Mechanism involved in the Circulation of oxidized LDL and its Ratio; An Early Risk Marker in Diabetic and Non-Diabetic Subjects with Coronary Heart Disease

Thirunavukkarasu Jaishankar, Meera Shivasekar*, Vinodhini V M

Department of Biochemistry, SRM Institute of Science and Technology, Kattankulthur, Tamilnadu-603203, India

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ABSTRACT

Under oxidative stress hardening of arteries is linked to oxidative variations in low density lipoproteins and imaginably more than one mechanism is involved in the atherosclerosis, where LDL is oxidized in all the cells of intimal wall during the progression of CHD. Ox-LDL act as a prognosticator of dysfunction in endothelium along with pro-thrombotic, pro-apoptotic, pro-inflammatory properties in subjects suffering from oxidative stress. Circulating ox-LDL is associated with the development of atherosclerosis but also numerous degenerative and age related disease. The objective of our study is to assess the levels of circulating oxidized LDL and its ratio in Diabetic and Non-Diabetic Subjects with CHD. This cross-sectional study was conducted in Department of General Medicine and Master Health check-up OP of SRM Medical College and Research Centre, Tamil Nadu, India. Totally 273 subjects in that 91 CHD patient without Diabetes, 91 CHD patient with diabetes and 91 healthy control in age group of 30 to 55 years and were age and sex matched. After overnight fasting blood samples were collected for analysis. ox-LDL were measured by ELISA method and Lipid Profile is measured using Auto Analyser AU480. Statistical analysis was done using student ‘t’ test and Pearson correlation analysis for the comparison between two groups. When compared to controls the mean level Low Density Lipoprotein and Plasma Oxidized LDL was significantly elevated in CHD group. Significantly positive correlation was observed between plasma oxidized LDL and LDL. The study concludes that increased circulating ox-LDL and its ratio are early risk marker and useful predictor of mortality in patients with CHD. For the diagnosis and treatment of coronary heart disease an appropriate method that reveal the mechanisms which increase circulating LDL and ox-LDL is needed.

INTRODUCTION

Coronary Heart Disease is the most important reason for death in India. CHD act as a multifactorial disorder related to the environmental and genetic aspect (Pranavchand et al., 2013). CHD is increasing rapidly in Indian people and manifesting a younger age (Strong et al., 1999). It has been estimated that approximately 10% of those CHD patients were very young ≤45 years olds, has high morbidity and long-term mortality. Thus it poses a severe burden on the family and society (Fournier et al., 2004).
Clinically, the risk factors for predicting is difficult to determine so far on account of traditional risk factors that are not uniquely presented in young adults with CHD (Cole et al., 2004). Epidemiological studies further indicated that some risk factors, including currently smoking, hypertension and dyslipidemia, played relatively more prominent roles in young subjects (Allen et al., 2001).

Among the pathogenesis of atherosclerosis, deposition of lipid is briefly studied. Various studies stated that statin therapy reduces LDL-C, which prevents cardiovascular disease. An elevated level of LDL-C is the leading risk aspect for cardiovascular disease (Steinberg et al., 2002). The chief transporter of cholesterol is Low-density lipoprotein cholesterol, which accumulates in intimal layer and promotes the chemoattractant and adhesion molecules expressions in the surface of endothelial cells, by triggering the adhesion of circulating monocytes to the endothelium. After adhesion, into the intima, the monocytes are migrated, which separated into macrophages, internalize and accumulate the cholesterol in cells to become foam cells (Steinberg et al., 2009). Cholesterol uptake by the LDL receptor, which cannot accumulate cholesterol in macrophages, because the receptor of LDL is down-regulated by increased intracellular cholesterol over the mechanism of sterol regulatory element-binding protein (SREBP).

In contrast, certain altered forms of LDL-C, such as acetylated LDL and oxidized LDL, are taken up by macrophages through scavenger receptors, cause accumulation of substantial cholesterol to form foam cell because the scavenger receptor is marginally up-regulated by oxidized LDL (Horton et al., 2002). The hypothesis of LDL oxidation predicts an early incident of coronary heart disease. This study is supported by several confirmations that oxidized LDL may promote the formation of foam cell in vitro and in vivo. Several pro-atherosclerotic potentials such as endothelial cell and monocytes present in ox-LDL prompt to increase and adhesion particles, inflammatory cytokines and chemokines. Stimulation of monocytes or macrophages toward the increased tissue factor, matrix metalloproteinase, and scavenger receptors lead to the formation of foam cell and progression of atherosclerotic lesions (Steinberg et al., 2009). By various hypothesized mechanisms LDL-C turns into oxidized LDL by several enzymes within the intimal wall. Lipooxygenase acts as a non-heme iron-containing dioxygenase intracellular oxidation enzymes, which directly oxygenate polyunsaturated fatty acids (Parhasarathy et al., 1989). Lipooxygenase enzyme undergoes LDL oxidation by (enzymatic) direct and (non-enzymatic) indirect reactions. Lipooxygenase later produces radical oxidants which contribute non-enzymatic lipid peroxidation.

