Tikrit Journal of Pure Science 22 (2) 2017 *ISSN: 1813 – 1662 (Print)*
E-ISSN: 2415 – 1726 (On Line)

Oxidized LDL and Risk of Coronary Heart Disease

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Abstract
Oxidized LDL cholesterol (ox-LDL) is considered to be a key factor of initiating and accelerating coronary heart disease CHD and diabetes Mellitus DM , is associated with several mechanisms one of them Oxidative stress. The aim of this study was to find the association between ox- LDL ,HDL-C , TG , LDL-C and arylesterase , with CHD risk in different age groups in males and females , the difference in these risk factors can explain the sex difference and how much the changes in the risk factors levels in CHD and CHD with DM patients between age groups.
Methods: determination biochemical parameters of serum blood of ox-LDL , TG , TC , HDL-c , LDL-c , S. Glucose and arylesterase activity were measured in cases male and females were carried CHD and CHD with DM .The cases divided in different the age groups (40-49) , (50-59) and (60-69) years and compered the result with control groups .
Results: mean Value of ox-LDL and oxidation ratio of LDL show significantly higher at ($p<0.05$) in CHD and ($p<0.001$) in CHD with DM patient for females in the age group (40-49) years but in the males show the results significant decrease and different significant in others groups ,beside to effect Sex and age some the studied biochemistry parameters levels and signification decrease in HDL-C and arylesterase levels as antioxidant enzymes in females and male patient selected compared with control groups.

Key words: Oxidized LDL, Atherosclerosis ,Diabetes Mellitus ,Coronary heart disease

Abbreviation list:
CHD: Coronary heart disease, DM: Diabetes Mellitus, HDL: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, TC: Total cholesterol, TG: Triglycerides, OX-LDL: Oxidized low density lipoprotein cholesterol .

Introduction
Despite the involvement of oxidized LDL (ox-LDL) [1] in all stages of atherosclerosis from the initiation of fatty streaks to the development of plaque instability and rupture, the potential association of systemically measured ox-LDL with coronary heart disease (CHD) is still a matter of controversy [2],[3]. ox-LDL has been hypothesized to induce foam cell formation, an early yet critical step in the development of atherosclerosis [2]. Furthermore, ox-LDL down regulates endothelial nitric oxide synthase, increases the formation of metalloproteinase, and induces apoptosis in human coronary endothelial cells [1],[5]. Nevertheless, the data from epidemiologic studies remain controversial Although several studies that investigated the association between circulating levels of oxidative biomarkers and CHD yielded fairly strong associations, others were not able to demonstrate any meaningful relationship with CHD. We sought to further elucidate whether ox-LDL is a predictor of incident CHD in a large prospective population-based cohort study of middle aged men and women[6].
Oxidative modification of low density lipoprotein (LDL) in the arterial wall is central to the pathogenesis of atherosclerosis. LDL oxidation has been shown to be inhibited by HDL in vitro, In co-culture HDL prevents the production of mildly oxidized LDL by artery wall cells[7]. This protective effect of HDL relates to enzymes associated with it. Paraoxonase [8], platelet activating factor acetyl hydrolase[9] and lecithin: cholesterol acyl transferase [10] are the HDL associated enzymes that retard the oxidation of LDL by preventing the generation of lipid peroxides.[7] Furthermore, logistic regression analysis revealed that the predictive value of ox-LDL was additive to that of the global risk assessment score for cardiovascular risk prediction that is based on Framingham risk factors: age, total cholesterol, HDL cholesterol, systolic blood pressure, diabetes mellitus, and smoking [11]. Atherosclerosis, a major degenerative disease of arteries involves a series of inflammatory and oxidative modification within the atrial wall(12) Atherosclerosis typically being with endotheial injury followed by low density lioprotien (LDL)oxidation and accumulation with vascular cell ,triggering the pro-inflammatory cascade (inter leukin (IL)-1,IL-6 and tumor necrosis factor (TNF)-a) and the subsequent proliferation of smooth muscle cell, Through this complex process, a sequence of events, including foam cell formation followed by fibrous cap and thrombus formation in the advanced plaque occurs leading to cardiovascular diseases, such as coronary heart diseases (angina, myocardial infarction, stroke, and peripheral vascular diseases [13] emerging researches shows that obesity, hypertension, diabetes mellitus, dyslipidemia , smoking, aging, diets rich in saturated fats, and reduced activity are the established risk factors for atherosclerosis[14],[15].
Oxidative stress is a state in which oxidation exceeds the antioxidant systems in the body, it is arises from an imbalance between the production of reactive oxygen species (ROS) and the body's antioxidant defenses against them, which intensifies cellular damage. The antioxidant defenses enable the body system to remove ROS, restore the prevailing

