

Postmortem Analysis of Cardiac Troponin I by High-Sensitivity Troponin I Assay

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ABSTRAK

Analisis troponin I postmortem dalam konteks autopsi boleh dijadikan sebagai alat yang bernilai untuk pakar patologi forensik. Kajian ini bertujuan untuk menentukan hubungan tahap troponin jantung postmortem dalam kematian berkaitan jantung, seperti infarksi miokardium akut (AMI), dan kes kematian berkaitan trauma menggunakan ujian troponin I jantung berkepekaan tinggi (hs-cTnI). Kajian keratan rentas retrospektif ini meneliti kematian medikolegal semula jadi dan tidak semula jadi dari Julai 2022 hingga Disember 2023. Laporan autopsi dikaji untuk mengumpul maklumat demografi, penemuan autopsi dan tahap troponin jantung. Tahap troponin jantung dianalisis menggunakan ujian hs-cTnI. Sebanyak 219 kes telah dianalisis, mendedahkan bahawa tahap median hs-cTnI lebih tinggi dalam kes kematian jantung (5914.80 pg/ml) berbanding kematian bukan jantung (1246.10 pg/ml). Perbezaan yang signifikan diperhatikan antara tahap median troponin jantung dalam kematian akibat AMI (12145.25 pg/ml) dan punca kematian yang lain. Analisis ciri operasi penerima menunjukkan kawasan di bawah lengkung sebanyak 0.723 untuk hs-cTnI dalam kematian akibat AMI. Selain itu, pelbagai jenis kematian berkaitan trauma juga menunjukkan perbezaan yang ketara dalam tahap troponin. Sebanyak 219 kes telah dianalisis, yang terdiri daripada 84 kematian jantung dan 135 kematian bukan jantung, termasuk 59 kes kematian berkaitan trauma. Tahap median hs-cTnI adalah lebih tinggi dengan ketara dalam kes kematian jantung (5914.80 pg/ml) berbanding kematian bukan jantung (1246.10 pg/ml) ($p < 0.01$). Perbezaan yang signifikan diperhatikan antara tahap median troponin jantung dalam kematian akibat AMI (12145.25 pg/ml) dan punca kematian lain. Analisis ciri operasi penerima menunjukkan kawasan di bawah lengkung sebanyak

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0.723 untuk hs-cTnI dalam kematian akibat AMI. Selain itu, pelbagai jenis kematian berkaitan trauma menunjukkan perbezaan yang ketara dalam tahap troponin. Walau bagaimanapun, kekurangan nilai rujukan yang ditetapkan memerlukan makmal individu untuk menetapkan penanda aras sendiri bagi menyokong pakar patologi forensik dalam ujian diagnostik pelengkap untuk kes postmortem. Kesimpulannya, hs-CTnI menawarkan ujian postmortem yang boleh dipercayai untuk membantu dalam mendiagnosis AMI.

Kata kunci: Biokimia postmortem; penyakit kardiovaskular; perubatan forensik; trauma; troponin I jantung kepekaan tinggi

ABSTRACT

Postmortem troponin I analysis in autopsy settings can be a valuable tool for forensic pathologists. This study aimed to determine the association of postmortem cardiac troponin levels in cardiac-related death, such as acute myocardial infarction (AMI), and traumatic-related death cases using high-sensitivity cardiac troponin I (hs-CTnI) assays. This retrospective cross-sectional study examined natural and unnatural medicolegal deaths from July 2022 to December 2023. Autopsy reports were reviewed to collect demographic information, autopsy findings, and cardiac troponin levels. The levels of cardiac troponin were analysed using high-sensitivity cardiac troponin assays. A total of 219 cases were analysed, revealing that the median level of hs-CTnI was higher in cardiac death cases (5914.80 pg/ml) compared to non-cardiac deaths (1246.10 pg/ml). Significant differences were noted between the median cardiac troponin level in deaths due to AMI (12145.25 pg/ml) and other causes of death. Receiver operating characteristic analysis showed an area under the curve of 0.723 for hs-cTnI in AMI deaths. Also, various types of traumatic deaths exhibited significant differences in troponin levels. A total of 219 cases were analysed, comprising 84 cardiac deaths, and 135 non-cardiac deaths, which included 59 traumatic death cases. The median level of hs-CTnI was significantly higher in cardiac death cases (5914.80 pg/ml) compared to non-cardiac deaths (1246.10 pg/ml) ($p < 0.01$). Significant differences were noted between the median cardiac troponin level in deaths due to AMI (12145.25 pg/ml) and other causes of death. Receiver operating characteristic analysis showed an area under the curve of 0.723 for hs-cTnI in AMI deaths. Also, various types of traumatic deaths exhibited significant differences in troponin levels. However, the lack of established reference values requires individual laboratories to establish their own benchmarks to support forensic pathologists in complementary diagnostic testing for postmortem cases. In conclusion, hs-CTnI offers reliable postmortem testing to assist in diagnosing AMI.

