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Role of lipid Profile, circulating Malondialdehyde (MDA) and Oxidized LDL as a biochemical risk marker for coronary artery disease

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Abstract

Background: *Coronary artery disease remains a significant cause of morbidity and mortality world-wide. Oxidized-LDL is a known contributor to atherosclerosis, a key pathological process underlying CAD. Malondialdehyde (MDA) is one of the main byproducts of lipid peroxidation. Cholesterol and other lipid parameters are regarded as important factors in coronary heart disease progression.*

Objective: *This study aims to investigate the levels of serum oxidized-LDL and MDA along with lipid levels and to find the association of Ox-LDL & serum MDA with lipid parameters across various categories of CAD as per the severity of disease.*

Material & Methods: *After obeying the inclusion and exclusion criteria, a total of 190 patients with an age group of >40 years diagnosed with coronary artery disease (CAD) attending the OPD/IPD of Teerthanker Mahaveer Hospital were finalized as the study population. The serum levels of Ox-LDL & MDA were assessed by using separate ELISA kits.*

Results: *After correction for age, sex, and lipid profile, serum values of Ox-LDL and MDA were elevated significantly in early-stage CAD. The increase in ox-LDL & MDA was more pronounced in advanced stages of CAD, with levels rising progressively as CAD stenosis severity ($p < 0.001$). Serum Ox-LDL ($r= 0.39$, $p< 0.001$) & MDA ($r= 0.45$, $p< 0.001$) were correlated positively with serum total cholesterol, LDL & TG. However, Ox-LDL ($r = -0.61$, $p < 0.01$) & MDA ($r= -0.49$, $p< 0.001$) showed inverse correlation with HDL levels.*

Conclusion: *Our findings indicate a significant correlation of elevated ox-LDL and MDA levels with adverse lipid profile changes and CAD progression, underscoring their potential as biomarkers for disease severity and progression in CAD. Our findings also support the concept that serum lipids are important CHD risk factors.*

INTRODUCTION

Cardiovascular diseases encompass a range of issues related to the heart and blood vessels, including ischemic heart disease, rheumatic heart disease, and cerebrovascular disease, commonly referred to as strokes [1].Coronary artery disease (CAD), a form of cardiovascular disease, is seen as one of the primary causes of illness and death, creating a significant socioeconomic burden globally [2].

Coronary artery disease (CAD) is the primary identifiable cause of unexpected fatalities. Historically, the prevalence of CHD was considered relatively low in affluent nations. However, recent studies have uncovered a surprisingly significant occurrence of both mild and severe CHD among certain patients. Men are more likely to be affected by CHD than women, although the rates differ with age. Among adults aged 18 to 45, the prevalence is around 0.7%. Conversely, it rises to 13.3% among those aged 55 and older. The Global Burden of Disease report indicates that India's age-

standardized cardiovascular disease (CVD) mortality rate stands at 272 per 100,000 people, which surpasses the global average of 235 per 100,000 people [3].

A lipid profile is a set of investigations that are frequently used in order to assess the risk of heart disease and can reveal whether a person is at risk for a heart attack or stroke because of hardening of arteries and blood vessel blockage. High blood cholesterol is strongly linked to the progression of heart disease [4].

The guidelines set by the American Heart Association recommend the following target values for cardiovascular disease risk factors: Total cholesterol >200 mg/dL, triglycerides >200 mg/dL, HDL <40 mg/dL, and LDL >130 mg/dL [5].

In recent years, several theories have been suggested to clarify the connection between high cholesterol levels and the onset of atherosclerosis (AS). It is now thought that the "oxidative modification hypothesis" is the key factor contributing to AS [6].

It has been determined that LDL-C plays a major role in the development of atherosclerosis (AS), which includes injuries to endothelial cells, inflammation, the creation of foam cells, and the rupture of unstable plaques [7], the NCEP ATP III guidelines have highlighted the importance of reducing LDL-C as a key objective in both the prevention and management of coronary heart disease [8].

Oxidized low-density lipoprotein (oxLDL) has been demonstrated to accumulate in atheromatous plaques. An expanding collection of research suggests that oxLDL plays a role in the mechanisms underlying coronary artery disease, acute coronary syndrome, and vulnerable plaques [9].

Malondialdehyde (MDA) results from the oxidative decomposition of polyunsaturated fatty acids initiated by free radicals, making it a widely used marker for oxidative stress [10]. Clinical research has indicated that MDA-type receptors are important and significant in cardiovascular diseases, rendering these antigens promising candidates for assessing immune responses related to atherosclerosis [11,12].

In order to lower the risk factors linked to coronary heart disease (CHD), a comprehensive strategy is necessary. Recognizing and controlling risk factors is essential for preventing CHD in people above 40 who show no symptoms, serving as primary prevention, and for mitigating recurring events in those already diagnosed, which is considered secondary prevention. Managing these risk factors should be approached as both preventive and therapeutic measures for the atherosclerotic disease process [13].

MATERIAL AND METHODS

Study design: - This study was conducted on 190 patients diagnosed with coronary artery disease and hospitalized to Teerthanker Mahaveer Hospital, Moradabad, India, between August 2023 and September 2024 During the specified period, all patients meeting the inclusion criteria were chosen as cases.

Inclusion & Exclusion Criteria: - The criteria for including participants were as follows: individuals aged over 40 experiencing typical chest pain, evidence of coronary issues following non-invasive assessments like physical examinations or imaging studies, and those who have had a myocardial infarction [14].

Exclusion criteria were as follow: Patients with renal failure, or on dialysis, organ transplant, either diagnosed chronic diseases like HIV /acquired immunodeficiency syndrome (AIDS), tuberculosis, autoimmune disorders, chronic obstructive pulmonary disease (COPD), or liver cirrhosis (chronic liver disease) were also excluded. All co-morbidities other than Metabolic Syndrome and subjects diagnosed with type 1 diabetes and familial dyslipidaemias were excluded. Pregnant females, psychiatric patients, who are severely ill will, be excluded from the study. Patients on Statins/Aspirin would also be avoided from the study. [15].