reducing environment and repair the tissue damage [16]. Oxidative stress plays an important role in the etiology and pathogenesis of many chronic diseases such as atherosclerosis, hypertension cancers [17] and diabetes mellitus which is a chronic metabolic condition leading to microvascular and macrovascular complications resulting in considerable morbidity and mortality, Patients with diabetes are at a two to four-fold increased risk of cardiovascular disease (CVD). However, the mechanism that predisposes these patients to increased risk of CVD is poorly understood. Inflammatory processes have been increasingly recognized to play a role in pathogenesis of both diabetes and heart disease, and may offer a biological link between the two diseases. Various circulating markers of inflammation have been extensively evaluated for their role as risk predictors of cardiovascular disease, Dietary intake of antioxidants can inhibit or delay the oxidation of susceptible cellular substrates so prevent oxidative stress.[18].

The Aim of study:

The present study was the effect of levels (ox-LDL, TG, TC, HDL-C, LDL-C, VLDL-C, S. Glucose, Arylesterase activity) in different age groups with development of heart disease ((CHD) and (CHD with DM)) for females and males .

Material and methods

Taking 163(93 samples from patient (female) and 70 (male)) who attend Rizgary Teaching Hospital, the province of Arbil, Iraq,between November 2014-march 2015 and the intensive care cardiac samples were divided into two section first to those who suffer from heart disease and blood vessels, other part with one heart and cardiovascular diseases, as well as type II diabetes from both disease and gender (male and female) than they were aged between 40-70 years. were divided in the age groups (40-49) , (50-59) and (60-69) years

Control group: 137 samples taken from healthy people with the same age of the patients group.

Blood collection and biochemistry analysis :

Blood samples (5ml) were collected before initiating the treatment in patients. fasting blood sample were used to analyze (total cholesterol, HDL-C , LDL-C , VLDL-C and triglycerides, S.glucose) commercial diagnostic kits from franc (BDH) company . serum sample collected to estimate OX-LDL and frozen at (-80) °C then were analyzed within one month the kit for estimating ox-LDL, the kit was obtained from MyBiosource company ,U.S.A. depended on ELISA. And Arylesterase was assayed by using 4 mM phenyl acetate as a substrate depending on (Allwsh and Jasim) method [19].

Statistical analysis :

All data have presented as mean ±SD. (One-way analysis of variance) (ANOVA) was performed on each variable and the Bonferroni statistics employed to compare the mean values from the different groups .un paired T-test was used to assess the effect between groups. differences were considered significant at ($P<0.05$,$P<0.01$,$P<0.001$), All statistical analysis were performed using SPSS statistical software (version 19) .

Results and Discussion

In this study, the patients were examined as a part of population based survey for lipid profile in Irbil city , serum lipid profile was measured for all subjects following 12 hours fasting . this profile includes TC,TG,HDL-C,LDL-C, ox-LDL, ox-LDL/TC, ox-LDL/HDL, ox-LDL/LDL-C , Arylesterase (U/mmol), S-Glucose. The results are presented in conventional units of mmol/l for control (female and males) in two groups were not different because the control were age and sex matched to cases .

A total of (93 females and 70 male) as patients individuals participated in the study the clinical and biochemical characteristics of the study population are show in the table(1), the age group (40-49) year show higher significantly in (ox-LDL; ox-LDL/TC; ox-LDL/HDL-C and ox-LDL/LDL-C) at($p<0.05$) for patients with CHD and significant increased at ($p<0.001$) for (ox-LDL; ox-LDL/LDL-C; ox-LDL/HDL-C) and ox-LDL at ($p<0.01$) for patients with (CHD+DM) when compared with control group. The age group (50-59) years show signification increase at ($P<0.05$) for (ox-LDL; ox-LDL/TC; ox-LDL/HDL-C) for CHD patients. in addition the high significantly at ($P<0.05$) for females with (CHD with DM) in (ox-LDL; ox-LDL/TC and ox-LDL/HDL-C) at($p<0.01$), wherever there was a significant decreases at ($p<0.05$) for ox-LDL/LDL-C for female with CHD and (CHD+DM) group where compare with control group for the same age .

The (60-69) years group show increase significantly at ($P<0.01$) for ox-LDL, and ox-LDL/HDL-C at ($p<0.05$) for females with (CHD) higher signification increases at ($p<0.001$) for females with (CHD with DM) for (ox-LDL; ox-LDL/TC and ox-LDL/HDL-C), wherever no signification difference in (ox-LDL/TC; ox-LDL/LDL-C) for females with (CHD) when compared with control and no signification in ox-LDL/LDL for female with (CHD+DM) when compared with control. the results shows for females in age group with (CHD) higher signification at ($p<0.05$) for ox-LDL and ox-LDL/TC and ox-LDL/HDL-C where compared with healthy females as a control group.

