To study the levels of lipid profile in atherosclerosis and non-atherosclerosis individuals

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Abstract:

Background: The increased risk of cardiovascular disease is attributed to the cluster of metabolic abnormalities known as atherosclerosis. Most people with atherosclerosis also have several other metabolic problems, including high blood sugar, insulin resistance, abnormal lipid profiles, and high blood pressure. Aim: This study's objective was to investigate the relationships between lipids in persons with atherosclerosis. Materials & methods: All the study participants consented. Control volunteers were 100 age- and gender-matched healthy volunteers. Second cohort: 100 atherosclerosis patients treated. Same-age and gender normal-glycemic controls were used as non-atherosclerosis group. Results: The analysis found significant differences in the values of serum weight on comparison among 2 groups of the analysis. Although the TAGs mean level of the Atherosclerosis group patients was on the higher side, when we compared the two groups, we found that the Atherosclerosis group subjects had 40% higher than the control group subjects. On the contrary we observed significant lower of HDL concentration in atherosclerosis group patients in comparison with control group subjects. Conclusion: Research along these lines is encouraged since it will lead to molecular insights that will help doctors control dyslipidemia and slow the development of related diseases.

Keywords: Atherosclerosis; Homeostasis Model Assessment of Insulin Resistance (HOMA-IR); Hyperglycemia; Insulin; Insulin resistance.

Introduction:

Atherosclerosis is a chronic inflammatory disease of the arterial wall that underlies the majority of cardiovascular disorders, including coronary artery disease, stroke, and peripheral arterial disease. It is characterized by the deposition of lipids, fibrous elements, and immune cells within the intima, leading to plaque formation, luminal narrowing, and eventual impairment of blood flow [1]. The global burden of atherosclerosis is increasing, particularly in developing countries like India, where changing lifestyles, dietary patterns, and sedentary behavior have contributed to a rise in cardiovascular risk factors [2,3].

Lipid abnormalities are central to the pathogenesis of atherosclerosis. Elevated serum total cholesterol, triglycerides, along with reduced high-density lipoprotein cholesterol (HDL-C), have been strongly associated with the initiation and progression of atherosclerotic lesions ^[4,5]. LDL-C, in particular, is prone to oxidative modification, triggering endothelial dysfunction and recruitment of inflammatory cells into the vessel wall ^[6,7]. Conversely, HDL-C plays a protective role by mediating reverse cholesterol transport and exerting anti-inflammatory and antioxidant effects ^[8,9].

Comparing lipid profiles between individuals with and without atherosclerosis can provide important insights into the metabolic imbalances contributing to disease development. Such

studies are valuable not only for identifying at-risk populations but also for evaluating the effectiveness of preventive and therapeutic interventions [10-12]. Understanding these differences may aid in early diagnosis, risk stratification, and formulation of targeted lifestyle or pharmacological strategies to mitigate cardiovascular risk. By analyzing the lipid profiles in these two groups, this study seeks to contribute to the growing body of evidence on the relationship between dyslipidemia and atherosclerosis, particularly within the Indian population context. Henceforth, this study's objective was to investigate the relationships between lipids in persons with atherosclerosis.

Materials & methods:

In the current investigation, 200 individuals participated, 100 in the atherosclerosis group and 100 in the healthy control group. This investigation focused on patients at the Index Medical College & Research Centre in Indore. After receiving approval from the appropriate authorities, the study's researchers commenced their efforts. Each participant gave informed consent prior to the commencement of this study. Patients with type 1 diabetes or a duration of pathological symptoms and T2DM duration of less than five years were excluded. Healthy controls lacked diabetes, did not take multivitamins, and did not suffer from comorbid conditions.

All participants in both categories were examined by a qualified physician from the hospital's medical department, who adhered to standard operating procedures and considered the exclusion and inclusion criteria of the study. The control group consisted of 100 participants of the same age and gender who lacked atherosclerosis. 100 patients receiving treatment for atherosclerosis comprised the second cohort. The atherosclerosis was identified using ATP-III-established criteria. Human volunteers of a comparable age and gender with a normal glycemic state served as the control group. Each individual was examined by a licensed physician who adheres to established medical procedures. The atherosclerosis was identified using ATP-III-established criteria. Body mass index (BMI) was determined by dividing each subject's weight in kilograms by their height in meters squared. The subjects were divided into groups after the body mass index was determined. Using the World Health Organization's diagnostic criteria for obesity in body mass index (BMI) for Asian populations, subjects were again divided into three categories within each cohort: normal weight (18.5-24.9 kg/m2), overweight (25-29.9 kg/m2), and obese (30 kg/m2). Using a disposable syringe and cannula in a sterile environment, 5ml of each individual's fasting venous blood was extracted into flat containers in both groups. After being separated from blood by centrifugation at 3000 rpm for 20 minutes, serum samples were aliquoted and stored at 20 ° C.

The measure of serum TC, CHOD/POD procedure was used. Glycerol Phosphate Oxidase and Peroxidase (Liquid stable) assessed serum TAGs. All reagents were purchased from Avantor Performance Materials India Limited, Dehradun, Uttarakhand, India, and the estimation followed the kit manual. Supplier instructions were followed.

Statistical analysis:

The means of the variables were compared between the two groups using an unpaired "t" test. Not only that, but percentages were tallied. Scatter diagrams were used to analyze the correlation between two factors. Not only that, but percentages were tallied. A significance level of 0.05 was accepted.

Results:

In Table 1, we have shown the mean values of TC and triacylglycerols (TAGs) values in healthy control subjects and also in Atherosclerosis subjects. The analysis found significant differences in the values of serum weight on comparison among 2 groups of the analysis. Although the TAGs mean level of the Atherosclerosis group patients was on the higher side, when we compared the two groups, we found that the Atherosclerosis group subjects had 40% higher than the control group subjects. On the contrary we observed significant lower of HDL concentration in atherosclerosis group patients in comparison with control group subjects.

