Clinical and Experimental Health Sciences

A Pilot Study on the Effect of Virgin Coconut Oil on Serum Lipid Profile and HS CRP Level Among Post-Acute Coronary Syndrome Patients: A Randomized Controlled Trial

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ABSTRACT

Background: Virgin Coconut Oil (VCO) is beneficial for human health. Introducing VCO is cost-effective in Acute Coronary Syndrome (ACS) management compared to currently available medication. This study examines the influence of VCO on serum lipid profiles and hs CRP level among ACS patients.

Methods: A randomized single-blinded study was conducted from March 2018 to May 2018 among 70 ACS patients. The random sampling technique was used to allocate participants in the control and intervention groups respectively. Participants in Group A received one bottle containing 100 pieces of virgin coconut oil soft gels and were required to ingest 5 soft gels (5ml/5g) twice per day. Group B participants received routine treatment and statin. Changes in serum lipid profiles were identified using a paired t-test and an independent t-test.

Results: The 70 ACS patients were 51 years old and older and were mostly male. The total cholesterol level and Low-Density Lipoprotein Concentration (LDL-C) were reduced from baseline for 30 days. Most participants (68%) reported feeling better doing daily activities although some (22%) reported feeling worse from consuming virgin coconut oil due to its oily odour and slightly oily taste during eructation.

Conclusion: Virgin coconut oil was able to reduce serum lipid profile based on total cholesterol, triglyceride, and LDL-C through its consumption of 5 grams per day for 30 days. Virgin coconut oil may have a positive effect on cholesterol levels and other cardiovascular risk factors.

Keywords: Acute Coronary Syndrome, Cardiovascular Risk Factors, Serum Lipid Profile, Hs Crp Level, Virgin Coconut Oil, Medium Chain Triglyceride

1. INTRODUCTION

Acute coronary syndrome (ACS) occurs due to decreased blood flow in the coronary artery, which hinders the heart from functioning properly. It varies from non-ST-segment elevation myocardial infarction (NSTEMI) to ST-segment elevation myocardial infarction (STEMI) (1). The association between NSTEMI and unstable angina is strongly observed due to their similarity in clinical presentations and pathophysiology origins. NSTEMI is diagnosed by severe myocardial ischemia, evidenced by ECG and the presence of necrosis biomarkers in the circulation (1,2). ACS occurs in the intima, the inner layer of the coronary artery wall. The blood flow to the myocardium is reduced due to the partially or completely blocked arteries. The patient's condition will worsen from a myocardium infarction when the heart suffers damage or death from the lack of nutrients and oxygen.

In Malaysia, ACS has emerged as one of the widespread causes of fatality in recent years. According to the Ministry of Health Malaysia (MOH), cardiovascular disease is the number one cause of death since early 1980s (4). As mentioned by the World Health Organization (WHO), there are several strategies for cardiovascular prevention which reduce morbidity and mortality. Cardiovascular diseases are managed by adhering to these strategies, which are developed by considering the cost-effectiveness and innovativeness of healthcare (4). Introducing VCO is cost-effective in ACS management compared to currently available medication. VCO is a natural resource available in Malaysia which lowers lipid level sand glucose levels, based on results of animal studies (5,6,7,8). However, there are still limited studies on human testing. Further human studies are highly required to confirm the effects of VCO on ACS patients.

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Coconut oil is considered a food source and is used as complementary medicine. It is one of the primary natural products produced from coconut, used as a food ingredient and known as a functional food (9). Researchers have confirmed that there are significant benefits experienced by the intervention group (subjects who consume VCO as an intervention). Several studies on animals claimed that VCO has a valuable benefit on health such as lowering lipid and glucose levels, serving as anti-inflammation and analgesic agent, and increasing the effectiveness of hepatoprotective activity [5,8,10,11,12,13,14).

Even though there exist many animal studies which tested the various effects of VCO (10,15,16,17), similar human studies are still lacking. In addition, despite there being a large body of literature on VCO's ability to reduce serum lipids, only a few of these studies were performed in Malaysia. There is no clear evidence of VCO's effect on ACS patients in Malaysia. The volume of research on the benefit of VCO on ACS patients does not match the magnitude of the problem that is posed in Malaysia. This study, therefore, aims to investigate the impact of VCO on serum lipid profiles among ACS patients in Malaysia.

2. METHODS

This is a single-blind randomized controlled trial and equal randomization study with a ratio of 1:1 for parallel groups. The study was conducted in the medical cardiac ward in University Malaya Medical Centre (UMMC), Malaysia.

