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Original Communication

# Biochemical blood markers and sampling sites in forensic autopsy

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**Abstract**

Forensic pathologists often hesitate to use biochemical blood markers due to the risk of large postmortem changes and deviations from healthy subjects. Biochemical analyses of postmortem blood, if possible, may help to evaluate pathological status and determining the cause of death in forensic diagnosis, for example, in sudden unexpected death without obvious cause, or young adults with no apparent cause of death or antemortem information. Even commercially available biochemical markers were re-evaluated in the blood samples of 164 forensic autopsy cases. Biochemical markers examined were HbA1c, fructosamine, blood nitrogen urea (BUN), creatinine, total protein, total bilirubin, γ-glutamyl transpeptidase (γ-GTP), triglyceride, total cholesterol, C-reactive protein (CRP) and pseudocholine esterase (pChE). We collected cardiac blood (left cardiac blood and right cardiac blood) and peripheral blood (femoral vein blood) to clarify the differences in measured values by sampling site. The measured values were analyzed in relation to postmortem interval, etiology of death and sampling sites. Of all eleven markers, HbA1c is the most useful and reliable because of its negligible postmortem changes and small deviation from healthy subjects. Total bilirubin, BUN, CRP and total cholesterol can be useful if we set appropriate limit ranges and pay attention to the interpretation. For the evaluation of changes due to postmortem intervals, none of the markers except for triglyceride showed significant changes up to three days postmortem. As for sampling sites, femoral vein blood is generally recommended considering postmortem changes, but left cardiac blood was suitable for creatinine, pChE, and total cholesterol. For clinical forensic diagnosis of biochemical blood markers, we must determine the "forensic abnormal value" after collecting more cases by known causes with more information about the population.


*Keywords:* Biochemical markers; Postmortem change; Right cardiac blood; Left cardiac blood; Femoral vein blood; Sampling sites

## 1. Introduction

In forensic autopsies, antemortem information such as present and past illnesses is rarely available, and the forensic pathologist must make a decision on the basis of autopsy findings. In clinical medicine, a lot of information, including biochemical markers in the blood, is available and contributes to the diagnosis of disease, in addition to physical findings. For forensic pathologists, biochemical analysis of the postmortem blood, if possible, may help in evaluating pathological status and determining cause of death in such cases. However, to date, forensic pathologists have often hesitated to use biochemical markers in the blood for forensic diagnosis due to concern about large postmortem changes and large deviations from healthy subjects.

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There is a complete review by Coe on autopsy samples, covering a lot of markers[1] and a report by Tsuji et al. on an animal study,[2] with respect to postmortem changes in the markers. Additionally, there are several reports on the usefulness of the individual markers in autopsy diagnosis.[3–15] However, there is insufficient information on commercially available blood markers and differences in sampling sites in forensic autopsy cases. We chose to measure eleven clinically available biochemical markers in the blood from three sampling sites. The cost for measuring these eleven markers by one sample was very low because these markers are routinely measured in clinical medicine. The cost/benefit factor is very important for low-budget forensic facilities.

Our aim was to re-examine and evaluate commercially available blood markers and their usefulness in forensic diagnosis. We investigated how biochemical markers in the blood suffer from postmortem changes and showed differences due to the etiology of death, while determining suitable sampling sites. These will be useful for taking postmortem changes into consideration when selecting markers and interpreting results.

## 2. Materials and methods

### 2.1. Blood samples

With the permission of the Ethics Committee of Graduate School of Medicine, The University of Tokyo (No. 690), blood was obtained from 164 consecutive autopsy cases in our department from April 2003 to March 2006 (age 0–98, average age 54.9 ± 21.8, median age 57.0, male 112, female 52). The postmortem interval of the sampling of specimens are as following: 0–12 h (25 cases), 13–69 h (69 cases), 25–48 h (54 cases), 49–72 h (16 cases). Causes of death were as follows: blunt injury (52 cases), sharp injury (seven cases), asphyxiation (18 cases), drowning (four cases), fire death (five cases), intoxication (nine cases), internal death (39 cases) and others (30 cases). Care was paid so that the deceased would not be identified from the data. The bodies were preserved refrigerated and forensic autopsies were performed within a day after they were found. The blood was sampled from the right and left heart cavities and femoral vein within 72 h postmortem, as far as possible. As soon as whole blood was obtained, the sera was separated by centrifugation at 1000*g*, 30 min, and stored at −20 °C, while the whole blood was stored at 4 °C as long as a day, until shipping to the laboratory of SRL, Co. Ltd. (Tokyo, Japan), where the samples were analyzed within a day.

