



Reduce The Risk of Atherosclerosis through the wet Cupping Therapy

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Abstract

Hypercholesterolemia is a high level of cholesterol in the blood. Patients must take anti-cholesterol drugs for a long time, so they are at risk of experiencing side effects from the drug. Apo-B and total cholesterol are indicators of cholesterol levels in the blood. Wet cupping therapy is a method of excreting metabolic waste in the blood through the surface of the skin. The study aims to prove the potential of wet cupping therapy as a complementary therapy to reduce Apo-B and total cholesterol.

Method: *This research is Quasy experimental research using humans as research subjects. The dependent variable is Apo-B, and total cholesterol gave wet cupping treatment. Cupping is done twice, 7 points, using a G21 needle. A large sample of 32 people with hypercholesterolemia divided into treatment groups and control groups. The research subjects were selected based on sample inclusion criteria. After 12 hours of fasting and still taking simvastatin, blood was taken through 5ml of the brachial vein. Put into a 2ml purple tube containing EDTA the rest inserted in a red tube. Apo-B measurement using ELISA sandwich method, elabscience reagent, Biopharma ELISA reader tool, in units of ng/ml. Total cholesterol uses the enzymatic colorimetry method, diasys reagent, Biolyzer100 spectrophotometry, in mg/dl units. Data analysis was carried out with the Wilcoxon Signed Ranks Test with a significance level of 5% ($\alpha = 0.05$), the pre-data compared with the post data.*

Results: *A significant reduction in Apo-B measurements with p-value 0.000 ($\alpha < 0.05$), SD 42. A significant reduction also occurred in the total cholesterol group. Obtained p-value 0.005 ($\alpha < 0.05$) SD 0.23.*

Conclusion: *Intervention of wet cupping therapy can reduce Apo-B levels and total cholesterol in the blood. Further research needs to be done to measure the potential for prevention of atherosclerosis.*

Keywords: *wet cupping, Apo-B, total cholesterol, blood.*

Background

Cupping has been used in medicine since ancient times. Even Hippocrates uses cupping in cases of internal disease (Mahdavi et al. 2012). The duration of this cupping history proves that cupping done correctly is safe and effective. There

is a misperception in interpreting wet cupping. The needle depth of the skin is only 0.05mm. The wound with the needle does not cause blood to bleed. New blood comes out after being withdrawn with a 200mmHg negative power pump (Subadi, I.2014). Cupping is not an act of

removing blood but removing metabolic waste called causative pathological substances (El-sayed 2013). In other words, wet cupping does not reduce circulating blood volume. The blood coming out of the wound is "bloodlike" which trashes cholesterol metabolism, old erythrocytes, etc. The amount of cupping blood done correctly does not reduce hemoglobin (Mouradet al. 2016). The study aims to prove the potential of wet cupping therapy as a complementary therapy to reduce Apo-B and total cholesterol.

Cholesterol is present in tissues and plasma lipoproteins in the form of free cholesterol or a combination of long chain fatty acids as cholesterol esters. Cholesterol is synthesized in many tissues from acetyl co-A and is removed from the body in the bile as a cholesterol salt. Cholesterol esters are a form of cholesterol storage in almost all body tissues. The main source of cholesterol comes from the synthesis in the body itself, namely endogenous cholesterol and from foods known as exogenous cholesterol. Acetyl CoA is the source of all carbon atoms in cholesterol (Murray et al. 2009).