Keywords: High-sensitivity cardiac troponin I; cardiovascular disease; forensic medicine; postmortem biochemistry; trauma

INTRODUCTION

Autopsies in cases of sudden death are typically conducted within two distinct contexts, either the forensic autopsy or clinical autopsy. A clinical autopsy is performed at the request of the treating doctor or the family, while a forensic autopsy is initiated by legal authorities, such as a magistrate or coroner, depending on the country, to determine the cause of death. According to the Association for European Cardiovascular Pathology, one of the purposes of an autopsy is to ascertain whether the cause of death is cardiac-related or non-cardiac-related (Basso et al. 2017).

Forensic practitioners primarily perform autopsies, often revealing that coronary atherosclerosis or atheroma is the main cause of death (Milroy 2017). However, determining the cause of death can be challenging when gross or histological findings are insufficient or absent. In such cases, postmortem cardiac biomarker analysis is necessary to provide a reliable postmortem diagnosis in the absence of more sophisticated molecular methods (Batalis et al. 2010; Campuzano et al. 2014; Cao et al. 2019).

The commonly used cardiac biomarkers for investigating myocardial infarction include Creatine Kinase-MB (CK-MB) and cardiac troponins (cTn), such as cardiac Troponin I (cTnI) or T (cTnT). These proteins function as biomarkers of myocardial necrosis (Rahimi et al. 2018). However, cTn protein is preferred over CK-MB for diagnosing acute myocardial infarction (AMI) because numerous studies have established that cTn is more specific for detecting myocardial injury than CK-MB. CK-MB can produce more

false-positive results, as its levels increase in over 50% of acute muscular injuries and 80% of patients with chronic muscle disease (Trost & Feldman 2015).

In contrast, cTnI and cTnT are contractile proteins found exclusively in cardiac myocytes, offering high specificity and sensitivity for myocardial damage (Jacob & Khan 2018). The development of high-sensitivity cardiac troponin (hs-cTn) assays to detect the level of the cTn has been beneficial in stratifying acute coronary syndrome (ACS) cases, facilitating both early and late detection. The analyte of cTnI or cTnT is referred to as hs-cTnI or hs-cTnT, respectively, when analysed by using the hs-cTn assay (Thygesen et al. 2012). Multiple studies demonstrated that the detection of cTn by hs-cTn assays are more efficient than the conventional or former assays to detect the cTn level particularly in the early detection of AMI cases (Freund et al. 2011; Lin et al. 2022). Furthermore, hs-cTn assays have a low coefficient of variation, enhancing their ability to detect slight changes in cTn levels over different time frames (Saenger et al. 2011; Thygesen et al. 2012).

In addition to its role as a specific indicator used in diagnosing ACS, cTn has also proven valuable in diagnosing patients with blunt chest trauma. Interestingly, it has shown elevations in cTn levels up to 70% in identifying blunt cardiac trauma (Dou et al. 2022). Elevated cTn levels have also been observed in non-cardiac conditions such as chronic kidney disease, heterophile antibodies, skeletal myopathies and pulmonary hypertension (Hong et al. 2023).