### 2.2. Biochemical analyses

We selected the biochemical markers on the basis of postmortem stability reported in previous reports.[1–4] The 11 markers included HbA1c (latex aggregation method, standard range: 4.3–5.8%) and the fructosamine (calorimetry method: 205–285 mM) for chronic hyperglycemia, blood nitrogen urea (BUN) (urease UV method: 6–20 mg/dL) and creatinine (enzyme method: male 0.61–1.04 mg/dL, female 0.47–0.79 mg/dL) for renal failure, total protein (Biuret method: 6.7–8.3 g/dL) for malnutrition, total bilirubin (vanadinate oxidation method: 0.2–1.0 mg/dL) and γ-glutamyl transpeptidase (γ-GTP) (JSCC standardization method: male < 70 IU/L, 37 °C, female < 30 IU/L, 37 °C) for liver function, triglyceride (enzyme method: 50–149 mg/dL) and total cholesterol (enzyme method: 150–219 mg/dL) for hyperlipidemia, C-reactive protein (CRP) (latex aggregation method, <0.3 mg/dL) for inflammation, pseudocholine esterase (pChE) (rate assay, male 242–495 IU/L, 37 °C, female 200–495 IU/L, 37 °C) for liver function and organic phosphate poisoning. The sera volume of required to measure the 11 markers was 2 mL.

The laboratory rejected 2.4% of samples for bilirubin measurement, but not those for other markers. However, we could not measure all the markers in substantial numbers of cases because of a lack of sample volume.

### 2.3. Statistical analyses

Data are expressed as the mean ± SD. Statistical significance was determined as follows: for postmortem interval, Spearman's rank correlation was carried out (Table 2). For etiology of death, one-way ANOVA was carried out. When there was a difference among groups, Scheffe's posthoc test between multiple groups was performed (Table 3). For regional differences, one-way repeated measures ANOVA was carried out. When there was a difference among groups, a paired *t*-test for pair-wise comparisons was performed (Table 4). The software for the above-mentioned statistical analysis was Statview Ver. 4.11 (Abacus Concepts Inc., Berkeley, CA). Significant level was 0.05 (5%).

## 3. Results

We summarized the data for right cardiac blood, which were obtained in almost all cases (Table 1). First, HbA1c showed almost the same mean value as healthy subjects, and a very low ratio of abnormal values (24.8%), as compared with fructosamine (77.7%), another marker for chronic hyperglycemia. The next group of markers showed much higher mean values than the healthy subjects, and a higher ratio of abnormal values (37.3–95.1%). This group included total bilirubin, triglyceride, BUN, CRP, γ-GTP, fructsamine and creatinine. In the third group, pseudocholine esterase (pChE) and total cholesterol showed lower mean values than the healthy subjects and a higher ratio of abnormal values (64.1%, 72.9%). In the last group, total protein showed almost the same mean value as healthy subjects, but a wide variability, and therefore, a high ratio of abnormal values (75.3%).

To evaluate changes due to postmortem intervals, we classified postmortem intervals into four groups (0–12 h, 13–24 h, 25–48 h, 49–72 h) and carried out correlation analysis between the results obtained for each of the

Table 1
Differences from clinical standards (right cardiac blood)

| Marker | Measured value | Value obtained from healthy subjects | Unit | *n* | Results outside the reference intervals of healthy subjects (%) |
|---|---|---|---|---|---|
| HbA1c | 5.23 ± 1.23 | 4.3–5.8 | % | 149 | 24.8 |
| *t*-Bilirubin | 1.32 ± 2.44 | 0.2–1.0 | mg/dL | 150 | 37.3 |
| Triglyceride | 129.9 ± 107.4 | 50–149 | mg/dL | 155 | 45.2 |
| BUN | 39.8 ± 40.6 | 6–20 | mg/dL | 162 | 59.3 |
| CRP | 7.54 ± 11.54 | <0.3 | mg/dL | 163 | 69.3 |
| γ-GTP | 154.1 ± 173.4 | Male < 70, female < 30 | IU/L, 37 °C | 148 | 74.3 |
| Fructsamine | 325.3 ± 147.1 | 205–285 | mM | 148 | 77.7 |
| Creatinine | 3.29 ± 2.35 | Male 0.61–1.04, female 0.47–0.79 | mg/dL | 163 | 95.1 |
| Pseudocholine esterase | 204.1 ± 120.7 | Male 242–495, female 200–459 | IU/L, 37 °C | 153 | 64.1 |
| *t*-Cholesterol | 142.3 ± 77.3 | 150–219 | mg/dL | 155 | 72.9 |
| *t*-Protein | 7.45 ± 2.09 | 6.7–8.3 | g/dL | 154 | 75.3 |

Values are expressed as the mean ± SD.

markers and the postmortem intervals. We summarized the results in Table 2. The triglyceride value significantly decreased according to the postmortem interval.

