Cardiac Troponin I Level in Multiple Traumatic Injuries and its Relation to type of Injury, site of Injury, and Postmortem Interval (PMI): A Cross Sectional Cadaveric Study in Babylon

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Abstract

Background: Cardiac troponins are regulatory proteins that govern the interaction between actin and myosin filaments in muscles, this interaction is mediated by calcium. Troponins form an integral part of the cardiac and skeletal muscles. High levels of Troponin I (cTnI) were reported to be common after trauma. Forensic investigation requires the use of more precise biochemical markers to diagnose acute myocardial infarction and other causes of death, particularly in cases involving trauma or violent fatalities. **Aim of the study:** This study is to measure level of troponin I in human cadavers who died after sever trauma, and to find its relation with type of injury, site of injury, postmortem interval (PMI), and cause of death. **Methodology and Participants:** A cross-sectional investigation was done on 50 cadavers, 33 males and 17 females. Ten ml of blood from each case were drawn directly from the heart. The samples of blood were centrifuged and the sera were submitted to chemical analysis. The cTnI and ELISA, test is utilized, employing the principle of a solid phase enzyme-linked immunosorbent assay. The spectrophotometric measurement of absorbance was conducted at a wavelength of 450 nm. Another 40-living people were also included in this study for measurement of cTnI level as control for dead people. Data processing was performed using SPSS statistical software. **Results:** Mean troponin level in males vs. females was 14.86ng/ml and 14.13ng/ml respectively. The difference between cadaveric troponin and living people. The results are statistically significant with a p-value of less than 0.0001. The highest levels of troponin were observed in bullet injuries; 19.44ng/l. and in trauma to thorax 16.38ng/l. The level of troponin showed increase with elapse of time, but this increase was not steady; no linear relationship. Before 12 hours the level of troponin reached was 10.4ng\\l and peaked at 48 hours to reach 22045ng/ml. The age group with the greatest troponin level was 41–50 years old, with a concentration of 15.91ng/l. Similarly, live individuals had a troponin level of 3.19ng/l . **Conclusion:** Cardiac troponin the serum levels of deceased individuals showed a marked elevation compared to those in healthy populations. Serum troponin level is elevated in all kind of trauma however; it is significantly higher in deaths due to chest trauma compared to other sites of trauma. The levels of troponin showed marked increase after 12 hours of death.

Key Words: Postmortem Cardiac Troponin, Traumatic Injuries, Site of Injury, Type of Injury, Postmortem Interval.

Introduction

Three components make up the protein molecule known as troponin that are responsible for regulation of the contraction of skeletal and cardiac muscles. It is an integral part of these muscles. Troponin consists of three subunits: troponin C, troponin I, and troponin T **(Hassoni Mahdi, et al., 2024)**. The bloodstream typically contains minimal concentrations of the cardiac-specific troponins, known as cTnI and cTnT. Injury to the heart muscle cells releases cardiac troponins

I and T into circulation **(Kitzman, D. W., et al.,1988)**. As the level of damage increases, there is a corresponding increase in the concentration of cardiac troponins I and T in the bloodstream. After experiencing a heart attack, the levels of troponins in the blood may increase within 3 or 4

hours and stay elevated for a period of 10 to 14 days **(Abdelzaher, M. A., 2024)**.

Elevate levels of Troponin I (cTnI) have been reported to be common after trauma **(Sjögren, A. L. 1971)**. Diagnosing blunt cardiac injury in cases with multiple traumatic injuries might be challenging due to the presence of distracting wounds that result from high-velocity chest trauma **(Marcomichelakis, J., et al., 1983)**. Troponin elevation has been observed in as many as 30% of critically injured patients, and the cTnI level of trauma patients gradually increased after admission and peaked at 48 hours **(Gardin, J. M., et al., 1997)**.

Most research investigating the function of troponin in trauma has mostly concentrated on blunt thoracic trauma, namely blunt cardiac injury **(Salton, C. J., et al., 2002)**. Recent research suggests that the level of physiological stress rather than the physical impact on the chest may be more closely associated with the increase in troponin levels following trauma **(Wooten, S. V., et al., 2021)**.

