POSTMORTEM ASSESSMENT OF CARDIAC TROPONIN-T (TROPOMYCIN) AND CARDIAC TROPONIN-I (INHIBITOR) LEVELS TO DIAGNOSE MYOCARDIAL INFARCTION IN VICTIMS OF SUDDEN CARDIAC DEATHS

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ABSTRACT

Background: Sudden cardiac death (SCD), which is caused by acute myocardial infarction (AMI), represents a considerable percentage of autopsy cases. In many situations in forensic medicine, the diagnosis of acute myocardial infarction based only on pathological findings is difficult, so the determination of biochemical markers in postmortem biological fluids may be of significant value. **Objective:** The present study has aimed to throw light upon the role of postmortem assessment of cardiac troponin-T and cardiac troponin-I levels in the diagnosis of AMI in SCDs. **Methodology:** This is a prospective study that was conducted on 50 deceased (who fulfilled inclusion criteria) of sudden death and a control group of 20 cases consisting of deaths due to causes other than sudden death, including poisoning, falling from height, and stab wounds, that were admitted to the Forensic Medicine Authority, Alexandria Department, from January 2018 to March 2019 for comparison. At the time of autopsy, hearts were exposed, from which blood was obtained during internal examination of the thoracic cavity. About 10 ml of blood was obtained from the heart left chamber by a sterile syringe and was used for the analysis of cardiac troponin T and cardiac troponin I levels after centrifugation to obtain plasma. **Results**: All the assessed parameters showed significant correlation with SCDs. The sensitivity of cTnI and cTnT for prediction of SCD was 100% with a specificity of 85%. **Conclusion:** Evaluation of the biocardiac enzymes of cTnI and cTnT represents a highly sensitive marker in postmortem determnination of myocardial lesions in SCD.

Keywords: sudden cardiac death, myocardial lesion, biomarkers, cardiac enzymes.

INTRODUCTION

The incidence of sudden cardiac death (SCD) has been steadily rising worldwide, now accounting for annual deaths of over 17 million persons, which constitutes about a third of mortality globally. Identifying the cause of SCD is a priority for physicians handling these cases; however, it presents a significant challenge for forensic pathologists. Acute myocardial infarction (AMI) is globally recognized as one of the principal causes of death and a major threat to human life. In clinical settings, the diagnosis of AMI is facilitated by electrocardiograms and specific serum biochemical markers that detect myocardial damage **(Han and Flavin, 2014; Taniguchi et al., 2016).**

In forensic pathology, SCD is recognized as a frequent cause of acute mortality. While conventional pathological techniques can readily identify typical myocardial lesions, quantitatively assessing the severity of myocardial damage remains challenging. In cases of sudden death occurring at the very early stages of infarction, positive pathological evidence is often absent, necessitating a diagnosis based on the exclusion of other potential causes of death **(Yasuda et al., 2005)**.

The autopsy is well known to be limited in diagnosing mortality caused by ischaemic heart disease (IHD) and is well recognized. Much research has explored using biochemical analysis to detect levels

of cardiac troponins in postmortem bodies as a method to differentiate cardiac from non-cardiac mortality **(Davies et al., 2005).**

In medico-legal casework, assessing myocardial damage is a crucial aspect of postmortem investigations for various causes of death. Recent trends in applying biochemical methods to forensic pathology have shown significant interest in utilizing cardiac troponins (proteins specific to cardiac myofibrils) for postmortem examinations of IHD, which is mainly implicated in sudden death in several populations **(Zhu et al., 2006).**

Troponin is a complex protein that is located on the actin filament, regulating the actin-myosin interaction during muscle contraction. Troponin is constituted of three subunits: tropomyosin binding (cTnT), inhibitory (cTnI), and calcium binding (TnC). It is typically absent from the serum unless there has been necrosis of cardiac cells, making it highly specific to cardiac tissue. Current research has demonstrated that the levels of cardiac troponins act as a reliable marker of AMI **(Bheeshma et al., 2015).**

The current study aimed to evaluate the use of postmortem cardiac troponin T (cTnT) and cardiac troponin I (cTnI) levels for diagnoses of acute myocardial infarction in medicolegal autopsy and correlate them with the histopathological findings.

SUBJECTS & METHODS

A) Subjects:

This is a prospective study that was conducted on 50 deceased (who fulfilled inclusion criteria) of sudden death and a control group of 20 cases consisting of deaths due to causes other than sudden death, including poisoning, falling from height, and stab wounds, that were admitted to the Forensic Medicine Authority, Alexandria Department, from January 2018 to March 2019 after an official permission from the director of the Alexandria Medical Authority, scientific and ethical committee with ethical approval number; **Ms. 16.1.2018.**

Inclusion Criteria:

- Sudden death of an adult with the cause of death unexplained by external examination (body found deceased at home, outdoors, or in the hospital emergency room).
- Autopsy performed within 48 hours of death**.**

Exclusion Criteria:

- All bodies subjected to autopsy more than 48 hours after death.
- Bodies found to have septicaemia, renal disease, pulmonary embolism, myopericarditis, or congestive heart failure.
- Bodies that have undergone cardiac surgery, cardiopulmonary resuscitation, or experienced cardiac concussion.

