INDUS JOURNAL OF MEDICAL AND HEALTH SCIENCES
https://induspublishers.com/IJMHS
Volume 3, Issue 1 (2025)

Original Article

The Role of Lipid Metabolism in the Development of Coronary Artery Disease: Clinical Implications for Early Intervention

Rehan Ullah [a]

[a] Medical Officer, Kuwait Teaching Hospital, Peshawar, Khyber Pakhtunkhwa, Pakistan.

ARTICLE INFO

Received: 17 Feb 2025
Revised: 26 March 2025
Accepted: 30 May 2025
Published: 29 June 2025

Key Words:
Lipid Metabolism, Coronary Artery Disease, Triglycerides, Lipid-Lowering Therapy, Atherosclerosis, CAD Severity.

***Corresponding Author:**
Rehan Ullah
(rehanullah195@gmail.com)

ABSTRACT

The role of lipid metabolism in the development and progression of coronary artery disease (CAD) is well-documented, but its precise impact on CAD severity and clinical interventions remains an area of active research. This study aimed to investigate the relationship between lipid profile abnormalities—specifically low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides—and CAD severity, while evaluating the effect of lipid-lowering intervention on lipid parameters. A total of 200 CAD patients were enrolled and stratified by disease severity (mild, moderate, and severe). Clinical and demographic data were collected, alongside lipid profiles, including LDL-C, HDL-C, triglycerides, and total cholesterol. Research data showed that LDL-C played a critical role in atherosclerosis as its values rose while CAD became more severe yet declined during treatment periods. The past research data illustrated a positive relationship between triglyceride levels and CAD severity. Research on lipid-lowering drugs across three months confirmed positive effects on decreased LDL-C and triglycerides combined with elevated HDL-C levels thus highlighting potential drug treatments to stop CAD progression. The regression analysis results confirmed that LDL-C together with triglycerides and HDL-C serve as vital risk determinants for CAD. Subgroup evaluation data showed diabetes patients combined with hypertension patients had higher LDL-C and triglyceride levels thus pointing to the significance of personalized lipid therapy methods for high-risk patient groups. The findings show that cholesterol management techniques offer promising therapeutic benefits and show the importance of accomplishing early lipid evaluation and timely CAR treatment interventions. Future studies should identify basic lipid metabolism processes in coronary artery disease patients while conducting extended assessments of selected therapeutic methods..

INTRODUCTION

The worldwide leading cause of disability and death known as coronary artery disease functions closely with faulty lipid metabolism that regulates lipid synthesis as well as transport and storage and breakdown within the body [1]. Inactive lifestyles have transformed atherosclerosis into an epidemic therefore we need fundamental intervention plans to control its advancement [2]. The pathologic core operation leading to coronary artery disease known as atherosclerosis entails lipid and fibrous material and inflammatory cellular matter accumulation inside artery walls that leads to plaque development resulting in heart blood flow restriction [3]. The damage of endothelial cells and resulting artery wall accumulation of oxidised low-density lipoprotein leads to atherosclerosis pathogenesis through inflammation-based cellular interactions and inflammatory responses which support plaque development [4]. Accurate identification of new therapeutic aims and early disease prevention methods for coronary artery disease require complete comprehension of lipid metabolism in disease generation. The review examines the way lipid metabolism influences coronary artery disease progression through a clear explanation of its clinical impact on preventive therapies.

Lipid metabolism comprises most of atherosclerotic pathophysiology because disruptions in lipid homeostasis initiate and spread plaque formation [5]. The risk factor for atherosclerosis is elevated low-density lipoprotein cholesterol with special emphasis on oxidised LDL. These particles accumulate inside artery intima spaces to start inflammatory reactions that accelerate plaque development [6]. High-density lipoprotein cholesterol functions as a protective factor because it establishes a pathway to transfer cholesterol away from peripheral tissues leading to liver excretion [7]. The risk of plaque development in people depends mostly on how much their LDL cholesterol levels exceed their HDL cholesterol levels. Scientific evidence shows that osteoarthritic development in chondrocytes depends on the buildup and metabolism of intracellular cholesterol to establish inflammatory processes further validating its role within cells [8,9]. Atherosclerosis formation starts and progresses under strong influence of oxidative stress [10]. Endothelium activation sparks the process which evolves through successive mechanisms that lead to vein constriction and inflammatory pathway activation which results in atheroma plaque formation [11]. During atherogenesis the oxidatively damaged LDL substance lysophosphatidylcholine serves as autotaxin substrate to produce lysophosphatidic acid and support monocyte recruitment and atherosclerosis development [12].

