Journal of Applied Veterinary Sciences, 9 (4): 10 - 16 (October, 2024).
ISSN: Online: 2090-3308, Print: 1687-4072
Journal homepage : https://javs.journals.ekb.eg

Implementation of Cardiac Troponin I as A Sensitive Biomarker for Myocardial Injury in Animals: Review Article

Mohamed Tharwat [1,2,*] and Mohamed Marzok [3,4]

[1]Department of Clinical Sciences, College of Veterinary Medicine, Qassim University, P.O. Box 6622, Buraidah, 51452, Saudi Arabia

[2]Department of Animal Medicine, Faculty of Veterinary Medicine, Zagazig University, 44519, Zagazig, Egypt

[3]Department of Clinical Sciences, College of Veterinary Medicine, King Faisal University, Al-Ahsa, Saudi Arabia

[4]Department of Surgery, Faculty of Veterinary Medicine, Kafr El Sheikh University, Kafr El Sheikh, Egypt

***Corresponding Author:** Mohamed Tharwat, **E-Mail:** atieh@qu.edu.sa

ABSTRACT

A biomarker is defined as a detectable biomarker of a specific state and is often measured in urine, blood, milk, sweat, tear or soft tissue, either in physiological or pathological conditions. Of these commonly used biomarkers specifically for heart injury, there are cardiac troponin I (cTnI) and N-terminal pro-B-type natriuretic peptide. The cardiac troponin I is a sensitive biomarker formed by the injured cardiac cells and hence is used in humans for the early discovery of cardiac injury, and in parallel, it is used for prognostic purposes. Veterinary medicine widely uses cTnI as a marker for myocardial injury. In equines, cTnI was found to be a useful marker for the diagnosis of either primary or secondary myocardial injuries. It has also been used in newborn dogs to predict hypoxia and cardiac injury as a result of perinatal asphyxia. In neonatal calves with congenital heart anomalies, it was reported that the affected animals had a higher concentration of cTnI. In lambs with myocarditis, it was reported that significant levels of serum cTnI were detectable in diseased cases compared to healthy ones. This review article was written to emphasize the clinical significance of cTnI as a sensitive biomarker for cardiac injury in animals either in physiological or pathologic states. It has been concluded from several pathological and physiological studies that cTnI is a highly specific cardiac biomarker. It can detect myocardial injury due to primary cardiac disorders such as pericarditis, endocarditis and myocarditis. Also, it can predict cardiac injury in some non-cardiac-affected cases that can affect the heart, such as racing competitions, long road transportation, extensive training, difficult parturition, Downer syndrome, general anesthesia, infestation with external parasites such as ticks, calcium injection in extremely high doses, injection of cardiac glycosides, and use of the electroejaculation method to collect semen.

Keywords: Animals, Biomarkers, Cardiac Troponin I, Myocardial injury, Pathophysiology.

Review Article:
DOI:https://dx.doi.org/10.21608/javs.2024.298632.1358
Received : 22 June, 2024.
Accepted: 21 August, 2024.
Published in October, 2024.

J. Appl. Vet. Sci., 9(4): 10-16.

INTRODUCTION

In the medical sciences, a biomarker is defined as a detectable marker for a specific condition and is often evaluated in urine, blood, milk, sweat, tear or soft tissue, either in physiological or pathological circumstances (**Jesuthasan *et al.*, 2022; Tharwat, 2023**). Of these markers, cardiac-derived biomarkers are currently used on a large scale in veterinary practice; the two most commonly used markers are cardiac troponin I (cTnI) and N-terminal pro-B-type natriuretic peptide (**Oyama, 2013**).

The cTnI is a sensitive biomarker used in human medicine to predict myocardial injury. It is formed by the injured myocytes and therefore is currently used in human medicine for the early detection of cardiac damage and, at the same time, for the prognosis of the affected cases (**La Vecchia *et al.*, 2000;**

Koureti, 2022). It was reported that in human patients with myocardial infarction, the blood level of cTnI is increased not only after the occurrence of the disease but elevated due to various pathologies affecting the myocardium (**Katrukha and Katrukha, 2021**). During the pandemic of COVID-19, the levels of cTnI have been extensively used in ten of the studies to predict the prognosis of the patients (**Al Abbasi *et al.*, 2020; Lippi *et al.*, 2020; Malik *et al.*, 2020; Salvatici *et al.*, 2020; Shi *et al.*, 2020; Ali *et al.*, 2021**).

In animals, the use of cTnI as a biomarker for heart injury is broadly applied (**Tharwat, 2020; Tharwat, 2023**). In horses, it was found that cTnI is a useful biomarker for the diagnosis of either primary or secondary myocardial injury cases compared to healthy horses (**Van Der Vekens *et al.*, 2015**). The biomarker has also been used in dogs to predict hypoxia and cardiac injury as a result of perinatal asphyxia in newborn dogs. It was concluded in the latter study that serum cTnI was higher in asphyxiated cases compared to non-asphyxiated neonatal dogs and therefore may be used as a sensitive biomarker of ischemic cardiac injury in neonatal dogs (**Nobre Pacífico Pereira *et al.*, 2022**).