Table(1) oxidized low density lipoprotein in females (Coronary heart disease and Diabetes type2)

Age year	Patients	ox-LDL (mmol/l)	ox-LDL /TC	ox-LDL /HDL-C	ox-LDL/LDL-C
40-49	CHD N=10	40.64±6.71 *	7.37±0.12 *	44.65±8.71 *	10.66±0.88 *
	CHD+DM N=13	47.09±9.03 ***	7.69±1.04 **	57.42±5.31 ***	11.92±2.12 ***
	control (N=30)	24.01±9.16	5.152±0.98	22.45±0.008	8.365±2.12
50-59	CHD N=18	45.091±9.11 **	8.07±0.81 **	62.89±10.09 **	11.75±1.27 **
	CHD+DM N=17	49.22±10.31 **	8.189±1.05 **	60.67±12.31 *	11.74±1.56 **
	control (N=22)	30.16±5.31	6.27±0.69	29.86±5.059	13.771±1.50
60-69	CHD N=20	55.19±7.91 **	9.306±2.03	66.49±8.71 *	12.51±1.47
	CHD+DM N=15	57.44±11.07 ***	11.08±1.56 ***	74.59±13.11 ***	12.35±2.06
	control (N=15)	38.4±8.72	8.30±0.02	38.4±6.67	13.24±2.28
All	CHD	47.33±10.11 *	8.549±1.033 *	58.72±7.13 *	11.74±1.18
	CHD+DM	54.31±12.11 ***	9.03±0.97 ***	61.71±6.09 ***	12.48±1.25 *
	control	30.87±6.06	6.63±0.99	30.22±2.11	11.80±0.166

P*<0.05 ,P**<0.01 , P***<0.001 (Compared females with control for same age)

Recently we show high signification at($p<0.001$) for all groups for different ages groups for example (CHD+DM) ox-LDL; ox-LDL/TC and ox-LDL/HDL compared with control, and signification increase at ($p<0.05$) in ox-LDL/LDL-C when compared with control group.

This results for oxidized low density lipoprotein may be due to the Lp-PLA_2 enzyme (lipoprotein associated phospholipase A2) activity is an important risk predictors for CHD, ox-LDL correlated positively with activity of Lp-PLA_2 in CHD because the ox-LDL being substrate of Lp-PLA_2 may be affects the activity of this enzyme [18] Lp-PLA_2 hydrolyses oxidized phospholipid of OX-LDL to produce lysophosphatidycholine (LysoPC) and oxidized non esterified fatty acid (OXNEFA) [20]. the two products are prion flammatory and a therogenic and are important contributors to the risk of CHD. In addition, a positive correlation was observed between enzyme activity and fasting glucose in diabetes group, the hyperglycemia may be effected the activity of the Lp-PLA_2 which is increase in ox-LDL levels [18] The enzyme activities which have an important role in lipoproteins synthesis such as cholesterol acyl. COA Carboxylase and reduced the activity of cholesterol Acyltransferase and lecithin and inhibiting the activity of lipoprotein lipase and liver lipase activity that led to increased synthesis of vL DLc and decreased the concentration of HDL-C [21].

In this study found a strong significant effect between ox-LDL and total cholesterol ratio and known that HDL levels are associated with age in women, HDL-cholesterol levels increase progressively to the fifth decade and then decrease with menopause, the mechanism may influence the data on HDL-C concentration [22]. One of the more prominent markers of oxidative stress in DM is ox-LDL ; increased production of free oxygen radicals, higher ox-LDL along with stimulation of ox-LDL uptake by macrophages and the ensuing promotion of early atherosclerotic changes might be contributed by insulin resistance (IR);hyperglycemia is associated by increased inflammatory burden and increased lipid peroxidation ,all leading to inhanced macrophage foam cell formation , low density lipoprotein (LDL) oxidation by macrophages was increased due to the activation of several pro-oxidant systems as well as the depletion of antioxidants [23] ox-LDL and oxidation ratio of LDL (ox-LDL/TC, ox-LDL/HDL-C, and ox-LDL/LDL-C) are closely related with CHD and they are better biomarkers for discrimination between patients with coronary heart disease and healthy[24].