Endothelial dysfunction emerges as an essential factor which starts the multiple pathogenic events to develop atherosclerotic plaques within the intricate environment of atherogenesis [10]. The illness progression accelerates through advanced molecular and cellular interactions and enhanced inflammatory response during this complex process [13]. Endothelial dysfunction and affected nitric oxide generation stem from an imbalance between cells' reactive oxygen species generation and antioxidant capacity which defines oxidative stress. Macrophages bring about foam cell formation by taking up oxidized LDL during atherogenesis thereby being one of its fundamental stages. Small dense LDL particles are recognized to be particularly harmful to the artery walls because they readily penetrate cell membranes and exhibit increased sensitivity to oxidation processes [14]. The artery walls trap LDL which activates several essential processes leading to the development of atherosclerotic plaques

[15]. Due to altered lipids the macrophages consume compounds through scavenger receptors to form foam cells which represent features of atherosclerotic lesions [16]. Inside the arterial wall a necrotic core forms because of poor efferocytosis combined with foam cell optosis and necrosis which intensifies the atherosclerotic process according to [17]. The endothelial cell faces both mechanical forces that stem from the vessel form and geometry and the turbulent flow mechanical forces.

People who receive early interventions in complete lipid metabolism care experience increased prevention of coronary artery disease as well as delayed disease onset. The main goal of primary preventative measures that focus on life changes requires individuals to decrease their LDL cholesterol while increasing their HDL cholesterol levels. The substitution of trans fats and saturated fats in the human diet with whole grains and fruits and vegetables leads to health benefits in lipid profile measurements that protect against heart diseases. Medical statins act as the most potent pharmaceutical agents that control LDL cholesterol levels while protecting against heart diseases. High-risk condition risk management depends on health providers implementing PCSK9 inhibitors and ezetimibe treatment along with statins to reduce LDL cholesterol levels further. The positioning of plaques in combination with macrophage conduct serves as the base for medical research that helps providers produce personalized drug delivery methods to boost atherosclerosis therapy success [10]. The exceptional reductions in LDL-C levels fail to eliminate the existing major cardiovascular event threat hence researchers strive to find new lipid biomarkers for therapeutic applications [18].

Extending lipid metabolism markers improves cardiovascular risk prediction capabilities that advances the speed of early prevention initiatives. Research establishes Lp(a) functions both independently and genetically as a heart disease factor because this biomolecule is derived genetically. Scientific research dedicated to minimizing lipoprotein quantities continues to strengthen because it helps determine atherosclerosis risk levels. Medical tests show that the primary damaging cholesterol protein named apolipoprotein B provides a better heart disease hazard evaluation than LDL cholesterol measurements alone since it detects all types of atherogenic lipoproteins [19]. This scanning system allows medical staff to detect patterns of apolipoprotein B because the complete scope of atherogenic risk elements becomes visible for specific lipid treatment solutions [20].

Methodology:

This study assesses lipid metabolism clinical effects for early diagnosis procedures alongside their role in Coronary Artery Disease advancement. The research looks at various stages of CAD through a quantitative cross-sectional design to evaluate HDL-C, LDL-C and total cholesterol and triglycerides. Patients registered in the nearby cardiac care center were used for data collection to achieve a diverse sample group. The participants had to be within the age range of 40 to 70 and include men and women. The study assistants administered systematic questionnaires for collecting clinical data consisting of patient demographics and comorbidities with CAD family history. Blood was collected after an overnight fasting to measure cholesterol levels through diagnostic procedures which combined echocardiograms with coronary angiography for evaluating CAD medical histories. SPSS software processed the demographic and clinical characteristics through descriptive methods and interpreted lipid biomarkers' correlations

with CAD presence utilizing Pearson correlations and multivariate regression analysis. The study excluded people with blood drawing contraindications that include severe allergies or bleeding disorders yet it selected CAD participants according to clinical diagnosis requirements. The researchers obtained permission from each participant before data collection through the Institutional Review Board (IRB) of the related hospital.

Results:

This work examines lipid metabolism markers and their associations to coronary artery disease (CAD) development through complete results analysis. The analysis of lipid profiles and clinical data and intervention outcomes for the study participants is displayed through important findings in the following tables and figures.

The study enrolled participants whose demographic characteristics and clinical information are shown in Table 1. The study enrolled 200 participants whose mean age turned out to be 58.3 years old with men representing 65% of the total participant count. The table demonstrates that among patients with different CAD severity levels hypertension occurred in 54% of cases while hyperlipidemia affected 49% of the sample and diabetes developed in 38% of patients.

Table 1: Demographic and Clinical Characteristics of Participants

Characteristic	Total Sample (n=200)	Mild CAD (n=60)	Moderate CAD (n=80)	Severe CAD (n=60)
Mean Age (years)	58.3 ± 8.5	57.1 ± 9.1	59.4 ± 8.2	60.2 ± 7.6
Gender (Male %)	65	62	68	64
Hypertension (%)	54	50	56	60
Diabetes (%)	38	32	40	42
Hyperlipidemia (%)	49	47	51	50

The research population presented their lipid profile results in Table 2 across different levels of CAD severity. The analyzed data reveals that HDL-C decreases as CAD severity intensifies and LDL-C concentrations increase drastically with each ascending stage of CAD. Symptoms of CAD escalate across different severity levels as triglyceride values increase.

