# Lipid Peroxidation and Lipid Profile in Dyslipidemia and Coronary Artery Disease

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**Abstract—** Dyslipidemia is one of the conventional risk factor whereas enhanced oxidative stress is one of the novel risk factor for coronary artery disease (CAD). This study was conducted to compare lipid peroxidation and lipid profile in dyslipidemia and coronary artery disease and also to correlate the lipid profile parameters with MDA, the product of lipid peroxidation. One hundred and twenty three clinically proved coronary artery disease patients and 109 age and sex matched subjects without coronary artery diseases were included in this study. The subjects were classified into four groups as CAD patients with dyslipidemia, CAD patients without dyslipidemia, subjects without CAD but with dyslipidemia and subjects without CAD and without dyslipidemia. Total cholesterol, triglycerides, high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol and MDA were estimated. Statistical analysis was done by using one –way analysis of variance (ANOVA) to test the significant difference of various parameters between groups of subjects. Correlation coefficient was calculated to test the correlation of various lipid profile parameters with MDA.In the present study total cholesterol and LDL cholesterol values were significantly higher for CAD patients with dyslipidemia and subjects with dyslipidemia only than the subjects without CAD and dyslipidemia ($p$ = 0.000). We found that HDL cholesterol levels were significantly low in CAD patients than the subjects without CAD ($p$ = 0.002). CAD patients with dyslipidemia had a significantly high level of triglyceride than the rest of the groups. MDA level was significantly higher in CAD patients ($p$ = 0.000) and in subjects with dyslipidemia but without CAD ($p$ = 0.000) than subjects without dyslipidemia and CAD. A statistically significant correlation was found between high levels of total cholesterol, LDL and triglyceride and low levels of HDL with MDA in the study subjects. From these findings it can be concluded that dyslipidemia especially low HDL is a predictor for CAD. Abnormal lipid profile parameters are significantly correlated with lipid peroxidation which is elevated in dyslipidemia. Both dyslipidemia and lipid peroxidation independently contribute to the development of CAD.

**Key Words—** Coronary artery disease, Dyslipidemia, Lipid peroxidation, Lipid profile, Oxidative stress

## 1 INTRODUCTION

Coronary artery disease (CAD) is a complex disease with several genetic and environmental risk factors. Dyslipidemia is a primary, major risk factor for CAD and may even be a prerequisite for CAD, occurring before other major risk factors come into play. Atherogenic dyslipidemia comprises a triad of increased blood concentrations of small dense low density lipoprotein (LDL) particles, decreased high-density lipoprotein (HDL) particles, and increased triglycerides (TG) [1]. According to NCEP - ATP III (National cholesterol education program - Adult treatment panel III) guidelines [2] hypercholesterolemia is defined as total cholesterol >200mg/dl, LDL as >100mg/dl, hypertriglyceridemia as triglyceride >150mg/dl and HDL <40mg/dl. Dyslipidemia is defined by presence of one or more than one abnormal serum lipid concentration.

Low-density lipoproteins (LDL) are known to promote cholesterol accumulation in macrophages as well as inflammatory responses within the vessel wall; leading to atherosclerosis progression [3]. In contrast to LDL cholesterol, HDL cholesterol plays a protective role. HDL promotes reverse cholesterol transport, and has other anti-inflammatory, antithrombotic and antioxidant effects that may contribute to inhibition of atherosclerosis. Elevated plasma triglyceride concentrations contribute to increased risk of cardiovascular disease, both directly and indirectly because such elevations combined with associated risk factors such as obesity, metabolic syndrome, proinflammatory and prothrombotic biomarkers, and type 2 diabetes mellitus [4].

An important role in atherogenesis is played by oxidative stress, which may be induced by common risk factors. Lipid peroxidation refers to the oxidative degradation of lipids. Many studies have shown that the free radicals, reactive oxygen species (ROS), are involved in the pathogenesis of atherosclerosis and CAD [5], [6].

Oxidative stress both promotes and is induced by vascular diseases and risk factors that lead to vascular disease [7]. A crucial step in the pathogenesis of atherosclerosis is believed to be the oxidative modification of low density lipoprotein (LDL). The effects of lipid peroxides i.e. endothelial cell damage, uncontrolled lipid uptake, decreased prostaglandin synthesis and associated thrombogenicity are strongly implicated in the pathogenesis of atherosclerosis. A lot of oxygenated compounds, particularly aldehyde such as malondialdehyde (MDA) are produced during the attack of free radicals to membranes lipoproteins and polyunsaturated fatty acids [8], [9].