Forensic investigation requires the use of more precise biochemical markers to diagnose acute myocardial infarction and other causes of death, particularly in cases involving trauma or violent fatalities. Cardiac troponin is one of the biomarkers used to detect necrosis. Is the preferred biomarker for the detection of myocardial infarction and injury to other skeletal muscles **(Bjerring, A. W., et al., 2018, Redfield, M. et al, 2005, Finocchiaro, G., et al., 2024)**? Clinical investigations have thoroughly demonstrated the superiority of cardiac troponin compared to other indicators of necrosis. Testing for cardiac troponin is more accurate in detecting myocardial damage when there is also a skeletal muscle injury present. Furthermore, it provides superior differentiation of heart muscle damage when the CK-MB level is normal or very slightly elevated **(Fitzgibbons, T. P., 2023)**.

This study aims to quantify the concentration of troponin I in the sera of cadavers who died after sustaining sever trauma as marker for diagnosis of traumatic non cardiac patients and to find its relation to type of injury, site sit of injury, and postmortem interval (PMI) and cause of death.

Methodology and Participants

A cross-sectional investigation was done 50 cadavers, 33 males and 17 females, referred to the dissection hall of the Institute of Forensic Medicine in Baghdad, for the period between 12th February 2019 and 1st May 2019. The cases were collected from cadavers referred to the Institute after fatal trauma. The inclusion criteria were:

(1) The postmortem interval is the time between death and the start of the autopsy should be less than 6 hours.

(2) there was no history of heart or coronary diseases.

(3) no pulmonary disease

(4) no history of hypertension, diabetes, and intracranial disease.

Data on the general demographic features; type of injury, site of injury, and cTnI, levels were collected. For measurement of cTnI, ten ml of blood for each case were drawn directly from the heart by mean of disposable syringe, and then each sample was placed in a tube containing anticoagulant. The samples were centrifuged and the serum was transferred to another test tube. Both cTnI, ELISA test is utilized, employing the principle of a solid-phase enzyme-linked immunosorbent assay. We directly correlated the level of troponin cTnI, with the observed color intensity in the test sample. The absorbance was quantified using spectrophotometry at a wavelength of 450 nm. Another sample of cases included in this study which consisted of 40 normal living people from whom samples of blood were collected as control for the ante mortem level of troponin I .

Data processing was conducted using SPSS statistical software (**version 22.0; IBM Corp., USA**). Data are presented as mean ± standard deviation. Descriptive statistics (frequency table, percentage and figures) the study employed inferential statistics, specifically the independent t-test, ANOVA, and Pearson correlation coefficient. We deemed a P-value less than 0.05 as statistically significant.

Results

The average troponin level in males was 14.86 ng/ml, whereas in females it was 14.13 ng/ml. There was no discernible disparity in the average troponin levels between males and females (Independent t-test: t -0.170, df 48, sig 0.866). Table (1), shows the distribution of cases based on age, gender, and average troponin level. The age group with the greatest number of instances was

identified 31-40 and 41-50 years, the highest level was in the same group; 15.91ng/l. Table (2), shows the distribution of cases based on the cause of death, gender, and age. Road traffic incidents accounted for the biggest number of fatalities. The majority of cases were males. Moreover, Table (3), Shows distribution according to time after death and level of troponin. The highest levels were observed after 12 hours of death and the lowest were before 2 hours. Tables (4-7), shows troponin level in various causes of death (n=50).

Table 1: Displays the distribution of cases based on age, gender, and average troponin level. The age group with the greatest number of instances was identified 31-40 and 41-50 years, the highest level was in the same group; 15.91ng\l.

Age	No. of cases	Gender		Level of troponin Mean ng/l
		Male	female	
1-10	4	3	1	11.835
11-20	10	7	3	11.65
21-30	7	3	4	10.98
31-40	12	7	5	10.22
41-50	11	8	3	15.91
51-60	5	4	1	10.74
61-70	1	1	0	>15

Table 2: Displays the distribution of cases based on the cause of death, gender, and age. Road traffic incidents accounted for the biggest number of fatalities. The majority of cases were males

Cause of death	Number of cases	Percent% from total	
RTA	28	56%	
Gunshot	11	22%	
Blunt Injury	6	12%	
Sudden death	5	10%	
Total	50	100%	
Mean age	32.28 years	No. Male	33 (66%)
		No. Female	17 (34%)

Table 3: Shows distribution according to time after death and level of troponin. The highest levels were observed after 12 hours of death and th lowest were before 2 hours.

