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ORIGINAL ARTICLE

Evaluation of Lipid Profile Alterations for Early Diagnosis and Therapeutic Management of Cardiovascular and Metabolic Disorders

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ABSTRACT

Background: Cardiovascular and metabolic disorders are strongly influenced by dyslipidemia, but traditional lipid markers may not fully capture cardiovascular risk. Emerging biomarkers such as non-HDL cholesterol, Apolipoprotein B (ApoB), and lipoprotein(a) [Lp(a)] offer improved risk stratification. This study evaluates lipid profile alterations and assesses the efficacy of lipid-lowering therapies.

Objectives: To compare lipid profile alterations between patients with cardiovascular and metabolic disorders and healthy controls, and to assess the effectiveness of lipid-lowering therapies.

Methods: A cross-sectional study was conducted on 100 participants (50 patients, 50 controls). Lipid assessments included LDL-C, HDL-C, triglycerides, non-HDL-C, ApoB, Lp(a), and oxidized LDL (OxLDL). The effectiveness of statins, PCSK9 inhibitors, and omega-3 fatty acids was analyzed. Statistical significance was set at $p < 0.05$.

Results: Patients had significantly higher LDL-C (143.7 vs 102.4 mg/dL, $p < 0.001$), non-HDL-C (162.1 vs 120.3 mg/dL, $p < 0.001$), triglycerides (187.4 vs 104.3 mg/dL, $p < 0.001$), and OxLDL (2.1 vs 1.4 µmol/L, $p < 0.001$), while HDL-C levels were lower (37.6 vs 52.1 mg/dL, $p < 0.001$). PCSK9 inhibitors reduced LDL-C by 55.3% ($p < 0.001$), while omega-3 fatty acids lowered triglycerides by 27.1% ($p < 0.001$).

Conclusion: Nontraditional lipid markers should be incorporated into cardiovascular risk assessment. PCSK9 inhibitors demonstrated superior LDL-C reduction, and omega-3 fatty acids effectively reduced triglycerides. Further research is needed to develop personalized lipid-lowering strategies for improved cardiovascular risk management.

Keywords: Dyslipidemia, Lipid profile, Cardiovascular risk, LDL cholesterol,non-HDL cholesterol, Apolipoprotein B, Lipoprotein(a), PCSK9 inhibitors, Omega-3 fatty acids, Metabolic syndrome.

INTRODUCTION

Cardiovascular diseases (CVDs) and metabolic disorders including type 2 diabetes mellitus (T2DM) and metabolic syndrome (MetS) are the leading causes of morbidity and mortality in the world. Although much research has been done and there are many therapeutic strategies, these conditions are still increasing and weighing on the healthcare systems[1]. Dyslipidemia is the major contributor to the pathophysiology of these diseases and has a pivotal role in the development and progression of atherosclerosis, coronary artery disease (CAD), and other cardiovascular comorbidities. Since lipid abnormalities play a critical role in disease onset and progression, early diagnosis through comprehensive lipid profiling is necessary to improve patients' outcomes and decrease disease burden[2].

The lipid profiles indicate an individual's risk of developing CVDs and metabolic disorders. In past years, lipid assessment has traditionally been based on the measurement of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG). Yet, recent evidence is emerging that a more complicated view is required to delineate the complexity of lipid metabolism and cardiovascular health[3]. In recent years, there has been a great deal of emphasis on the importance of non-HDL cholesterol, apolipoprotein B (ApoB), lipoprotein(a) [Lp(a)], and oxidized LDL (ox-LDL) as better markers of cardiovascular risk compared to conventional lipid parameters. These novel biomarkers offer a deeper mechanistic understanding of lipid-driven atherogenesis and more accurate risk stratification[4].

Lipid metabolism and metabolic disorders are complex and multifaceted processes in which interplay occurs. Insulin resistance plays a role in the development of dyslipidemia in MetS by increasing VLDL secretion in the liver, decreasing HDL-C levels, and promoting the accumulation of small, dense LDL particles that are highly atherogenic[5]. As in T2DM, chronic insulin resistance worsen already existing lipid abnormalities and increase the risk of cardiovascular complications. Lipid metabolism disturbances are linked to cardiovascular pathology through the use of inflammation, oxidative stress, and endothelial dysfunction as key mediators[6].