Next, to evaluate differences of obtained values by etiology of death, we classified the cause of deaths into eight categories (blunt injury, sharp injury, asphyxiation, drowning, fire death, intoxication, internal death and others). We carried out a comparison by etiology of death and summarized the results in Table 3. There were significant differences in pseudocholine esterase, total cholesterol and total protein. For total cholesterol, there were differences in asphyxiation-blunt injury, and asphyxiation-internal death. For total protein, there were differences in fire death-blunt injury, fire death-sharp injury, and fire death-internal death.

Differences by site of blood sampling are summarized in Table 4. Triglyceride, BUN and fructsamine showed no difference by sampling site, but eight other markers showed significant differences. Creatinine showed the lowest value in the left cardiac blood, but seven other markers showed the lowest values in the femoral vein blood.

## 4. Discussion

Of all eleven markers examined, HbA1c showed the smallest deviation from healthy subjects (24.8%, Table 1), negligible postmortem changes (Table 2) and no difference due to etiology of death (Table 3). We confirmed that HbA1c showed negligible postmortem changes. Glycated albumin,[9] glycated hemoglobin,[10–14] and fructsamine[14] were shown to indicate chronic hyperglycemia in autopsy samples. However, in this study fructsamine showed a large deviation from healthy subjects. We recommend HbA1c as a good marker for chronic hyperglycemia in forensic practice because of its commercial availability and reliability. We can interpret the negligible postmortem changes in HbA1c as follows: the level of glycation of hemoglobin is a cumulative process the rate of which is determined by the level of prevailing glucose concentration under the lifespan of erythrocytes. By contrast, the other markers evaluated are components of tissues reflecting their destruction, or components normally eliminated from the blood by functioning organs.

Table 2
Postmortem interval (right cardiac blood)

| Marker | Measured value | | | | *p*-value[a] | Postmortem change |
|---|---|---|---|---|---|---|
| | Postmortem time | | | | | |
| | 0–12 h | 13–24 h | 25–48 h | 49–72 h | | |
| HbA1c | 5.43 ± 1.78 (21) | 5.35 ± 1.05 (66) | 4.92 ± 1.05 (49) | 5.39 ± 1.59 (13) | 0.1571 | Unchanged |
| t-Bilirubin | 1.25 ± 2.53 (24) | 1.26 ± 2.28 (64) | 1.34 ± 2.38 (48) | 1.66 ± 3.32 (14) | 0.9305 | Unchanged |
| Triglyceride | 158.8 ± 82.5 (25) | 139.4 ± 138.0 (66) | 114.1 ± 73.5 (48) | 93.4 ± 65.0 (16) | 0.0083 | Decrease |
| BUN | 29.5 ± 29.2 (25) | 39.1 ± 34.1 (69) | 48.6 ± 51.8 (52) | 36.2 ± 40.8 (16) | 0.2752 | Unchanged |
| CRP | 7.12 ± 12.54 (25) | 9.30 ± 11.55 (69) | 6.67 ± 12.22 (53) | 3.44 ± 5.42 (16) | 0.7540 | Unchanged |
| γ-GTP | 131.0 ± 113.3 (25) | 135.8 ± 162.9 (61) | 165.1 ± 172.6 (49) | 229.6 ± 282.8 (13) | 0.2545 | Unchanged |
| Fructsamine | 304.9 ± 122.5 (22) | 308.2 ± 107.7 (66) | 360.4 ± 201.7 (45) | 301.8 ± 149.3 (15) | 0.2711 | Unchanged |
| Creatinine | 3.14 ± 2.93 (25) | 3.21 ± 2.11 (69) | 3.53 ± 2.48 (53) | 3.06 ± 2.02 (16) | 0.3442 | Unchanged |
| Pseudocholine esterase | 235.3 ± 105.0 (24) | 200.9 ± 133.1 (66) | 191.4 ± 114.9 (47) | 207.4 ± 106.0 (16) | 0.2599 | Unchanged |
| *t*-Cholesterol | 165.9 ± 59.3 (25) | 135.0 ± 83.5 (66) | 143.6 ± 79.3 (48) | 131.8 ± 67.1 (16) | 0.2212 | Unchanged |
| *t*-Protein | 7.16 ± 1.92 (25) | 7.23 ± 1.89 (66) | 7.63 ± 1.96 (47) | 8.34 ± 3.15 (16) | 0.0998 | Unchanged |

Values are expressed as the mean ± SD. (*n*): *n* is the sample number.
[a] Spearman's rank correlation test.

