Kafkas Univ Vet Fak Derg
20 (6): 921-927, 2014
DOI: 10.9775/kvfd.2014.11403

Journal Home-Page: http://vetdergi.kafkas.edu.tr
Online Submission: http://vetdergikafkas.org

RESEARCH ARTICLE

The Rapid Analyses of Cardiac Troponins in Dogs with Dilated Cardiomyopathy, Distemper or Parvoviral Infection [1]

Vehbi GÜNEŞ [1] Fatma UYANIK [2] Meryem EREN [3]
Murat KİBAR [4] Öznur ASLAN [1] Ali C. ONMAZ [1]

[1] *The present study was financially supported by Scientific Research Project Unit of Erciyes University (Project Code: VA-06-10)*
[1] Department of Internal Medicine, Faculty of Veterinary Medicine, University of Erciyes, TR-38090 Kayseri - TURKEY
[2] School of Health Care Vocational Education, University of Onsekiz Mart, TR-17100 Çanakkale - TURKEY
[3] Department of Biochemistry, Faculty of Veterinary Medicine, University of Erciyes, TR-38090 Kayseri - TURKEY
[4] Department of Veterinary Surgery, Faculty of Veterinary Medicine, University of Erciyes, TR-38090 Kayseri - TURKEY

Article Code: KVFD-2014-11403 Received: 16.04.2014 Accepted: 10.07.2014 Published Online: 06.08.2014

Abstract

In this study, concentrations of cardiac troponins (cTnI and cTnT) and using a cassette kit which is a qualitative assay were used to investigate the diagnosis of myocardial damages due to different diseases in dogs. Study groups were composed of dogs with distemper infection (DI) (Group I, n=8), parvoviral infection (PVI) (Group II, n=18) and dilated cardiomyopathy (DCM) (Group III, n=18). Healthy dogs were included for each group as control. Levels of troponins (cTnI, cTnT) were determined by commercially available ELISA kits and cassette kits were used for qualitative assays. The mean concentrations of cTnI in Groups I, II and III were 2.26±0.41 ng/ml, 2.41±0.98 ng/ml and 5.076±1.32 ng/ml, respectively. Positive cTnI and cTnT expressions were determined in groups as 5/8 (62.5%) and 4/8 (50%) in group I; 5/18 (27.7%) and 5/18 (27.7%) in group II and 13/18 (72.2%) and 6/18 (33.3%) in group III, respectively. In healthy control dogs, cTnI and cTnT were determined to be negative. The cTnI concentrations were more reliable cardiac marker in dogs with cardiomyopathy. Moreover, cTnI and cTnT casette kits used in this study may be valuable diagnostic tools to diagnose the myocardial damage in dogs with DI, PVI and DCM for the small animal practioners.

Keywords: *Cardiac troponin, Cardiomyopathy, Distemper, Parvoviral infection, Dog*

Dilate Kardiyomiyopati, Distemper ya da Parvoviral İnfeksiyonlu Köpeklerde Kardiyak Troponinin Hızlı Analizi

Özet

Bu çalışmada, köpeklerdeki farklı hastalıklara bağlı miyokardial dejenerasyonların teşhisinde kardiyak troponin (cTnI, cTnT) konsantrasyonları ve kalitatif analiz yapan kaset kitlerinin kullanımı araştırıldı. Çalışma grupları distemper enfeksiyonlu (DE) (Group I, n=8), parvoviral enfeksiyonlu (PVE) (Group II, n=18) ve dilate kardiyomiyopatili (Group III, n=18) köpeklerden oluşturuldu. Her bir grubun kontrolü için sağlıklı köpekler çalışmaya dahil edildi. Ticari ELISA kitleri ile troponin (cTnI, cTnT) düzeyleri belirlendi ve kalitatif analizler için kaset kitler kullanıldı. Grup I, II ve III'de ortalama cTnI düzeyleri sırasıyla 2.26±0.41 ng/ml, 2.41±0.98 ng/ml ve 5.076±1.32 ng/ml idi. Pozitif cTnI ve cTnT varlığı da sırasıyla grup I'de 5/8 (62.5%) ve 4/8 (50%); grup II'de 5/18 (27.7%) ve 5/18 (27.7%), ve grup III'te 13/18 (72.2%) ve 6/18 (33.3%) olarak belirlendi. Sağlıklı kontrol köpeklerinde cTnI ve cTnT testlerinin negatif olduğu belirlendi. Kardiyomiyopatili köpeklerde kalp biyobelirteçleri daha güvenilirdi. Ayrıca cTnI ve cTnT kaset kitlerinin küçük hayvan pratisyenleri için; DE, PVE ve kardiyomiyopatili köpeklerde miyokarditisi belirlemek için faydalı bir teşhis aracı olabileceği belirlendi.

