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Evaluation of postmortem urea nitrogen, creatinine and uric acid levels in pericardial fluid in forensic autopsy

Bao-Li Zhu*, Takaki Ishikawa, Tomomi Michiue, Dong-Ri Li, Dong Zhao, Li Quan, Hitoshi Maeda

Department of Legal Medicine, Osaka City University Medical School, Asahi-machi 1-4-3, Abeno, 545-8585 Osaka, Japan

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Abstract

In postmortem biochemistry, there is insufficient data available for the practical analysis of factors in the pericardial fluid. The aim of the present study was to examine postmortem pericardial fluid for urea nitrogen (UN), creatinine (Cr) and uric acid (UA) levels to investigate the pathophysiology of death in forensic autopsy cases (total, $n=409$; within 48 h postmortem), which included blunt, sharp instrument injury, asphyxiation, drowning, fire fatalities, hyperthermia, hypothermia, methamphetamine-related fatalities, other poisoning, delayed death from trauma and natural diseases. There was a significant elevation in the three markers for chronic renal failure, gastrointestinal bleeding, hyperthermia, hypothermia, methamphetamine fatalities and delayed traumatic death, which was comparable with the clinical criteria for their serum levels. These postmortem findings showed azotemia due to renal failure, elevated protein catabolism and rhabdomyolysis. Although the pericardial levels were otherwise similar to the clinical serum reference ranges, only the drowning fatalities showed significantly lower levels for each marker. These observations suggested the stability of UN, Cr and UA in the pericardial fluid within 48 h postmortem and their usefulness for the pathophysiological investigation of death involving azotemia.

Keywords: Forensic pathophysiology; Postmortem biochemistry; Urea nitrogen; Creatinine; Uric acid; Pericardial fluid

1. Introduction

Postmortem biochemistry is one of the most productive ancillary procedures available to forensic pathologists [1–5]. During the biochemical investigation of cadaveric body fluids, agonal and postmortem interference should be taken into consideration. Using a classical procedure to compare the postmortem findings with clinical data, it was suggested that the most useful blood samples are of peripheral origin [1]. However, previous study suggested that the distribution of nitrogenous compounds in the cardiac and peripheral blood may depend on the cause of death [5]. Further investigations are necessary for more detailed pathophysiological analyses of the blood levels for individual cases, possibly using a reference material. In this respect, the vitreous and pericardial fluids may be investigated for alternative materials [6–19]. Although postmortem interference is also inevitable for vitreous fluid, nitrogenous compounds including urea nitrogen (UN), creatinine (Cr) and uric acid (UA) are relatively stable [5]. Pericardial fluid can also be easily collected without significant contamination. However, there is insufficient published data regarding the practical analysis of these nitrogenous compounds in the pericardial fluid.

In the present study, we comprehensively examined postmortem BUN, Cr and UA levels in the pericardial fluid as markers for investigating the pathophysiology of death.

2. Materials and methods

2.1. Materials

The pericardial fluid of 409 forensic autopsy cases within 48 h postmortem at our institute were examined. The cases

* Corresponding author. Tel.: +81 6 6645 3767; fax: +81 6 6634 3871.
E-mail address: baolizhu@med.osaka-cu.ac.jp (B.-L. Zhu).

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included 304 males and 105 females, 2 months to 94 years in age (mean of 58.8 years of age) with postmortem intervals between 5 and 47 h. Samples were aseptically collected using syringes and stored at −20 °C, and were centrifuged before assay.

The causes of death were classified as follows: acute traumatic death from blunt injury ($n=65$), sharp instrument injury ($n=15$), mechanical asphyxiation ($n=36$: hanging, $n=6$; strangulation, $n=11$; aspiration, $n=13$; others, $n=6$), drownings ($n=31$: freshwater, $n=15$; saltwater, $n=16$), fire fatalities ($n=80$) consisting of those with blood carboxyhemoglobin (COHb) below 60% ($n=47$) and above 60% ($n=33$), hyperthermia ($n=7$), hypothermia (cold exposure, $n=6$), methamphetamine (MA)-related fatalities (fatal intoxication, $n=7$; other causes of abuser deaths, $n=8$), other poisoning ($n=17$), delayed death from traumas (multiple organ insufficiency, $n=45$; survival time, 3–90 days), acute myocardial infarction/ischemia (AMI, $n=70$), spontaneous cerebral hemorrhages (CH, $n=10$), gastrointestinal (GI) bleeding ($n=5$) and chronic renal failure (uremia, $n=7$) (details shown in Table 1). The above-mentioned causes of death were classified on pathological and toxicological bases. The AMI group consisted of cases of sudden death, which showed macro- and microscopical findings of acute ischemic heart diseases without any evidence of the cause of death being other than due to a cardiac attack [20].

