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CORRELATION OF HIGH LDL WITH ANGIOGRAPHY FINDINGS IN PATIENTS WITH UNDERLYING CORONARY ARTERY DISEASE

Dr Ayesha Shahid Butt[*1], Maj. Gen. (R) Dr. Sohail Aziz[2], Dr Zoha Ijaz[3], Dr Sana Hassan[4], Dr Wajeeh Ul Hassan[5], Dr Taj Mahal[6]

*[*1]Postgraduate Resident FCPS – Cardiology, Department of Cardiology, Fauji Foundation Hospital Rawalpindi*
[2]HI(M), BSc, MBBS, FCPS, FRCP (Edin), MRCP (UK), FSCAI(USA), M. Phil. Cardiology, Head Department of Cardiology, Fauji Foundation Hospital Rawalpindi,
[3,4,5,6]Postgraduate Resident FCPS – Cardiology, Department of Cardiology, Fauji Foundation Hospital Rawalpindi

[*1]buttayesha80@gmail.com, [2]soaziz@hotmail.com, [3]zohaijaz@gmail.com, [4]sana582468@gmail.com, [5]wajeehassan31@hotmail.com, [6]taj.mehdi94@gmail.com

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Corresponding Author: *
Dr Ayesha Shahid Butt

Abstract

Introduction: *Coronary artery disease (CAD) remains a leading cause of global morbidity and mortality, with elevated low-density lipoprotein (LDL) cholesterol being a well-established risk factor. However, the relationship between LDL levels and angiographic severity across different patient subgroups remains incompletely understood. This study aimed to determine the correlation of high LDL with angiography findings in patients with underlying coronary artery disease.*

Methodology: *In this cross-sectional study, 462 patients with confirmed CAD underwent coronary angiography and LDL measurement. Angiographic severity was categorized as single-, double-, or triple-vessel disease. Spearman's correlation analysis evaluated LDL-angiography associations overall and across subgroups including gender, age, BMI, comorbidities, and CAD subtypes.*

Results: *The overall population showed a significant positive correlation ($r=0.579$, $p<0.001$). Stronger associations emerged in males ($r=0.629$) versus females ($r=0.512$), smokers ($r=0.689$), and diabetics ($r=0.614$). Normal-weight patients (BMI<25) demonstrated higher correlations ($r=0.605$) than overweight/obese patients ($r=0.561$). Late presenters (≥12 hours post-symptom onset) had stronger correlations ($r=0.607$) than early presenters ($r=0.547$). Among CAD subtypes, stable angina ($r=0.596$) and NSTEMI ($r=0.595$) showed marginally stronger associations than STEMI ($r=0.533$).*

Conclusion: *Our findings highlight the critical role of LDL cholesterol in the severity of CAD, while demonstrating notable differences among patient subgroups. The findings support tailored lipid management approaches, especially for high-risk populations such as smokers and diabetics, who exhibited the most significant connections with LDL-atherosclerosis*

INTRODUCTION

Coronary heart disease (CHD), also known as CAD, is a heart disease in which atherosclerotic plaques form in the lumen of the coronary arteries, causing impaired blood flow and oxygen supply to the heart muscle.[i] Cardiovascular disease is one of the leading causes of death worldwide. According to the World

Health Organization (WHO), an estimated 17.9 million people will die from cardiovascular disease in 2021, accounting for 32% of all deaths worldwide.[ii] Research from the Global Burden of Disease states that worldwide, people with CHD are 126 million individuals (1,655 per 100,000), about 1.72% of the world's population. There were 9 million deaths due to CHD worldwide. Men are more at risk than women. Incidence usually begins in the fourth decade of life and increases with age. The global prevalence of CHD is increasing. It is estimated that the prevalence rate will increase to 1,845 by 2030.[iii]