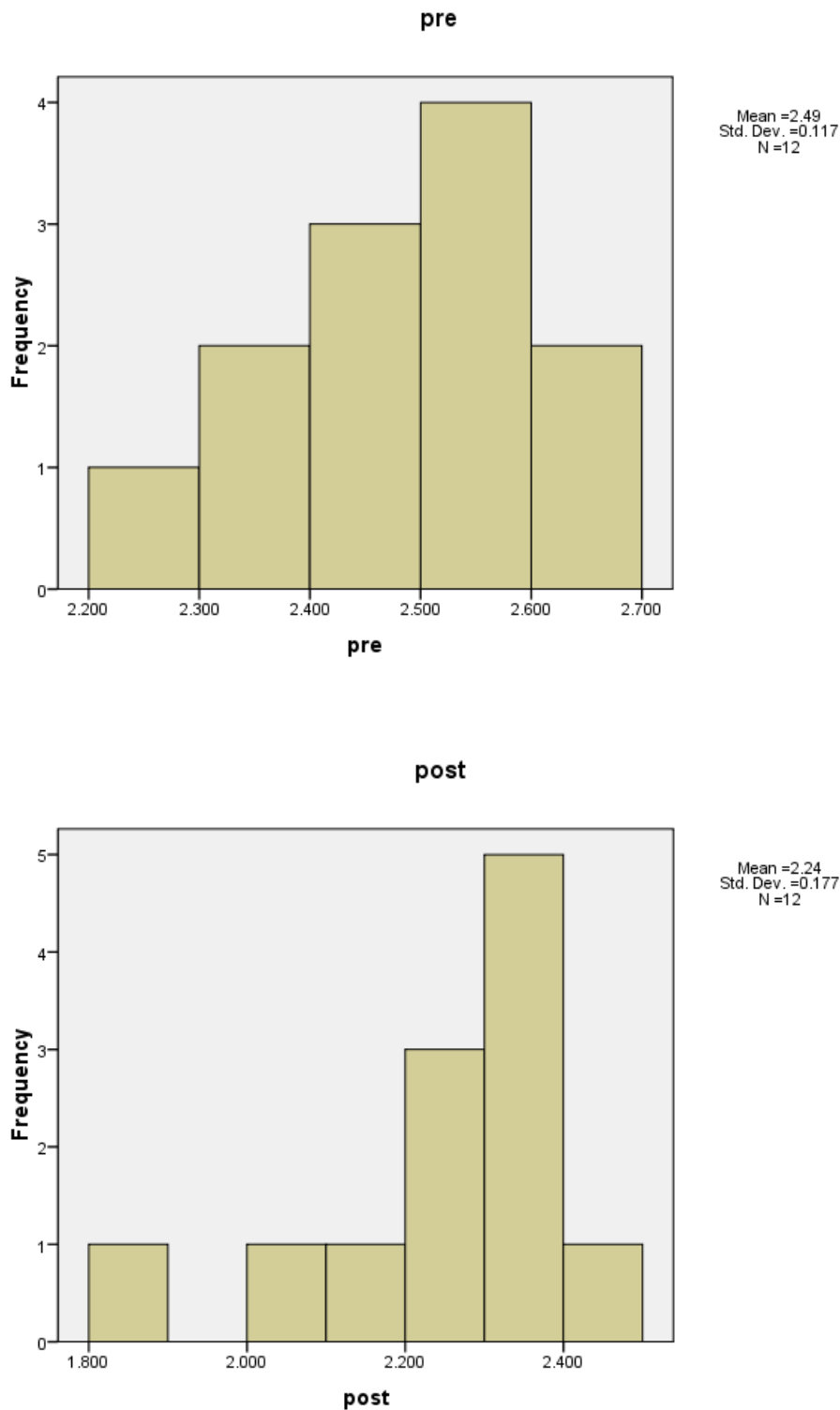
Cholesterol is not soluble in blood fluids, for it to be sent to the whole body needs to be packaged with proteins into particles called lipoproteins, which can be considered as carriers of cholesterol in the blood. The main proteins that makeup LDL are Apo-B (Apolipoprotein-B) (Walldius G, and Jungner I. 2004). In contrast, HDL in its operation clears excess cholesterol from the walls of blood vessels by transporting it back to the liver. The main protein that forms HDL is Apo-a (Apolipoprotein-A). The involvement of HDL cholesterol in reserve cholesterol transport is a mechanism to protect the endothelium against the risk of atherosclerosis. HDL has anti-inflammatory, antioxidant, antithrombotic properties. HDL is also antiatherogenic (Niasariet al. 2007).

Methodology

This research is Quasy experimental research using humans as research subjects. The independent variables were wet cupping therapy, seven coats in the back area, negative pump 5 minutes then wound with a G21 needle as many as 15 punctures with a depth of 0.05mm. The dependent variable is Apo-B and total cholesterol. Measurements are carried out twice, pre and post. A large sample of 33 people with hypercholesterolemia was divided into treatment groups and control groups. The research subjects were selected based on sample inclusion criteria, aged 45-55 years, not suffering from chronic diseases, total cholesterol > 200mg. After 12 hours of fasting and still taking statin anti-cholesterol drugs, blood was taken through 5ml of the brachial vein. Put into a 2ml purple tube containing EDTA the rest was inserted in a red container. Apo-B measurement using ELISA sandwich method, elabscience reagent, Biopharma ELISA reader tool, in units of ng/ml. Total cholesterol uses the enzymatic colorimetry method, diasys reagent, Biolyzer100 spectrophotometry, in mg/dl units. Data analysis was carried out with the Wilcoxon Signed Ranks Test with a significance level of 5% ($\alpha = 0.05$), the pre-data was compared with the post data. The study was conducted at the Biochemistry Laboratory of the Faculty of Medicine, University of Jember. The research ethics test was obtained from the University of Jember Ethics Committee in December 2017

Results and Discussion

Results: measurement of pre-Apo-B data in 12 study subjects obtained mean 2.49 SD 0.117. The post data obtained mean 2.24 SD 0.177. The Wilcoxon Signed Ranks Test was obtained p-value 0.005, because p-value < 0.05, this decrease was significant.