Table(2) Biochemical variables in females (Coronary heart disease & Diabetes type2)

Age year	Patients	TG mmol/l	TC mmol/l	HDL-C mmol/l	LDL-C mmol/l	HDL-C/TC	HDL-C /LDL-C	Aryl-esterase U/mmol	S.Glucose mmol/l
40-49	CHD N=10	2.55±1.93 *	5.5± 0.31	0.91±0.031	3.51± 1.10	0.165±0.031	0.259±0.031 *	50.31± 9.91 *	5.95±1.21
	CHD+DM N=13	2.91±0.11 **	6.12±0.92 **	0.82±0.031 *	3.75± 0.95 *	0.133±0.021 *	0.218±0.021 **	46.93± 8.17 **	7.71±0.01 ***
	control N=30	1.8± 0.74	4.66±0.92	1.09± 0.28	2.87± 0.77	0.23± 0.03	0.379± 0.021	68.92± 4.31	5.51± 0.38
50-59	CHD N=18	3.04±0.44 **	5.81±0.92 *	0.78± 0.07 *	3.99± 0.91 *	0.134±0.071	0.195± 0.08 **	42.71±12.01 *	5.85±1.13 *
	CHD+DM N=17	3.33±0.05 ***	6.01±1.21 **	0.81± 0.07 **	4.19± 0.96 ***	0.134±0.041	0.193± 0.04 **	33.41±12.44 **	7.31±1.35 ***
	control N=22	2.036±0.78	4.81±0.91	1.106±0.257	2.91± 0.87	0.229± 0.0289	0.380± 0.0295	59.87± 12.1	4.59±0.25
60-69	CHD N=20	4.32±1.05 **	5.93±1.21 **	0.83±0.044	4.41± 0.91 *	0.1399±0.03 *	0.188± 0.06 *	55.41±11.31	6.70±1.12 *
	CHD+DM N=15	4.53±0.88 ***	5.18±1.03	0.77±0.031 *	4.65± 0.78 **	0.148±0.008	0.165±0.016 **	42.32±10.21 **	7.71±2.26 **
	control N=15	1.851±0.85	4.622± 1.22	1.0± 0.233	2.90±1.191	0.216± 0.09	0.344±0.019	55.42± 9.91	5.98±0.07
All	CHD	3.96±0.79 ***	5.77±1.31 *	0.84± 0.04 **	4.20± 0.92 *	0.145±0.044 *	0.2± 0.033 *	52.33± 8.72 *	6.12±1.11
	CHD+DM	3.25±0.21 *	6.01±1.21 **	0.88±0.025 *	4.35±0.871 **	0.146±0.071 *	0.202±0.047 *	43.01± 7.01 ***	7.31±2.71 **
	control	1.863±0.78	4.659±0.90	1.096±0.252	2.836±0.83	0.235±0.028	0.386± 0.03	61.403± 9.21	5.36± 1.13

P*<0.05 ,P**<0.01 , P***<0.001 (Compared females with control for same age)

The results in table(2) show signification for females the age group (40-49) years which increase signification at ($p<0.05$) for TG and decrease signification at ($p<0.05$) for (HDL/LDL), Arylesterase and no signification in (TC,HDL-C; LDL;HDL/TC; S.Glucose) when compare with control, for females (CHD) and the results show for females (CHD with DM) high significantly increases at($p<0.01$) for (TG,TC) and (S.Glucose) at($p<0.001$), and decrease signification at ($p<0.05$) in HDL-C,HDL-C/TC and HDL-C/LDL-C, Arylesterase at($p<0.01$) when compared with control group.

On the other hand the age group (50-59) years show high signification increase at ($p<0.05$) in level of LDL-C, S.Glucose, TC, and TG at($p<0.05$), while there was a decrease signification at ($p<0.01$) for HDL-C/LDL-C and (HDL-C; Arylesterase) at ($p<0.01$) for females with (CHD) compared with control group. For females with (CHD and DM) show increase signification at ($p<0.001$) in (TC, LDL-C, S.Glucose) as well as TG at ($p<0.01$) and decrease signification at ($p<0.01$) in HDL-C, as well as decrease signification in HDL-C/LDL and Arylesterase when compared with control group.

The third age group (60-69) years the female with (CHD) show high significantly increases at ($p<0.01$) in TG, LDL-C, TC and S.Glucose at ($p<0.05$), but decrease signification at ($p<0.05$) in HDL-C/TC, HDL-C/LDL-C, arylesterase , while no signification in HDL-C level. For females with (CHD and DM) show high signification at ($p<0.01$) in S.Glucose while signification at ($p<0.001$) for TG, LDL-C, but signification decrease at ($p<0.05$) for HDL-C , HDL-C/ LDL-C, arylesterase. while no signification in TC and HDL-C/TC levels.