Many studies have employed conventional cTn assays in the postmortem investigation of cardiac deaths, particularly

in cases suspected of AMI (Cao et al. 2019). Various study results have demonstrated the effectiveness of conventional cTn assays in diagnosing AMI in postmortem cases. However, there remains a need for more sensitive methods to diagnose early ischemic heart lesions in postmortem (González-Herrera et al. 2016).

Only a limited number of studies have utilised hs-cTn, most of which involve postmortem samples such as serum or pericardial fluids (Beausire et al. 2018; González-Herrera et al. 2016; Zribi et al. 2021). One preliminary study conducted in Malaysia focused on postmortem analysis of cTnI using a hs-cTnI assay and concluded that hs-cTnI might be a potential biomarker in cardiac cases with AMI (Wan et al. 2023).

This study aimed to analyse postmortem cTnI levels using the hs-cTnI assay regardless of the cause of death. It also explored the potential role of hs-cTnI as a biomarker in cases of cardiac-related deaths. Additionally, it sought to investigate any potential association between hs-cTnI levels and traumatic death cases.

MATERIALS AND METHODS

Study Design

This retrospective cross-sectional study was conducted at the Forensic Unit of Hospital Canselor Tuanku Muhriz (HCTM) in Kuala Lumpur, Malaysia, from July 2022 to December 2023. The study included samples from cases of natural and unnatural medicolegal deaths brought in by the police or Emergency Department of HCTM, which underwent medicolegal postmortem examination.

Natural deaths encompassed fatalities

due to conditions such as myocardial infarction, haemorrhagic stroke or infection, whereas unnatural deaths included accidents like falls from heights or motor vehicle crashes, suicides and homicides. The study included deceased persons over 18 years old with documented cardiopulmonary resuscitation (CPR) status, histopathological examination reports and hs-cTnI results as inclusion criteria. Exclusion criteria comprised postmortem intervals (PMI) of more than 24 hours, gross kidney abnormalities and cases involving decomposed, charred or skeletonised human remains.

Study Sample

Subjects were categorised into two primary groups, i.e. cardiac and non-cardiac deaths. The cardiac death group was subdivided into two main categories, i.e. deaths due to ischaemic heart disease (IHD) and deaths from other heart pathologies. IHD deaths were further classified into four categories i.e. AMI, coronary thrombosis, old myocardial infarction (MI) and death solely attributable to coronary atherosclerosis. Other heart pathologies included anomalous coronary artery, hypertensive heart disease, cardiomyopathy, valvular heart disease and cardiac tamponade resulting from a ruptured dissecting aortic aneurysm.

Non-cardiac deaths were categorised into asphyxia/hanging, intoxication, infection (excluding myocarditis, which is classified as a cardiac-related death), trauma and other causes. Examples of other categories included malignancy, electrocution, brain pathology, perforated viscus and ruptured oesophageal varices. Traumatic deaths were further categorised

into fatal chest injuries, fatal head injuries and multiple fatal injuries.

Statistical Analysis

Statistical analyses were conducted using IBM SPSS Statistics 28.0.0.0 (190) software for Windows (IBM SPSS Statistics, Inc., Chicago, IL, USA). Descriptive analysis of demographic data was presented using numbers and percentages. Correlation analysis was performed to examine the relationship between age and hs-cTnI levels. Differences in hs-cTnI levels among groups were assessed using the Mann-Whitney U or Kruskal-Wallis tests. Furthermore, the reliability of cTnI measured by the hs-cTnI assay as a potential biomarker was evaluated using Receiver Operating Characteristic (ROC) analysis to determine the area under the curve (AUC).

Ethics Approval

Ethical approval for this study was granted by the Universiti Kebangsaan Malaysia Review Board and Ethics Committee under approval code UKMPPI/111/8/JEP-2022-465.

RESULTS

Out of 783 postmortem cases, 219 were included in this study over 18 months, consisting of 84 cases of cardiac deaths and 135 cases of non-cardiac deaths. The median age of participants was 43 years, ranging from 18 to 88 years old. Despite the lack of a significant relationship between age and hs-cTnI levels, the correlation analysis showed a positive Pearson correlation coefficient ($r=0.126$).