2.1. Participants

The targeted population was subjects admitted to the cardiology clinics who met the inclusion criteria and agreed to participate in this study. Patients who are aged 18 to 65, male or female, have ACS, at post-acute phase, and can understand Malay and English were included in this study. However, patients who are pregnant, have uncontrolled hypothyroidism, have a renal failure with creatinine >2mg/dL, and have liver failure were excluded from this study.

2.1.1. Sample Population

ACS consists of both STEMI and non-ST-segment elevation ACS (NSTE-ACS). It demonstrates the existence of anginal syndrome associated with electrocardiographic ST-segment elevation in two or more limbs of at least 0.1 mV or two or more precordial leads of at least 0.2 mV. The newly elevated and developed chest syndromes are explained through unstable angina pectoris irrespective of a significant increase in cardiac-specific troponin I values.

2.1.2. Sampling Method

Only subjects who met the eligibility requirements were randomized into the study. After consent was obtained, block randomization (1:1) and random number table were used for the allocation of treatment. The sample size for the study was calculated based on the formula by Sakpal, 2010 (18). Sample size estimation in a clinical trial with an alpha value of .05, power of 80%, and a clinically significant difference of 0.8 and standard deviation of 0.74 was calculated based on the result of the previous study by Thaw et al. (19)A lipid level change of 0.5 mmol/L was detected in a sample size of 40 subjects, 20 in each group with 5% level of significance and 80% power. Therefore, the sample size needed to be60 subjects (30 per group), considering a dropout rate of 10%.

2.2. Data collection and Randomization

The participants were recruited from the cardiac ward with the supervision and permission of the PPUM Cardiologist. Eligible subjects were given the subject information sheet and consent form. An explanation of the study procedure was given by the researcher. Participants were given sufficient time to decide whether to participate in this study. They were free to withdraw from the study at any time. Initial screening in the ward was performed by the researchers. A random sampling technique was used to assign participants to the control group and the intervention group.

Participants in Group A each received a bottle labelled A containing 100 pieces of VCO soft gels and were instructed to consume 5 capsules of VCO twice per day (0.5ml/capsule = 10 capsules/5 ml) for 30 days. They were also continuing routine treatment, including consuming statin. Group B remained on routine treatment and statin.

After 30 days, there followed up with all subjects for a blood test and anthropometry measurements in the ward. They were instructed to fast for at least 10 hours before the blood test. Group A was instructed to return the labelled bottles and the number of soft gels left was measured to monitor compliance. Participants who experienced any adverse effect would notify the medical officer and would receive appropriate treatment and be allowed to withdraw from the study (however, no adverse effect of VCO had been reported). This is a single-blind study where participants and medical laboratory technicians (outcome assessors) were not assigned the coded key. VCO soft gels were provided by local companies who helped in preparing, packing, and distributing them.

2.3. Preparation of VCO Soft Gel

VCO was extracted through the fermentation process (no heat was applied to retain its beneficial fatty acids). Each ml of VCO contains 0.46gm of lauric acid, C:12. Raw VCO from Company B was certified by UKM UNIPEQ. The local company has agreed to provide soft gels with 0.5ml of VCO (0.23gm of lauric acid) in each. In this study, all participants were supplied with 5ml of VCO with a total 1.15gm of lauric acid (C:12). Each VCO soft gel contained 0.23gm of lauric acid. The soft gel shells are a combination of gelatin, water, and opacifiers such as glycerin or sorbitol. The authors and the clinical supervisor (a cardiologist) inspected the bottles and were satisfied with the amount, appearance, and fatty acid compositions of the VCO soft gels. The researcher received the soft gels in boxes, which were kept

in the National Cardiovascular (NCVD) room located in Level 7, Menara Utama, UMMC. The NCVD room was locked throughout the experiment and the key was kept by the main author.

2.4. Ethical Considerations

The medical research ethics committee from the University of Malaya had provided ethical approval (reference number: 2017.528.5276, 17 July 2018). The study had been registered under ANZCTR with a unique number (ACTRN126.180.01736235).

2.5. Data Analysis

Each randomized group was summarized separately based on baseline characteristics. An intention-to-treat (ITT) analysis was used to fulfill primary outcomes, which included all randomized patients in the groups they were randomly assigned while disregarding anything that happens after randomization. A per-protocol (PP) analysis was used to fulfill secondary outcomes. Patients who reported >75% in the follow-up were included in the PP population.