In calves with congenital heart anomalies, it was reported that the diseased calves had a higher serum concentration of cTnI (**Suzuki *et al.*, 2012**). In lambs with myocarditis, a significant increase in serum cTnI was measured and reported in diseased versus healthy lambs (**Karapinar *et al.*, 2012**). More recently, it was reported that with the development of embryos, the level of cTnI decreased towards parturition in sheep (**Jonker and Louey, 2024**).

The uses of cTnI in veterinary medicine were found to be highly applicable in both diseased and sound animals for the evaluation of the myocardium's functional status. This review article was therefore designed to emphasize the importance of cTnI as a sensible biomarker for cardiac injury in animals, either in physiological or pathologic states.

Cardiac troponin I and physiological conditions

A report conducted on healthy racing greyhounds with a 7-kilometer competition race showed that serum cTnI concentration increased in 97% of the dogs with a highly significant difference when compared to pre-arcing concentration, then declined a day after the competition ended. However, the factors behind such increases in healthy greyhounds are not strictly identified, and therefore the detected high levels were attributed to racing **(Fig. 1) (Tharwat *et al.*, 2013b).**

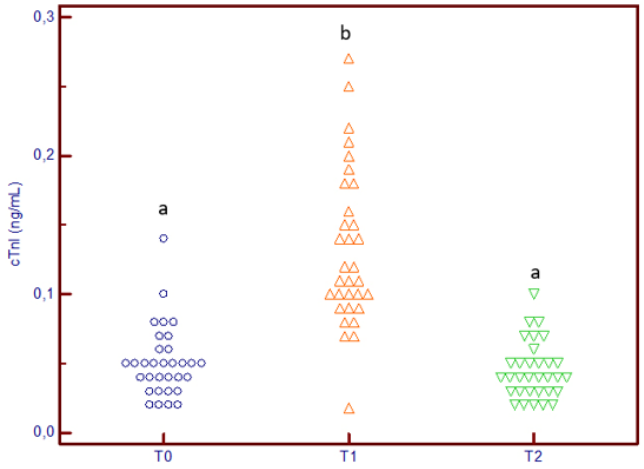

Fig. 1. Comparison of serum cardiac troponin I levels in 32 racing greyhounds' pre-race (T0), 2 hours post-race (T1) and 24 hours post-race (T2) (Tharwat et al., 2013b). [a,b] Different letters indicate statistical significance ($P < 0.05$).

The high cTnI concentrations were detected in healthy sled dogs who participated in exercise of different intensities (**McKenzie *et al.*, 2007**). In another study carried out on healthy racing camels, the level of cTnI was also significantly raised in 91.3% of the animals after a competition of 5 km but dropped a day post-race to pre-race concentrations (**Fig. 2**) (**Tharwat *et al.*, 2013d**).

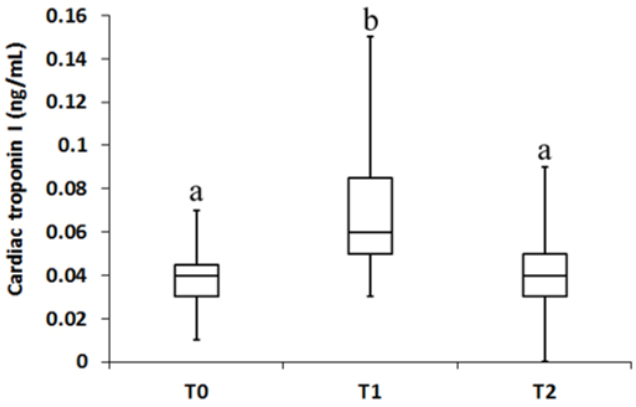

Fig. 2. Alterations of cardiac troponin I values in camels before (T0), 2 h after (T1) and 24 h after (T2) a 5 km race (Tharwat et al., 2013d). [a,b] Different letters indicate statistical significance ($P < 0.05$).

Nostell and Haggström (2008) suggested that the increases in cTnI in horses may be due to the hypoxia that accompanied exercise, resulting in a change in myocardial permeability and finally leakage of cTnI into the bloodstream. The decline in cTnI after racing suggested that the myocardial injury in racing competitions was reversible and therefore should be

explained in future studies as an adaptation for the race. Interestingly, the level of cTnI did not show any significant changes after 8 km of training in healthy racing camels versus base line levels (**Tharwat, 2021**). The latter findings support the theory that cTnI is released only during intense exercise or racing competitions as a result of cardiac hypoxia, as reported by **Nostell and Haggström (2008).**

In 25 healthy camels transported for a round trip of 500 km that took approximately 5 hours, the serum level of cTnI increased significantly 2 hours post-loading compared to pre-loading concentrations; the levels returned to pre-experiment values 24 hours later (**Fig. 3**) (**Tharwat *et al.*, 2013c**). The reason why cTnI elevated significantly after transportation is not clear. However, because the transported camels were healthy, cTnI release may be due to stress during transportation.