Anahtar sözcükler: *Kardiyak troponin, Kardiyomiyopati, Distemper, Parvoviral enfeksiyon, Köpek*

INTRODUCTION

Cardiovascular diseases in dogs are commonly encountered by pet veterinarians. Cardiac valve diseases, cardiac arrhythmias and myocardiopathies are among the most common causes of heart failure in dogs [1].

İletişim (Correspondence)
+90 352 3372740
aonmaz@yahoo.co.uk

Damage of the myocardium for a variety of reasons such as infections, toxications, nutritional deficiencies and idiopathic disorders also occurs in dogs. One of the mechanisms of damage of the myocardium is acute inflammation in the myocardial wall due to thromboembolic diseases caused by bacterial, viral or other parasitic microorganisms [2]. Myocardial damage may also occur due to bacteremia, sepsis, pericarditis or endocarditis. It is difficult to determine the presence of acute myocardial damage and in many occasions it can not be diagnosed [1-3].

Myocardium damage may be classified according to etiopathogenesis [1-3]. *ßeta*hemolytic *Streptococcus, Staphylococcus, Neosporacaninum, Distemper, Parvovirus, Dirofilaria imminitis*, micotic factors, various drugs (Adriamycin, Isoprenalin) and tumors can cause myocardial damages in dogs [1,2]. Physical examination, electrocardiography, echocardiography and analysis of serum biochemical parameters are important in the clinical evaluation of myocardial diseases in dogs. The levels of myoglobin, creatin kinase - myocardial band (CK-MB), lactate dehydrogenase (LDH) and aspartat aminotransferase (AST) enzyme activities are commonly used in the diagnosis of myocardial diseases. The levels of these biomarkers can be used alone or together with to evaluate patients with cardiac injury and they are the most important indicators of cardiac cell death or necrosis in humans or animals [4].

Cardiac troponins (cTn) can also be important indicators of myocardial damage since they leak out of the myocardial cells under inflammatory conditions [4-7]. The troponin complex consists of a group of proteins. There are three different types of troponins and they are classified according to their functions: cardiac troponin I (cTnI), cardiac troponin T (cTnT) and cardiac troponin C (cTnC). Troponin is important in mediating the interaction between actin and myosin in the sarcomere. When there is a damage of the myocardium, the levels of these enzymes increase in the circulation. As a result of acute myocardial syndrome and necrosis, cTnT and cTnI are released into circulation [4,8]. The kits and assays that used to detect the levels of the troponins in humans have also been used in dogs [9-13]. In recent publications, the concentration of cTnI has been investigated in different canine cardiovascular diseases, including congenital cardiac diseases [12,14,15], acquired cardiac heart diseases such as arrhythmogenic right ventricular dysplasia [16], pericardial effusion [15,17,18], cardiac contusion [19], experimental infarction [20], cardiovascular injury, pacing, cardiotoxic drugs with positive and inotropic effects [21] neoplasia [22] dogs with dilated cardiomyopathy [23] and parvoviral enteritis [24]. A previous study also showed to use cTnI to evaluate the efficacy of antiarrhythmia treatment in dogs [25].

However, to the best of our knowledge, the clinical use of troponin cassette kits and cTnT concentration values have not been established in dogs with distemper and parvovirus infections.

In this preliminary study, our goal was to determine the expression of cTnI and cTnT by using qualitative immune chromatographic cassette kits and to measure the serum concentrations of cTnI and cTnT by quantitative ELISA methods in dogs with dilated cardiomyopathy (DCM), parvovirus infection (PVI) and distemper. With the aim of diagnosis of myocardial injury in clinical practice, accuracy of quick cTn kits was investigated. In addition, we wished to determine the advantages and disadvantages of using either cTnT or cTnI to determine myocardial injury in dogs with cardiomyopathy.

MATERIAL and METHODS

Animals

The study group consisted of dogs of various ages and breeds that had different cardiac disorders with cardiac and/or non-cardiac causes. Dogs with DCM, Distemper and PVI were provided from the clinic of the Faculty of Veterinary Medicine of Erciyes University between 2007 and 2009. These dogs were grouped according to their diagnosis as follows: Dogs with Distemper infection (Group I, n=8), dogs with Parvoviral infection (Group II, n=18) and dogs with Dilated Cardiomyopathy (Group III, n=18). The control group consisted of healthy mixed breed dogs (40 male, 29 female) from the Kayseri Municipal dog shelter. The control group was divided into 4 groups according to age: 1-6 months (n= 17), 6-12 months (n= 10), 1-5 years (n= 21) and 5-10 years (n= 21). The general appearance, hair coat, lymph nodes, mucosa (mouth, conjunctive, vaginal), body temperature, heart rate, respiratory rate were examined. Regular clinical examinations of all systems were performed. To determine presence of parvovirus and distemper infections, canine parvovirus casette test kit 10 (Orgenics, Israel) and canine distemper casette test kit (Orgenics, Israel) were utilized.

