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Evaluation of high-sensitivity cardiac troponins I and T as biomarkers for subclinical myocardial injury in canine distemper virus infection: a preliminary study

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Abstract

Canine distemper virus is a multisystemic pathogen known to affect various organs, including the heart. However, traditional cardiac biomarkers may lack the sensitivity to detect minor myocardial damage in the early or subclinical stages of the disease. This preliminary study aimed to evaluate the levels of high-sensitivity cardiac troponin I and high-sensitivity cardiac troponin T in dogs naturally infected with canine distemper virus and to assess their potential as indicators of subclinical myocardial injury. The study included 15 dogs diagnosed with distemper and 15 healthy control dogs. The diagnosis was confirmed by rapid antigen testing. Serum high-sensitivity cardiac troponins and haematological indices were analysed. The results revealed significantly higher concentrations of both troponin I (0.381 ± 0.085 ng/ml) and troponin T (0.297 ± 0.074 ng/ml) in the infected group compared to healthy controls ($P < 0.05$), along with characteristic haematological changes, including leukocytosis and lymphopaenia. Although overt clinical signs of heart failure were not the primary complaint in these patients, the significant elevation of high-sensitivity troponins suggests the presence of subclinical myocardial injury associated with the infection. These findings indicate that high-sensitivity assays could serve as valuable screening tools to identify patients at risk of cardiac complications, warranting further cardiac monitoring even in the absence of severe clinical symptoms.

Viral myocarditis, early diagnosis, heart damage, dog

Canine distemper virus (CDV) remains a major cause of mortality worldwide, characterized by its multisystemic nature (Greene and Appel 1998; Beineke et al. 2009). While clinicians are typically familiar with the respiratory, gastrointestinal, and severe neurological manifestations of the disease (Thulin et al. 1992), myocardial involvement remains a critical complication that is frequently overlooked or underdiagnosed (Higgin et al. 1981). Previous research using electrocardiography (ECG) has identified abnormalities, such as R-wave prolongation and T-wave amplitude changes, in infected dogs, suggesting potential cardiac damage (Areshkumar et al. 2018). However, detecting this involvement during the early or subclinical phases is difficult, as the severity of systemic symptoms often masks overt signs of heart failure.

Currently, cardiac troponins (cTnI and cTnT) serve as the most specific biomarkers for myocardial injury (Undhad et al. 2012). Nevertheless, standard assays may lack the necessary sensitivity to identify minor myocardial cell death. In contrast, high-sensitivity cardiac troponin (hs-cTn) assays can detect concentrations far below the limits of conventional methods, enabling the identification of silent injury (Klüser et al. 2019). Since there is currently no data evaluating both hs-cTnI and hs-cTnT specifically in CDV cases, as indicated by the lack of such reports in recent literature (Güneş et al. 2014), this study aims to determine their concentrations in naturally infected dogs to assess the extent of subclinical myocardial injury.

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Materials and Methods

The present study was conducted following approval from the Local Ethics Committee of Kafkas University (Protocol Number: 2023/149) on 5.12.2023. Informed written consent was obtained from the dogs' owners before inclusion in the study.

Animals

A total of 30 dogs were enrolled in the study, divided into two groups: 15 patients with CDV (7 males, 8 females) and 15 healthy control dogs (4 males, 11 females). The infected group consisted of client-owned dogs presented to the Iğdır University Tuzluca Animal Hospital with clinical signs consistent with distemper.

Sample collection and diagnostic procedures

The diagnosis of CDV was established based on clinical findings and confirmed using a rapid immunochromatographic test kit (ASAN Easy Test CDV Ag, Seoul, Korea, Cat. No: 022321), following the manufacturer's protocol. For biochemical and haematological analyses, venous blood samples (5 ml) were drawn from the cephalic vein into serum separator and EDTA tubes. Serum was separated by centrifugation at 1,000 *g* for 10 min and stored at −20 °C until cardiac biomarker analysis. Troponin concentrations remained stable at −20 °C for 3 months (Langhorn and Willesen 2016), serum samples were stored at −20 °C for a maximum of 45 days, and tests were performed.