Table 3
Etiology of death (right cardiac blood)

| Marker | Measured value | | | | | | | | *p*-value[a] |
|---|---|---|---|---|---|---|---|---|---|
| | Blunt injury | Sharp injury | Asphyxiation | Drowning | Fire death | Intoxication | Internal death | Others | |
| HbA1c | 5.05 ± 0.93 (44) | 5.44 ± 1.01 (7) | 5.39 ± 1.43 (17) | 6.05 ± 1.52 (4) | 5.80 ± 2.37 (5) | 5.23 ± 1.50 (8) | 5.33 ± 1.34 (36) | 4.99 ± 1.09 (28) | 0.6044 |
| t-Bilirubin | 1.25 ± 1.84 (49) | 0.38 ± 0.41 (6) | 0.39 ± 0.25 (18) | 0.40 ± 0.26 (3) | 0.70 ± 0.88 (4) | 1.99 ± 3.91 (7) | 1.34 ± 2.63 (36) | 2.21 ± 3.52 (29) | 0.2808 |
| Triglyceride | 142.5 ± 148.1 (49) | 135.6 ± 46.9 (7) | 163.1 ± 94.5 (18) | 155.0 ± 116.2 (3) | 134.8 ± 79.4 (5) | 111.0 ± 61.1 (8) | 117.1 ± 85.8 (36) | 101.9 ± 77.1 (29) | 0.5737 |
| BUN | 37.5 ± 35.0 (52) | 10.4 ± 6.9 (7) | 22.0 ± 20.3 (18) | 18.4 ± 3.5 (3) | 34.0 ± 29.2 (5) | 25.2 ± 15.3 (8) | 58.1 ± 54.1 (39) | 47.5 ± 42.5 (30) | 0.0094 |
| CRP | 12.35 ± 15.40 (52) | 1.49 ± 3.30 (7) | 1.82 ± 3.44 (18) | 0.29 ± 0.42 (3) | 1.15 ± 2.43 (5) | 3.59 ± 9.43 (9) | 7.24 ± 7.67 (39) | 7.47 ± 11.23 (30) | 0.0063 |
| γ-GTP | 130.3 ± 152.8 (49) | 152.4 ± 132.1 (7) | 162.9 ± 265.5 (18) | 102.3 ± 76.5 (3) | 138.3 ± 166.3 (4) | 117.3 ± 59.9 (7) | 176.2 ± 173.3 (32) | 182.3 ± 179.1 (28) | 0.9079 |
| Fructsamine | 293.9 ± 103.9 (48) | 247.6 ± 115.6 (5) | 336.6 ± 55.8 (17) | 259.0 ± 214.6 (3) | 258.0 ± 131.4 (5) | 365.2 ± 268.9 (6) | 366.8 ± 189.1 (36) | 346.7 ± 149.6 (28) | 0.2433 |
| Creatinine | 3.35 ± 2.25 (52) | 1.91 ± 1.19 (7) | 2.99 ± 3.07 (18) | 4.09 ± 2.29 (3) | 2.69 ± 1.30 (5) | 3.16 ± 1.37 (9) | 3.64 ± 2.28 (39) | 3.29 ± 2.73 (30) | 0.7557 |
| Pseudocholine esterase | 193.0 ± 91.0 (49) | 197.2 ± 96.1 (5) | 285.6 ± 151.6 (17) | 274.0 ± 109.3 (3) | 277.6 ± 148.8 (5) | 288.8 ± 185.5 (8) | 183.7 ± 117.4 (36) | 159.5 ± 98.6 (30) | 0.0039 |
| *t*-Cholesterol | 128.9 ± 53.1[b](49) | 98.3 ± 32.3 (7) | 219.3 ± 108.0 (18) | 183.7 ± 22.6 (3) | 200.2 ± 92.9 (5) | 131.3 ± 60.8 (8) | 130.1 ± 69.4[b] (36) | 119.5 ± 66.7[b] (29) | <0.0001 |
| *t*-Protein | 6.96 ± 2.02[c] (50) | 6.06 ± 1.87[c] (7) | 8.64 ± 1.23 (18) | 9.35 ± 0.78 (2) | 10.76 ± 3.88 (5) | 9.24 ± 1.51 (7) | 7.14 ± 1.55[c] (35) | 7.17 ± 2.04[c] (30) | <0.0001 |

Values are expressed as the mean ± SD. (*n*): *n* is the sample number.
[a] One way ANOVA.
[b] $p < 0.05$ (Scheffe's posthoc test; vs asphyxiation).
[c] $p < 0.05$ (Scheffe's posthoc test; vs fire death).