Time after death	No. of cases	Level of troponin ng/l
1-12	11	10.36
13-24	24	11.73
25-36	13	12.96
36-48	2	22.45

Table4: Mean troponin level in various causes of death (n=50).

Causes of death	N	Mean troponin level (ng/ml)	Std. Deviation
RTA	28	15.04*	14.16
Gunshot	11	19.44	17.30
Blunt injuries	6	11.49	18.54
Sudden death	5	5.73	3.28
Total	50	14.61	14.25
* There is no statistically significant change in the average level of troponin between most causes of death RTA and gunshot (Independent T-test: t -0.819, df 37, Sig. 0.418). ** ANOVA test not applicable due to small sample size.			

Table 5: level of troponin according to site of trauma (n=50). The highest level of troponin was observed in trauma to the chest. The difference in levels in in different causes was not significant. ANOVA: F0.364 df 3, Sig.0.792.

Site of trauma	N*	Mean troponin level (ng/ml)	Std. Deviation
Abdomen	10	11.22	13.18
Thorax	5	16.38	19.13
Extremities	22	11.89	17.75
Head injuries	13	14.22	11.80
Total	50	14.61	14.25
ANOVA: F 0.346, df 3, Sig. 0.792			

Table 6: Shows mean level of troponin and site of trauma; the highest level of troponin was observed in ches trauma

Site of trauma	Number of cases	Mean level of troponin; ng//l
Head injuries	19	11.95
Abdomen	10	10.87
Thorax	10	14.14
Extremities	11	11.89

Table 7: Shows mean level of troponin and age in living peoples who unstained no injury. The highest levels were observed in age group 41-50 years

Age group	Number of cases	Mean of troponin (40 cases)
1-10	4	0.23
11-20	10	0.338
21-30	7	0.371
31-40	12	0.276
41-50	11	3.16
51-60	5	0.498
61-70	1	0.29