For inflammation and oxidative stress, Myeloperoxidase act as the primary enzyme. Myeloperoxidase is a highly cationic protein have a capability of binding in leukocytes endothelial cells, leukocytes and LDL-C. Myeloperoxidase is regulated through reactive oxygen species which oxidize lipid and protein. Relationship between MPO. and LDL-C might improve the oxidation of LDL (Schindhelm et al., 2009).

Myeloperoxidase is linked with the progression of atherosclerosis (Hazen et al., 1997). Some convincing suggestions on the significance of MPO to atherosclerosis were found to be related to lipooxygenase.

Modification of LDL to oxidized LDL is a significant occurrence in the oxidation hypothesis of atherogenesis (Leopold et al., 2009). Because of overproduction of reactive oxygen species through endothelial cells, by oxidative modifications cause endothelial dysfunction and plaque disruption which plays a critical role in the progression of coronary heart disease (Hadi et al., 2005). A study by Huang et al. stated that the ratio of LDL oxidation is strongly correlating with the severity of coronary heart disease. They found the ox-LDL and LDL oxidation ratio (ox-LDL/T.C., ox-LDL/HDL-C and ox-LDL/ LDL-C) are significantly greater in CHD subjects when compared to controls (P < 0.001) (Huang et al., 2008). At present, it is not possible to screen ox-LDL directly in human blood vessel although efforts are in progress. Therefore, we hypothesized that ox-LDL levels in younger CHD patients due to its pathophysiological role. Unfortunately, there were no such data available until now. Therefore, the objective of our current study was to assess the role of ox-LDL in predicting the future occurrence of CHD.

**MATERIALS AND METHODS**

This cross-sectional study was done from Jun 2019 to Dec 2019 at SRM. Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India, on subjects attending the Cardiology and medicine outpatient. Totally 273 subjects were included who were age and sex match in the age group 30-55 years.

Group-I = 91 Normal Healthy Control

Group-II = 91 Non-Diabetes subjects with CHD

Group-III = 91 Diabetes subjects with CHD

The control subjects were also taken from Master health check-up Programme and General Medicine.
O.P. in SRM. Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India. The study protocol was approved by the institutional ethical committee (ECN: 1513/ICE/2018).

**Inclusion criteria**

The CHD Subjects including both males and females selected based on coronary angiography

Group – I (Healthy Controls): The control group consists of persons with no clinical and ECG. evidence of CHD and negative history of the past event of CHD or stroke, Diabetes Mellitus, Hypertension, smoking, Dyslipidemia, and family history of CHD.

Group – II (Non-diabetic CHD): Serum glucose value are above the normal and below the diabetic level and patients with chest pain, ECG. changes, increased cardiac markers such as creatinine phosphokinase (CPK-MB) and troponin level.

Group – III (Diabetic CHD): Previously known diabetic patients with CHD.

**Exclusion criteria**

The subjects who were on treatment for renal failure, cancer, autoimmune diseases were excluded.

**Anthropometric measurement**

The study was explained to all the patients registered in the study; written informed consent was taken. The baseline examination included physical examination, medical history, health habits, family history of CHD and Diabetes. The physical examination comprised of the 12-lead resting electrocardiogram. Anthropometrics indices Weight (Kg), Height (Meters), Waist Circumference (cm) and Hip Circumference (cm) along with BMI and waist-hip circumference ratio were measured and calculated (Misra et al., 2009). Systolic and diastolic blood pressures were measured. Cigarette smoking was evaluated by questionnaire.

After overnight fasting Blood sample (5ml) was collected in sodium citrate and plain vacationer under aseptic precaution. 2ml of blood was taken for the measurement of Glucose. Lipid profile includes (Total cholesterol by Cholesterol Oxidase method, Triglycerides by Glycerol peroxidase method, HDL-C and LDL-C by Direct method high-density lipoprotein (HDL) cholesterol (HDLC), TC/HDL-C ratio, LDL-C/HDL-C ratio were measured using Beckman Coulter Auto analyzer (AU480).