Total bilirubin showed a tendency towards postmortem increase time-dependently, but not significantly (Table 2), and a relatively small deviation from healthy subjects (37.3%, Table 1). This suggests total bilirubin can be used as a marker. γ-GTP and pseudocholine esterase, other markers of liver function, are not appropriate for forensic diagnosis. They showed a high portion of deviation from healthy subjects (74.3%, 64.1%, respectively, Table 1) and no postmortem increase, although γ-GTP showed a tendency to increase time-dependently (Table 2).

For creatinine and BUN, kidney injury markers, we recommend BUN as a good marker for renal injury. The cases with BUN > 100 mg/dL showed renal diseases or acute renal failure due to hemorrhagic shock after intestinal bleeding. We must be careful when considering the background since high BUN was reported in association with severe hypoxia or skeletal muscle damage.[15] Creatinine cannot be recommended because of the extremely high ratio of abnormal values (95.1%), suggesting a postmortem increase.

The level of CRP showed no significant postmortem changes (Table 2). Clinical standard CRP is <0.3 mg/dL, which is almost equal to zero. In our study, a large standard deviation was observed (7.54 ± 11.54), but 30.7% of measured samples were within the limit of healthy subjects. Considering these results, it is concluded that CRP is a good marker for inflammation, which is also affected by survival time, as reported previously.[6]

High total cholesterol can be a marker for hyperlipidemia, since it tends to decrease postmortem (Table 2). There were significant differences in asphyxiation-blunt injury, and asphyxiation-internal death (Table 3). Postmortem time intervals were 25.7 ± 13.6 h (asphyxiation), 22.9 ± 15.6 h (blunt injury), and 29.7 ± 16.3 h (internal death), and there was no significance among the three groups in terms of postmortem interval. We concluded that differences in asphyxiation-blunt injury and asphyxiation-internal death reflect the different causes of death. By contrast, the mean triglyceride value was higher than that of healthy subjects, but there was a significant time-dependent decrease (Table 2). This marker can easily affected by ingestion or starvation, which are sometimes identified in autopsy cases and cannot be used as postmortem markers.

Total protein tended to increase time-dependently, though not significantly (Table 2). Intravascular fluid is affected either by overhydration caused by infusion or postmortem extravasation. In our data on etiology of death, there were differences in fire death-blunt injury, and fire death-sharp injury, fire death-internal death (Table 3). In all cases, fire death is related to this difference, reflecting intravascular extravasation due to heat.

As for sampling sites, we recommend the following. For HbA1c, any site can be used. This is because the values of the three sampling sites were substantially the same, although femoral vein blood showed a significantly lower value statistically. For triglyceride, BUN, fructsamine, any site can be used because there were no significant

Table 4
Regional differences in biochemical markers obtained from postmortem blood

| Marker | Measured value | | | *n* | *p*-value[a] | Regional difference[b] | Recommended sampling site |
|---|---|---|---|---|---|---|---|
| | Right cardiac blood | Left cardiac blood | Femoral vein blood | | | | |
| HbA1c | 5.26 ± 0.94 | 5.23 ± 0.97 | 5.20 ± 0.99 | 40 | 0.0452 | r > fe (r = l, l = fe) | r, l, fe |
| *t*-Bilirubin | 1.27 ± 2.12 | 1.35 ± 2.18 | 1.06 ± 1.84 | 34 | 0.0001 | r = l > fe | fe |
| Triglyceride | 131.4 ± 144.7 | 147.4 ± 183.1 | 130.6 ± 104.6 | 36 | ns | | r, l, fe |
| BUN | 45.3 ± 42.6 | 44.1 ± 42.3 | 45.1 ± 42.5 | 39 | ns | | r, l, fe |
| CRP | 7.94 ± 10.38 | 8.12 ± 10.74 | 6.96 ± 9.27 | 41 | 0.0032 | r = l> fe | fe |
| γ-GTP | 161.3 ± 192.4 | 158.4 ± 183.6 | 128.9 ± 154.6 | 38 | 0.0397 | l > fe (r = l, r = fe) | fe |
| Fructsamine | 355.9 ± 119.3 | 355.6 ± 127.0 | 334.8 ± 106.9 | 29 | ns | | r, l, fe |
| Creatinine | 3.12 ± 2.32 | 2.95 ± 2.34 | 3.30 ± 2.38 | 40 | 0.0005 | l < r = fe | l |
| Pseudocholine esterase | 262.0 ± 137.1 | 277.3 ± 163.4 | 250.7 ± 134.0 | 35 | 0.0079 | l > r = fe | l |
| *t*-Cholesterol | 178.6 ± 96.6 | 195.4 ± 118.5 | 174.6 ± 94.7 | 34 | 0.0057 | l > r = fe | l |
| *t*-Protein | 7.69 ± 1.70 | 7.78 ± 1.84 | 6.97 ± 1.89 | 37 | <0.0001 | r = l > fe | fe |