Cardiac Examinations and Sample Collection

Rightlaterolateral (L/L) dorsoventral (D/V) radiographs, and echocardiographic and electrocardiographic (ECG) records were taken from dogs with cardiologic disorders. Referance values were assessed by the animal's body weight. Cases of DCM also have been identified as outside of FS 25-40%. In dogs with distemper or PVI only ECG findings were recorded.

P and T waves amplitudes, duration of P, T and QRS complex, PQ and QT interval were determined on the ECG recording. Transmission gel applied by shaving the hairs on the 4^{th}-6^{th} intercostal area for echocardiographic examinations. The contraction of the heart muscle and valves were evaluated with 2-D echocardiography. The interventricular septum thickness (IVS) left ventricular

diameter (LVD), left ventricular posterior wall thickness (LVW) and LA/Ao ratio were measured by M-mode echocardiography in systolic and diastolic phases separately. From these values, The fractional shortening (FS%) and the ejection fractions (EF%) were calculated automatically.

Blood samples (2 mL) were collected into a tube containing heparin for the troponin cassette test. Four mL of blood was also collected in tubes without anti-coagulants for the biochemical analyses. To obtain serum samples, blood samples were centrifuged at 3.000 rpm for 15 min. In addition, 2 mL of blood samples were collected in EDTA containing vacuum tubes for hematological examination.

The Results of cTnI and cTnT Cassette Tests, cTnI and cTnT Concentrations and Selected Biochemical Parameters

In order to determine the presence of heart-derived troponin I (cTnI) in the blood of the dogs, Card-I kit Combo test kits (BioMarket, Finland) were used. For determination of troponin-T (cTnT) in the blood Trop-T Sensitive Rapid Assay (Roche, Germany) were employed. Clinical examination, echocardiography, radiography and ECG tests on the dogs were all done on the same day and all results were recorded. The concentration of cardiac TnI was determined using an ELISA reader (Bio-Tek ELX 50; USA). cTnI analysis was performed using a commercial ELISA kit containing 96-well plates which were coated with a monoclonal antibody according to manufacturer's recommended protocol (Multiscan Spectrum Thermo, Finlandiya). Quantitative assessment of cardiac TnT was done at the Kayseri Research and Teaching Hospital Emergency laboratory using Troponin T Stat Elecsys 04660307 (Lot No: 15263501, Roche) kits with an electrochemiluminescence immunoassay technique using a Cobas e 411 device. Some selected biochemical parameters such as; aspartate transaminase (AST), lactate dehydrogenase (LDH) and creatinine kinase (CK), were measured with spectrophotometric commercial kits (Biolabo, France) in serum samples**.**

Statistical Analysis

Statistical analysis was performed using One-way ANOVA followed by Dunnett's t-test for comparison the patient and control groups and a Tukey test was used to compare cTn positive and negatives within the groups. Statistical significance was considered to be $P<0.05$. The results are expressed as means ± standard deviations (SD).

RESULTS

Findings of Serum Biochemical Analysis

Apart from CK, LDH and AST no other significant changes in biochemical parameters were detected *(Table 1)*. Particularly in group III, the mean CK and LDH parameters were found to be increased statistically significantly compare to other groups. Based on this, the CK and LDH activities in cTn I positive dogs were significantly higher when compared to both the cTn negative dogs and the control group ($P<0.001$). The mean AST activity in the cTn positive group was also found to be significantly higher than the cTn negative and control groups ($P<0.001$).

Cardiologic Findings in Group I and II

The elongation of the R wave length, the expansion of the T wave amplitude, P-wave amplitude, sinus tachycardia and sinus arrhythmia findings were determined in ECG examinations in group I. Sinus Tachycardia, arrhythmia and P wave abnormalities were also observed in dogs with Parvovirus infection.

Cardiologic Findings in Group III

In right L/L and D/V radiographs of dogs with DCM,

Table 1. *Mean levels of selected biochemical parameters in cardiac Troponin kit positive and negative dogs. Results are present as mean±SD*
Tablo 1. *Gruplarda kardiyak Troponin kit negatif ve pozitif olan köpeklerin diğer biyokimyasal bulguları. Sonuçlar ortalama±SD olarak verildi*

Groups	Casette Kit Result	LDH (IU/L)	CK (IU/L)	AST (IU/L)
Group I	Tn (+) n=5	21.87±11.50	27.21±12.12	4.33±3.99 [a]
	Control n=15	28.51±13.33	30.88±10.97	0.55±0.31 [b]
Group II	Tn (+) n=5	44.96±17.35	22.635±7.95	2.11±1.20 [c]
	Control n=12	38.72±18.89	22.61±15.31	0.70±0.58 [d]
Group III	Tn (+) n=13	89.94±27.58 [a]	500.02±125.14 [a]	21.05±1.21 [e]
	Control n=42	35.78±41.30 [b]	27.76±7.90 [b]	0.84±0.95 [f]

