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Status of Serum Lipid Profile Parameters in Atherosclerosis: A Comparative Study with Healthy Controls

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ABSTRACT

Background: Atherosclerosis is a chronic inflammatory disease caused by lipid accumulation in arteries, leading to coronary artery disease, stroke, and peripheral vascular disease. Dyslipidemia is a major modifiable risk factor. This study compares lipid profiles of patients with atherosclerosis and healthy controls.

Methods: A prospective study was conducted at Index Medical College, Indore (Feb 2012–May 2013). It included 53 atherosclerosis patients and 50 age- and sex-matched healthy controls, divided into the 30–50 and 51–70-year groups. Ultrasound/CT confirmed diagnosis. Fasting blood samples were analyzed for TC, TG, HDL-C, LDL-C, and VLDL-C using standard methods. Statistical analysis used an unpaired t-test ($p<0.05$ significant).

Results: Atherosclerosis patients had a higher proportion of smokers, especially males aged 51–70 years. They showed significantly increased TC, LDL-C, TG, and BMI, and decreased HDL-C compared to controls ($p<0.0001$). In males, HDL-C, TG, and BMI varied with age, while TC and LDL-C did not. No significant age-related lipid variation was seen in females.

Conclusion: Atherosclerosis is associated with abnormal lipid profiles (↑TC, LDL-C, TG, VLDL-C and ↓HDL-C) in both sexes. Age-related lipid variation is mainly seen in males. Regular lipid profile testing is important for early detection and management.

Key-words: Atherosclerosis, Serum lipid profile, Total cholesterol, LDL-cholesterol, HDL-cholesterol, Triglycerides, Dyslipidemia, Healthy controls

INTRODUCTION

Cardiovascular diseases (CVDs) are the leading cause of death worldwide, accounting for an estimated 19.8 million deaths in 2022 or approximately 32% of all global deaths. More than 3/4 of CVD deaths globally took place in low- and middle-income countries [1]. Among the various pathological entities that make up CVD, atherosclerosis is at the core of the main underlying mechanisms of coronary artery disease, ischemic stroke, and peripheral artery disease.

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Atherosclerosis is a chronic and progressive disease of the arteries. It is characterized by the deposit of fat inside the arteries. Damage to diseased arteries restricts blood flow. This sclerotic and fatty tissue occupies space within the arterial intima. As a result, arterial diameter decreases. An increase in thrombus formation occurs due to a weakened wall [2]. Also, a thrombus is the formation of a blood clot within a blood vessel. A global increase in disease burden caused by atherosclerosis results in significant deaths. Thus, identification of its modifiable risk factors is an urgent necessity. Under identification of modifiable risk factors, dyslipidemia is the major risk factor that must be tackled as a priority.

The development of the lesion involves several mechanisms, but disorders of lipid metabolism remain at the core of initiation and progression.[3] At the cellular level, the process starts with endothelial dysfunction, allowing the entry of atherogenic lipoproteins into the

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subendothelial space, especially low-density lipoprotein (LDL). Once retained in the arterial intima, LDL is oxidatively modified to produce oxidized LDL (ox-LDL), which initiates a series of pro-inflammatory phenomena, including monocyte recruitment, macrophage activation, and the formation of lipid-loaded foam cells, the characteristic histological feature of early atherosclerotic lesions. Considerable experimental and clinical data supporting that "LDL modification hypothesis" of atherogenesis, confirming ox-LDL as a major culprit in endothelial damage and plaque formation. The most widely measured serum lipid profile parameters in clinical practice for atherosclerotic risk prediction include total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), and high-density lipoprotein cholesterol (HDL-C) [4]. TC, LDL-C, and TG elevations are independent pro-atherogenic factors that cause lipid deposition, endothelial activation, and plaque progression. Of note, VLDL-C, which carries triglyceride-rich particles, is also implicated in atherogenesis. In contrast, HDL-C has clear beneficial effects on the heart. This effect is mainly due to reverse cholesterol transport (RCT). Herein, excess peripheral cholesterol is mobilized for hepatic clearance. Thus, there is a reduction in atheromatosis.[5] Low HDL-C is one of the most powerful independent coronary risk factors. In addition, HDL has several other beneficial effects. It has anti-inflammatory, anti-oxidative, and vasodilatory properties. All these effects together prevent the formation of atheroma [6]. Various lipid ratios, such as TC/HDL-C (i.e., Castelli Risk Index I) and LDL-C/HDL-C (i.e. Castelli Risk Index II), are more sensitive indicators of cardiovascular risk than any individual lipid fraction, as they reflect the changing relationship between atherogenic and atheroprotective lipoprotein fractions. The identification of non-traditional additional biomarkers, such as lipoprotein (a), apolipoproteins, and the atherogenic index of plasma (AIP), is enabling a redefined, more accurate risk stratification beyond the conventional lipid profile.