And the remaining 3ml of plasma was allowed to clot for 30 minutes and then centrifuged at 2500 RPM for 10 minutes for the quantification of oxidized LDL was done by ELISA method.

**Statistical analysis**

All the data were analyzed using Statistical Package for Scientific Studies (SPSS) version 21. The results were denoted as Mean ± Standard Deviation. Student’s t-test was used to evaluate the variance between the mean levels of various parameters. Correlation between various variables was assessed using Pearson’s correlation equation. The p-value <0.05 was considered statistically significant.

**RESULTS**

A total of 273 Subjects, 39(41%) of male and 52(59%) of the female subject have Non-Diabetic CHD. 58(64%) of male and 33(36%) of the female subject have Diabetic CHD. And 34 (37%) of male and 57(63%) of the female subject were selected as control Table 1. Majority of Non-Diabetic CHD subjects are in the age group of 40-50 years and Diabetic Subjects with CHD in the age group of 40-50 years. At the same time, most of the control subjects fall in the age group of 30-45 years. In the subject group, 27 Non-Diabetic CHD subjects having a Family history of CHD and 20 Diabetic CHD subject having a Family history of CHD. A lifestyle habit comprises of alcohol consumption and type of diet consumed. Incidence and measurement of all these lifestyles are depicted in Table 1.

![Comparison of BMI in CHD and Healthy Control](image1)

**Figure 1: Comparison of BMI in CHD and Healthy Control**

![Comparison of ox-LDL in CHD and Healthy Control](image2)

**Figure 2: Comparison of ox-LDL in CHD and Healthy Control**

In both Diabetic and Non-Diabetic CHD patients the BMI, Waist Circumference, Waist Hip Ratio, systolic blood pressure were significantly increased
Table 1: Demographics and baseline characteristics Coronary Heart Disease Subject and Healthy Controls

| Parameters                          | Controls (n=91) | Non-Diabetic CHD patient (n=91) | Diabetic CHD patient (n=91) |
|-------------------------------------|----------------|---------------------------------|-----------------------------|
| Mean age (years, mean ± S.E.M.)     | 41.8 ± 9.7     | 42.3 ± 10.5                     | 40.6 ± 6.4                  |
| Male Sex (%)                        | 34 (37%)       | 39 (41%)                        | 58 (64%)                    |
| Female Sex (%)                      | 57 (63%)       | 52 (59%)                        | 33 (36%)                    |
| Body Mass Index (kg/m²)             | 21.9 ± 0.37    | 23.47 ± 0.35                    | 24.03 ± 0.19                |
| Waist circumference (cm)            | 90.9 ± 10.1    | 98.8 ± 9.6                      | 98.8 ± 4.3                  |
| Waist to Hip Ratio                  | 0.94 ± 0.02    | 1.01 ± 0.01                     | 1.05 ± 0.03                 |
| Waist to Height Ratio               | 0.56 ± 0.01    | 0.62 ± 0.02                     | 0.65 ± 0.01                 |
| Systolic Blood Pressure (mm Hg)     | 109.73 ± 18.32 | 122.38 ± 16.57                  | 122.26 ± 13.95              |
| Diastolic Blood Pressure (mm Hg)    | 77.69 ± 6.95   | 81.58 ± 13.26                   | 82.16 ± 16.47               |
| Diet                                |                |                                 |                             |
| Non-vegetarian                      | 55 (61%)       | 65 (70%)                        | 70 (76%)                    |
| vegetarian                          | 36 (39%)       | 26 (30%)                        | 21 (24%)                    |
| Alcohol drinking                    |                |                                 |                             |
| Drinkers                            | 4 (6%)         | 31 (34%)                        | 38 (41%)                    |
| Non-Drinkers                        | 87 (94%)       | 60 (66%)                        | 53 (59%)                    |
| Diabetes                            |                |                                 |                             |
| Diabetic                            | 0              | 0                               | 91 (100%)                   |
| Non-Diabetic                        | 91 (100%)      | 91 (100%)                       | 0                           |
| Family history of CHD               | Yes            | 0                               | 27 (31%)                    |
| No                                  | 91 (100%)      | 64 (69%)                        | 71 (78%)                    |

Values are expressed in Mean ± Standard Deviation.

