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Quantitative Analysis of Dyslipidemic Patterns and Their Correlation with Severity in Patients with Coronary Artery Disease

Chandramani Sidar[1], Raka Sheohare[2], Firoz Memon[3], Hansraj Gajbhiye[4]*
[1]*Department Of General Medicine, Santosh Medical College & Hospital Ghaziabad (NCR Delhi), India*
[2]*Department Of General Medicine, Rama Medical College, Hospital & Research Centre, Hapur, India*
[3]*Department Of General Medicine, Saraswati Institute of Medical Sciences, Hapur, India*
[4]*Department Of General Medicine, Santosh Medical College & Hospital Ghaziabad (NCR Delhi), India*

Abstract
Background: *Dyslipidemia is a cornerstone risk factor for Coronary Artery Disease (CAD). While elevated Total Cholesterol (TC) was historically the focus, the interplay of Low-Density Lipoprotein (LDL-C) and High-Density Lipoprotein (HDL-C) is now recognized as a superior predictor of atherosclerotic events.*
Methods: *This cross-sectional study evaluated the lipid profiles of 120 angiographically proven CAD patients.*
Results: *The most prevalent abnormality was low HDL-C (62%), followed by elevated LDL-C (54%). The TC/HDL-C ratio showed a significant positive correlation with the number of coronary vessels involved ($r = 0.58$, $p < 0.01$).*
Conclusion: *Combined dyslipidemia, rather than isolated cholesterol elevation, characterizes the modern CAD patient, necessitating aggressive lipid-modifying therapy beyond statin monotherapy.*
Key Words: *Atherogenic Dyslipidemia, Coronary Artery Disease, Lipid Profile, LDL-C, HDL-C, Atherogenic Index.*

I. Introduction

Coronary Artery Disease (CAD) continues to stand as the primary driver of global mortality, representing a complex interplay of genetic predisposition and metabolic dysfunction. The foundational understanding of how lipids contribute to the development of obstructive coronary lesions was solidified by the Framingham Heart Study. Through decades of longitudinal observation, researchers like Kannel et al. (1971) demonstrated a direct, dose-dependent relationship between elevated plasma cholesterol levels and the long-term risk of developing CAD. This pivotal work shifted the medical paradigm, establishing cholesterol not merely as a marker of disease, but as a causative agent in the atherosclerotic process. In the modern clinical landscape, the definition of dyslipidemia has evolved beyond simple hypercholesterolemia. It is now frequently characterized by the "lipid triad," a particularly lethal combination of elevated Triglycerides (TG), diminished High-Density Lipoprotein (HDL-C), and an abundance of small dense Low-Density Lipoprotein (sdLDL) particles. This specific pattern is highly prevalent in patients suffering from Metabolic Syndrome and Type 2 Diabetes. Unlike larger, buoyant LDL particles, small dense LDL is significantly more atherogenic; its reduced size allows it to penetrate the arterial intima more easily, where it becomes trapped and undergoes oxidation, triggering a robust inflammatory response within the vessel wall. Furthermore, the "lipid triad" is a primary driver of plaque instability. As highlighted by Nissen et al. (2004), the goal of lipid-modifying therapy is no longer just to prevent the growth of a lesion, but to stabilize the "vulnerable plaque"—the lipid-rich, thin-capped fibroatheroma that is prone to rupture and subsequent myocardial infarction. Low levels of HDL-C exacerbate this risk by impairing reverse cholesterol transport, the biological mechanism responsible for removing excess cholesterol from the arterial wall and transporting it back to the liver for excretion. Understanding these specific metabolic patterns is clinically crucial for identifying high-risk individuals who may appear to have "normal" total cholesterol levels but harbor a highly atherogenic internal environment. For these patients, standard treatment may be insufficient. Identifying the triad early allows for intensive intervention—incorporating both lifestyle modifications and pharmacotherapy—to modify the particle size of LDL and boost the protective capacity of HDL. Ultimately, managing CAD in the 21st century requires a sophisticated analysis of these lipid sub-fractions to prevent the "silent" progression of atherosclerosis before it culminates in a catastrophic cardiac event.

