



Comparison of Lipid Profile Between Obese and Non-Obese Adults Without Known Vascular Comorbidities in a Tertiary Care Setting in North Kerala

Dr. Subinth S^{1*}, Dr. Jog Antony², Dr. Reeta James³, Dr. Mohammed Shamnas P P⁴, Dr. N.K. Thulaseedharan⁵

¹ Postgraduate, Department of General Medicine, KMCT Medical College, Calicut, India

²⁻⁵ Department of General Medicine, KMCT Medical College, Calicut, India

*Corresponding author: Dr. Subinth S

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ABSTRACT:

Background: Obesity is a well-established risk factor for dyslipidemia and cardiovascular disease. The present study, conducted at a tertiary care hospital in North Kerala, examines lipid profile variations between obese and non-obese individuals without known vascular comorbidities. Given the high obesity prevalence and unique dietary habits in this region, this study aims to elucidate obesity-driven lipid abnormalities and their potential implications for cardiovascular risk.

Methodology: This cross-sectional study analyzed 462 participants, categorizing them into obese and non-obese groups based on body mass index (BMI) criteria. Fasting lipid profiles, including 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), were measured and compared between the groups. Statistical analyses assessed lipid profile differences, sex-specific variations, and associations with lifestyle factors such as diet, physical activity, and smoking status.

Results: This cross-sectional study analyzed 462 participants, categorizing them into obese and non-obese groups based on body mass index (BMI) criteria. Fasting lipid profiles, including 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), were measured and compared between the groups. Statistical analyses assessed lipid profile differences, sex-specific variations, and associations with lifestyle factors such as diet, physical activity, and smoking status.

Conclusion: This study confirms obesity's association with atherogenic dyslipidemia, aligning with global findings while highlighting region-specific dietary and lifestyle influences. The observed lipid abnormalities underscore the need for routine lipid screening in obese individuals, even in the absence of vascular comorbidities. Lifestyle modifications, including dietary interventions and increased physical activity, are crucial in mitigating obesity-related dyslipidemia. Future research should explore genetic predispositions, longitudinal lipid variations, and potential interventions tailored to the North Kerala population.

Introduction:

Obesity is a well-established metabolic disorder with significant implications for lipid metabolism and cardiovascular risk. Dyslipidemia is one of the most frequently observed metabolic abnormalities in obese individuals, characterized by alterations in serum lipid levels, including elevated triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and decreased high-density lipoprotein cholesterol (HDL-C). Conventional anthropometric indices, such as body mass index (BMI), waist circumference (WC), and waist-to-hip ratio (WHR), are commonly used to define obesity and assess its metabolic consequences.¹

With the increasing burden of obesity, newer adiposity indices such as waist-to-height ratio (WHtR), visceral

adiposity index (VAI), and body adiposity index (BAI) have been proposed to provide a more refined assessment of obesity-related metabolic disturbances. Recent studies suggest that WHtR, a relatively simple measure of central obesity, may be a useful predictor of metabolic complications, including dyslipidemia, and may have prognostic value in assessing cardiovascular risk in obese individuals.²

Although dyslipidemia is a recognized feature of obesity, the specific lipid abnormalities observed in obese individuals without known vascular comorbidities require further exploration. While dyslipidemia is frequently studied in obese individuals with established cardiovascular or metabolic disorders, limited research has been conducted in populations devoid of overt vascular comorbidities. Investigating



lipid abnormalities in this subgroup is critical for understanding the early metabolic derangements associated with obesity and may provide insights into primary prevention strategies aimed at reducing long-term cardiovascular risk.³

Epidemiological data on obesity-related dyslipidemia in different Indian populations remain scarce, particularly in the North Kerala region, where distinct dietary habits, genetic predispositions, and lifestyle factors may influence metabolic health. Understanding lipid profile variations between obese and non-obese individuals in this population may help bridge existing knowledge gaps and improve early risk stratification. Additionally, assessing lipid patterns in individuals without diabetes, hypertension, or clinically apparent vascular disease could provide valuable information for designing targeted preventive and therapeutic interventions.⁴ The present study aims to evaluate the relationship between obesity and serum lipid parameters, including TC, LDL-C, HDL-C, TGs, and compare the lipid profile of obese vs non obese individuals without known vascular comorbidities.