B) Methods:

Autopsy was performed with a single midline incision that extended from the chin to the pubic bone, and then the skin, muscles, and soft tissue were peeled back, exposing the rib cage, which was pulled from the skeleton.

The abdomen was further opened by dissecting the abdominal muscles away from the bottom of the rib cage and diaphragm. The organs were removed individually, but before that, each organ was examined in situ for any gross pathology. For removal of the opening in, an incision was made from one ear to the other: through the vertex, the scalp was cut and then pulled forward and backward, and the open of the skull was removed by using a vibrating saw. Then the brain was exposed and examined in situ before removal. After removal of the body organs, each one was weighted. At the time of autopsy, hearts were exposed, from which blood was obtained during internal examination of the thoracic cavity. Approximately ten mililiters of blood were obtained from the heart's left chamber using a 21G sterile syringe and needle. The blood was then placed in a test tube and centrifuged immediately to obtain plasma at a frequency of 3,000 rpm. The obtained plasma was stored at 4°C for no more than 24 hours before being sent to

the department laboratory for analysis to measure levels of cardiac troponin T and cardiac troponin I.

A-Biochemical analysis:

Cardiac troponin T was measured using Elecsys Troponin T kits, and cardiac troponin I was measured using Elecsys Troponin I kits; both were measured using Cobas e 601 analyzers by the Sandwich principle, according to **Anderson (2013).**

B-Histopathological study:

After removal of the heart, a gross examination was done for any pathology, and then it was put in a container filled with 10% formalin. The containers were covered, labeled, and sent for histopathological examination. For histopathological studies, formalin-fixed paraffin 5 micrometer sections from the heart were stained with hematoxylin and eosin (H & E) according to **Lamberg & Rothstein (1978)** for examination by light microscope.

Statistical Analysis:

The collected data were organized, tabulated, and statistically analyzed using IBM SPSS statistical software, version 20.0 (Armonk, NY: IBM Corp). Categorial variables were expressed in numbers and percentages **(Kirkpatrick and Feeney, 2012)**, and the data normality was tested using Kolmogorov-Smirnov. Numerical data were presented in median, range (minimum-mmaximum), mean, and standard deviation. Student t-test, chi square (2), ANOVA (F-test), Fisher exact test, Monte Carlo (MC) correction, Kruskal-Wallis test, Spearman coefficient, Pearson correlation coefficients, and post hoc test. The accepted level of significance of the obtained results was stated at 0.05. Sensitivity and specificity were assessed.

a- **Sensitivity**

Sensitivity assesses the percentage of the correctly determined actual positive cases **(Tom, 2006).** In this study, we calculate sensitivity according to the following formula:

$$\text{Sensitivity} = \frac{\text{Total MI cases with positive cTnI test}}{\text{Total of all MI cases tested}}$$

b-Specificity

Specificity assesses the percentage of correctly determined actual negative cases ***(Tom, 2006*)**. In our study, we calculated sensitivity according to the following formula:

$$\text{Specificity} = \frac{\text{Negative cTnI test}}{\text{Total of all MI cases tested}}$$

RESULTS

This study included 70 cases who were divided into two groups as regard the cause of death: group (I) included 50 (71.4%) cases who had sudden cardiac death, and group (II) the control group included 20 (28.6) cases who died due to other causes such as poisoning [7 (10%) cases], falling from height [8 (11.42%) cases], and stab wound [5 (7.14%) cases] as shown in **Table 1 and fig. 1.**

The results of the current study illustrated a higher prevalence in the number and percentage of males among both the studied and control cases. However, the MI studied group experienced a statistically insignificant difference in the gender distribution as compared with the control group **(Table 2 and fig. 2).**

Data analysis among the MI studied group showed a higher prevalence of sudden cardiac deaths in the seventh- and eighth-decade age group (age between 60-70 and 70-80 years old), as shown in **Table 3.** The mean age of MI and the control group was 61.14 ± 12.79 (range 34.0 – 81.0years) and 33.15± 13.12 (range 16.0 – 57.0 years), respectively. Statistical analysis of the previous data showed significant differences in the mean age between the control group and the MI studied group **(Table 4).**

In our study, the biochemical analysis results of cTnI and cTnT documented a markedly significant increased level of cTnT and cTnI in MI cases compared to control. The mean values of cTnT and cTnI were [752.0 ±271.6 (range: 190.0 – 1310) and 1711.2 ± 1024.4 (range: 213.0 – 3519.0), respectively, among the MI group **(Table 5).**