Different letters in the same column represent statistical difference. P value is P<0.001

generalized or ventricular expansion of the heart, dorsal deviation of the trachea, steepen of the posterior border, loss of details in the thorax, pleural effusion, and diffuse radiopacity in the thorax were observed (n=18). In dogs with right heart dilatation (6 dogs); pouch formations on the dorsal posterior border of myocardium and pulmonary venous congestion were observed. Arrhythmia and LA/Ao ratio were >2 in M-mode graphs in all dogs. In the right parasternal short axis view of the interventricular septum (IVS) and posterior wall (PW), the reduction in the wall thickness, increase in values of left ventricular space (LVID) were observed in echocardiography in all dogs of group III.

Doppler echocardiographic examinations determined in sufficiency of mitral and tricuspid valves with varying degrees. In addition, above 2 mm pericardial fluid was observed in the 2-D ultrasound in 8 dogs.

Results of cTnI and cTnT Measurements

The cTnI and cTnT concentrations of cTn positive animals in each group are given in *Table 2*. In all three groups, the cTnI concentrations were found to be statistically significantly higher in the cTn positive cases than in the cTnI negative cases and the control group. The mean cTnT concentrations were not statistically significantly different in all groups. Mean concentrations of cTnT positive cases were higher than those of cTnI negative cases in the group I and III but were not statistically significant.

Results of cTn Casette Tests

A total of 44 sick dogs admitted to the clinics of Veterinary Hospital between 2007 and 2009 and diagnosed as Distemper infection, Parvovirus infection and dilated cardiomyopathy were included in this study. Positive cTnI and cTnT expressions were determined in groups as 5/8 (62.5%) and 4/8 (50%) in group I; 5/18 (27.7%) and 5/18 (27.7%) in group II and 13/18 (72.2%) and 6/18 (33.3%) in group III, respectively *(Table 3)*. In healthy control dogs, cTnI and cTnT were negative. Cardiac troponins were determined within 30 min after admission to the clinic of Internal Medicine. cTn kits results with the number of cTn positive animals are given in the *Table 1*. Pictures of cTn kits used in the study are shown in *Fig. 1, 2,* and *3*.

Table 2. *Mean cardiac Troponin I and Troponin T concentrations in dogs with distemper infection (group I), parvovirus infection (group II) and dilated cardiomyopathy (group III). Results are present as mean±SD*

Tablo 2. *Distemper, Parvoviral enfeksiyonlu ve dilate kardiyomyopatili köpeklerde ortalama kardiyak Troponin I ve Troponin T konsantrasyonları. Sonuçlar ortalama±SD olarak verildi*

Groups	Casette Kit Results	cTnIng/ml Mean ±SD	cTnTng/ml Mean ±SD
Group I	Tn (+) n= 5	2.26±0.41 [a]	0.15±0.24
	Control n=15	0.27±0.13 [b]	0.01±0.00
	P value	0.025	NS
Group II	Tn (+) n= 5	2.41±0.98 [a]	0.01±0.00
	Control n=12	0.09±0.03 [b]	0.01±0.00
	P value	0.001	NS
Group III	Tn (+) n= 13	5.076±1.32 [a]	0.08±0.06
	Control n=42	0.18±0.09 [b]	0.01±0.00
	P value	0.001	NS

P value was determined between cTn (+) and cTn (-), and was set at P<0.05. Different letters (a, b, c) in each group show statistical difference. NS, not significant

Table 3. *Cardiac Troponin cassette kit findings in dogs with distemper infection (group I), parvovirus infection (group II) and dilated cardiomyopathy (group II)*

Tablo 3. *Distemper (group I), Parvoviral enfeksiyonlu (grup II) ve dilate kardiyomiyopatili (grup III) köpeklerde kardiyak troponin kaset kit bulguları*

Groups	n	cTnI		cTnT	
		+ (%)	-	+ (%)	-
Group I	8	5 (62.5)	3	4 (50)	4
Group II	18	5 (27.7)	13	5 (27.7)	13
Group III	18	13 (72.2)	5	6 (33.3)	12

Numbers describes proportionof positivity or negativity determined by the cassette kits. cTnI: cardiac Troponin I, cTnT: cardiac Troponin T

DISCUSSION

In the present study, commercial cassette kits were successfully used to assess myocardial damage in dogs. To the best of our knowledge, the usage of these cassette kits has not been reported in dogs before. As the sequence of amino acids in these proteins is very much alike in different species [26], it was possible to diagnose myocardial disorders in dogs by kits used in humans. Additionally these cTn kits have been validated for use in the cow [27], calf [28,29] and lambs [30]. Recently, an high-sensitivity immunoassay (direct chemiluminometric method) has been described in clinically affected dogs [12,13]. But these