Methodology

This cross-sectional study was conducted over a period of 12 months from June 1st 2024- June 1st 2025, at the Department of General Medicine, KMCT Medical College, a tertiary care center in Kozhikode, Kerala. A total sample size of 528 participants was calculated based on the study of Rao et al⁵ using the formula for a two-sample t-test, considering an effect size of 0.9, a power of 0.80, a significance level of 0.05, and a standard deviation of 5 and a non-response rate of 10%. The inclusion criteria comprised patients aged between 15 to 95 years attending the General Medicine OPD. The exclusion criteria included patients with known vascular comorbidities such as diabetes, hypertension, heart disease, hypercholesterolemia, peripheral vascular disease, or malignancies; those with endocrine conditions that could lead to obesity or dyslipidemia, such as hypothyroidism; patients taking medications that could affect lipid profile or body weight, such as oral contraceptives; and pregnant women. Patients were enrolled through convenience sampling based on inclusion and exclusion criteria until the required sample size was achieved. Participants were then sequentially allocated into two groups: obese and non-obese, based on BMI classification.

After obtaining informed consent, anthropometric

measurements were taken. Weight was measured using standardized digital scales and height using a drop-down tape measure fixed on a wall. BMI was calculated as weight in kilograms divided by the square of height in meters and categorized as per standard cutoffs. Waist and hip circumferences were also measured according to WHO protocol. Fasting blood samples (5 ml) were collected after 8–12 hours of overnight fasting, centrifuged for 10 minutes at 1000 rpm to separate serum, and analyzed for lipid profile parameters. Total cholesterol was estimated using the CHOD-PAP method, HDL by immune inhibition method, and triglycerides by GPO method. LDL levels were calculated using the Friedewald formula: $LDL = TC - (HDL + TG/5)$.

All data, including demographic details and laboratory parameters were recorded in a structured proforma. Data entry was done using Microsoft Excel and statistical analysis was performed using SPSS version 26. Quantitative variables were summarized using mean and standard deviation, while categorical variables were presented as frequencies and proportions. Appropriate statistical tests such as Student's t-test, Chi-square test, and ANOVA were employed to compare variables between the two groups. A p-value less than 0.05 was considered statistically significant.

Results

Around 462 individuals participated in the study. The age distribution of 462 participants ranges from 15 to 95 years, with a mean age of 56.74 years and a standard deviation of 14.623. Among the participants, 59.5% were male and 40.5% were female. Regarding obesity status, 38.1% were obese while 61.9% were non-obese. In terms of physical activity, 50.2% reported being active and 49.8% had a sedentary lifestyle. Dietary patterns revealed that 38.7% followed a moderate diet, 35.1% had an unhealthy diet, and only 26.2% maintained a healthy dietary pattern. Alcohol consumption was reported by 28.8% of the participants, whereas 71.2% did not consume alcohol. Smoking was present in 21.2% of individuals, while the majority, 78.8%, were non-smokers. (Table 1)

Table 1: Socio-demographic and Lifestyle Characteristics of the Study Population (N=462)

Variable	Categories	Frequency (%)
Gender	Male	275 (59.5%)



	Female	187 (40.5%)
Obesity Status	Obese	176 (38.1%)
	Non-obese	286 (61.9%)
Personal History	Active	232 (50.2%)
	Sedentary	230 (49.8%)
Dietary Pattern	Healthy	121 (26.2%)
	Moderate	179 (38.7%)
	Unhealthy	162 (35.1%)
Alcohol Consumption	Yes	133 (28.8%)
	No	329 (71.2%)
Smoking Status	Yes	98 (21.2%)
	No	364 (78.8%)