So far many studies were conducted to evaluate lipid profile and oxidative stress in CAD. But no systematic studies were conducted to compare lipid peroxidation and lipid profile in both dyslipidemia and CAD. Hence the present study was undertaken to compare and to correlate these factors in dyslipidemia and CAD so as to bring new light in the field of prevention and management of both dyslipidemia and CAD.

## 2 MATERIALS AND METHODS

One hundred and twenty three clinically proved coronary artery disease patients referred from the Pushpagiri Heart Institute (Inpatient and outpatient departments) and 108 age and sex matched subjects without coronary artery disease from the staffs of Pushpagiri Medical College Hospital were selected for this study. On the basis of NCEP- ATP III criteria, the subjects were classified into

Group-1: CAD patients with dyslipidemia.

Group-2: CAD patients without dyslipidemia.
Group-3: Subjects without CAD and with dyslipidemia.
Group-4: Subjects without CAD and without dyslipidemia.

Ethical approval from the Institutional ethics committee, Pushpagiri Institute of Medical Sciences and Research Centre and informed written consent were obtained. Five ml of fasting blood was collected from all the subjects and estimation of total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol and MDA a lipid peroxidation product were carried out. Total cholesterol was estimated by cholesterol oxidase-peroxidase (CHOD- PAP) method [10], Triglyceride by glycerol phosphate oxidase-peroxidase (GPO - PAP) method [11], HDL cholesterol by homogenous enzymatic colorimetric test [12] and LDL cholesterol by homogenous enzymatic colorimetric test [13]. MDA estimation was based on the reaction of MDA with thiobarbituric acid (TBA), producing a MDA-TBA adduct according to a modified version of Satoh's [14] and Yagi's methods [15].

### 2.1 Statistical Analysis

Results are presented as mean ± standard deviation. Statistical analysis was done by using one-way analysis of variance (ANOVA) to test the significant difference of various parameters between groups of subjects. Bonferronic was used as a Post-Hoc test. Correlation coefficient was calculated to test the correlation of various lipid profile parameters with MDA.

## 3 RESULTS

The mean values of total cholesterol, HDL, LDL, triglyceride and MDA of different groups were calculated and the results are shown in Table 1. The statistical data are given in Tables 2 and 3. The mean level of total cholesterol was significantly higher in group 1 subjects with CAD and dyslipidemia compared to group 2 and group 4. No such difference was observed in group 3 and subjects in group 1. The mean total cholesterol level was much lower in group 2 than group 4.

HDL cholesterol level was significantly lower in group 1 compared to other groups. Subjects in group 2 has significantly higher HDL (42.73±10.30) than in group 1 (36.37±9.469) but significantly lower than group 3 (50.98±10.489) and group 4 (52.13±11.280) subjects. No significant difference was observed between group 3 and group 4.

LDL cholesterol level was significantly higher in group 1 and group 3 compared to group 2 and group 4. No significant difference was observed among group 1 and group 3. The level of triglyceride was significantly higher in CAD patients with dyslipidemia compared to other groups. No significant difference was observed between the subjects in group 2 and group 4. Subjects in group 3 has significantly higher mean triglyceride level than group 2 and group 4 but the level was significantly lower than group 1.

MDA level was significantly higher in group 1, group 2 and group 3 compared to group 4. No significant differences were observed between group 2, group 3 and group 4. The correlation coefficient of total cholesterol, HDL, LDL and triglyceride with MDA was calculated in the study subjects and statistical data is given in the Table 4. A statistically significant correlation was found between high levels of total cholesterol, LDL and triglyceride and low levels of HDL with MDA in the study subjects. The correlation coefficient of total cholesterol, HDL, LDL and triglyceride with MDA within the groups were calculated and significant correlations were observed only for total cholesterol and LDL with MDA in subjects with dyslipidemia and not for CAD patients.