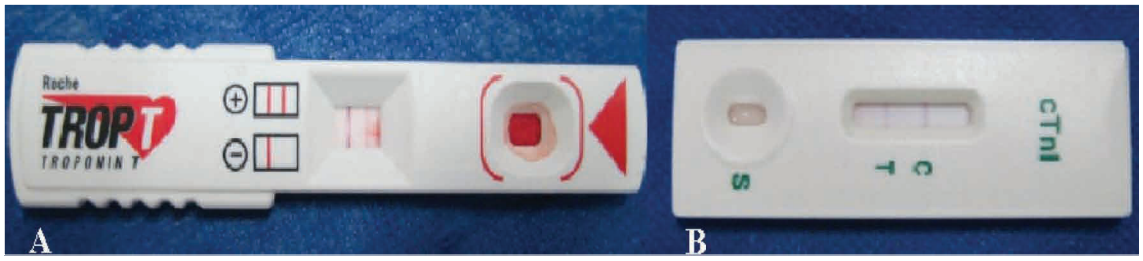

Fig 1. A terrier presented with bloody diarrhea and bloody vomit (hemorrhagic gastroenteritis), **A-** cTnT positive; **B-** cTnI positive *(double line on reading window)*

Şekil 1. Kanlı ishal ve kanlı kusma ile başvuran 14 yaşındaki terrier (hemorajik gastroenteritis), **A-** cTnT Pozitif; **B-** cTnI pozitif *(okuma penceresi üzerindeki çift çizgi)*

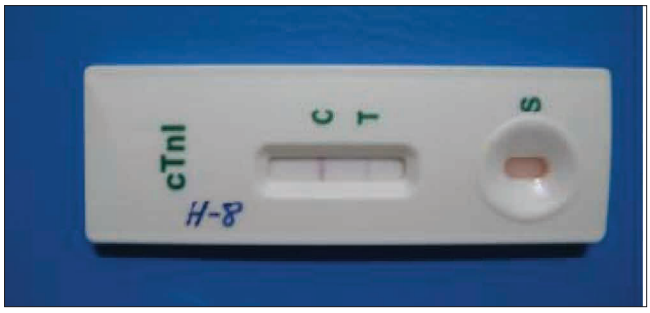

Fig 2. Positive cTnI test in a dog with distemper

Şekil 2. Distemperli bir köpekte pozitif cTnI test

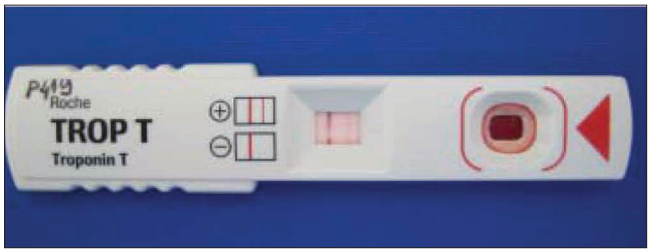

Fig 3. Positive cTnT results obtained from a dog with respiratory difficulties and fatigue symptoms due to mild DCM

Şekil 3. Hafif DCM nedeni ile solunum güçlüğü ve yorgunluk semptomu gösteren bir köpekten elde edilen pozitif cTnT sonuçları

assay process different from the process reported in this study.

In this preliminary study, positivity rates for both cTnI and cTnT casette kits were determined in dogs with distemper infection parvoviral infection and DCM. Positive cTnI rates were higher than those of cTnT in group I and III, however, cTnT were determined as similar rates in group II *(Table 3)*. These data suggest that cardiac cTnT may be positive in more acute cases such as parvoviral infection (group II). Although the concentration of cTn I has been reported to be increased in pericardial effusions [17], in our study the three cases with defined diastolic heart failure and pericardial effusion were negative for cTn I and cTn T based on the results from the casette kits. Since the clinical symptoms were mild, it is possible that in these cases, the myocardium was not yet fully affected.

Acccording to the casette kits results of our study, where result of cTn I cassette kits was higher than those of cTnT in all groups, we speculate that cTnI kit is more sensitive marker than cTnT kits in dogs with myocardial damage *(Table 3)*. In previous studies with healthy dogs [14,15,17,18,31], dogs with myxomatous mitral valve disease, arrhythmia [12,13,25] and dogs with congenital heart failure [12,16], the concentrations of myocardium troponins and especially cTnI were determined [30].

It was reported that the life expectancy of the dogs was longer when the troponin concentrations were low and that the life expectancy was drastically reduced when the troponin concentrations increased [13,24]. The low troponin levels indicate that there is no distinct, active cardiomyocyte death. The dogs that survived cardiac diseases and lived for 1, 2 or 3 years had cTnI concentrations of 0.18 ng/mL, 0.07 ng/mL and 0.05 ng/mL, respectively. In this study, it was determined that positive cassette cTnI kit results compatible with high cTnI concentrations *(Table 2)*.