3. RESULTS

The researcher and a research assistant had conducted the recruitment process following the CONSORT diagram (Figure 1). Only 71 out of 80 patients with ACS were invited and randomized for baseline evaluation. On the 30th day of follow-up, 61 patients attended; 10 patients were lost to follow-up and another two patients did not attend the follow-up due to disease progression. The total compliance was 86%.

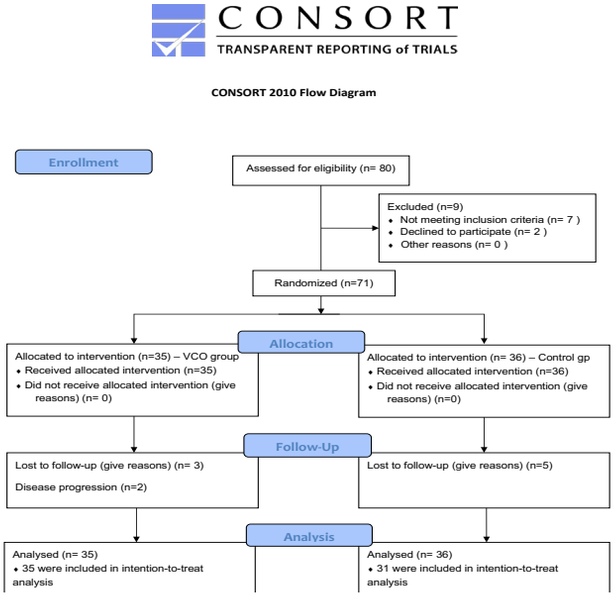

Figure 1. *CONSORT 2010 flow diagram*

Table 1 shows a description of the characteristics of the participants. The overall mean age was 50 and two-thirds of the participants were male. Mean changes for the serum lipid level on the 30th day of follow-up are shown in Table 2, for within each randomized group and comparison between groups. Total cholesterol (TC) concentrations were significantly reduced by 15.1%in the VCO group (95%CI – .19, 0.33, p< 0.001), low-density lipoproteins (LDL-C) and triglyceride concentrations also decreased from baseline to day 30 by 19.1% and 11.8% respectively. High-density lipoproteins concentration (HDL-C) showed no significant increase (but had reduced by 3.4%); however, there was a small increment from baseline to day 30. Serum lipid level of the control group did not show any statistically significant difference from baseline to day 30; however, there was a small clinical reduction in all lipid parameters. All participants showed no changes in serum Hs-CRP concentration.

Table 1. *Demographic and Clinical Characteristics of Participants*

Characteristic	VCO group (n =35) M± SD	Control group (n =35) M± SD	P-value t-test/ X^2/Fisher
Age (year)	50.5 ± 8.9	53.8±10.1	0.18[a]
Gender			
Male	24	23	
Female	11	12	0.10[b]
Race			
Malay	18	16	
Chinese	8	8	0.23[b]
Indian	9	11	
Education level			
Tertiary	17	16	
Secondary	18	19	0.24[b]
Marital status			
Married	30	32	0.61[c]
Single	5	3	
Diagnoses			
Unstable angina	14	16	
NSTEMI	11	10	0.92[b]
STEMI	5	5	
Medications			
Statin+ anticoagulant	5	5	
Statin + antihpt+anticougulant	13	13	0.98[b]
Statin+Antihpt+ Anti DMt+Anticoagulant	12	13	
Baseline Body Weight (kg)	77.5±14.7	74.1±10.5	0.2[a]
NUTRITICS (Nutrition analysis software)			
Total calories	2422 ± (956.9)	2367 ± (784.6)	0.91[a]
Total energy Kilojoules	9941± (4388)	14519 ± (2566)	0.15[a]
Protein %	17.8 ± (2.4)	17.8 ± (2.47)	0.91[a]
Carbohydrate %	56.9± (5.2)	50.5± (6.5)	0.45[a]
Saturated fat %	24.6 ± (5.0)	26.6 ± (4.6)	0.10[a]
Alcohol %	0.6 ± (1.5)	1.1 ± (2.0)	0.72[a]
Level of activities			
• None – little or no regular exercise	0	0	
• Light – 1-3 days per week	14	13	0.92[x]
• Moderate – 1-3 days light week or 5 days week hard	11	12	
• Very active – 6 days week hard	6	6	