TABLE 1
SERUM LIPID PROFILE AND MDA OF THE STUDY SUBJECTS

| Lipid Profile | Group 1 $n$ = 78 | Group 2 $n$ = 45 | Group 3 $n$ = 41 | Group 4 $n$ = 67 |
|---|---|---|---|---|
| TC mg/dl | 247.50 ± 30.67 | 153.98 ± 27.56 | 254.10 ± 41.000 | 169.30 ± 21.562 |
| TG mg/dl | 167.59 ± 50.74 | 99.56 ± 35.06 | 127 ± 66.16 | 100.04 ± 32.42 |
| HDL-C mg/dl | 36.37 ± 9.47 | 42.73 ± 10.13 | 56.12 ± 13.27 | 52.13 ± 11.28 |
| LDL-C mg/dl | 177.61 ± 31.73 | 91.36 ± 27.11 | 177.62 ± 36.953 | 96.82 ± 25.543 |
| MDA nmol/mL | 1.5672 ± .71167 | 1.3742 ± .47102 | 1.5400 ± .63735 | 0.8739 ± .29326 |

*The values are given in mean± standard deviation*

TABLE 2
STATISTICAL DATA

ANOVA

| | | Sum of Squares | Df | Mean Square | F | Sig. |
|---|---|---|---|---|---|---|
| Total cholesterol | Between Groups | 440492.722 | 3 | 146830.907 | 163.568 | .000 |
| | Within Groups | 203772.117 | 227 | 897.675 | | |
| | Total | 644264.840 | 230 | | | |
| HDL | Between Groups | 10900.510 | 3 | 3633.503 | 34.058 | .000 |
| | Within Groups | 24217.785 | 227 | 106.686 | | |
| | Total | 35118.294 | 230 | | | |
| LDL | Between Groups | 398147.139 | 3 | 132715.713 | 145.145 | .000 |
| | Within Groups | 207560.521 | 227 | 914.364 | | |
| | Total | 605707.660 | 230 | | | |
| TG | Between Groups | 211000.890 | 3 | 70333.630 | 31.883 | .000 |
| | Within Groups | 500757.092 | 227 | 2205.978 | | |
| | Total | 711757.983 | 230 | | | |
| MDA | Between Groups | 20.214 | 3 | 6.738 | 21.638 | .000 |
| | Within Groups | 70.685 | 227 | .311 | | |
| | Total | 90.899 | 230 | | | |