Dyslipidemia, especially elevated low-density lipoprotein cholesterol (LDL-C), is one of the major risk factors for cardiovascular disease. Thus, LDL-C has always been the most important target in the treatment of patients with coronary artery disease (CAD).[iv] Previous studies have shown that for every 1 mmol/L reduction in LDL-C, coronary events can be decreased by 23%. Even if LDL-C concentration is maintained at 1.99 mmol/L by intensive therapy with high doses of statin drugs, the incidence of cardiovascular events is lowered from 10.9 to 8.7%.[v] This means that despite implementing standard lipid-lowering therapy and controlling risk factors such as blood pressure and glycaemia, patients may still develop cardiovascular events, which is also known as residual cardiovascular risk.[vi] Hence, applying LDL-C to assess the risk of CAD still faces limitations. It is necessary to look for new blood lipid parameter to better predict cardiovascular diseases.[vii]

The rationale of this study is to highlight the extent of the problem which will help us in planning the preventive and management strategies. Results of this study will pave the way for further research in our local population. Recognition of high LDL levels is important, due to the imminent danger of severe coronary artery disease. The correlation of high LDL and severe CAD is suggested by several studies. But locally studies are lacking in this regard. I am expecting different results locally as our living styles and eating habits are different from western population. The results of this study will not only provide us with the local stats of the problem but will also help the clinicians for early screening of high LDL in these particular patients. Early identification of the high LDL in patients with coronary artery disease may be beneficial for patients' risk stratification.

METHODOLOGY

This descriptive cross-sectional study was conducted at the Department of Cardiology, Fauji Foundation Hospital, Rawalpindi, over a period of six months from January 2025 to June 2025. The study population comprised 100 consecutively enrolled patients aged 18-70 years with confirmed coronary artery disease (CAD), while excluding those with advanced heart failure, prior coronary bypass surgery, pregnancy, contrast allergy, or chronic kidney disease (serum creatinine >2 mg/dL). Diagnostic criteria for CAD subtypes were strictly applied: unstable angina required typical chest pain >30 minutes without enzyme elevation; NSTEMI was defined by ischemic symptoms with ST-depression/T-wave inversion and elevated troponins; while STEMI diagnosis followed standard ECG criteria incorporating gender-specific ST-elevation thresholds. Coronary angiography findings were categorized by vessel involvement (SVD/DVD/TVD) using ≥50% stenosis cutoff, with LAD lesions further stratified anatomically. Comprehensive clinical profiling included documentation of hypertension (controlled on treatment), diabetes (FBS <110 mg/dL), smoking history (≥10 cigarettes/day for >2 years), and family CAD history in first-degree relatives. Anthropometric measurements followed standardized protocols, with BMI calculated from weight (digital scale) and height (stadiometer). Following ethical approval and informed consent, fasting venous blood samples were processed for LDL quantification using standardized laboratory methods. All angiographic procedures employed femoral access with iohexol contrast, interpreted by a blinded interventional cardiologist to ensure objective assessment of disease severity. Statistical analysis utilized SPSS v25, employing descriptive statistics for baseline characteristics and Spearman's correlation to evaluate LDL-angiography associations. Rigorous subgroup stratification accounted for potential confounders including demographic factors, comorbidities and CAD presentation patterns. The 0.05 significance threshold was applied throughout the analytical process.

RESULTS

The study population consisted of 462 individuals with coronary artery disease, exhibiting a mean age of 48.6 years (SD ±9.5), indicating a primarily middle-aged group. The statistics demonstrated a moderate male predominance relative to female patients. Analysis of cardiovascular risk factors revealed hypertension as the most common comorbidity, closely followed by a familial history of ischemic heart disease. Metabolic diseases were prevalent, with about one-third of individuals being diabetic, and a smoking history was noted in 37.2% of cases. The clinical presentation patterns revealed a rather uniform distribution among acute coronary syndrome subtypes, with STEMI patients forming the biggest category, followed by NSTEMI, while stable angina cases comprised the remainder. The equitable distribution among CAD subtypes establishes a solid basis for comparative assessments of disease severity and progression trends. Detailed analysis of clinical and demographic profile of the patient is presented in figure 1, table 1 and table 2.