Males predominated among the subjects, accounting for 183 cases (83.6%), compared to 36 cases (16.4%) for females (Table 1). Significant differences were observed among ethnicities, with Chinese being the majority at 74 cases (33.8%), followed by Malay at 52 cases (23.7%) and Indian at 41 cases (18.7%) ($p < 0.05$). CPR was performed in approximately one-fifth of the cases (42 cases, 19.18%), but there was no significant association found with hs-cTnI levels ($p > 0.05$).

The subjects were categorised into two main groups i.e. cardiac deaths (84 cases) and non-cardiac deaths (135 cases). These groups were further subdivided based on the data collection results (Table 2). Among cardiac deaths, IHD predominated with 71 cases, while other heart pathologies accounted for 13 cases. Within the IHD subgroup, AMI cases comprised nearly half of the IHD cases, totalling 30 cases. In the non-cardiac death group, trauma was the most frequent cause (59 cases), followed by deaths due to infection (31 cases). No significant differences were observed in the non-cardiac death group.

Significant differences were noted in median levels of hs-cTnI, with higher levels observed in cardiac deaths (5914.80 pg/ml) compared to non-cardiac cases (1246.10 pg/ml). Differences were significant when comparing IHD, other heart pathologies and non-cardiac deaths. However, post-hoc analyses revealed significant differences only between IHD and non-cardiac death groups.

In the cardiac death group, significant differences were observed in median levels of hs-cTnI. Post-hoc analyses indicated significant differences between AMI vs old MI ($p < 0.01$), AMI vs coronary atherosclerosis ($p < 0.01$), coronary

TABLE 1: Median with interquartile range (IqR) across different subject characteristics and Hs-cTnI levels

Variables	Frequency (%)	Median (IqR)	Median Hs-cTnI level (IqR) (pg/ml)
Age		43 (21)	-
Sex			
Male	183 (83.6)		2407.30 (16627.60)
Female	36 (16.4)		6613.40 (25073.58)
Ethnicity			
Chinese	74 (33.8)		7237.70 (24165.95)
Malay	52 (23.7)		1139.25 (12975.18)
Indian	41 (18.7)		1561.50 (21602.90)
Indonesian	14 (6.4)		2355.75 (15963.90)
Others	11 (5.0)		557.40 (18771.50)
Bangladeshi	9 (4.1)		8469.10 (24714.80)
Burmese	7 (3.2)		4368.50 (11663.90)
Pakistani	6 (2.7)		5106.55 (18240.25)
Nepalese	5 (2.3)		407.10 (17994.40)
Cardiopulmonary (CPR) status			
CPR performed	42 (19.2)		2287.50 (27501.15)
CPR not performed	177 (80.8)		3062.40 (18732.05)

atherosclerosis versus valvular heart disease (VHD) ($p < 0.05$), old MI vs VHD ($p < 0.05$) and VHD versus cardiomyopathy ($p < 0.05$). Significant differences were also noted between AMI deaths and non-AMI cardiac deaths. In the assessment of hs-cTnI levels in AMI deaths, ROC curve analysis yielded an AUC of 0.723 with a 95% confidence interval ranging from 0.647 to 0.79 (Figure 1).

Regarding the extent of stenosis caused by atheromatous plaque in the coronary arteries among cardiac death cases, severe stenosis was predominant, accounting for 67 cases (79.76%) out of 84 cardiac deaths, followed by normal coronary artery appearance in 9 cases (10.71%) (Table 3), but no differences were observed.

Several significant findings emerged from the analysis of hs-cTnI levels in traumatic deaths (Table 4). The median hs-cTnI level was notably higher in cases, where fatal trauma involved the chest area (15509.85 pg/ml) compared to those

without fatal chest trauma (188.40 pg/ml). Pure fatal head trauma (86.20 pg/ml) showed a lower median hs-cTnI level compared to other types of fatal injuries (15509.85 pg/ml). Similarly, cases involving a single fatal injury (109.40 pg/ml) exhibited a lower median hs-cTnI level than cases with multiple fatal injuries (21146.90 pg/ml).

