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Postmortem cardiac troponin T levels in the blood and pericardial fluid. Part 2: Analysis for application in the diagnosis of sudden cardiac death with regard to pathology

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Abstract

Although previous forensic pathological studies have suggested the possible application of cardiac troponins in the diagnosis of myocardial infarction, there appears to be insufficient data with regard to its cardiac pathology. The present study analyzed the heart blood, peripheral blood and pericardial fluid levels of cardiac troponin T (cTnT) in sudden cardiac deaths ($n=96$) within 48 h postmortem in relation to pathological findings of acute myocardial infarction (AMI, $n=34$), recurrent myocardial infarction (RMI, $n=23$), ischemic heart disease without any pathological evidence of infarction (IHD, $n=24$) and other heart diseases (OHD, $n=15$). Control groups ($n=75$, survival time <24 h) within 48 h postmortem consisted of asphyxiation ($n=35$), drowning ($n=27$) and cerebrovascular diseases ($n=13$). There was a marked correlation in the cTnT levels between right and left heart blood samples. The pericardial level was usually higher than either heart blood level, and the external iliac venous blood level was the lowest. Although postmortem time-dependent increases in heart and pericardial blood cTnT levels were observed in most groups, they were most evident for AMI and asphyxiation. In the early postmortem period (<12 h) there was no significant difference between AMI or RMI and the other groups except for drowning. After 12 h postmortem, significantly elevated heart blood and pericardial cTnT levels were observed for AMI and RMI showing multiple interstitial hemorrhages and necrosis compared to those with localized eosinophilic changes or patchy interstitial hemorrhages, IHD and OHD. These differences were the smallest for peripheral blood. For sudden cardiac death cases, the difference in cTnT level at each site among the causes of death was independent of gender, age, heart or lung weight and pathologies of affected coronary artery and severity of coronary stenosis. These observations suggest that the elevation in postmortem blood and pericardial cTnT levels in sudden cardiac death may depend on the severity of ischemic myocardial damage including the size and intensity of myocardial lesions involving multiple interstitial hemorrhages and necrosis, and also the postmortem period for heart and pericardial levels.

Keywords: Forensic pathology; Postmortem biochemistry; Blood; Pericardial fluid; Cardiac troponin T; Sudden cardiac death

1. Introduction

Sudden cardiac death is a common cause of acute death in forensic pathology [1–6]. Although it is not difficult to detect typical myocardial lesions using conventional pathological methods, quantitative evaluation of the severity of myocardial damage is not easy. Since positive pathological evidence cannot be detected in cases of sudden death in the very early stage of infarction, the diagnosis usually depends on negative findings excluding other causes of death [5–7]. Cardiac troponins (cardiac myofibril-specific proteins) are specific markers of myocardial damage [5–12]. Previous studies have suggested the possible application of these markers in the postmortem diagnosis of acute myocardial infarction [6–13]. However, the preceding report (Part 1) [7] showed that elevated postmortem blood and pericardial cardiac troponin T (cTnT) levels may

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depend on the severity of myocardial damage from various causes of death at the time of death, including hyperthermia, methamphetamine (MA) abuse and carbon monoxide (CO) poisoning. Thus, blood and pericardial cTnT levels should be discussed based on careful comparison with the pathological findings and in consideration of the postmortem period [7]. Although a comparative study of biochemical and morphological findings is also necessary for sudden cardiac death, few such investigations exist [10,14 –16].

The present report (Part 2) describes the findings of postmortem blood and pericardial cTnT levels in sudden cardiac death cases and discusses them with regard to macro- and micro-pathology.

2. Materials and methods

2.1. Materials

Medicolegal autopsy cases (within 48 h postmortem) at our institutes ($n=171$) were examined comprising 115 males and 56 females, 2 month–94 years of age with a median of 64.0 years of age, and a postmortem interval of 5–48 h. Specimens were aseptically collected using syringes. Blood was drawn separately from the left heart, right heart chambers and external iliac vein, and pericardial fluid was also collected. The blood samples were immediately centrifuged to separate the serum, which was subsequently stored at −20 °C until use.

The causes of death were classified based on routine macromorphological, micropathological and toxicological examinations as described in the preceding report [7]. The subjects involved in the present study were as follows: sudden cardiac death groups ($n=96$, within 48 h postmortem) consisting of acute myocardial infarction (AMI, $n=34$), recurrent myocardial infarction (RMI, $n=23$), ischemic heart disease without any pathological evidence of infarction (IHD, $n=24$), other heart diseases (OHD, $n=15$: idiopathic myopathy, $n=9$; chronic congestive heart disease, $n=6$), and control groups ($n=75$, survival time < 24 h, within 48 h postmortem) consisting of asphyxiation ($n=35$), drowning ($n=27$) and cerebrovascular diseases (CVD, $n=13$) selected from the cases in the preceding report [7]. Morphological severity of myocardial damage for sudden cardiac death was examined on the left anterior, left posterior, septal and right lateral myocardial sections by routine hematoxylin–eosin (HE) staining, and were scored: score 1, not evident except for interstitial congestion, edema and patchy myocardial eosinophilic changes; score 2, localized eosinophilic changes with patchy interstitial hemorrhages; score 3, localized necrosis; score 4, localized necrosis with multiple interstitial hemorrhages. Case profiles and examples of cardiac pathology for sudden heart diseases are shown in Tables 1 and 2, and Fig. 1a–d, respectively.

2.2. Biochemical analyses

cTnT was measured using an electro-chemiluminescence immunoassay [17]. Hemoglobin contamination at <0.5 g/dl did not interfere with the measurements. The clinical reference blood range was 0.1 ng/ml for the peripheral venous blood. In cases of strong hemolysis, which may have influenced the measurements, the findings were not used in the analyses.

Table 1
Case profiles ($n=171$)

Cause of death	n	Male/female	Age (years)		Survival time (h)		PMI (h)	
			Range	Median	Range	Median	Range	Median
Cardiac death								
AMI	34	21/13	45–94	67.0	<0.5–24	<0.5	5–42	19.3
RMI	23	18/5	55–85	70.0	<0.5–20	<0.5	8–38	18.0
IHD	24	17/7	47–85	71.5	<0.5–3	<0.5	5–34	18.5
Other heart diseases[a]	15	8/7	17–87	64.0	Unknown		10–34	19.9
Asphyxiation								
Strangulation/hanging	25	17/8	9–84	48.5	<0.5		6–44	20.3
Aspiration	10	7/3	12–78	56.5	<0.5–3	<0.5	8–42	16.0
Drowning								
Freshwater	10	8/2	5–72	47.0	<0.5		9–36	27.3
Saltwater	17	12/5	0–73	56.0	<0.5		8–48	20.3
Cerebrovascular disease[b]	13	5/8	43–81	63.0	<0.5–36	10	5–30	22.3
Total	171	115/56	0–94	59.0			5–48	19.7

PMI, postmortem interval; AMI, acute myocardial infarction; RMI, recurrent myocardial infarction; IHD, ischemic heart disease.
[a] Idiopathic myopathy ($n=9$) and chronic congestive heart disease ($n=6$).
[b] Spontaneous cerebral hemorrhage ($n=9$) and subarachnoid hemorrhage ($n=4$).