The data analysis demonstrated a statistically significant moderate association between high LDL levels and angiographic observations ($r=0.579$, $p<0.001$). Male patients had a more robust correlation ($r=0.629$) than females ($r=0.512$), indicating gender-specific disparities in the atherogenic potential of LDL. Age stratification revealed consistent correlations in both younger (<45 years, $r=0.564$) and older (≥45 years, $r=0.585$) patients. Diabetics had a notably stronger connection ($r=0.614$) than non-diabetics ($r=0.562$), underscoring the significance of LDL in diabetic vasculopathy. Among CAD subtypes, stable angina ($r=0.596$) and NSTEMI ($r=0.595$) had greater correlations than STEMI ($r=0.533$). Smokers exhibited the most robust correlation overall ($r=0.689$), whereas non-smokers displayed a more moderate association ($r=0.516$). Patients having a familial history of CAD exhibited greater correlations ($r=0.604$) compared to those without such a history ($r=0.559$). Detailed analysis of stratification is illustrated in table 3.

Table 1: Details of clinical and demographic quantitative variables n=462

Variable	Minimum	Maximum	Mean	Std. Deviation
Age (years)	48.63	9.48	25.00	70.00
Duration of symptoms (hours)	12.22	4.05	1.00	24.00
Height (cm)	168.09	10.07	140.00	201.00
Weight (kg)	75.36	15.30	35.20	130.40
BMI (kg/m²)	26.97	6.37	10.40	53.20
LDL levels (mg/dL)	139.19	25.04	70.00	226.20

Table 2: Details of clinical and demographic qualitative variables n=100

Variable	Category	Frequency	Percent
Gender	Male	259	56.1%
	Female	203	43.9%
Family History of IHD	Yes	194	42.0%
	No	268	58.0%
Hypertension	Yes	199	43.1%
	No	263	56.9%
Diabetes Mellitus	Yes	147	31.8%
	No	315	68.2%
Smoking	Yes	172	37.2%
	No	290	62.8%
Type of CAD	STEMI	171	37.0%
	NSTEMI	154	33.3%
	Stable Angina	137	29.7%

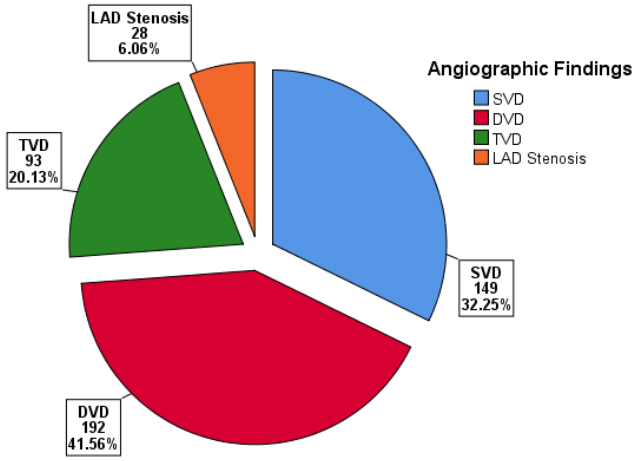

Figure 1: Distribution of patients on the basis of angiographic findings n=462

Table 3: Correlation analysis between LDL levels and angiography findings (Overall population and stratification for various clinical and demographic variables n=462

Study Group		Number of Patients (n)	Correlation Coefficient (r)	p-value
Overall Population		462	0.579	<0.001
Gender	Male	259	0.629	<0.001
	Female	203	0.512	<0.001
Age Groups	<45 years	172	0.564	<0.001
	≥45 years	290	0.585	<0.001
BMI Categories	BMI<25	182	0.605	<0.001
	BMI≥25	280	0.561	<0.001
Symptom Duration	<12 hours	234	0.547	<0.001
	≥12 hours	228	0.607	<0.001
Comorbidities	Hypertensive	199	0.505	<0.001
	Non-Hypertensive	263	0.636	<0.001
	Diabetic	147	0.614	<0.001
	Non-diabetic	315	0.562	<0.001
	Smoker	172	0.689	<0.001
	Non-smoker	290	0.516	<0.001
Family History of CAD	Yes	194	0.604	<0.001
	No	268	0.559	<0.001
Subtypes of CAD	Stable angina	137	0.596	<0.001
	NSTEMI	154	0.595	<0.001
	STEMI	171	0.533	<0.001