The total the age groups the results show signification increase at ($p<0.001$) in TC, LDL-C, TG levels at ($p<0.05$), but there was significant decrease at($p<0.05$) in arylesterase, HDL-C, HDL-C/LDL-C and HDL-C/TC while no signification in S.Glucose for females with CHD, The females with (CHD and DM) shows the results significant increase at($p<0.05$) in TG, LDL-C($p<0.01$), S.Glucose and TC, while decrease significant at($p<0.05$) in HDL-C, HDL-C/LDL-C,and arylesterase, but no significant in HDL-C/TC level when compare with healthy women.

The results in table (2) shows increased in TG, TC,HDL-C/LDL-C levels . lipid abnormalities, high blood pressure, and smoking are major risk factors for coronary heart disease . obesity and diabetes also contribute to CHD risk, the role of major cardiovascular risk factors in the development of CHD[25], male and female phenotypes are developed through the action of sex hormones, Among women storgen is the predominant sex hormone, the decrease in estrogen production after menopause changes the females lipid metabolism toward a more atherogenic form by decreasing the HDL cholesterol level and by increasing LDL-C and total cholesterol, triglyceride and lipoprotein (a) levels [26],[27]. In addition to the lipid effect, estrogen may have cardio protective effects through glucose metabolism and the hemostatic system, and it may be also have direct effect on endothelial cell function [25] .Some of studies show the strong association between high TG to HDL molar ratios and high levels of circulating ox-LDL, even after adjustment for LDL cholesterol

levels , thus suggests astrong association between the small, dense LDL phenotype and oxidation of LDL [11] .

The characteristic dyslipidemia in metabolic syndrome which favors atherogenesis is an elevated vLDL, lowered HDL and qualitatively altered LDL which becomes small and dense which is more readily oxidized and more atherogenic, bing non recognized by LDL receptors but taken up by macrophages which intern become the foam cell, the increased free fatty acid fluxes to the liver resulting in increased synthesis of vLDL because of hepatic insulin resistance even postprandial vLDL synthesis , its clearance too is impaired because of impaired lipoprotein lipas activity due to the insulin resistance , normal activity of cholesterol ester transport protein results in transfer of cholesterol esters between vLDL-C, LDL-C and HDL-C. thus vldl-C becomes cholesterol rich and LDL-C and HDL-C becomes rich in triglycerides then they reach liver, lose the triglyceride merely by the action of hepatic lipase, whose activity is normal despite insulin resistance , thus converting intor small dense LDL small dense HDL which are poor cholesterol scavengers because of inpaired reversed cholesterol transforms [28]. Diabetes is associated with an increased risk of cardiovascular death and higher incidence of CHD [29]. One of the features of diabetes is abdominal obesity and visceral adipose tissue plays an importance role in the progress of diabetes [28]. in addition to change in sex hormone levels with aging or illness may lead to metabolic disorders. more ever, the ratio changes in men versus women may have distinct pathological responses [30] .

The results show decrease in arylesterase level, arylesterase can in turn hydrolyze lipid peroxides in ox-LDL and convert them to a less atherogenic LDL-C thus causing further reduction in ox-LDL content [16] . Arylesterase is a calcium dependent HDL associated ester hydrolase that catalyese the hydrolysis of organophosphates , aromatic carboxylic acid esters and carbamates purified human HDL associated arylesterase retards the oxidation of LDL by preventing the generation of lipid peroxides [7]. The reason for decrease arylesteras activity enzyme may be due to decreased ability of HDL to protect erythrocyte membranes could be related to lipid composition of HDL and to this low arylesterase enzyme activity, and diabetic is associated with oxidative damage, the higher lipid peroxidation products in diabetic patients are ascribed to higher susceptibility of plasma lipoproteins to oxidation and decrease of antioxidant defenses [31]. Oxidative-imbulance has been implicated in the etiology various disorders, including cancers, renal disease, parkinson's disease and diabetes mellitus. arylesterase activity has been shown to be modulated by lifestyle and dietary factors such as short – term caloric restriction , same studies suggests the increase in the susceptibility of low–density lipoprotein for oxidation is due to the decrease in serum arylesterase activity with age [32].

