Journal of Forensic and Legal Medicine 58 (2018) 126–129

Contents lists available at ScienceDirect

Journal of Forensic and Legal Medicine

journal homepage: www.elsevier.com/locate/yjflm

Research Paper

Value of the serum thyroglobulin level for diagnosing neck compression in postmortem cases

Marin Takaso, Hajime Tsuboi, Noriko Komeda, Mami Morimoto, Hiroshi Ikegaya*

Department of Forensic Medicine, Graduate School of Medical Science, Kyoto Prefectural University of Medicine, Japan

ARTICLE INFO

Keywords:
Mechanical asphyxia
Thyroglobulin
Postmortem
Right heart blood
Diagnosis
Neck compression

ABSTRACT

To investigate the relationship between blood thyroglobulin (Tg) levels and neck compression, the Tg levels of right cardiac blood were measured using a chemiluminescence immunoassay in 256 autopsy cases.

There were 11 cases in which neck compression was confirmed based on autopsy findings and other information, in which the mean Tg level was 3155 ng/mL (range: 179–16,500 ng/mL). In the remaining cases, the mean Tg level was 4160 ng/mL (range: 0.3–139,000 ng/mL). There was no significant difference between the mean Tg levels of the two groups.

In a comparison between the case groups with Tg levels of ≥200 ng/mL and < 200 ng/mL, it was found that the frequency of neck compression was significantly higher ($P < 0.05$) in the ≥200 ng/mL group.

The frequency of high Tg levels (≥200 ng/mL) was increased among the cases in which death was caused by neck compression or asphyxia.

In a comparison of the median Tg values of right heart blood, left heart blood, whole blood, and femoral venous blood, the median Tg values of whole blood and right heart blood were shown to be about 10 times higher than those of left heart blood and peripheral blood.

It is said that high postmortem blood Tg levels are caused by mechanical compression of the thyroid gland. However, high Tg levels were detected in the half of the cases without neck compression. Therefore, neck compression should be diagnosed carefully based on autopsy findings and other information.

1. Introduction

Thyroglobulin is a glycoprotein, which is synthesized and secreted by the follicular epithelial cells in the follicular wall of the thyroid gland. The follicular cavity of the thyroid gland is filled with a substance called colloid, most of which is composed of thyroglobulin. Thyroid hormones; i.e., triiodothyronine (T3) and thyroxin (T4), are synthesized by the condensation of monoiodotyrosine and diiodotyrosine (by iodinating the tyrosine residues in thyroglobulin). The synthesized T3 and T4 molecules are combined with thyroglobulin and stored in follicles. T3 and T4 are reabsorbed into the follicular epithelium by endocytosis, hydrolyzed by proteases, and secreted into the blood. In the blood, most T3 and T4 molecules are bound to plasma proteins, but the free forms of T3 and T4 exhibit physiological activity. It is rare for thyroglobulin to be detected in the blood.[1]

In the field of forensic medicine, it has been reported that postmortem blood Tg levels are useful for diagnosing neck compression.[2–5]

Among these studies, Tamaki et al.[3] quantified the Tg level of whole heart blood and found that it was > 200 ng/mL in 12 out of 14 cases of mechanical asphyxia and < 200 ng/mL in 15 cases that did not involve mechanical asphyxia. In their following study, they quantified the Tg level of right heart blood and found that 35 of 42 cases of hanging, strangulation, or throttling exhibited right heart blood Tg levels of > 200 ng/mL, whereas the other 36 cases all had right heart blood Tg levels of < 200 ng/mL.[4] This result is cited in a famous forensic medicine textbook.[6]

However, it has recently been reported that among autopsy cases that did not involve neck compression the right cardiac blood Tg levels of 43% cases and the left cardiac blood Tg levels of 23% cases were > 200 ng/mL.[7] In addition, Tg levels have been reported to rise in cases of brain injury.[8]

Also, the detailed mechanism responsible for high right heart blood Tg levels has not been fully elucidated. Recently, Hayakawa et al.[9] examined autopsy cases that did not involve neck compression. In the latter study, the Tg levels of peripheral arteriovenous blood, right heart blood, and left heart blood were measured. They concluded that congestion of the venous blood system promotes the diffusion of Tg after death and that heart blood aids the diffusion of Tg away from the

* Corresponding author. Department of Forensic Medicine, Kyoto Prefectural University of Medicine, 465 Kajiicho, Kamigyo, Kyoto, 602, Japan.
E-mail addresses: ikegaya@koto.kpu-m.ac.jp, ikegaya-tky@umin.ac.jp (H. Ikegaya).

https://doi.org/10.1016/j.jflm.2018.04.016
Received 22 February 2018; Received in revised form 5 April 2018; Accepted 28 April 2018
Available online 22 June 2018
1752-928X/

thyroid gland more readily than peripheral blood because of the heart's proximity to the thyroid gland. Therefore, they recommended that peripheral arterial blood should be used to obtain Tg measurements.

Based on our experience, we recognize that the Tg level of right heart blood is elevated in some autopsy cases that do not involve neck compression.

Although blood congestion occurs in cases of heart failure, in all previous studies of heart failure-related autopsy cases some of the control cases displayed high Tg right heart blood levels.[3,4] Therefore, it is questionable whether a right heart blood Tg level of > 200 ng/mL can be used to diagnose neck compression.