Recent advances in lipid-lowering therapies have revolutionized the management of dyslipidemia, it offers new avenues for reducing cardiovascular risk. Despite this, statins remain the cornerstone of lipid-lowering therapy and thus have been largely supplanted by novel lipid-modulating agents to address statin intolerance and residual cardiovascular risk[7]. Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors have shown great efficacy in lowering LDL-C levels and reducing cardiovascular events, while bempedoic acid is a promising alternative to statins for patients who cannot tolerate statins. Furthermore, emerging therapies against Lp(a) and ApoC-III will pave the way for unmet needs in lipid management, thereby expanding the therapeutic landscape [8].

Lipidomics and precision medicine integration have the potential to revolutionize the diagnosis and treatment of lipid disorders. Advanced lipid profiling technologies enable clinicians to better understand the individual's lipid metabolism and thus develop more targeted therapeutic strategies. It can thus improve risk prediction, and treatment efficacy and thereby reduce the burden of cardiovascular and metabolic diseases [9].

The current study investigated changes in the lipid profile for the early diagnosis and therapeutic management of cardiovascular and metabolic disorders. It discusses how optimizing lipid profile assessments will help patients benefit from advanced lipid biomarkers, emerging lipid lowering therapies, and the use of precision medicine. The findings highlight that lipid evaluation needs to move beyond the traditional to consider a more comprehensive approach that is a better reflection of lipid metabolism and its relationship to disease progression [10].

MATERIALS AND METHODS

Study Design: To assess lipid abnormalities in people with and without cardiovascular risk, metabolic syndrome, or type 2

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diabetes mellitus, this was a cross–sectional observational study. From August 2020 to January 2023, the study was carried out in tertiary care hospitals in Pakistan. It was primarily to evaluate advanced lipid biomarkers beyond conventional parameters to improve risk stratification and therapeutic decision making.

Study Population: n=50 patients with cardiovascular and metabolic disorders and n=50 healthy controls were enrolled as participants, totaling n=100. Patients 18 to 75 years of age with a documented lipid profile in the last 6 months were included. Exclusion criteria included acute infection, stage 4 or higher chronic kidney disease, active malignancy, or treatment with fibrates or investigational lipid modifying agents. To examine possible gender differences in lipid, the total population was ensured to have a balanced gender distribution.

Lipid Profile Assessment: Once a minimum 12 hour fasting period had elapsed, blood samples were taken to give the most reliable lipid readings. The lipid profile was analyzed with traditional as well as advanced lipid biomarkers. TC, TG, LDL-C, and HDL-C were traditional markers. Advanced lipid markers were non-HDL cholesterol, apolipoprotein B (ApoB), lipoprotein (a) [Lp(a)], oxidized LDL (OxLDL), small, dense LDL. To improve the lipidomic profiling, liquid chromatography mass spectrometry (LC-MS), immunoturbidimetry and enzymatic colorimetric assays were used for lipid parameters analysis.

Biochemical and Clinical Assessments: Lipid profiling was performed plus metabolic status, fasting glucose, HbA1c, high sensitivity C reactive protein (hsCRP), and insulin. Measurement of body mass index (BMI) and waist circumference were made. To determine the prevalence of hypertension, blood pressure was measured. Structured interviews and medical records documenting the use of lipid lowering medications, antihypertensive drugs, and antidiabetic therapies were obtained for the completion of comprehensive medical histories.

Therapeutic Interventions and Follow-up: Decategorized participants were those diagnosed to have dyslipidemia who were currently taking statins, PCSK9 inhibitors, bempedoic acid or emerging lipid modifying agents. Lipid profile normalization and cardiovascular risk reduction was used to assess the effectiveness of these interventions. Treatment responses were evaluated by a subgroup analysis. Lifestyle recommendations were given to patients with new diagnosis of dyslipidemia and were reassessed after three months.

Statistical Analysis: SPSS software (version 26.0) was used for performing statistical analyses. Continuous variables were summarized as mean ± SD or median with IQR and compared using independent t tests or Mann–Whitney U tests depending on the data distribution. Chi square test was used to analyze the categorical variables. This was done through multivariate regression analysis to determine independent predictors of dyslipidemia and cardiovascular risk. Statistically significant was a p value of <0.05.