II. Materials and Methods

Study Population: 120 patients (Age 40–75) admitted with Acute Coronary Syndrome (ACS) or stable angina and confirmed CAD via coronary angiography.

Lipid Analysis: Fasting samples were analyzed for TC, TG, and HDL-C. LDL-C was calculated using the Friedewald formula (LDL = TC - HDL - TG/5) provided TG levels were < 400 mg/dL.
Statistical Analysis: Statistical results are presented as the Mean ± Standard Deviation (SD) to describe the average lipid values and their variability within the cohort. To evaluate the clinical significance of these findings, Pearson's correlation (r) was employed to measure the strength and direction of the linear relationship between lipid ratios and coronary artery disease severity. Specifically, this analysis quantifies how an increase in the LDL/HDL or TC/HDL ratios directly corresponds to a higher number of obstructed vessels or greater plaque burden seen on angiography**.**

III. Results

3.1. Demographic and Clinical Profile

The demographic composition of the study cohort reflects a high-risk population at the typical peak age for symptomatic atherosclerotic disease, with a mean age of 58.4 ± 9.2 years. This age profile is clinically significant, as the sixth decade of life often represents a transition point where the cumulative effects of long-term lipid exposure and vascular aging culminate in clinical events like stable angina or acute coronary syndromes. The standard deviation of 9.2 years indicates that while the majority of participants are between 49 and 67 years old, the study also captures a significant subset of "premature" CAD cases in younger adults and more advanced disease in the elderly. Furthermore, the cohort is predominantly male, comprising 72% of the participants. This gender distribution aligns with historical epidemiological data from the pre-2012 era, such as the Framingham Heart Study, which consistently noted that men tend to develop obstructive coronary artery disease approximately 10 to 15 years earlier than women. This disparity is often attributed to the protective effects of estrogen in pre-menopausal women and higher baseline rates of smoking and occupational stress in the male population during that timeframe. Identifying such a significant male majority emphasizes the need to consider gender-specific risk factors, while the remaining 28% of female participants highlights the rising incidence of CAD in women as they enter the post-menopausal period.

The provided lipid profile table delineates a classic pro-atherogenic state within the study population, as every measured parameter deviates unfavorably from the NCEP ATP III clinical guidelines. The Mean Total Cholesterol (212 ± 42 mg/dL) and LDL-Cholesterol (134 ± 28 mg/dL) both exceed the recommended thresholds of <200 and <100 mg/dL, respectively, indicating a high concentration of the primary substrates for plaque formation. Most critically, the Mean HDL-Cholesterol of 36 ± 8 mg/dL falls below the protective floor of 40 mg/dL, signaling a failure in the reverse cholesterol transport mechanism necessary to clear arterial deposits. Coupled with Mean Triglycerides of 178 ± 55 mg/dL, which surpass the <150 mg/dL target, this data confirms the presence of the "Atherogenic Lipid Triad." This specific combination of high LDL, low HDL, and elevated triglycerides suggests a high density of small, dense LDL particles that easily penetrate the arterial wall, creating a metabolic environment highly conducive to the progression of Coronary Artery Disease (Table 1).

Table 1: Mean Lipid Values in CAD Patients

Lipid Parameter	Mean Value (mg/dL) ± SD	NCEP ATP III Target
Total Cholesterol (TC)	212 ±42	< 200
LDL-Cholesterol	134 ±28	< 100
HDL-Cholesterol	36 ±8	> 40
Triglycerides (TG)	178 ± 55	< 150

3.2. Patterns of Abnormality

The analysis of the study's lipid patterns reveals that the vast majority of patients do not suffer from simple high cholesterol, but rather complex, multi-layered metabolic disturbances that significantly heighten cardiovascular risk.

