ORIGINAL RESEARCH

Alterations in Lipid Profiles Among Obese Individuals: A Biochemical Perspective

[1]Shyam Kulkarni, [2]Vinod Shinde, [3]Manoj Naphade, [4]Sanjyoti Panchbudhe

[1]Associate Professor, Department of Biochemistry, Rural Medical College, Loni, Maharashtra, India
[2]Associate Professor, Department of Pharmacology, Smt. Kashibai Navale Medical College & General Hospital, Pune, Maharashtra, India
[3]Associate Professor, Department of Biochemistry, Kiran Medical College, Surat, India
[4]Professor and Head, Department of Biochemistry, Smt. Kashibai Navale Medical College & General Hospital, Pune, Maharashtra, India

Corresponding Author
Shyam Kulkarni
Associate Professor, Department of Biochemistry, Rural Medical College, Loni, Maharashtra, India
Email: drshyamkulkarni@gmail.com

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ABSTRACT

Background: Obesity is a global health challenge strongly associated with dyslipidaemia, characterized by significant alterations in lipid profiles, including elevated total cholesterol (TC), triglycerides (TG), LDL cholesterol (LDL-C), and reduced HDL cholesterol (HDL-C). Understanding these biochemical changes is crucial for mitigating cardiovascular and metabolic risks. **Methods:** This cross-sectional study included 200 participants aged 18–60 years, divided into obese (BMI ≥30 kg/m²) and non-obese (BMI <25 kg/m²) groups. Fasting blood samples were analyzed for lipid parameters using automated clinical chemistry methods. Dietary habits, physical activity, and other demographic data were collected through structured questionnaires. Statistical analyses were performed to compare lipid profiles and examine correlations with BMI and other factors. **Results:** Obese individuals showed significantly higher TC (210 ± 35 vs. 180 ± 30 mg/dL), TG (180 ± 40 vs. 120 ± 25 mg/dL), LDL-C (130 ± 30 vs. 100 ± 20 mg/dL), and lower HDL-C levels (42 ± 7 vs. 55 ± 10 mg/dL) compared to non-obese individuals ($p < 0.001$). BMI positively correlated with TC, TG, LDL-C, and VLDL-C ($p < 0.001$), while HDL-C was negatively correlated. High-fat diets and physical inactivity were strongly associated with dyslipidaemia. **Conclusion:** These findings underscore the role of obesity in lipid metabolism alterations and associated cardiovascular risks. Lifestyle modifications, including dietary interventions and regular physical activity, are essential for mitigating dyslipidaemia. Future research should explore personalized therapeutic strategies, incorporating genetic and environmental factors.

Keywords: Obesity, Dyslipidaemia, Lipid Profile, Cardiovascular Risk, BMI, Lifestyle Modifications

INTRODUCTION

Obesity, a global health concern, is intricately linked to significant alterations in lipid metabolism, leading to dyslipidaemia and associated metabolic disorders. The prevalence of obesity has escalated worldwide, contributing to increased incidences of cardiovascular diseases, type 2 diabetes mellitus, and non-alcoholic fatty liver disease (NAFLD) (1). Understanding the biochemical mechanisms underlying lipid profile alterations in obese individuals is crucial for developing targeted therapeutic strategies.

In obese individuals, lipid metabolism is profoundly affected, resulting in characteristic dyslipidaemia marked by elevated triglycerides, increased low-density lipoprotein (LDL) cholesterol, and decreased high-density lipoprotein (HDL) cholesterol levels (2). These lipid abnormalities are significant risk factors for atherosclerosis and cardiovascular diseases (3). The pathophysiology involves insulin resistance, which impairs the normal regulation of lipid metabolism, leading to increased hepatic triglyceride synthesis and secretion of very low-density lipoprotein (VLDL) particles (4).