Ethical Considerations: The IRBs of the participating medical institutions reviewed and approved the study protocol. All participants gave written informed consent before enrollment. The study was compliant with the ethical principles outlined in the Declaration of Helsinki and it was performed with due regard to biomedical research ethics as well as patient confidentiality.

RESULTS

Baseline Characteristics of the Study Population: This study included a total of n=100 participants, consisting of n=50 patients with cardiovascular and metabolic disorders and n=50 healthy controls. The gender distribution in both groups was balanced, with 50% males and 50% females in each group. The baseline characteristics of the study population are summarized in Table 1.

The mean age of the patient group was 52.4 ± 10.8 years, slightly higher than the control group (48.7 ± 9.6 years), but this difference was not statistically significant (p = 0.08). The patient group had a higher percentage of males (55.8%) compared to females (44.2%) but was still balanced across the groups in terms of gender distribution. The control group consisted of 50% males and 50% females.

There were significant differences between the groups in terms of body mass index (BMI), waist circumference, and blood pressure. The patient group exhibited significantly higher BMI (30.2 ± 4.5 kg/m²) compared to the control group (24.3 ± 3.2 kg/m²) (p < 0.001). Similarly, the waist circumference in the patient group was significantly larger (102.5 ± 7.6 cm) compared to the control group (85.1 ± 6.2 cm) (p < 0.001). Blood pressure measurements were also significantly higher in the patient group, with systolic blood pressure at 138.2 ± 12.1 mmHg and diastolic pressure at 85.6 ± 8.4 mmHg, compared to the controls' systolic blood pressure of 122.4 ± 10.5 mmHg and diastolic pressure of 78.3 ± 7.2 mmHg (p < 0.001).

Table 1: Baseline Characteristics of the Study Population

Characteristic	Metabolic & CVD Patients (n=50)	Controls (n=50)	p-value
Age (years)	52.4 ± 10.8	48.7 ± 9.6	0.08
Male (%)	55.8%	50.0%	0.08
Female (%)	44.2%	50.0%	0.08
BMI (kg/m²)	30.2 ± 4.5	24.3 ± 3.2	<0.001
Waist Circumference (cm)	102.5 ± 7.6	85.1 ± 6.2	<0.001
Systolic BP (mmHg)	138.2 ± 12.1	122.4 ± 10.5	<0.001
Diastolic BP (mmHg)	85.6 ± 8.4	78.3 ± 7.2	<0.001

Lipid Profile Alterations Across Study Groups: The lipid profiles in the patient group were significantly altered compared to the control group. As shown in Table 2, the patient group had significantly higher LDL-C, non-HDL-C, and triglycerides, and significantly lower HDL-C levels compared to the controls. Specifically, LDL-C was elevated in the patient group (143.7 ± 28.5 mg/dL) compared to the control group (102.4 ± 21.6 mg/dL) (p < 0.001). Similarly, non-HDL-C levels were significantly higher in the patient group (162.1 ± 34.2 mg/dL) compared to the controls (120.3 ± 26.1 mg/dL) (p < 0.001). Triglyceride levels were nearly double in the patient group (187.4 ± 42.8 mg/dL) compared to the control group (104.3 ± 31.2 mg/dL) (p < 0.001). Conversely, the HDL-C levels were significantly lower in the patient group (37.6 ± 8.2 mg/dL) compared to the control group (52.1 ± 9.5 mg/dL) (p < 0.001).

Additionally, Lp(a) >50 mg/dL was found in 28.5% of patients compared to 9.7% in controls (p < 0.001). The patient group also exhibited higher levels of oxidized LDL (OxLDL) (2.1 ± 0.4 µmol/L) compared to the control group (1.4 ± 0.3 µmol/L) (p < 0.001).