TABLE 3
POST HOC TESTS

| Dependent Variable | (I) Group | (J) Group | Mean Difference (I–J) | Std. Error | Sig. | 95% Confidence Interval Lower Bound | 95% Confidence Interval Upper Bound |
|---|---|---|---|---|---|---|---|
| Total cholesterol | 1 | 2 | 93.522* | 5.609 | .000 | 78.59 | 108.45 |
| | | 3 | -6.598 | 5.780 | 1.000 | -21.98 | 8.79 |
| | | 4 | 78.201* | 4.991 | .000 | 64.92 | 91.48 |
| | 2 | 1 | -93.522* | 5.609 | .000 | -108.45 | -78.59 |
| | | 3 | -100.120* | 6.469 | .000 | -117.34 | -82.90 |
| | | 4 | -15.321 | 5.775 | .051 | -30.69 | .05 |
| | 3 | 1 | 6.598 | 5.780 | 1.000 | -8.79 | 21.98 |
| | | 2 | 100.120* | 6.469 | .000 | 82.90 | 117.34 |
| | | 4 | 84.799* | 5.941 | .000 | 68.99 | 100.61 |
| | 4 | 1 | -78.201* | 4.991 | .000 | -91.48 | -64.92 |
| | | 2 | 15.321 | 5.775 | .051 | -.05 | 30.69 |
| | | 3 | -84.799* | 5.941 | .000 | -100.61 | -68.99 |
| HDL | 1 | 2 | -6.362* | 1.934 | .007 | -11.51 | -1.22 |
| | | 3 | -14.604* | 1.992 | .000 | -19.91 | -9.30 |
| | | 4 | -15.763* | 1.720 | .000 | -20.34 | -11.18 |
| | 2 | 1 | 6.362* | 1.934 | .007 | 1.22 | 11.51 |
| | | 3 | -8.242* | 2.230 | .002 | -14.18 | -2.31 |
| | | 4 | -9.401* | 1.991 | .000 | -14.70 | -4.10 |
| | 3 | 1 | 14.604* | 1.992 | .000 | 9.30 | 19.91 |
| | | 2 | 8.242* | 2.230 | .002 | 2.31 | 14.18 |
| | | 4 | -1.159 | 2.048 | 1.000 | -6.61 | 4.29 |
| | 4 | 1 | 15.763* | 1.720 | .000 | 11.18 | 20.34 |
| | | 2 | 9.401* | 1.991 | .000 | 4.10 | 14.70 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | 3 | 1.159 | 2.048 | 1.000 | -4.29 | 6.61 |
| | | 2 | 86.246* | 5.661 | .000 | 71.18 | 101.31 |
| | 1 | 3 | -.014 | 5.833 | 1.000 | -15.54 | 15.51 |
| | | 4 | 80.789* | 5.037 | .000 | 67.38 | 94.20 |
| | | 1 | -86.246* | 5.661 | .000 | -101.31 | -71.18 |
| | 2 | 3 | -86.260* | 6.528 | .000 | -103.64 | -68.88 |
| | | 4 | -5.456 | 5.828 | 1.000 | -20.97 | 10.06 |
| LDL | | 1 | .014 | 5.833 | 1.000 | -15.51 | 15.54 |
| | 3 | 2 | 86.260* | 6.528 | .000 | 68.88 | 103.64 |
| | | 4 | 80.803* | 5.996 | .000 | 64.85 | 96.76 |
| | | 1 | -80.789* | 5.037 | .000 | -94.20 | -67.38 |
| | 4 | 2 | 5.456 | 5.828 | 1.000 | -10.06 | 20.97 |
| | | 3 | -80.803* | 5.996 | .000 | -96.76 | -64.85 |
| | | 2 | 68.034* | 8.792 | .000 | 44.63 | 91.44 |
| | 1 | 3 | 40.102* | 9.060 | .000 | 15.99 | 64.22 |
| | | 4 | 67.545* | 7.823 | .000 | 46.72 | 88.37 |
| | | 1 | -68.034* | 8.792 | .000 | -91.44 | -44.63 |
| | 2 | 3 | -27.932* | 10.140 | .038 | -54.92 | -.94 |
| | | 4 | -.489 | 9.052 | 1.000 | -24.58 | 23.60 |
| TG | | 1 | -40.102* | 9.060 | .000 | -64.22 | -15.99 |
| | 3 | 2 | 27.932* | 10.140 | .038 | .94 | 54.92 |
| | | 4 | 27.443* | 9.313 | .021 | 2.66 | 52.23 |
| | | 1 | -67.545* | 7.823 | .000 | -88.37 | -46.72 |
| | 4 | 2 | .489 | 9.052 | 1.000 | -23.60 | 24.58 |
| | | 3 | -27.443* | 9.313 | .021 | -52.23 | -2.66 |
| | | 2 | .19296 | .10446 | .396 | -.0851 | .4710 |
| | 1 | 3 | .02718 | .10764 | 1.000 | -.2593 | .3137 |
| | | 4 | .69330* | .09295 | .000 | .4459 | .9407 |
| | | 1 | -.19296 | .10446 | .396 | -.4710 | .0851 |
| | 2 | 3 | -.16578 | .12048 | 1.000 | -.4864 | .1549 |
| | | 4 | .50034* | .10755 | .000 | .2141 | .7866 |
| MDA | | 1 | -.02718 | .10764 | 1.000 | -.3137 | .2593 |
| | 3 | 2 | .16578 | .12048 | 1.000 | -.1549 | .4864 |
| | | 4 | .66612* | .11065 | .000 | .3716 | .9606 |
| | | 1 | -.69330* | .09295 | .000 | -.9407 | -.4459 |
| | 4 | 2 | -.50034* | .10755 | .000 | -.7866 | -.2141 |
| | | 3 | -.66612* | .11065 | .000 | -.9606 | -.3716 |

*The mean difference is significant at the 0.05 level.*

TABLE 4
CORRELATION OF LIPID PROFILE WITH MDA

| | R | *p* |
|---|---|---|
| Total Cholesterol | 0.449 | 0.001 |
| Triglyceride | 0.344 | 0.000 |
| HDL | -0.229 | 0.002 |
| LDL | 0.446 | 0.000 |

## 4 DISCUSSION

Dyslipidemia has been found to be one of the most important contributing factors for CAD. High total

cholesterol, low HDL cholesterol, high LDL cholesterol and high triglycerides play an important role in the causation of CAD [16], [17], [18], [19].