DISCUSSION

With the higher incidence of sudden cardiac death (SCD), particularly due to AMI in forensic contexts, extensive research has been conducted on the potential use of cTn as a biochemical marker in postmortem (González-Herrera et al. 2016). Hence, careful selection of data based on specific inclusion and exclusion criteria is crucial to ensure the quality and reliability of this retrospective analysis. The inclusion criteria in the present study specifically required cases

TABLE 2: Kruskal-Wallis test and Post hoc Mann-Whitney tests for Hs-cTnI levels in cardiac deaths and non-cardiac deaths.

	Frequency (%)	Median Hs-cTnI level (IqR) (pg/ml)	'p' value		Cardiac vs non-cardiac death 'p' value	AMI vs other cardiac death 'p' value	AMI vs all death 'p' value
			Within categories	Overall categories			
Cardiac deaths			5914.80 (18278.43)	0.032**	<0.001 ^b	0.001 ^b	0.001 ^b
Ischaemic heart disease	71 (32.42)	5680.70 (18533.00)					
Acute myocardial infarction	30 (13.70)	12145.25 (32397.73)					
Coronary thrombosis	3 (1.37)	7619.00 (-)					
Old myocardial infarction	15 (6.85)	2933.10 (9862.90)					
Coronary atherosclerosis alone	23 (10.5)	2229.90 (15565.70)					
Other heart pathology	13 (5.94)	6352.60 (24155.20)					
Anomalous coronary artery	5 (2.28)	9752.80 (24760.90)					
Hypertensive heart disease	2 (0.91)	2494.45 (-)					
Cardiomyopathy	3 (1.37)	2910.60 (-)					
Valvular heart disease	1 (0.46)	-					
Cardiac tamponade	2 (0.91)	20315.75 (-)					
Non-cardiac deaths			1246.10 (20684.90)	0.625 ^a			
Asphyxia/Hanging	8 (3.65)	23181.15 (29366.55)					
Intoxication	11 (5.02)	633.90 (11212.10)					
Infection	31 (14.16)	460.60 (6074.00)					
Trauma	59 (26.94)	814.20 (24137.20)					
Other	26 (11.87)	2072.95 (17548.70)					

^a Kruskal-Wallis; ^b Mann-Whitney test

*Post-hoc analyses revealed significant differences between AMI vs old MI (p=0.005), AMI vs coronary atherosclerosis (p=0.003), coronary atherosclerosis vs valvular heart disease (p=0.045), old MI vs valvular heart disease (p=0.04) and valvular heart disease vs cardiomyopathy (p=0.044).

Post-hoc analyses revealed significant differences only between IHD and non-cardiac death (p value=0.02). AMI = acute myocardial infarction, MI = myocardial infarction

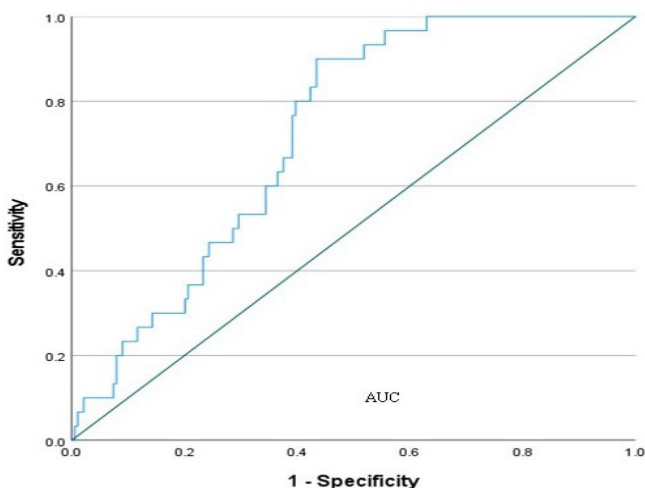


FIGURE 1: Area under the curve for the evaluation of Hs-cTnI in AMI deaths

TABLE 3: Kruskal-Wallis test and interquartile range (IqR) between severity of coronary atheroma and blood Hs-cTnI levels in cardiac deaths

Stenosis severity	Frequency (%)	Median Hs-cTnI level (IqR) (pg/ml)	z-statistic	'p' value
Normal	9 (10.71)	2910.60 (8769.55)	5.19	0.158 ^a
Mild to moderate	8 (9.52)	14864.85 (43942.30)		
Severe	67 (79.76)	5680.70 (17514.70)		