Cardiac and haematological analysis

Serum concentrations of high-sensitivity cardiac troponin I (hs-cTnI) and troponin T (hs-cTnT) were quantified using species-specific commercial ELISA kits (BT Lab, Shanghai, China, Cat. No E0294Ca) compatible with the Thermo Scientific Multiscan GO microplate reader. Haematological indices, including white blood cell (WBC) counts and differential leukocyte ratios, were analysed using an automated veterinary haematology analyser (Mindray BC-5000 Vet, Shenzhen, China).

Statistical analysis

Data distribution was assessed using normality tests in SPSS software (Version 20.0). Differences between the infected and healthy groups were evaluated using Student's *t*-test for continuous variables, while categorical data were compared using Chi-square test. A P value of ≤ 0.05 was considered significant.

Results

The comparative analysis of cardiac biomarkers demonstrated significant differences between the groups. Serum concentrations of both hs-cTnI and hs-cTnT were found to be significantly higher in CDV-infected dogs compared to the healthy control group ($P < 0.05$) (Table 1). Specifically, mean hs-cTnI concentrations reached 0.381 ± 0.085 ng/ml in the infected group versus 0.151 ± 0.006 ng/ml in controls. Similarly, hs-cTnT concentrations were elevated to 0.297 ± 0.074 ng/ml in the patient group, compared to 0.119 ± 0.014 ng/ml in healthy dogs.

Regarding haematological indices, the infection induced profound changes in the blood profile. A significant leukocytosis, primarily driven by neutrophilia ($P < 0.001$), was observed in the CDV group. In contrast, significant decreases were recorded in lymphocyte counts (lymphopaenia), as well as in red blood cell (RBC) counts, haemoglobin (HGB), haematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), and platelet (PLT) counts compared to healthy controls ($P < 0.001$) (Table 1).

Spearman's correlation analysis revealed a significant positive correlation between hs-cTnI and hs-cTnT concentrations ($r = 0.530$, $P < 0.01$). Furthermore, when the data were stratified by sex, no significant differences were observed in troponin concentrations between male and female dogs within either the healthy or the infected groups ($P > 0.05$) (Table 2).

Discussion

The most important finding of the present study is the significant elevation of high-sensitivity cardiac troponin I (hs-cTnI) and T (hs-cTnT) in dogs naturally infected with CDV compared to healthy controls. While cardiac involvement in CDV, including cardiomyopathy and electrocardiographic abnormalities, has been previously documented

Table 1. Concentrations of hs-cTnT, hs-cTnI, and selected blood indices in healthy and CDV-infected dogs (data are presented as means ± SEM).

Indicator	Healthy	CDV	*P* value
Hs-cTnT (ng/ml)	0.1193 ± 0.0140	0.2971 ± 0.0740	< 0.05
Hs-cTnI (ng/ml)	0.1514 ± 0.0060	0.3809 ± 0.0850	< 0.05
WBC (10^9/l)	16.20 ± 1.80	24.74 ± 2.21	< 0.001
NEU (10^9/l)	11.48 ± 1.35	27.74 ± 1.83	< 0.001
MON (10^9/l)	1.96 ± 0.25	1.63 ± 2.11	< 0.001
LYM (%)	0.12 ± 1.24	0.03 ± 1.32	< 0.001
RBC (10^{12}/l)	7.11 ± 1.36	3.75 ± 0.76	< 0.001
HGB (g/dl)	14.60 ± 1.23	5.30 ± 1.15	< 0.001
HCT (%)	35.00 ± 0.90	14.40 ± 2.13	< 0.001
MCV (fl)	64.70 ± 1.70	41.30 ± 1.26	< 0.001
MCH (Pg)	26.50 ± 1.30	15.90 ± 0.92	< 0.001
PLT (10^9/l)	281.00 ± 2.42	201.00 ± 2.31	< 0.001

Hs-cTnT: high sensitivity cardiac troponin T; hs-cTnI: high sensitivity cardiac troponin I; CDV: canine distemper virus; SEM: standard error of the mean; WBC: white blood cells; NEU: neutrophils; MON: monocytes; LYM: lymphocytes; RBC: red blood cells; HGB: haemoglobin; HCT: haematocrit; MCV: mean corpuscular volume; MCH: mean corpuscular haemoglobin; PLT: platelet

Table 2. Concentrations of hs-cTnT and hs-cTnI by sex.