The histopathological findings revealed that infarction was documented in the whole MI cases 50, where 43 of them (86%) had acute MI evidenced by wavy myocardiocytes that signify necrosis or cell death and inflammation caused by necrosis, which is characterized by the existence of several immune cells, particularly neutrophils, and 7 (14%) cases had old MI evidenced by replacing the necrotic myocardium with fibrous and vascular tissue**.** Whereas in the control group, old MI was evident only in 3 (15%) cases, with a significant difference between the two groups regarding prevalence or absence of MI **(Table 6 and fig. 3, 4 & 5).**

The findings of the current study showed that the sensitivity of both cTnT and cTnI for prediction of sudden cardiac death was 100% with a specificity of 85% **(Table 7).**

Considering studying the relation between cTnT levels and cardiac histopathology **(Table 8 and fig. 6)** and between cTnI levels and cardiac histopathology **(Table 9 and fig. 7)** in both the MI group and the control group, there was a significant difference in the mean values of cTnT regarding histological findings between both groups. Additionally, the results revealed a significant positive moderate correlation between both cTnT and cTnI in the MI group ($r_s = 0.591$, $p < 0.001$), meaning the correlation was insignificant in the control group, as shown in **Table 10.**

Declaring the postmortem interval (PMI), the present study results illustrated that PMI in the MI group ranged from 7 to 43 hours with a mean SD of 24.12 to 10.51, while the control group ranged from 8 to 41 hours with a mean SD of 24.05 to 9.09, with an insignificant difference between both groups as demonstrated in **Table 11.**

As regard correlation between PMI and both cTnT and cTnI levels, there was insignificant weak to fair positive correlation between PMI and cTnT levels in both MI groups and in the control group ($r = 0.058$ and $r = 0.225$, respectively). However, this correlation with cTnI was insignificantly weak positive with time interval in the MI group and control group ($r = 0.094$ and $r = 0.231$, respectively, as demonstrated in **Table 12.**

Table (1): Distribution of the studied cases according to cause of death.

Cause of death	Particular subtype	No.	%	Total No.	Total %
MI group	50	50	100%	50	100
Control group	Poisoning	7	35%	20	100
	Falling from	8	40%		
	Stab wound	5	25%		
Total		70	100.0	70	100.0

MI= Myocardial infraction

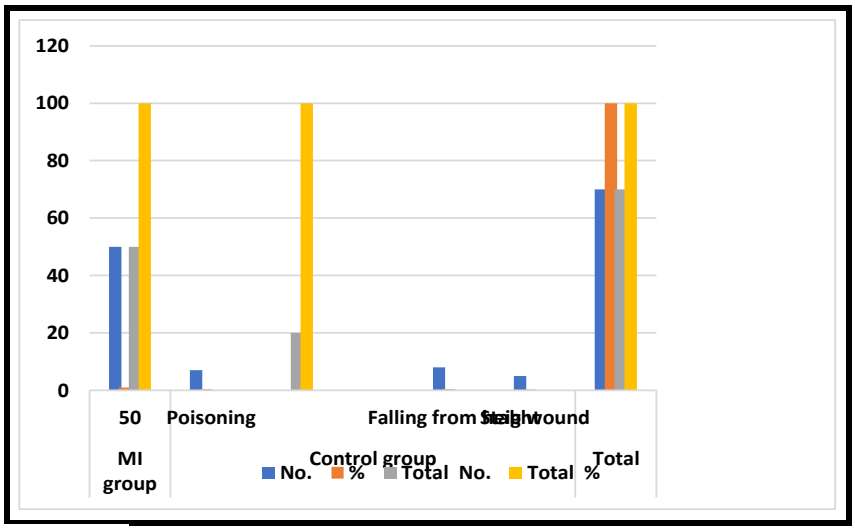

Figure (1): Distribution of the studied cases according to cause of death

Table (2): Comparison between the studied cases according to sex.

Sex	MI group (n = 50) No.	%	Control group (n = 20) No.	%	Test of Sig.	P value
Male	42	84.0	16	80.0	$\chi 2$=	FEp=
Female	8	16.0	4	20.0	0.161	0.732

p: p value for comparing between the studied groups, ***:** Statistically significant

