Elevation of blood viscosity as a putative risk factor for sudden death during bathing

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Deaths which occur in the bathtub are common in Japan, amounting to approximately 14000 per year. To clarify the risk of sudden death while taking a bath, we analyzed the physiological changes induced by bathing. Thirty volunteers participated in our study, and hemorheological factors such as whole blood and plasma viscosity, hematocrit, total protein and fibrinogen, etc. were measured before and after taking a bath at 42°C for 20 minutes. A body weight reduction was observed after bathing (mean 550 g) and whole blood viscosity levels were significantly increased for all the cases with a mean of 0.46 cP (p<0.0001). Whole blood viscosity reached over 5.5 cP in four cases. This viscosity value corresponds to the highly elevated levels seen in cerebral ischemic disease patients, and could be explained by their increased hematocrit. We also investigated the hemoglobin levels by analyzing the postmortem blood of 10 deaths during bathing and 11 acute deaths stemming from traffic accidents. These results suggest that the dehydration induced by bathing can cause serious hyperviscosity, which may sometimes be fatal for the elderly people with less compensative ability.

Key words: Sudden death, bathtub death, blood viscosity, hematocrit

1. Introduction

Japanese people enjoy taking baths and their bathing style greatly differs from the Western style. One Japanese investigation found that Japanese people take baths 5 times a week during the winter for an average of 26 minutes each time, and spend 11 minutes in the bathtub filled with hot water1. Elderly people in Japan prefer bathing in hot water above 42°C, while Europeans and Americans prefer 40°C or lower. The estimated number of sudden deaths during bathing is approximately 14000 per year in Japan2.

In countries other than Japan, the major causes of fatalities in the bathtub are suicide or carbon monoxide poisoning rather than natural disease3. A study from Finland pointed out the risk of sudden death due to arrhythmia in a
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sauna bath. In some reports, increased heart rate and ejection fraction of the left ventricle, elevated body temperature, increased perspiration, and decreased mean blood pressure were observed during bathing. The major causes of fatalities in the bathtub are due to hyperthermia and decreased blood pressure that leads to unconsciousness resulting in drowning.

In this report, we investigated the changes in hemorheological factors (i.e. whole blood viscosity, hematocrit, plasma viscosity, total protein, fibrinogen, etc.) in 30 adult males after bathing. A postmortem blood viscosity study comparing sudden deaths during bathing and acute traffic accidental deaths was also performed. In this study, the increased level of hemorheological factors during bathing is demonstrated, and its relationship with the risk of sudden death is discussed.

2. Materials and methods

2.1 Hemorheologic study of the blood before and after bathing

2.1.1 Subjects and protocols

Thirty healthy male volunteers between 20 and 50 years of age (20 to 47-year-old, median of 21) participated in this study. The volunteers were not taking any medications. Informed written consent was obtained from each subject according to the Tokai University School of Medicine Ethic Committee’s Guidelines. Prior to bathing, a doctor examined each subject’s pulse rate and blood pressure to determine the suitability of each volunteer for participating in this study considering the risk factors involved in bathing. Experimental bathing (42°C) was performed for 20 minutes with the temperature in the bathroom set to 20°C. Hemodynamic conditions of the volunteers were measured. They were all within normal ranges. The depth of water in the bath was about 45-50 cm. The water temperature was maintained by thermostat and the room temperature was maintained by air conditioner. The blood pressure and pulse rate were measured in a sitting position outside the bathtub.

The volunteers were supplied with sufficient hydration after the experiment was over and carefully followed up afterward by the doctor. The blood pressure and pulse rate were measured in a sitting position outside the bathtub.

2.1.2 Blood sampling and measurement of hemorheologic factors

Blood samples were collected by the puncturing the antecubal vein and withdrawing 15 ml of blood before and after bathing. A part of each sample (10 ml) was anticoagulated with 12.5 U/ml sodium heparin added for hemorheological studies, and the remaining 5 ml of the sample was used for haemostasis and fibrinogenolysis studied after adding 0.5 ml of 3.8% trisodium citrate solution. The viscosity of the whole blood and plasma was measured at 37°C using a digital cone-plate viscometer (Brookfield, USA).

Whole blood viscosity and plasma viscosity were determined at a share rate of 225 sec^{-1}, which corresponds to the wall shear rate of arteries for a man during exercise.