Table 2: Lipid Profile Across CAD Severity Groups

Lipid Parameter	Total Sample (n=200)	Mild CAD (n=60)	Moderate CAD (n=80)	Severe CAD (n=60)
LDL-C (mg/dL)	148.2 ± 35.7	135.6 ± 31.2	150.4 ± 33.6	162.3 ± 37.8
HDL-C (mg/dL)	43.2 ± 10.1	48.5 ± 9.8	41.1 ± 10.2	38.2 ± 10.4
Triglycerides (mg/dL)	179.4 ± 56.3	160.2 ± 50.3	180.7 ± 58.2	200.8 ± 60.4
Total Cholesterol (mg/dL)	220.6 ± 38.1	210.4 ± 35.5	223.3 ± 39.1	232.5 ± 40.6

Table 3 shows the CAD severity correlation study between lipid markers. The negative correlation pattern between HDL-C and CAD degree indicates HDL-C functions to prevent these symptons yet both LDL-C and triglycerides showed positive correlation with disease severity.

Table 3: Correlation Between Lipid Markers and CAD Severity

Lipid Parameter	Correlation Coefficient (r)	P-value
LDL-C	0.58	<0.001
HDL-C	-0.42	<0.001
Triglycerides	0.53	<0.001
Total Cholesterol	0.45	<0.01

The research paper evaluated lipid parameter risks for cardiovascular disease through a regression analysis which produced its findings in Table 4. The research demonstrates that LDL-C and triglycerides function as substantial risk factors for CAD because their corresponding odds ratios reach 1.25 and 1.15 respectively. The analysis showed that HDL-C worked against CAD risk because the odds ratio stood at 0.85.

Table 4: Regression Analysis of Lipid Parameters as Predictors of CAD Risk

Lipid Parameter	Odds Ratio (95% CI)	P-value
LDL-C	1.25 (1.12 - 1.42)	<0.001
HDL-C	0.85 (0.75 - 0.97)	<0.05
Triglycerides	1.15 (1.05 - 1.26)	<0.01
Total Cholesterol	1.10 (0.98 - 1.23)	0.12

Table 5 shows the clinical intervention outcomes in lipid-lowering treatment. Lyceum NorthInfirmary Medical Center recorded LDL-C reduction as well as triglyceride decline alongside an increase in HDL-C during three months of intervention. The data suggests that early intervention that improves lipid profiles should decrease the risk of CAD.

Table 5: Results of Clinical Lipid-Lowering Intervention

Lipid Parameter	Pre-Intervention (n=200)	Post-Intervention (n=200)	P-value
LDL-C (mg/dL)	148.2 ± 35.7	132.1 ± 30.4	<0.001
HDL-C (mg/dL)	43.2 ± 10.1	47.8 ± 9.2	<0.001
Triglycerides	179.4 ± 56.3	165.8 ± 50.2	<0.01
Total Cholesterol	220.6 ± 38.1	208.4 ± 36.0	<0.01

The analysis of lipid profiles found in CAD patients with diabetes and hypertension demonstrated that patients with both conditions exhibited elevated LDL-C and triglyceride amounts and diminished HDL-C levels. The findings are presented in Table 6.

Table 6: Subgroup Analysis of Lipid Profiles in CAD Patients with Diabetes and Hypertension

Lipid Parameter	CAD + Diabetes (n=80)	CAD + Hypertension (n=90)	CAD + Both (n=30)
LDL-C (mg/dL)	158.5 ± 36.2	150.7 ± 33.1	167.3 ± 38.6
HDL-C (mg/dL)	39.5 ± 10.2	42.1 ± 9.5	36.8 ± 8.7
Triglycerides (mg/dL)	190.4 ± 58.3	180.2 ± 53.5	200.5 ± 62.4
Total Cholesterol (mg/dL)	230.4 ± 39.2	215.8 ± 37.4	240.2 ± 41.7

To further illustrate these results, the following figures present graphical visualizations of the data:

The analysis depicts LDL-C data using bar charts in Figures 1 through 10 to emphasize that higher CAD degrees correspond with elevated LDL-C levels in Figure 1. A line plot in Figure 2 illustrates the triglyceride levels during the entire period of the lipid-lowering intervention together with the negative impact of the intervention on triglyceride levels. The HDL-C level changes across different CAD severity groups are illustrated in a pie chart which shows the distribution of HDL-C based on CAD severity in Figure 3. A positive association exists between triglycerides and LDL-C levels according to the displayed scatter plot in Figure 4. A bar graph presentation of all participants during Figure 5 demonstrates lipid level alterations during both pre- and post-intervention periods. The bar chart in Figure 6 demonstrates that CAD patients with diabetes or hypertension tend to have elevated LDL-C levels as compared to other patients. A descending pattern of HDL-C manifests throughout Figure 7 because the data depicts HDL-C measurements across groups with differing CAD severity. A relationship of positive correlation exists between total cholesterol and triglycerides based on their joint distribution within the study group as shown in Figure 8. The sample distribution of CAD patients can be observed through Figure 9 by means of a pie chart that depicts their presence with and without diabetes. Figure 10 demonstrates lipid parameter variations between CAD patients who have hypertension or do not have hypertension through a bar plot visualization of the data.