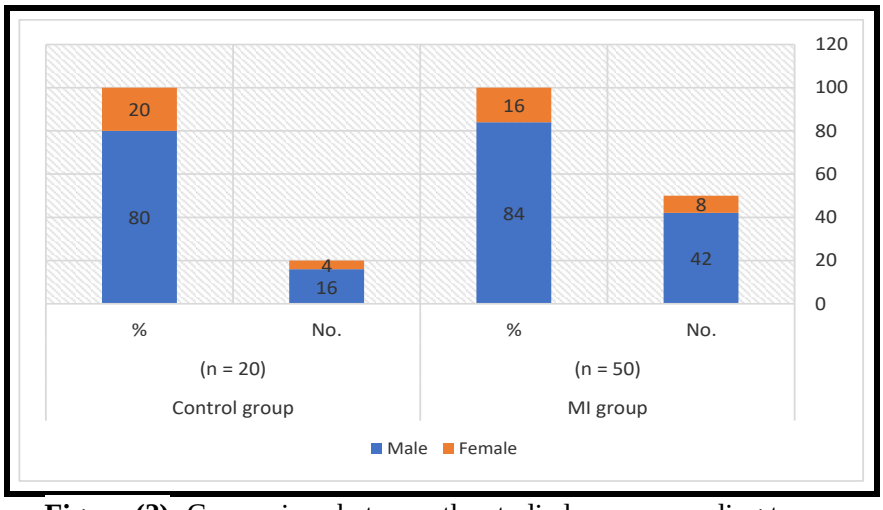

Figure (2): Comparison between the studied cases according to sex

Table (3): Comparison between MI studied and control cases according to different age groups.

Age (decade)	MI (n = 50)		Control (n = 20)		Test of Sig.	p
	No.	%	No.	%		
Frist	0	0.0	0	0.0		
Second	0	0.0	3	15.0		
Third	0	0.0	7	35.0		
Fourth	3	6.0	4	20..0	χ^2=	^{MC}p
Fifth	9	18.0	3	15.0	35.884*	<0.001*
Sixth	11	22.0	3	15.0		
Seventh	12	24.0	0	0.0		
Eighth	12	24.0	0	0.0		
Ninth	3	6.0	0	0.0		

χ2: Chi square test, **MC:** Monte Carlo, p: p value for comparing between the studied groups, *****: Statistically significant at p ≤ 0.05.

Table (4): Comparison between MI & control cases as regard to the mean age.

Age	MI (n = 50)	Control (n = 20)	Test of Sig.	p
Min. – Max.	34.0 – 81.0	16.0 –57.0		
Mean ± SD.	61.14 ± 12.79	33.15 ±13.12	8.210*	<0.001*
Median	63.0	30.0		

MI→ myocardial infraction, SD→standard deviation, Min→minimum, Max→maximum, *: Statistically significant at p ≤ 0.05, p: p value for comparing between the studied groups.

Table (5): Comparison between the two studied groups according to post mortem troponin level.

		MI (n = 50)	Control (n = 20)	U	p
cTn T	Min. – Max.	190.0 –1310.0	4.20 – 423.0		
	Mean ± SD.	752.0 ±271.6	72.74 ±125.6	11.50*	<0.001*
	Median	732.5	22.55		
cTn I	Min. – Max.	213.0 –3519.0	2.50 –345.0		
	Mean ± SD.	1711.2 ±1024.4	61.54 ±94.11	9.00*	<0.001*
	Median	1729.0	28.40		

U: Mann Whitney test, *: Statistically significant at p ≤ 0.05,

Table (6): Comparison of the two studied groups according to histopathological findings.

Histopathology	MI (n = 50)		Control (n = 20)		χ^2	^{MC}p
	No.	%	No.	%		
Normal myocardium histology	0	0.0	17	85.0		
Old MI	7	14.0	3	15.0	64.531*	<0.001*
Early MI	43	86.0	0	0.0		

χ2: Chi square test, **MC:** Monte Carlo, p: p value for comparing between the studied groups**.**

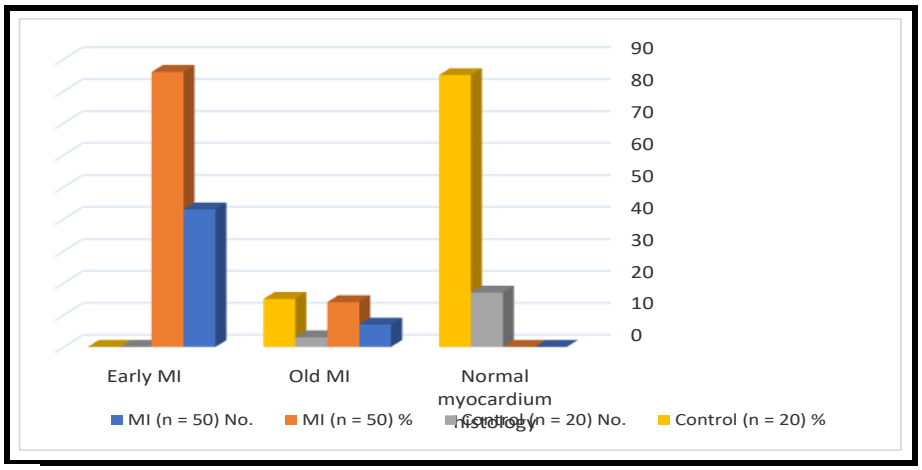

Figure (3): Comparison between the two studied groups according to histopathological findings.