A positive correlation between plasma total cholesterol levels and risk of CAD was demonstrated in many earlier studies [20]. In the present study total cholesterol values were significantly higher for CAD patients with dyslipidemia and subjects with dyslipidemia only than the subjects without CAD and dyslipidemia (p= 0.000) which is in agreement with the above studies. CAD patients without dyslipidemia were shown to be low total cholesterol than the other three groups because most of the CAD patients may take cholesterol lowering drugs.

Low HDL-C levels are stronger predictor of occurrence and reoccurrence of MI and stroke and are also associated with premature and severe CAD [21], [22]. Low HDL is considered as a major risk factor for coronary artery disease even in individuals whose LDL levels are low; HDL remains a strong independent predictor of coronary artery disease risk [23], [24]. Findings of our study are also in well agreement with all the above studies as we found that HDL cholesterol levels were significantly low in CAD patients. Even though the LDL values are lower for CAD patients without dyslipidemia the HDL cholesterol levels were significantly lower than the subjects without CAD but with dyslipidemia (p=0.002). Therefore, HDL might be cardioprotective because it prevents cholesterol accumulation in cells of the artery wall. Animal and human studies have raised the possibility that HDL also slows vascular disease by blocking inflammation [25]. In our study subjects without CAD had significantly high HDL levels than the CAD patients (p=0.000).

Oxidative modification of LDL cholesterol is a key process of atherosclerosis and elevated LDL cholesterol has been recognized as primary risk factor for CAD by NCEP–ATP III [26]. In a study by Sawant et al (2008) increased LDL cholesterol has been found to be contributing considerably to dyslipidemia irrespective of age and gender. In the present study also CAD patients and subjects with dyslipidemia had significantly high LDL levels (p=0.000)

Elevated plasma triglyceride (TG) concentration is becoming increasingly established as an independent risk factor for premature coronary artery disease (CAD) [27], [28], [29]. ROS are involved in oxidation of LDL, which is considered as a fundamental step in the initiation and progression of atherosclerosis [30]. Malondialdehyde is a decomposition product of autooxidation of polyunsaturated fatty acids which is used as an index of oxidative damage. Many studies have linked excess generation of reactive oxygen species (ROS) with cellular damage and atherogenesis. Cavalca et al [31] have found increased MDA levels in CAD patients. The same results were observed by Das et al [20], Koutur et al [32], Kaur et al [33], Shaikh and Suryakar [34] and Simon et al [35]. Findings of this study is in well agreement with all the above mentioned studies. We observed significantly high MDA levels both in CAD patients with dyslipidemia and without dyslipidemia. These results indicate that even though lipid levels are normal there is an elevated oxidative stress in CAD patients.

An increased level of plasma malondialdehyde (MDA) is an indicator of elevated lipid damage [36] at the same time dyslipidemia may also contribute to increase in oxidative stress which accelerates the process of atherosclerosis [37] and the subjects with dyslipidemia when compared to normal subjects (p=0.000). We also found an elevated level of oxidative stress in subjects with dyslipidemia.

A study by Rao and Kiran [38] showed that statistically significant linear relationship could not be established between increased oxidative stress and abnormal lipid profile parameters. But in a study by Mahapatra [39] MDA level correlated better with total cholesterol and triglyceride levels, poorly correlated with LDL and in inverse relationship was observed with HDL. In the present study we observed a significant correlation between abnormal lipid profile parameter with MDA in the study subject. But within the different groups, significant correlations were observed only for total cholesterol and LDL for subjects with dyslipidemia.

From these findings it can be concluded that dyslipidemia especially low HDL is a predictor for CAD. Abnormal lipid profile parameters are significantly correlated with lipid peroxidation. Both dyslipidemia and lipid peroxidation independently contribute to the development of CAD. Control of hyperlipidemia through lifestyle modifications such as exercise, diet and drugs may help to reduce oxidative stress and CAD.

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