The Effect of Wet Cupping on Serum Lipid Concentrations of Clinically Healthy Young Men: A Randomized Controlled Trial

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ABSTRACT

Objective: The aim of this study was to determine if a reduction in serum lipoproteins, especially LDL cholesterol, is a preventive approach against atherosclerosis. Phlebotomy has been a recommended method to reduce serum lipoprotein levels. The present study was conducted to investigate the effects of wet cupping on serum lipoprotein concentrations.

Subjects and Methods: In this randomized controlled trial, 47 men (18 to 25 years old), without chronic disease or a history of hyperlipidemia and antihyperlipidemic drug consumption were randomly assigned into control (N = 24) and treated (N = 23) groups. Men in the treated group were subjected to wet cupping, whereas men in the control group remained untreated. The serum concentrations of lipids, collected from brachial veins, were determined at the time of wet cupping and then once a week for 3 weeks. Data were analyzed using a repeated measure ANOVA.

Results: A substantial decrease in LDL cholesterol (p < 0.0001) and in the LDL/HDL ratio (p < 0.0001) was found in the treated group compared to the control. There were no significant changes in serum triglyceride between groups (p > 0.05). Although there were no statistically significant variations in total cholesterol and HDL cholesterol (p > 0.05), a 7% decrease in total cholesterol and 3% increase in HDL cholesterol may be clinically important.

Conclusions: Wet cupping may be an effective method of reducing LDL cholesterol in men and consequently may have a preventive effect against atherosclerosis.

INTRODUCTION

Wet cupping is an operation for drawing blood by applying a heated cup to scarified skin. 1 Bleeding and cupping have been used in medicine since ancient times in the treatment of fevers and local inflammatory disorders. Local bleeding was effected by a scarificator or leeches. 2 Bloodletting reached the height of its popularity between 1825 and 1835, particularly in Europe. 3 However, as a result of the introduction of modern scientific methods, bloodletting as an accepted therapeutic practice went out of vogue in the middle of the 19th century. 2 Today, however, bloodletting is being restored in modern medicine as the most effective method of treating the increasingly frequent disorders caused by iron overloading such as polycythemia, hemochromatosis, and porphyria cutanea tarda. 3 The effectiveness of repeated blood donation in lowering blood lipids has been studied. Patients with hyperlipidemia who donated blood and received Gemfibrozil had nearly a twofold reduction in serum total cholesterol, LDL cholesterol, and triglyceride levels by comparison with patients who were treated with Gemfibrozil alone. 4 In other words, prolonged repeated bloodletting is associated with a reduction in cardiovascular events. 5 Because of the preventive effects of lipoprotein-lowering trials on atherosclerosis progression, 6 the present study was conducted to investigate the effects of wet cupping as a method of bloodletting on serum lipids.

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Forty-seven (47) men, 18 to 25 years old, without chronic disease, coagulation disorders, or a history of antilipid drug consumption and with total cholesterol and triglyceride concentrations of less than 250 mg/dL, were randomly assigned into case (N = 23) and control (N = 24) groups. For the duration of the experiment, the subjects were not on a high-energy diet. The men in the control group were subjected to wet cupping. A hygienic procedure was conducted using consistent incisions (seven incisions with approximate depth of 2 mm and length of 1 cm) and sucking (3 minutes) in the interscapular area. On average, a total quantity of 50 mL was collected from each case. Four blood sampling events through brachial veins were conducted, 1 week apart, commencing at the time of wet cupping on both experimental groups. Serum concentrations of triglyceride, total cholesterol, and HDL cholesterol were measured using a chemistry analyzer (Roche Cobas Mira, Roche Instrument Center, Rotkrenz, Switzerland) with the enzymatic methods. Indirect serum concentration of LDL cholesterol was calculated via the equation: \[ \text{total cholesterol} \div (\text{HDL} + \text{TG}/5) \]. For the purposes of this particular preliminary study, the importance of the observation of all of the effects of the wet cupping procedure on lipid parameters was of utmost importance. Therefore, the authors did not include any placebo or blindness for the control group.