Indicator	Group	Sex	n	Mean ± SEM	*F/P* value
Hs-cTnT	Healthy	Male	4	0.1132 ± 0.0100	*F* = 3.658
		Female	11	0.1216 ± 0.0190	*P* > 0.05
	CDV	Male	7	0.2728 ± 0.0960	*F* = 0.573
		Female	8	0.3183 ± 0.1180	*P* > 0.05
Hs-cTnI	Healthy	Male	4	0.1500 ± 0.0030	*F* = 3.003
		Female	11	0.1519 ± 0.0080	*P* > 0.05
	CDV	Male	7	0.3303 ± 0.1180	*F* = 1.232
		Female	8	0.4252 ± 0.1260	*P* > 0.05

Hs-cTnT: high sensitivity cardiac troponin T; hs-cTnI: high sensitivity cardiac troponin I; CDV: canine distemper virus; SEM: standard error of the mean

(Higgin et al. 1981; Areshkumar et al. 2018), these conditions are often challenging to diagnose in the subclinical phase. By utilizing high-sensitivity assays, which offer superior detection limits compared to standard methods (Klüser et al. 2019), our study provides the first quantitative biochemical evidence of myocardial injury in CDV-infected dogs that do not exhibit overt clinical signs of heart failure.

Troponins are structural proteins of the contractile apparatus, and their release into the circulation is a definitive sign of myocyte necrosis (Undhad et al. 2012; Langhorn and Willesen 2016). Even slight increases indicate myocardial damage (Carretón et al. 2017). In studies evaluating standard cTnT concentrations in healthy dogs, values have been reported to range from 0.01 to 0.024 ng/ml (Shaw et al. 2004; Burgener et al. 2006; Bakirel and Guneş 2009). For high-sensitivity assays, Kızıltepe et al. (2024) reported hs-cTnT concentrations between 0.0177–0.1223 ng/ml. Our findings in the healthy control group (0.1193 ± 0.014 ng/ml) are consistent with these reference intervals, confirming the absence of myocardial injury in our control subjects.

In the infected group, our study revealed significantly higher hs-cTnT concentrations than in healthy dogs ($P \leq 0.05$). Previous research using standard cTnT assays in CDV-infected dogs reported concentrations of approximately 0.15 ± 0.24 ng/ml (Güneş et al. 2014). To the best of our knowledge, the present study is the first to determine hs-cTnT concentrations specifically in CDV-infected dogs (Ayvazoğlu et al. 2022; Yaşar et al. 2024). Although cTnT is cardiac-specific, concerns exist regarding its re-expression in skeletal muscle during severe disease states, which might confound results (Jaffe et al. 2011). However, cTnI is widely considered a more specific marker for myocardial damage than cTnT (Langhorn and Willesen 2016; Carretón et al. 2017).

Parallel to cTnT, hs-cTnI concentrations were also significantly elevated in our CDV-infected group. The literature indicates that cTnI concentrations in healthy dogs typically range from 0.004 to 0.153 ng/ml (Oyama and Sisson 2004; Langhorn et al. 2013; Polizopoulou et al. 2014), with hs-cTnI specifically reported between 0.0709 and 0.1446 ng/ml (Kızıltepe et al. 2024). Our control group's mean hs-cTnI (0.1514 ± 0.006 ng/ml) aligns with these findings. The significant elevation observed in the infected group (0.3809 ± 0.085 ng/ml) is consistent with previous reports of increased standard cTnI in CDV cases (Kang et al. 2009; Güneş et al. 2014). The strong correlation found between hs-cTnT and hs-cTnI ($P \leq 0.01$) in our study further validates that the leakage is of cardiac origin. These elevations suggest the presence of asymptomatic or subclinical myocardial damage, a phenomenon increasingly recognized in human and veterinary cardiology (De Lemos and Berry 2023; McEvoy et al. 2023; Fanaroff and Sun 2023; Barac et al. 2024).