Cardiac TnI results of this study were in agreement with those of Kocaturk et al.[24]. They postulated that high cTnI levels were likely to contribute to increasing mortality rate and shortening survival length in dogs with parvoviral enteritis. O'Brien et al.[21] compared the cTnI in tissues from different species using a new immunoassay method with the goal of determining human cTnI. Their data showed that cTnI is a perfect candidate to be a biomarker of cardiac injury in mammals. In our study, we also used the diagnostic systems that were developed for human. In healthy dogs, the plasma cTnI levels were determined to be between 0.03 ng/mL and 0.07 ng/mL (average 0.02 ng/mL). Guglielmini et al.[33] found that average cTnI was 0.10 ng/mL (range 0.10-0.17 ng/mL) in healthy dogs. The mean cTnI concentrations of the 1-6 months, 6-12 months and 1-5 years old dogs were 0.09±0.03 ng/mL, 0.27±0.13 ng/mL and 0.18±0.09 ng/mL, respectively, in the present study. These findings were consisted with previous studies [33,34]. There were no significant differences in dogs with different ages. It seems that age might not influence to these parameters. A cTnI level of approximately 1 ng/mL and lower were determined to be normal for healthy dogs.

However, the normal cTnI levels in our study were close to the results obtained by Fonfara et al.[32] where dogs without distinct cardiac disease had a cTnI concentration of 0.21-0.26 ng/mL. The higher values reported in previous studies might be due to methodology and the use of different animals. In a study where the cTnI concentrations of dogs with cardiac diseases were determined using three different analyzers, it was shown that the values obtained by each method were similar but were impossible to compare with each other [35].

The reliability of cTnT concentrations was interpreted as quite low in the present study. As mean cTnT levels were very close to those of control, group I, group II and III. We supposed that cTnT analyses had an insufficient sensitivity in the present study. Moreover, Apple et al.[36] reported that cTnI assays providing a measurable signal in the absence of a cTnT signal in rat, dog and monkey. Additionally, cTnI is more sensitive than cTnT and its values 3-7-fold higher than from cTnT with any given heart damage [37].

In our study, the mean LDH levels in all groups were found to either be within the limits or lower than the normal range. On the other hand, the CK activity was found to be significantly higher in dogs with cardiomyopathy using the cassette kits for troponin (500.02±125.14 IU/L) compared to the control data from the same group (27.76±7.90 IU/L) and to other groups. This difference was interpreted as the evidence of cardiac damage in the present study which similarly reported by Jurlander et al.[4]. In addition, this study showed that cTnI positive dogs had increased CK activity that parallels with an increase in the cTnI concentration and these parameters can be used as specific markers for diagnosis of myocardium damage in dogs. Our results were in agreement with a study that showed cTnI is important and highly specific serological marker [38].

When the cTnI, CK and LDH values compared, it was found that they were not correlated. The cTnI concentrations were significantly higher in the cTnI positive cases compared to cTnI negative cases, whereas there were no distinct differences in the CK and LDH values in these groups. Furthermore, the lacks of tissue specifity and sensitivity and short half time in blood stream of above enzymes have major limitation factors [37] for the diagnosis. Due to this reason, analysis of serum cTnI is more sensitive in diagnosing myocardial damages than the other conventional biochemical markers such as CK and LDH.

A usual cardiovascular system examination includes: disease history, physical examination, complete blood count, serum biochemical analysis, ECG and chest radiographs [39]. In our study, dogs with cardiac diseases were subjected to all the tests listed above, their clinical findings and mean LDH, CK and AST results *(Table 1)* were consistent with the literature [3]. These parameters could be used to differentiate between cardiac and non-cardiac patients, but the cTn parameters were more potent biomarkers for the diagnosis of myocardial diseases. In addition, the results from the cassette cTn kits made diagnosis stronger along with evidence obtained from the physical examination, electrocardiography, echocardiography and biochemical analyses.

In conclusion, expression of cTnI with practice kits and analysis of cTnI concentrations are sensitive determinant of both myocardial damage due to parvoviral myocarditis and distemper and dilated cardiomyopathy in dogs. The concentration of cTnI and cTn cassette kits can be utilized by small animal practioners for the diagnosis of developing myocardial defects. Additionally due to the rapid diagnosis of myocardiopathies with cassette kits without the need for sophisticated laboratory techniques, these quick kits are an important helper diagnostic tool for small animal practioners. Cardiac troponin analyzes should not be disregarded in assessing the prognosis of abovementioned diseases. These cassette kits may also be beneficial to evaluate mortality rate in veterinary emergency and critical care medicine.

Conflict of Intereststatement

None of the authors of this paper has a financial or personal relationship with other people or organizations that could inappropriately influence or bias the content of the paper.