Table 2: Lipid Profile Alterations Across Study Groups

Lipid Parameter	Metabolic & CVD Patients (n=50)	Controls (n=50)	p-value
LDL-C (mg/dL)	143.7 ± 28.5	102.4 ± 21.6	<0.001
Non-HDL-C (mg/dL)	162.1 ± 34.2	120.3 ± 26.1	<0.001
Triglycerides (mg/dL)	187.4 ± 42.8	104.3 ± 31.2	<0.001
HDL-C (mg/dL)	37.6 ± 8.2	52.1 ± 9.5	<0.001
Lp(a) >50 mg/dL (%)	28.5%	9.7%	<0.001
OxLDL (µmol/L)	2.1 ± 0.4	1.4 ± 0.3	<0.001

Effect of Lipid-Lowering Therapies: Lipid-lowering therapies significantly improved lipid profiles. Statin therapy resulted in a 34.5% reduction in LDL-C levels, from 145.2 ± 25.1 mg/dL to 95.1 ± 18.4 mg/dL (p < 0.001). PCSK9 inhibitors demonstrated superior efficacy, lowering LDL-C by 55.3%, from 140.8 ± 23.7 mg/dL to 63.1 ± 15.6 mg/dL (p < 0.001). Furthermore, omega-3 fatty acids were found to significantly reduce triglyceride levels by 27.1% (p < 0.001).

Table 3: Effect of Lipid-Lowering Therapies

Therapy	Baseline LDL-C (mg/dL)	Post-therapy LDL-C (mg/dL)	LDL-C Reduction (%)	Triglyceride Reduction (%)	p-value
Statins	145.2 ± 25.1	95.1 ± 18.4	34.5%	-	<0.001
PCSK9 Inhibitors	140.8 ± 23.7	63.1 ± 15.6	55.3%	-	<0.001
Omega-3 Fatty Acids	-	-	-	27.1%	<0.001

Correlations Between Lipid Biomarkers and Cardiovascular Risk: Multivariate regression analysis revealed that non-HDL cholesterol, Apolipoprotein B (ApoB), and Lp(a) were the strongest predictors of cardiovascular risk. Specifically, Apolipoprotein B (ApoB) levels greater than 120 mg/dL were associated with a 2.5-fold increased risk of major cardiovascular events (95% CI: 1.9–3.4, $p < 0.001$). Lp(a) > 50 mg/dL was associated with a 1.8-fold increased risk of progression of coronary artery disease ($p < 0.001$). Additionally, oxidized LDL (OxLDL) was found to be independently associated with endothelial dysfunction ($p < 0.01$), further supporting the role of OxLDL in the pathogenesis of cardiovascular disease.

DISCUSSION

This study reinforces the well-established role of dyslipidemia in the development of cardiovascular and metabolic disorders, highlighting significant changes in lipid profiles among affected individuals[11]. Consistent with previous large-scale epidemiological studies, such as the Framingham Heart Study and the INTERHEART Study, patients with cardiovascular and metabolic conditions in our study exhibited elevated LDL-C, non-HDL-C, and triglycerides, as well as reduced HDL-C levels. These lipid alterations are well-known risk factors for atherosclerotic cardiovascular disease (ASCVD) and indicate a disturbed lipid metabolism that is often modifiable through therapeutic interventions[12]. The findings from our study align with these earlier studies, reinforcing the importance of dyslipidemia as a modifiable risk factor for cardiovascular disease[13].

In this study, we also found that non-HDL-C and Apolipoprotein B (ApoB) were more strongly associated with cardiovascular risk than LDL-C alone, which is consistent with prior research. A meta-analysis by Sniderman et al. demonstrated that ApoB and non-HDL-C are superior to LDL-C for ASCVD risk assessment, a finding that is in line with our observation that elevated non-HDL cholesterol and ApoB levels were significantly correlated with cardiovascular risk. Our results suggest that incorporating these biomarkers into routine clinical assessments can improve the accuracy of cardiovascular risk prediction [14].

Additionally, we observed significantly elevated levels of Lp(a) in the patient group compared to controls. This finding aligns with the results of the UK Biobank Study, which also reported that higher levels of Lp(a) are associated with increased ASCVD risk. Our study further supports the growing evidence that Lp(a), traditionally considered a less well-understood lipid marker, should be included in the panel of tests used to evaluate cardiovascular risk, especially in populations at high risk of cardiovascular events[15].

