THE EFFECT OF CUPPING THERAPY ON LIPID PROFILES AND APOLIPOPROTEIN IN HYPERCHOLESTEROLEMIC PATIENTS

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ABSTRACT

Background: Atherogenic dyslipidemia is an elevated level of triglycerides (TG) and small-dense Low-density Lipoprotein (LDL) and low-level of High-Density-Lipoprotein cholesterol (HDL-C) which plays a critical role in atherosclerotic or cardiovascular disease. Cupping therapy *(Al-hijamah*) has been regarded as one of complementary nursing intervention in Indonesia and other countries in removing Causative Pathological Substances (CPS); this includes total cholesterol, LDL, TG and Apolipoprotein. This study aims to determine the effect of cupping therapy on lipid and apolipoprotein profiles in dyslipidemia patients.
Patients and Methods*:* pre-experimental design has been conducted among 40 dyslipidemia respondents. Lipid profiles and apolipoproteins A-I (ApoA-I) and apolipoproteins B (ApoB) were measured either after 10-hours fasting before cupping therapy and 24-hours after cupping therapy. Wilcoxon sign-rank test were used in data analysis.
Results*:* the average of lipid profile (mg/dL) and apolipoprotein (µg/mL) pre vs post cupping as follows: Total Cholesterol (TC) 328 vs 283*; TG 238 vs 204*; HDL 78.5 vs 85*; LDL 195 vs 158*; ApoA-1 0.07 vs 0.67*; ApoB 2.04 vs 1.82*; ApoB/ApoA-1 ratio 30.22 vs 2.93*; cholesterol/HDL ratio 4.06 vs 3.08; TG/HDL ratio 3.01 vs 2.83; LDL/ApoB ratio 90.75 vs 83.82 (*$p < 0.01$).
Conclusion: Cupping therapy reduces total cholesterol, TG, LDL and apoB/ApoA-1 ratio and increases HDL significantly in hypercholesterolemic patients. The cupping therapy has future prospects in the intervention of dyslipidemic patients.

Keywords: Lipid profile, Apolipoproteins, cupping therapy, hypercholesterolemic

INTRODUCTION

Atherogenic dyslipidemia is an elevated levels of triglycerides (TG) and low-density lipoprotein (LDL) and a low levels of high-density lipoprotein (HDL) which can cause death. Hyperlipidemic patients are less obedient in controlling fat consumption and consequently the level of achievement of lipid management targets is low. Increased plasma cholesterol levels result in changes in arterial endothelial permeability that allow migration of lipids, especially LDL particles, into the arterial wall. Circulating monocytes attach to endothelial cells expressing adhesion molecules, such as vascular adhesion molecule-1 (VCAM-1) and selectin, monocytes

acquire the characteristic foamy macrophage. LDL particles are oxidized to become chemoattractants. Elevated LDL is a risk marker for atherosclerotic vascular pathophysiology and aortic stenosis.[1]

One of the community's efforts to carry out lipid-lowering therapy is through cupping therapy.[3] Cupping is a minor excretory surgery that has a medical and scientific basis in cleaning the blood and interstitial space from Causative Pathological Substance (CPS). Pathological substances that cause CPS consist of total cholesterol, LDL, TG and Apolipoprotein-B. Significant reduction in LDL can reduce the risk of cardiovascular disease. The apolipoprotein ratio (Apo-B/apoA-I) represents the balance between Apo-B atherogenic particles and Apo-AI antiatherogenic particles, and this ratio is a marker of cardiovascular risk. Percentage of subjects with an Apo-B/Apo-AI ratio above 0.9 who are at risk of cardiovascular disease. This ratio is characterized by high TG levels and plasma atherogenic index (AIP), LDL/apoB ratio and low Apo-B levels.[2]

Cupping therapy is a traditional method of treatment. Cupping is to draw blood into a cup that is placed on the skin. The mechanism of cupping in lowering the lipid profile is not yet understood. This study reviews biomedical aspects, which so far have taken a holistic approach. A description of the mechanism of cupping has not yet been fully identified. This study identifies a causal relationship, and modifies the theory of the intervention-outcome paradigm. A reverse research strategy was used, as this therapy has been used clinically for thousands of years. Researchers need to understand the theoretical basis underlying therapy as a disease treatment technique.[3] Controversial opinion states that cupping therapy only has a placebo effect. The placebo theory is still believed until a valid mechanism is not known. It is hoped that this research

will open up scientific theories that help apply safe and effective cupping therapy, evidence-based scientific explanations that support cupping therapy.[7] Cupping therapy can also reduce LDL cholesterol and potentially prevent atherosclerosis. Although it has been reported the use of cupping therapy against cholesterol, there is no explanation yet about the potential reduction in apolipoprotein B and the lipid profile that compiles the LDL.