The hematocrit level was measured by the hematocrit centrifuge. Blood was collected from the peripheral vein and centrifuged to obtain plasma, which was stored at -80°C for subsequent analysis. The plasma concentrations of cholesterol and triglyceride were determined enzymatically by Determiner L-TC and Determiner L-TG (Kyowa Medix) on a Roche Auto-Analyzer COBAS MIRA plus (Roche, USA). The plasma concentrations of HDL-C and LDL-C were determined enzymatically by Cholestest N HDL and Cholestest N LDL (Dai-ichi Pure chemicals, Tokyo) and by Roche Auto-Analyzer COBAS MIRA plus (Roche, USA). The plasma concentration of total protein was determined enzymatically by the Biuret method (Wako, Osaka) and by Roche Auto-Analyzer COBAS MIRA.
plus (Roche, USA). The plasma concentration of fibrinogen was determined by automated coagulation method using Thrombocheck Fib (L) (Sysmex, Kobe) and Coagrex-800 (Sysmex, Kobe). Haemostasis and fibrinogenolysis laboratory data were obtained by an automated method in the clinical laboratory.

Antiplasmin activity was detected using the synthetic chromogenic substrate method. D-dimer, α2 plasmin inhibitor-plasmin complex, and total plasminogen activator inhibitor-1 were assayed by latex photometric immunoassay, while thrombin-antithrombin III complex was assayed by enzyme immunoassay.

2.2 Postmortem blood study of sudden deaths

The blood sample was collected from the right atrium, within 24 hours after death. Total hemoglobin levels of postmortem blood samples were measured for 10 victims of sudden death in bath-tub (47 to 88 year-old male with a mean age of 64.2 years) and 11 victims of acute deaths due to traffic accidents (20 to 81 year-old males with a mean age of 55.5 years).

2.3 Statistical analysis

All data are expressed as the mean value ± SD. Statistical analysis was performed using Mann-Whitney's U-test (Statview 5.0 software Abacus Concept Corporation). A value of p<0.05 was considered statistically significant.

3. Results

3.1 Changes in hemorheologic factors before and after bathing

The healthy male volunteers had a mean age of 24.5 ± 7.3 years, ranging from 20 to 50 years. The subjects' average height was 170.7 cm (median: 170 cm), weight was 79.9 kg (median: 71 kg) and BMI was 26.4 (median: 25). Changes in the hemorheologic factors before and after bathing are summarized in Table 1. For 28 of the subjects, a reduction in body weight was observed, ranging from 0 to 1000 g (n = 30, mean; 550 g). Whole blood viscosity was increased (0.03 to 0.96, mean; 0.46 cP) after bathing (p<0.0001) for all cases (Fig.1), as well as plasma viscosity (0 to 0.15, mean; 0.59 cP) (p<0.0001), hematocrit level (0 to 4, mean; 1.66%) (p<0.0001), and total protein (-0.8 to 1.8, mean; 0.46 g/dL) (p<0.001), and fibrinogen at -21 to 101 with a mean of 28 mg/dL (p<0.05). For most subjects, total cholesterol, triglyceride, HDL-C, and LDL-C were increased, although not significantly so.

<table>
<thead>
<tr>
<th>Hemorheological factors</th>
<th>Before bathing</th>
<th>After bathing</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole blood viscosity (cP)</td>
<td>4.33±0.46</td>
<td>4.78±0.51</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Plasma viscosity (cP)</td>
<td>1.42±0.09</td>
<td>1.48±0.07</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>45.9±2.98</td>
<td>47.6±3.25</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Total protein (g/dL)</td>
<td>7.92±0.72</td>
<td>8.39±0.57</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>185±40</td>
<td>193±36</td>
<td>-</td>
</tr>
<tr>
<td>Triglyceride (mg/dL)</td>
<td>160±125</td>
<td>166±122</td>
<td>-</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>71±21</td>
<td>75±23</td>
<td>-</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>109±32</td>
<td>113±20</td>
<td>-</td>
</tr>
<tr>
<td>Fibrinogen (mg/dL)</td>
<td>262±65</td>
<td>293±65</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Data are expressed as the mean value ± SD
(-): not statistically significant
There was a significant ($r = 0.67$) correlation between the elevation of whole blood viscosity and hematocrit (Fig.2). There were also significant correlations between whole blood viscosity and hematocrit before ($r = 0.73$) and after ($r = 0.84$) taking a bath (Fig.3). There were also correlations between the elevation of whole blood viscosity and body weight loss and between the elevation of hematocrit and weight loss (Fig.4).