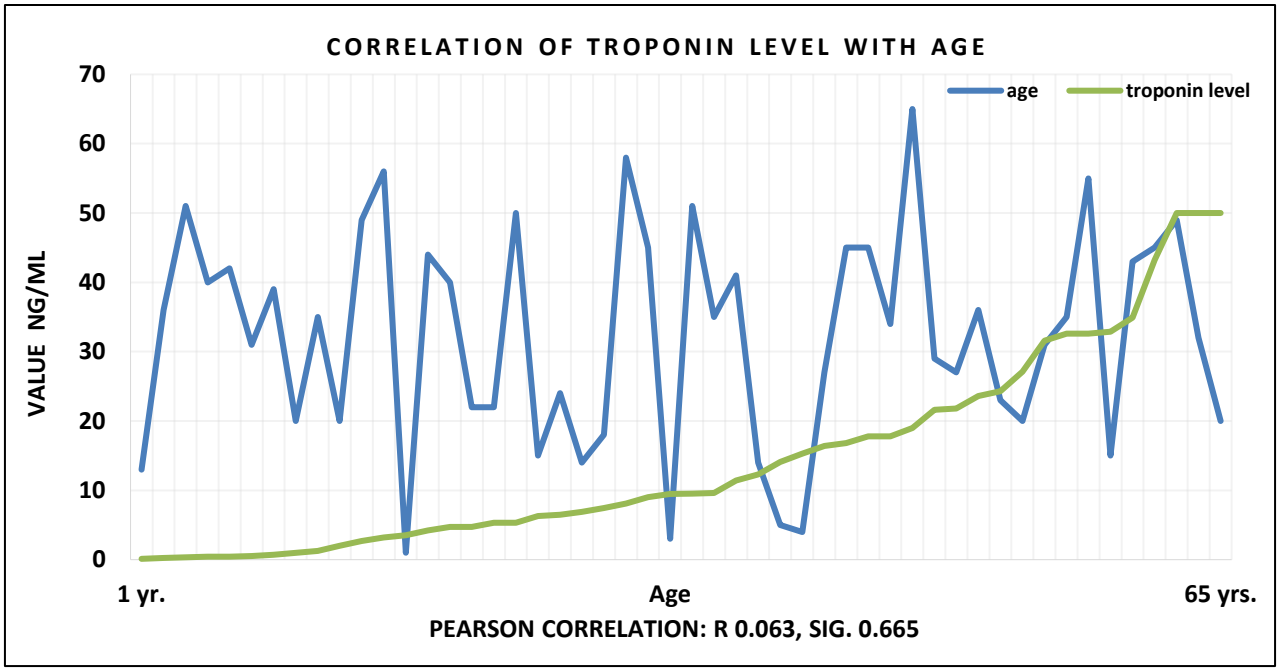

Figure 1: Correlation of troponin level (ng/ml) with age (yrs.) in cadavers (**n=50**).

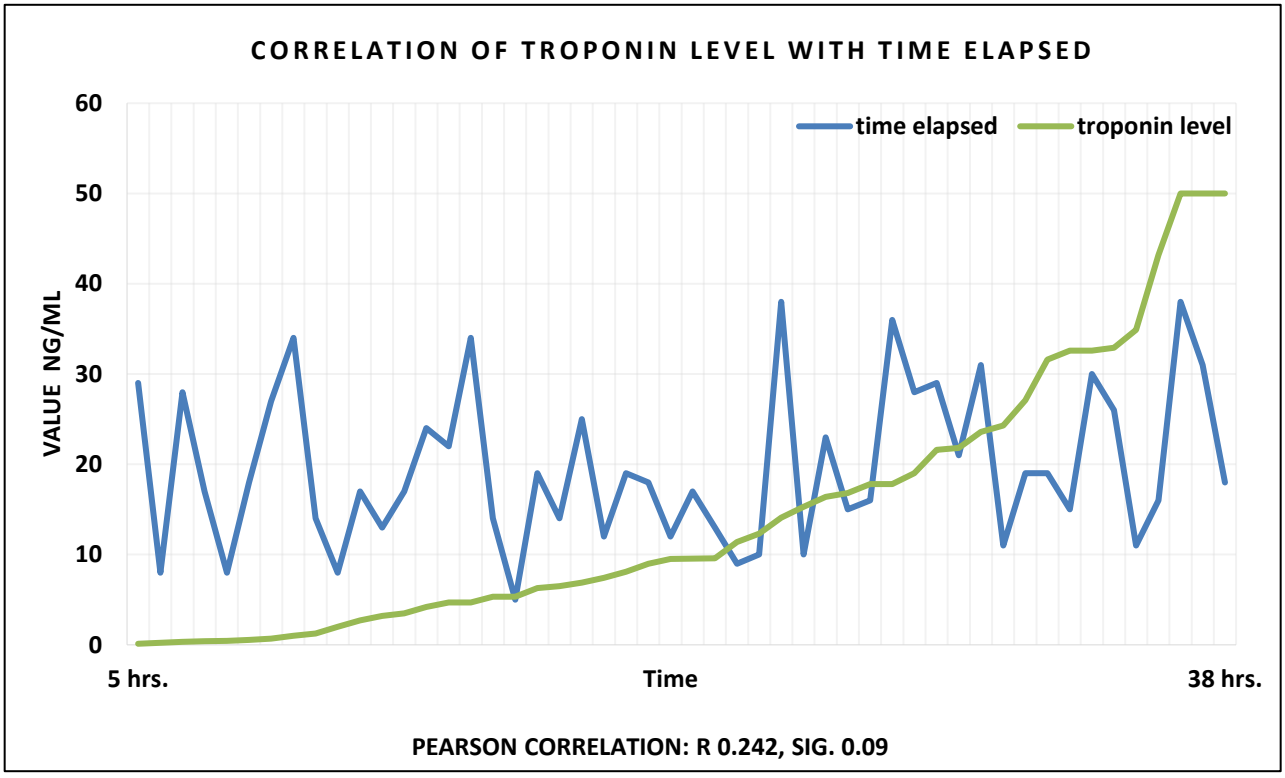

Figure 2: Correlation of troponin level (ng/ml) with time elapsed (hrs.) in dead (**n=50**).