It is also evident that this process is reversible, as the cTnI level returned to its baseline concentration within 24 hours of the end of transportation. On the contrary, no significant differences were detected in 10 horses subjected to a 300-kilometer round-trip journey that took about 3.5 hours (**Fig. 4**) (**Tharwat and Al-Sobayil, 2014c**). Why the level of cTnI did not increase significantly as camels did may be explained on the basis of the method of transportation applied in both trials. In the camel experiment, animals were transported for 500 km, or about 5 hours, loaded in a recumbent in an open truck, and loaded and unloaded by a winch; therefore, this method is stressful. On the other hand, horses were transported for 300 km, or about 3 hours, standing throughout the experiment, loaded in a closed truck, and the climatic conditions were optimal; that is why cTnI did not differ significantly.

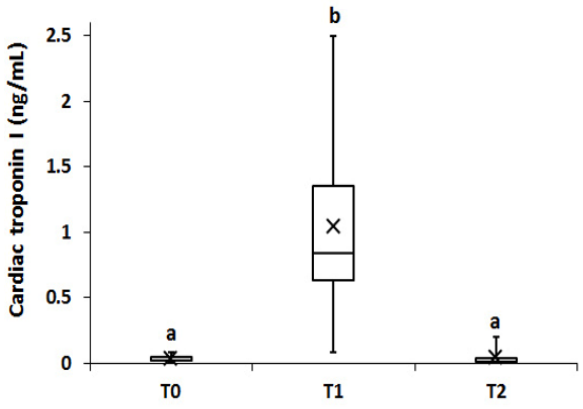

Fig. 3. Box and whiskers plot of cardiac troponin I and effect in camels transported for a round trip of 500 km; before (T0), within 2 hours of the end of transport (T1) and 24 hours after transportation (T2). **(Tharwat *et al.*, 2013c).** [a,b] Different letters indicate statistical significance ($P < 0.001$).

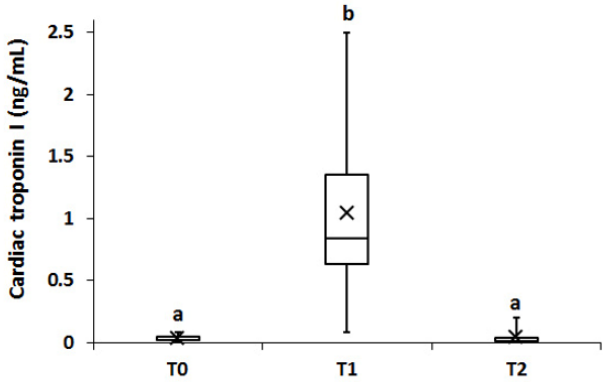

Fig. 4. Box and whiskers plot of cardiac troponin and effect of transportation in horses transported for 300 km. T0, 24 hours before transport; T1, just before transport; T2–T5 at 50,100, 200, and 300 km of transport; T6, 24 hours after transport. The circle and 6 indicates that the animal number 6 is an outlier at T4 (0.04 ng/ ml) **(Tharwat and Al-Sobayil, 2014c).**

Cardiac troponin I and pathological conditions

Primary cardiac pathologies

Cardiac troponin I was especially elevated in animals affected by primary cardiac diseases. In cattle, it was reported that cTnI was significantly elevated in animals with pericarditis in comparison to healthy cows **(Mellanby *et al.*, 2009; Buczinski and Bélanger, 2010).** In a *Dorcas gazelle* with vegetation of the mitral valve, cTnI measured 2.18 ng/ml; it declined to 0.09 ng/ml after therapeutic intervention **(Tharwat *et al.*, 2014).** In newborn goat kids with a cardiac form of nutritional muscular dystrophy, the serum concentration of cTnI was significantly higher than values in healthy kids (11.18±20.07 ng/ml vs. 0.02±0.05 ng/ml; P = 0.017) **(Fig. 5) (Tharwat *et al.*, 2013e).** Twenty-three percent of diseased goat kids had a cTnI level of more than 50 ng/mL. Pathologic findings in the latter study showed macroscopic epicardial and endocardial degeneration, microscopic advanced cardiac degeneration, Zenker's necrosis, remarkable necrotic myocarditis and fibrosed plaques between the muscles and sub-endocardium **(Tharwat *et al.*, 2013e).**

Secondary cardiac pathologies

Cardiac troponin I is elevated in numerous disorders of non-cardiac origin. In 10 out of 19 cases (53%) of goats with prolonged parturition, the serum concentration of cTnI was significantly increased compared to those with normal birth (0.094±0.155 ng/ml vs. 0.008 ± 0.013 ng/ml). However, 16 out of 18 (89%) goats with pregnancy toxemia showed remarkable increases in serum cTnI that reached a value of 0.852 ± 1.472 ng/ml **(Fig. 6) (Tharwat *et al.*, 2012).** The significantly elevated cTnI in goats with pregnancy toxemia pointed out the occurrence of severe cardiac

injury, an important factor that explains the deaths of 12 goats with pregnancy toxemia just after admission. Therefore, it was postulated that significantly higher levels of cTnI in goats with pregnancy toxemia suggested a guarded prognosis **(Tharwat *et al.*, 2012).** In a recent study, it was found that the serum level of cTnI increased significantly in dromedary camels with dystocia compared to those with normal parturition (0.18±0.12 ng/ml vs. 0.03±0.02 ng/ml, P = 0.0007) **(Tharwat *et al.*, 2024).**