Haemostasis and fibrinogenolysis laboratory data were within normal ranges for almost all subjects, although two showed higher antiplasmin level, one higher $\alpha_2$-plasmin inhibitor-plasmin complex and three higher total plasminogen activator inhibitor-1 after bathing.

3.2 Postmortem blood study of sudden death victims in the bathtub and victims of other acute deaths.

For the autopsy of cases of male bathtub deaths, the hemoglobin levels (8.3-22.0 g/dL, mean: 15.2 g/dL) tended to be higher than those of acute deaths due to traffic accidents at 10-18.7 g/dL, with a mean of 13.8 g/dL. The difference in hemoglobin between bathtub deaths and traffic accident victims was not statistically significant.
4. Discussion

After bathing, a reduction of weight and changes of rheological factors were observed. Whole blood viscosity, plasma viscosity, hematocrit, total protein and fibrinogen levels were significantly increased. The mean whole blood viscosity was 4.33 ± 0.46 cP before bathing, which was almost the same as that of our previous study which involved 181 males. Hyperviscosity has been suggested to be a major risk factor for stroke. Increased blood viscosity causes decreased blood flow to the peripheral organs in addition to significantly decreased brain blood-flow. In our study, the viscosity was increased to more than 5.00 cP after bathing in eight subjects. Furthermore, in four subjects, the viscosity rose to a very high level, higher than 5.50 cP, which corresponds to the elevated viscosity levels observed by Wong et al. for 127 cerebral infarction patients. Elevated blood viscosity is also a major risk factor for ischemic heart disease.

A higher viscosity in cerebral infarction patients has been attributed to elevated fibrinogen in some reports, while others have emphasized the risk of increased hematocrit levels. Whole blood viscosity is mainly determined by hematocrit and serological factors. The present data indicated that whole blood viscosity was strongly correlated with the hematocrit level. In our study, increased whole blood viscosity (>0.2 cP) was observed for 24 subjects. However, the increase in the plasma viscosity for these cases accounts for only a small fraction of that of the whole blood viscosity in all these cases. The mean ratio of the increase of plasma and the whole blood viscosity was 12.3 ± 9.5%. Thus, it is assumed that the increase in the hematocrit level plays a major role in the elevation of whole blood viscosity observed after bathing.

Increased hematocrit level resulting in hyperviscosity may be due to dehydration, which is consistent with the reduction in weight observed after bathing in our study. Perspiration increases when taking a bath because of the high temperature and moisture, increased cardiac output, and dilatation of the peripheral blood vessels. Hyperviscosity is also a major risk factor for ischemic heart disease.

In Japan, Kuroyanagi et al. reported that hyperviscosity related illnesses such as ischemic heart and cerebral disease account respectively for 37.8% and 2.1% of sudden deaths that occur in the bathtub. In his report, 28.8% of the cases were simply diagnosed as drowning, but as they mentioned, many drowning cases with a hidden cause such as transient ischemic attack were
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Our postmortem analysis revealed that the mean hemoglobin levels of the victims who died in the bathtub (15.2 g/dl) tend to be higher than those of the victims of acute death due to traffic accidents, which was 13.8 g/dl. It was also higher than that of the 181 acute deaths reported by Fujita et al. which was 12.4 g/dl. These data suggested that the death during bathing is accompanied by elevation of hematocrit at the time of death.

Four subjects showed highly elevated whole blood viscosity of more than 5.5 cP after bathing, corresponding to the mean viscosity levels of cerebral infarction patients. The common feature among these four was a relatively high hematocrit level even before bathing; their levels were 51.0%, 51.4%, 52.0% and 52%

Although bathing is an effective activity for keeping the body clean and is relaxing, it induces acute hyper-dehydration resulting in blood hyperviscosity. We analyzed these changes using young males for ethical reasons. These changes may have been more serious in older people with less compensation ability. In fact, most bathtub deaths are observed in the elderly, at a mean age of 74.3 ± 13.8 years.

In summary, we have provided evidence that those with high whole blood viscosity and a high hematocrit level should be aware of these risks associated with bathing. Sufficient intake of water before bathing such as isotonic sports drink may aid in preventing acute dehydration, the major cause of the fatal rheological changes in the elderly.

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