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The Effect of the Blood Cupping Therapy on High Density Lipoprotein (HDL) and Low Density Lipoprotein (LDL) in Hypercholesterol Patients

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Abstract

Introduction: Hypercholesterol is a high cholesterol level in the blood. Blood cupping therapy is a technique of excreting metabolic waste in the blood through the skin. The study aimed to measure any effect of blood cupping therapy to HDL and LDL cholesterol. **Method**: It was a Quasi-Experimental study using humans as research subjects. Non random consecutive sampling. Patiens with hypercholesterol after 12 hours of fasting were treated with blood cupping, 9 points. Research subjects were 51 men divided into three groups, the cupping group only consisted of 17 men, the drug group without cupping consisted of 17 men, and the cupping and drug consisted 17 men. Collecting data from third of the groups as pre and post data. The data were analyzed by Mann Whitney, Wilcoxon and Kruskal Wallis. The research took place at the Laboratory of Biochemistry, Universitas Jember. **Results**: Measurement of HDL cholesterol on the cupping group was pretest=33.03±3.73; posttest=37.58±6.54; p=0.000. The drug group was pretest=33.40±3.18; posttest=33.56±3.50; p=0.788. The cupping and drug group was pretest=34.32±3.38; posttest=37.61±2.01; p=0.002. Kruskal Wallis test on pretest group was 0.534 and on posttest group was 0.002. Measurement of LDL cholesterol on the cupping group was pretest=154.70±39.68; posttest=123.89±41.86; p=0.000. The drug group was pretest=151.24±44.17; posttest=151.24±44.17; p=0.019. The cupping and drug group was pretest=147.48±62.66; posttest=105.57±57.94; p=0.001. Kruskal Wallis test on pretest group was 0.439 and on posttest group was 0.082. This means that there was no difference in the average pretest and posttest LDL levels in the cupping, drug, cupping and drug groups. Conclusion: The intervention of blood cupping therapy can increase HDL cholesterol level, and reduce LDL cholesterol level. Further research needs to be done to measure the potential prevention of atherosclerosis.

Keywords: *blood cupping; HDL; LDL; cholesterol*

Introduction

Blood cupping therapy or the wet cupping therapy has been used for medication since ancient times. Even Hipocrates used blood cupping in cases of internal drug.[(1)] The long history of blood cupping therapy proves that the blood cupping therapy when performed correctly is a safe and effective method.

There is a misperception in interpreting blood cupping therapy. The depth of the needle into the skin is only 0.05mm. Injuries with such needles do not cause blood flow. Blood comes out after being pulled with a pump of negative strength -200 mmHg.[(2,3)] Actually, blood cupping therapy is not a treatment for removing blood circulation but for removing metabolic waste or

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the so-called causative pathological substances.[4] In other words, blood cupping therapy does not reduce circulating blood volume. The bloodlike substance that comes out of the injury is cholesterol metabolism wastes, old erythrocytes, etc. The blood amount due to the conduct of a correctly performed blood cupping therapy does not reduce hemoglobin.[5]

We have understood that high levels of total blood cholesterol, especially in the form of LDL cholesterol, are a major risk factor for developing coronary heart diseases.[6] Some patients who receive LDL cholesterol reduction therapy still have complaints of coronary heart disease.[7] The aim of the study was to measure effect of blood cupping therapy to HDL and LDL cholesterol. The existence of HDL cholesterol in the reserve cholesterol transport is a mechanism to protect endothelium against the risk of atherosclerosis. HDL has anti-inflammatory, antioxidant, antithrombotic properties. It is also antiaterogenic.[8]

Method

It was a Quasi-Experimental study using humans as research subjects. Non random consecutive sampling. Patiens with hypercholesterol after 12 hours of fasting were treated with blood cupping, 9 points. Research subjects were 51 men divided into three groups, the cupping group only consisted of 17 men, the drug group without cupping consisted of 17 men, and the cupping and drug consisted 17 men. Collecting data from third of the groups as pretest and posttest. The research subjects were selected based on sample inclusion criteria, such as being aged between 45-55 years old, and having total cholesterol> 160mg/dl. After fasting for 12 hours and still consuming statin anti-cholesterol drugs, blood is taken through the cubiti vein as much as 5ml. To measure the HDL and LDL cholesterol, the enzymatic colourimetry method, diasys reagent, Biolyzer100 spectrophotometric device, in units of mg/dl were implemented. Data analysis was performed with the Wilcoxon, Mann Whitney, Kruskall wallis test with a significance level of 5%, in which the data were pretest-posttest and compared fom thid groups. The research was carried out at the Laboratory of Biochemistry, Universitas Jember.