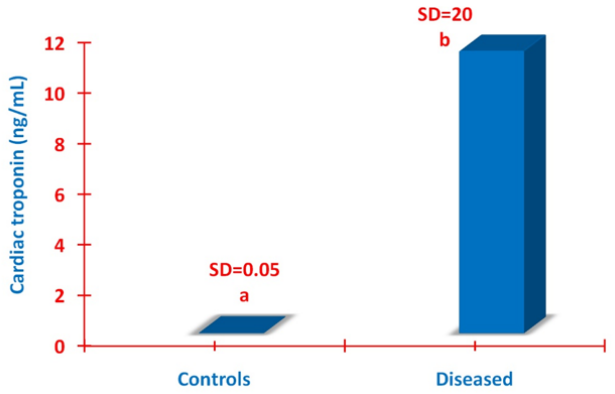

Fig. 5. Cardiac troponin I (cTnI) in goats with cardiac nutritional muscular dystrophy versus healthy controls. [a,b] different letters indicate a significant difference (P<0.05) **(Tharwat et al., 2013e).**

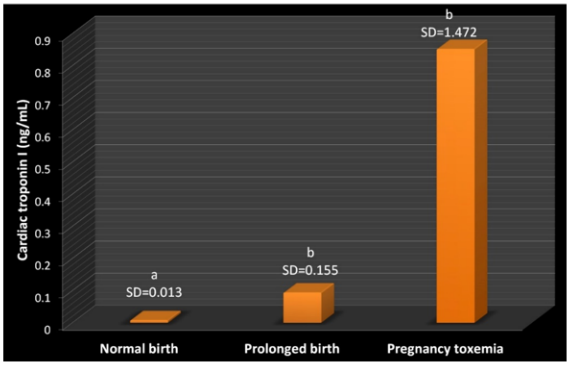

Fig. 6. Comparative mean ± standard deviation (SD) of cardiac troponin I in goats with normal birth, prolonged birth and in those with pregnancy toxemia. [a,b] different letters indicate a significant difference (P<0.05) **(Tharwat *et al.*, 2012).**

The level of cTnI was reported to be high in animals with long-standing chronic conditions. In dromedary camels with Downer syndrome, the serum concentration of cTnI was 10-fold higher than that in controls (0.01–2.20 ng/ml vs..00–0.08 ng/ml; P≤0.05), revealing a severe degree of myocardial injury (**Fig. 7**) (**Tharwat, 2012**). The level of cTnI was also reported to be significantly high in animals with external parasites producing toxins. A special example was reported in camels with tick infestation (1.7±1.6 ng/ml vs. 0.03±0.02 ng/ml in the healthy camels; P<0.0001), pointing out myocardial injury in those animals (**Tharwat and Al-Sobayil, 2014b**).

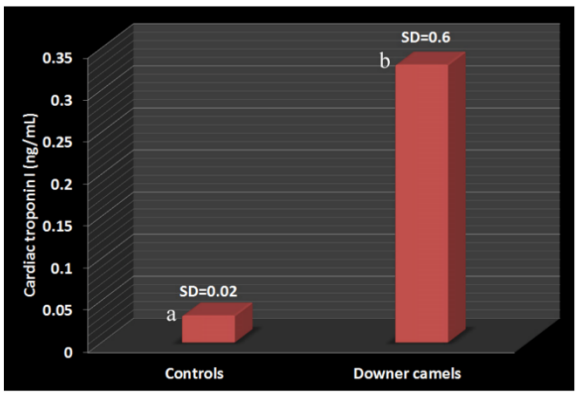

Fig. 7: Cardiac troponin I values in downer camels compared to control healthy camels. [a,b] Different letters indicate a significant difference (P=0.019). SD = standard deviation.

In the latter report, cTnI serum levels were used to predict the prognosis of camels with ticks, where the level in recovered camels was 0.36±0.53 ng/ml vs. 3.0±1.1 ng/ml in the camels that did not recover (**Fig. 8**) (**Tharwat and Al-Sobayil, 2014b**). The increases in cTnI in camels with tick infestation may be due to toxins present in tick saliva (**Wernery and Kaaden, 2002),** leading to damage to myocardium and skeletal muscles; this was proven by significant elevations in cTnI, creatine kinase and aspartate aminotransferase in the same study (**Tharwat and Al-Sobayil, 2014b**). The results of the latter study are supported by another with high cTnI levels in trials of experimentally induced myocardial damage (**O'Brien, 2006**). Furthermore, **Miranda *et al.*, (2006)** found that increased consumption of oxygen by myocardial muscles during long periods of increased heart rate is linked with a decreased supply of oxygen to the myocardium.