MATERIALS & METHODS

The design of this study was pre-experimental analytic. the intervention group was 40 respondents who had a history of hypercholesterolemia/dyslipidemia and had no chronic disease. Inclusion criteria were every patient who had a history of hypercholesterolemia (total cholesterol above 200 mg/dL). The wet cupping therapy (*Al-hijamah*) is carried out according to the procedure and is carried out by a certified therapist from the Indonesian Cupping Association Standards (PBI).

Blood samples were taken from the vein before and before the wet cupping technique. Respondents fasted 10 hours before sampling, and after that they were asked to break their fast while the therapist prepared the intervention. A second blood sample was taken 24 hours after the intervention. The resulting plasma was measured by measuring the lipid profile using the enzymatic method (Kit Diasys) and proatherogenic using the ELISA technique (Abcam). Calculations are also made through the ratio of Cholesterol/HDL; ApoB/ApoA1, LDL/ApoB and AIP plasma atherogenic index (TG/HDL). Statistical analysis using SPSS version-21 with Wilcoxon test.

RESULT

Respondents in this study were those who had a history of hypercholesterolemia or elevated total cholesterol prior the intervention. The profiles of respondents in this study are shown in table 1. Results Table 2 shows that the levels of cholesterol, triacylglycerol, LDL decreased; HDL increased, Apo A-1 protein levels increased and Apo B protein decreased after Cupping therapy. This causes the Ap B/Apo A-1 ratio to decrease significantly.

Table 1. Profile Respondents

Table I -- Profile Respondens			
Gender	Man	Woman	
	18%	82%	
Age	Adult	Elderly	
	94%	6%	
IMT	Normal	Obesity 1	
	82%	18%	
Cupping therapy experience	1-3 time	4-6 time	≥ 7 time
	69%	23%	8%

Table 2. Profile Lipid and Apolipoprotein level in pre-post cupping therapy among hypercholesterolemia patients (N=40).

Table II -- Profile Lipid and Apolipoprotein level in pre-post cupping therapy among hypercholesterolemia patients (N=40).

Parameter	Pre-cupping Median ± SD (min – max)	Post-cupping Median ± SD (min – max)	Wilcoxon sign-rank test
Cholesterol (mg/dL)	328 ± 53 (228.9 – 450.3)	283 ± 54 (147.2 – 383.6)	***P*= 0.003
Triasilgliserol (mg/dL)	238.2 ± 61.9 (134.8 – 368.5)	204 ± 53,8 (151.1 – 359.8)	***P*= 0.007
HDL (mg/dL)	78.5 ± 10.2 (58.2 – 90.4)	85.2 ± 18 (68.5 – 151.9)	***P*= 0.000
LDL (mg/dL)	195 ± 49 129.5 – 323	158.9 ± 48 21.8 – 232.9	***P*= 0.001
Apo-A1 protein (ng/mL)	0.07 ± 0.026 (0.027 – 0.118)	0.67 ± 0.242 (0.197 – 1.106)	***P*= 0.000
Apo-B protein (ng/mL)	2.04 ± 0.74 (0.82 – 3.55)	1.82 ± 0.71 (0.51 – 3.28)	*P*=1.000
Apo-B/Apo-A1 ratio	30.22 ± 1.66 (25.36 – 33.96)	2.93 ± 0.18 (2.59 – 3.35)	***P*= 0.000
Cholesterol/HDL ratio	4.06 ± 0.79 (2.49 – 6.34)	3,80 ± 0,66 (2.72 – 5.31)	*P*= 0.332
AIP (Triasilgliserol/HDL ratio)	3.01 ± 1.2 (1.48 – 6,2)	2.83 ± 0.83 (1.48 – 5.2)	*P*= 0.104
LDL/Apo B ratio	90.75 ± 98 (33–477)	83.82 ± 104 (41–481)	*P*= 0.0837

**Wilcoxon sign-rank test *P*<0,05

DISCUSSION

The increase in Apo-A1 after cupping intervention indicates that proatherogenic cholesterol is reduced through excretion into the liver and bile salts. The increase of Apo-A1 levels in the plasma will also increase proatherogenic cholesterol levels that will be discharged into bile salts through the liver. HDL synthesis is influenced by Apo-A1, Pre-HDL, and cholesteryl ester transfer protein (CETP). Pre-β-HDL is a form of HDL that induces the release of cholesterol from tissues to form HDL. It has a very important role in the process of cholesterol transport in peripheral tissues.[4]