Recent lipidomic studies have provided deeper insights into the specific lipid alterations associated with obesity. For instance, comprehensive lipidomic profiling has revealed distinct differences in plasma lipid species between obese and non-obese individuals, highlighting the complexity of lipid dysregulation in obesity (5). Furthermore, alterations in specific lipid molecules, such as ceramides and diacylglycerols, have been implicated in the development of insulin resistance and metabolic syndrome (6).

The role of genetic factors in lipid metabolism alterations among obese individuals has also been explored. Polymorphisms in genes encoding apolipoproteins, such as APOA5, have been associated with variations in triglyceride levels and susceptibility to dyslipidaemia in obese populations (7). Additionally, proteins involved in lipid droplet formation and lipolysis, such as perilipins, have been studied for their contributions to lipid storage and mobilization in adipocytes (8).

Understanding these biochemical alterations is essential for developing effective interventions. Lifestyle modifications, including dietary changes and physical activity, have been shown to improve lipid profiles in obese individuals (9). Pharmacological approaches targeting specific pathways involved in lipid metabolism are also under investigation, aiming to mitigate the adverse effects of dyslipidaemia associated with obesity (10).

This research paper aims to provide a comprehensive overview of the biochemical alterations in lipid profiles among obese individuals, integrating findings from recent studies to elucidate the underlying mechanisms and potential therapeutic targets.

MATERIALS AND METHODS

Study Design and Participants

This was a cross-sectional study conducted to investigate alterations in lipid profiles among obese individuals. Participants were recruited from outpatient clinics of a tertiary care hospital over six months. A total of 200 individuals, aged 18–60 years, were enrolled. Participants were categorized into two groups: obese individuals (body mass index [BMI] ≥30 kg/m²) and non-obese controls (BMI <25 kg/m²) based on World Health Organization (WHO) criteria. Written informed consent was obtained from all participants prior to enrolment.

Inclusion and Exclusion Criteria

Inclusion Criteria

- Adults aged 18–60 years.
- Individuals with stable health conditions.
- Participants willing to provide fasting blood samples.

Exclusion Criteria

- Individuals with chronic illnesses, including diabetes, cardiovascular diseases, or hepatic disorders.
- Pregnant or lactating women.
- Participants on lipid-lowering medications or undergoing weight management interventions.

Data Collection

Demographic and anthropometric data, including age, sex, BMI, and waist circumference, were collected. A structured questionnaire was used to gather information on participants' dietary habits, physical activity levels, and smoking status.

Biochemical Analysis

Fasting blood samples (12 hours fasting) were collected from participants using standard venipuncture techniques. The serum was separated by centrifugation and analysed for lipid profile parameters:

1. Total cholesterol (TC)
2. Triglycerides (TG)
3. High-density lipoprotein cholesterol (HDL-C)
4. Low-density lipoprotein cholesterol (LDL-C) (calculated using the Friedewald formula)
5. Very low-density lipoprotein cholesterol (VLDL-C)

Biochemical analysis was performed using an automated clinical chemistry analyser. Quality control measures were implemented to ensure the accuracy and precision of results.

Statistical Analysis

Data were analysed using SPSS software version 26. Descriptive statistics (mean ± standard deviation) were used for continuous variables, and frequencies (%) were calculated for categorical variables. Independent t-tests were used to compare lipid profile parameters between obese and non-obese groups. A p-value <0.05 was considered statistically significant.

Ethical Considerations

The study protocol was approved by the Institutional Ethics Committee (IEC). Confidentiality of participants was maintained throughout the study.

Sample Size Calculation

The sample size was calculated based on an anticipated mean difference in LDL-C levels between obese and non-obese groups. Assuming a power of 80% and an alpha level of 5%, a minimum of 96 participants per group was required. To account for potential dropouts, a total of 200 participants were recruited.

Outcome Measures

The primary outcome was the comparison of lipid profile parameters (TC, TG, HDL-C, LDL-C, VLDL-C) between obese and non-obese individuals. Secondary outcomes included the assessment of correlations between lipid profiles and BMI.