Table(3) oxidized low density lipoprotein in males (coronary heart disease& Diabetes type2)

Age (year)	Patients	ox-LDLmmol/l	ox-LDL/TC	ox-LDL/HDL-C	ox-LDL/LDL-C
40-49	CHD N=10	45.81±4.21 ++	7.691±0.159 ++	53.26±4.47 +++	13.05±1.08 ++
	CHD+DM N=12	46.33±2.01 ++	7.55±0.911 +++	61.691±1.97 +++	11.789±2.41 +
	control (N=25)	26.33±2.05	13.21±2.30	25.81±2.51	8.955±1.02
50-59	CHD N=11	29.11±5.31 +	4.892±1.052 ++	42.189±6.91 ++	6.60±3.87 ++
	CHD+DM N=14	30.41±2.66 +	4.606±1.681 ++	39.966±5.42 ++	6.83±3.591 +
	control (N=30)	33.11±9.12	1.413±0.81	26.02±2.87	11.223±2.7
60-69	CHD N=13	38.03±2.31 +	6.76±2.316 +	54.25±8.031 ++	8.335±1.351 ++
	CHD+DM N=10	40.01±4.61 ++	6.941±1.44 +	58.715±12.817 +++	6.697±2.571 +++
	control (N=15)	39.77±7.41	8.707±0.53	39.02±5.61	14.95±2.65
All	CHD	37.97±4.24 ++	6.154±1.709 +	50.553±8.831 ++	9.127±1.031 +
	CHD+DM	38.97±3.03 +	5.985±1.711 ++	54.12±11.351 +++	8.113±2.98 +
	control	32.66±7.16	7.11±1.01	30.28±3.41	11.69±1.21

$P^{+}<0.05$,$P^{++}<0.01$, $P^{+++}<0.001$ (Compared between males with control for same age)

The results for males (CHD and CHD+DM) table (3) for oxidized low density lipoprotein for age group (40-49) years male with (CHD) significant increase at ($p<0.01$) for ox-LDL, ox-LDL/HDL-C($p<0.001$) and ox-LDL/LDL-C. but significant decrease at ($p<0.01$)for ox-LDL/TC, the male with (CHD & DM) the results show significant increase ($p<0.01$) for ox-LDL, ox-LDL/TC, ox-LDL/HDL-C and ox-

LDL/LDL-C when compared with control group for the age year (40-49).

The age group (50-59) years with CHD disease the results show significant decrease for ox-LDL at ($p<0.05$), ox-LDL/TC at($p<0.01$), but the significant increase at ($p<0.01$) in ox-LDL/HDL-C, ox-LDL/LDL-C. (CHD with DM) group the results show significant decrease at($p<0.05$) for ox-LDL, ox-LDL/LDL-C and ox-LDL/TC on other side significant increase at($p<0.01$) for ox-LDL /HDL-C when compared with control group for the same age group.

The age group (60-69) years for (CHD) patients the results indicated significant increase at ($p<0.05$) for ox-LDL/TC, ox-LDL/LDL-C while ox-LDL/HDL-C at($p<0.01$), the results show significant decrease in ox-LDL. the (CHD and DM) group the result show significant increase at ($p<0.01$) for ox-LDL and ox-LDL/HDL-C, while significant decrease at($p<0.05$) ox-LDL/LDL-C, and ox-LDL/TC when compared with normal females as a control group .

The results shows for all age groups (CHD) highly significant increases at ($p<0.01$) for ox-LDL, ox-LDL/HDL-C, and significant decrease at($p<0.05$) for ox-LDL/TC, ox-LDL/LDL-C, (CHD with DM) group the result show significant increase ox-LDL at($p<0.01$) and ox-LDL/HDL-C at($p<0.001$) while significant decrease in ox-LDL/LDL-C at ($p<0.05$), ox-LDL/TC at($p<0.01$) when the result compared with control group for the same age .

The results show highly significant for ox-LDL level in patients with (CHD with DM) the reason may be due low density lipoprotein cholesterol (LDL-C) has been confirmed to play an important role in development of CHD, including endothelial cell injury, inflammation, foam cell, formation, and unstable plaque rupture [33].and the NCEP ATPIII (National Cholesterol Education Program Adult Team Panel III) have emphasized that lowering LDL-C is an important target in the prevention and treatment of coronary heart disease some evidence indicate that oxidized low–density lipoprotein cholesterol (ox-LDL) plays a more crucial role in the process of CHD. ox-LDL was increased in patients with coronary heart disease, found that unstable plaques were filled with ox-LDL which elevated levels was positively correlated with acute coronary syndrome, and level of ox-LDL is regarded as an index severity of acute coronary syndrome[33].

The indicate serum ox-LDL level and oxidation ratio of LDL are closely related with CAD, more interesting date form the study revealed that the correlation ship between CAD and relative degree of LDL oxidation is stronger than with the level of ox-LDL in *vivo* [34].

Hyperglycemia may lead to intracellular changes in redox state resulting in depletion of cellular NADpH+H pool, leading to increased tendency for oxidative stress and high level of oxidized lipoprotein especially LDL , in addition the macrophage express scarenger receptors for modified lipoprotein (ox-LDL) permitting themnto ingest lipids and become foam cells , the activated macrophage can generate reactive oxygen species that augment oxidant stress [35].