### Table 1. Serum Triglyceride, Total Cholesterol, LDL-C and HDL-C Concentrations, and LDL/HDL Ratio

<table>
<thead>
<tr>
<th>Variable</th>
<th>0</th>
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<tr>
<td><strong>Triglyceride (mg/dL)</strong></td>
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<tr>
<td>Case</td>
<td>116.2 ± 57.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>122.5 ± 54.7&lt;sup&gt;a&lt;/sup&gt;</td>
<td>107 ± 49.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>117.9 ± 66.6&lt;sup&gt;a&lt;/sup&gt;</td>
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<td>Control</td>
<td>108.2 ± 48.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>92.3 ± 32.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>88.3 ± 30.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>76.5 ± 25.3&lt;sup&gt;a&lt;/sup&gt;</td>
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<td><strong>Total cholesterol (mg/dL)</strong></td>
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<td>Case</td>
<td>163 ± 39.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>146.5 ± 35.9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>146.3 ± 30.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>138.9 ± 31.5&lt;sup&gt;a&lt;/sup&gt;</td>
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<td>Control</td>
<td>147.3 ± 39.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>141.5 ± 35.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>140.2 ± 31.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>134.4 ± 34.6&lt;sup&gt;a&lt;/sup&gt;</td>
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<td><strong>LDL cholesterol (mg/dL)</strong></td>
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<tr>
<td>Case</td>
<td>96.7 ± 36.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>76.9 ± 36.8&lt;sup&gt;b&lt;/sup&gt;</td>
<td>76.1 ± 32&lt;sup&gt;b&lt;/sup&gt;</td>
<td>70.7 ± 34&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>Control</td>
<td>83.3 ± 35.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>80.4 ± 37.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>74.1 ± 29.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>76.9 ± 31.8&lt;sup&gt;a&lt;/sup&gt;</td>
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<td><strong>HDL cholesterol (mg/dL)</strong></td>
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<tr>
<td>Case</td>
<td>43.2 ± 9.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>45.2 ± 5.9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>47.8 ± 8.9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>44.5 ± 8.8&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td>Control</td>
<td>42.4 ± 9.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>42.6 ± 6.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>45.7 ± 5.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>42.4 ± 6.6&lt;sup&gt;a&lt;/sup&gt;</td>
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<td><strong>LDL/HDL</strong></td>
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<td>Case</td>
<td>2.34 ± 0.21&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.81 ± 0.19&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.7 ± 0.2&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.72 ± 0.22&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>Control</td>
<td>2.07 ± 0.19&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.92 ± 0.18&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.67 ± 0.15&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.85 ± 0.14&lt;sup&gt;b&lt;/sup&gt;</td>
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Values are shown at the onset and 1, 2, and 3 weeks after wet cupping on healthy young men who were subjected to wet cupping (case; N = 23) and others who remained untreated (control; N = 24). Data are presented as mean ± SEM. <sup>a,b</sup>Values with different superscripts within rows differ significantly (p < 0.05).

**MATERIALS AND METHODS**

Forty-seven (47) men, 18 to 25 years old, without chronic disease, coagulation disorders, or a history of antilipid drug consumption and with total cholesterol and triglyceride concentrations of less than 250 mg/dL, were randomly assigned into case (N = 23) and control (N = 24) groups. For the duration of the experiment, the subjects were not on a high-energy diet. The men in the control group were subjected to wet cupping. A hygienic procedure was conducted using consistent incisions (seven incisions with approximate depth of 2 mm and length of 1 cm) and sucking (3 minutes) in the interscapular area. On average, a total quantity of 50 mL was collected from each case. Four blood sampling events through brachial veins were conducted, 1 week apart, commencing at the time of wet cupping on both experimental groups. Serum concentrations of triglyceride, total cholesterol, and HDL cholesterol were measured using a chemistry analyzer (Roche Cobas Mira, Roche Instrument Center, Rotkrenz, Switzerland) with the enzymatic methods. Indirect serum concentration of LDL cholesterol was calculated via the equation: \[ \text{total cholesterol} \div (\text{HDL} + \text{TG}/5) \]. For the purposes of this particular preliminary study, the importance of the observation of all of the effects of the wet cupping procedure on lipid parameters was of utmost importance. Therefore, the authors did not include any placebo or blindness for the control group.

**FIG. 1.** Serum triglyceride concentrations at the onset and 1, 2, and 3 weeks after wet cupping on healthy young men who were subjected to wet cupping (case; N = 23) and others who remained untreated (control; N = 24). Data are presented as mean ± standard error of the mean.

**FIG. 2.** Serum total cholesterol concentrations at the onset and 1, 2, and 3 weeks after wet cupping on healthy young men who were subjected to wet cupping (case; N = 23) and others who remained untreated (control; N = 24). Data are presented as mean ± standard error of the mean.
Blood sampling and the measurement of blood parameters was undertaken by a laboratory technician without bias toward the experimental groups: He had no knowledge of the identity of the groups during the procedures. Data were analyzed using a repeated measures ANOVA in SAS/STAT and presented as mean ± SEM.

RESULTS

Serum concentrations of triglyceride, total cholesterol, LDL and HDL cholesterol, and the LDL/HDL ratio are presented in Table 1.

The concentrations of triglyceride did not show any interaction between bloodletting times and groups \( (p > 0.05) \), whereas this was significantly different between groups \( (p < 0.05; \text{Fig. 1}) \). Total cholesterol serum concentrations did not show any significant interaction between times and groups \( (p > 0.05) \), whereas the differences between times were significant \( (p < 0.001) \). There was significant difference between weeks 0 and 1 \( (p < 0.001) \), 2, and 3 \( (p < 0.01; \text{Fig. 2}) \). However, there was no difference between weeks 1 and 2.

LDL cholesterol produced a significant interaction between times and groups \( (p < 0.002) \) and between times \( (p < 0.0001) \). In treated groups, the significant difference in times was noted between weeks 0 and 1 \( (p < 0.001) \), weeks 0 and 2 \( (p < 0.001) \), and weeks 0 and 3 \( (p < 0.001) \). The difference between weeks 1 and 2, and weeks 2 and 3 was not significant \( (p > 0.05) \). There was no significant difference at any time within the control group \( (p > 0.05) \). Considering the insignificant difference of times one and four within the control group, subtracting times one and four in the treated group revealed a 20% reduction in LDL cholesterol (Fig. 3).