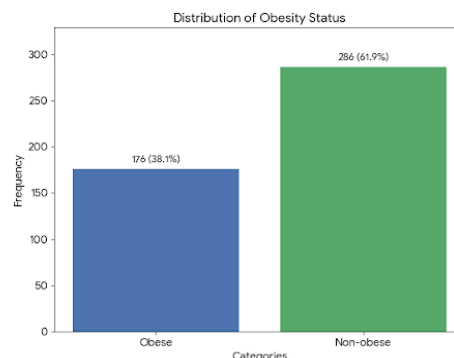


Figure 2: Distribution of obesity status of the study participants

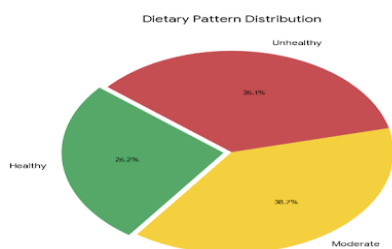


Figure 1: Dietary pattern distribution of the study subjects

The comparison of lipid profiles between obese and non-obese individuals revealed statistically significant differences across all parameters. Obese participants had higher mean levels of total cholesterol (222.78 ± 54.42 mg/dL) compared to non-obese individuals (175.89 ± 48.61 mg/dL; $p=0.001$). Triglyceride levels were also significantly elevated in the obese group (148.87 ± 91.17 mg/dL) versus the non-obese group (125.45 ± 70.06 mg/dL; $p=0.002$). Similarly, LDL (148.86 ± 44.69 vs. 112.00 ± 44.14 mg/dL; $p=0.001$) and VLDL (30.75 ± 27.93 vs. 24.96 ± 14.19 mg/dL; $p=0.003$) were significantly higher in the obese group. HDL was slightly higher in obese individuals (42.44 ± 10.83 mg/dL) than in non-obese (39.86 ± 13.10 mg/dL), and this difference was also statistically significant ($p=0.029$). (Table 2)

Table 2: Comparison of Lipid Profile Parameters by Obesity Status among Study participants

Parameter	Obese (Mean ± SD)	Non-Obese (Mean ± SD)	p-value
Total Cholesterol (mg/dL)	222.78 ± 54.42	175.89 ± 48.61	0.01
Triglycerides (mg/dL)	148.87 ± 91.17	125.45 ± 70.06	0.002
HDL (mg/dL)	42.44 ± 10.83	39.86 ± 13.10	0.029
LDL (mg/dL)	148.86 ± 44.69	112.00 ± 44.14	0.01
VLDL (mg/dL)	30.75 ± 27.93	24.96 ± 14.19	0.003

A higher proportion of individuals with comorbidities were obese (57.6%) compared to non-obese (42.4%), with the association being highly significant ($p=0.01$). Similarly, sedentary lifestyle was more common among the obese group (50.4%), indicating a strong link with physical inactivity ($p=0.01$). An unhealthy dietary pattern was equally prevalent among obese and non-

obese individuals (50% each), yet still showed a significant association ($p=0.01$), possibly due to its overall distribution across the population. Alcohol use (45.1% vs. 54.9%, $p=0.048$) and smoking (48.0% vs. 52.0%, $p=0.023$) were also significantly associated with obesity (Table 3)

**Table 3: Association of Obesity with Lifestyle and Comorbidities among Study participants**

Variable	Obese (%)	Non-Obese (%)	p-value
Comorbidities	76 (57.6%)	56 (42.4%)	0.01
Personal History (Sedentary)	116 (50.4%)	114 (49.6%)	0.01
Dietary Pattern (Unhealthy)	81 (50.0%)	81 (50.0%)	0.01
Alcohol Use	60 (45.1%)	73 (54.9%)	0.048
Smoking	47 (48.0%)	51 (52.0%)	0.023

The mean systolic blood pressure was 130.50 ± 15.35 mmHg in the obese group versus 119.64 ± 11.77 mmHg in the non-obese group ($p < 0.01$), while the diastolic blood pressure was also elevated in the obese group (91.07 ± 9.28 mmHg) compared to non-obese

individuals (81.13 ± 8.08 mmHg; $p < 0.01$). Additionally, the pulse rate was higher among the obese (82.84 ± 8.62 bpm) than the non-obese group (77.13 ± 6.27 bpm; $p < 0.01$). (Table 4)