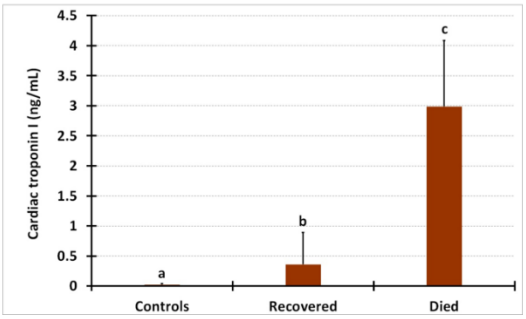

Fig. 8: Mean serum concentrations of serum cardiac troponin I in camels with tick infestation. [a,b,c] Different letters indicate a significant difference.

In experimental studies, induction of hypercalcemia in goats led to significant increases in the cTnI significantly during the 4 hours after calcium injection and peaked to 2.1 ng/mL 24 hours post-induction, then dropped gradually till the normal level 6 days after induction. Similar, induction of hypocalcemia in the goats resulted in a significant elevation in serum cTnI during the 4 hours after disodium EDTA injection but decreased at days 1 and 2 after induction and at normal levels thereafter (**Fig. 9**) (**Tharwat and Al-**

Sobayil, 2015). Contrary to this, injection of cardiac glycoside into donkeys did not reveal any significant differences in cTnI serum levels compared to pre-injection values (**Tharwat and Al-Sobayil, 2014a**). However, the effect of general anesthesia had adverse effects on cardiac functions and consequently on serum levels of cTnI.

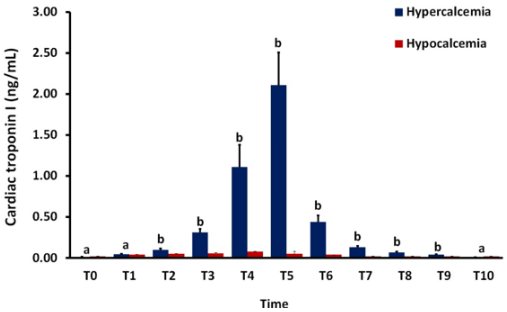

Fig. 9: Mean ±SD serum concentrations of cardiac troponin I in goats with experimentally induced hyper- and hypocalcemia. T0, immediately before calcium or EDTA injection; T1-T4 at 30, 60, 120 and 240 minutes after injection; T5-T10 at 1, 2, 3, 4, 5 and 6 days after injection (Tharwat and Al-Sobayil, 2015). [a,b] Differ significantly between goats with induced hyper- and hypocalcemia.

In a trial of halothane and isoflurane general anesthesia in camels, the cTnI level did not increase over 0.04 ng/ml in the isoflurane group. On the other hand, in camels anesthetized with halothane, cTnI values increased greatly 40 minutes and 80 minutes after recovery (0.2 ng/ml and 0.47 ng/ml, respectively); they also increased significantly at days 1 and 2 post-recovery. Serum concentrations of cTnI differed significantly between the halothane and isoflurane groups at 40 minutes, 80 minutes, 24 hours and 48 hours after recovery (**Fig. 10**) (**Tharwat *et al.*, 2013a**). The latter report concluded that the anesthetic halothane has an adverse effect on cardiomyocytes in comparison to isoflurane, and hence, halothane should not be used in camels suspected of having a cardiac disorder.

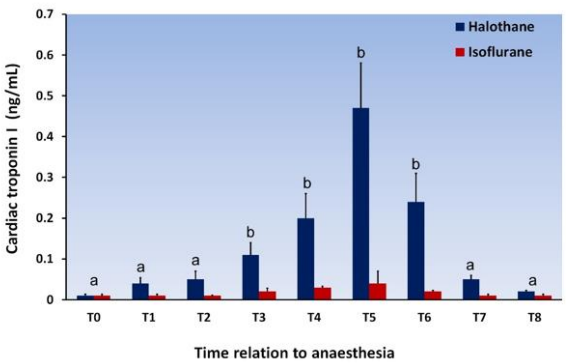

Fig. 10: Pre-anaesthetic, anaesthetic and post-anaesthetic serum concentration of cardiac troponin I (means ± SEM) in camels (n = 6) undergoing isoflurane and halothane anesthesia. T0, immediately before anesthesia; T1, 20 min after xylazine administration; T2, 20 minutes after ketamine administration; T3, 60 minutes during inhalation anesthesia; T4, 40 minutes of recovery; T5, 80 minutes of recovery; T6-T8, 24 hours, 48 hours and 72 hours after anesthesia (Tharwat et al., 2013a). [b]Differs significantly between the two anesthetic agents at P<0.05.

It was also reported that semen collection through stimulation by electroejaculation (EEJ) affects levels of cTnI. The concentration of serum cTnI increased significantly (P≤0.0001) in dromedary camels subjected to EEJ; the concentration declined to pre-experimental levels 24 h later. However, in the camel group with natural mating, serum levels of cTnI did not change significantly post-mating versus pre-mating levels (**Fig. 11**; **Tharwat *et al.*, 2014**). The significant increase of cTnI due to stimulation by EEJ suggested myocardial injury by this procedure; this explanation was supported by the non-significant differences in cTnI after natural mating in the same species.