Multiple previous comparative studies have demonstrated significantly elevated levels of TC, LDL-C, TG, and VLDL-C, and significantly reduced levels of HDL-C, in patients with atherosclerosis compared to healthy individuals. Hence, dyslipidemia is an important biochemical feature of atherosclerosis disease.[7] Although this has been established, the exact characterization of the degree of lipid derangement in each parameter in various clinical populations will help in optimizing preventive and therapeutic targets.[8] Thus, the present study was planned to assess and compare serum lipid profile parameters between patients with established atherosclerosis and age- and sex-matched healthy controls.

MATERIALS AND METHODS

Study Design- The present study was conducted as a prospective, consecutive observational study. Patients clinically diagnosed with atherosclerosis, attending the outpatient department or admitted to the Department of Medicine ward of Index Medical College Hospital and Research Centre, Indore (M.P.), were enrolled from the date of Dean's permission. The study period extended from February 2012 to May 2013.

Study Population- The study comprised 53 patients with atherosclerosis (cases) and 50 age- and sex-matched healthy individuals serving as controls. The control group consisted of apparently healthy males and females with no known systemic illness, recruited from the same institution.

Inclusion Criteria- Patients clinically and/or investigatively confirmed to have atherosclerosis were included in the study.

Exclusion Criteria- Patients with concomitant conditions, including diabetes mellitus, myocardial infarction, cerebrovascular stroke, and other major systemic illnesses, were excluded from the study to minimize confounding variables.

Blood Sample Collection- Venous fasting blood samples (3 ml) were collected from each subject — both cases and controls — under strict aseptic precautions using disposable syringes. Blood samples were collected into plain vials and used for serum separation and subsequent lipid profile estimation.

Serum Separation- Blood samples collected in plain vials were incubated at 37°C for 45 minutes (during winter) or 20 minutes (during warmer months) to allow clot formation. Following incubation, samples were transferred to centrifuge tubes and centrifuged at 3000 rpm for 10–20 minutes. The clear supernatant (serum)

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was carefully aspirated into clean, dry serum test tubes and used for lipid profile analysis.

Sample Preservation- Serum samples were stored and analyzed as per the following protocol:

- Total Cholesterol: 2–8°C, 7 days
- HDL-Cholesterol: 2–8°C, 7 days
- LDL-Cholesterol: Calculated (Friedewald equation)
- Triglycerides: 2–8°C, 7 days

Lipid Profile Estimation

Total Cholesterol — Enzymatic Method- Total serum cholesterol was estimated by the enzymatic colorimetric method. Cholesterol esterase (CHE) hydrolyzes cholesterol esters to free cholesterol, which is oxidized by cholesterol oxidase (CHO) to cholest-4-en-3-one and hydrogen peroxide (H_2O_2). The H_2O_2 reacts with 4-aminoantipyrine and phenol in the presence of peroxidase (POD) to produce a pink-colored quinoneimine dye proportional to cholesterol concentration.

Reactions:

Cholesterol ester + H_2O → Cholesterol + Fatty acid

Cholesterol + O_2 → Cholest-4-en-3-one + H_2O_2

H_2O_2 + 4-aminoantipyrine + Phenol → Quinoneimine dye + H_2O

Procedure: 10 µl of distilled water (Blank), standard, or serum (Test) was added to 1.0 ml enzyme reagent. Incubated at 37°C for 5 minutes (or 15 minutes at room temperature). Absorbance measured at 505 nm.

Calculation: Cholesterol (mg/dl) = (A Test / A Standard) × 200

Normal range: 130–250 mg/dl

HDL-Cholesterol — Phosphotungstate Method: HDL was estimated after precipitation of LDL, VLDL, and chylomicrons using phosphotungstate/Mg^{2+}. The supernatant was analyzed enzymatically.

Procedure: Serum mixed with reagent, centrifuged, and the supernatant used for analysis. Absorbance measured at 505 nm.

Calculation: HDL-C (mg/dl) = (A Test / A Standard) ×50 ×2

Reference values:

Males: 30–75 mg/dl; Females: 35–85 mg/dl

LDL-Cholesterol — Friedewald Equation: LDL-C (mg/dl) = Total Cholesterol − HDL-C − (Triglycerides / 5)

Normal range: 80–150 mg/dl

Serum Triglycerides — GPO-PAP Method: Triglycerides were estimated using enzymatic end-point method involving LPL, GK, GPO, and POD reactions, forming a colored complex measured at 505 nm.