The myocardial injury observed may be attributed to both direct viral effects and systemic consequences of the infection. Haematological analysis in our study showed significant leukocytosis (neutrophilia) and lymphopaenia, alongside decreases in RBC, HGB, HCT, and PLT concentrations. Lymphopaenia is a hallmark of CDV due to viral lymphotropism (Willi et al. 2015), while the concurrent neutrophilia and anaemia are consistent with previous findings (Daldaban et al. 2021; Pekmezci et al. 2022). These alterations suggest a profound effect on the haematopoietic system (Bohn 2013; Buragohain et al. 2017; Saaed and Al-Obaidi 2021). However, these findings should be interpreted with caution. While cardiac troponins are highly specific for myocardial tissue, they can also be elevated in a wide range of non-cardiac systemic conditions, including sepsis and systemic inflammatory response syndrome. In such contexts, troponin release is not invariably associated with irreversible cell necrosis; rather, it can be driven by mechanisms of reversible ischaemia and cellular stress, such as increased cell membrane permeability, oxidative stress, or the presence of inflammatory cytokines (Hickman et al. 2010; Hori et al. 2018). Severe anaemia and systemic inflammation can lead to 'demand ischaemia' in the myocardium, contributing to troponin release independent of direct viral replication. Importantly, the high-sensitivity assays (hs-cTnI and hs-cTnT) utilized in this study are exceptionally sensitive biomarkers capable of detecting even microscopic, low-grade myocardial injury or subtle cellular perturbations that conventional assays might overlook (Xu et al. 2013). Therefore, in the absence of functional imaging or histopathology, these elevations should be viewed as biochemical evidence suggestive of subclinical myocardial involvement—representing a crucial milestone in clinical risk stratification—rather than definitive structural damage.

Furthermore, we evaluated the effect of sex on biomarker concentrations. Some veterinary studies have noted non-significant trends of higher hs-cTn concentrations in male dogs (Ayvazoğlu et al. 2022; Yaşar et al. 2024), potentially due to the cardioprotective antioxidant effects of oestrogen in females (Kim et al. 1996). In our study, although concentrations appeared numerically higher in some subgroups, no significant difference was found between sexes. This lack of difference might be influenced by the fact that CDV infection rates and severity can vary by sex, with some studies reporting higher prevalence in females (Joshi et al. 2022; McDermott et al. 2023; Mousafarkhani et al. 2023), potentially masking physiological sex differences in troponin release during the acute phase of infection.

This study has several limitations. Primarily, the relatively small number of animals included characterizes this work as a preliminary study. Secondly, although the research was conducted in a university facility, complementary cardiac diagnostic methods such as echocardiography and electrocardiography could not be performed. Due to CDV's highly contagious nature, infected dogs were maintained under strict biosecurity and sanitation protocols in an infectious disease isolation unit. Transporting these patients to the central cardiology unit or utilizing shared, high-end diagnostic equipment within the isolation ward was restricted to prevent nosocomial cross-contamination. Consequently, we could not correlate the biochemical findings with functional cardiac deficits. Furthermore, repeated testing of biomarker values over time was not performed. Because we relied on single-time-point baseline measurements, postmortem histopathology was not evaluated in mortality cases; without longitudinal data, it would be impossible to differentiate whether histological lesions at death were present during initial sampling or developed later due to disease progression. Therefore, the structural damage could not be definitively confirmed. Despite these limitations, this preliminary study provides the first data utilizing high-sensitivity assays for CDV, suggesting that subclinical myocardial involvement—evidenced by elevated hs-cTnI and hs-cTnT—may be a significant component of the disease pathogenesis.

In conclusion, our preliminary study broadens the perspective on myocardial involvement in CDV-infected dogs. The significant increase in both hs-cTnI and hs-cTnT suggests the potential presence of subclinical myocardial stress or injury, which may be overlooked in routine examinations. While elevated troponin concentrations alone do not definitively confirm irreversible structural damage without imaging or histopathology, these high-sensitivity biomarkers should be considered valuable tools for the early detection of cardiac involvement, prompting closer monitoring of cardiovascular health in dogs battling distemper.

Conflict of interest

The authors declare no conflict of interest for this study.