Values are expressed as the mean ± SD.
r, right cardiac blood; l, left cardiac blood; fe, femoral vein blood; ns, not significant.
[a] One-way repeated measures ANOVA.
[b] Paired *t*-test.

differences among sampling sites. For total bilirubin, CRP, γ-GTP, total protein we recommend femoral vein blood since these markers tend to increase postmortem and femoral vein blood showed a significantly lower value than the others. For creatinine, we recommend left cardiac blood since it showed a tendency to increase time-dependently, and there was a lower level in left cardiac blood than femoral vein blood. For pseudocholine esterase and total cholesterol, we recommend left cardiac blood since it showed a tendency to decrease time-dependently, and showed a higher level in left cardiac blood than femoral vein blood. In fact, left cardiac blood was suitable for measuring the value of creatinine, pseudocholine esterase, total cholesterol, while femoral vein blood was good for measuring the other eight markers (Table 4).

When biochemical blood markers are used in forensic practice, we need the reliability of the standard values. In clinical medicine, standard values are calculated from the samples obtained from healthy adults (usually 20–40 years of age). Needless to say, there is no perfect standard value with blood samples obtained from forensic autopsies because the samples are from the deceased with various causes of death, agonal states, complications, treatments and postmortem changes. These factors generally affect the level of markers. This is a serious problem. We tentatively tried to calculate a forensic standard value of HbA1c, which shows negligible postmortem changes, and obtained a value of 2.77–7.69% (mean ± 2SD), although our data included abnormal values due to etiology of death. However, the limitation of this kind of study is derived from the lack of information on death scene circumstances, present and past illness, as well as the various backgrounds of the victims. Therefore, we must be aware that above-mentioned standard value is only a guideline. A more accurate forensic standard value should be determined after collecting more samples and excluding apparently abnormal data due to pathological changes on the basis of complete analysis of all cases. Then, we can obtain a genuine forensic standard.

In our study we re-examined commercially available blood markers and presented the possibility of forensic diagnosis. For clinical forensic diagnosis using biochemical blood markers, we must collect more samples by known causes with more information about the population, and thereby determine the "forensic abnormal value". Situation that often require biochemical assessment include sudden unexpected death without obvious cause, such as alcoholics with a low post mortem blood alcohol level, young adults with no apparent cause of death and antemortem information such as present and past illnesses.

## 5. Conclusion

In our postmortem biochemistry study of 164 consecutive autopsy cases, HbA1c was clearly a reliable marker. Total bilirubin, BUN, CRP and total cholesterol would have been useful if we had set an appropriate limit range and been careful in the interpretation. For the evaluation of changes due to postmortem intervals, the triglyceride value decreased according to the postmortem interval, but other markers did not show significant changes up to three days of postmortem. As for the etiology of deaths, the value of asphyxiation showed a higher value that of blunt injury and internal death, reflecting the difference in the cause of death. Additionally, femoral vein blood is generally a suitable sampling site for measuring blood biochemical markers because of its relatively slight postmortem changes.

Postmortem biochemistry is poorly understood, under used at present because of concerns about postmortem changes and large deviations from healthy subjects, while it has great potential for forensic service work and future research into the sudden unexpected death without obvious cause, or young adults with no apparent cause of death or antemortem information. As the next step for clinical

forensic diagnosis on the basis of biochemical blood markers, we must determine the "forensic abnormal value" after collecting more samples by known causes with more information about the population.

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