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Assessment of small, dense LDL particles among subjects consuming coconut oil or sunflower oil as cooking medium by using LDL-cholesterol/ LDL-apo B ratio as a surrogate marker

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ABSTRACT

Background: Coconut oil is the major cooking oil used by the people of Kerala. On account of its hypercholesterolemic effects, people are shifting to sunflower oil as an alternate cooking medium. The effect of dietary fats on small, dense low-density lipoprotein (LDL) particles, a newly recognized cardiovascular risk factor, has not been thoroughly investigated in this population.

Materials and Methods: We compared the effect of these two cooking oils on apolipoprotein B (apo B) and LDL-cholesterol/LDL-apo B ratio, which is a surrogate for small, dense LDL particles, among 62 control subjects and 64 subjects with type 2 diabetes. The subjects were divided into two subgroups based on the type of cooking medium used (coconut oil/sunflower oil). Total cholesterol, triglycerides, LDL-cholesterol, high density lipoprotein (HDL)-cholesterol and apo B were estimated, and the LDL-cholesterol/LDL-apo B ratio was calculated from these estimations.

Results: Triglycerides, LDL-cholesterol, and apo B concentrations were significantly higher among diabetic subjects compared to the control groups. A predominance of small, dense LDL particles indicated by low LDL-cholesterol/LDL-apo B ratio was observed among subjects of both control and diabetic groups. These parameters were not significantly different between subjects with the same clinical condition, but using different cooking media.

Conclusions: Small, dense LDL particles could be a major risk factor in this population, given its high prevalence in the study population. As the parameters studied did not differ significantly between the subgroups, it may be concluded that a change in the type of cooking medium has not considerably affected these parameters.

Key words: Apo B, coconut oil, small, dense LDL, sunflower oil, type 2 diabetes

INTRODUCTION

Coronary Heart Disease (CHD) is the prime cause of socioeconomic burden in India, characterized by premature occurrence, severity, and a high rate of incidence.[1,2] In spite of the tremendous improvement in the quality of health care and access to tertiary medical care to a vast majority of the population, cardiovascular disease remains the prime cause of mortality among the population of Kerala (South West India). It has been reported that the prevalence of risk factors for coronary heart disease (CHD) and its incidence is high in Kerala compared to the other regions of India.[3-6] Dietary fats affect lipid metabolism and have been considered as a major factor influencing CHD risk. Coconut is an important constituent of a typical Kerala diet and a majority of the population uses coconut oil as the cooking medium. Although saturated fats constitute 90% of coconut oil, most of these are medium chain fatty acids having 10 to 12 carbon atoms. Medium chain fatty acids are not dependent on carnitine

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transport[7] and they are metabolized rapidly by the body for energy requirements.[8] On account of the peculiar composition and characteristics of the fatty acids of coconut oil, its health effects and association with CHD still remains disputed. There is a perception among the general population of Kerala that the high saturated fat content of coconut oil may have contributed to the rise in the incidence of CHD in this region. As a result, a tendency to change the cooking medium to oils rich in polyunsaturated fat has been observed among people; sunflower oil being the most widely used. The major fatty acid of sunflower oil is linoleic acid and the fact that polyunsaturated fats reduce serum cholesterol has been well established.[9]

Lipid parameters such as apo B, small dense LDL, and the like, are known to affect CHD risk.[10-13] The occurrence of CHD in spite of normal LDL-cholesterol concentrations may be explained based on the particle size of LDL. Small, dense LDL particles are more atherogenic than normal LDL particles, as they are more susceptible to oxidation and have a decreased affinity for LDL receptor.[14] As dietary fats may affect cardiovascular risk through parameters other than elevated serum cholesterol and LDL-cholesterol, it is important to understand the effects induced by different types of fats on other lipoprotein parameters also. The effects of the replacement of saturated fat with polyunsaturated fat as cooking medium on these parameters have not been completely elucidated. This study was conducted to evaluate small, dense LDL particles using an equation among coconut oil and sunflower oil consumers of Kerala and to assess whether it is a major risk factor among this population. For this, we estimated the lipid profile, apo B, and LDL-cholesterol/LDL-apo B ratio as a surrogate for small, dense LDL particles among normal and type 2 diabetes subjects, using either coconut oil or sunflower oil as their cooking medium.

