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INTRODUCTION

Cardiac troponin I (cTnI) is presently recognized as one of the most valuable biochemical markers of myocardial damage. Biochemical markers have been an integrative part of non-invasive diagnostic work-up in cardiology for almost a decade, and with the more recent focus on cardiac troponins this field is now experiencing a renaissance. Indications for cardiac marker analysis are non-invasive diagnosis of both non-ischemic and ischemic myocardial injury. The utilization of cardiac troponins as valuable biochemical markers with high specificity for myocardial muscle has been investigated in common companion and laboratory animals such as the dog, monkey, pig and rat, and normal values for cTnI have been established in pet-owned dogs. Cardiac troponin I and T are considered the new “gold markers” of ischemic myocardial injury in human medicine according to the guidelines from the National Academy of Clinical Biochemistry and the International Federation of Clinical Chemistry.

CARDIAC TROPONIN I

The cardiac troponins are contractile proteins located within each individual myofibril and existing as different isotypes that vary between cardiac and skeletal muscles. These key proteins are an integrated part of the sarcomeric unit, which includes actin, myosin, tropomyosin and troponin I, T and C. The troponin-complex includes proteins involved in the process of myofibril contraction and relaxation, and display quite simple tissue isotype distribution. Cardiac troponin I is uniquely located in the myocardium, where it is the only existing isotype. Since it is not found in skeletal muscle it remains 100% specific for the heart. There is a uniform distribution of cTnI throughout atrial and ventricular myocardium at all developmental ages. cTnI becomes elevated in the plasma within hours after cardiac injury, peaks within the first day and remains elevated for up to a week after the insult. The sensitivity and specificity for cTnI is reported to 97% and 95% respectively.

STUDIES

The cardiac troponins have become a quite popular agenda for research in companion animal medicine in the past five years. Studies have focused on assessment of changes in cardiac troponin levels in different types of pathology. The role of cTnI as a useful biochemical marker for myocardial cell injury has been studied by several different research groups. Assessment of cTnI levels has been made in dogs with various types of cardiac disease including endocardiosis, dilated cardiomyopathy, myocarditis, and pericarditis. cTnI and cTnT levels have furthermore been evaluated in studies on dogs presented with blunt chest trauma and gastric dilatation volvulus. Furthermore changes in cTnI levels have been evaluated in dogs with infectious disease, including Babesiosis and Ehrlichia canis. Our group has furthermore evaluated cTnI levels in a pilot study involving dogs with pericardial effusion of varying etiologies. In addition, cTnI has been assessed in feline hypertrophic cardiomyopathy.

SUMMARY

The general consensus based on the studies that are currently available is that cardiac troponins are useful markers of myocardial damage in the dog, and that cTnI is a more sensitive marker of myocardial insult when compared to cTnT. Data also indicates that cats with hypertrophic cardiomyopathy have ongoing myocardial damage based on cardiac troponin levels. Despite these proteins apparent usefulness as indicators of recent or ongoing damage to the myocardium, the level of cTnI does not seem to be decisive in differentiating types of etiology in dogs with pericardial effusion according to our preliminary findings, even though cTnI levels are significantly elevated in both plasma and pericardial effusion from these dogs. cTnI may eventually prove a valid clinical tool, and as such, might facilitate the decision of appropriate treatment at an early stage in different types of disease processes with a potential negative impact on the myocardial tissue.

REFERENCES

References available upon request

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