Demographic details of participants were collected through a standardized questionnaire that covered age, gender, and risk factors of cardiovascular diseases, including smoking, family history of heart disease, and hypertension.

Coronary Angiography

Procedure for coronary angiography was done utilizing the catheter approach. The catheters used were TIG-5Fr, JL 3.5 6Fr, and JR 3.5 6Fr, as well as Visipaque dye. Two cardiologists collected the angiographic results, and as per the

findings of report, the patients were grouped into three categories based on the degree of blockage among the vessels involved as well as the severity of the disease, as determined by the CAD-RADS score.

Patient Stratification

This research evaluates the severity of coronary artery stenosis using the CAD-RADS score, which reflects the extent and intensity of coronary atherosclerosis. The first classification established was the Mild CAD group, determined by the interpretation of the disease following CAD-RADS criteria. The second category identified was the Moderate CAD group, based on the percentage of blockage in the affected vessels, including the left anterior descending artery (LAD), right coronary artery (RCA), left circumflex artery (LCX), and the diagonal or marginal artery. Third classification was the Severe CAD group, defined by the level of stenosis in the affected vessels, indicating stenosis percentages such as 50%, 50–75%, and over 75% [16].

Biochemical Assay

Following a 12-hour fasting period, 10 ml of venous blood was drawn from each patient. The serum specimens were spun at 4000 RPM for 10 minutes and then preserved at −70 °C. Standard laboratory methods were employed to analyze lipid parameters, triglycerides (TGs), cholesterol (Chol), low density lipoprotein cholesterol (LDL-C), and high density lipoprotein cholesterol (HDL-C). LDL-C levels were determined using an autoanalyzer. ELISA technique was done to evaluate the concentrations of serum MDA and Ox-LDL, with the assays conducted according to the guidelines provided by the manufacturer.

Statistical Analysis

The quantitative data is expressed in the form of mean ± SD values. The Kolmogorov-Smirnov and Shapiro-Wilk tests were employed to assess the normality of data. A one-way ANOVA was conducted to compare all parameters among various groups (CAD-RADS groups, age groups) for hypothesis testing. Post hoc ANOVA tests were performed for pairwise comparisons. The correlation of plasma MDA, Ox-LDL with lipid profile was determined using Pearson's correlation coefficient. The p-value of 0.05 or less was deemed statistically significant for all variables. The analysis was carried out using IBM SPSS Statistics for Windows.

RESULTS

This study investigated the lipid profile parameters, oxidized low-density lipoprotein (Ox-LDL) and malondialdehyde (MDA) levels among 190 patients diagnosed with CAD. The analysis aimed to elucidate the relationships between these biochemical markers and the severity of CAD. By comparing the mean values across different CAD severity groups—mild, moderate, and severe—we aimed to identify trends that would improve our knowledge of the underlying pathophysiological mechanisms and their potential implications for clinical practice.

Table 1: Descriptive statistics of different Parameters among all patients of CAD

Parameters	Minimum	Maximum	Mean ± SD
CHOLESTEROL	200.80	363.10	228.167 ± 35.021
TG	160.30	335.30	189.809 ± 40.081
HDL	13.00	45.50	27.078 ± 4.973
LDL	125.16	275.24	178.631 ± 31.361
VLDL	32.06	121.06	45.406 ± 10.508
OX-LDL	413.40	682.67	555.907 ± 48.974
MDA	5.94	39.64	19.363 ± 6.797

Table 1 summarizes the descriptive statistics, highlighting mean values, standard deviations, and ranges for each parameter among the 190 patients diagnosed with coronary artery disease (CAD).

Table 2: Comparative analysis of oxidative stress parameters among different groups of CAD patients categorized as per CAD-RADS criteria using ANOVA analysis

Biomarkers	CAD Interpretation by CAD-RADS	Mean ± SD	P value Sig. (ANOVA)
OX-LDL	**Group 1 CAD**	490.55 ± 32.560	<0.05*
	Group 2CAD	540.074 ± 35.115	
	Group 3 CAD	578.257 ± 34.432	
MDA	**Group 1 CAD**	13.057±4.762	<.001**
	Group 2CAD	16.757 ± 4.462	
	Group 3 CAD	22.059 ± 6.327	

Table 3: Comparative analysis of lipid profile among different groups of CAD patients categorized as per CAD-RADS criteria using ANOVA analysis

Lipid Biomarkers	CAD Interpretation by CAD-RADS	Mean ± SD	P value Sig. (ANOVA)
CHOLES	**Group 1 CAD**	204.95 ± 2.681	<.001**
	Group 2CAD	210.288 ± 13.122	
	Group 3 CAD	242.566 ± 41.421	
TG	**Group 1 CAD**	164.812 ± 2.784	<.001**
	Group 2CAD	168.719 ± 4.248	
	Group 3 CAD	209.138 ± 48.187	
HDL	**Group 1 CAD**	33.559 ± 5.772	<.001**
	Group 2CAD	27.980 ± 2.952	
	Group 3 CAD	25.136 ± 3.479	
LDL	**Group 1 CAD**	147.428 ± 24.773	<.001**
	Group 2CAD	173.381 ± 25.007	
	Group 3 CAD	190.614 ± 30.224	
VLDL	**Group 1 CAD**	35.937± 6.722	<.001**
	Group 2CAD	43.410 ± 8.271	
	Group 3 CAD	49.705 ± 11.003	

Table 2&3 depicts comparative analysis of lipid profiles, Ox-LDL, and MDA levels among different groups of CAD by one-way ANOVA test in CAD patients. Our findings revealed that both mean and standard deviation values were significantly different in CAD patients among all three different groups. Notably, those with severe CAD exhibited markedly higher mean values than their counterparts in the moderate and mild CAD groups.