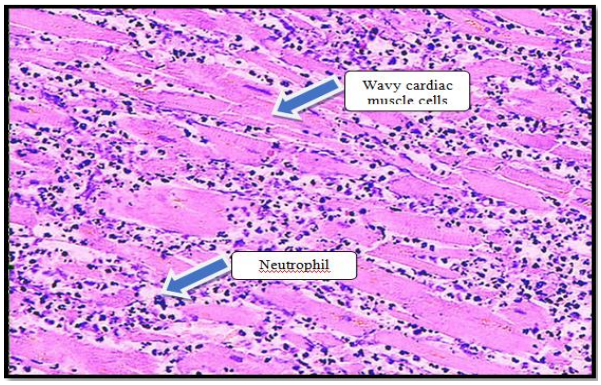

Figure (4): Acute myocardial infarction shows wavy cardiac muscle cells and immune cells, especially neutrophils.

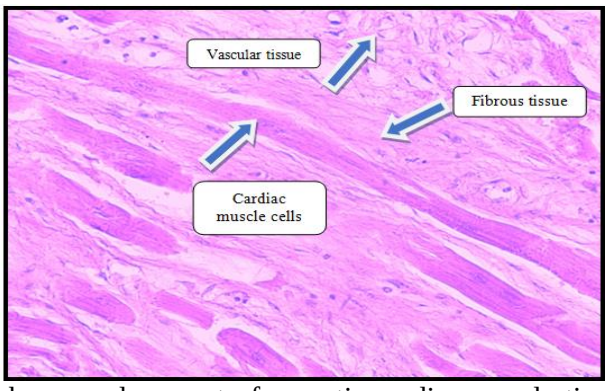

Figure (5): Old MI shows replacement of necrotic cardiac muscle tissue with vascular and fibrous tissue.

Table (7): Sensitivity and specificity of cTnT and cTnI in MI and control groups.

	cTnT test results		**cTnI test results**	
	MI	**Control**	**MI**	**Control**
Positive cTnT	50	3	50	3
Negative cTnT`	0	17	0	17
Total cases	50	20	50	20
Sensitivity	100%		100%	
Specificity		85%		85%

Table (8): Relation between cTnT and pathology among both MI & control group.

Pathology	N	cTnT Min. – Max.	Mean ± SD.	Median	U	p
MI (n = 50)						
Normal myocardium histology	**0**	–	–	–	54.0*	0.005*
Old MI	**7**	323.0– 1290.0	522.1±343.7	419.0		
Early MI	**43**	190.0–1310.0	789.4±242.8	790.0		
Control(n = 20)						
Normal myocardium histology	**17**	4.20–42.30	22.05±11.58	19.70	0.000*	0.002*
Old MI	**3**	312.0 – 423.0	360.0 ± 57.0	345.0		
Early MI	**0**	–	–	–		

U: Mann Whitney test, p: p value for association between **cTnT** and **Pathology** in each group**,** *: Statistically significant at $p \leq 0.05$

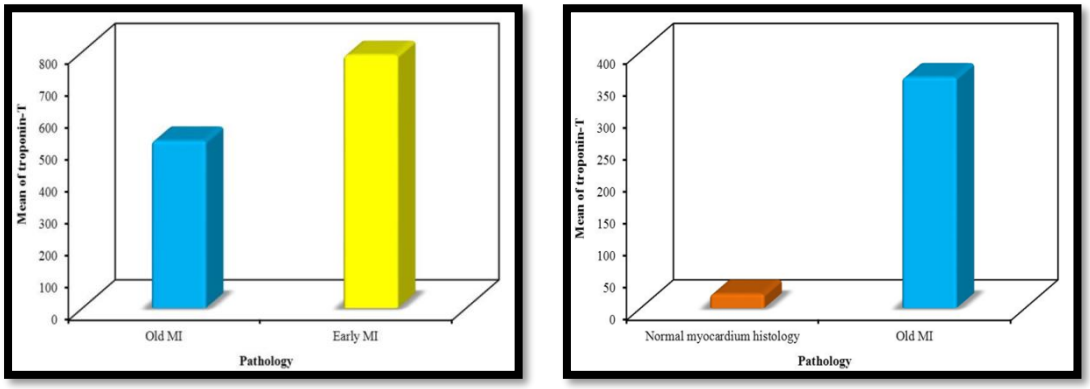

Figure (6): Relation between Troponin-T and pathology in MI group (n = 50) & control group (n = 20).

Table (9): Relation between Troponin-I and pathology among both MI & control group.