REFERENCES

1. Ettinger SJ, Feldman EC: Textbook of Veterinary Internal Medicine: Diseases of the Dog and Cat. 4th ed., 996-1005, WB Saunders Company, Philadelphia, 1995.

2. Fox PR: Canine myocardial disease. **In,** Fox PR (Ed): Canine and Feline Cardiology. 467-493, Churchill Livingstone, New York, 1988.

3. Fox PR: Myocardial diseases. **In,** Ettinger SJ (Ed): Textbook of Veterinary Internal Medicine. 3rd ed., 1097-1131, WB Saunders, Philadelphia, 1989.

4. Jurlander B, Clemensen P, Wagner GS, Grande P: Very early diagnosis and risk stratificationof patients admitted with suspected acute myocardial infarction by the combined evaluation of a single serum value of cardiactroponin-T, myoglobin and creatinekinase MB. *Eur Heart J*, 21, 382-389, 2000.

5. Christenson RH, Apple FS, Morgan DL, Alonsozana GL, Mascotti K, Olson M, McCormack RT, Wians FH Jr, Keffer JH, Duh SH: Cardiac troponin-I measurement with the Access immunoassay system: Analytical and clinical performance characteristics. *Clin Chem,* 44, 52-60, 1998.

6. Boccara G, Pouzeratte Y, Troncin R, Bonardet A, Boularan AM, Colson P, Mann C: The risk of cardiac injury during laparoscopic fundoplication Cardiac troponin I and ECG study. *Acta Anaesth Scand*, 44, 398-402, 2000.

7. Ooi DS, Isotalo PA, Veinot JP: Correlation of antemortem serum creatinekinase-MB, troponin-I and troponin-T with cardiac pathology. *Clin Chem*, 46, 338-344, 2004.

8. McAuliffe JJ, Robbins J: Troponin T expression in normal and pressure-loaded fetal sheep. *Heart Pediatr Res,* 29, 580-585, 1991.

9. Bader D, Kugelman A, Lanir A, Tamir A, Mula E, Riskin A: Cardiac troponin I serum concentrations in newborns: A study and review of the literature. *Clin Chemica Acta,* 371, 61-65, 2006.

10. Hagman R, Lagerstedt AS, Fransson BA, Bergström A, Häggström J: Cardiac troponin I levels in canine pyometra. *Acta Vet Scand*, 49, 6, 2007.

11. Petric AD and Tomsic K: Diagnostic methods of cardiomyopathy in dogs-old and new perspectives and methods. *Slov Vet Res,* 45, 5-14, 2008.

12. Winter RL, Saunders AB, Gordon SG, Miller MW, Sykes KT, Suchodolski JS, Steiner JM: Analytical validation and clinical evaluation of a commercially available high-sensitivity immunoassay for the measurement of troponin I in humans for use in dogs. *J Vet Cardiol,* 16, 81-89, 2014.

13. Polizopoulou ZS, Koutinas CK, Dasopoulou A, Patsikas M, York M, Roman I, Gandhi M, Patel S, Koutinas AF, O'Brien PJ. Serial analysis of serum cardiac troponin I changes and correlation with clinical findings in 46 dogs with mitral valve disease. *Vet Clin Pathol,* 43, 218-225, 2014.

14. Oyama MA and Sisson DD: Cardiactroponin-I concentration in dogs with cardiac disease. *J Vet Int Med,* 18, 831-839, 2004.

15. Spratt DP, Mellanby RJ, Drury N, Archer J: Cardiactroponin I: Evaluation I of a biomarker for the diagnosis of heart disease in the dog. *J Small Anim Pract,* 46, 139-145, 2005.

16. Baumwart RD, Orvalho J, Meurs KM: Evaluation of serum cardiac troponin I concentration in Boxers with arrhythmogenic right ventricular cardiomyopathy. *Am J Vet Res,* 68, 524-528, 2007.

17. Shaw SP, Rozanski EA, Rush JE: Cardiactroponins I and T in dogs with pericardial effusion. *J Vet Int Med,* 18, 322-324, 2004.

18. Linde A, Summerfield NJ, Sleeper MM, Wright FB, Clifford CA, Melgarejo T, Knight DH: Pilot study on cardiac troponin I levels in dogs with pericardial effusion. *J Vet Cardiol,* 8, 19-23, 2006.

19. Schober KE, Kirbach B, Oechtung G: Non-invasive assessment of myocardial cell injury in dogs with suspected cardiac contusion. *J Vet Cardiol*, 1, 17-25, 1999.

20. Ricchiuti V, Sharkey SW, Murakami MM, Voss EM, Apple FS: Cardiac troponin I and T alterations in dog hearts with myocardial infarction: Correlation within farct size. *Am J Clin Pathol,* 110, 241-247, 1998.