The analysis of HDL cholesterol did not reveal any significant interaction between times and groups \( (p > 0.05) \). The significant increase from week 1 to 2 and decrease from week 2 to 3 was noted in both groups \( (p = 0.002; \text{Fig. 4}) \).

The LDL/HDL ratio did not show any significant interaction between times and groups \( (p = 0.07) \), whereas a significant difference was noticed between times \( (p < 0.0001) \). The significant difference in times of treated group was noted between weeks 0 and 1 \( (p < 0.001) \), weeks 0 and 2 \( (p < 0.001) \), and weeks 0 and 3 \( (p = 0.001) \). In the control group, a significant difference was found between weeks 0 and 2 \( (p = 0.01) \). Considering the insignificant difference of weeks 0 and 3 within the control group, subtracting weeks 0 and 3 in treated group revealed a 17% reduction in the LDL/HDL ratio (Fig. 5).

Blood sampling and the measurement of blood parameters was undertaken by a laboratory technician without bias toward the experimental groups: He had no knowledge of the identity of the groups during the procedures. Data were analyzed using a repeated measures ANOVA in SAS/STAT and presented as mean ± SEM.

FIG. 3. Serum low-density lipoprotein (LDL)-cholesterol concentrations at the onset and 1, 2, and 3 weeks after wet cupping on healthy young men who were subjected to wet cupping (case; \( N = 23 \)) and others who remained untreated (control; \( N = 24 \)). Data are presented as mean ± standard error of the mean.

FIG. 4. Serum high-density lipoprotein (HDL)-cholesterol concentrations at the onset and 1, 2, and 3 weeks after wet cupping on healthy young men who were subjected to wet cupping (case; \( N = 23 \)) and others who remained untreated (control; \( N = 24 \)). Data are presented as mean ± standard error of the mean.

FIG. 5. Serum LDL/HDL [low-density lipoprotein/high-density lipoprotein] ratio at the onset and 1, 2, and 3 weeks after wet cupping on healthy young men who were subjected to wet cupping (case; \( N = 23 \)) and others who remained untreated (control; \( N = 24 \)). Data are presented as mean ± standard error of the mean.
DISCUSSION

The present study was conducted to evaluate the effect of wet cupping on serum lipids concentrations. The results of the study have shown that wet cupping is an effective method in reducing LDL cholesterol and the LDL/HDL ratio. Wet cupping did not show a significant influence on total cholesterol, triglyceride, and HDL cholesterol; therefore, any changes in the LDL/HDL ratio seems to be related to the reduction in LDL cholesterol.

Intensive lipid-lowering therapy is considered as a vaso-protective treatment for selected patients in early stages of coronary atherosclerosis, with the potential of preventing further disease progression. More than 70 clinical trials examining the effects of cholesterol reduction have been reported. These studies demonstrate that lowering LDL cholesterol reduces fatal and nonfatal heart attacks. The treatment of elevated LDL cholesterol can have either of two aims: First, the prevention of complications of atherosclerosis; second, the treatment after complications have occurred. Clinical and experimental data demonstrate that LDL cholesterol shows progressive reduction and may actually induce regression of atherosclerosis lesions. HMG-COA reductase inhibitor treatment was associated with: (1) a 20% decrease in total cholesterol, a 28% decrease in LDL cholesterol, a 13% decrease in triglyceride, and a 5% increase in HDL cholesterol; (2) a 31% decrease in all-cause mortality. Primary prevention goals include LDL-C <130 mg/dL (3.36 mmol L⁻¹), triglyceride <150 mg/dL (1.69 mmol L⁻¹), and HDL-C ≥40 mg/dL (1.03 mmol L⁻¹) for men and ≥50 mg/dL (1.29 mmol L⁻¹) for women. A goal of lowering plasma LDL concentration to <100 mg/dL (2.59 mmol L⁻³) is advocated for coronary heart disease and diabetic patients. However, optimal LDL cholesterol values are <100 mg/dL in both women and men. All high-risk patients with LDL-C >100 mg/dL should be treated with drug therapy with the goal of reducing LDL-C to <100 mg/dL.

According to the results of the present study, the main effect of wet cupping is within the first week. During the next 2 weeks, the changes in lipids are not significant. How long the effect of wet cupping on LDL cholesterol maintains, remains to be investigated. On average, during skin puncture, the amount of cholesterol is 3.9% less than venous blood cholesterol concentration. The effect of wet cupping, as a method of skin puncture plus cupping, on LDL cholesterol does not seem to result from the amount of blood (average 50 mL) collected from the subjects. Therefore, another mechanism rather than the amount of blood volume has to be involved in reduction of LDL cholesterol. The effect of wet cupping on different age groups, hyperlipidemic patients, and women are subjects for further investigations. The authors’ current working hypothesis is the question of how the effect of wet cupping is being exerted. Is it caused by simple cup installation, scarification, or the whole process?

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REFERENCES


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