Table 4: Pair wise Post Hoc analysis of parameters of Oxidative stress and Lipid Biomarkers among 3 different groups of cases of CAD using Tukey's HSD testing

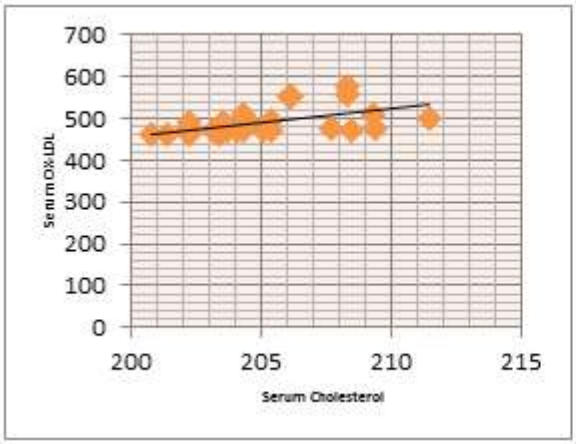
Parameters	P value Sig. (Post hoc ANOVA)		
	Group 1 Vs Group 2	**Group 2 Vs Group 3**	**Group 3 Vs Group 1**
OX-LDL	0.005	0.643	0.000
MDA	0.020	0.07	0.000
CHOLESTEROL	0.04	0.764	0.001
TG	0.003	0.895	0.001
HDL	0.001	0.09	0.000
LDL	0.002	0.307	0.000

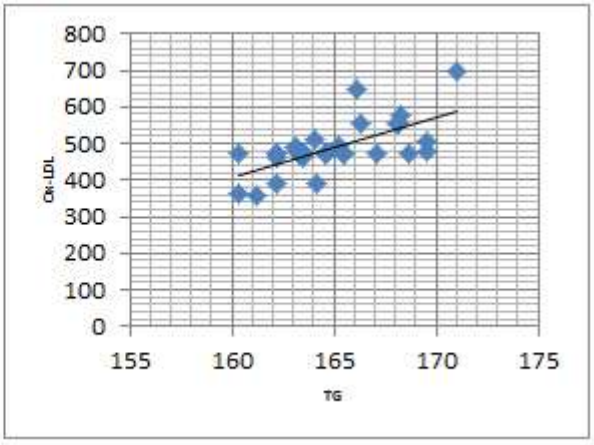
VLDL	0.005	0.875	0.000

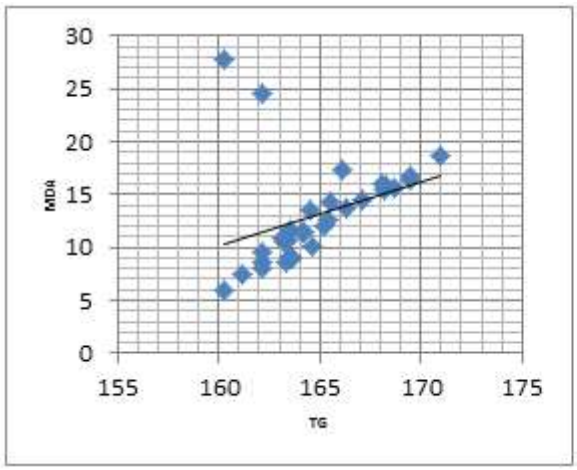
Table 4 represents the post hoc analysis for pair-wise comparison of all parameters to determine the specific difference in means of 3 groups of CAD.

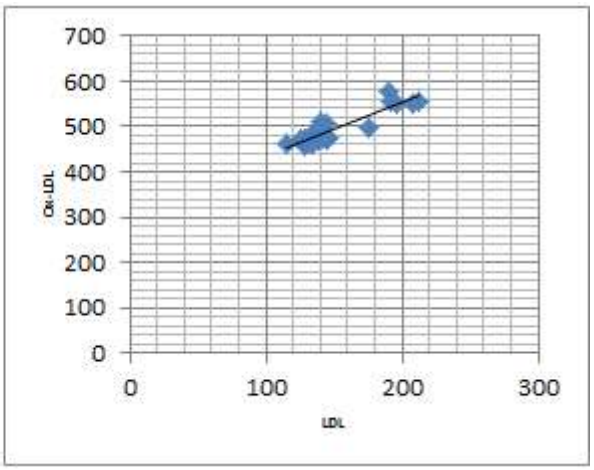
Table 5: Correlation of lipid profile with parameters of oxidative stress (Ox-LDL & MDA) among patients of Group 1 CAD categorized as per CAD-RADS criteria

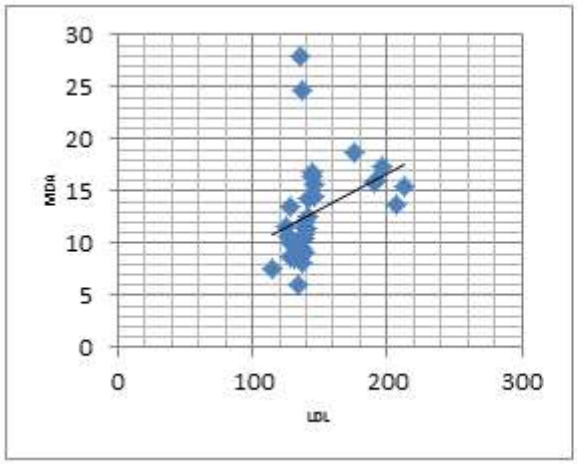
Parameters		**CHOL**	**TG**	**HDL**	**LDL**	**VLDL**
Ox-LDL	**Pearson Correlation**	0.402*	0.504*	-0.354	0.557**	0.402*
	Sig (2-tailed)	0.03	0.01	0.346	0.000	0.034
	N	32	32	32	32	32
MDA	**Pearson Correlation**	0.301	0.453 *	-0.254	0.407*	0.354*
	Sig (2-tailed)	0.148	0.054	0.473	0.04	0.042
	N	32	32	32	32	32