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References

Areshkumar M, Vijayalakshmi P, Selvi D 2018: Evaluation of prognostic indicator based on the electrical conductivity of the heart in canine parvovirus enteritis. Int J Livest Res **8**: 41-46

Ayvazoğlu C, Kiziltepe Ş, Yaşar Ü, Yaşar ZG, Demir P, Acar A 2022: High sensitivity cardiac troponin T (hs-cTnT) and I (hs-cTnI) levels in dogs with *Dirofilaria immitis*. Turk J Vet Anim Sci **46**: 718-723

Bakirel U, Guneş S 2009: Value of cardiac markers in dogs with chronic mitral valve disease. Acta Vet (Beograd) **59**: 223-229

Barac A, Wadlow RC, Deeken JF, de Filippi C 2024: Cardiac troponin I and T in ICI myocarditis screening, diagnosis, and prognosis. Cardio Oncology **6**: 804-807

Beineke A, Puff C, Seehusen F, Baumgärtner W 2009: Pathogenesis and immunopathology of systemic and nervous canine distemper. Vet Immunol Immunopathol **127**: 1-18

Bohn AA 2013: Diagnosis of disorders of iron metabolism in dogs and cats. Vet Clin North Am Small Anim Pract **43**: 1319-1330

Buragohain M, Goswani S, Kalita DJ 2017: Clinicopathological findings of canine distemper virus infection in dogs. J Entomol Zool Stud **5**: 1817-1819