Pathology	N	cTnI Min. – Max.	Mean ± SD.	Median	U	p
MI (n = 50)						
Normal myocardium histology	**0**	–	–	–	50.0*	0.003*
Old MI	**7**	213.0– 3315.0	739.3±1137.7	312.0		
Early MI	**43**	280.0–3519.0	1869.5±925.4	1784.0		
Control(n = 20)						
Normal myocardium histology	**17**	2.50– 55.0	24.45±14.78	23.0	0.000*	0.002*
Old MI	**3**	214.0 – 345.0		256.0		
Early MI	**0**	–	–	–		

U: Mann Whitney test, p: p value for association between **Troponin-I** and **Pathology** in each group *: Statistically significant at $p \leq 0.05$.

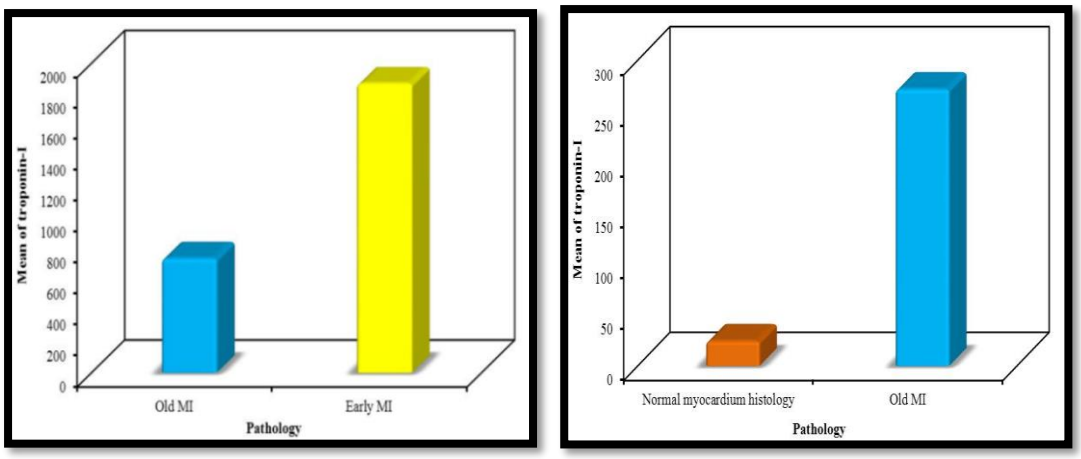

Figure (7): Relation between Troponin-I and pathology in MI group (n = 50) & control group (n = 20).

Table (10): Correlation between cTnT and cTnI among the studied cases.

Variables	cTnT			
	MI (n = 50)		Control (n = 20)	
	r_s	p	r_s	p
cTnI	0.591*	<0.001*	0.374	0.104

r_s: Spearman coefficient, *: Statistically significant at $p \leq 0.05$

Table (11): Comparison between the two studied groups according PMI.

PM Interval	MI (n = 50)	Control (n = 20)	t	p
Min. – Max.	7.0 –43.0	8.0 –41.0		
Mean ± SD.	24.12 ±10.51	24.05 ±9.09	0.026	0.979
Median	23.0	22.50		

t: Student t-test**, p: p** value for comparing between the studied groups

Table (12): Correlation between postmortem interval (PMI) and both cTnT and cTnI among the studied groups.

Variables	PM Interval			
	MI (n = 50)		Control (n = 20)	
	r	p	r	p
cTnT	0.058	0.687	0.225	0.340
cTnI	0.094	0.517	0.231	0.327

r: correlation coefficient (r 0-0.24 weak, r 0.25-0.49 fair, 0.50-0.74 moderate, >0.75 strong

DISCUSSION

Postmortem biochemical analysis of fluids and tissues has proven to be valuable in identifying the causes of death, particularly if the pathophysiologic alterations are undetectable through morphological methods **(Gonza´lez-Herrera et al., 2016).**

Most often, sudden death is caused by cardiovascular disorders (CVD), with IHD being the commonest. In about 80% of cases, the assumed cause of SCD is myocardial ischaemia, which is primarily associated with coronary atherosclerosis **(Michaud et al., 2014)**.

Sudden cardiac death is commonly encountered in forensic practice, significantly challenging forensic pathologists, particularly in cases of short-timeframe death that follows an ischaemic insult **(Sabatasso et al., 2018).** Most commonly, SCD happens shortly after the onset of ischaemic injury. Therefore, histolopatholgic investigation does not demonstrate reliable indicators of necrostic changes that manifest after a certain duration of individual survival **(Jasra et al., 2012).**

Cardiac troponin I (cTnI), being exclusively found in the myocardium, serves as a highly specific biomarker of myocardial injury. Postmortem serum and pericardial fluid levels of cTnI are significantly elevated in individuals who experienced acute myocardial ischaemia compared to control subjects **(Belsey & Flanagan, 2016).**

Data analysis of the current study among the MI group showed a higher prevalence of sudden cardiac death in the seventh- and eight-decade age group between 60-70 and 70-80 years and a higher prevalence in the number and percentage of male cases as compared to females with the MI studied group, which experienced a statistically insignificant difference in the gender distribution as compared with the control group. The results of this study revealed that infarction was evident by histopathology in all cases in the MI group, whereas in the control group, old MI was evident in 3 (15%) cases, with a significant difference between the two groups.