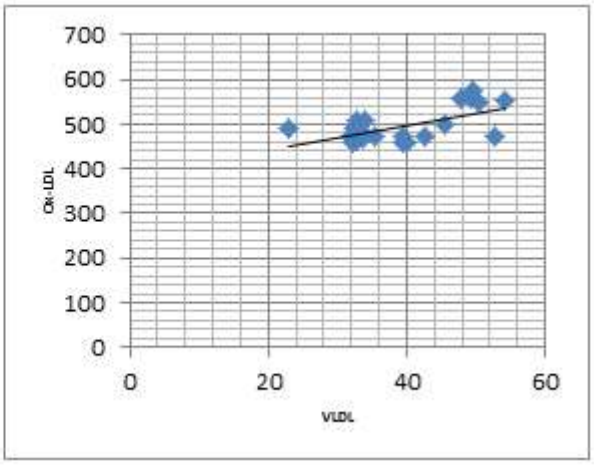












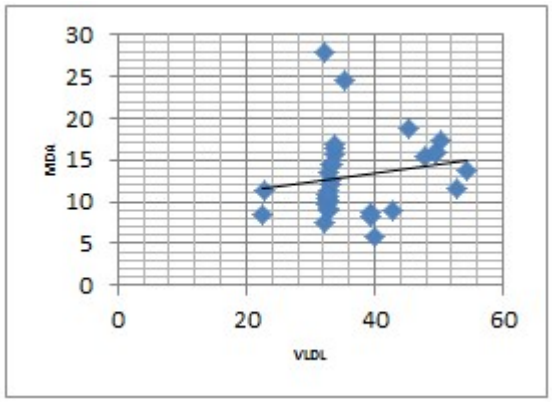
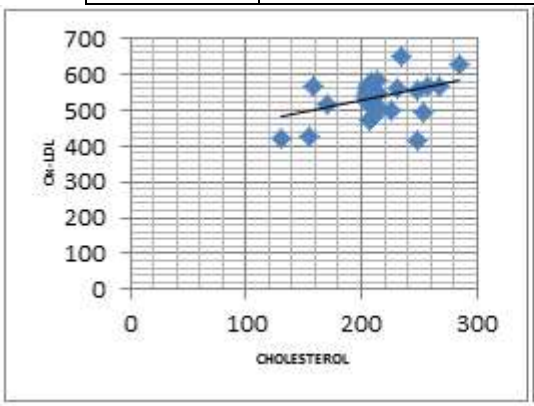
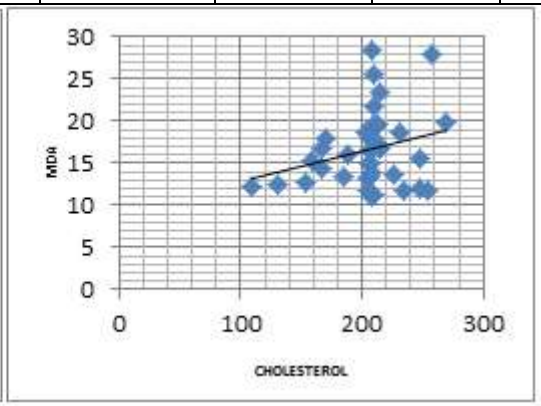


Table 6: Correlation of lipid profile with parameters of oxidative stress (Ox-LDL & MDA) among patients of Group 2 CAD categorized as per CAD-RADS criteria

Parameters		**CHOL**	**TG**	**HDL**	**LDL**	**VLDL**
Ox-LDL	**Pearson Correlation**	0.507**	0.603**	-0.456	0.656**	0.507**
	Sig (2-tailed)	0.02	0.001	0.354	0.005	0.002
	N	36	36	36	36	36
MDA	**Pearson Correlation**	0.459*	0.554 *	-0.40*	0.506*	0.452**
	Sig (2-tailed)	0.05	0.003	0.05	0.03	0.005
	N	36	36	36	36	36





