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Effect of Virgin Coconut Oil on Lipid Profile and Other CVD Risk Factors

Research Article

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Abstract

Background & objectives: Effect of coconut oil on lipid profile and atherosclerosis is controversial. Saturated fat appears to have favourable effect on high density lipoproteins (HDL). There are no long-term studies on Virgin coconut oil (VCO) used for human consumption. This study was intended to evaluate its effect on lipids.

Methods & study design: In a single centre non-randomized study subjects with coronary artery diseases (CAD) (group 1) and without proven CAD (group 2) were assigned to receive VCO for 6 months. They were followed up with lipid profile, ApoB/ApoA ratio, antioxidants, high sensitive C-reactive protein (CRP) and Glycosylated haemoglobin (HbA1c) at regular intervals

Results: 42 subjects in group 1 and 32 in group 2 completed 6 months study. There was significant increase in total cholesterol in group 1 ($p = 0.033$). There were no changes (Group 1 and group 2 respectively) in low density lipoproteins (LDL) ($p = 0.12$ and 0.21), Apo B/A ratio ($p = 0.47$ and 0.97), total antioxidants ($p= 0.24$ and 0.57), waist hip ratio ($p= 0.39$ and 0.229) and hs CRP ($p = 0.25$ and 0.011) in either group. There was statistically significant increase in HDL ($p = 0.011$ and 0.025) levels in both groups

Conclusion: VCO does not change lipid profile in an atherogenic direction. VCO increases HDL cholesterol without changes in LDL, Apo B/A ratio, redox potential, inflammatory markers, central obesity and HbA1C. There is an increase in total cholesterol in those with CAD.

Keywords: Virgin coconut oil; Lipids; Atherogenic; Anthropometry; Antioxidants

Introduction

Increased risk of cardiovascular disease has been attributed to consumption of saturated fatty acid rich food among many other reasons [1]. There are many guidelines recommending reduction of SFA for cardiovascular disease prevention [2-5]. Coconut oil and its effect on the composition of lipids in human is still under debate. Coronary artery diseases incidence is very high among population of Kerala state due to many known and unknown reasons [6]. We, in an attempt to understand the biochemical role of this most commonly used oil media in this part of the country, conducted many studies including one randomized study [7-10]. Fatty acid constitution of coconut oil also is debatable, some of the researchers feels that the major fatty acid lauric acid can be no more classified as medium chain fatty acid [11,12]. While others are of different opinion, Even though these are debatable facts 2017American Heart Association Presidential advisory on dietary fats and cardiovascular disease recommends to limit coconut oil with LDL rising property in order to prevent cardiovascular diseases [13]. Fatty acid content in virgin coconut oil (VCO) is essentially the same as in regular oil but additionally there are phytochemicals and the exact role of these ingredients in metabolism is not known. However, the changes in the chemical composition during the bleaching and deodorization

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of the conventional oil are not seen in virgin coconut oil. There are many short-term studies with VCO claiming the beneficial effect and consuming about 30 ml of VCO daily has been reported to have HDL enhancing capability [14-16]. In the context of people consuming the VCO for its beneficial effect, we conducted a long-term study to evaluate its metabolic effect on lipids.

Material & Methods

Approval from scientific committee and institutional ethics committee was obtained as per guidelines. All subjects signed informed consent before entering the study.

Clinical Trials Registry of India CTRI/2015/04/005678.

Setting

Participants were identified from those attending Cardiology outpatient department as well as from the community.

Design

Non -randomized open label study

Sample size

Since it is a pilot study, we recruited 50 subjects in each group

Participants

Group I had proven CAD on medication including statins. CAD was diagnosed by either one of the following. ECG changes consistent with old myocardial infarction, echocardiogram with regional wall motion abnormality, coronary angiogram, myocardial perfusion scan, multi detector CT angiography) on medication including statins. Group II included subjects without known CAD and not on cholesterol reducing medications. Both groups were with low HDL cholesterol (<40 mg /Decilitre in men and less than 50 mg/Decilitre in female). Patients with uncontrolled hypothyroidism, renal failure creatinine >2 mg/dl and liver failure, and other illness limiting the life expectancy <1 year were excluded.

Intervention

On the day of recruitment after signing the informed consent and initial assessment they were allocated 4 grams of VCO for daily consumption. They were called at specific time periods for blood test, anthropometry and for dispensing VCO capsules.

Main outcome measures

The primary outcome was change in serum lipid profile especially the HDL level, ApoB/Apo A ratio, antioxidant levels and C reactive protein. The secondary objectives were the change in anthropometric measures and glycosylated haemoglobin.