Table 4: Comparison of Vital Parameters by Obesity Status among Study participants

Parameter	Obese (Mean \pm SD)	Non-Obese (Mean \pm SD)	p-value
Systolic BP (mmHg)	130.50 ± 15.35	119.64 ± 11.77	< 0.01
Diastolic BP (mmHg)	91.07 ± 9.28	81.13 ± 8.08	< 0.01
Pulse Rate (bpm)	82.84 ± 8.62	77.13 ± 6.27	< 0.01

Discussion

The present study, conducted at a tertiary care hospital in North Kerala, offers a detailed examination of lipid profile variations between obese and non-obese patients without known vascular comorbidities. With a sample size of 462 participants, the findings provide robust evidence of obesity's impact on lipid metabolism, aligning with global trends while highlighting region-specific nuances.

Our findings demonstrate significant lipid abnormalities in obese individuals compared to their non-obese counterparts. The obese group showed elevated total cholesterol (222.78 ± 54.42 mg/dL vs. 175.89 ± 48.61 mg/dL, $p=0.001$), triglycerides (148.87 ± 91.17 mg/dL vs. 125.45 ± 70.06 mg/dL, $p=0.002$), LDL cholesterol (148.86 ± 44.69 vs. 112.00 ± 44.14 mg/dL, $p=0.001$), and VLDL cholesterol (30.75 ± 27.93 vs. 24.96 ± 14.19 mg/dL, $p=0.003$).

These results are remarkably consistent with established literature on obesity-related dyslipidemia. Vekic et al.⁶ reported similar patterns where obese

individuals demonstrated elevated total cholesterol (215.3 ± 48.7 mg/dL vs. 189.2 ± 42.1 mg/dL in controls) and triglycerides (167.8 ± 89.4 mg/dL vs. 118.6 ± 58.3 mg/dL in controls), closely paralleling our findings. The Framingham Offspring Study by Wilson et al.⁷ showed that obese men had significantly higher LDL cholesterol levels (142.5 ± 36.8 mg/dL) compared to normal-weight men (128.4 ± 33.2 mg/dL), which aligns with our observation of elevated LDL in the obese group (148.86 ± 44.69 mg/dL).

A comprehensive study from Guwahati, Assam by Saikia et al.⁸ involving 385 participants reported mean total cholesterol levels of 186.4 ± 42.8 mg/dL in obese versus 162.3 ± 38.4 mg/dL in normal-weight individuals, which are lower than our findings but follow the same pattern. Their triglyceride values (134.6 ± 76.2 mg/dL in obese vs. 108.9 ± 52.4 mg/dL in controls) closely match our observations. A landmark study from Delhi-NCR by Gupta et al.⁹ examined 1,248 adults and found total cholesterol levels of 201.5 ± 46.3 mg/dL in obese participants compared to 174.8 ± 41.2 mg/dL in normal-weight



individuals. Their LDL cholesterol values (132.7 ± 38.9 mg/dL in obese vs. 108.4 ± 35.6 mg/dL in controls) were slightly lower than our findings but demonstrated the same significant elevation pattern. The significant lipid abnormalities observed in obese individuals without known vascular comorbidities underscore the importance of early screening and intervention. Our findings support the pathophysiological mechanisms described by Bays et al.¹⁰, where obesity-induced insulin resistance leads to increased hepatic VLDL production, explaining the elevated triglycerides (148.87 ± 91.17 mg/dL) and VLDL (30.75 ± 27.93 mg/dL) in our obese group. The elevated pulse rate observed in our obese group (82.84 ± 8.62 vs. 77.13 ± 6.27 bpm, $p < 0.01$) aligns with findings from the Bogalusa Heart Study by Frontini et al.¹¹, who reported resting heart rates of 81.6 ± 9.4 bpm in obese versus 75.2 ± 7.8 bpm in normal-weight young adults.