Findings and Discussion

Table 1. Result of analysis HDL level on the cupping, drug, cupping and drug group

Groups	n	Levels HDL Mean±SD Median ± IQR (Min-Max)		p
		Pretest	Posttest	
Cupping	17	33.03 ± 3.73 32.49 ± 5.54 (27.81-39.97)	37.58 ± 6.54 35.64 ± 4.34ab (31.59-55.35)	0.000
Drug	17	33.40 ± 3.18 32.67 ± 5.26 (29.27-39.96)	33.56 ± 3.50 34.56 ± 5.13a (25.92-38.61)	0.788
Cupping and drug	17	34.32 ± 3.38 34.84 ± 5.20 (26.19-39.16)	37.61 ± 2.01 37.57 ± 3.25b (35.11-42.17)	0.002
p		0,534	0.002	

Add : * significan on a = 0,05

IQR = Inter Quartile Range

Measurement of HDL cholesterol on the cupping group was pretest= 33.03±3.73; posttest=37.58±6.54; p=0.000. The drug group was pretest=33.40±3.18; posttest=33.56±3.50; p=0.788. The cupping and drug group was pretest=34.32±3.38; posttest 37.61±2.01; p=0.002. Kruskal Wallis test on pretest group was 0.534 and on posttest group was 0.002.

This data shows an average increase in HDL levels from 33.03 to 37.58. There was an increase of 4.55 point (13.8%).

Measurement of LDL on the cupping group was pretest=154.70±39.68; posttest=123.89±41.86; p=0.000. The drug group was pretest=151.24±44.17; posttest=151.24±44.17; p=0.019. The cupping and drug group was pretest=147.48±62.66; posttest=105.57±57.94 ; p=0.001. Kruskal Wallis test on pretest was 0.439 and on posttest was 0.082. This means that there was no difference in the average pretest and posttest LDL levels in the cupping, drug, cupping and drug groups.

Table 2. Result of analysis LDL Level on the cupping, drug, cupping and drug group

Groups	n	LDL level Mean±SD Median ± IQR (Min-Maks)		p
		Pretest	Posttest	
Cupping	17	154.70 ± 39.68 151.03 ± 79.77 (94.34-214.92)	123.89 ± 41.86 116.45 ± 81.13 (67.45-193.18)	0.000
Drug	17	151.24 ± 44.17 138.34 ± 40.19 (112.16-284.03)	136.40 ± 42.12 130.71 ± 42.40 (83.08-264.89)	0.019
Cupping and Drug	17	147.48 ± 62.66 136.96 ± 28.65 (74.84-337.66)	105.57 ± 57.94 95.41 ± 74.37 (38.08-256.47)	0.001
p		0,439	0.082	

Add : * significan on a = 0,05

IQR = Inter Quartile Range

This data shows an a decrease in LDL levels from an average of 154 to 123. There was a decrease of 31 point (20%).

The National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) guidelines recommend checking non-HDL cholesterol levels containing apolipoprotein B when TG levels elevated.[9]

The main action of statin anti cholesterol drugs in reducing blood cholesterol is inhibiting HMG CoA reductase, which reduces the concentration of hepatocyte cholesterol and stimulates absorption of liver LDL cholesterol from the circulation.[10] However, there is an increasing evidence stating that statins can also have pleiotropic effects. Statins can alter the synthesis and metabolism of lipoproteins and accelerate the degradation of intracellular apolipoprotein B, resulting in a reduction in apopoprotein B secretion.[11] The Atorvastatin Comparative Cholesterol Efficacy and Safety Study

(ACCESS) in patients with hypercholesterolaemia reported reductions in plasma apopoprotein B levels following statin therapy.[12]

This measurement follows the research conducted by Saryono[13], that blood cupping therapy can reduce cholesterol levels. As a result of keratinocyte enrichment in the skin the body can experience hypoxia and induce Hipoxia Inducible Factor (HIF-1α) as a self-defense effort.[14] HIF-1α will activate macrophages in the skin which subsequently induce proinflammatory genes such as IL-1. IL-4, IL-6 and TNF-α.[3] Interleukin-6 which is secreted by macrophages acts to stimulate the body's immune response, for example after trauma or tissue damage that leads to inflammation. The release of IL-6 stimulates young macrophage cells to mature and be able to do phagocytosis more efficiently. IL-6 also stimulates monocytes to produce inflammatory cytokines that play a role in local and systemic inflammation, resulting in accelerated proliferation and differentiation of macrophages.[1]