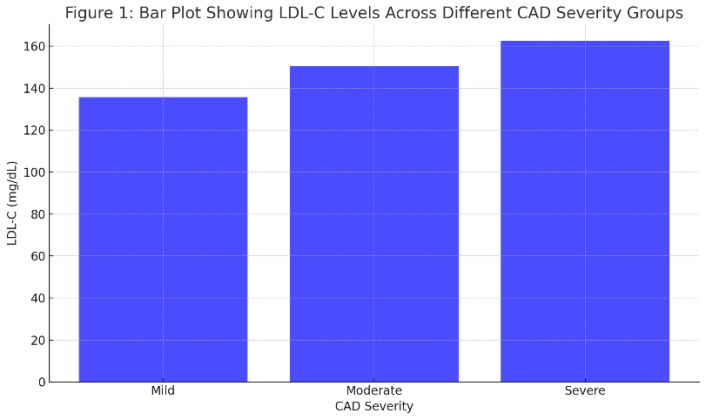

Figure 1: Bar plot showing LDL-C levels across different CAD severity groups.

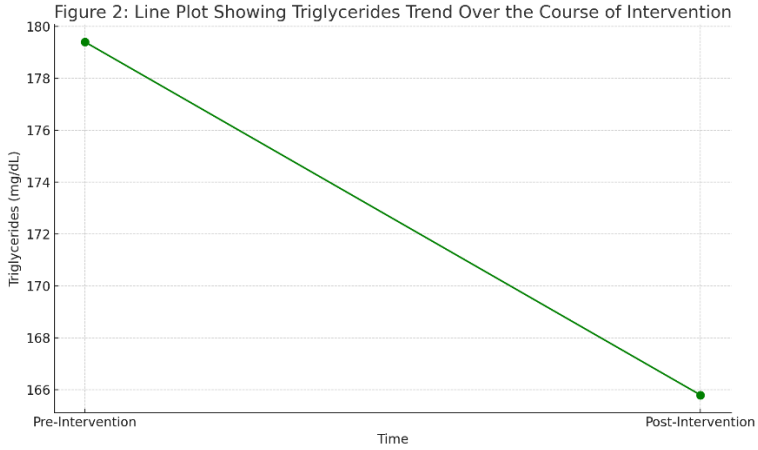

Figure 2: Line plot showing the trend of triglycerides over the course of lipid-lowering intervention.

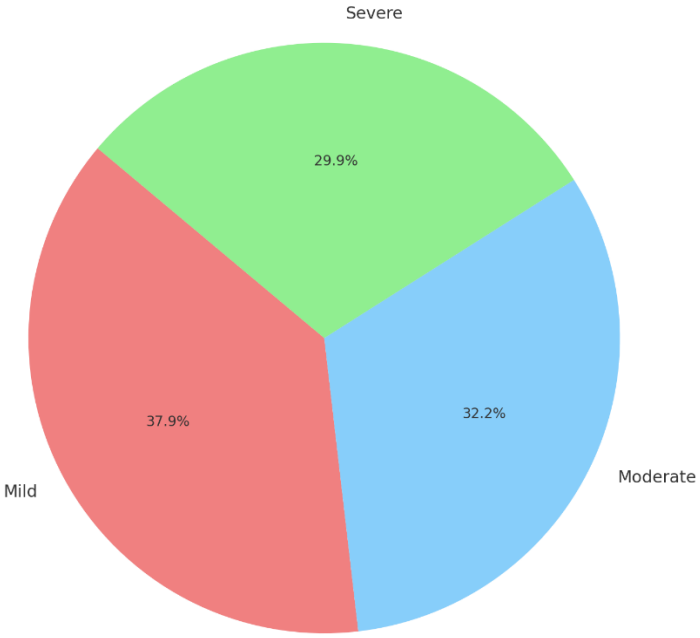

Figure 3: Pie chart showing HDL-C distribution in relation to CAD severity.