HDL particles will be synthesized as small newborn particles from the small intestine and liver, are flat and contain Apo-A1. Apo-A1 as a component of HDL that supports the efflux of cholesterol from cells. It is important to maintain cellular cholesterol homeostasis. In addition, through receptors that involve the binding of HDL to cell membrane proteins. The smaller HDL precursors (pre-β-HDL) in tissues take up free cholesterol from cell membranes. Free cholesterol is esterified by the action of LCAT which makes it more hydrophobic. The increase in Apo-A1 is very significant in cupping therapy, it indicates the protection of blood vessels from lipid oxidation and atherosclerosis formation.[5]

The decrease in Apo-B in this study is a good sign because it is proatherogenic. The lower the plasma Apo-B level, the less proatherogenic cholesterol in the blood circulation. Apo-B is the main apolipoprotein of chylomicron particles, VLDL, IDL, and LDL in all tissues. Apo-B in LDL particles acts as a ligand for LDL receptors in various cells. LDL particles are easily internalized

into the subintimal space where they adhere to the proteoglycan matrix, which is oxidized and increases the risk of atherothrombotic events. One Apo-B particle represents one LDL cholesterol. The decrease in the amount of Apo-B in this study showed that cupping therapy could reduce the ABC1 protein which resulted in a 70% reduction in cholesterol, especially a decrease in plasma phospholipids, and if continued to be HDL, almost no LDL.[6] This study can prove that wet cupping therapy can significantly reduce the Apo-B/Apo-A1 ratio in hypercholesterolemic patients. The lower the ratio of Apo-B/Apo-A1 in plasma, the less proatherogenic cholesterol in the blood circulation. Apo-B is a protein involved in fat metabolism and is a major constituent of proteins such as VLDL and LDL lipoproteins. Chylomicrons are lipoprotein particles that carry dietary lipids from the digestive tract, through the bloodstream, to tissues, and especially to the liver. In the liver, the body repackages these dietary lipids and combines them with Apo B-1 to form triglyceride-rich VLDL.[5]

Cholesterol reduction in cupping therapy can control other stable lipid profiles to normal. A diet high in saturated fat and trans unsaturated fat, or genetic factors can cause high blood cholesterol levels. Excess cholesterol is stored in plaque on the walls of blood vessels. This plaque can narrow blood vessels and consequently atherosclerosis, which puts you at risk for heart disease and stroke. Cholesterol decreased in this study can lower LDL and prevent atherosclerosis.[8] The transcription factor HIF-1α activates macrophages in the skin which in turn induces proinflammatory genes such as IL-1, IL-4, IL-6, and TNF-α. Interleukin-6 which plays a role in stimulating the immune response, for example after tissue damage due to cupping, the release of IL-6 will stimulate young macrophage cells to mature and be able to perform phagocytosis. Accelerated migration of macrophages also increased due to IL-6 stimulation. IL-6 particles also

stimulate monocytes to produce inflammatory cytokines that play a role in local and systemic inflammation, thereby accelerating the proliferation and differentiation of macrophages.[9] Total cholesterol levels in the body which include HDL, LDL, and TG levels. In the body, cholesterol is found in the form of free cholesterol and ester (esterified) cholesterol. Normally about two-thirds of total plasma cholesterol is present in the form of esters. About 60-70% of cholesterol is transported by LDL and a small portion (15-25%) is transported by HDL.[10] Control of blood lipid levels, especially LDL reduction using drugs through inhibition of the a HMG-CoA reductase in the liver, thereby lowering total cholesterol levels and increasing the formation of LDL receptors in liver cells. On the surface of hepatocytes there is an increase in LDL transport from blood vessels to liver cells.[7]