BMI- Body Mass Index, SBP- Systolic Blood Pressure DBP-Diastolic Blood Pressure

Table 2: Assessment of Biochemical Parameter between Diabetic and Non-Diabetic Coronary Heart Disease Subject with Healthy Controls

| Parameters              | Controls (n=91) | CHD patient (n=91) | p-Value | CHD Patients with Diabetes (Mean ± SD) | p-Value |
|-------------------------|----------------|--------------------|---------|---------------------------------------|---------|
| FBG (mg/dl)             | 90.24±4.18     | 94.29±6.98         | 0.1249  | 169.98±66.28                          | <0.0001*** |
| Total cholesterol (mg/dl)| 168.8±16.3     | 218±41.42          | <0.0001*** | 235.73±40.33                          | <0.0001*** |
| Triglyceride (mg/dl)    | 84.6±30.5      | 159.7±69           | <0.0001*** | 178.86±90.08                          | <0.0001*** |
| HDL-C (mg/dl)           | 46±9           | 34±7               | <0.0001*** | 37.83±4.25                            | <0.0001*** |
| LDL-C (mg/dl)           | 106.4±12.59    | 159.4±11.46        | <0.0001*** | 164.64±27.32                          | <0.0001*** |
| VLDL (mg/dl)            | 17.26±8.77     | 28.06±12.14        | <0.0001*** | 34.08±14.29                           | <0.0001*** |
| TC/HDL-C Ratio          | 3.71±0.70      | 6.17±1.14          | <0.0001*** | 6.50±1.36                             | <0.0001*** |
| LDL/HDL Ratio           | 2.35±0.53      | 4.22±0.75          | <0.0001*** | 4.41±0.90                             | <0.0001*** |
| Ox-LDL (U/L)            | 16.73±3.55     | 41.53±8.72         | <0.0001*** | 42.82±10.03                           | <0.0001*** |
| Ox-LDL/LDL-C Ratio      | 0.15±0.016     | 0.25±0.012         | <0.0001*** | 0.25±0.015                            | <0.0001*** |
| Ox-LDL/HDL-C Ratio      | 0.37±0.11      | 1.12±0.29          | <0.0001*** | 1.14±0.30                             | <0.0001*** |
| HbA1c (%)               | 4.9±0.17       | 5.21±0.28          | <0.0001*** | 8.49±2.32                             | <0.0001*** |

Values are expressed in Mean ± Standard Deviation.

FBG- Fasting Blood Glucose, TG- Triglyceride, TC- Total Cholesterol, HDL-C- High-density lipoprotein, LDL-C- Low-density lipoprotein, VLDL-C- Very low-density lipoprotein, Lp(a)- Lipoprotein (a) and HbA1c- Hemoglobin A1c

*P value < 0.05 is considered significant; NS-Not significant; ***Very highly significant ; **Highly Significant.
The study shows FBG, Total cholesterol, Triglyceride, LDL-C, VLDL-C, LDL/HDL ratio, Total Cholesterol/HDL ratio, HbA1c are significantly elevated in these patients compared to control depicted in the Tables 2 and 3. The mean levels of HDL-C levels did not differ significantly among the two groups in diabetic and non-diabetic subject with CHD. In non-diabetic CHD subjects, the mean level of ox-LDL (41.53±8.72) and HbA1c (5.21±0.28) values show a statistically significant increase when compared to controls (P < 0.001). In a diabetic subject with CHD, the mean level of ox-LDL (42.82±10.03) and HbA1c (8.49±2.32) values were significantly elevated when compared (4.9±0.17) % with controls (P < 0.001) Table 2 and Figure 2.

Pearson correlations analysis between ox-LDL [41.53±8.72] and HbA1c with other biochemical parameters in subjects with Non-Diabetic CHD

| Variables                  | ox-LDL       | P-Value       |
|----------------------------|--------------|---------------|
| BMI                        | 0.111        | <0.0001***    |
| Waist circumference        | 0.035        | <0.0001***    |
| Waist Hip ratio            | 0.296        | <0.0001***    |
| FBG                        | -0.106b      | <0.0001***    |
| Total cholesterol          | 0.920        | <0.0001***    |
| Triglyceride               | 0.145        | <0.0001***    |
| HDL-C                      | -0.148b      | <0.0001***    |
| LDL-C                      | 0.996        | <0.0001***    |
| VLDL-C                     | 0.144        | <0.0001***    |
| Cardiac Risk Ratio - I     | 0.634        | <0.0001***    |
| Cardiac Risk Ratio - II    | 0.743        | <0.0001***    |
| HbA1c                      | -0.030b      | <0.0001***    |

a - Positive Correlation
b - Negative Correlation

(p<0.05) as compared to controls as depicted in Table 1 and Figure 1.