Regarding biochemical analysis, these results showed that cTnI and cTnT had significantly higher values in MI cases than control cases. These findings are in accordance with **Carvajal-Zarrabal et al. (2017),** who measured cTn-I and cTn-T in blood samples from the pulmonary veins of twenty dead bodies; those were clinically diagnosed to have SCD, and eight bodies with variable causes of violent death found that cTn-I and cTn-T levels were significantly elevated in cardiac versus noncardiac deaths. Further, **Peter et al.'s (2006)** study measured troponins cTnT and cTnI in twenty-five cases diagnosed with cardiac injury and eleven control cases without significant trauma to myocardium and revealed that there are significant differences in the cases with cardiac injury and those without for cardiac troponin T & I. In contrast, **Rahimi et al. (2018)** reported no significant difference in cTnT level measured in 140 cases with PMI less than 24 hours and had different causes of death ($p \geq 0.05$) such as CVD, thoracic or non-thoracic injury, sudden unexplained death, and others. This may have been attributed to microangiopathies and increased permeability of the myocardial cells; oxygen supply is reduced, and demand is increased. Also, **Bheeshma et al. (2015)** studied 12 cases with myocardial infarction and 6 control cases and showed that troponin T was elevated (>2.000 ng/ml) in all cases of sudden death except one case and in control cases except 2 cases; however, the p value was not studied for comparison, which disagrees with these results.

This study reported that the sensitivity of both cTnI and cTnT in prediction of sudden cardiac death is 100% with a specificity of 85%, which coincides with **Ikeda et al. (2002),** who reported that the sensitivity of cTnI and cTnT was 83.1% and 84.8%, respectively, and the specificity for cTnI and cTnT was 90.9% and 81.3%, respectively. Moreover, the study by **Bheeshma et al. (2015)**

revealed that the cTnT sensitivity and specificity were 91.66% and 66.66%, respectively, in the diagnosis of sudden cardiac death, which is close to the results of this study; however, **Rao et al. (2017)** reported that cTnT had a sensitivity of 92% and a specificity of 5%, which is much less than this study, as remarkable hypoxia or systemic hypovolemic states might cause coronary ischaemia, with subsequent elevation in the levels of troponin; also cardiopulmonary resuscitation could contribute to myocardial injury, leading to elevated cTnT levels, as mentioned by **Rao et al. (2017).**

The correlation between both troponins was significant, positive, and moderate in the MI group while the correlation was insignificant in the control group. This significant correlation may be due to the same kinetics of cTnT and cTnI, as they are released within 6 hours of a myocardial cellular lesion and persist in plasma for several hours. No studies have been done to investigate the correlation between both troponins for comparison.

Concerning the relation between cTnT and cTnI levels and pathology, the results showed that there was a significant difference between both in the MI group and the control group; this goes in line with **Bheeshma et al. (2015),** who concluded that there was an association between postmortem cTnT reactivity in death cases caused by MI with a significant correlation with histopathologic alterations. Another agreement with **Vanhaebost et al. (2017),** who showed that no cases had exclusive elevation of cTnT and cTnI levels without morphologic findings of myocardial ischaemic changes. **Khalifa et al. (2006)** also reported that all bodies with morphologically apparent myocardial infarction had increased cTnT levels, which agree with these results.

Regarding PMI, in the MI group it ranged from 7 to 43 hours, while the control group ranged from 8 to 41 hours, with a significant difference between both groups. Studying the correlation of cTnI and cTnT with PMI demonstrated insignificant weak to fair positive correlation in both groups. Increased troponin T and I levels with time interval occur as autolysis causes cellular membrane damage, which leads to leakage of troponin outside of cells, but this increase is insignificant ($p > 0.05$) as revealed by these results.

These results agree with **Peter et al. (2006),** who concluded that cardiac blood cTnI exhibited a nonsignificant positive correlation with the time of autolysis; also, **Vanhaebost et al. (2017)** proved that there were no statistically significant correlations between troponin T & I concentrations collected from peripheral blood (femoral vein) and postmortem interval. Contrary, these results disagree with **Gonza´lez-Herrera1 et al. (2016),** who studied cTnT levels in pericardial fluid and serum obtained from fifty-eight cases at defined postmortem intervals and concluded that cTnT showed stability in levels up to 34 hours after death.