Materials and Methods

Subjects

This observational study was conducted at a tertiary care university hospital after obtaining approval from the Institutional Ethics Committee and was in accordance with the Helsinki Declaration of 1975, as revised in 2000. Informed consent of all the enrolled subjects was taken. Men aged 35 to 65 years, not on lipid-lowering therapy, attending the Outpatient Departments for routine health evaluation, consuming the respective oil as the predominant cooking medium for more than two years, and consenting to participate in the study were included. Healthy subjects without a prior history of diabetes, hypertension, renal or thyroid abnormalities, who were free from a history of CHD, as confirmed by a normal resting 12-lead electrocardiogram (ECG), and absence of inducible ischemia on stress test were recruited as controls. Subjects with type 2 diabetes based on the World Health Organization (WHO) diagnostic criteria for diabetes: Fasting blood glucose levels >6.99 mmol/L[15] without renal or thyroid abnormalities, were recruited in the diabetic groups, and they were on insulin therapy or oral hypoglycemic agents. On enrollment, the subjects were interviewed to obtain details regarding their diet and lifestyle. Details regarding their medical history and medications were procured from the medical records.

The subjects satisfying the above criteria formed four groups based on the type of cooking oil used and presence/absence of type 2 diabetes. Groups 1 and 2 consisted of 31 control subjects each, consuming coconut oil and sunflower oil, respectively. Groups 3 and 4 consisted of 32 subjects each with type 2 diabetes consuming coconut oil and sunflower oil, respectively [Table 1]. The average oil consumption per day for each subject was calculated by dividing the daily oil usage by the family with the number of family members, after adjusting for children.

Laboratory methods

Two milliliters of fasting blood sample was drawn from each subject; the serum was separated for biochemical analyses. Total cholesterol, triglycerides, HDL-cholesterol and LDL-cholesterol were measured, using kits from Roche, in Hitachi 912 auto analyzer. Apo B was analyzed by immunoturbidimetry, using kits from Daiichi Pure Chemical Co., Tokyo, Japan, using Systronics UV-Vis Double Beam Spectrophotometer 2201. Briefly, apo B in the serum reacts with antihuman apo B antibody, causing turbidity, which was measured against blank. Apo B concentrations of the serum were calculated using a single-point calibrator. Apo B concentrations above 0.9 g/L (gram per liter) were considered to be pathogenic.[16] LDL-cholesterol/

Table 1: Clinical profile of the subjects

Groups	Age (mean±SD)	Hypertensives (%)	Tobacco users (%)	Oil used (g/day) (mean±SD)
1, *n*=31 (coconut oil controls)	48.3±6		7 (22.5)	29±10
2, *n*=31 (sunflower oil controls)	49.1±9		4 (12.9)	25±7
3, *n*=32 (coconut oil diabetics)	54.5±11	14 (43.8)	4 (12.5)	24±5
4, *n*=32 (sunflower oil diabetics)	52.9±8	12 (37.5)	6 (18.7)	22±9

SD: Standard deviation

LDL-apo B ratio was calculated from concentrations of total cholesterol, triglycerides, HDL-cholesterol, and apo B, as described by Hattori, Suzuki, *et al.*[17] This formula provides information on the qualitative and quantitative alteration of LDL, without performing ultracentrifugation. The equations LDL-cholesterol = 0.94 total cholesterol - 0.94 HDL-cholesterol - 0.19 triglycerides; and LDL-apoB = apoB - 0.09 total cholesterol + 0.09 HDL-cholesterol - 0.08 triglycerides, is used for the calculation of LDL-C/LDL-apoB ratio. A lower ratio of LDL-cholesterol/LDL-apo B (<1.2) is postulated to indicate the preponderance of small, dense LDL particles.

Statistical analysis

Statistical analysis of the data was done using IBM SPSS software 19.0. The prevalence of hypertension and smoking among the study subjects, were summarized as percentages and absolute numbers. All other values (age, mean oil consumed/day, results of biochemical analysis) were expressed as mean and standard deviation. The analysis of variance (ANOVA) was used to compare the mean values of the parameters between different groups and a $P < 0.05$ was considered to be statistically significant. Post-hoc analysis was carried out by Tukey's test.

Results

Among Group 3 subjects, 43.8%, and among Group 4 subjects, 37.5%, were hypertensives and were on antihypertensive drugs. The percentage of subjects using tobacco were 22.5, 12.9, 12.5, and 18.7%, respectively, in the four groups [Table 1].