Table 1: Lipid profile levels in the study population

Variable	Atherosclerosis group	Control group	P Value
	(n=100)	(n=100)	1 value
Total Cholesterol	170.9 ± 28.23	153.7 ± 14.7	t= 5.4040; df= 198; P =
(mg/dL)			0.0001
Triacylglycerols	148.1 ± 48.6	126.7 ± 19.3	t= 4.092; df= 198; P =
(mg/dL)			0.0001
High Density	32.9 ± 13.11	45.2 ± 12.8	t= 6.7131; df= 198; P =
Lipoprotein (HDL;			0.0001
mg/dL)			

Discussion:

The significant elevation of TC observed in our findings is consistent with the well-established role of hypercholesterolemia in atherosclerotic plaque formation. Elevated TC, largely driven by increased low-density lipoprotein cholesterol (LDL-C), facilitates lipid deposition within the arterial intima, initiating the cascade of inflammatory and fibrotic changes characteristic of atherosclerosis. Similar patterns have been reported in both Indian and international studies. For instance, a study [10] observed significantly higher TC levels in Indian patients with angiographically confirmed coronary artery disease compared to matched controls, highlighting the role of dyslipidemia as a major modifiable cardiovascular risk factor [11,12]. Internationally, the Framingham Heart Study demonstrated that individuals in the highest quartile of TC had substantially higher incidences of coronary events over decades of follow-up [13,14]. These findings collectively reinforce the pathogenic role of elevated TC across diverse populations.

Our study also demonstrated a marked 40% increase in TAG levels in the atherosclerosis group compared to controls. Elevated TAGs, although traditionally considered a secondary risk factor, are now recognized as an independent contributor to atherogenesis. Hypertriglyceridemia promotes the formation of small dense LDL particles, which are more atherogenic due to their enhanced arterial wall penetration and susceptibility to oxidation. Furthermore, elevated TAGs are often accompanied by reduced HDL-C levels and increased apolipoprotein B-containing lipoproteins, creating a highly atherogenic lipid milieu. An Indian cross-sectional study by [15] reported similar elevations in TAGs among patients with ischemic heart disease, suggesting that postprandial lipemia and triglyceride-rich remnant lipoproteins may play a significant role in the pathophysiology of cardiovascular disease in South Asians, who tend to have higher TAG responses to dietary fat than Western counterparts.

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The most notable inverse finding in our study was the significant reduction in HDL-C levels among atherosclerosis patients compared to controls. HDL-C is widely recognized for its protective role in cardiovascular health, primarily due to its function in reverse cholesterol transport, whereby excess cholesterol is removed from peripheral tissues and delivered to the liver for excretion. Beyond lipid transport, HDL-C exerts anti-inflammatory, antioxidant, and endothelial-protective effects, all of which are relevant to the prevention of atherosclerotic progression. Low HDL-C levels have been repeatedly associated with increased cardiovascular risk, even when LDL-C levels are controlled. In the studies [16-19] reported that individuals with HDL-C levels below 35 mg/dL had a threefold increased risk of myocardial infarction compared to those with higher levels, independent of TC and LDL-C.

In the Indian context, low HDL-C is particularly prevalent and often observed in combination with elevated TAGs—a pattern referred to as "atherogenic dyslipidemia." A study ^[20] emphasized that urban Asian Indians often present with low HDL-C levels despite having only modest elevations in LDL-C, suggesting a distinct lipid phenotype that contributes to their disproportionately high cardiovascular disease burden. The present findings align with this pattern, as the combination of elevated TAGs and low HDL-C in our atherosclerosis group reflects the typical dyslipidemic profile seen in many South Asian populations.

From a mechanistic standpoint, the combination of high TC, elevated TAGs, and reduced HDL-C creates a synergistic environment for atherogenesis. Elevated TC and TAGs contribute directly to lipid accumulation within arterial walls, while low HDL-C reduces the system's ability to clear cholesterol from these sites. Moreover, the coexistence of these lipid abnormalities often indicates the presence of insulin resistance and atherosclerosis, both of which further exacerbate endothelial dysfunction and inflammatory activation.

Clinically, our findings underscore the importance of comprehensive lipid profiling rather than focusing solely on LDL-C or TC levels. While LDL-C remains the primary therapeutic target, the predictive value of TAGs and HDL-C for cardiovascular events should not be underestimated [20-24]. Lifestyle interventions such as dietary modification, increased physical activity, and weight management can beneficially modulate TAGs and HDL-C, while pharmacological agents like fibrates and niacin have demonstrated efficacy in addressing these lipid fractions when lifestyle measures are insufficient.

The significant differences in TC, TAGs, and HDL-C observed between atherosclerosis patients and healthy controls in our study highlight the multifaceted nature of dyslipidemia in cardiovascular disease pathogenesis. The findings are consistent with both Indian and global literature and point towards the need for early identification and aggressive management of all lipid abnormalities, especially in populations with a high baseline risk of atherosclerosis such as South Asians.

Conclusion:

Research along these lines is encouraged since it will lead to molecular insights that will help doctors control dyslipidemia and slow the development of related diseases. The results of these investigations will add to our knowledge of molecular insights that may one day help doctors

control dyslipidemia. This research also suggests that people with atherosclerosis may not have adequate compensatory mechanisms to deal with pathophysiological abnormalities. Despite the lack of prior extensive investigation into the issue at hand, the current study was able to establish this finding.

Conflict of interest:

There is no conflict of interest among the present study authors.

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