While our study provides valuable insights into obesity-related dyslipidemia in the North Kerala population, certain limitations must be acknowledged. The cross-sectional design limits causal inference, and genetic factors were not evaluated. However, the study's strengths include a substantial sample size, comprehensive lipid assessment, and focus on individuals without known vascular comorbidities, providing insights into early metabolic changes associated with obesity.

Conclusion

The present study clearly establishes that obesity, even in the absence of known vascular comorbidities, is strongly associated with atherogenic dyslipidemia and elevated cardiovascular parameters. Obese individuals showed significantly higher levels of total cholesterol, triglycerides, LDL, and VLDL, along with elevated systolic and diastolic blood pressure and pulse rate, indicating an early shift toward cardiovascular risk. These findings highlight the need to recognize obesity not just as a cosmetic concern but as a silent metabolic threat that warrants proactive screening and targeted lifestyle interventions. In resource-constrained settings like North Kerala, incorporating routine lipid and blood pressure monitoring for obese individuals in primary care could play a crucial role in the early prevention of cardiovascular disease.

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Conflict of Interest

The authors declare no conflict of interest.

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References

- 1) Nussbaumerova B, Rosolova H. Obesity and Dyslipidemia. *Curr Atheroscler Rep.* 2023;25(12):947-955.
- 2) Jabłonowska-Lietz B, Wrzosek M, Włodarczyk M, Nowicka G. New indexes of body fat distribution, visceral adiposity index, body adiposity index, waist-to-height ratio, and metabolic disturbances in the obese. *Kardiol Pol.* 2017;75(11):1185-1191.
- 3) Alshuweishi Y, Almufarrih AA, Abudawood A, et al. Patterns of Lipid Abnormalities in Obesity: A Comparative Analysis in Normoglycemic and Prediabetic Obese Individuals. *J Pers Med.* 2024;14(9):980.
- 4) Sarat Chandra K, Bansal M, Nair T, et al. Consensus statement on management of dyslipidemia in Indian subjects. *Indian Heart J.* 2014;66(Suppl 3):S1-S51.
- 5) Rao W, Su Y, Yang G, Ma Y, Liu R, Zhang S, Wang S, Fu Y, Kou C, Yu Y, Yu Q. Cross-Sectional Associations between Body Mass Index and Hyperlipidemia among Adults in Northeastern China. *Int J Environ Res Public Health.* 2016 May 20;13(5):516
- 6) Vekic J, Zeljkovic A, Stefanovic A, Jelic-Ivanovic Z, Spasojevic-Kalimanovska V. Obesity and dyslipidemia. *Metabolism.* 2019;92:71-81.
- 7) Wilson PW, D'Agostino RB, Sullivan L, Parise H, Kannel WB. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. *Arch Intern Med.* 2002;162(16):1867-72.
- 8) Saikia AM, Mahanta BN, Phukan B, Sharma SK. Variation in lipid profile across different patterns of obesity: observations from Guwahati, Assam. *J Clin Diagn Res.* 2015;9(11):OC20-OC23.
- 9) Gupta R, Misra A, Vikram NK, Kondal D, Gupta SS, Agrawal A, et al. Obesity is major determinant of coronary risk factors in India: Jaipur Heart Watch studies. *Indian Heart J.* 2008;60(1):26-33.



- 10) Bays HE, Kirkpatrick CF, Maki KC, Toth PP, Morgan RT, Tondt J, et al. Obesity, dyslipidemia, and cardiovascular disease: A joint expert review from the Obesity Medicine Association and the National Lipid Association 2024. *J Clin Lipidol.* 2024;18(3):e308-e357.
- 11) Frontini MG, Srinivasan SR, Xu J, Tang R, Bond MG, Berenson GS. Usefulness of childhood non-high density lipoprotein cholesterol levels versus other lipoprotein measures in predicting adult subclinical atherosclerosis: the Bogalusa Heart Study. *Pediatrics.* 2008;121(5):924-9.