Remmer et al. (2013), who studied postmortem cTnT in the serum and pericardial fluid of 101 autopsied cases with PMI of 8–141 h, reported a significant positive correlation between pericardial fluid and serum cTnT level and PMI, which disagrees with these results; this may be attributed to the long PMI of cases in their study.

CONCLUSIONS

In view of the previous results, it could be concluded that:

- To properly evaluate the cardiac lesions of persons who died suddenly, including cases where death involves myocardial infarction, you have to estimate both cTnI and cTnT. Using this procedure, it should be possible to rule out a cardiac cause of death with high accuracy, i.e., evaluation of the biocardiac enzymes of cTnI and cTnT represents a highly sensitive marker of myocardial lesion.
- Determination of cTnI and cTnT levels will be important to exclude false positive results of diagnosing cases of

sudden death by routine autopsy and histopathology study due to other causes than myocardial infarction.

- Both cTnI and cTnT have the same sensitivity and specificity for diagnosis of myocardial infarction in postmortem cases.

The findings from our study confirmed that cTnI and cTnT are sensitive markers in detection of MI at autopsy; however, they are less specific.

RECOMMENDATION

Based on the present study, the following recommendations were proposed:

- Taking a larger sample of population for better accuracy.
- Study of both cTnI and cTnT in blood from other diseases (electric shock, pulmonary oedema, renal failure) to decrease the false positivity.
- Further research is needed with more standardization of pre-postmortem variables. We acknowledge that the current work is a pilot study in such a scarcely addressed issue, paving the way for more elaborated studies.
- More collaboration between cardiac care units in hospitals and medical legal institutes for better follow-up of cases.

CONFLICT OF INTEREST

The authors declare that they have no competing interests

ETHICAL CONSIDERATIONS

- This study was reviewed and approved in accordance with the Research Ethical Committee Recommendations of the Faculty of Medicine, Banha University **(Ms. 16.1.2018).**
- Also, official permission was taken from the director of the Alexandria Medical Authority. Informed consent was taken from each legal guardian of the studied case. All personal data was kept anonymous to ensure confidentiality of the data studied parameters.

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الملخص العربى

تقييم ما بعد الوفاة لمستويات التروبونين القلبي (التروبومايسين) و التروبونين القلبي (المثبط) لتشخيص احتشاء عضلة القلب لدى ضحايا الوفيات القلبية المفاجئة

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الخلفية العلمية: الموت القلبي المفاجئ ، والذي يحدث بسبب احتشاء عضلة القلب الحاد ، يمثل نسبة كبيرة من حالات التشريح. في العديد من حالات الطب الشرعي، و يكون تشخيص احتشاء عضلة القلب الحاد استنادًا إلى النتائج المرضية فقط أمرًا صعبًا، وبالتالي فإن تحديد العلامات البيوكيميائية في السوائل البيولوجية بعد الوفاة قد يكون ذا قيمة كبيرة. **الهدف:** تهدف الدراسة الحالية إلى إلقاء الضوء على دور تقييم ما بعد الوفاة لمستويات التروبونين القلبي- التروبومايسين والتروبونين القلبي - المثبط في تشخيص احتشاء عضلة القلب الحاد في الموت القلبي المفاجئ. **المنهجية:** هذه دراسة استطلاعية أجريت على ٥٠ متوفى (الذين استوفوا معايير الاشتمال) من الموت المفاجئ ومجموعة مراقبة مكونة من ٢٠ حالة تتكون من وفيات لأسباب أخرى غير الموت المفاجئ، بما في ذلك التسمم، والسقوط من ارتفاع، والطعنات والتي تم قبولها بمصلحة الطب الشرعي قسم الإسكندرية خلال الفترة من يناير ٢٠١٨ إلى مارس ٢٠١٩ للمقارنة. في وقت تشريح الجثة، تم الكشف عن القلوب، والتي تم الحصول عليها من الدم أثناء الفحص الداخلي للتجويف الصدري. تم الحصول على حوالي ١٠ مل من الدم من غرفة القلب اليسرى بواسطة حقنة معقمة واستخدمت لتحليل مستويات التروبونين القلبي التروبومايسين والتروبونين القلبي المثبط بعد الطرد المركزي للحصول على البلازما. **النتائج:** أظهرت جميع المعلمات التي تم تقييمها وجود علاقة كبيرة مع الموت القلبي المفاجئ وكانت حساسية التروبونين القلبي- التروبومايسين والتروبونين القلبي - المثبط للتنبؤ بـ الموت القلبي المفاجئ 100% مع خصوصية ٨٥%. **الخلاصة:** إن تقييم إنزيمات القلب الحيوي (التروبونين القلبي- التروبومايسين والتروبونين القلبي – المثبط) يمثل علامة حساسة للغاية في تحديد إصابات عضلة القلب بعد الوفاة في حالات الموت القلبي المفاجئ.