VCO product used in this study

The VCO capsules used in this study were manufactured as per norms and have been certified by food safety and standards authority of India and registered under worldwide quality assurance.

Subject compliance

Each subject was interviewed by the dietician at the beginning of the study and a diary was provided to them to note down the capsule consumption. The balance capsules were returned during each visit and tallied with consumption.

Anthropometric measurements

Body mass index: Height was measured with subjects on bare foot using standardized extendable measuring rod. Weight was measured with an electronic Dura weighing machine on empty stomach.

Waist hip ratio: Waist circumference was measured with the patient standing, at the end of expiration midway between the twelfth rib and anterior superior iliac spine as per WHO STEPS protocol. The hip measurement was taken at the level of greater trochanter and the ratio was calculated.

Biochemical parameters

Lipid profile was estimated after 12 hours fasting as per institution protocol in Roche COBAS-8000**.** Apo lipoprotein B, Apo lipoprotein A, and ultrasensitive C reactive protein were estimated by immuno-turbidometry in Roche COBAS-8000 Glycosylated haemoglobin was measured by HPLC in Bio rad- Variant. Four clinically relevant antioxidants catalase (CAT), Glutathione peroxidase (GP) Glutathione redutcase (GR) and total anti-oxidant status were estimated. Catalase was estimated by CELLBIOLABS ,INC ELISA KIT manually. Total antioxidant status (TA), Glutathione reductase and Glutathione peroxidase were estimated by photometric method using RANDOX kit in. in Roche COBAS-8000

Safety outcome measured

Both renal (serum creatinine) and liver functions (aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase) were estimated at one month, three month and at end of the study

Statistical analysis

Statistical Analysis was done using IBM SPSS statistics 20 windows (SPSS Inc., Chicago, USA). For all the continuous variables the results are given in mean ± standard deviation and for categorical variables as percentage.

To compare the mean of numerical variable between cases and control two sample t- test was applied for parameter test. To test the statistical significance of association of categorical variables chi square test was applied.

Probability value (p value) less than 0.05 is considered for statistical significance. To study the statistical significance of difference in mean value of continuous variables from the baseline to different time periods Repeated Measure of ANOVA is used.

Results

We included 50 subjects with known CAD status on medication (Group I) and 39 non-CAD subjects (Group II) in to the study over a period of 2 years. Out of these 43 subjects in group I and 32 in group II completed the study. The baseline characteristics are shown in table 1.

The number of male participants were high in group I and the second group participants were younger than group one. Both groups had low HDL cholesterol. CAD group subjects were on cholesterol

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Table 1: Baseline characteristics.

Parameters	Group I	Group II
Number	43	32
Age	58.65 ± 9.91	45.28 ± 14.01
Gender		
Male (%)	39 (90.7)	15 (46.9)
Female (%)	4 (9.3)	17 (53.1)
Hypertension (%)	52.4	21.9
Dyslipidemia (%)	100	100
Diabetes mellitus (%)	48.8	9.4
Oil consumption at baseline		
Coconut oil (%)	69.8	81
Sunflower oil (%)	9.3	6.3
Coconut oil + sunflower oil (%)	16.3	0
Others (%)	4.6	12.7
Statin therapy	100%	0

reducing statins. Risk factors were high in group I compared to group II (Table1). Both groups were consuming the oil of their choice at the time of recruitment and thereafter. Significant number of subjects was taking coconut oil at baseline.

The total cholesterol levels showed steady increase in group I at the same time there was no change in group II. HDL cholesterol shows statistically significant increase in both groups. The LDL cholesterol in group I subjects, who were consuming the statins was significantly lower compared to non-CAD group (Table 2). LDL Cholesterol was not significantly elevated in either group during the study period. No statistically significant changes occurred in triglycerides and VLDL level.

The best indicator of non-atherogenic versus atherogenic lipids, the ApoB/A ratio did not change during the study (Table 3).

In both groups the main anti-oxidants did not show statically significant changes (Table 4).

Ultra-sensitive CRP did not show statistically significant changes in group I but the high USCRP in group II declined toward the end of the study (Table 5).

Glycosylated haemoglobin levels were little high in CAD group as result of their diabetic status but there were no changes during the study period (Table 6).

The consumption of VCO over 6 months did not show any changes in the anthropometric measurements (Table 7)

There were no changes in the liver function and renal function at different stages of the study (Table 8).

Discussion

Virgin coconut oil extracted directly from the coconut milk which varies significantly from traditional coconut oil with respect to the various phytochemicals and Vitamin E content. Large number of populations started using VCO as food supplement and in this context, we studied the metabolic effect this oil.