[a] *Independent t test;* [b] *Chi Square test;* [c] *Fisher exact test*

Table 2. *Comparison between VCO group and Control group for baseline data and Day 30 (post intervention)*

Clinical parameter	VCO group (n=30)				Control group (n = 31)				Comparison between two groups	
Serum lipid profile	Baseline (Day 0)	Post intervention (Day 30)	t (df)	*p	Baseline (Day 0)	Post intervention (Day 30)	t (df)	*p	t (df)	**p
Total Cholesterol	4.66±1.47	3.94 ± 0.86	3.06 (34)	0.00	4.30±1.13	4.23±1.14	-.55 (35)	0.32	-1.5(69)	0.58
Triglyceride	1.61±0.96	1.42 ± 0.74	2.10 (34)	0.04	1.81±0.89	1.78±1.13	0.15 (35)	0.87	-0.79 (69)	0.87
HDL-C	1.17 ±0.43	1.13 ±0.28	-15 (34)	0.88	1.14±0.60	1.07±0.25	-.74 (35)	0.46	0.87(69)	0.46
LDL-C	2.61 ±1.21	2.11 ±0.71	2.67 (34)	0.01	2.61±0.83	2.47±1.07	1.02 (35)	0.46	-2.1(69)	0.31
HS CRP	2.63 ± 4.2	5.48±18.8	-.90(34)	0.34	1.92±4.6	2.4±5.1	-1.19 (35)	0.24	0.93 (69)	0.35

**Paired t test; ** Independent t test*

Over 30 days, self-reported compliance was high among 87% of the patients in the intervention group, similarly in the statin group (86%). For feedback, 68% of the participants reported feeling better in doing daily activities and 22% reported feeling worse in consuming virgin coconut oil due to oily odour and slightly oily taste during eructation.10% of the patients had reported stomach discomfort during the first two days of VCO consumption but symptom relief on day three. The dietary intake levels were presented using the nutrition analysis software (NUTRITICS) at baseline across the intervention group. Total fat intake, protein, and carbohydrate did not differ among the intervention group. Most participants reported no changes in regular physical activity. The regular physical activity increased by approximately 18% and 16% for patients in the VCO group and control group respectively.

The VCO fatty acid composition used in the intervention is presented in Figure 2. Lauric acid C12:0 (48%) was the major element found in 94% of the saturated fatty acid, followed by myristic acid C14:0 (19%), and palmitic acid C16:0 (9%).

Fatty acid profile		Concentration (%)
C6	Caproic	2.215
C8	Caprylic	12.984
C10	Capric	6.806
C11	Undecanoic	0.028
C12	Lauric	47.280
C13	Tridecanoic	0.030
C14	Myristic	15.803
C15	Pentadecanoic	0.006
C16	Palmitic	6.688
C16:1	Heptadecanoic	0.011
C17	Stearic	0.011
C18	Oleic	1.481
C18: 1n9c	Elaidic	5.073
C18: 1n9t	Linoleic	0.231
C18: 2n6c	Linolelaidic	1.168
C18: 3n6g	γ Linolenic	0.045
C18: 3n3a	α Linolenic	0.007
C20	Cis-11-Eicosenoic	0.039
C20: 1n9	Behenic	0.039
C22	Cis-13,16-Docisadienoic	0.006
C24	Lignoceric	0.020

Figure 2. *Fatty acid composition of VCO (30)*

4. DISCUSSION

In this trial, middle-aged men and women with ACS who were admitted in the cardiology unit in UUMC were randomly assigned to consume 5ml (5grams) of VCO per day while continuing routine treatment and medication for 30 days. The control group would only continue routine treatment and medication. Significant changes were reported in triglyceride and LDL-C concentrations, as well as total cholesterol between the two groups at the end of the trial. VCO consumption also resulted in statistically significant differences in TC, triglyceride, and LDL-C. However, it did not significantly raise HDL-C concentration, though

there was a small increment in pre – and post-means. All lipid parameters showed no statistically significant increase in the control group.

The results agreed with the recent study by Khaw et al. (19), who found that VCO was able to reduce LDL-C and increase HDL-C. However, this study showed no changes in HDL-C. Several previous studies have reported that coconut oil was not favoured in lowering lipid level (20,21). These studies have reported their findings on the effect of coconut oil instead of VCO. There is a lot of difference between regular coconut oil and VCO. The process of extraction is different, and the end product also contains different compositions of fatty acids. In general, the process of extracting coconut oil uses the bleaching and deodorizing processes where chemical agents are added, which makes the oil lose beneficial fatty acid. On the other hand, VCO can be extracted using either the heat-free process of fermentation, cold pressing, or through minimal heat exposure, processes which retain all-important fatty acids that are beneficial to health. The process of extraction plays an important role in why some coconut oil studies seem to have harmful effects on health, particularly on lipid profiles.

