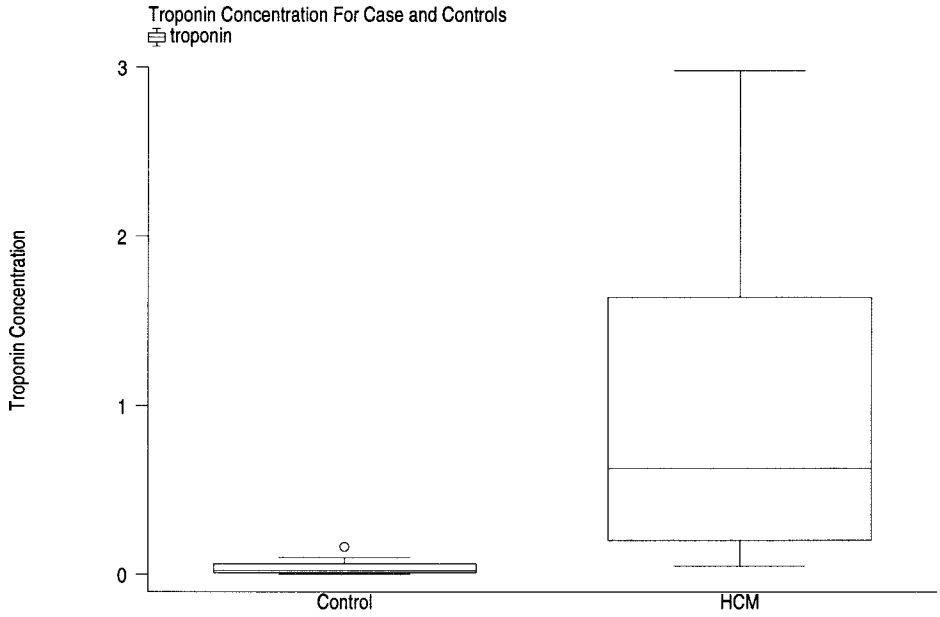

Fig 1. Box-and-whisker plots of cardiac troponin I (cTnI) concentration (ng/mL) in normal cats (n = 33) and cats with hypertrophic cardiomyopathy (n = 19). The cat with the highest cTnI concentration (10.93 ng/mL) is not shown on the graph. The horizontal line in each box represents the median value. The boxes themselves represent the 25th to 75th percentiles (ie, the middle 50% of the data). The whiskers represent the 10th to 90th percentiles. The difference between groups was significant ($P < .0001$).

with regard to gender and age, but they did differ significantly by breed (Table 1). The normal group consisted of 28 domestic shorthair and 5 domestic longhair cats, whereas the HCM group consisted of 13 domestic shorthair cats and 1 each of 7 other breeds. Twelve of the 20 cats with HCM had no clinical signs referable to HCM at the time of [cTnI] determination. Three of these cats had experienced congestive heart failure in the past (all had pleural effusion) and were on cardiac medications (atenolol, n = 2; aspirin, n = 1; enalapril, n = 1; diltiazem, n = 1; furosemide, n = 3) at the time of [cTnI] determination. The remaining 8 cats had clinical signs referable to their cardiac disease. Six presented in congestive heart failure, and 2 presented with systemic thromboembolic disease, 1 of which also had congestive heart failure. Nineteen of the 20 HCM cats had abnormal cardiac auscultation findings. Seventeen and 4 of these 19 cats had systolic heart murmurs and gallop sounds, respectively. Two of the 4 cats with gallop sounds had concurrent systolic heart murmurs. All cats with gallop sounds had congestive heart failure.