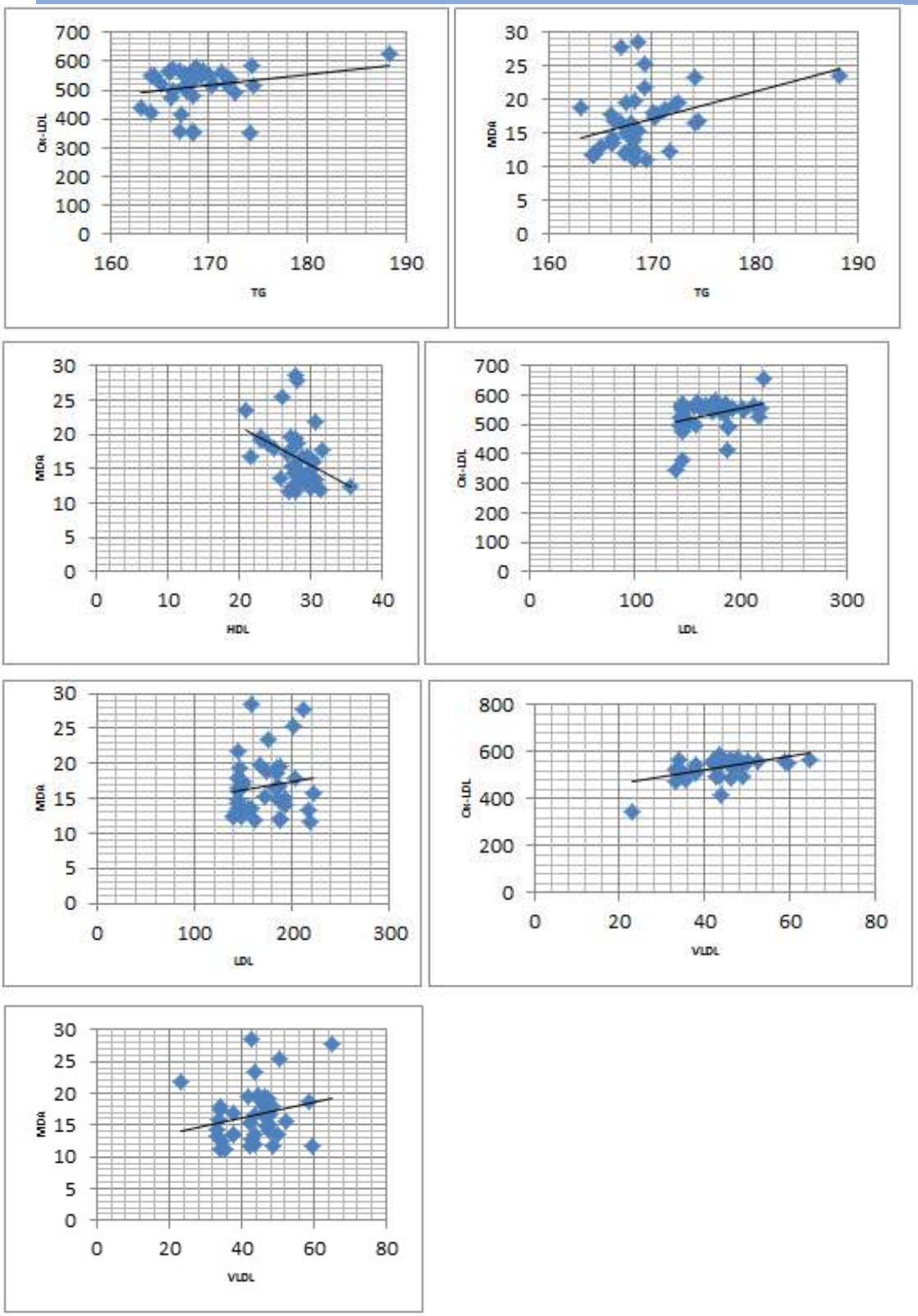
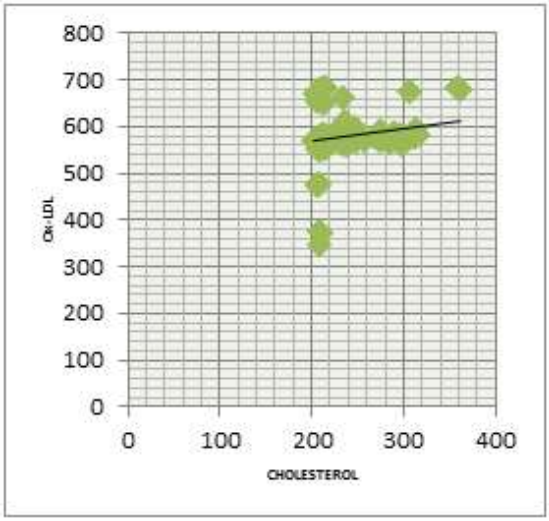
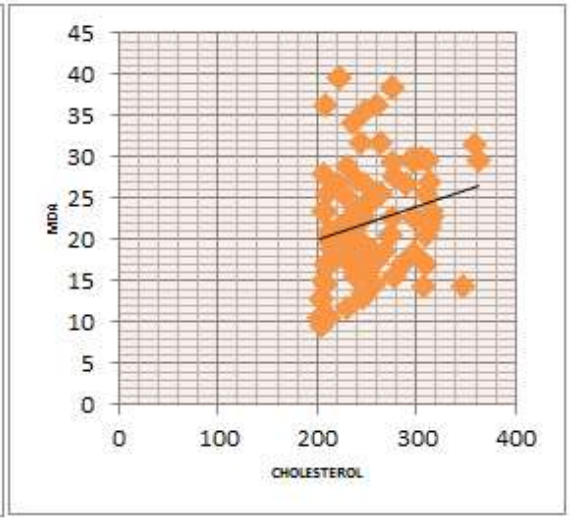
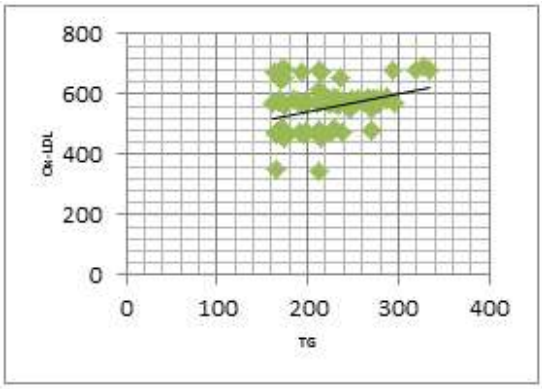


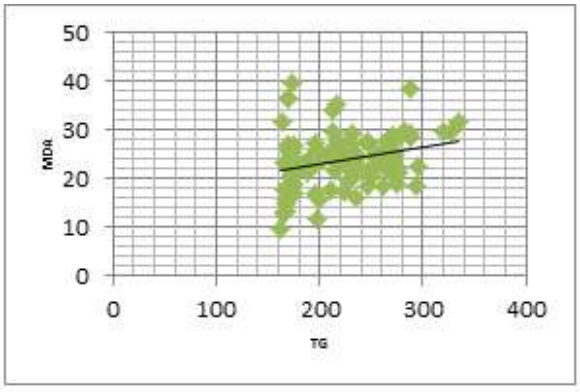
Table 7: Correlation of lipid profile with parameters of oxidative stress (Ox-LDL & MDA) among patients of Group 3 CAD categorized as per CAD-RADS criteria

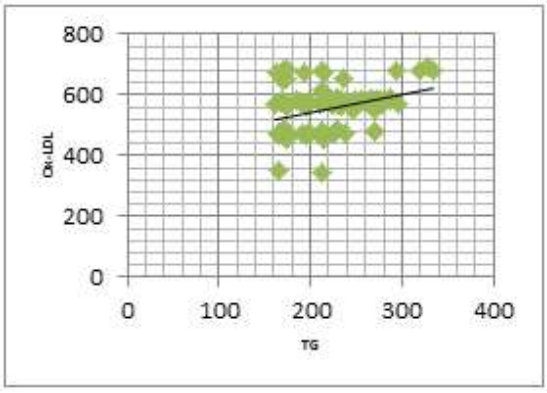
Parameters		CHOL	TG	HDL	LDL	VLDL
Ox-LDL	Pearson Correlation	0.554**	0.705**	-0.508**	0.756**	0.654**
	Sig (2-tailed)	0.001	0.001	0.000	0.0005	0.005
	N	82	82	82	82	82
MDA	Pearson Correlation	0.356**	0.604**	-0.503**	0.654**	0.604**
	Sig (2-tailed)	0.000	0.01	0.001	0.005	0.01
	N	82	82	82	82	82