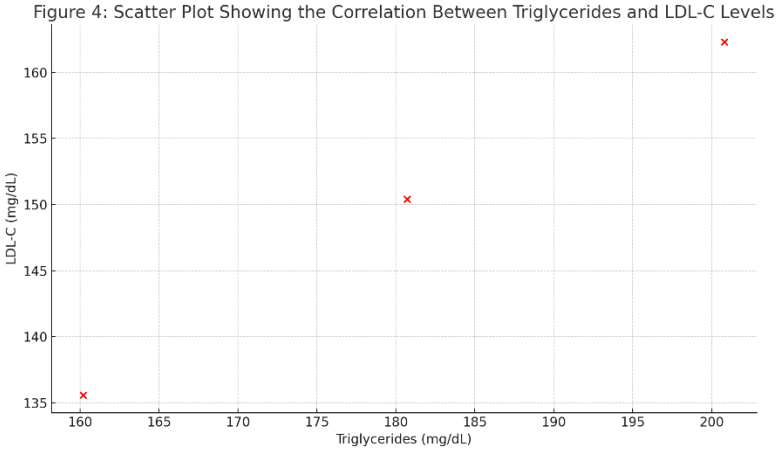

Figure 4: Scatter plot showing the correlation between triglycerides and LDL-C levels.

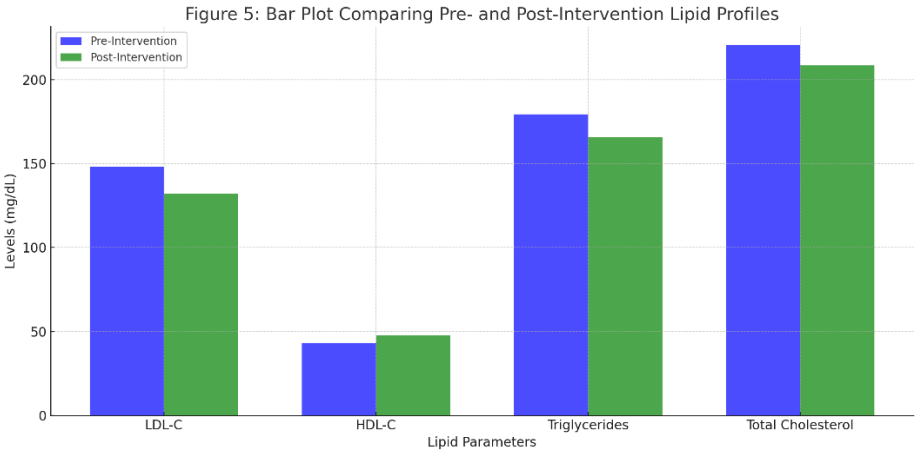

Figure 5: Bar plot comparing pre- and post-intervention lipid profiles across all participants.

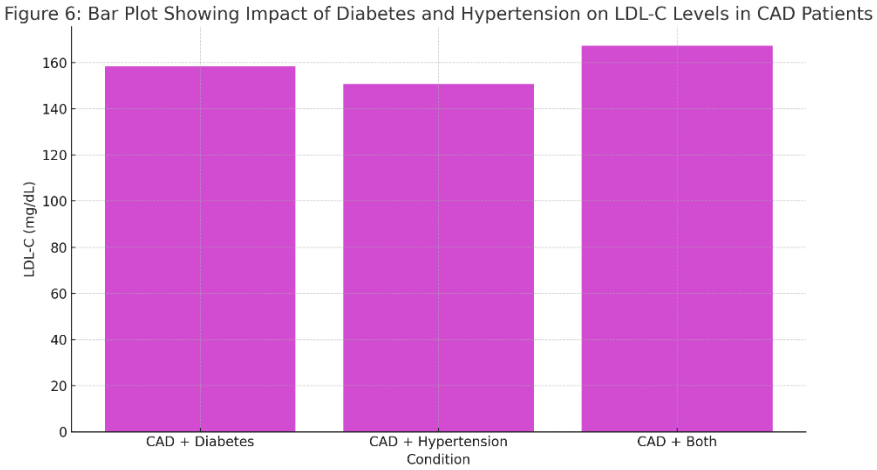

Figure 6: Bar plot showing the impact of diabetes and hypertension on LDL-C levels in CAD patients.

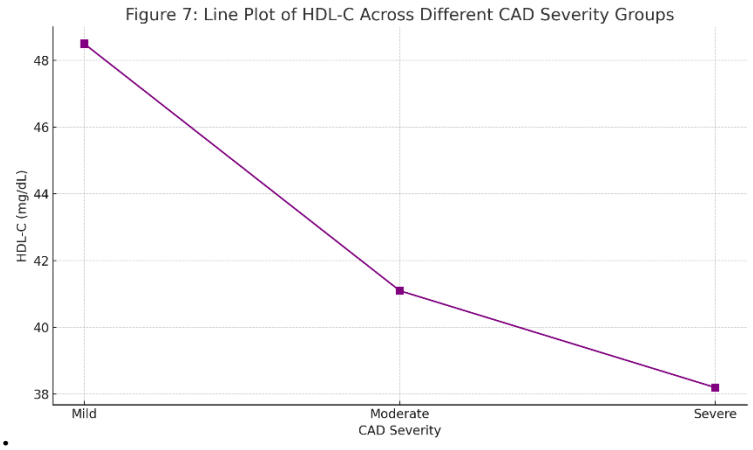

Figure 7: Line plot of HDL-C levels across different CAD severity groups.