2.2. Biochemical analyses

UN was measured by a urease–glutamate dehydrogenase method [21], Cr by an alkali–picric acid method (modified Jaffe method) [22], and UA by a uricase peroxidase method using an L-type UA Kit (Wako, Tokyo) [23]. Hemoglobin contamination (<0.5 g/dl) did not interfere with the measurements. The clinical reference serum ranges were: 6–20 mg/dl for UN, 0.61–1.04 mg/dl (adult male) and 0.47–0.79 mg/dl (adult female) for Cr, and 3.7–7.6 mg/dl (male) and 2.5–5.4 mg/dl (female) for UA.

2.3. Toxicological analyses

The blood COHb concentrations were determined using a CO-oximeter system [24,25] for all fire fatalities. Volatile chemicals including alcohol were analyzed using head-space gas chromatography for all cases, and drug analyses were performed by gas chromatography/mass spectrometry when preliminary screening tests were positive.

2.4. Statistical analyses

Regression equation analysis was used to compare two parameters including biochemical markers, the age of the victims, the survival time and the postmortem interval. Comparisons between groups were performed using the

Table 1
Case profiles ($n=409$)

Cause of death	n	Male/Female	Age (years)		Survival time (h)		PMI (h)	
			Range	Median	Range	Median	Range	Median
Blunt injury	65	53/12	10–94	55.0	<0.5–24	1.5	9–44	19.8
Sharp instrument injury	15	11/4	38–90	60.0	<0.5–16	<0.5	7–42	16.9
Asphyxia	36	22/14	0–93	55.0	<0.5		6–44	18.3
Drowning								
Freshwater	15	10/5	45–79	60.0	<0.5		9–36	20.0
Saltwater	16	11/5	0–73	47.0	<0.5		7–47	18.7
Fire fatality								
COHb <60%	47	39/8	23–93	66.0	<0.5		6–36	16.3
COHb >60%	33	23/10	1–87	68.0	<0.5		5–42	12.3
Hyperthermia	7	5/2	15–88	72.0	3–14	5.0	9–31	20.5
Hypothermia	6	5/1	44–76	57.0	3–6	3.0	6–44	16.3
MA-related fatalities								
MA poisoning	7	6/1	20–52	38.0	3–24	4.5	22–45	29.0
other MA-abuser fatalities	8[a]	6/2	20–59	45.0	<0.5–22	<0.5	10–24	13.8
Other poisoning	17[b]	12/5	20–87	56.0	<0.5–24	5.0	13–42	25.0
Delayed traumatic death	45[c]	33/12	1–83	59.0	72–2,160	59.0	5–30	13.1
Natural diseases								
Acute myocardial infarction/ischemia	70	53/17	43–94	67.0	<0.5–11	<0.5	6–42	19.7
Spontaneous cerebral hemorrhages	10	5/5	45–81	64.0	0.5–19	1.5	5–39	22.4
Chronic renal failure	7	5/2	46–89	78.0	unknown		9–27	17.3
Gastrointestinal bleeding	5	5/0	50–67	61.0	1–8	2.0	10–31	14.6

PMI, postmortem interval; COHb, carboxyhemoglobin concentration; MA, methamphetamine.

[a] Asphyxia ($n=3$), death due to injury ($n=2$), burns ($n=1$), acute myocardial infarction ($n=1$) and spontaneous cerebral hemorrhage ($n=1$).

[b] Sedative–hypnotics ($n=4$), sodium cyanide ($n=2$), ethanol ($n=3$), carbon monoxide ($n=3$) and others ($n=4$).

[c] Multiple organ insufficiency and secondary infection from head injury ($n=39$) and chest injury ($n=6$).