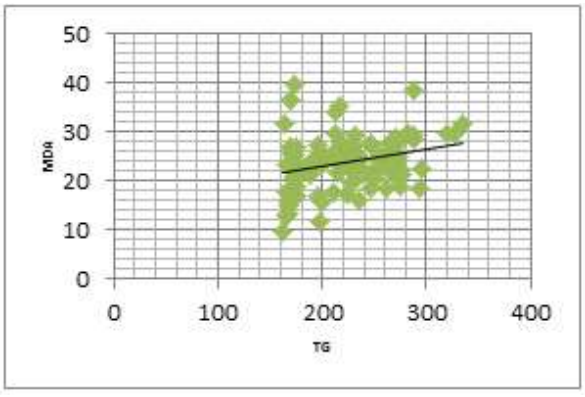


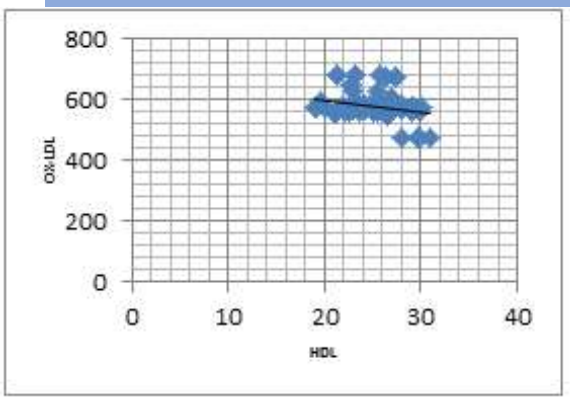


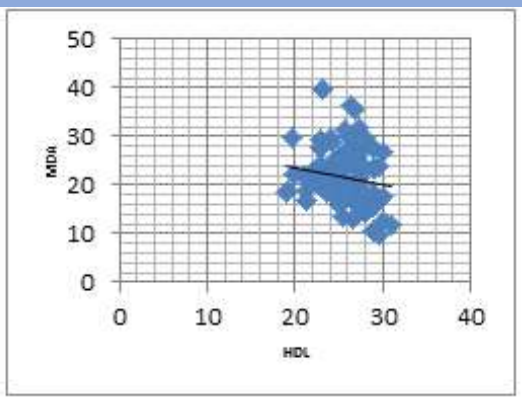


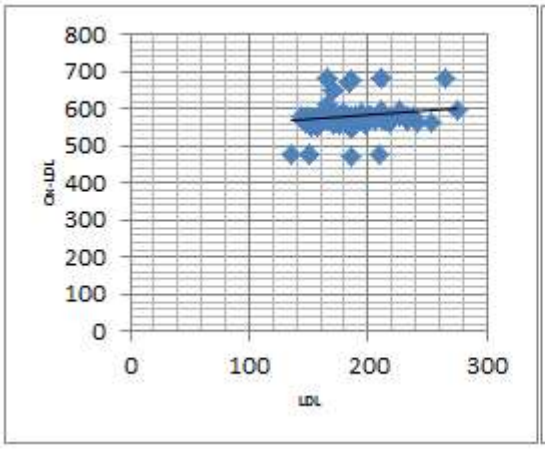


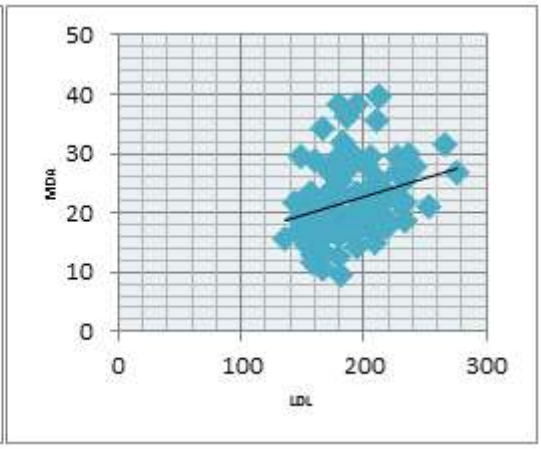


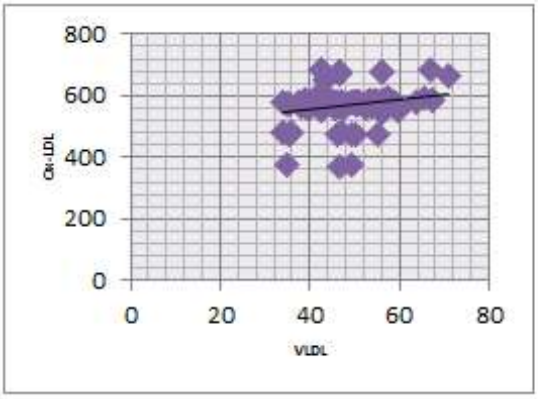


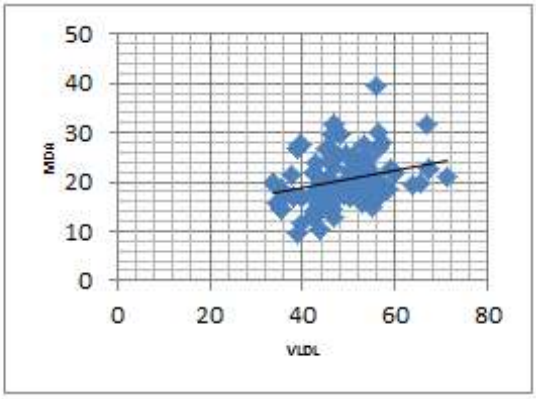












DISCUSSION

Coronary artery disease (CAD) occurs when there is insufficient blood and oxygen delivery to cardiac muscle due to blockages in the coronary arteries, leading to a mismatch between oxygen demand and supply [17]. The onset of CAD is attributed to the buildup of plaque in arterial wall supplying blood to the heart, a process referred to as atherosclerosis, which progressively constricts the arteries [18].