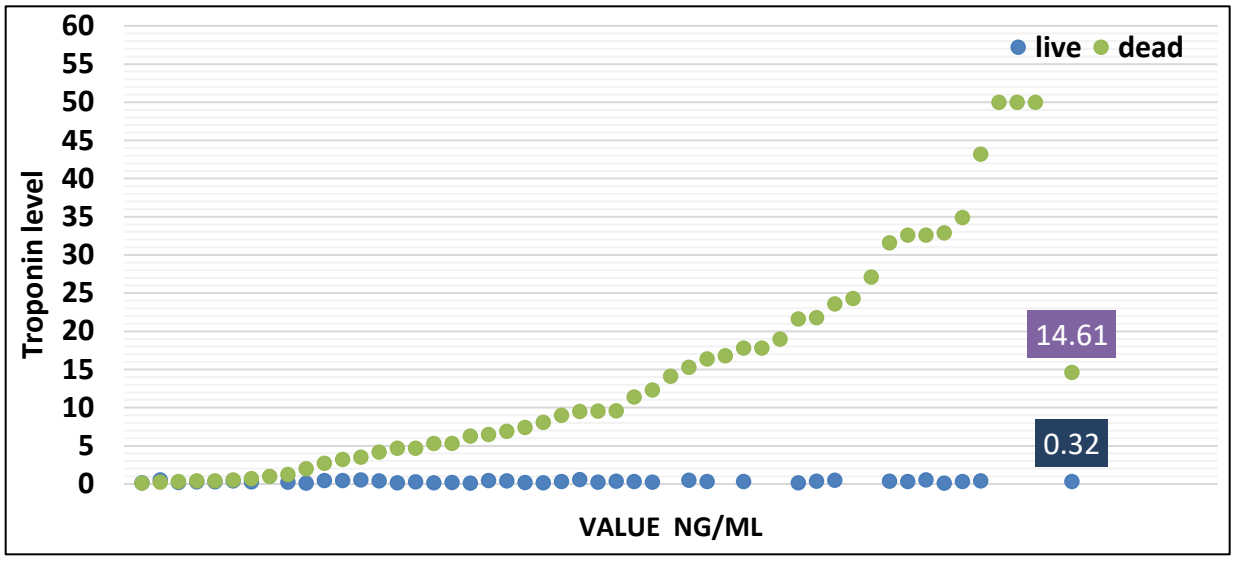

Figure 3: Mean troponin level: living person 0.32 ng/ml, SD+ 0.1. mean troponin level in dead persons 14.61 ng/ml, SD +14.25. Independent T-test: t 7.087, df 50, sig. 0.0001

Discussion

This study showed that the mean troponin (cTn1) level in cadaveric blood was significantly higher than that in living persons. It was 14.61ng/ml, SD 14.25 in dead persons, while in living it was 0.32ng/ml, SD 0.13. Independent T-test: t 7.087, def. 50, sig. 0.0001(Figure 3). This difference can be explained by the liberation of large amounts of troponin after sever cell destruction following trauma especially that involved multiple sites of the body. The result of this study showed that about, in 50% of the cases, The cause of death was a Road Traffic Accident (RTA), (Table 4). These results are in line with many studies which reported that RTA is the leading cause of accidental deaths, constituting about 76% of accidental death all over the world. **(Kajstura, J., et al., 2010, Mohammadi, S., et al., 2016, Tanna, J. A., et al., 2011)**.

The current investigation found no statistically significant association between the level of troponin I and age, this result is in line with other study **(Garland, J., et al., 2023, Marcomichelakis, J., et al.,1983)**. It has been reported that Cardiac troponin the link between age and cardiovascular risk is not biologically equal for individuals with different genetic backgrounds, denoted as I and T **(al'Absi, M., Devereux, R. B., et al., 2006)**. The present study showed no correlation between sex and level of troponin I. These results are in disagreement with other study; it was reported that Studies have shown that women have lower cardiac troponin I and T levels than males.

Furthermore, women showed a stronger correlation between these levels and cardiovascular events compared to men **(Petersen, S. E., Aung, N., et al., 2016, Krumholz, H. M., et al., 1993)**. The discrepancy in findings can be attributed to the fact that one study focused on a certain group of deceased patients, while the second study examined live individuals.