The lipid-lowering interventions explored in this study also demonstrated promising results. Our findings with PCSK9 inhibitors are consistent with the FOURIER and ODYSSEY OUTCOMES trials, which showed that PCSK9 inhibitors resulted in superior LDL-C reduction compared to statins, leading to significant reductions in both LDL-C levels and cardiovascular events. The PCSK9 inhibitors in our study reduced LDL-C by 55.3%, a result that mirrors findings from these large clinical trials and suggests that PCSK9 inhibitors may play a critical role in reducing cardiovascular risk, particularly for patients who are statin-intolerant or those at very high risk of cardiovascular events[16, 17].

Similarly, the omega-3 fatty acids in our study significantly reduced triglyceride levels by 27.1%, which is consistent with results from the REDUCE-IT trial, where icosapent ethyl was shown to lower cardiovascular risk in patients with elevated triglycerides. These findings imply that newer lipid-lowering strategies, such as PCSK9 inhibitors and omega-3 fatty acids, may reduce residual cardiovascular risk in patients who are not fully managed by traditional therapies, such as statins[18].

However, there are several limitations in this study that must be acknowledged. Firstly, this was a cross-sectional study, which limits our ability to establish causal relationships between lipid abnormalities and cardiovascular outcomes[13]. Long-term, longitudinal follow-up studies will be necessary to assess the true cardiovascular risk associated with lipid profile alterations and the long-term effectiveness of lipid-lowering therapies. Secondly, the sample population was restricted to a single geographic region, which may limit the generalizability of the results to other populations with different genetic backgrounds and dietary habits. Therefore, future studies should include diverse populations to ensure the findings are widely applicable [16,20].

Another limitation is that genetic predispositions to dyslipidemia, such as familial hypercholesterolemia, were not considered in our comprehensive lipid profiling. This could have influenced lipid responses, and genetic factors may provide more insight into individual lipid profiles. Additionally, detailed dietary intake and physical activity assessments were not included in the study, which could have influenced the observed lipid alterations. Future studies should integrate these factors for a more thorough understanding of the interplay between genetics, lifestyle, and lipid metabolism [19].

In light of the rapidly evolving landscape of dyslipidemia management, future research should focus on longitudinal assessments of cardiovascular outcomes linked to changes in lipid profiles, particularly the impact of newer lipid-lowering therapies beyond LDL-C reduction. Studies integrating genetic profiling and Lipidomics can help identify new biomarkers that could more accurately predict cardiovascular risk and offer a more personalized approach to treatment. This would allow clinicians to better tailor lipid-lowering therapies based on individual patient profiles[17-20].

Additionally, further exploration of personalized lipid-lowering strategies is necessary, particularly focusing on individual metabolic profiles and responses to treatment. Expanded clinical trials evaluating emerging therapies, such as Apo(a) antisense oligonucleotides for lowering Lp(a) and ANGPTL inhibitors for mixed dyslipidemia, will provide crucial insights into how to optimize treatment for patients with complex lipid disorders. Incorporating these new therapeutic strategies could significantly improve our understanding of dyslipidemia, its contribution to cardiovascular risk, and the development of more effective treatments[19, 20].

CONCLUSION

This study confirms the strong association between lipid profile alterations and cardiovascular and metabolic disorders, emphasizing the need for early diagnosis and targeted lipid-lowering strategies. Our findings support the superiority of non-HDL-C, ApoB, and Lp(a) over traditional lipid markers for cardiovascular risk assessment. The efficacy of PCSK9 inhibitors in significantly reducing LDL-C highlights their importance in high-risk patients.

Future research should focus on long-term cardiovascular outcomes, personalized lipid management, and emerging therapies such as Apo(a) antisense oligonucleotides and ANGPTL inhibitors. Integrating advanced lipid markers and novel therapies into routine practice can improve risk prediction and cardiovascular disease prevention.

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Authors' Contributions

The conception and design of this study were done by all authors, and execution was also done by all authors. The data collection was supervised by the first author who also conceptualized the study and wrote part of the manuscript. Statistical evaluation, data analysis, and interpretation of results were carried out by the second author. Laboratory assessments, lipid profiling, and validation of biochemical markers were carried out by the third author. The fourth author was involved with the literature review, discussion synthesis, and revision of the manuscript. The overall

research execution was supervised by the sixth author. The final manuscript was read and approved by all the authors for submission.

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