Total cholesterol and HDL-cholesterol did not show significant difference between any of the groups. The triglycerides and LDL-cholesterol levels were significantly higher for the type 2 diabetic groups compared to both the control groups [Table 2]. Hypertriglyceridemia (>2.26mmol/L) was observed in 3.2% (n = 2) of the controls and 14% (*n* = 9) of the type 2 diabetes subjects, but no significant differences were observed between clinically comparable coconut oil consumers and sunflower oil consumers. Increased LDL-cholesterol level (>2.6 mmol/L) was observed among 27% (n = 17) of the controls and 68.7% (*n* = 44) of the diabetic subjects.

Among the control subjects, 35.5% (*n* = 22) had apo B levels >0.9 g/L, while it was 56.3% (*n* = 36) among the type 2 diabetes subjects. The percentage of subjects with apo B levels >0.9 g/L did not differ significantly between groups with similar clinical conditions. The apo B concentrations were higher for type 2 diabetes subjects compared to the control groups, but the value was statistically significant only for Group 4 [Table 2].

Table 2: Results of biochemical estimations and LDL-cholesterol/LDL-apo B ratio

Parameters	Group 1 (mean±SD)	Group 2 (mean±SD)	Group 3 (mean±SD)	Group 4 (mean±SD)
Total cholesterol (mmol/L)	4.43±0.67	4.26±0.65	4.58±0.94	4.68±0.91
Triglycerides (mmol/L)	1.47±0.45	1.36±0.41	1.86±0.50*	1.80±0.38*
HDL-cholesterol (mmol/L)	1.22±0.24	1.12±0.23	1.15±0.26	1.05±0.23
LDL-cholesterol (mmol/L)	2.31±0.54	2.27±0.60	3.01±0.85*	2.98±0.74*
Apo B(g/L)	0.85±0.18	0.85±0.18	0.94±0.19	0.97±0.17*
LDL-cholesterol/ LDL-apo B ratio	1.28±0.47	1.25±0.38	1.10±0.39	1.18±0.54

'*' represents *P*<0.05 compared to control groups, SD: Standard deviation, LDL: Low-density lipoprotein

LDL-cholesterol/LDL-apo B ratio below 1.2, which is a surrogate for small, dense LDL particles, was seen in 43.5% (*n* = 27) of the control subjects and 57.8% (*n* = 37) of the type 2 diabetes subjects, but the ratio did not show significant difference on comparing the control and diabetic groups [Table 2]. The mean value of LDL-cholesterol/ LDL-apo B ratio for both diabetic groups was below the stipulated value. The percentage of subjects with normal LDL-cholesterol concentrations (below 2.6 mmol/L), but an LDL-cholesterol/LDL-apo B ratio below 1.2 was 38.7% (*n* = 24) for Group 1, 22.6% (*n* = 14) for Group 2, 31.3% (*n* = 21) for Group 3, and 21.9% (*n* = 14) for Group 4 subjects. Also, the parameters studied did not show any considerable difference between groups with similar clinical conditions, that is, between Groups 1 and 2 and between Groups 3 and 4.

Discussion

Although both the quantity and quality of fat influence the risk of cardiovascular disease, saturated fats in general are considered to be hypercholesterolemic compared to polyunsaturated fats.[18] It has been proposed that saturated fatty acids and cholesterol in the diet act synergistically to raise the serum cholesterol level.[19] There are various reports that have shown that coconut oil raises total cholesterol and LDL-cholesterol, when compared to other fats.[20] However, studies conducted in some populations habitually consuming a diet rich in coconut and coconut oil have failed to establish that coconut oil increases the risk for cardiovascular events.[21] It is noteworthy in our study that the serum total cholesterol and LDL-cholesterol concentrations of coconut oil users did not show a significant elevation compared to the respective groups using sunflower oil.