Elevated oxidative stress has been proven as a likely shared cause in numerous cardiovascular diseases (CVDs). Strong evidence supports the role of free radicals (ROS/RNS) in the development of various health issues. A considerable amount of information exists regarding the molecular mechanisms that induce oxidative stress and CVD. These diseases exhibit a highly intricate pathogenesis, and no single mechanism can fully account for the underlying pathophysiology. Therefore, oxidative stress and inflammation ought to be viewed as contributing factors rather than primary pathophysiological causes [19].

In view of above statements, the current study had been undertaken to evaluate the serum levels of Ox-LDL & MDA

along with lipid profile among 190 CAD patients, that were classified into three groups as Mild, moderate and severe CAD as per the chosen CAD-RADS criteria.

Out of total diagnosed 190 coronary artery disease (CAD) patients in this study; 141 of them were men (74.2%) and 49 were women (25.8%). The predominance of males in our research aligns with previous findings, which have consistently indicated that coronary artery disease (CAD) is more common among men than women, especially in younger populations. Various studies demonstrate that men are considerably more likely to develop CAD compared to women, largely due to elevated levels of conventional cardiovascular risk factors like smoking, hypertension, and high cholesterol ***(Gao Z et al. ; Wada H et al.)***. Conversely, because of estrogen's protective effects, women typically experience the onset of CAD later in life, usually following menopause. ***(Xiang D et al., 2021)*** [20,21,22].

Table 1 demonstrates the descriptive statistics of oxidative stress markers and lipid profile in CAD patients. The mean value of oxidative stress markers, Ox-LDL and MDA were elevated along with dysregulated lipid profile, such as the mean values for Ox-LDL was **555.907 ± 48.974 nmol/ml** and it was increased from the normal range. Numerous researches have shown elevated levels of serum Ox-LDL among patients of CAD serves to endothelial dysfunction, atherogenesis, and the formation of plaques. Comparable results were found by ***Zhang Q. and colleagues in 2019,*** [23]. This research revealed significantly higher concentrations of Ox-LDL in CAD patients compared to healthy individuals, emphasizing the critical role of oxidized lipoproteins in atherosclerosis development. They observed that Ox-LDL triggers endothelial dysfunction, raises arterial wall permeability, and stimulates foam cell formation, all contributing to plaque accumulation. Additionally, oxidative stress increases the atherogenic capacity of LDL particles by promoting their oxidation.

Our results align with the study by ***Ehara S. et al.*** which found that levels of Ox-LDL were significantly elevated in relation to the extent of severity of coronary artery disease (CAD), indicating that higher Ox-LDL levels are associated with more advanced atherosclerotic conditions. They proposed that oxidative stress facilitates the transformation of LDL particles into Ox-LDL. Macrophages identify the Ox-LDL via scavenger receptors, leading to the generation of foam cells and greater plaque instability. Increased Ox-LDL concentrations are vital to the inflammatory mechanisms that contribute to the development of atherosclerosis in CAD [24].

Additionally, ***Holvoet P et al***. found that individuals suffering from CAD exhibited elevated ox-LDL [25]. ***Huang H et al.*** identified the presence of ox-LDL in unstable plaques and established a connection between ACS and increased ox-LDL levels. It is believed that the amount of ox-LDL may reflect the severity of acute coronary syndrome. Collectively, these findings imply that ox-LDL is a crucial factor in the development of coronary artery disease [26].

Average mean value of serum MDA was **19.363 ± 6.797 nmol/ml**, and this is again elevated from its normal range. ***Shaafi S. et al.*** found comparable results regarding serum MDA levels. This research demonstrated significantly elevated MDA levels, which is an indicator of lipid peroxidation, among patients of CAD in contrast to healthy subjects. MDA levels were directly correlated with the severity of CAD. They described that the interaction of free radicals interact with polyunsaturated fatty acids in cellular membranes, causes lipid peroxidation, leading to the formation of reactive aldehydes like MDA. The heightened oxidative stress experienced by CAD patients results in increased lipid peroxidation, which damages the arterial walls and encourages plaque development. The elevated MDA levels in CAD indicate the extent of oxidative injury present [27].

Khaki-Khatibi F. et al. reported significant findings. They found that MDA levels were notably higher among CAD cases in comparison with controls, and elevated levels correlated with more severe coronary artery obstruction. Increased MDA levels indicate heightened oxidative stress and lipid peroxidation. The ongoing oxidative conditions in CAD promote lipid oxidation, resulting in endothelial damage, infiltration of inflammatory cells, and the development of atherosclerotic plaques. MDA acts not only as an indicator of oxidation of lipids but also contributes to CAD by enhancing plaque instability and vascular inflammation [28].

Furthermore, for the lipid profile the mean cholesterol, LDL-C and triglycerides levels were **228.167 ± 35.021 mg/dl,**

178.631 ± 31.361 mg/dl and 189.809 ± 40.081 mg/dl, respectively, all of which were raised from their normal range. In contrast, HDL-C levels were **27.078 ± 4.973 mg/dl,** which was decreased from its normal range. Supporting results are demonstrated by ***Abera A., et al. and Gonzalez-Pacheco H et al.*** where, CAD have notably elevated total cholesterol, LDL, and TG along with low levels of HDL, when compared to control groups. This observation helps us to understand dyslipidemia as a risk factor for CAD. Elevated levels of LDL and triglycerides contribute to the buildup of cholesterol in the arterial walls, whereas low HDL levels hinder the removal of excess cholesterol from these vessel walls. By altering LDL levels and reducing HDL activity, oxidative stress worsens the lipid metabolic imbalance in individuals with CAD, leading to a higher risk of atherogenesis. Consequently, the changed lipid profile accelerates plaque formation and the development of atherosclerosis. [29, 30]

Table 2 and 3 summarizes the findings of the ANOVA analysis comparing oxidative stress markers ***(Ox-LDL and MDA)*** along with lipid profile across three groups of CAD patients. Group 1, 2 an 3 represents different sub-groups based on interpretation of severity of CAD by CAD-RADS criteria.