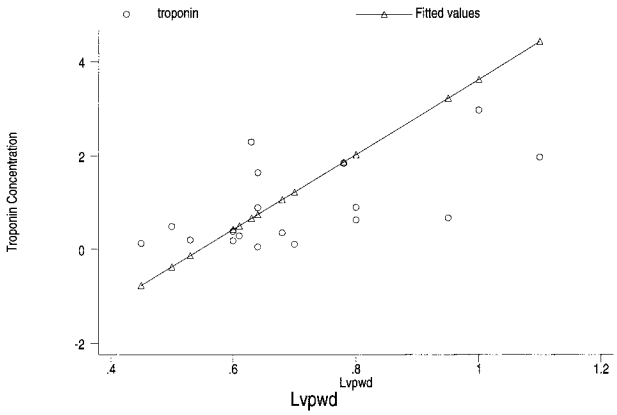

Fig 2. Linear regression analysis showing that cardiac troponin I (cTnI) concentration (ng/mL) varies as a function of left ventricular posterior wall (LVPW) thickness (cm) in diastole in the cats with hypertrophic cardiomyopathy (n = 19; $r^2 = .3539$, $P = .0092$). The highest cTnI concentration (10.93 ng/mL) has been removed from the analysis.

Median [cTnI] in the control group was <0.03 ng/mL (ie, below the level of detection), with maximal and minimal values of 0.16 ng/mL and <0.03ng/mL, respectively. All cats with HCM had detectable [cTnI]. Median [cTnI] in cats with HCM was 0.66 ng/mL, with maximal and minimal values of 10.93 ng/mL and 0.05 ng/mL, respectively. [cTnI] was significantly higher in the group of cats with HCM than in control cats ($P < .0001$; Fig 1). There was a weak linear correlation between [cTnI] and LVPWd thickness ($P = .0092$, $r^2 = .3539$; Fig 2). There was no relationship between IVSd and La/Ao and [cTnI] in the cats with HCM ($P = .8467$ and .0652, respectively). Cats with congestive heart failure at the time of [cTnI] measurement had significantly higher [cTnI] than did cats with no clinical signs ($P = .0095$) and cats with a history of congestive heart failure ($P = .0201$) (Fig 3). [cTnI] in cats with historical congestive heart was not significantly different from that in cats with no clinical signs ($P = .0518$) (Fig 3).

Two of the 20 cats with HCM had thromboembolic disease. These cats had the highest [cTnI] of any of the cats studied (10.93 ng/mL and 2.98 ng/mL). Because of the low numbers of affected cats, such high [cTnI] might skew interpretation of HCM subgroup analysis, and consequently these cats were excluded from this analysis. The cat with the highest [cTnI] had a right femoral artery thromboembolus, pulmonary edema, IVSd thickness of 6.0 mm, LVPWd thickness of 10.1 mm, and LA/Ao ratio of 2.7.

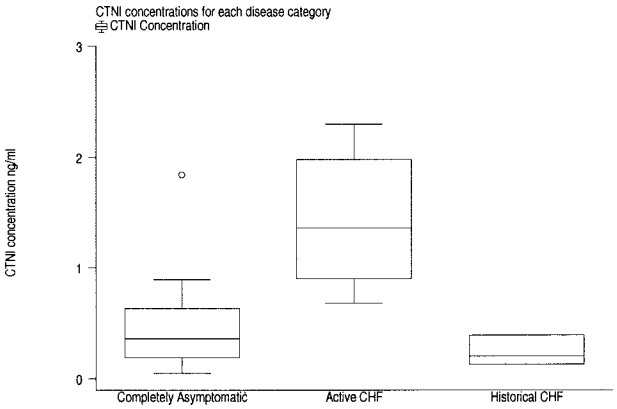

Fig 3. Box-and-whisker plots of cardiac troponin I (cTnI) concentration (ng/mL) in cats with HCM. The disease categories are no history of clinical signs (n = 9), active congestive heart failure (n = 6), and historical congestive heart failure (n = 3). The two cats with thromboembolic disease who had the highest cTnI concentrations (10.93 and 2.98 ng/mL) are not included in the statistical comparison. The horizontal line in each box represents the median value. The boxes themselves represent the 25th to 75th percentiles (ie, the middle 50% of the data). The whiskers represent the 10th to 90th percentiles. The circle represents 1 cat.