^aKruskal-Wallis

TABLE 4: Mann-Whitney test and interquartile range (IqR) between types of injury and blood Hs-cTnI level in traumatic deaths

Types of injury	Frequency (%)	Median Hs-cTnI level (IqR) (pg/ml)	z-statistic	'p' value	
Fatal chest trauma	Fatal chest trauma	28 (47.46)	15509.85 (38858.03)	-2.520	0.012 ^b
	No chest trauma	31 (52.54)	188.40 (1274.40)		
Fatal head trauma	Fatal head trauma	25 (42.37)	86.20 (558.65)	-3.544	<0.001 ^b
	Others	34 (57.63)	15509.85 (36805.38)		
Multiple injuries	Multiple injuries	27 (45.76)	21146.90 (38810.20)	-3.393	<0.001 ^b
	Single fatal injury	32 (54.24)	109.40 (827.70)		

^bKruskal-Wallis

to have histopathological examination reports. This is an essential criterion because, in some cases, the cause of death is determined solely based on gross findings, which may not provide sufficient detail for accurate diagnosis. By including only cases with histopathological reports, we aim to ensure that the cause of death is accurately determined based on both macroscopic and microscopic findings. This improves the reliability of the data collected, ensuring that the cases included in the present study reflect a well-substantiated and precise cause of death. CPR status was included as an essential inclusion criterion in the present study because previous research has shown that CPR can lead to increased cTn levels (Polena et al. 2005). However, it is important to note that while some studies have demonstrated this association, others have shown no significant impact of CPR on cTn levels (Cao et al. 2019; Chen et al. 2015; Moridi et al. 2022).

Exclusion criteria, such as the presence of gross kidney abnormalities in postmortem findings and a PMI of more than 24 hours, were applied because these factors can only be reliably assessed retrospectively and are known to impact result's accuracy (Chaulin 2022; Rahimi et al. 2018). PMI refers to the time that has elapsed between a person's death and the examination of their body. Other factors that could potentially affect hs-cTn analysis accuracy such as hyperbilirubinemia, lipemia, presence of heterophile antibodies and interference from biotin and cardiac troponin antibodies, could not be determined from the postmortem reports (Sharma et al. 2023).

In the present study, age demonstrated a weak positive linear correlation with hs-

cTnI levels. These findings suggest that age may not be useful in predicting hs-cTnI levels in this population, consistent with other study findings, which showed no correlation between age and cTn levels (Rahimi et al. 2018). Interestingly, this study identified a significant association between ethnicity and cTnI levels, contrasting with other research findings (Rahimi et al. 2018; Sthaneshwar et al. 2010). It is possible that this association may be influenced by confounding factors such as underlying health conditions rather than ethnicity itself. Further research is required to determine whether this association is genuine or an artefact of the study design.

Previous studies have shown that CPR is inconclusive in causing elevated cTnI levels (Moridi et al. 2022). The present study exhibited no differences in hs-cTnI levels between cases with or without CPR, aligning with the findings from other studies and meta-analyses (Cao et al. 2019; Chen et al. 2015; Moridi et al. 2022). In our retrospective study, the availability of CPR status was recorded as a binary variable, indicating whether CPR was performed. Due to the nature of the retrospective data collection, we were unable to obtain detailed information on the duration or specific timing of the CPR procedure. As a result, while we can determine whether CPR occurred, we cannot assess how the length or timing of the procedure might have influenced the outcomes.

Generally, cardiac death cases exhibited significantly higher median hs-cTnI levels compared to non-cardiac death cases. However, these results contrast with another study that showed no difference between cardiac and non-cardiac death groups (Rahimi et al. 2018). When comparing IHD deaths with

other cardiac deaths and non-cardiac deaths, the Kruskal-Wallis test revealed a significant association among all groups. However, post-hoc analysis indicated a significant association only between IHD and non-cardiac death cases. Contrary to expectations, there was no difference between IHD and other cardiac death cases. This discrepancy suggests that the sample size for other cardiac deaths may be insufficient to detect smaller group differences.