21. O'brien PJ, Smith DE, Knechtel TJ, Marchak MA, Pruimboom-Brees I, Brees DJ, Spratt DP, Archer FJ, Butler P, Potter AN, Provost JP, Richard J, Snyder PA, Reagan WJ: Cardiac troponin I is a sensitive, specific biomarker of cardiac injury in laboratory animals. *Lab Anim,* 40, 153-171,2006.

22. Farace G, Beardow A, Carpenter C, Yeung K, Zieba M, Ettinger SJ, Forney SD: Troponin concentrations in patients with massesor tumors. *J Vet Int Med,* 22, 718, 2008.

23. Çakıroğlu D, Meral Y, Bakırel U, Kazancı D: Cardiac troponin levels in dogs with dilate cardiomyopathy. *Kafkas Univ Vet Fak Derg,* 15 (1): 13-17, 2009.

24. Kocaturk M, Martinez S, Eralp O, Tvarijonaviciute A, Ceron J, Yilmaz Z: Tei index (myocardial performance index) and cardiac biomarkers in dogs with parvoviral enteritis. *Res Vet Sci,* 92, 24-29, 2012.

25. Noszczyk-Nowak A, Paslawska U, Cepiel A, Staszczyk M, Janiszewski A, Nicpon J: 24-hour holter monitoring and Troponin I level in Boxers with arrhythmogenic right ventricular cardiomyopathy. *Kafkas Univ Vet Fak Derg,* 19 (Suppl-A): A99-A104, 2013. DOI: 10.9775/kvfd.2012.8041.

26. O'brien PJ, Landt Y, Ladenson JH: Differential reactivity of cardiac and skeletal muscle from various species in a cardiac troponin I immunoassay. *Clin Chem,* 43, 2333-2338, 1997.

27. Gunes V, Erdogan HM, Citil M, Ozcan K: Assay of cardiac troponins in the diagnosis of myocardial degeneration due to foot-and-mouth disease in a calf. *Vet Rec,* 56, 714-715, 2005.

28. Gunes V, Atalan G, Citil M, Erdogan HM: The use of qualitative cardiac troponin kits in the determination of myocardial cell damage caused by traumatic reticuloperitonitis in cattle. *Vet Rec,* 162, 514-517, 2008.

29. Tunca R, Sozmen M, Erdogan H, Citil M, Uzlu E, Ozen H, Gokçe E.: Determination of cardiac troponin I in the blood and heart of calves with foot-and-mouth disease. *J Vet Diagn Invest,* 20, 598-605, 2008.

30. Tunca R, Erdogan HM, Sozmen M, Citil M, Devrim AK, Erginsoy S, Uzlu E: Evaluation of cardiac troponin I and inducible nitricoxide synthase expressions in lambs with white muscle disease. *Turk J Vet Anim Sci,* 33, 53-59, 2009.

31. Sleeper MM, Clifford CA, Laster LL: Cardiac troponin-I in the normal dog and cat. *J Vet Int Med,* 15, 501-503, 2001.

32. Fonfara S, Loureiro J, Swift S, James R, Cripps P, Dukes-McEwan J: Cardiac troponin I as a marker for severity and prognosis of cardiac disease in dogs. *Vet J,* 184, 334-339, 2010.

33. Guglielmini C, Civitella C, Diana A, Di Tommaso M, Cipone M, Luciani A: Serum cardiac troponin I concentration in dogs with precapillary and postcapillary pulmonary hypertension. *Vet Int Med,* 24, 145-152, 2011.

34. Fredericks S, Merton GK, Lerena MJ, Heining P, Carter ND, Holt DW: Cardiac troponins and creatinekinase content of striated muscle in common laboratory animals. *Clin Chim Acta,* 304, 65-74, 2001.

35. Adin DB, Oyama MA, Sleeper MM, Milner RJ: Comparison of canine cardiac troponin I concentrations as determined by 3 analyzers. *J Vet Int Med,* 20, 1136-1142, 2000.

36. Apple FS, Murakami MM, Ler R, Walker D, York M: Analytical characteristics of commercial cardiac troponin I and T immunoassays in serum from rats, dogs, and monkeys with induced acute myocardial injury. *Clin Chem,* 54, 1982-1989, 2008.

37. O'brien PJ: Cardiac troponin is the most effective translational safety biomarker for myocardial injury in cardio toxicity. *Toxicol,* 245, 206-218, 2008.

38. Giuliani I, Bertinchant JP, Lopez M, Coquelin H, Granier C, Laprade M, Pau B, Larue C: Determination of cardiac tropoin-I forms in the blood of patients with unstable anginapectoris. *Clin Biochem,* 32, 111-117, 2002.

39. Aiello SE: The Merck Veterinary Manual, Circulatory System. 1-101 Merckand Co Inc, White house Station, NJ, 1998.