LDL is a cholesterol source for extrahepatic tissue. When LDL is very excessive, its capture system will be saturated so that excessive LDL can be taken by macrophages. Some LDL cholesterol is captured by macrophages before being oxidized. The more LDL cholesterol levels exist in the plasma, the more cells will be captured by macrophages. Furthermore, macrophages will experience efflux and nascent HDL will approach the macrophage to take LDL cholesterol. Then, nascent HDL becomes adult HDL. After taking cholesterol free from macrophage cells, free cholesterol will be esterified to cholesterol ester by the enzyme Lechitin Cholesterol Acyl Tranferase (LCAT). So HDL here serves as an absorber of LDL cholesterol from macrophages and as a carrier of LDL cholesterol back to the liver so that cholesterol levels in plasma decrease.[7]

According to El-Sayed et al.[4], blood cupping therapy is an excretory minor surgical procedure that has medical and scientific basis for cleaning blood and interstitial space from cholesterol causative pathological substanses (CPS) as metabolic waste production. Many studies have reported that blood cupping therapy can reduce LDL cholesterol. HDL cholesterol functions as an absorber of LDL cholesterol from macrophages and as an LDL cholesterol carrier back to the liver with the help of pre-HDL.[15] Pre-HDL has a role in the reverse cholesterol transport to increase excess cholesterol efflux from peripheral tissue back to the liver to be excreted through bile. The acceleration of migration of macrophages also increases due to stimulation of IL-6.[16,17]

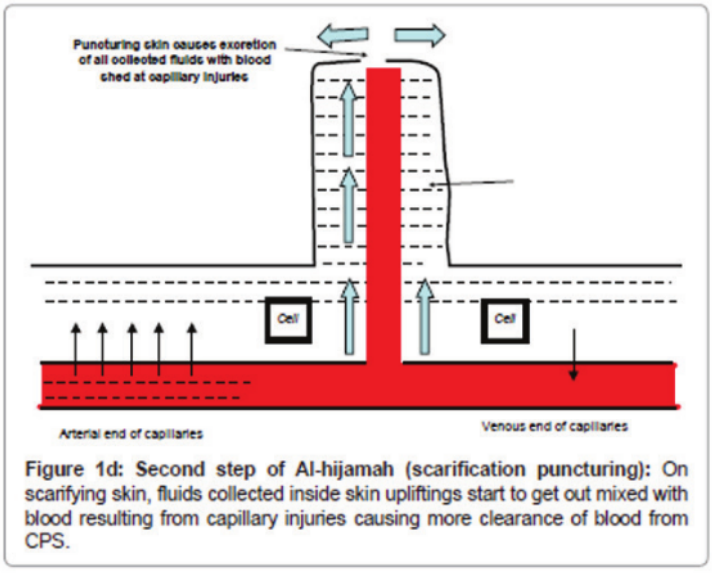
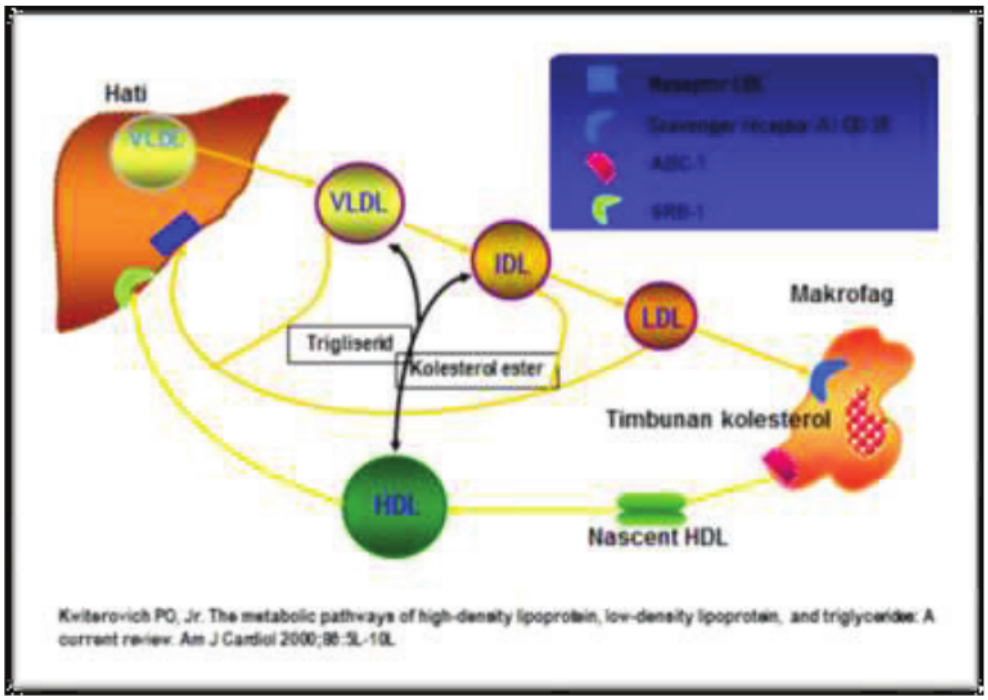