Procedure: 10 µl serum added to the reagent, incubated at 37°C for 10 minutes. Absorbance measured at 505 nm.

Calculation: Triglycerides (mg/dl) = (A Test / A Standard) × 200

Normal range: <200 mg/dl

Statistical Analysis- Data were expressed as mean±standard deviation (SD). Comparison between atherosclerosis patients and healthy controls was performed using the Student's unpaired *t*-test. A $p<0.05$ was considered statistically significant.

Ethical Considerations- The study was initiated following approval from the institutional authority (Dean's permission). Informed consent was obtained from all participants before enrollment.

RESULTS

Fifty normal healthy controls and fifty-three atherosclerosis patients were assembled in two age groups. Males constitute 54% of the normal health control group (NHC) and 56.6% from atherosclerosis group. Smokers among the atherosclerosis patients are at a much higher level in males (35.84%) as well as females (9.43%). In the estimation of NHC males (12%) and females (2%). The maximum smoker rate in the atherosclerosis sub-group was 37.93% among male smokers (Table 1).

Table 1: Subject Characteristics and Smoking Status

Age Group	Group	n	Male (%)	Female (%)	Male Smokers (%)	Female Smokers (%)
30–50 yr	NHC	26	14 (53.85)	12 (46.15)	07.69	0
30–50 yr	Atherosclerosis	24	13 (54.16)	11 (45.83)	33.33	08.33
51–70 yr	NHC	24	13 (54.16)	11 (45.83)	16.66	04.16

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51–70 yr	Atherosclerosis	29	17 (58.62)	12 (41.37)	37.93	10.34
Total	NHC	50	27 (54.00)	23 (46.00)	12.00	02.00
Total	Atherosclerosis	53	30 (56.60)	23 (43.39)	35.84	09.43

NHC = Normal Healthy Controls

In atherosclerotic males compared with their NHC counterparts of the same age groups, total cholesterol, LDL cholesterol, and triglycerides were extremely significantly elevated, while HDL cholesterol was significantly decreased. In the 30–50-year age group (cholesterol: 293.62±18.12 mg/dl; HDL: 23.77±1.24 mg/dl) and the 51–70-year age group (cholesterol: 300.59±16.68 mg/dl; HDL: 25.94±1.09 mg/dl), all parameters were extremely significantly different ($p<0.0001$) (Table 2).

Table 2: Lipid Profile Comparison in Males — NHC vs. Atherosclerosis Patients (Mean±SD)

Biochemical Parameter	NHC Male 30–50 yr (n=14)	Athero. Male 30–50 yr (n=13)	p-value	NHC Male 51–70 yr (n=13)	Athero. Male 51–70 yr (n=17)	p-value
Cholesterol (mg/dl)	181.36±21.38	293.62±18.12	<0.0001**	181.54±20.35	300.59±16.68	<0.0001**
HDL-Cholesterol (mg/dl)	45.71±5.64	23.77±1.24	<0.0001**	52.00±4.98	25.94±1.09	<0.0001**
LDL-Cholesterol (mg/dl)	93.71±25.35	164.23±6.03	<0.0001**	100.00±19.90	166.47±5.56	<0.0001**
Triglyceride (mg/dl)	91.57±19.44	167.62±5.30	<0.0001**	92.92±18.13	170.63±3.81	<0.0001**
BMI (kg/m²)	25.15±4.67	38.62±1.56	<0.0001**	25.54±4.35	40.13±1.26	<0.0001**

***p<0.0001 = Extremely significant (unpaired t-test). NHC = Normal Healthy Controls; Athero. = Atherosclerosis.*

A similar pattern was seen among females, where the atherosclerotic patients of both age groups had significantly higher levels of total cholesterol (293.40±18.21 mg/dl and 298.00±14.83 mg/dl for 30–50 years and 51–70 years respectively), higher levels of LDL-cholesterol, higher levels of triglycerides, and higher values of BMI (>39 kg/m²), along with significantly lower levels of HDL-cholesterol (25.00±1.18 and 25.82±1.33 mg/dl) than that of female NHC groups ($p<0.0001$ for all parameters) (Table 3).