This cat was being treated with aspirin, enalapril, and atenolol. Postmortem examination did not reveal any myocardial infarction, but myocardial and intimal fibrosis were present. The other cat had a right brachial artery thromboembolus, IVSd thickness of 8 mm, LVPWd thickness of 10 mm, LA/Ao ratio of 1.9, and no congestive heart failure. This cat was not on medication at the time of evaluation.

[cTnI] in the healthy cats in this study was used as the comparison reference interval. In studies in humans with coronary artery disease, the upper limit of the normal reference interval has been regarded as 2 SD above the mean of the control population (which encompasses 97.5% of the population).[43] Using our data and following this standard, a [cTnI] of $\geq$0.157 ng/mL would be considered abnormal in an otherwise healthy cat. Only 3 cats diagnosed with HCM had a [cTnI] of $<$0.157 ng/mL. Only one normal cat had a [cTnI] of $\geq$0.157 ng/mL. Consequently, the sensitivity and specificity of [cTnI] for moderate to severe HCM was 85 and 97%, respectively.

One cat in the study initially was included in the control group because of a normal physical examination and clinical history. Initial [cTnI] at this time was 0.36 ng/mL. Evaluation 4 months later disclosed a systolic heart murmur. Thoracic radiographs revealed a mildly enlarged cardiac silhouette without evidence of congestive heart failure. Echocardiographic examination revealed systolic anterior motion of the septal mitral valve leaflet, IVSd and LVPWd thicknesses of 7.5 and 6.8 mm, respectively. The La/Ao was 1.5. [cTnI] at the time of echocardiography and radiography was 0.42 ng/mL. Consequently, in this one cat measurement of [cTnI] detected an abnormality before clinical evidence of HCM was present.

Discussion

In the present study, we compared [cTnI] in normal cats and in cats with moderate to severe HCM. [cTnI] was significantly higher in cats with HCM than in control cats. Histopathology was not performed in the affected cats, and therefore a correlation of high [cTnI] with myocardial damage cannot be definitively made. However, a high [cTnI] has been associated with myocardial damage in other species. There have been no reports of [cTnI] in humans with HCM, but our findings are consistent with those of previous investigations in humans in which myocardial damage was secondary to nonischemic myocardial disease, including in humans with idiopathic dilated cardiomyopathy.[31–34]

We did not attempt to determine left ventricular mass noninvasively (echocardiographically) because many cats have asymmetric HCM, and such methods are inaccurate when the concentric hypertrophy is not uniform. [cTnI] was correlated weakly with the thickness of the LVPWd. This finding suggests a possible association between the extent of hypertrophy, or disease severity, and ongoing myocardial damage. However, this association was not strong, and definitive conclusions regarding its relevance cannot presently be made. Subgroup analysis of the cats with HCM indicated that [cTnI] was correlated with the presence of congestive heart failure when compared with both asymptomatic HCM cats and those with a history of congestive heart failure. Although no attempts have been made to specifically define a relationship between [cTnI] and the presence or absence of congestive heart failure in humans, [cTnI] has been shown to decrease after resolution of heart failure.[32]

In the present study, the [cTnI] in cats with HCM was markedly higher than that in healthy cats whether or not congestive heart failure was present. A study in humans using a similar second-generation cTnI assay determined that [cTnI] was abnormally high in patients with severe congestive heart failure compared with healthy individuals.[31] Although there is still some debate, current data indicate that troponin release represents irreversible cardiomyocyte injury.[44] There are several possible pathophysiologic mechanisms for the increase in [cTnI] in cats with HCM in this study, but no specific attempts were made to identify such causes. Microvessel or intramural coronary artery disease occurs in cats with HCM.[45] Intramural coronary arterial disease could contribute to regions of microscopic myocardial ischemia sufficient to cause cell necrosis by rendering myocardial oxygen delivery inadequate to meet tissue demands.[46] The histologic association between myocardial fibrosis in cats with HCM and intramural vessel changes supports this explanation.[45] The presence of myocardial hypertrophy in the absence of adequate increases in myocardial capillary density also could contribute to ischemia (ie, limited coronary reserve).[47] Furthermore, as in humans with HCM, abnormal coronary flow dynamics, decreased coronary vasodilatory reserve, and systolic compression of the septal perforator arteries could contribute to myocardial ischemia and necrosis in cats with HCM.[48–50] In humans, myocardial infarction due to coronary artery atherosclerosis (macrovessel disease) and secondary myocyte necrosis are the most common causes of increased [cTnI]. Although such disease is common in humans, its occurrence in domestic cats is rare. Consequently, it is highly unlikely that large coronary artery atherosclerosis or thrombosis was present in the cats in this study. Although postmortem examination results for most of the cats were not