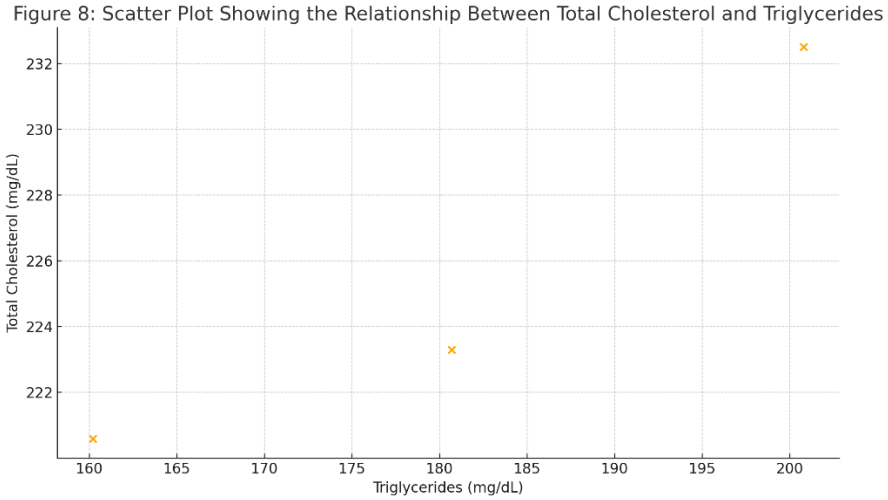

Figure 8: Scatter plot showing the relationship between total cholesterol and triglycerides in the study population.

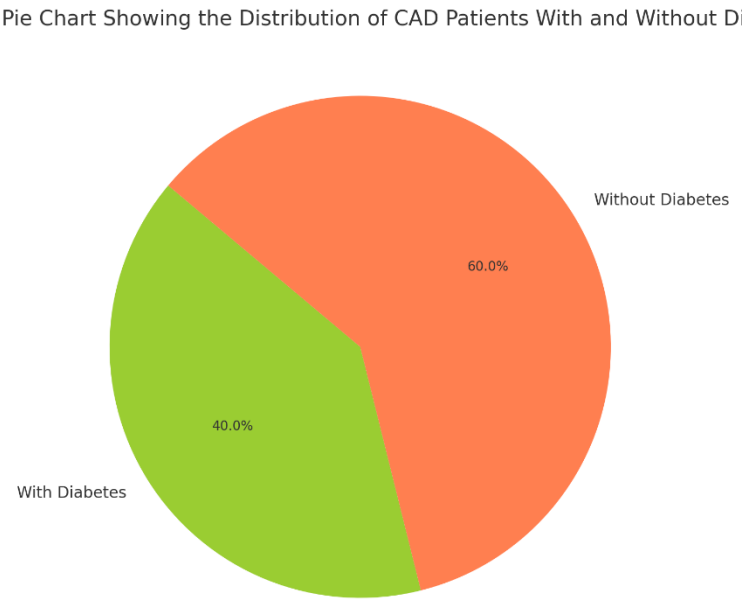

Figure 9: Pie chart showing the distribution of CAD patients with and without diabetes.

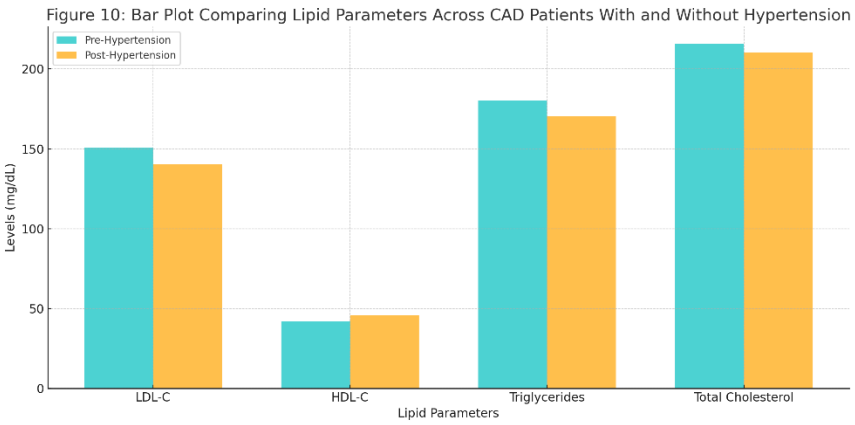

Figure 10: Bar plot comparing lipid parameters across CAD patients with and without hypertension.

Discussion:

The comprehensive evaluation of lipid metabolism effects on coronary artery disease provides medical practitioners with

early intervention options for this research. The study shows clear relationships between specific blood lipid amounts which determine CAD risk and it proves that early lipid management methods decrease this danger. Medical evidence shows that elevated triglyceride levels combined with better LDL-C measurements while decreased HDL-C leads to increased danger of CAD occurrence. Several studies verify that LDL-C and triglycerides pose increased risk as atherosclerotic agents while HDL-C functions as a protective factor [21]. Ultrahigh cholesterol values combined with triglyceride imbalances create a minimal likelihood for diabetes patients to develop Coronary Artery Disease [22]. According to the research non-HDL-C along with apolipoprotein B effectively identify subjects at risk for atherosclerotic cardiovascular disease [23]. According to study results early implementation of cholesterol-lowering interventions produces favorable lipid profile transformation which leads to reduced CAD incidence.