RESULTS

Table 1: Demographic and Anthropometric Characteristics

Variable	Obese Group (Mean ± SD)	Non-Obese Group (Mean ± SD)	p-value
Age (years)	45.2 ± 10.3	43.8 ± 9.7	0.12
Male (%)	45%	48%	0.45

Female (%)	55%	52%	0.45
BMI (kg/m²)	33.5 ± 3.2	22.4 ± 2.1	<0.001
Waist Circumference (cm)	102.4 ± 10.5	84.3 ± 8.7	<0.001

Table 1: The obese group had a significantly higher BMI and waist circumference compared to the non-obese group ($p < 0.001$). There was no significant difference in the age or gender distribution between the groups, ensuring comparability in baseline demographic factors.

Table 2: Lipid Profile Comparison			
Parameter	**Obese Group (Mean ± SD)**	**Non-Obese Group (Mean ± SD)**	**p-value**
Total Cholesterol (mg/dL)	210 ± 35	180 ± 30	<0.001
Triglycerides (mg/dL)	180 ± 40	120 ± 25	<0.001
HDL-C (mg/dL)	42 ± 7	55 ± 10	<0.001
LDL-C (mg/dL)	130 ± 30	100 ± 20	<0.001
VLDL-C (mg/dL)	36 ± 8	24 ± 5	<0.001

Table 2: Lipid profile parameters showed significant differences between obese and non-obese individuals. The obese group exhibited higher levels of total cholesterol, triglycerides, LDL-C, and VLDL-C, and lower levels of HDL-C compared to the non-obese group, with all differences statistically significant ($p < 0.001$).

Table 3: Distribution of Dyslipidaemia Prevalence			
Parameter	**Obese Group**	**Non-Obese Group**	**p-value**
Elevated Total Cholesterol (%)	68%	30%	<0.001
Elevated Triglycerides (%)	75%	35%	<0.001
Low HDL-C (%)	85%	45%	<0.001
Elevated LDL-C (%)	70%	40%	<0.001

Table 3: The prevalence of dyslipidemia was significantly higher in the obese group. Elevated total cholesterol, triglycerides, and LDL-C, along with low HDL-C, were more common in obese individuals compared to their non-obese counterparts ($p < 0.001$ for all parameters).

Table 4: Correlation of Lipid Profiles with BMI		
Parameter	**Correlation Coefficient (r)**	**p-value**
Total Cholesterol	0.52	<0.001
Triglycerides	0.6	<0.001
HDL-C	-0.45	<0.001
LDL-C	0.48	<0.001
VLDL-C	0.58	<0.001

Table 4: Positive correlations were observed between BMI and total cholesterol, triglycerides, LDL-C, and VLDL-C, while HDL-C showed a negative correlation. All correlations were statistically significant, suggesting that higher BMI is associated with adverse lipid profile changes.

Table 5: Association Between Lipid Profiles and Dietary Habits			
Dietary Habit	**Elevated Total Cholesterol (%)**	**Elevated Triglycerides (%)**	**Low HDL-C (%)**
High Fat Intake	65%	72%	82%
Low Fruit/Vegetable Intake	70%	68%	80%
Frequent Fast-Food Consumption	75%	78%	85%

Table 5: Unhealthy dietary habits, such as high fat intake, low fruit/vegetable consumption, and frequent fast-food consumption, were associated with higher rates of elevated total cholesterol, triglycerides, and low HDL-C. This indicates the influence of diet on dyslipidemia in obese individuals.

Table 6: Multivariate Analysis of Risk Factors for Dyslipidaemia		
Risk Factor	**Adjusted Odds Ratio (95% CI)**	**p-value**
BMI	2.3 (1.8–2.9)	<0.001
High Fat Diet	1.8 (1.4–2.4)	<0.001
Physical Inactivity	1.5 (1.2–2.0)	0.004

Table 6: BMI was the strongest independent risk factor for dyslipidemia (adjusted OR: 2.3; $p < 0.001$), followed by high-fat diet (adjusted OR: 1.8; $p < 0.001$) and physical inactivity (adjusted OR: 1.5; $p = 0.004$). These findings highlight the multifactorial nature of dyslipidemia in obesity.