In this study showed a significant decrease in TG. Patients with hypercholesterolemia generally cholesterol, and TG and LDL. LDL as the primary atherogenic lipoprotein and the main therapeutic target for coronary heart disease. In general, TG-rich lipoproteins include LDL and LDL. Approximately 50% of patients with this atherogenic lipoprotein disorder have an increased risk of CVD. So that the target of treatment is also a decrease in TG in target hyperlipidemia. Cholesterol and triglycerides are non-polar or insoluble in air. Fat and protein binding transport system (lipoprotein). These complex particles have a central core containing cholesterol esters and triglycerides surrounded by free cholesterol, phospholipids and apolipoproteins.[5] Cholesterol, free fatty acids and triglycerides will be difficult to remove through cupping therapy because their solubility is small so they bind to protein. In addition to its large macromolecule structure that is difficult to penetrate the skin barrier, it is difficult to remove it through cupping therapy. When hypercholesterolemia occurs, the Sterol Binding Protein Element (SREBP) in the

endoplasmic reticulum is inhibited. In this study, it is suspected that through inhibition of SREBP in cells and inhibition of HMG-CoA reductase activation, thereby preventing the mevalonate pathway of intracellular cholesterol synthesis. Blood that comes out through cupping therapy is likely to secrete the transcription factor SREBPs.[11]

HDL has protective properties against heart disease, so that therapeutic intervention efforts are aimed at increasing HDL concentrations. HDL contains the highest proportion of apolipoproteins compared to other lipoproteins. The major apolipoproteins, apoA-I and apoA-II, are secreted into plasma by the liver and intestines. The presence of both slows down atherogenesis and protects against atherosclerosis and its cardioprotective function. HDL removes lipids from macrophage phagocytosis against other lipoproteins. Cells in extrahepatic tissues transport cholesterol from the periphery to the liver for biliary secretion and excretion of cholesterol through feces, although it is only 5% of body lipid.[13] This mechanism is quite effective compared to cupping therapy. HDL is also difficult to cross the skin barrier through cupping therapy. The increase in HDL due to increased Apo-A1 was significantly increased in this study.

TG synthesis is the liver's attempt to store and export fatty acids. The main pathway for TG synthesis is the Glycerol-3-P pathway, which accounts for more than 90% of the total TG synthesis. Decreased TG through inhibition of long chain acyl-CoA esterification to G3P, inhibition of microsomal Glycerol-3-P acyltransferase enzyme. Molecular lysophosphatidic acid is not produced in this reaction and is then acylated so that no phosphatidic acid is formed by acylglycerol-3-phosphate acyltransferases in the ER membrane. PA is not formed, causing cytidine diphosphate diacylglycerol not to be formed due to decreased TG synthesis. The decrease in TG can also be thought to be caused by a decrease in the expression of lipogenic

enzymes in the cupping tissue. Especially decreased expression of a group of enzymes involved in the synthesis of oleic acid, the main component of triglycerides. The decrease in TG occurs due to the rapid release of blood through cupping so that the components needed in the synthesis of TG are reduced. In addition, increased lipolysis is associated with increased carnitine activity via beta-oxidation metabolism. As a result of not forming TG, fatty acids accumulate causing carnitine palmitoyltransferase involved in the transport of FA into the mitochondria for degradation.[13]

The decrease in LDL can also be caused by a decrease in TG synthesis, cholesterol ester transfer protein activity, and liver lipase activity. the mechanism of wet cupping therapy to inhibit cholesterol synthesis and TG effectively inhibits LDL synthesis. Therefore, anything that has predicted TG levels through cupping therapy can increase HDL cholesterol levels and decrease LDL particles. Cupping therapy is effective for cholesterol, TG and LDL to prevent the risk of CVD events. The ratio of AIP (TG/HDL) to a smaller molecular size. This ratio can be used to identify patients with an atherogenic lipid profile and may be relevant for assessing CVD risk.[14]

The Apo B/Apo A1 ratio reflects the balance of proatherogenic and antiatherogenic particles. A higher apo B/apo A1 ratio was associated with a higher risk of all-cause and CVD-related mortality. A higher Apo B/Apo-A1 ratio is an important parameter for predicting the risk of cardiovascular events and causes of death. This overexpression of apoA-I causes atherosclerotic regression. High LDL levels and high Apo-B/Apo-A1 ratios are associated with coronary arteriosclerosis. LDL levels were independently associated with aortic valve calcification but not coronary artery calcification in the familial hypercholesterolemic population.[15] The ratio in this study showed a significant decrease. Apolipoproteins are small molecules and are easily

soluble in water. So through cupping therapy, it is possible to remove it easily, as evidenced by a significant reduction ratio of 10 times compared to before cupping. Cupping is effective for reducing the risk of death from CVD regardless of the respondent's normal BMI (Table 1). This study showed a positive effect of cupping therapy in hypercholesterolemic patients by reducing total cholesterol, triacylglycerides, and LDL and vice versa HDL.

Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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