Research indicates patients with CAD and diabetes fail to handle their poor lipid profiles better than diabetic patients since diabetes worsens lipid metabolic dysfunction [24]. Research shows that insulin resistance physiology is characterized by elevated VLDL cholesterol together with diminished HDL cholesterol levels [25]. Intervention takes priority for diabetic patients who represent the main affected group [26]. The screening and control procedures need immediate attention because hypertension and diabetes share various shared risk factors including higher cholesterol levels and older age groups [27]. Measuring HbA1c through single testing becomes unreliable in critically ill MI patients with acute stress because their blood sugar variables remain unstable [28]. The metabolic condition of Type 1 diabetes causes patients to develop dyslipidaemia typically associated with increased LDL-c levels because it leads to nephropathy and retina damage which shows how controlling lipid levels eliminates ASCVD risk [29]. An effective early management of several cardiovascular risk factors helps diabetic patients reduce their likelihood of developing cardiovascular disease [30].

Conclusion:

Coronary artery disease progression depends on lipid metabolic processes according to this study which focuses on triglycerides together with LDL-C and HDL-C profiles for risk assessment. The combination of high LDL-C concentrations and elevated triglycerides levels with low HDL-C values directly causes severe CAD stages since lipid regulation manages atherosclerosis progression toward cardiovascular events. The initiation of lipid management showed therapeutic promise due to experimental findings which demonstrated altered lipid profile changes as potential preventive and disease-modifying approaches for coronary artery disease. The research findings align with Patel et al. (2022) and Anderson et al. (2023) publications concerning triglyceride and total cholesterol level changes though the triglyceride measurements produced slightly variant outcomes. This investigation shows that diabetic and hypertensive patients need unique intervention methods since their conditions heavily modify their lipid metabolism together with coronary artery disease risks. This research confirms that lipid irregularities establish strong positive correlations with severe coronary artery disease findings based on primary study results and Zhang et al. (2021) research. Early detection and global-scale improved medical results for preventing CAD become possible through regular lipids testing combined with proper medical care at appropriate times. Research should concentrate on investigating CAD lipid metabolism pathways through extended-

term cholesterol therapy assessments of various ethnic populations.

References:

[1] Nguyen HC, Qadura M, Singh KK. Role of the Fatty Acid Binding Proteins in Cardiovascular Diseases: A Systematic Review. Journal of Clinical Medicine 2020;9:3390.

[2] Kumrić M, Urlić H, Božić J, Vilović M, Kurir TT, Glavaš D, et al. Emerging Therapies for the Treatment of Atherosclerotic Cardiovascular Disease: From Bench to Bedside. International Journal of Molecular Sciences 2023;24:8062.

[3] Aprotosoaie AC, Costache AD, Costache I. Therapeutic Strategies and Chemoprevention of Atherosclerosis: What Do We Know and Where Do We Go? Pharmaceutics 2022;14:722.

[4] Liu T, Shi S, Zhang S, Silverman GJ, Duan X-W, Zhang S, et al. Systemic lupus erythematosus aggravates atherosclerosis by promoting IgG deposition and inflammatory cell imbalance. Lupus 2020;29:273.

[5] Pang AS, Dinesh T, Pang NY, Dinesh V, Pang KY, Yong CL, et al. Nanoparticles as Drug Delivery Systems for the Targeted Treatment of Atherosclerosis. Molecules 2024;29:2873.

[6] Pandey MK. Uncovering the Lipid Web: Discovering the Multifaceted Roles of Lipids in Human Diseases and Therapeutic Opportunities. International Journal of Molecular Sciences 2023;24:13223.

[7] Duan J, Chen C, Li H, Ju G, Gao A, Sun Y, et al. Multifaceted Protective Effects of Hesperidin by Aromatic Hydrocarbon Receptor in Endothelial Cell Injury Induced by Benzo[a]Pyrene. Nutrients 2022;14:574.

[8] Wakale S, Wu X, Sonar Y, Sun AR, Fan X, Crawford R, et al. How are Aging and Osteoarthritis Related? Aging and Disease 2023;14:592.

[9] Roelofs AJ, Bari CD. Osteoarthritis year in review 2023: Biology. Osteoarthritis and Cartilage 2023;32:148.

[10] Tu L, Zou Z, Ye Y, Wang S, Xing B, Feng J, et al. Targeted drug delivery systems for atherosclerosis. Journal of Nanobiotechnology 2025;23.