On the other hand the (50-59) years group the result show decrease in ox-LDL level the reason may be that was HMG-COA(statins) are considered as the frontline medication which can reduce cardiovascular morbidity and mortality in both primary and secondary preventation [36]. By lowring LDL, studies have also shown that statins can slow down progression or even regress coronary CHD, and found that statins also have pleiotropic effect such as antioxidant, anti-inflammatory, and stabilization of plaques which are independent of LDL lowering [37]. As well as the simulation can significantly reduce circulating ox-LDL level in subjects with coronary artery disease (CAD) and that agree with researcher Pasterkamp *et al*. which show the effect statins on reducing level of ox-LDL and oxidation ratio of LDL (ox-LDL/TC, ox-LDL/HDL-C, ox-LDL/LDL-C in long), as well as short – period (within 2 weeks) therapy [33].

From the other side the result indicated decrease in ox-LDL, ox-LDL/TC, ox-LDL/ LDL-C,for male with CHD and DM , the reason may be due to diabetes mellitus is characterized by disturbance in insulin secretion, act of it enzyme or both in which lead to hyperglycemia condition, clinically the glucose – in blood stream cannot move efficiently from vascular in to cell, so that the glucose level in blood stream remain high this will harm the entire organ in the body and certain tissue because of hyperglycemia [38], so ox-LDL can be formed by several causes one of them is through non enzymatic glycation, glycation process can occur in Apo ß as well as LDL-C-phospholipid, advanced glycosylation End products (AGEs) which formed by oxidative stress and hyperglycemia can oxidize LDL-C, glycated LDl-C will bind with scavenger receptor in macrophage which it can be a foam cell later-the foam cell is a fundamental for forming atherosclerotic lesion [39].

There are several method that can be done as a step of palliative therapy for type 2 diabetes mellitus , one of them through inhibition of lipoprotein –associated phospholipase A_2 (Lp-PLA_2) which is very specifically to the inflammation in vascular has low biologically variability, and has a role in atherosclerotic plaque inflammation .new finding suggests that Lp-PLA2 can be substantial risk factor on CHD plaque formation and on it's rupture [40] . one of the drugs which act through selective inhibition of Lp-PLA_2 is Dara pladib which some patents with DM used treatment hyperglycemia, the drug is irreversible inhibitor for Lp-PLA_2 enzyme by formed from two hydrogen bonding interaction with tyr160 and gln 352 side chain and a couple of pi-pi interaction with aromatic and aliphatic hydrophobic remainder of Lp-PLA_2 [41]. as well as the drugs has

the ability as a selective inhibitor for Lp-PLA_2 receptor, by inhibiting the receptor of Lp-PLA_2, it also inhibits the formation of lysophosphatidylcholine (Lyso-pc) and oxidized free fatty acid (ox-FA) which is derived from ox-LDL [42].

The results table(4) show the patients(40-49) years with CHD highly significant increases at ($p<0.05$) for TG, TC, LDL-C and decrease significantly at($p<0.05$) in HDL-C/TC, HDL-C/LDL-C, and Arylesterase but there is no significant difference in S.glucose level . The patient group with (CHD with DM) the results show significant increase at ($p<0.01$) in TG, LDL-C and (TC ,S.glucose) at ($p<0.001$), while a significant decrease at ($p<0.01$) for HDL-C, at ($p<0.05$) HDL-C/TC and arylesterase activity the results compared with healthy males group.

The result of (50-59) years group shows highly significant at($p<0.01$) in (CHD) patients at for TG, S.glucose, TC, LDL-C as well as significant decrease at($p<0.05$) HDL-C, HDL-C/TC and HDL-C/LDL-C, while no significant difference in arylasterase. while (CHD with DM) group show highly significant increases at ($p<0.01$) for TG, LDL-C, TC and S.glucose, while decrease significant at($p<0.05$) in HDL-C, HDL-C/TC and HDL-C/LDL-C, all this result compared with normal group for the age (50-59) year .

From the other side the results show for (60-69) year age group for CHD patients significant increase at ($p<0.01$) for TC,LDL-C, S.glucose and TG at ($p<0.05$), while significant decrease at ($p<0.05$) for HDL-C while no significant difference in HDL-C/TC and arylesterase. the patients of (CHD with DM) group the result show significant increase at ($p<0.01$) for TC,TG, LDL-C and S.glucose, while there was a significant decrease at ($p<0.001$) for HDL/TC, arylesterase as well as HDL-C and HDL-C/LDL-C, as well as no significant difference in arylesterase levels. all this result are compared with healthy male as a control group.