In addition, this study showed VCO consumption having a good effect on the lipid profile at only 5ml, containing 1.3grams of lauric acid, compared to previous studies that used 30ml (24grams of lauric acid) [20]. In this study, 1.15grams of lauric acid had significantly reduced the lipid level. The VCO compositions are listed in Figure 2. They contain fatty acid at more than 50%, an ingredient which played a role in lowering

lipid level. As reported by several previous studies, lauric acid contributes to lowering the lipid level.

The transmission of lauric acid is direct to the liver in the form of energy and other metabolites (23,24). Ketone bodies are a type of these metabolites that can be used by extra hepatic tissues in the form of energy. This shows that synthetic MCT oil and dietary fats might have different physiological effects. Due to different experimental doses, feeding periods, and experimental designs, there are inconsistent findings of lauric acid observed on serum cholesterol levels.

There are several different ways in which lauric acid can emerge which moderate serum cholesterol levels. One of the most highly oxidized substance is lauric acid, which contributes minimally to fat accumulation and obesity. Half of the fatty acids in coconut oil are lauric acid. Long-chain fatty acids are predominantly used for allowing the triglyceride structure to be consumed rapidly. Lauric acid is transported directly to the liver using the portal vein (23) Lauric acid is not found in phospholipids, but it might be present minimally in chylomicrons (24)

Across the mitochondrial membrane, lauric acid is rapidly transported by passive diffusion. It is rapidly metabolized in a number of ways in the liver. Two acyl-CoA dehydrogenase enzymes oxidize lauric acid (25). It can metabolize ketone bodies, which are important energy sources in the body for extra hepatic organs (23,24,25,26,27). It is contrary to the study by Vijayakumar et al.(22) who found no difference in blood lipid levels among 200 participants. They reported difficulty in interpreting findings as all participants were on statin therapy. In the current study, the statin group showed some reduction in lipid level; however, there was no statistically significant outcome observed. The statin functions to inhibit Hydroxy-Methyl-Glutaryl-CoA reductase (HMGCR), which acts as the main enzyme of the cholesterol biosynthesis pathway (28,29). This function can lead to several side effects on the human body.

There were several confounding factors like eating out, duration of consumption, and physical activities which could not be assessed accurately since the study was conducted on free-living subjects (Vijayakumar et al. 2016a). The difference in population, research methodology and VCO dosage are the main factors that contribute to the different findings across VCO studies.

The strength of this study is the use of randomized design with a high completion rate (85%) and self-reported dietary compliance over 30 days. However, it was a short-term trial of 30 days of intervention, which is the major limitation. There lacks an examination throughout a longer duration of intervention. The findings of the study are not generalizable for the wider population.

5. CONCLUSION

In this trial, VCO with rich lauric acid appears to have a beneficial effect on serum lipid levels. The actions of lauric acid as MCT in reducing serum lipid and its numerous benefits have been reported in the previous two years and the current study. This study focuses on the individual with coronary problems already treated with a statin. Intervention groups received 5ml of VCO (1.13grams of lauric acid) in soft gel form and would continue on a statin for 30 days whereas the control group would continue solely on a statin. Baseline and 30th-dayfollow-up were recorded and compared. The findings revealed that the group consuming VCO showed a reduction of TC by 15.1% (95%CI – .19, 0.33, $p < 0.001$), LDL-C and triglyceride concentrations also decreased from baseline to day 30 by 19.1% and 11.8% respectively. HDL-C concentration showed an insignificant increase in the control group.

MCT is advantageous for CHD risk, which implies that different dietary sources of fats can be reflected from observational trials. VCO as a natural product can reduce cholesterol parameters better compared to statin. Therefore, medical personnel should consider this natural product as one of the front-line treatments for cardiovascular disease patients as well as a method of prevention for CVD. The existing dietary suggestions cannot be changed by the findings of this study, such as reducing saturated fat intake, but they call for further clarification to develop a scientific relationship between health and dietary fats.

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Conflict of Interest

The authors declare that there are no conflicts of interest regarding the publication of this article.

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