Burgener IA, Kovacevic A, Mauldin GN, Lombard CW 2006: Cardiac troponins as indicators of acute myocardial damage in dogs. J Vet Intern Med **20**: 277-283
Carretón E, Morchón R, Montoya-Alonso JA 2017: Cardiopulmonary and inflammatory biomarkers in heartworm disease. Parasit Vectors **10**: 151-163
Daldaban F, Karaca Bekdik I, Aslan Ö, Akyüz B, Akçay A, Arslan K 2021: Investigation of TLR1-9 genes and miR-155 expression in dogs infected with canine distemper. Comp Immunol Microbiol Infect Dis **79**: 101711
De Lemos JA, Berry JD 2023: Comparisons of multiple troponin assays for detecting chronic myocardial injury in the general population: redundant or complementary? Eur Heart J **44**: 2606-2608
Fanaroff AC, Sun LL 2023: High-sensitivity troponin in patients with cancer. JACC Cardio Oncol **5**: 610-612
Greene CE, Appel MJ 1998: Canine distemper. In: Greene CE (Ed.): Infectious Diseases of the Dog and Cat. 2nd Edition. WB Saunders Co., Philadelphia, pp. 9-22
Güneş V, Uyanık F, Eren M, Kibar M, Aslan Ö, Onmaz AC 2014: The rapid analyses of cardiac troponins in dogs with dilated cardiomyopathy, distemper or parvoviral infection. Kafkas Univ Vet Fak Derg **20**: 921-927
Hickman PE, Potter JM, Aroney C, Koerbin G, Southcott E, Wu AH, Roberts MS 2010: Cardiac troponin may be released by ischemia alone, without necrosis. Clin Chim Acta **411**: 318-323
Higgin RJ, Krakowka S, Metzler AE, Koestner A 1981: Canine distemper virus-associated cardiac necrosis in the dog. Vet Pathol **18**: 472-486
Hori Y, Iguchi M, Heishima Y, Yamashita Y, Nakamura K, Hirakawa A, Kitade A, Ibaragi T, Katagi M, Sawada T, Yuki M, Kanno N, Inaba H, Isayama N, Onodera H, Iwasa N, Kino M, Narukawa M, Uchida S 2018: Diagnostic utility of cardiac troponin I in cats with hypertrophic cardiomyopathy. J Vet Intern Med **32**: 922-929
Jaffe AS, Vasile VC, Milone M, Saenger AK, Olson KN, Apple FS 2011: Diseased skeletal muscle: a non-cardiac source of increased circulating concentrations of cardiac troponin T. J Am Coll Cardiol **58**: 1819-1824
Joshi VR, Bhanderi BB, Mathakiya RA, Jhala MK, Desai DN 2022: Sero-surveillance of canine distemper in dogs. Indian J Vet Sci Biotechnol **18**: 100-103
Kang JH, Cheon HB, Kim JH, Park JU, Han TS, Han JI, Kim GH, Chang DW, Na KJ, Kang HG, Yang, MP 2009: Conversion of ventricular tachyarrhythmia by propafenone and atenolol administration in a dog with high serum cardiac troponin I concentration. J Vet Clin **26**: 239
Kim YD, Chen B, Beauregard J, Kouretas P, Thomas G, Farhat MY, Myers AK, Lees DE 1996: 17β-Estradiol prevents dysfunction of canine coronary endothelium and myocardium and reperfusion arrhythmias after brief ischemia/reperfusion. Circulation **94**: 2901-2908
Kızıltepe Ş, Ayvazoğlu C, Yaşar Ü, Yaşar ZG, Aydin N, Tasci GT 2024: Determination of cardiac troponin levels in dogs with babesiosis. Van Vet J **35**: 164-167
Klüser L, Maier ET, Wess G 2019: Evaluation of a high-sensitivity cardiac troponin I assay compared to a first-generation cardiac troponin I assay in Doberman Pinschers with and without dilated cardiomyopathy. J Vet Intern Med **33**: 54-63
Langhorn R, Willesen JL 2016: Cardiac troponins in dogs and cats. J Vet Intern Med **30**: 36-50
Langhorn R, Willesen JL, Tarnow I, Kjelgaard-Hansen M 2013: Evaluation of a high-sensitivity assay for measurement of canine and feline serum cardiac troponin I. Vet Clin Pathol **42**: 490-498
McDermott I, Gilbert M, Shah MK, Sadaula A, Anderson NE 2023: Seroprevalence of canine distemper virus (CDV) in the free-roaming dog (*Canis familiaris*) population surrounding Chitwan National Park, Nepal. Plos One **18**: e0281542
McEvoy JW, Daya N, Tang O, Fang M, Ndumele CE, Coresh J, Christenson RH, Selvin E 2023: High-sensitivity troponins and mortality in the general population. Eur Heart J **44**: 2595-2605
Mousafarkhani F, Sarchahi AA, Mohebalian H, Khoshnegah J, Arbabi M 2023: Prevalence of canine distemper in dogs referred to Veterinary Hospital of Ferdowsi University of Mashhad, Mashhad, Iran. Vet Res Forum **14**: 153-160
Oyama MA, Sisson DD 2004: Cardiac troponin-I concentration in dogs with cardiac disease. J Vet Intern Med **18**: 831-839
Pekmezci D, Konaçoğlu GN, Çolak ZN 2022: A preliminary study for determination of neutrophil to lymphocyte, monocyte to lymphocyte and platelet to lymphocyte ratios in dogs with canine distemper virus infection. J Anatol Environ Animal Sci **7**: 437-443
Polizopoulou ZS, Koutinas CK, Dasopoulou A, Patsikas M, York M, Roman I, Gandhi M, Patel S, Koutinas AF, O'Brien PJ 2014: 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
Saaed MM, Al-Obaidi QT 2021: Clinical, hematological and some biochemical changes in dogs infected with canine distemper. IOSR-JAVS **14**:26-33
Shaw SP, Rozanski EA, Rush JE 2004: Cardiac troponins I and T in dogs with pericardial effusion. J Vet Intern Med **18**: 322-324
Thulin JD, Granstrom DE, Gelberg HB, Morton DG, French RA, Giles RC 1992: Concurrent protozoal encephalitis and canine distemper virus infection in a raccoon (*Procyon lotor*). Vet Rec **130**: 162-164
Undhad VV, Fefar DT, Jivani BM, Gupta H, Ghodasara DJ, Joshi BP, Prajapati KS 2012: Cardiac troponin: an emerging cardiac biomarker in animal health. Vet World **5**: 508-511

Willi B, Spiri AM, Meli M, Grimm F, Beatrice L, Riond B, Bley T, Jordi R, Dennler M, Hofmann-Lehmann R 2015: Clinical and molecular investigation of a canine distemper outbreak and vector-borne infections in a group of rescue dogs imported from Hungary to Switzerland. BMC Vet Res **11**: 154

Xu RY, Zhu XF, Yang Y, Ye P 2013: High-sensitive cardiac troponin T. J Geriatr Cardiol **10**: 102–109

Yaşar Ü, Ayvazoğlu C, Yasar ZG, Kiziltepe Ş, Aydin N 2024: High-sensitivity cardiac troponin (hs-cTnI and hs-cTnT) levels in dogs with *Hepatozoon canis*. J Agric Sci **8**: 482-492