Table 3: Lipid Profile Comparison in Females — NHC vs. Atherosclerosis Patients (Mean±SD)

Biochemical Parameter	NHC Female 30–50 yr (n=12)	Athero. Female 30–50 yr (n=11)	p-value	NHC Female 51–70 yr (n=11)	Athero. Female 51–70 yr (n=12)	p-value
Cholesterol (mg/dl)	168.67±14.61	293.40±18.21	<0.0001**	181.18±19.81	298.00±14.83	<0.0001**
HDL-Cholesterol (mg/dl)	43.50±5.85	25.00±1.18	<0.0001**	46.91±6.38	25.82±1.33	<0.0001**
LDL-Cholesterol (mg/dl)	104.74±22.45	162.70±5.06	<0.0001**	95.55±23.14	164.55±5.63	<0.0001**
Triglyceride (mg/dl)	99.50±28.13	170.17±4.65	<0.0001**	87.36±20.54	171.18±3.95	<0.0001**
BMI (kg/m²)	27.08±4.89	39.20±1.40	<0.0001**	27.82±4.62	39.18±1.33	<0.0001**

***p<0.0001 = Extremely significant (unpaired t-test). NHC = Normal Healthy Controls; Athero. = Atherosclerosis.*

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Inter-group variation within the age groups of atherosclerotic patients was not significant for any biochemical or anthropometrical parameter in males, although HDL-cholesterol ($p<0.0001$), triglycerides ($p<0.05$), and BMI ($p<0.01$) were significantly different between 30–50 and 51–70-year age groups. At the same time, total cholesterol and LDL-cholesterol showed no significant differences across age groups. Results, on the other hand, showed that there was no significant inter-group difference in any biochemical parameter among atherosclerotic females; this suggests that age-related lipid variation in atherosclerosis is sex-specific (Table 4).

Table 4: Age-Stratified Comparison of Lipid Parameters Within Atherosclerosis Patients (30–50 yr vs. 51–70 yr)

Biochemical Parameter	Male 30–50 yr (n=13)	Male 51–70 yr (n=17)	p-value	Female 30–50 yr (n=11)	Female 51–70 yr (n=12)	p-value
Cholesterol (mg/dl)	293.62±18.12	298.63±15.06	0.42 (NS)	296.82±20.66	298.00±14.83	0.87(NS)
HDL-Cholesterol (mg/dl)	23.77±1.24	26.06±1.00	<0.0001**	25.00±1.18	25.82±1.33	0.14(NS)
LDL-Cholesterol (mg/dl)	164.23±6.03	165.94±5.27	0.42 (NS)	163.73±5.88	168.58±7.09	0.08(NS)
Triglyceride (mg/dl)	167.62±5.30	170.63±3.81	<0.05*	169.55±4.32	171.18±3.95	0.36(NS)
BMI (kg/m²)	38.62±1.56	40.13±1.26	<0.01†	39.45±1.57	39.18±1.33	0.66(NS)

**p<0.05 = Statistically significant; †p<0.01 = Moderately significant; **p<0.0001 = Extremely significant; NS = Not significant (unpaired t-test).*

DISCUSSION

The current investigation consisted of 53 patients with atherosclerosis and 50 normal healthy controls (NHC), matched for age and sex, and equally distributed across two age groups: 30–50 years and 51–70 years. 54% and 56.60% of participants in the NHC and Atherosclerosis groups were male, respectively. The smoking prevalence was significantly higher among atherosclerosis patients, including 35.84% in males and 9.43% in females, as compared to NHC males (12%) and females (2%). The group of patients with atherosclerosis aged 51 – 70 had the highest prevalence of smoking, whose 37.93% are males and 10.34% are females. According to Ugur *et al.* [9], male sex, younger age, and lower education level were significant factors that determined smoking behaviour in subjects who have cardiovascular disease. Also, the history of smoking had a significant association with BMI. Smoking promotes endothelial dysfunction, oxidative stress, and an atherogenic lipid profile, and is a well-established independent risk factor for atherosclerosis [10].

In both age groups (30-50 years and 51-70 years), a very high level of significance ($p<0.0001$) was found for elevated serum total cholesterol (TC), LDL-cholesterol (LDL-C), triglycerides (TG), and BMI in atherosclerotic males compared to NHC males of corresponding age groups. In addition, HDL- cholesterol (HDL-C) was found to be very significantly decreased ($p<0.0001$). In the 30-50 age group, Mean TC was significantly higher in atherosclerosis (293.62±18.12 mg/dl) than in NHC (181.36±21.38 mg/dl), and HDL-C was also significantly different (23.77±1.24 mg/dl in atherosclerosis and 45.71±5.64 mg/dl in NHC). Likewise, in the 51–70 years group, TC was 300.59±16.68 mg/dl in patients and 181.54±20.35 mg/dl in NHC, and HDL-C was 25.94±1.09 mg/dl and 52.00±4.98 mg/dl, respectively.