The current investigation revealed a progressive rise in troponin levels following death, however this increase was not steady (Figure 2). Troponin I level reached the highest values in 48 hours after death (22,57ng/ml which is significantly higher than its level in the first 12 hours after death (10,4ng/ml), that is about double (Table 3). However, Statistical research indicated a little positive association between troponin levels and the time that has passed since death, which means that the rise in troponin level after death is not regular. This may be explained by the fact that the cadaver after 24 hours under goes decomposition which does not occur in all the organs of the body at the same time or at regular time intervals. Therefore, the high level of troponin after 48 hours represents the summation of all troponin released from all decomposed organs after that time. The current study aligns with previous research. According to the findings, various factors, such as the duration between death and examination and the extent

of damage at the moment of death, can influence post-mortem troponin readings **(Goble, M. M., Mosteller, M., et al., 1992)**.

Comparison of ante-mortem troponin to post-mortem troponin showed marked disproportionate rise in troponin level with post-mortem interval (PMIs) and the value of troponin. It was reported that, the level of troponin in 84 hours after death was significantly higher than that before 12 hours after death **(Redfield, M. M., Jacobsen, S. J., et al., 2005)**. These findings are in line with the result of the present study: the level of troponin before 12 hours was 10.36ng/l, while after 48 hours it was 22.45ng/l.

Inquiring about the correlation between troponin levels and the cause of death (Table 4) the present study shows that the highest levels were recorded in cases died after firearm injuries (19.4ng/ml) and the lowest (5.7ng/ml) were in cases died suddenly. This may be attributed to the large amount of troponin released from the injured muscles to the blood after sever muscle destruction, which is usually sever and massive in polytrauma. While in sudden death there is no time for injured muscle to release troponin, which usually occurs after muscle autolysis. The troponin detected in blood after sudden death may be due to MI those results in sudden death. In this study the difference in mean level of troponin between sudden death and other causes is highly significant, Nevertheless, the multivariate statistical analysis of the result (ANOVA) indicated that there were no significant variations in troponin levels across different sources other than sudden death. Previous studies have shown increased level of troponin in various blunt and open traumatic injuries **(Bairey Merz, C. N., et al., 2020)**. Recent data suggest that the level of physiological stress experienced rather than the mechanical impact on the chest may primarily influence the increase in troponin levels in trauma cases **(Bairey Merz, C. N., et al., 2017)**. There is a scarcity of literature on the correlation between traumatic brain injury (TBI) and elevated levels of troponin .

Regarding the level of postmortem troponin I in relation to the site of trauma, the present study showed that the highest level of troponin was detected in trauma involved the thorax (table 6). For the chest a possible cardiac contusion may be associated with release of troponin I **(Ji, H., Kim, A., Ebinger, J. E., et al., 2020, Kararigas, G., Dworatzek, et al., 2014, Westaby, J. D., Zullo, et al., 2023)**. In a premortal study on patients who sustained severe multiple traumas it was found that A prolonged and substantial discharge of troponin was observed. In 82% of cases, I was commonly associated with chest trauma and consistently linked to electrocardiographic abnormalities **(Pfaffenberger, S., Bartko, P., et al., 2013)**. In another study on post traumatic troponin level, it was reported that the All-trauma types, with the exception of abdominal trauma, showed elevated levels of cTnI, **(Pelà, G., Crocamo, A., et al., 2016, Jesmin, S., Hattori, Y., et al., 2005)**. The results of this study are in line with our results.

Conclusion

The results of the present study showed that the measurement of cardiac troponin levels in the blood after death. The levels of I were markedly elevated in cadavers compared to those in normal populations. Serum troponin level is elevated in all kind of trauma however; it is significantly higher in death due to chest trauma compared to other sites of trauma .The levels of troponin showed marked increase after 12 hours of death. But the increase was disproportional, there is no positive correlation between postmortem time interval and troponin I levels. Levels of postmortem troponin higher than 20ng\l may indicate that the PMI is more than 24 hours.

Conflict of Interest

The authors declare no conflict of interest.

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