As per ANOVA findings there was significant diiference (***p <0.05)*** in the mean values of serum Ox-LDL & MDA between the 3 CAD groups. Group 3 (Table 2) had the highest Ox-LDL (**578.257 ± 34.432**) & MDA levels (**22.059 ± 6.327**) suggesting that the patients of Group 3 had more severe oxidative damage ***(p < 0.05)*** than those of Groups 1 and 2, confirming the association of elevated oxidative stress and the severity of ***CAD (p < 0.01)***. Total cholesterol, LDL, and triglyceride levels varied significantly (***p value < 0.05)***, across the three groups (Table 3). While Group 1 had the lowest levels of these lipid markers, Group 3 had noticeably increased amounts of total cholesterol, triglycerides, LDL. HDL and cholesterol levelswere lowest in Group 3 which is in line with the dyslipidemic profile linked to advanced CAD.

Post-hoc analysis presented in ***Table 4*** further clarifies the significance of differences for oxidative stress markers (Ox-LDL & MDA) and lipid profile parameters observed in ANOVA testing between the three groups of CAD.

The findings of Post hoc analysis revealed a highly statistical significant difference among ***Group 1 and Group 3 CAD groups (p = 0.001)*** among oxidative stress markers and lipid biomarkers.

Furthermore, this difference was also statistically significant among the ***Group 1 and Group 2 CAD groups (p = 0.05)*** between various parameters. However, no significant difference was found between the ***2nd and 3rd CAD groups (p = 0.07)*** among oxidative stress parameters and lipid profile.

These results indicate that the severity of CAD impacts the measured outcome, with more pronounced differences observed ***between "Group 1 and Group 3 cases".***

For example, **Stocker R et al.** reported that elevated levels of Ox-LDL were correlated with adverse lipid profiles, characterized by increased LDL and triglycerides, along with more pronounced atherosclerosis [31]. Furthermore, Tanaga K et al. revealed that in patients with CAD, greater oxidative stress indicated by MDA is correlated with heightened lipid peroxidation and instability of plaques. [32].

Table 5, 6 and 7 present the correlation coefficients between parameters of oxidative stress (Ox-LDL & MDA) and lipid profile in Group 1 (mild CAD), Group 2 (moderate CAD) and Group3 (severe CAD) , respectively.

The correlation analysis revealed a positive relationship between Ox-LDL and total cholesterol and LDL cholesterol, including triglycerides across all the three groups, indicating that elevated Ox-LDL values are closely related with lipid dysregulation in patients with CAD. And a strong negative relationship was revealed between Ox-LDL and *HDL* among group 3 patients, this reflects that these relationship are particularly pronounced in Group 3 (severe CAD), as compare to Group 1(mild CAD) and Group 2 (moderate CAD), thus highlighting the progressive role of oxidative stress in lipid abnormalities as the disease advances.

Similarly, MDA levels were significantly associated with triglycerides only in Group 1. Among Group 2 MDA was significantly correlated positively with total cholesterol, triglycerides and negatively with HDL-C, suggesting that oxidative damage, as indicated by MDA, is linked to lipid accumulation in the earlier stages of CAD.

In Group 3, the correlation between MDA and lipid biomarkers became even more robust ***(r = 0.356, 0.604, -0.503 and 0.654*** with a highly significant ***p < 0.01*** for cholesterol, triglycerides, LDL-C and HDL respectively), further reinforcing the idea that lipid peroxidation is a key contributor to lipid disturbances in severe CAD.

Interestingly, MDA and HDL cholesterol demonstrated an inverse correlation across all three groups. Group 3 exhibited the strongest negative relationship, with a p-value below 0.01, suggesting that as the severity of CAD rises, oxidative stress contributes to the reduction of HDL levels.

Relevant literature that supports these findings is a study by ***Heitzer T., et al.*** **Findings**: The research discovered a strong link between increased oxidative stress markers (MDA, Ox-LDL) and changed lipid profiles (reduced HDL, elevated LDL, and triglycerides) in patients with coronary artery disease (CAD). This trend is likely due to the significant impact of oxidative stress on lipid metabolism. Free radicals modify lipoproteins, enhancing their likelihood of becoming atherogenic. Increased Ox-LDL leads to dysfunction of the endothelium and fosters the buildup of fat within the arterial walls. Lipid irregularities that facilitate LDL accumulation and disrupt the function of reverse cholesterol transport (HDL function) are associated with heightened oxidative damage in CAD, which accelerates atherosclerosis progression. [33]. The research discovered a strong link between increased oxidative stress markers (MDA, Ox-LDL) and changed lipid profiles (reduced HDL, elevated LDL, and triglycerides) in CAD patients. This trend is likely due to the significant impact of oxidative stress on lipid metabolism. Free radicals modify lipoproteins, enhancing their likelihood of becoming atherogenic. Increased Ox-LDL leads to dysfunction of the endothelium and fosters the buildup of fat within the arterial walls. Lipid irregularities that facilitate LDL accumulation and disrupt the function of reverse cholesterol transport (HDL function) are associated with heightened oxidative damage in CAD, which accelerates atherosclerosis progression [34].

CONCLUSION

This research highlights the relationship between lipid profiles in individuals exhibiting various lipid levels and markers of oxidative stress (ox-LDL and MDA). Our findings indicate that abnormal lipid profiles, characterized by elevated total cholesterol and LDL-C levels along with a decrease in HDL-C levels, are significantly linked to elevated oxidative markers, particularly ox-LDL and MDA.

The significant connections between these oxidative markers and lipid components support the growing evidence that oxidative stress and an imbalance in lipids are closely related with the progression of cardiovascular disease. Additional research is needed to pinpoint the specific causal mechanisms and possible therapeutic implications, although these results provide valuable insights into the relationship between oxidative stress and lipid metabolism. Future research should investigate the benefits of pharmacological treatments, dietary changes, and antioxidant interventions in lowering oxidative stress and improving lipid profiles in vulnerable populations.

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