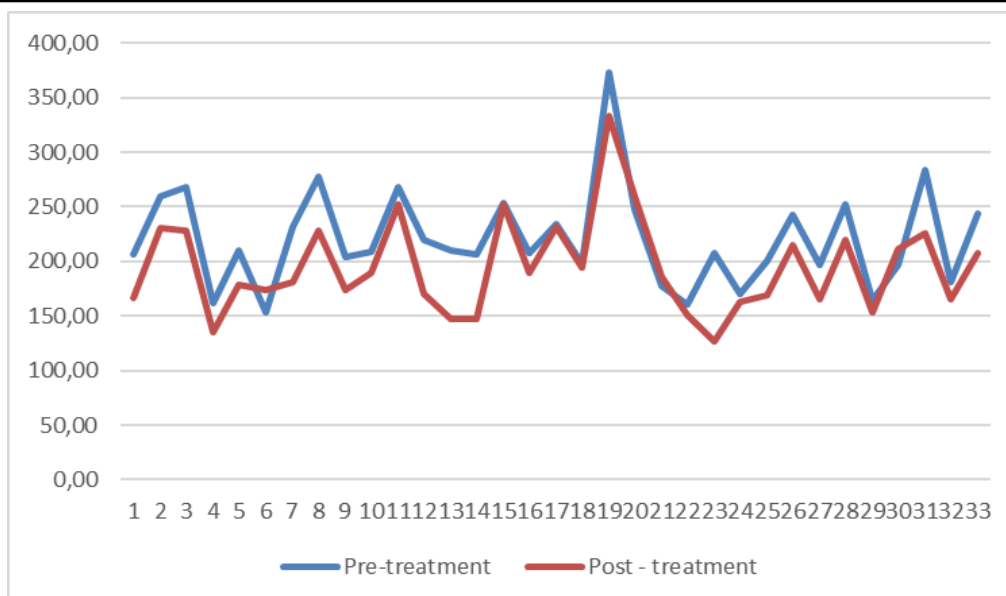


Source: 2018 primary data

Figure 1: Apo-B measurement results pre and post, n = 12 people

Results: measurement of total pre cholesterol data in 33 study subjects obtained mean 226.6 min 153.3 max 373.33. The post data obtained mean 199.3 min 126.6 max 333.3. Wilcoxon Signed

Rank Tests obtained p-value 0.000, Cor 0.772. Because p-value < 0.05, this decrease is meaningful.



Source: 2018 primary data

Figure 2: Results of the measurement of total cholesterol, pre, and post, n = 33 people

This measurement is by research conducted by Saryono (2010), Mustafa L et al. (2012), Niasari M, et al. (2007) that cupping can reduce cholesterol levels. As a result of keratinocyte clotting in the skin will experience hypoxia and induce hypoxia-inducible factor (HIF-1 α) as an effort to self-defense (Ontoseno, 2004). HIF-1 α activates macrophages in the skin which then produces proinflammatory genes such as IL-1, IL-4, IL-6, and TNF- α (Subadi, 2014). Interleukin-6 secreted by macrophages acts to stimulate the 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 (Ilkay, 2005).

LDL (low-density lipoprotein) is a source of cholesterol for an extrahepatic tissue. If LDL is very excessive, the LDL uptake system will be saturated so macrophages can take that excess LDL. Macrophages capture some LDL cholesterol before it is oxidized. The more LDL cholesterol levels in the plasma, the more macrophage cells will be achieved. Furthermore, macrophages will

experience efflux, and nascent HDL will approach the macrophage to take LDL cholesterol.

Furthermore, the nascent HDL becomes adult HDL. After taking free cholesterol from macrophage cells, free cholesterol will be esterified to cholesterol ester by the enzyme Lecithin Cholesterol Acyl Transferase (LCAT). So HDL here functions as an absorbent of LDL cholesterol from macrophages and as a carrier of LDL cholesterol back to the liver so that cholesterol levels in the plasma decrease (Kwiterovic et al. 2000).

According to El-Sayed, et al., (2013), cupping is a minor excretory surgical procedure that has a medical and scientific basis in cleansing the blood and interstitial spaces of causative pathological substances (CPS) cholesterol as the production of metabolic waste. Many research results report that cupping can reduce LDL cholesterol. HDL cholesterol functions as an absorbent of LDL cholesterol from macrophages and as a carrier of LDL cholesterol back to the liver with the help of pre-HDL (Praningsih.2017). Pre β -HDL has a role in the process of transporting back cholesterol (reverse cholesterol transport) which can increase the excess cholesterol efflux from the peripheral tissue back to the liver to be excreted through bile. The acceleration of macrophage migration also

increases due to IL-6 stimulation (Walldius et al.200).

Wet cupping treatment is a non-infectious inflammatory reaction that stimulates the release of chemical mediators including IL-1, IFN- γ , IL-6, IL-8, IL-18 which will activate macrophages so that cholesterol efflux occurs. Wet cupping treatment will enable LCAT (Lecithin Cholesterol Asil Transferase) which converts HDL to HDL3. Cholesterol binds to HDL3 to be carried to the liver and is formed as bile acids which will then be excreted through the intestine (Siadat et al. 2004). Through this process, the cholesterol in the circulation will decrease to be expelled through the gut (Kwiterovicet al. 2000).

Conclusion

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

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