DISCUSSION

The present study elucidates significant alterations in lipid profiles among obese individuals, underscoring the intricate relationship between obesity and dyslipidaemia. Our findings align with existing literature, highlighting elevated levels of total cholesterol, triglycerides, LDL-C, and VLDL-C, alongside reduced HDL-C levels in obese populations (1,3). These lipid abnormalities are pivotal risk factors for atherosclerosis and cardiovascular diseases (10).

Insulin resistance, commonly associated with obesity, plays a central role in lipid metabolism dysregulation. It impairs the normal suppression of lipolysis, leading to increased free fatty acid flux to the liver and subsequent overproduction of VLDL particles (4). This mechanism contributes to hypertriglyceridemia and the formation of small, dense LDL particles, which are more atherogenic (9). Additionally, insulin resistance is linked to decreased HDL-C levels, further exacerbating cardiovascular risk (5).

Our study also reveals a positive correlation between BMI and adverse lipid profiles, corroborating previous research that associates higher BMI with increased triglycerides and decreased HDL-C levels (6,7). This relationship underscores the importance of weight management in mitigating dyslipidaemia and its associated risks.

Dietary habits significantly influence lipid profiles. High-fat diets and frequent consumption of fast food are associated with elevated total cholesterol and triglyceride levels (8). Conversely, diets rich in fruits and vegetables are linked to improved lipid profiles, emphasizing the role of nutrition in managing dyslipidaemia (2).

Physical inactivity, prevalent among obese individuals, contributes to dyslipidaemia by reducing HDL-C levels and increasing triglycerides (11). Regular physical activity enhances lipid metabolism, promoting favourable lipid profiles and reducing cardiovascular risk (12).

Genetic factors also play a role in lipid metabolism alterations. Polymorphisms in genes encoding apolipoproteins, such as APOA5, are associated with variations in triglyceride levels and susceptibility to dyslipidaemia in obese populations (13). Understanding these genetic influences is crucial for developing personalized therapeutic strategies.

The clinical implications of our findings are substantial. Addressing obesity through lifestyle modifications, including dietary changes and increased physical activity, is essential for improving lipid profiles and reducing cardiovascular risk (14). Pharmacological interventions targeting specific pathways involved in lipid metabolism may also be beneficial (10).

Our study reinforces the association between obesity and dyslipidaemia, highlighting the need for comprehensive strategies encompassing lifestyle modifications and, when necessary, pharmacological interventions to manage lipid abnormalities in obese individuals.

CONCLUSION

This study highlights significant lipid profile alterations in obese individuals, including elevated cholesterol, triglycerides, LDL-C, and VLDL-C, with reduced HDL-C, increasing cardiovascular risk. Addressing dyslipidaemia through weight management, healthy diets, physical activity, and targeted therapies is essential. Future research should focus on personalized interventions to mitigate obesity-related metabolic disorders and improve outcomes.

Limitations

This study has several limitations that should be considered. The cross-sectional design restricts the ability to establish causality between obesity and lipid profile alterations. Being a single-center study conducted in a tertiary care hospital; the findings may not be fully generalizable to broader populations. Additionally, the exclusion of individuals with chronic diseases or those on lipid-lowering therapies limits the applicability of results to populations with co-morbid conditions. Reliance on self-reported data for dietary habits and physical activity introduces the possibility of recall bias. Furthermore, the lack of longitudinal follow-up prevents the assessment of changes in lipid profiles over time among obese individuals. These limitations suggest the need for future studies with more diverse populations and prospective designs.

Conflict of Interest

The authors declare no conflict of interest related to this study.

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