The Pearson correlations analysis between ox-LDL [42.82±10.03] with other biochemical parameters in subjects with Diabetic CHD

| Variables                  | ox-LDL       | P-Value       |
|----------------------------|--------------|---------------|
| BMI                        | 0.037        | <0.0001***    |
| Waist circumference        | -0.051b      | <0.0001***    |
| Waist Hip ratio            | 0.073        | <0.0001***    |
| FBG                        | 0.208        | <0.0001***    |
| Total cholesterol          | 0.797        | <0.0001***    |
| Triglyceride               | 0.018        | <0.0001***    |
| HDL-C                      | -0.046b      | <0.0001***    |
| LDL-C                      | 0.995        | <0.0001***    |
| VLDL-C                     | 0.110        | <0.0001***    |
| Cardiac risk ratio - I     | 0.646        | <0.0001***    |
| Cardiac risk ratio - II    | 0.805        | <0.0001***    |
| HbA1c                      | 0.246        | <0.0001***    |

a - Positive Correlation
b - Negative Correlation
Pearson correlations analysis between ox-LDL [42.82 ±10.03] with other biochemical parameters in subjects with Diabetic CHD

ox-LDL positively correlated with BMI (r = 0.037), Waist Hip Ratio (r = 0.073), FBG (r = 0.208), Total Cholesterol (r = 0.797), Triglyceride (r = 0.018), LDL-C (r = 0.995), VLDL-C (r = 0.110), Cardiac Risk Ratio-I (r= 0.646, p=0.0001), Cardiac Risk Ratio-II (r= 0.805, p=0.0001) and HbA1c (r = 0.246). And ox-LDL negatively correlated with Waist Circumference (r = -0.051), HDL-C (r = -0.046) Table 4.

DISCUSSION

The risk of cardiovascular disease is more in CHD subject associated with increased ox-LDL along with elevated LDL-C level compared to normal healthy individuals. This is enlightened by various relations like the integrity of intimal wall impairment along with lipid and lipoprotein concentrations rearrangements (Singh et al., 2016). The present study gives more information on the role of ox-LDL in the progression of CHD. An elevated level of ox-LDL suggests the augmented inflammatory events in patients who are positively correlated with the progression of CHD.

In our study ox-LDL levels was elevated in both Non-diabetic and Diabetic subject with CHD. And a significant difference in Total Cholesterol, Triglyceride, HDL-C and LDL-C was observed. However, the plasma level of LDL-C cholesterol was significantly higher in CHD patients compared to controls an essential risk factor in the progression of atherosclerosis.

Oxidation of LDL occur in two main stages. In the early stages oxidation of LDL in invitro, show a lack of changes in apolipoprotein B100 which undergo oxidative modifications of LDL. Such altered LDL-C is called minimally oxidized LDL. In oxidized LDL lipids present in it are cytotoxic and pro-apoptotic, but not in the mildly oxidized LDL. It stimulate the smooth muscle cells proliferation contribute to persist macrophage foam cells by a PI3 kinase/Akt-dependent mechanisms. For the development and progression of the atherosclerotic lesion, living macrophages play an essential role, loss of macrophage in the initial phase of atherosclerosis can contribute to reducing the burden of macrophage and progression of the slow lesion. In contrast, macrophage death in the late phase of the atherosclerotic lesion may cause necrotic core formation of lesions, leading to the raise of plaque rupture (Yoshida et al., 2005).

In our study, we observed a negative correlation between oxidized LDL and HDL-C. HDL-C cholesterol levels are conversely related to the risk of CHD because it prohibits atherosclerosis by degenerating the oxidized LDL stimulatory effect on monocyte aggregation (Ali et al., 2012). A study conducted on middle age group found a negative correlation between oxidized LDL and HDL-C (Sigurdardottir et al., 2002). This is due to the inverse antioxidant action of HDL components. Therefore ox-LDL is shown to be the strongest predictor of future CHD. LDL oxidative modification plays a crucial role in the progression of atherosclerosis.