available, those for the cat with HCM that had the highest [cTnI] (10.93 ng/mL) showed no evidence of macroscopic myocardial infarction.

If high [cTnI] occurs after an isolated event, plasma half-life is an important criterion in establishing a cutoff concentration. In patients with acute myocardial infarction, cTnI is released as the cardiomyocytes undergo ischemic damage. Generally, [cTnI] is above cutoff concentrations for 5–7 days after myocardial infarction.[35] The cats with HCM likely experience chronically high [cTnI] from ongoing damage rather than a single acute insult.

The clinical course of congestive heart failure in humans usually involves activation of the renin-angiotensin-aldosterone axis, the sympathetic nervous system, and the cytokine cascade.[51,52] Such chronically activated pathways are thought to contribute to the long-term progression of myocardial disease and its sequelae. These processes could contribute to chronically high [cTnI]. Reports in the veterinary literature on activation of the renin-angiotensin-aldosterone axis and the sympathetic nervous system in naturally occurring heart disease are limited, but an increase in activity is generally reported.[53–56] Others have not demonstrated activation of these pathophysiological processes.[57] Chronic activation of these pathways might contribute to myocardial damage in cats with HCM, although this possibility has not been investigated.

The 2 highest [cTnI] values were from cats with systemic arterial thromboembolic disease (10.93 and 2.98 ng/mL). There are at least 2 potential explanations for this finding. First, it is assumed that skeletal troponin proteins are released into systemic circulation as a result of ischemic damage to skeletal muscle secondary to embolization. Myofibrillar proteins found in human skeletal muscle do not interfere with the cTnI analysis method used in this study even at an extremely high concentration (skeletal troponin T, 1,000 ng/mL; skeletal troponin I, 280 ng/mL).[41] Percentages of cross-reactivity, ([cTnI]/concentration of cross-reactant) $\times$ 100, for troponin T (skeletal) and troponin I (skeletal) are 4% and 4%, respectively.[41] Although the monoclonal antibody used to detect cTnI may have cross-reacted with the skeletal form of feline troponin I, no attempt was made to quantify such cross-reactions. Further study is justified based on our findings. cTnI has a unique 31–amino acid sequence on the N-terminus of the protein, which is specific to the human cardiac form.[35] The remaining amino acid sequence of cTnI is nearly 40% dissimilar from the human skeletal muscle isoforms.[10,31] Therefore, cTnI determination is considered to be highly specific for detecting myocardial damage in humans.[31–35] Unfortunately, the amino acid sequence of feline cTnI has not been published. The second possible explanation for the marked increase in the [cTnI] in these 2 cats is the possibility of concurrent myocardial infarction due to thromboembolic disease of the myocardium. Although most cats with thromboembolic disease secondary to cardiomyopathy form only 1 large thrombus in the left atrium, both of these cats may have formed additional smaller thrombi that embolized to the coronary vasculature. However, a postmortem examination was performed on only 1 of the cats with thromboembolic disease, and gross evidence of coronary artery occlusion was not found.