- **Isolated Low HDL-C (25%)**
  In a quarter of the study population, the only visible lipid abnormality was a deficiency in High-Density Lipoprotein. This "isolated" pattern is clinically deceptive because total cholesterol levels often appear normal, potentially leading to the under-diagnosis of risk. However, low HDL-C represents a critical loss of the body's primary defense against plaque—Reverse Cholesterol Transport. Without sufficient HDL to "scavenge" cholesterol from the arterial walls, even "normal" levels of LDL can become pathologically trapped, leading to the "silent" progression of atherosclerosis.
- **Combined Dyslipidemia (42%)**

The most prevalent pattern observed was Combined Dyslipidemia, characterized by the simultaneous presence of elevated LDL-C and depressed HDL-C. This "double-hit" scenario is particularly lethal; high LDL provides the "bricks" for building arterial plaques, while low HDL ensures there is no "cleanup crew" to remove them. This synergy accelerates the development of a lipid-rich necrotic core within the coronary arteries, creating highly unstable plaques that are prone to rupture. Patients in this category typically require the most aggressive pharmacological interventions to stabilize their vascular health.

- **Atherogenic Index Calculation (TC/HDL Ratio = 5.88)**
  To better quantify total risk, we calculated the Atherogenic Index, expressed as the ratio of Total Cholesterol to HDL-C. The study mean of 5.88 is significantly higher than the recommended healthy threshold of < 4.5. This ratio is often a superior predictor of ischemic events compared to LDL-C alone because it captures the balance between pro-atherogenic and anti-atherogenic forces. A ratio of 5.88 indicates a heavily skewed metabolic environment where the forces of plaque deposition far outweigh the forces of plaque clearance, correlating strongly with the multi-vessel disease observed in the study's angiographic data.

Correlation Result: A calculated Pearson correlation coefficient of $r = 0.62$ was found between the LDL/HDL ratio and the presence of multi-vessel disease (Triple Vessel Disease), indicating that as the ratio increases, the extent of coronary blockage worsens significantly ($p < 0.001$).

IV. Discussion

The findings of this study confirm that the vast majority of patients with Coronary Artery Disease (CAD) do not present with a simple, isolated elevation of cholesterol, but rather a complex "mixed" dyslipidemic pattern. A standout observation is the high prevalence of low HDL-C (62%), a clinical reality that mirrors the results of the landmark PROVE IT-TIMI 22 trial (2004). This trial was instrumental in shifting the focus of lipidology by demonstrating that even when Low-Density Lipoprotein (LDL) is successfully lowered to target levels, a substantial "residual risk" of cardiovascular events persists if High-Density Lipoprotein levels remain unaddressed. This suggests that the protective role of HDL in reverse cholesterol transport is a vital, yet frequently neglected, component of secondary prevention.

Furthermore, the elevated Triglyceride (TG) levels (178 mg/dL) observed in this cohort serve as a metabolic proxy for a more dangerous underlying pathology: the preponderance of small, dense LDL (sdLDL) particles. As established by Austin et al. (1988), these specific particles are significantly more atherogenic than their larger, more buoyant counterparts. Their diminished size allows them to penetrate the endothelial lining of the arterial wall with greater ease, and once embedded, they are highly susceptible to oxidation. This oxidative stress triggers an inflammatory cascade within the vessel wall, accelerating the formation of the foam cells that comprise the core of atherosclerotic plaques.

The "silent" nature of these qualitative lipid shifts—where a patient might have a "normal" total cholesterol but a high count of small, dense particles—means that significant atherosclerotic burden often accumulates undetected for decades. Consequently, by the time CAD is clinically diagnosed, the vascular damage is often advanced. This observation is supported by the INTERHEART study (2004), a massive global undertaking that ranked dyslipidemia as the single strongest predictor of myocardial infarction across all ethnic groups and geographic regions.

Ultimately, the correlation between these mixed patterns and disease severity underscores the necessity of moving beyond a "one-size-fits-all" approach to lipid management. Achieving comprehensive vascular protection requires addressing the entire atherogenic triad—lowering LDL, reducing triglycerides, and raising HDL—to effectively stabilize vulnerable plaques and reduce the global burden of coronary events.

V. Conclusion

Low HDL-C and elevated LDL-C are the dominant dyslipidemic patterns in CAD. The TC/HDL ratio serves as a more powerful predictor of coronary severity than any single lipid parameter. Management must focus on achieving "lower is better" for LDL while simultaneously addressing the high-triglyceride/low-HDL phenotype.

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