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Serum cardiac troponin I in canine syncope and seizures

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Abstract *Objective:* To determine if serum cardiac troponin I (cTnI) concentration distinguishes between cardiogenic syncope and collapsing dogs presenting with either generalized epileptic seizures (both with and without cardiac disease) or vasovagal syncope.
Animals: Seventy-nine prospectively recruited dogs, grouped according to aetiology of collapse: generalized epileptic seizures (group E), cardiogenic syncope (group C), dogs with both epileptic seizures and cardiac disease (group B), vasovagal syncope (group V) or unclassified (group U).
Methods: Most patients had ECG (n = 78), echocardiography (n = 78) and BP measurement (n = 74) performed. Dogs with a history of intoxications, trauma, evidence of metabolic disorders or renal insufficiency (based on serum creatinine concentrations >150 μmol/L and urine specific gravity <1.030) were excluded. Serum cTnI concentrations were measured and compared between groups using non-parametric statistical methods. Multivariable regression analysis investigated factors associated with cTnI. Receiver operator characteristic curve analysis examined whether cTnI could identify cardiogenic syncope.

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Results: Median cTnI concentrations were higher in group C than E (cTnI: 0.165 [0.02–27.41] vs. 0.03 [0.01–1.92] ng/mL; $p<0.05$). Regression analysis found that serum cTnI concentrations decreased with increasing time from collapse ($p=0.015$) and increased with increasing creatinine concentration ($p=0.028$). Serum cTnI diagnosed cardiogenic syncope with a sensitivity of 75% and specificity of 80%.
Conclusions: Serum cTnI concentrations were significantly different between groups C and E. However, due to the overlap in cTnI concentrations between groups cTnI, measurement in an individual is not optimally discriminatory to differentiate cardiogenic syncope from collapse with generalized epileptic seizures (both with and without cardiac disease) or vasovagal syncope.

Abbreviations

ANOVA	analysis of variance
BP	blood pressure
CSF	cerebrospinal fluid
cTnI	cardiac troponin I
GA	general anaesthesia
Group B	group with both cardiac disease and seizures
Group C	group with cardiogenic syncope
Group E	group with epilepsy
Group U	group in which no specific diagnosis was reached (unclassified)
Group V	group with vasovagal syncope
MRI	magnetic resonance imaging
ROC	receiver operator characteristic
SE	status epilepticus
SUDEP	sudden unexpected death in epilepsy
TLOC	transient loss of consciousness
USG	urine specific gravity

Introduction

The two major mechanisms of collapse with transient loss of consciousness (TLOC) are global cerebral hypoperfusion leading to syncope, and asynchronous discharge of cerebral neurons causing a seizure. Syncope and seizures can mimic one another in veterinary patients [1–3]. In human patients, this can also occur, with a consequent high rate of misdiagnosis, particularly as clinical examination on affected patients is often unremarkable at presentation [4–11]. Examples of disorders which may lead to seizure activity and can be confused with syncope include idiopathic epilepsy, intracranial disease, encephalopathies and metabolic disorders such as hypocalcaemia, hypoxia or hypoglycaemia. In human patients, studies suggest that one in four patients with 'epilepsy' may be misdiagnosed [4–8].

In veterinary medicine, published case reports illustrate the challenges of differentiating syncope from seizures [1,3]. Structural cardiac disease and arrhythmias (with or without underlying structural cardiac disease) can cause syncope [2,12]. Methods for differentiating benign causes of weakness and fainting from malignant cardiac arrhythmias, which may degenerate into ventricular fibrillation or cardiac arrest, are clinically important but currently relatively limited. Clinicians are often reliant on an incomplete medical history.

One cardiac biomarker, cardiac troponin I (cTnI), is useful for detecting cardiac myocyte damage [13] and is easy to sample. Increased circulating troponin concentrations have been associated with both cardiac [14,15] and non-cardiac conditions [16–22]. Studies in humans show that epileptic patients with generalized tonic–clonic seizures do not have raised circulating cTnI concentrations [23–26]. However, increased troponin concentrations may occur with more severe seizures, such as status epilepticus (SE), possibly due to seizure-associated catecholamine release causing tachyarrhythmias, coronary ischaemia and thereby myocardial injury [27]. There is little published information on the association of serum cTnI concentration with naturally occurring seizures in dogs, other than single case reports [3,28] and an oral presentation.[f] It is important to gain information regarding cTnI measurements following syncope or seizures to enable correct interpretation of data.

There are no data available regarding the clinical utility of serum cTnI for differentiating cardiac causes of syncope from seizures in dogs. Circulating

[f] Kim J, Song R, Lee D, Lee H, Park J & Park C. Changes of serum cardiac troponin I concentration in 13 dogs with intracranial disorders. J Vet Intern Med 2012; American College of Veterinary Internal Medicine Research abstract C-29.

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