In this study, to address this question, we measured the Tg level of right heart blood in much more cases that previous studies and discussed whether it can be used as a tool for diagnosing neck compression. Furthermore, the Tg levels of right heart blood, left heart blood, and femoral venous blood were measured, and we discussed the differences among them and the most appropriate sampling site for obtaining Tg level measurements.

2. Materials and methods

Right heart blood was obtained in 256 postmortem cases (175 males, 81 females, age range: 0–98 years old, mean age: 56.3 years, postmortem time: within two days) encountered at Kyoto Prefectural University of Medicine between 2008 and 2018. The T3, T4, and thyroglobulin levels of serum samples obtained by centrifugation were measured using chemiluminescence immunoassays by an external organization (LSI Medience Corporation). In each case, an autopsy was performed, and the cause of death was diagnosed. Cases in which thyroid disease was suspected based on the autopsy findings were excluded.

The relationships among the Tg level of right heart blood, causes of death, thyroid weight, T3 levels, and T4 levels were examined.

In another 30 cases, in which it was concluded that neck compression had not occurred based on autopsy findings and other information, right heart, left heart, and femoral blood samples were collected separately, and their Tg levels were measured and compared.

Statistical analysis was performed using the Student's *t*-test, with the significance set at the 1% level. A chi-square test with Yates revision or Fisher's exact test was used to compare differences between two groups. Values of $P < 0.05$ were considered significant. Statistical analysis was performed using StatMate version 4.01.

This research was approved by the institutional review board of the Kyoto Prefectural University of Medicine (ERB-C-1080).

3. Results

The median Tg level for all 256 cases was 342 ng/mL (range: 0.3–139,000 ng/mL) (clinical reference value: ≤33.7 ng/mL). The median weight of the thyroid gland was 14.5 g (range: 1.1–69 g). The mean T3 level was 270.9 ± 205.6 (clinical reference value: 58–159 ng/dL), and the mean T4 level was 7.01 ± 4.62 (clinical reference value: 4.87–11.72 μg/dL) (Table 1).

3.1. Effect of cervical compression on the Tg level

There were 11 cases in which it was concluded that neck compression had occurred based on autopsy findings and other information. The mean Tg level was 3155 ng/mL (range: 179–16,500 ng/mL) in these cases. In the remaining 245 patients without neck compression, the mean Tg level was 4160 ng/mL (range: 3.2–89,000 ng/mL). There was no significant difference between the mean Tg levels of the 2 groups.

There were no significant differences in Tg, T3, or T4 levels, or the weight of the thyroid gland between the groups with and without neck compression (Table 2).

3.2. Cut-off value for the diagnosis of neck compression

Of the 245 cases without neck compression, only 113 (46.1%) had Tg levels of < 200 ng/mL. On the other hand, of the 11 cases with neck compression only one (9.1%) had a Tg level of < 200 ng/mL.

In a comparison of the cases with Tg levels of ≥200 ng/mL and those with Tg levels of < 200 ng/mL, it was found that the frequency of neck compression was significantly higher among the cases with Tg levels of ≥200 ng/mL ($P > 0.05$).

The mean weight of the thyroid gland was 16.4 ± 8.75 g in the group with Tg levels of ≥200 ng/mL and 15.4 ± 7.13 g in the group with Tg levels of < 200 ng/mL. There was no significant difference in the mean weight of the thyroid gland between these groups (Table 3).

3.3. Causes of death and Tg levels

Table 4 shows the frequency of high Tg levels among various causes of death. There was a tendency for high Tg levels to be more common among the cases in which the cause of death was asphyxia or hypothermia. However, only the cases involving asphyxia due to neck compression exhibited a significantly increased frequency of high Tg levels.

3.4. Effects of the blood sampling site on the Tg level

The Tg levels of right heart blood, left heart blood, whole blood, and peripheral blood differed significantly in the 30 cases without cervical compression ($P < 0.05$). High Tg levels were detected in whole blood and right heart blood, and the median values of these samples were nearly 10 times higher than those of left cardiac blood and peripheral blood (Table 5).

4. Discussion

In our study of 256 cases, it is considered that the fact that there were a large number of cases involving high Tg levels among both the cases with and without neck compression explains why there was no significant difference in the Tg level of right heart blood between the cases with and without neck compression.

Various blood biochemical parameters are reported to change after death.[10–15] In the present study, there was no relationship between the postmortem time and Tg level (data not shown). This is because the postmortem times of our cases were limited to a maximum of 48 h, and Tg levels are reported to remain stable after death.[16] Therefore, the high Tg levels detected in some of our cases were not considered to have been caused by postmortem changes.

Recently, Hayakawa et al.[9] reported that the Tg level of right heart blood was elevated in 19 of 44 (43%) autopsy cases that did not involve neck compression. In our study, it was elevated in 132 of 245 (53.9%)

Table 1
Tg, T3, and T4 levels and weight of the thyroid gland in this study.

	T3 (ng/dL)	T4 (μg/dL)	Tg (ng/mL)	Weight of thyroid gland (g)
Mean ± SD (median)	270.9 ± 205.6 (157)	7.01 ± 4.62 (6.8)	3686 ± 10,868 (342)	15.9 ± 8.1 (14.5)

n = 256.