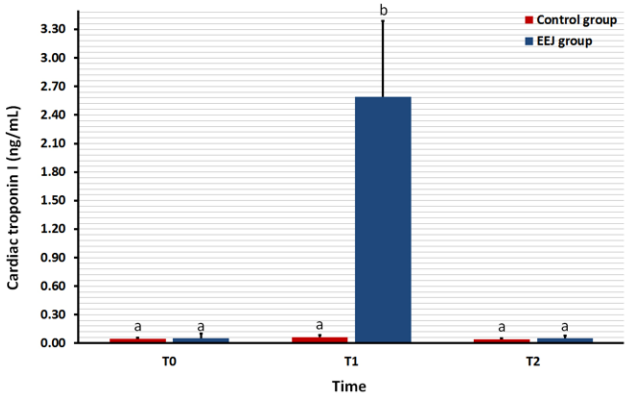

Fig. 11: Effect of stimulation by electroejaculation (EEJ) on cardiac troponin I in male dromedary camels compared with control group. T0: just before EEJ; T1: directly after EEJ; T2: 24 hours after EEJ **(Tharwat *et al.*, 2014).** [a,b]Values differ significantly (P=0.0001).

CONCLUSION

It is evident from various pathophysiologic reports that cTnI is a highly specific biomarker. It can detect myocardial damage due to primary cardiac diseases such as pericarditis, endocarditis and myocarditis. It can also detect cardiac injury in some non-cardiac states that can affect the heart, such as racing competitions, long road transportation, extensive training, difficult parturition, Downer syndrome, general anesthesia, infestation with external parasites such as ticks, calcium injection in extremely high doses, injection of cardiac glycosides, and use of EEJ to collect semen.

Conflict of Interest Statement

The authors declare that there is no conflict of interest.

REFERENCES

AL ABBASI, B., TORRES, P., RAMOS-TUAREZ, F., DEWASWALA, N., ABDALLAH, A., CHEN, K., ABDUL QADER, M., JOB, R., ABOULENAIN, S., DZIADKOWIEC, K., BHOPALWALA, H., PINO, J.E., and CHAIT, R.D., 2020. Cardiac Troponin-I and COVID-19: A Prognostic Tool for In-Hospital Mortality. Cardiology Research. 11(6), 398-404. http://dx.doi/10.14740/cr1159.

ALI, J., KHAN, F.R., ULLAH, R., HASSAN, Z., KHATTAK, S., LAKHTA, G., ZAD, G.U.L. N., ULLAH, R., 2021. Cardiac Troponin I Levels in Hospitalized COVID-19 Patients as a Predictor of Severity and Outcome: A Retrospective Cohort Study. Cureus. 13(3), e14061. http://dx.doi/10.7759/cureus.14061

BUCZINSKI, S., and BÉLANGER, A.M., 2010. Bovine tricuspid endocarditis as a cause of increased serum concentration of cardiac troponins. Canadian Veterinary Journal. 51(2), 195-7.

JESUTHASAN, A., ALI, A., LEE, J.K.W., and RUTHERFURD-MARKWICK, K. 2022. Assessment of Changes in Physiological Markers in Different Body Fluids at Rest and after Exercise. Nutrients. 14(21), 4685. http://dx.doi/10.3390/nu14214685

JONKER, S.S., and LOUEY, S., 2024. Fetal cardiac troponin I levels decline toward birth in sheep. American Journal of Physiology-Heart and Circulatory Physiology. 326(6), H1538-H1543. http://dx.doi/10.1152/ajpheart.00224.2024

KARAPINAR, T., EROKSUZ, Y., BEYTUT, E., SOZDUTMAZ, I., EROKSUZ, H., and DABAK, M., 2012. Increased plasma cardiac troponin I concentration in lambs with myocarditis. Veterinary Clinical Pathology. 41(3), 375-81. http://dx.doi/10.1111/j.1939-165X.2012.00448.x

KATRUKHA, I.A., and KATRUKHA, A.G., 2021. Myocardial Injury and the Release of Troponins I and T in the Blood of Patients. Clinical Chemistry. 67(1), 124-130. http://dx.doi/10.1093/clinchem/hvaa281

KOURETI, G., 2022. Correlation of Troponin I Level in Patients with Acute Myocardial Infarction and its Prognostic Implication in Heart Failure. Journal of the Association of Physicians of India. 70(4), 11-12.

LA VECCHIA, L., MEZZENA, G., ZANOLLA, L., PACCANARO, M., VAROTTO, L., BONANNO, C., and OMETTO, R., 2000. Cardiac troponin I as diagnostic and prognostic marker in severe heart failure. Journal of Heart and Lung Transplantation. 19(7), 644-52. http://dx.doi/10.1016/s1053-2498(00)00120-0

LIPPI, G., LAVIE, C.J., SANCHIS-GOMAR, F., 2020. Cardiac troponin I in patients with coronavirus disease 2019 (COVID-19): Evidence from a meta-analysis. Progress in Cardiovascular Diseases. 63(3), 390-391. http://dx.doi/10.1016/j.pcad.2020.03.001

MALIK, P., PATEL, U., PATEL, N.H., SOMI, S., and SINGH, J., 2020. Elevated cardiac troponin I as a predictor of outcomes in COVID-19 hospitalizations: a meta-analysis. Infezioni in Medicina. 28(4), 500-506.