Pre-screening for this study has shown that 65% CAD patients uses coconut oil for their dietary purpose and still higher numbers in group II. In both arms subjects with low HDL was recruited to see the effect of VCO on low HDL.

There are no previous long-term studies to understand the dose of VCO capable of making metabolic effect and a short-term study done in the past used 15 grams twice daily for 8 weeks [17]. The dose of 4 grams/day of VCO and the capsule form are selected mainly for long term convenience of the subject. Four grams/day of VCO constitutes about 720 grams of oil over 6-month time equivalent to 6500Kcal which is sufficient to produce metabolic effects.

The groups were significantly different in many aspects regarding the risk factors, diseases and the drugs they are consuming. Group I had higher incidents of risk factors (50% hypertension, 48% diabetes and 100% dyslipidaemia) compared to group II. In group I all the subjects were on different class of statins in spite of achieving desired level for their secondary prevention.

The lipid profile at baseline shows significantly less total cholesterol and LDL in group I because of their statin therapy. The total cholesterol in group I was significantly elevated at the end of 6 months but not in group II. The non -HDL cholesterol, which represents the cholesterol content present in all the atherogenic lipoproteins and was used in Helsinki study [18]. Elevated levels of non-HDL-C in combination with normal levels of LDL-C identify a subset of patients with elevated levels of LDL particle number, elevated apo B concentrations, and LDL of small, dense morphology [19]. In our study the non-HDL cholesterol didn't show significant changes. In group II the non-HDL cholesterol even though high compared to group I didn't change significantly at the end of the study indicating that VCO is not altering the lipids in atherogenic direction in the dose used.

Table 3: Apo B/A ratio.

Parameter		Group 1		
	Baseline Mean ± SD	3 months	6 months	p Value
Apo B/A ratio	0.80 ± 0.42	0.76 ± 0.28	0.72 ± 0.18	0.469
	Group II			
Apo B/A ratio	0.83 ± 0.19	0.83 ± 0.22	0.82 ± 0.27	0.973

Table 2: Lipid profile.

		Group I					Group II	
Parameter	Baseline Mean ± SD	3 months	6 months	p	Baseline	3 months	6 months	p
TC	135.19 ± 27.62	143.14 ± 22.15	144.5 ± 28.96	0.033	171.32 ± 31.51	176.66 ± 30.40	175.91 ± 29.62	0.372
HDL	33.64 ± 5.21	35.33 ± 6.27	35.94 ± 6.28	0.011	39.31 ± 6.00	41.73 ± 6.30	40.1 ± 7.20	0.025
LDL	85.53 ± 20.88	92.06 ± 17.80	90.68 ± 23.36	0.126	110.25 ± 30.36	117.04 ± 31.60	122.92 ± 27.78	0.210
TG	136.72 ± 57.67	143.35 ± 58.03	144.38 ± 62.16	0.644	116.04 ± 55.98	127.70 ± 58.87	135.59 ± 54.50	0.144
VLDL	27.35 ± 11.54	30.66 ± 15.09	29.51 ± 12.69	0.308	24.07 ± 10.74	25.51 ± 11.85	27.00 ± 11.00	0.260
TC-HDL	100.37 ± 28.54	107.80 ± 21.40	108.55 ± 28.42	0.052	132.01 ± 30.14	134.92 ± 29.31	135.81 ± 29.17	0.60

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Table 4: Antioxidants.

Parameter		Group 1					Group II	
	Baseline Mean ± SD	3 months	6 months	p Value	Baseline	3 months	6 months	p Value
CAT	53.06 ± 29.29	50.61 ± 28.16	42.72 ± 18.97	0.147	56.02 ± 17.81	55.49 ± 23.24	55.09 ± 28.70	0.988
GP	5389.51 ± 2039.20	7436.46 ± 9196.14	6724.95 ± 2335.48	0.235	6885.78 ± 2481.10	9630.24 ± 10518.30	7477.04± 2874.29	0.279
GR	53.06 ± 11.16	54.15 ± 13.76	52.86 ± 12.67	0.865	56.43 ± 14.15	57.32 ± 12.28	57.40 ± 13.35	0.948
TA	1.60 ± 0.27	1.57 ± 0.35	1.50 ± 0.24	0.243	1.48 ± 0.26	1.54 ± 0.29	1.47 ± 0.26	0.57

CAT- catalase, GP- glutathione peroxidases, GR- glutathionereductase TA- Total Antixodants

Table 5: Ultra-sensitive CRP.