DISCUSSION

This study offers substantial evidence of a strong correlation between increased LDL cholesterol levels and the severity of coronary atherosclerosis as evaluated by angiography. The total group exhibited a moderate yet extremely significant connection ($r=0.579$, $p<0.001$) between LDL levels and the degree of angiographic illness. This corresponds with the documented pathophysiological role of LDL in atherosclerosis, wherein higher circulating LDL

particles penetrate the artery intima, undergo oxidative alteration, and initiate a series of inflammatory responses resulting in plaque development and progression. The robustness of this link in our study underscores pivotal role of LDL in coronary atherosclerosis and corroborates existing guideline recommendations for vigorous LDL reduction in CAD patients.[viii,ix] The connection strength significantly differed among patient groupings, highlighting critical patterns regarding atherogenicity due to LDL in various clinical situations. Our observation of a moderate but significant correlation ($r=0.579$, $p<0.001$) between LDL levels and angiographic severity is consistent with multiple studies. Tarchalski et al. (2003) reported a similar positive correlation ($r=0.484$, $p<0.001$) between LDL and the Gensini score, reinforcing LDL's role as a key atherogenic driver.[x] Jin et al. (2006) further supported this, demonstrating that LDL-C levels were significantly higher in patients with multi-vessel disease compared to single-vessel CAD.[xi] In a study, there is no statistically significant relationship between LDL levels and the results of angiography, as indicated by a p-value of 0.888 and a correlation coefficient of -0.025.[xii] In another study, there is statistically significant relationship between LDL levels and the results of angiography, as indicated by a p-value of 0.052 and a correlation coefficient of 0.13.[xiii]

The most significant correlations were observed in smokers ($r=0.689$), individuals with diabetes ($r=0.614$), and those with a familial predisposition to CAD ($r=0.604$). These data indicate that LDL may synergistically combine with other cardiovascular risk factors to expedite coronary atherosclerosis. The robust connection in diabetics ($r=0.614$) underscores the specific susceptibility of diabetic vasculature to LDL-induced damage. In diabetes, glycated LDL is more susceptible to oxidation and foam cell production, whereas diabetic dyslipidemia typically presents with elevated tiny dense LDL particles that are particularly atherogenic. This underscores the necessity for rigorous LDL management in diabetic CAD patients, possibly aiming for objectives lower than those for non-diabetics. For smokers, the pronounced correlation may indicate cumulative endothelial damage from both smoking-related toxins and LDL infiltration. In individuals with diabetes, the glycation of LDL particles and related metabolic abnormalities likely increase their atherogenic potential. The significant association among individuals with a familial history indicates possible genetic influences that enhance the vascular effects of LDL.[xiv,xv]

A notable finding was the gender difference in correlation strength, with males exhibiting a larger connection ($r=0.629$) compared to females ($r=0.512$). Various biological and methodological factors may account for this disparity. The cardioprotective effects of estrogen in premenopausal women may partially counteract the atherogenic potential of LDL, possibly via improved reverse cholesterol transport or antioxidant properties. Conversely, diagnostic difficulties in female CAD patients, who frequently have more diffuse illness or microvascular dysfunction, may result in an underappreciation of actual angiographic severity. This gender disparity has significant clinical ramifications, indicating that LDL control thresholds and treatment objectives may require gender-specific attention.[xvi,xvii] Our stratified analysis revealed gender disparities, with males exhibiting a stronger LDL-atherosclerosis correlation ($r=0.629$) than females ($r=0.512$). This finding parallels Gupta et al. (2024),[xviii] who noted that lipid profiles may differentially predict CAD severity between sexes, though hormonal and vascular factors likely modulate this relationship. Additionally, the stronger association in diabetics ($r=0.614$) aligns with Pradhan et al. (2022),[xix] who highlighted the amplified atherogenicity of LDL in diabetic patients due to glycation and small dense LDL particles.