Further analysis of hs-cTnI levels revealed that AMI cases exhibited higher hs-cTnI levels compared to other cardiac deaths and most other cases, except for those involving cardiac tamponade due to ruptured dissecting aneurysm and asphyxia. These findings were consistent with meta-analyses indicating that both postmortem cTnI and cTnT levels increased in the serum and pericardial fluid in SCD (Cao et al. 2019).

Patients with thoracic aortic dissection were known to have elevated cTn levels, with the elevation mechanism linked to the proximal intimal flap of the dissection extending over the coronary ostia, disrupting blood flow and leading to myocardial ischemia (Ranasinghe & Bonser 2010; Zhao et al. 2022). While asphyxia was also associated with higher cTn levels based on several studies in live patients, this study found no differences when comparing asphyxia/hanging deaths with other death categories ($p > 0.05$) (Issa et al. 2021; Volobuev 2021).

The AUC for diagnosing AMI in this study was significant, falling between 0.7 and 0.8. This result was consistent with the study by Moridi et al. (2022), which reported an AUC of 0.744. This indicates that the hs-cTnI level has moderate

discriminatory power and is acceptable for diagnosing AMI-related deaths (Moridi et al. 2022). Based on these findings, hs-cTnI can be considered a reliable test in cases of SCD.

In traumatic deaths, cTn has been recommended as an aid, particularly in cases of cardiac injury resulting from minor blunt force trauma to the chest with unremarkable or subtle histopathological findings (Akbar et al. 2024). The present study showed significant elevations in hs-cTnI levels when the injury involved the chest and multiple injuries. Interestingly, fatal head injuries alone showed significantly lower hs-cTnI levels compared to other types of injuries. The possible explanation of low cTn level in fatal head injury because the primary damage occurs in the brain instead of the heart. Since cTn is a marker specific to cardiac muscle damage, levels may not significantly increase unless the head injury leads to secondary cardiac issues, such as stress-induced cardiomyopathy or direct trauma to the heart.

The exact cut-off value for postmortem cTn levels has yet to be established, as elevations were observed in almost all cases for reasons that remained unclear. Several hypotheses have been proposed such as myocardial damage during the agonal event, CPR or leakage from the cardiomyocyte membrane, but there was no consensus (Rahimi et al. 2018). However, the present study demonstrated significant differences in hs-cTnI levels between various causes of deaths, which could help to suggest the possible cause, particularly in AMI cases. Also, the present findings indicated that hs-cTnI has a good accuracy in diagnosing AMI in postmortem cases. A meta-analysis by Cao et al.

(2019) recommended that each forensic laboratory establishes its reference values for postmortem cTn levels.

The postmortem analysis of hs-cTnI in this study presented several challenges due to its retrospective, cross-sectional design, which limited access to detailed information in the postmortem records. These limitations included reliance on available postmortem findings and the inconsistent collection of hs-cTnI samples, as troponin testing was not routinely performed by all doctors. Additional limitations involved the inability to control for confounding factors such as medication use, underlying medical conditions and illicit drug history. Furthermore, variables like sample collection time, the volume of blood taken, resuscitation history, and patient age, while recognised as potential confounders, were outside of our control.

CONCLUSION

The study investigated the potential use of hs-cTnI as a postmortem biochemical marker in forensic settings, particularly for SCD cases. Cardiac deaths showed significantly higher median hs-cTnI levels compared to non-cardiac deaths, with AMI cases exhibiting higher hs-cTnI levels than most other cases. The study also found that hs-cTnI levels were significantly elevated in traumatic deaths, especially those involving chest injuries and multiple injuries, while fatal head injuries alone had the least hs-cTnI levels. In brief, hs-cTnI measured by the hs-cTnI assay provided reliable postmortem blood sample testing for diagnosing AMI and traumatic injuries. Although no established reference value exists yet, it is recommended that each laboratory establishes its reference values.

This would help forensic pathologists to use complementary tests to aid in diagnosing and resolving postmortem cases.

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CONFLICT OF INTEREST

The authors declare there is no conflict of interest

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