Figure 1d: Second step of Al-hijamah (scarification puncturing): On scarifying skin, fluids collected inside skin upliftings start to get out mixed with blood resulting from capillary injuries causing more clearance of blood from CPS.

Figure 1. The process of taking out metabolic waste from the blood

Source: Elsayed, 2013[4]

HDL is released as small cholesterol-poor particles containing apolipoprotein (apo) A, C, and E, and is called HDL nascent. Nascent HDL comes from the small intestine and liver, has a flat shape and contains apolipoprotein A1. Nascent HDL will approach macrophages to take cholesterol stored in macrophages. After taking cholesterol in macrophages, the nascent HDL turns into a rounded adult HDL. In order to be retrieved by nascent HDL, cholesterol (free cholesterol) on the inside of the macrophage must be brought to the surface of the macrophage cell membrane by a transporter called Adenosine Triphosphate-Binding Cassette Transorter-1 (ABC-1). After taking free cholesterol from macrophage cells, free cholesterol is esterified into cholesterol ester by the enzyme Lechitin Cholesterol Acyl Tranferase (LCAT). Furthermore, some cholesterol esters carried by HDL take two pathways. The first path is to the heart and is captured by a class B type 1 receptor known as SR-B1. The second pathway is the cholesterol esters in HDL will be exchanged with TG from VLDL and IDL with the help of Cholesterol Esters Transfer Protein (CETP). Thus the function of HDL as a cholesterol absorber from macrophages has two pathways namely direct to the liver and indirect pathways through VLDL and IDL to carry cholesterol back to the liver.



Source: Kwiterovich PO, 2000[18]

Figure 2. Reverse Cholesterot Transport Pathway

Metabolism of apolipoprotein is related to the development of atherosclerosis. Increased levels of apolipoprotein B or low levels of apolipoprotein A-I related to cardiovascular disease. Because apolipoprotein B and apolipoprotein A-I have opposite effects namely atherogenic and atheroprotective.[19]

Currently, the inflammatory mediators implicated in the pathogenesis of atherosclerosis include cytokines, chemokines, vasoactive molecules and growth factors. The anti-inflammatory effects of statins are attributed to multifaceted mechanisms including inhibition of cell cycle progression, induction of apoptosis, reduction of cyclooxygenase-2 activity and a biphasic, dose-dependent effect on angiogenesis.[1]

Cholesterol in nascent HDL is then esterified as fatty acids derived from lecithin by lecithin acyl cholesterol

transferase (LCAT) and its co-factor, apo A-1, producing spherical, mature HDL particles. Cholesterol esters in the HDL nucleus are then returned to the liver, either through the interaction of HDL with SR-B1 receptors, or transferred to lipoproteins containing B apo by cholesterol transfer proteins (CETP). (Reproduced with permission from H. Bryan Brewer Jr., personal communication, National Heart, Lung, and Blood Institute.[18]

Conclusion

The intervention of blood cupping therapy therapy has the potential to reduce levels of Apolipoprotein-B and total cholesterol in the blood. Bloodcupping therapy can be considered as an intervention that can reduce cholesterol, in addition to the use of anti-cholesterol drugs. Further research needs to be done to measure the potential prevention of atherosclerosis.

The main effect of statins decreasing the serum level of low-density lipoprotein (LDL) cholesterol, due to the inhibition of intracellular cholesterol biosynthesis. A minor effect is the decrease of serum triglycerides. Statins inhibit HMG-CoA reductase and decrease the production of mevalonate, geranyl pyrophosphate, and farnesyl pyrophosphate, and subsequent products on the way to construction of the cholesterol molecule. Thus, statins could inhibit inflammation, by inhibition of the cholesterol pathway and intracellularly interfering with Ras superfamily protein function. Mutations in transporter molecules or receptors can lead to an accumulation of cholesterol and a breakdown in the normal process of reverse cholesterol transport and cholesterol metabolism.

Ethical Clearance: The research ethical approval was obtained from the Ethics Committee of the University of Jember in Agustus 2019

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