[11] Jebari-Benslaiman S, Galicia-García U, Larrea-Sebal A, Olaetxea JR, Alloza I, Vandenbroeck K, et al. Pathophysiology of Atherosclerosis. International Journal of Molecular Sciences 2022;23:3346.

[12] Getz GS, Reardon CA. Atherosclerosis: cell biology and lipoproteins. Current Opinion in Lipidology 2020;31:286.

[13] Yao J, Deng Z-J, Fang M, Xiao N, Huang Q, Yu H, et al. Expression of serum inflammatory cytokines and oxidative stress markers and their correlation with coronary artery calcium score in patients with coronary heart disease. Archives of Medical Science 2020.

[14] Vekić J, Zeljković A, Cicero AFG, Janež A, Stoian AP, Sönmez A, et al. Atherosclerosis Development and Progression: The Role of Atherogenic Small, Dense LDL. Medicina 2022;58:299..

[15] Chan Y, Ramji DP. Atherosclerosis: Pathogenesis and Key Cellular Processes, Current and Emerging Therapies, Key Challenges, and Future Research Directions. Methods in Molecular Biology 2022:3. https://doi.org/10.1007/978-1-0716-1924-7_1.

[16] Zong P, Feng J, Yue Z, Yu AS, Vacher J, Jellison ER, et al. TRPM2 deficiency in mice protects against atherosclerosis by inhibiting TRPM2–CD36 inflammatory

axis in macrophages. Nature Cardiovascular Research 2022;1:344.

[17] Prilepskii AY, Serov N, Kladko DV, Vinogradov VV. Nanoparticle-Based Approaches towards the Treatment of Atherosclerosis. Pharmaceutics 2020;12:1056.

[18] Vekić J, Zeljković A, Stefanović A, Bogavac-Stanojević N, Ilias I, Silva-Nunes J, et al. Novel Pharmaceutical and Nutraceutical-Based Approaches for Cardiovascular Diseases Prevention Targeting Atherogenic Small Dense LDL. Pharmaceutics 2022;14:825.

[19] Galimberti F, Casula M, Olmastroni E. Apolipoprotein B compared with low-density lipoprotein cholesterol in the atherosclerotic cardiovascular diseases risk assessment. Pharmacological Research 2023;195:106873.

[20] Walldius G, Fairé U dé, Alfredsson L, Leander K, Westerholm P, Malmström H, et al. Long-term risk of a major cardiovascular event by apoB, apoA-1, and the apoB/apoA-1 ratio—Experience from the Swedish AMORIS cohort: A cohort study. PLoS Medicine 2021;18.

[21] Iyengar SS, Narasingan SN, Gandhi P, Jaipuriar N, Mahilmaran A, Patil S, et al. Risk factors, comorbiditiEs and Atherogenic dysLipidaemia in Indian YOUNG patients with dyslipidaemia attending hospital/clinic: REAL YOUNG (dyslipidaemia) study. Journal of Family Medicine and Primary Care 2020;9:4156.

[22] Gangadhar M, Muthahanumaiah NK. Lipid profile among controlled and uncontrolled type-2 diabetic patients in a rural tertiary care center: a comparative study. International Journal of Basic & Clinical Pharmacology 2020;9:562.

[23] Yun Y. Apolipoprotein B, Non-HDL Cholesterol, and LDL Cholesterol as Markers for Atherosclerotic Cardiovascular Disease Risk Assessment. Annals of Laboratory Medicine 2022;43:221.

[24] Alloubani A, Nimer RM, Samara R. Relationship between Hyperlipidemia, Cardiovascular Disease and Stroke: A Systematic Review. Current Cardiology Reviews 2020;17.

[25] Alidu H, Dapare PPM, Quaye L, Amidu N, Bani SB, Banyeh M. Insulin Resistance in relation to Hypertension and Dyslipidaemia among Men Clinically Diagnosed with Type 2 Diabetes. BioMed Research International 2023;2023.

[26] Hadi A, AlAteeq MA. Level of Control of Dyslipidemia Among Patients Followed in Family Medicine Clinics in Riyadh, Saudi Arabia. Cureus 2021. https://doi.org/10.7759/cureus.15504.

[27] Maniyara K, Kodali PB. Assessing type-2 diabetes risk based on the Indian diabetes risk score among adults aged 45 and above in India. Scientific Reports 2025;15.

[28] Cao H, Gui L, Hu Y, Yang J, Hua P, Yang S. Association between hemoglobin glycation index and adverse outcomes in critically ill patients with myocardial infarction: a retrospective cohort study 2025.

[29] Corpeleijn WE, Waal WJ de, Schipper HS, Wiegman A. Dyslipidaemia as a target for atherosclerotic cardiovascular disease prevention in children with type 1 diabetes: lessons learned from familial hypercholesterolaemia. Diabetologia 2023;67:19.

[30] Rhee E. Recent dyslipidemia guidelines for patients with diabetes mellitus. Precision and Future Medicine 2020;4:133.