On the other hand, inconsistent finding have been reported on the mechanism by which LDL oxidation contributes to atherogenesis in premature stage of the disease. Glycation of LDL cholesterol may facilitate oxidative modification added by advanced glycosylated end products can induce the production of ox-LDL from macrophage (Levitan et al., 2010). It happens by endothelial cells due to overproduction of reactive oxygen species, by oxidative modifications play crucial in clinical aspects of coronary heart disease such as dysfunction in endothelium and plaque disruption (Mundi et al., 2018).

Subjects with high LDL-C, also showed a significant increase in ox-LDL compared to controls. In the pathophysiology of atherosclerosis Oxidation of LDL-C plays a vital role. There is no conclusive proof associated with the protective effects of antioxidant therapy to avoid damage caused by vital molecules such as lipid, protein and DNA. (Linton et al., 2019). In our study we found a positive correlation between ox-LDL with FBG in diabetic CHD subject due to hyperglycemia-induced oxidative stress. In the pathogenesis of diabetic vascular problems, the increased production of ox-LDL and endothelial dysfunction are essential aspects.

Hyperlipidemia, i.e. endothelial dysfunction caused by oxidized LDL through eNOS uncoupling, which results in the production of increased superoxide anions (O2·−). Peroxynitrite anion (ONOO−) is formed by the superoxide when it reacts with NO. Peroxynitrite anion is highly reactive and cytotoxic, which induces peroxidation of lipid and cause endothelial dysfunction (Sukhovershin et al., 2015) by reactive oxygen species formation. Cytotoxic oxidant peroxynitrite formed when R.O.S reduces the NO production. Reduced NO availability promote the progression of atherosclerosis (Honing et al., 1998).

In diabetic subjects Increased production of superoxide with hypertriglyceridemia produce higher levels of oxygen radical by which diabetes undergo oxidation of LDL. Decreased serum NO levels due to
Increase in Blood Pressure are the major severities of diabetes. Tessari et al. In type 2 diabetes mellitus production of NO from arginine are decreased. (Tessari et al., 2010). Weide et al. stated that diabetes patients with HbA1c > 9% show decreased levels of NO associated with improvement of diabetic vascular problems (Aerdoso et al., 2014).

In our study, high Glucose and HbA1c levels were poorly controlled in all diabetic patients. The positive correlation between LDL-C oxidation and HbA1c were reliable with persistent hyperglycemia which might increase LDL oxidation. Previous studies confirm the role of LDL glycation is increased in invitro oxidation. In our diabetic patients, poor glycemic control may be the reason for the elevated LDL oxidation sensitivity. Due to increased oxidative stress, plasma LDL and ox-LDL in patients were elevated and accompanied with the severity of CHD. Increase in ox-LDL observed in our study is a predictor of sub-clinical and clinical atherosclerosis, suggesting that monitoring ox-LDL level in the initial stage may inhibit the development of atherosclerotic problems.

Holvoet et al. stated that a higher level of circulating ox-LDL was seen in CHD patients compared to controls (Holvoet et al., 2001). Galvan et al. found that oxidative processes increase in CHD cause elevation in the serum Ox-LDL level (Quiñones-Galvan et al., 1999).

And more than a few studies concentrated on tactics for lowering the plasma ox-LDL. Ndrepepa et al., found the patients who received statins had lesser levels of circulating ox-LDL and less severe CHD compared to the patients with CHD who did not receive statins (Ndrepepa et al., 2005). The treatment for decreasing circulating ox-LDL levels remains challenging. ox-LDL and oxidation LDL ratio (ox-LDL/T.C., ox-LDL/HDL-C and ox-LDL/LDL-C) are closely related to coronary heart disease are observed in our study. Our observation suggest that ox-LDL and its ratio may be an early marker of oxidative stress in CHD subjects (Yoshimoto et al., 2011).

CONCLUSIONS

The study concludes that the oxidation of LDL-C plays a pivotal role in atherosclerosis. LDL oxidation act as a direct contributor to atherogenesis. The patients with diabetes had increased sensitivity to oxidation of LDL due to poor glycemic control when compared to the non-diabetic subject with CHD. For diminishing oxidative stress and atherogenicity of LDL in diabetic patients, the enhancement of glycemic control is a valuable feature. Oxidative LDL susceptibility is increased with established cardiovascular threat aspects such as smoking, diabetes, hypercholesterolemia etc. Therefore consistent diet, the progress of glycemic control and intake of antioxidant in a diabetic patient could maintain antioxidant defence and reduce oxidative stress.

Conflict of interest

The authors declare that they have no conflict of interest for this study.

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