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Estimation of cardiac troponin I level in canine parvovirus infected dogs

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Abstract

Canine parvovirus (CPV) primarily targets gastrointestinal tract, bone marrow and myocardial tissues, with myocarditis mainly seen in foetus and very young puppies. Cardiac troponin I (cTnI) serves as a sensitive biomarker of cardiomyocyte injury. The present study assessed cTnI as a marker of myocardial injury in 36 CPV infected dogs under one year of age. Serum cTnI measured on day of presentation was within the normal range in 86.1 percent of CPV-infected dogs (<30 ng/L; mean 8.81 ± 1.12 ng/L), and 13.9 percent exhibited significantly elevated levels (>30 ng/L; mean 36.5 ± 1.74 ng/L), indicating myocardial involvement. These findings suggest that puppies suffering from myocardial injury due to CPV infection was generally low but when myocardial involvement occurred it would be severe in nature.

Keywords: *Canine parvovirus, troponin, myocarditis, biomarker*

The disease caused by CPV is one of the most common infectious diseases in dogs. The main target-tissues of CPV are the gastrointestinal tract, bone marrow and myocardium. The myocarditis associated with CPV develops from infection in utero or in dogs less than six weeks of age (Gulersoy *et al.*, 2020; Dines *et al.*, 2023; Oikonomidis *et al.*, 2023). Cardiac troponin is the most commonly utilised serum biomarker for detecting myocardial injury (Langhorn and Willesen, 2016). The serum cTnI concentration was elevated in non-surviving CPV-infected dogs, suggesting the presence of myocardial injury (Oikonomidis *et al.*, 2023). This study evaluated serum cTnI concentration in CPV infected dogs on the day of presentation to assess myocardial injury in the CPV infected dogs

The study population comprised of 36 dogs below one year of age, confirmed for CPV infection by conventional polymerase chain reaction (PCR), that were presented to the Department of Veterinary Medicine, Veterinary College, Bengaluru. Control group comprised of six apparently healthy dogs which were tested negative for CPV by PCR. Two millilitres of whole blood was aseptically collected from each dog , either from the cephalic or the lateral saphenous vein, and was dispensed into a sterile vacutainer containing clot activators and the serum was separated. The separated serum was utilised for estimation of cTnI using commercial kit (CHEMICOS VET-CHF100, Guangzhou Herun Bio-Pharm, China).

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The levels of cTnI in CPV infected dogs are presented in Table 1. Out of 36 CPV infected dogs, 31 had cTnI values within the normal range (<30 ng/L), with a mean concentration of 8.81 ± 1.12 ng/L. A statistically significant elevation in cTnI levels (>30 ng/L) was observed in five dogs, with a mean concentration of 36.5 ± 1.74 ng/L. The dogs of control group had mean cTnI levels of 3.69 ± 0.97 ng/L.

Table 1. Level of cardiac troponin I in CPV infected dogs

Parameter	Normal (<30 ng/L)	High (>30ng/L)	Control group
Number of animals	31	5	6
Mean ± S. E	8.81 ± 1.12	36.5 ± 1.74**	3.69 ± 0.97

** = $p < 0.01$ (highly significant)

Cardiac troponin I is a cardiac-specific regulatory protein released into circulation following myocardial damage (Langhorn and Willesen, 2016). In animals with myocardial infarction, elevated cTnI levels have been associated with myocardial injury and systemic inflammatory response syndrome (SIRS) (Gulersoy *et al.*, 2020; Dines *et al.*, 2023). In the present study, 86.1 percent of CPV infected dogs showed normal cTnI levels, suggesting the absence of myocardial involvement. However, 13.9 percent of CPV infected dogs had significantly elevated cTnI levels, indicating myocardial involvement. These findings suggest that the myocardial injury in puppies suffering from myocarditis due to CPV infection occurs in severe conditions. Needle and Mietelka (2014), Ford *et al.* (2017), Oikonomidis *et al.* (2023), Liu *et al.* (2025) and Stancu *et al.* (2025) also reported elevated cTnI levels in CPV infected dogs. The study highlights the use of cTnI in assessing the myocardial injury in CPV infected dogs. The serial assessment of cTnI in CPV infected dogs can act as a prognostic marker in CPV infected dogs and helps to assess the progression of myocardial injury.

Conclusion

Majority of CPV infected puppies showed normal serum cTnI level at presentation, with a smaller subset of CPV infected dogs exhibiting marked elevation indicative of myocardial involvement. These findings support cTnI as a screening biomarker for cardiomyocyte injury in CPV cases, while suggesting that clinically significant myocarditis is uncommon but potentially severe when present.

Conflict of interest

Authors declare no conflict of interest.

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