Parameter		Group I					Group II	
	Baseline Mean ± SD	3 months	6 months	P value	Baseline Mean ± SD	3 months	6 months	P value
URCP	0.44 ± 0.98	0.27 ± 0.32	0.28 ± 0.36	0.25	1.56 ± 1.63	0.69 ± 0.45	1.08 ± 1.05	0.011

Table 6: Glycosylated haemoglobin.

Parameter		Group I					Group II	
	Baseline Mean ± SD	3 months	6 months	p Value	Baseline	3 months	6 months	p Value
HbAIC1	6.71 ± 1.25	6.70 ± 1.22	6.73 ± 1.29	0.938	5.73 ± 0.68	5.78 ± 0.79	5.79 ± 0.88	0.843

Table 7: Waist hip ratio.

Parameter		Group 1			
	Baseline Mean ± SD	1 month	3 months	6 months	p Value
Waist-hip ratio	0.99 ± 0.05	0.99 ± 0.06	0.99 ± 0.04	0.98 ± 0.04	0.39
	Group II				
Waist-hip ratio	0.94 ± 0.05	0.94 ± 0.074	0.96 ± 0.06	0.94 ±0.06	0.23

Table 8A: Safety parameters.

Parameter		Group I			
	Baseline Mean ± SD	1 month	3 months	6 months	p value
ALP	75.44 ± 23.71	74.42 ± 24.95	76.76 ± 22.78	70.30 ± 24.41	0.268
ALT	36.57 ± 21.63	39.38 ± 25.50	36.98 ± 20.17	42.29 ± 23.43	0.086
AST	37.79 ± 19.95	31.83 ± 15.29	30.65 ± 13.34	33.10 ± 15.24	0.53
Creatinine	1.15 ± 0.16	1.13 ± 0.16	1.14± 0.18	1.13 ± 0.18	0.782

Table 8B: Safety parameters.

Parameter		Group II			
	Baseline Mean ± SD	1 month	3 months	6 months	p value
ALP	69.39 ± 19.42	66.94 ± 21.81	68.04 ± 20.18	69.67 ± 19.39	0.699
ALT	24.58 ± 10.94	28.38 ± 18.02	24.80 ± 10.38	24.17 ± 13.13	0.444
AST	23.78 ± 6.80	23.74 ± 5.76	23.96 ± 7.10	22.37 ± 9.21	0.63
Creatinine	0.88 ± 0.26	0.88 ± 0.26	0.89 ± 0.25	0.92 ± 0.24	0.375

ApoB/A ratio, the best reflector of the atherogenic lipids in the blood and the cut-off values for the apoB/apoA-I ratio that define a high cardiovascular risk were proposed to be 0.9 for men and 0.8 for women [20,21]. Elevated LDL Cholesterol and non -HDL cholesterol does not indicate the so called particle size which is more important for the genesis of atherosclerosis and can be better indicated by Apo B levels The MESA study showed that discordantly elevated Apo B is associated with coronary calcification [22]. The ratio of this remains unaltered in both groups at the end of the study and is in accepted level for cardiovascular risk.

Diseases like atherosclerosis results from the damage of cell membrane and nucleic acid by these ROS (Reactive oxygen species) molecules. There are many data to support this redox potential involvement in CAD [23,24].

The metabolic effect of the major median chain fatty acid like lauric acid in coconut oil on redox potential is not clear. Unsaturated fatty acids in oils like sunflower oil is likely to increase the oxidative stress compared to coconut oil [7]. Our study also shows that there is no change in oxidation related parameters on long term consumption of virgin coconut oil, even in group I with elevated total and LDL cholesterol probably indicating that the lauric acid rich coconut oil is not susceptible for oxidation. The anti-oxidants in both groups remain unchanged during the study period indicating that the VCO use during 6-month time does not alter the redox balance.

Ultra-sensitive CRP is a marker of inflammation in the body and is very much correlated with the atherogenic CAD. There were few reports stating that the VCO is an anti-inflammatory property. In our study there was no significant change in this very important biomarker at the end of the study in those with CAD but shows reduction in group II. The major concern about the oil was the safety on prolonged use and it has been shown that there was no hepatic or renal function alteration during 6-month period.

Limitation

Small study and confounding factors were not considered in analysis.

Conclusion

Considering the overall effects of VCO on lipoprotein profile it seems that it does not change the profile in atherogenic direction

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even though there is an increase in total cholesterol in subjects with CAD but not in others. Long term use of VCO in our subjects did not show change in redox potential, inflammatory markers, central obesity and glycosylated haemoglobin.

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