INTERNATIONAL JOURNAL OF ADVANCED BIOLOGICAL RESEARCH

www.scienceandnature.org

USE OF CARDIAC TROPONIN I FOR EARLY DETECTION OF MYOCARDIAL DAMAGE IN CATS

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ABSTRACT
The present study aimed to investigate the cardiac troponin I (cTn I) and biochemical parameters (CK, AST and LDH) levels in cats. The study involved 90 cats (45 male, 45 female). The cats were grouped as 0-5 years, 5-10 years and 10 - 15 years and over. Troponin I values were 0 .01- 0.33 ng/ml (mean 0.17 ng/ml) in cats, AST levels were higher in cats at 5-10 years of age and, CK levels were higher in cats at 10-15 years of age. Analysis of cardiac troponin I was considered the "gold standard "for the non-invasive diagnosis of myocardial injury in people and small animals. It was replaced traditionally used cardiac biomarkers such as creatine-kinase and its isoenzymes due to its high sensitivity and specificity for the detection of myocardial injury. The purposes of this study were to evaluate a commercially available immunoassay for the detection of cats' cTnI and to show that cTnI will increase in cats with myocardial injury. Increased cTnI of ≥ 0.32 ng/ml was highly specific for the presence of myocardial damage .

KEY WORD: Iraqi cats, Troponin I, CK, AST, LDH.

INTRODUCTION

Cardiac troponin (cTn), is an inhibitory protein complex located on the actin filament in all striated muscles, consists of three subunits T, I, and C [1]. These subunits are cTn I is the largest subunit and bound to actin, cTn T is bound to tropomyosin and cTn C is placed between Tn I and Tn T and has a high affinity against Ca^{+2} ion, Troponins don't exist in the blood of healthy persons or are suggested to exist in very small amounts[2]. Previously used biomarkers of myocardial damage, such as creatine-kinase (CK) and the isoenzyme of CK (CK-MB), have limited value in detecting myocardial injury due to their lack of tissue specificity and sensitivity [3]. The use of cTnI as a biomarker for the detection of myocardial injury has resulted in a substantial increase in the frequency of diagnosing acute myocardial infarction in people[4] Increased membrane permeability may be generated by reversible oxygen deficits as seen in inflammation or toxic damage which leads to degradation and leakage of free cTnI [5]. Several studies in small animals reported increased circulating cTnI concentrations in cats with hypertrophic cardiomyopathy[6].

MATERIALS & METHODS

The animals in this study consisted of 90 Cats. All animals were clinical examination by complete physical examination, checking body temperature, respiratory and heart beat frequency and general clinical appearance. The animals were grouped according to sex (male and female) and age. Each group comprised of 45 male and 45 female and animals were also categorized as. 30 animals in 0-5 years, old (n=30), 5-10 years old (n=30), and 10 -15 years old (n=30) .The blood samples from all animals were obtained from jugular vein into anticoagulant-free container. Sera were obtained by centrifugation at (3000 rpm for 10 min). Serum samples were preserved at -20˚C until analyses .Serum cardiac troponin I (cTn-I) values was determined calorimetrically using commercial test kits (Troponin I kit - DRG Diagnostic) on an ELISA readers (ELISA reader®-DAS for cTn-I). Serum lactate dehydrogenase, aspartate aminotransferase, creatin kinase and myocardial originated creatine kinase levels were measured spectrophtometrically (Photometer® 5010 Boehringer Mannheim) using commercial test kits (Randox®-UK) as instructed by producr. The statistical analysis of data was made using SPSS statistical package[7] and means were compared by student's t test.

RESULTS

Clinical Findings

All animals included in the study were determined as healthy and not healthy on clinical examination.

Serum Troponin I Consantration: Results of cTn-I concentrations showed no significant difference between each of groups and sex, but there was a significant differences between ages of cats($P<0.05$) (Table 1).

Biochemical Results

AST, CK and LDH concentration in sex groups and ages are given in Table 2 and Table 3 respectively.

TABLE 1. Serum troponin I levels in healthy and not healthy cats

Groups	N	Serum ctni (Xmin-Xmax) ng/ml	Mean Ng/ml
Total (ng/ml)	90	0.01 – 0.33	0.17
male (ng/ml)	45	0.02 – 0.032	0.17
female (ng/ml)	45	0.01 – 0.32	0.17
0- 5 years(ng/ml)	30	0.01 – 0.04	0.02
5-10 years (ng/ml)	30	0.02 – 0.27	0.14
10-15 years(ng/ml)	30	0.02 – 0.33	0.17

TABLE 2. The Serum AST, CK,and LDH enzymes according to sex in cats.

Parameters	Sex	N	Serum ctni Mean
AST (IU/L)	Male	15	30.06±1.21
	Female	15	27.96±0.96
CK (IU/L)	Male	15	69.90±10.0
	Female	15	26.60 ±2.8
LDH (IU/L)	Male	15	171.15±11.25
	Female	15	156.65±9.65

TABLE 3. The Serum AST, CK, and LDH enzymes according to age in cats

Parameters	Age	N	Serum ctni Mean
AST(IU/L)	0 – 5 years	10	13.55±2.36[c]
	5 – 10 years	10	33 .00±1.18 [a]
	10 – 15 years	10	26.55±4.72 [b]
CK(IU/L)	0 – 5 years	10	54.7±3.98[c]
	5 – 10 years	10	2.6±5.23 [b]
	10 – 15 years	10	94.6±11.3[a]
LDH(IU/L)	0 – 5 years	10	267.1±19.2
	5 – 10 years	10	297.3 ±30.43
	10 – 15 years	10	208.0±21.15

Means with different subscript letters in the same column differ significantly (P< 0.01) .

AST values obtained in cats aged 5-10 years was significantly (P<0.01) higher than the other age groups. CK was higher in cats aged 10-15 years old (P<0.01) compared with aged 5-10 years and 0-5 years old. A fluctuation of LDH was determined in cats in all age groups.

DISCUSSION

Studies in dogs have reported on the diagnostic use of cTnI and concluded that cTnI is both sensitive and specific for the diagnosis of acute myocardial injury [8]. Dogs with experimental acute myocardial infarction revealed a similar release pattern of cTnI [9]. In human cardiomyocytes, approximately 6 % to 8 % of total cardiac troponin is cytosolically dissolved and thus unbound in cytoplasm [10]. Early after myocardial cell injury affecting cell membrane permeability, parts of this free pool are leech into the blood stream, but the majority of cTnI is retained intracellular because of structural linkage to the contractile apparatus. Thus, release of cTnI may occured mono-physically, with only minor elevation after reversible myocyte injury, or bi- or polyphasically with more severe injury of the myocardium affecting the structurally bound portion of cTnI. The latter is characterized by an early peak of serum cTnI a and a subsequent second, more significant increase[11]. Moreover, we identified a higher sensitivity of cTnI to detect myocardial injury as compared to CK as described by other investigators [12]. In conclusion, the present study confirmed that cTnI is a specific and sensitive biomarker

for the detection of myocardial cell damage in cats. A serum concentration of cTnI > 0.33 ng/ml is an indicator of detectable myocardial necrosis in cats. The measurement of serum cTnI may become a clinically useful tool for the non-invasive diagnosis of myocardial cell injury in cats such as cardiomyopathy. Early detection of cardiac damage may also help in the risk stratification and prognostication in such patients. Further clinical studies are needed to evaluate the diagnostic benefit of the assessment of circulating cTnI in cats with naturally occurring heart disease

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