Interpretation of the [cTnI] from study to study is confounded by the variety of different assay techniques. Assays differ in the antibody configuration used for different epitopes. There are also nonuniform assay standardizations.[44,58,59] Consequently, normal reference intervals and established cutoff concentrations differ among assays. In this study, all assays were conducted using a Stratus® CS cTnI analyzer, which is a second-generation cTnI assay. The determination of [cTnI] of cats using different techniques likely would require a separate referencing system. The major difference among the first-, second-, and third-generation troponin assays is improved sensitivity and specificity.

Lack of echocardiography results for all of the healthy cats at the time of sample collection limits the conclusions that can be drawn from this study. Although the apparent lack of abnormalities upon reexamination and auscultation months later does not rule out the presence of HCM or other cardiomyopathy, it is unlikely that any of these healthy cats had moderate to severe HCM. Nonetheless, definitive conclusions regarding the normal range for [cTnI] in domestic cats and the sensitivity of [cTnI] in detecting HCM in cats remain illusive because of the screening protocol used for the healthy cats and the small sample sizes. Furthermore, the group of cats with HCM in this study represents a predetermined sample of cats. That is, determination of [cTnI] was only pursued in cats that had moderate to severe HCM diagnosed at a referral center. These cats may represent a more severely diseased sample of the total population of cats with HCM. Therefore, [cTnI] values obtained in this study may not be representative of all cats with HCM, especially those with mild disease. However, a large percentage of the cats with HCM in this study had never exhibited clinical signs (n = 9, 45%) at the time of [cTnI] determination.

No hospitalized cats without cardiac disease and no cats with other forms of cardiac disease were included in the study. The effects of such illnesses could result in high [cTnI] and could reduce the predictive value of a positive test result. Disease processes such as moderate to severe pulmonary embolism and sepsis can result in high concentrations of cTnT and cTnI, respectively, in humans.[60,61] However, in 1 study the difference between healthy people and those hospitalized without evidence of cardiac diseases was minimal, even when using sensitive second-generation cTnI assays.[31] [cTnI] is abnormally high in a variety of cardiovascular disease states and may not be specific for HCM but rather may arise from myocardial damage of any cause. Whereas the utility of [cTnI] as a screening tool requires further investigation, the results of this preliminary investigation indicate that [cTnI] is increased in most cats with moderate to severe HCM examined at the participating veterinary hospitals. Furthermore, because cats with congestive heart failure due to HCM had significantly higher [cTnI] than did those with a history of congestive heart failure, [cTnI] might be useful for assessing therapy.

In only 1 other published study was [cTnI] evaluated to determine the presence of myocardial damage in cats.[24] In that study, cats with blunt thoracic trauma were assessed. Not only was [cTnI] high, but it also was a more sensitive means of detecting myocardial injury than were cTnT or

CK-MB. Although there was no ''gold standard'' (histopathologic evaluation), those findings agree with findings in humans and dogs that indicate cTnI is more sensitive for detecting myocardial damage.[17,18,23]

The results of a second-generation assay indicated higher [cTnI] in cats with moderate to severe HCM than in healthy cats. In the sample of cats with HCM studied, almost all cats had high [cTnI] whereas almost no normal cat had increased [cTnI], indicating that most cats with moderate to severe HCM have some degree of ongoing cardiomyocyte damage. This ongoing damage probably is responsible for the replacement fibrosis seen histologically in cats with HCM.[62] Cats with congestive heart failure had significantly higher [cTnI] than did cats with no clinical signs and those with a history of heart failure. Further research is needed to determine whether [cTnI] is useful for differentiating HCM from other cardiomyopathies in cats or as a screening tool for detecting less severe forms of HCM in cats. Studies to determine whether [cTnI] may be used to indicate disease severity, assist in determining therapeutic efficacy, or determine prognosis also may be warranted.

Footnotes

[a] Vacutainer Systems, Becton Dickinson, Franklin Lakes, NJ
[b] Stratus® CS stat fluorometric analyzer, Dade Behring Inc, Newark, DE

Acknowledgments

The authors thank Dr Fe Wright for her technical and clinical support. This research was supported in part by a Department of Clinical Science research grant from the Veterinary Hospital of the University of Pennsylvania and Dade Behring Inc.

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