MCKENZIE, E.C., JOSE-CUNILLERAS, E., HINCHCLIFF, K.W., HOLBROOK, T.C., ROYER, C., PAYTON, M.E., WILLIAMSON, K., NELSON, S., WILLARD, M.D., and DAVIS, M.S., 2007. Serum chemistry alterations in Alaskan sled dogs during five successive days of prolonged endurance exercise. Journal of the American Veterinary Medical Association. 230(10), 1486-1492. http://dx.doi/10.2460/javma.230.10.1486

MELLANBY, R.J., HENRY, J.P., CASH, R., RICKETTS, S.W., BEXIGA, R., TRUYERS, I., and MELLOR, D.J., 2009. Serum cardiac troponin I concentrations in cattle with cardiac and noncardiac disorders. Journal of Veterinary Internal Medicine. 23(4), 926-930. http://dx.doi/10.1111/j.1939-1676.2009.0330.x

MIRANDA, R.C., MACHADO, M.N., TAKAKURA, I.T., DA MATA, P.F., DA FONSECA, C.G., MOUCO, O.M., HERNANDES, M.E., LEMOS, M.A., and MAIA, L.N., 2006. Elevated troponin levels after prolonged supraventricular tachycardia in patient with normal coronary angiography. Cardiology. 106(1), 10–13. http://dx.doi/10.1159/000092449

NOBRE PACÍFICO PEREIRA, K.H., HIBARU, V.Y., FUCHS, K.D.M., CRUZ, D.O.S., SANTOS CORREIA, L.E., LOPES, M.D., FERREIRA, J.C.P., FERREIRA, D.E., SOUZA, F., MACHADO, L.H.A., CHIACCHIO, S.B., and GOMES LOURENÇO, M.L., 2022. Use of cardiac troponin I (cTnI) levels to diagnose severe hypoxia and myocardial injury induced by perinatal asphyxia in neonatal dogs. Theriogenology. 180:146-153. http://dx.doi/10.1016/j.theriogenology.2021.12.027

NOSTELL, K., and HAGGSTRÖM, J., 2008. Resting concentrations of cardiac troponin I in fit horses and effect of racing. Journal of Veterinary Cardiology. 10(2), 105–109. http://dx.doi/10.1016/j.jvc.2008.10.001

O'BRIEN, P.J., 2006. Blood cardiac troponin in toxic myocardial injury: archetype of a translational safety biomarker. Expert Review of Molecular Diagnostics. 6(5), 685-702. http://dx.doi/10.1586/14737159.6.5.685

OYAMA, M.A., 2013. Using cardiac biomarkers in veterinary practice. Vet Clin North Am Small Anim Pract. 43(6), 1261-1272, vi. http://dx.doi/10.1016/j.cvsm.2013.07.010

SALVATICI, M., BARBIERI, B., CIOFFI, S.M.G., MORENGHI, E., LEONE, F.P., MAURA, F., MORIELLO, G., and SANDRI, M.T., 2020. Association between cardiac troponin I and mortality in patients with COVID-19. Biomarkers. 25(8), 634-640. http://dx.doi/10.1080/1354750X.2020.1831609

SHI, S., QIN, M., SHEN, B., CAI, Y., LIU, T., YANG, F., GONG, W., LIU, X., LIANG, J., ZHAO, Q., HUANG, H., YANG, B., and HUANG, C., 2020. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. JAMA Cardiology. 5(7), 802-810. http://dx.doi/10.1001/jamacardio.2020.0950

THARWAT, M., 2023. Advanced biomarkers and its usage in Arabian camel medicine – a review, Journal of Applied Animal Research, 51(1), 350-357, http://dx.doi/10.1080/09712119.2023.2203749

THARWAT, M., ALI, A., AL-SOBAYIL, F., DERAR, R., and AL-HAWAS, A., 2014. Influence of stimulation by electroejaculation on myocardial function, acid-base and electrolyte status and haematobiochemical profiles in male dromedary camels. Theriogenology. 82(6), 800–806. http://dx.doi/10.1016/j.theriogenology.2014.06.023

THARWAT, M., ALI, A., DERAR, D., OIKAWA, S., and ALMUNDARIJ, T.I., 2024. Effects of dystocia on the cardiac biomarker troponin I, acid-base balance and blood gases alongside the hematobiochemical profiles in female dromedary camels. International Journal of Veterinary Science. 13(1): 115-119. https://doi.org/10.47278/journal.ijvs/2023.070

THARWAT, M., AL-SOBAYIL, F., and AHMED, A.F., 2013a. Effect of isoflurane and halothane on myocardial function in healthy dromedary camels as assessed by cardiac troponin I. Journal of Camel Practice and Research. 20(2), 289-294.