The findings are in close agreement with Yadav *et al.* [11], who reported that atherosclerotic patients have significantly higher serum levels of TC, LDL-C, and TG, while having significantly lower HDL-C than normal healthy controls in the age groups of 30-50 and 51-70 years. Likewise, Faridi *et al.* [12] had shown that the prevalence of coronary atherosclerosis is higher at higher levels of serum lipoproteins in both sexes. Research conducted at BMIIMS, Pawapuri, by Kumar *et al.* [13] documented mean TC of 220±30 mg/dl and LDL-C of 140±35 mg/dl in atherosclerosis patients, values that are

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roughly similar to those in the present study. Similar results were observed among patients with CAD, as reported by Gupta *et al.* [14]. Their TC was 258.92±46.67 mg/dl, whereas controls had 175.98±21.67 mg/dl. Further corroborating the finding in this study that HDL-C has cardioprotective properties, Kohsaka *et al.* [15] confirmed that HDL-C level and Apo B/A-I ratio were the strongest predictors of atherosclerotic plaque (p<0.001).

Across both age groups, atherosclerotic females showed a pattern that was similar to that of males. The mean total cholesterol level of patients in the 30-50 years age group was 293.40±18.21 mg/dl vs 168.67±14.61 mg/dl in NHC. In the 51-70 years, patients had 298.00±14.83 mg/dl vs 181.18±19.81 mg/dl (p<0.0001). As per the report, HDL-C was significantly lower in atherosclerotic females (25.00±1.18 and 25.82±1.33 mg/dl) in comparison to NHC females (43.50±5.85 and 46.91±6.38 mg/dl) in both age groups (p<0.0001). Atherosclerotic women in both age groups had significantly higher BMI (>39 kg/m²) than NHC (p<0.0001).

The observational study by Ponnamalla *et al.* [16] has similar findings. The mean TC, LDL-C, and HDL-C were 201.3±26.8 mg/dl, 132.5±21.6 mg/dl, and 50.3±12.1 mg/dl, respectively. High LDL-C and poor lipid ratios were strongly related to greater cardiovascular risk in patients with ASCVD. In addition, a recent study conducted by Shewa *et al.* [17] An independent-samples t-test confirmed that ASCVD patients had significantly higher TC and LDL-C and lower HDL-C than healthy controls, which is fully consistent with the present findings. The same authors Bozkurt *et al.* [10], have reported elevated female BMI in atherosclerosis and raised female LDL-C, showing that raised obesity may be another atherogenic risk modifier in females.

The two subgroups of atherosclerosis age groups (30-50 v 51-70 years) were compared for lipid parameters. It revealed a sex-specific pattern. Among males with atherosclerosis, HDL-C (p<0.0001), TG (p<0.05), and BMI (p<0.01) were significantly different between the two age groups; however, there was no significant age-related variation in TC and LDL-C levels. In atherosclerotic females, there was no significant difference in any parameter across age groups (p>0.05). The result suggests that the age-associated alteration of lipids in atherosclerosis differs between males and females: the former display a progressive worsening of certain lipid fractions with age, while the latter have deranged lipid fractions that are comparable to those of males across age groups.

According to Ugur *et al.* [9], age was a determinant of the cardiovascular risk factor profile in males (p=0.001), whereas age groups in females were uniformly dyslipidaemic. The absence of age-dependent variation in females is likely due to the expedited lipid derangement resulting from estrogen withdrawal post-menopause, which tends to equalize lipid risk profiles among all adult females. In addition, Baral *et al.* [18], while conducting a comparative lipid profile analysis, found no significant age-related differences in LDL-C and TC levels in atherosclerosis patients, which supports the current data.

CONCLUSIONS

The present study conclusively demonstrates that the serum lipid profile of atherosclerotic patients was significantly disturbed. Total cholesterol, LDL-cholesterol, triglycerides, and VLDL-Cholesterol were highly elevated, and HDL-cholesterol was low in both sexes and all age groups compared with normal healthy controls (p<0.0001). The lower HDL-C levels observed in patients with atherosclerosis reinforce the importance of this protective lipoprotein as an independent cardioprotective marker. After controlling for age, our data for females confirm earlier studies showing lower LDL-C levels with higher Lp(a) levels. Increased body mass index (BMI) in both male and female patients with atherosclerosis also indicates obesity as an additional and exacerbating atherogenic risk factor. The observations confirm that periodic testing of serum lipid profile parameters can be a simple, reliable and cost-effective means for early detection of dyslipidaemia, better cardiovascular risk stratification and maximisation of therapeutic intervention in atherosclerotic patients.

CONTRIBUTION OF AUTHORS

Research concept- Dr Kapil Shrivastava
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