Elevated triglyceride concentration can exert its atherogenic potential by promoting the formation of small, dense LDL particles.[14] High triglyceride concentration may be particularly atherogenic if it occurs in association with other risk factors such as low HDL-cholesterol and the presence of small, dense LDL particles. It has been reported earlier that coconut oil, rich in medium chain fatty acids, raises the triglyceride level,[22] although only a marginal rise in triglyceride concentration among coconut oil users was observed in our study. Earlier investigators have observed that polyunsaturated fats decrease HDL-cholesterol level,[23] but, a substantial reduction in HDL-cholesterol concentration occurs only when polyunsaturated vegetable oils contribute significantly to the dietary energy.[24] HDL-cholesterol concentrations were only non-significantly reduced for groups using sunflower oil in this study. Most of the earlier studies mentioned here have been done in controlled settings in the Western population, where the intake of fat is much higher (≈35% of total calories) compared to our population (≈10%). As the lipid parameters are also influenced by the quantity of fat consumed, the non-significant results observed between the coconut oil and sunflower oil groups in the present study may also be due to the low fat intake.

Linoleic acid (*n-6* fatty acid), which constitutes about 65% of sunflower oil, has proven ability to lower cholesterol, especially LDL-cholesterol.[25] Earlier researchers have proposed that the lipid-lowering action of linoleic acid may be because of its capability to decrease the cholesterol content of LDL particles.[26] But more recent studies have demonstrated that polyunsaturated fats also reduce the number of LDL particles in circulation, as they have noticed a decline in LDL-apo B concentrations, which indicates a reduction in the number of LDL particles.[25,27] In contrast, there are also studies that have shown that a diet rich in polyunsaturated fats does not reduce apo B concentrations significantly when compared to saturated fat in men, but it is significantly reduced in women subjects.[28] Investigators have reported that a significant reduction in the apo B level can be attained in both men and women by replacing saturated fat in the diet with monounsaturated fat.[29] The sunflower oil consumers of our study have not shown a considerable reduction in the apo B concentration compared to coconut oil consumers. Irrespective of the type of oil taken, diabetic subjects have shown a higher apo B level compared to the controls, as has been observed by the earlier investigators.[30,31]

It is important to note that a considerable proportion of healthy as well as type 2 diabetes subjects of this study has lower LDL-cholesterol/LDL-apo B ratios, which denote a higher prevalence of small, dense LDL particles in this population. The percentage of subjects with normal LDL-cholesterol levels, but lower LDL-cholesterol/LDL-apo B ratio is also substantial (28.6%) in this study population. Increased concentrations of small, dense LDL particles have been reported in type 2 diabetes subjects earlier.[32] The percentage of diabetic subjects with low LDL- cholesterol/LDL-apo B ratio is high among our study population also. Occurrence of small, dense LDL particles among type 2 diabetes subjects may aggravate the threat for future cardiovascular events in these subjects. Analysis of the results from Quebec Cardiovascular Study suggests that the presence of hyper apo B and small, dense LDL particles are associated with a marked increase in CHD risk.[33] These conditions occur simultaneously in 24.2% of the controls and 34.4% of type 2 diabetes subjects in this study.

When the subject groups were compared based on the type of oil intake, it was observed that the proportion of healthy coconut oil consumers with a low LDL-cholesterol/LDL-apo B ratio was high, compared to the sunflower oil users (48.4 vs. 38.7%) and a similar result was observed between the type 2 diabetes groups (65.6 vs. 50%). This could be attributed partially to the statistically non-significant, yet higher triglyceride concentration observed among coconut oil consumers compared to the respective sunflower oil groups. There were studies that proved otherwise as well; wherein, the dietary fat changed from saturated to polyunsaturated, both n-3 and n-6, reduced the LDL particle diameter.[34] The number of coconut oil consumers with normal LDL-cholesterol levels, but lower LDL-cholesterol/LDL-apo B ratio was higher compared to the respective sunflower oil groups in this study. More aggressive therapeutic management of small, dense LDL particles using fibrates/statins or a combination of these drugs, known to shift the LDL size toward more buoyant particles, could be recommended in high-risk subjects, that is, patients with atherogenic lipid profile or a lipid profile within the normal range, but with small, dense LDL particles.

This study has certain major limitations. The methodology adopted as an indicator of small, dense LDL particles has not been validated, and a cut off value has not been established in our population. Also there was intra- and intergroup variation in the quantity of fat consumed, and the subjects may have been consuming other types of fat also (while eating out). These factors was not considered and this could have influenced the outcome. As this observational study was conducted in a small number of subjects, the conclusions arrived at from this study have to be confirmed with prospective studies, with a larger sample size, in controlled settings.

Despite the limitations, this study provides an insight into the high prevalence of small, dense particles even among healthy individuals of this population. This may warrant the need for screening people of this ethnic group for this parameter routinely, with classic lipid parameters, for risk assessment.

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