The analysis demonstrated significant associations among age groups, but marginally greater in younger patients (<45 years, $r=0.564$ vs ≥45 years, $r=0.585$). The strong correlation in younger individuals highlights notably harmful impact of LDL on early coronary artery disease, since sustained exposure to high levels presumably accelerates atherosclerotic alterations. The slightly diminished correlation in older patients may indicate the growing influence of age-related vascular alterations independent of LDL, like arterial stiffness or the formation of senescent cells. These findings underscore the paramount significance of early LDL management, particularly in young persons with additional risk factors.[xx]

The BMI paradox, characterized by greater

correlations in normal-weight individuals (r=0.605) compared to overweight or obese patients (r=0.561), contradicts traditional beliefs on the metabolic hazards associated with obesity. This suggests that in obese individuals, non-LDL routes (such as inflammation and insulin resistance) play a more significant role in the evolution of atherosclerosis, whereas in lean individuals, LDL continues to be the primary factor. Alternatively, changes in lipoproteins associated with obesity may render routine LDL values less indicative of the actual atherogenic particle load. As normal-weight patients had a stronger correlation (r=0.605) than obese patients (r=0.561), echoes Li et al. (2021), who found that LDL's predictive value diminishes in metabolic syndrome, where non-LDL pathways (e.g., inflammation) dominate.[xxi] Similarly, our smoking subgroup showed the strongest correlation (r=0.689), corroborating Yang et al. (2011), who identified smoking as a synergistic risk factor that exacerbates LDL-mediated atherosclerosis.[xxii]

The discovery of more robust correlations with extended symptom duration (≥12 hours, r=0.607 compared to <12 hours, r=0.547) may indicate various processes. A postponed presentation may provide greater LDL-mediated plaque advancement prior to angiography. Conversely, people exhibiting elevated LDL levels may form more permanent, progressive plaques that present with chronic symptoms instead of sudden rupture. The comparable correlation coefficients among CAD subtypes (stable angina r=0.596, NSTEMI r=0.595, STEMI r=0.533) imply that LDL plays a role in both chronic atherosclerotic progression and acute plaque destabilization; however, the marginally weaker association in STEMI may suggest a heightened influence of thrombotic factors in this context. Notably, our study's weakest correlation in STEMI patients (r=0.533) contrasts with Hindoro et al. (2017), who reported significant lipid-angiography associations across all CAD subtypes.[xxiii] This discrepancy may stem from differences in study populations or the predominance of thrombotic over atherosclerotic mechanisms in STEMI. Finally, our findings on LDL's predictive value in familial CAD (r=0.604) resonate with Gupta et al. (1996), who emphasized lipoprotein(a) [Lp(a)] as a genetic risk amplifier.[xxiv] However, Wilkinson et al. (2023) demonstrated that LDL management post-angiography significantly impacts outcomes, suggesting that our results could inform tailored lipid-lowering strategies.[xxv]

This study offers significant insights into the correlation between LDL cholesterol and the angiographic severity of CAD, although numerous limitations should be recognized. The cross-sectional design, although effective for identifying relationships, precludes definitive causal inferences regarding LDL's contribution to atherosclerosis progression. Our single-center recruiting technique, while providing comparable protocols, may restrict the generalizability of findings to wider populations, especially those with varying ethnic or socioeconomic backgrounds. Furthermore, coronary angiography, although the definitive method for evaluating obstructive CAD, may underestimate non-obstructive or diffuse atherosclerosis, hence potentially overlooking early-stage pathology. Ultimately, despite extensive modifications, residual confounding from unmeasured variables, such as eating patterns, physical activity, or genetic predispositions, may affect the reported associations.

On the other hand, thorough documentation of risk factors, such as smoking status, diabetes, hypertension, and family history, facilitates detailed categorization. The standardized angiographic evaluation, conducted by seasoned interventional cardiologists unaware of cholesterol levels, reduces interpretative bias. Moreover, the stringent statistical methodology, utilizing Spearman's correlation and stratification for essential factors, guarantees methodological integrity. Collectively, these qualities mitigate the limitations, enhancing the reliability of the findings.

CONCLUSION

This study establishes that elevated LDL levels significantly correlate with angiographic disease severity, reinforcing central role of LDL in atherosclerosis progression. The novel contribution of this study lies in identifying critical subgroup variations, particularly the amplified associations in males, smokers, and diabetics, contrasted with attenuated relationships in obese patients and STEMI presentations. These findings have important clinical implications. Our results advocate for a precision medicine approach to lipid management, where

treatment intensity considers not just absolute LDL levels but also patient-specific characteristics that modify LDL's atherogenicity. Future research should explore whether subgroup-specific LDL targets improve cardiovascular outcomes.

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