THARWAT, M., AL-SOBAYIL, F., AL-HAWAS, A., and BUCZINSKI, S., 2014. Increased serum concentration of cardiac troponin I in a Dorcas gazelle (*Gazella dorcas*) with mitral vegetation. Comparative Clinical Pathology. 23:469–473. https://doi.org/10.1007/s00580-013-1847-9

THARWAT, M., AL-SOBAYIL, F., and AL-SOBAYIL, K., 2012. The cardiac biomarkers troponin I and CK-MB in nonpregnant and pregnant goats, goats with normal birth, goats with prolonged birth, and goats with pregnancy toxemia. Theriogenology. 78(7), 1500–1507. https://doi.org/10.1016/j.theriogenology.2012.06.013

THARWAT, M., AL-SOBAYIL, F., and BUCZINSKI, S., 2013b. Influence of racing on the serum concentrations of the cardiac biomarker's troponin I and creatine kinase myocardial band (CK-MB) in racing greyhounds. Veterinary Journal. 197(3), 900–902. https://doi.org/10.1016/j.tvjl.2013.01.023

THARWAT, M., AL-SOBAYIL, F., and BUCZINSKI, S., 2013c. Cardiac biomarkers changes in camels (*Camelus dromedarius*) secondary to long road transportation. Journal of Veterinary Cardiology. 15(1), 15-22. https://doi.org/10.1016/j.jvc.2012.08.004

THARWAT, M., AL-SOBAYIL, F., and BUCZINSKI, S., 2013d. Effect of racing on the serum concentrations of cardiac troponin I and CK-MB in racing camels (*Camelus dromedarius*). Veterinary Research Communications. 37(2), 139–144. https://doi.org/10.1007/s11259-013-9556-z

THARWAT, M., AL-SOBAYIL, F., and EL-SAYED, M., 2013e. Cardiac troponin I in healthy newborn goat kids and in goat kids with cardiac nutritional muscular dystrophy. Acta Veterinaria Hungarica. 61(4), 442–453. https://doi.org/10.1556/AVet.2013.041

THARWAT, M., and AL-SOBAYIL, F., 2014a. Influence of the cardiac glycoside digoxin on cardiac troponin I, acid–base and electrolyte balance, and haematobiochemical profiles in healthy donkeys (*Equus asinus*). BVC Veterinary Research. 10, 64. https://doi.org/10.1186/1746-6148-10-64

THARWAT, M., and AL-SOBAYIL, F., 2014b. The effect of tick infestation on the serum concentrations of the cardiac biomarker troponin I, acid–base balance and haematobiochemical profiles in camels (*Camelus dromedarius*). Tropical Animal Health and Production. 46(1), 139–144. https://doi.org/10.1007/s11250-013-0464-6

THARWAT, M., and AL-SOBAYIL, F., 2015. Effect of experimentally induced hyper- and hypocalcaemia on myocardial function in goats as assessed by the serum concentration of cardiac troponin I. Global Veterinaria. 14(1), 124-128. https://doi.org/10.5829/idosi.gv.2015.14.01.9241

THARWAT, M., and AL-SOBAYIL, F., 2014c. Influence of transportation on serum concentration of the cardiac biomarkers troponin I and creatine kinase myocardial band in horses. Journal of Equine Veterinary Science. 34, 662–667. http://dx.doi.org/10.1016/j.jevs.2013.12.008

THARWAT, M., 2012. The cardiac biomarker troponin I and other hematological and biochemical variables in downer camels (*Camelus dromedarius*). Journal of Camel Practice and Research. 19(2), 123-128.

THARWAT, M., 2020. The cardiac biomarkers troponin I and creatine kinase myocardial band in camels (*Camelus dromedaries*) - review. Journal of Camel Practice and Research. 27(1), 121-128. http://dx.doi/10.5958/2277-8934.2020.00004.1

THARWAT, M., 2021. Influence of 8 km training on cardiac biomarkers alongside hematobiochemical profiles in race camels. Journal of Camel Practice and Research. 28(1), 79-84. http://dx.doi/10.5958/2277-8934.2021.00012.6

VAN DER VEKENS, N., DECLOEDT, A., VEN, S., DE CLERCQ, D., and VAN LOON, G., 2015. Cardiac troponin I as compared to troponin T for the detection of myocardial damage in horses. Journal of Veterinary Internal Medicine. 29(1), 348-54. http://dx.doi/10.1111/jvim.12530

WERNERY, U., and KAADEN, O.R., 2002. Infestations with ectoparasites. In: Infectious Diseases in Camelids. Blackwell Science, Berlin, pp. 312-342.

How to cite this article:
Mohamed Tharwat and Mohamed Marzok, 2024. Implementation of Cardiac Troponin I as A Sensitive Biomarker for Myocardial Injury in Animals: Review Article. Journal of Applied Veterinary Sciences, 9 (4): 10-16.**: https://dx.doi.org/10.21608/javs.2024.298632.1358**