Table (4) Biochemical variables in males (coronary heart disease& Diabetes type2)

Age year	Patients	TG mmol/l	TC mmol/l	HDL-C mmol/l	LDL-C mmol/l	HDL-C/TC	HDL-C /LDL	Arylesterase U/mmol	S.Glucose mmol/l
40-49	CHD N=10	2.61±0.41 +	5.95±0.75 ++	0.86±0.03	3.51±1.03 +	0.144±0.021	0.245±0.011 +	52.33±3.31 +	4.61±1.93
	CHD+DM N=12	3.11±0.61 ++	6.13±1.21 +++	0.751±0.07 +	3.95±0.98 ++	0.122±0.061 +	0.190±0.026 ++	55.36±2.03 +	6.97±0.67 +++
	control N=25	1.924± 0.704	4.715± 0.89	1.02± 0.02	2.942± 0.288	0.216± 0.02	0.346± 0.03	65.91± 8.01	4.41± 0.80
50-59	CHD N=11	3.36±0.15 +	5.95±0.91 ++	0.691±0.061 +	4.41±1.04 ++	0.116±0.057 +	0.156±0.049 ++	49.76±3.45	5.67±0.99 +
	CHD+DM N=14	3.71±1.09 ++	6.61±0.96 +++	0.761±0.031 +	4.45±0.77 ++	0.116±0.012 +	0.171±0.062 ++	45.43±3.44 ++	7.71±1.13 +++
	control N=30	2.09± 0.61	4.84± 0.878	1.072± 0.22	2.958± 0.841	0.221± 0.012	0.362±0.0261	52.71± 5.17	4.98± 0.67
60-69	CHD N=13	3.95±1.06 ++	5.63±0.86 +	0.701±0.04 +	4.56±1.03 +	0.136±0.081	0.153±0.079 ++	40.88±2.91	5.96±0.04
	CHD+DM N=10	3.99±1.33 +++	5.77±1.03 ++	0.661±0.07 +++	5.98±0.31 +++	0.114±0.022 +	0.110±0.53 +++	39.98±3.41 +	7.91±1.41 +++
	control N=15	1.979± 0.674	4.41± 0.83	1.019± 0.214	2.622± 0.786	0.231±0.0257	0.383± 0.027	49.78± 2.03	5.70± 0.51
All	CHD	3.30±0.061 ++	6.176±0.871 ++	0.751±0.051 +	4.163±0.96 ++	0.180±0.034	0.1807±0.04 +	47.656±4.32 +	5.53±1.23
	CHD+DM	3.60±0.33 ++	6.50±1.91 +++	0.724±0.041 +	4.80±1.09 +++	0.111±0.096 +	0.167±0.089 +	32.926±3.41 +++	7.506±1.12 +++
	control	1.983± 0.73	4.596± 0.922	1.045± 0.225	2.779± 0.861	0.227± 0.091	0.376± 0.026	56.13± 8.11	5.03±0.991

$P^{+}<0.05$,$P^{++}<0.01$, $P^{+++}<0.001$ (Compared between males with control for same age)

The patients for all the age groups show result for CHD highly increase significant at($p<0.01$) for TG,TC,LDL-C while significant decrease at ($p<0.05$) for HDL-C,HDL-C/TC, HDL-C/LDL-C and aryl-esterase , the patients of (CHD with DM) the results indicated increase for TG at($p<0.01$) and TC, LDL-C, S.glucose at($p<0.001$), as well as significant decrease for HDL-C ,HDL-C/TC,HDL-C/LDL-C and arylesterase this result compared with control group .

The results shows CHD dyslipidemia is checterized by -3-lipid abnormalities: elevated TG , reduced HDL-C and small LDL particle size ,LDL is heterogeneous in terms of size and density [11] , After the age of 20 years , low density lipoprotein cholesterol (LDL-C) increase significantly in both men and women or is in a flat state between the age of (50-60) years (male) and (60-70) years (female) on the other hand (HDL-C) levels decrease during puberty to young adulthood (in male) throughout their lives women have lower TC compared to men but the levels will rise sharply after menopause and will be higher in the age 60 year as compared to men [43].

Insulin resistance and type 2 diabetes are associated with interrelated plasma lipid and lipoprotein abnormalities, which include reduced HDL

cholesterol, and a predominance of elevated triglyceride levels is associated with an increased risk of cardiovascular disease, HDL-C the anti atherogenic lipoprotein influences the retardation of CHD process and it low levels contribute to CADin Diabetes Mellitus [28] The antioxidant roles to oxidative imbalance has been implicated in the etiology of various disorders , including cancer , renal disease and diabetic mellitus [32], Hyperglycemia generates free radicals , increasing oxidative stress, which is proven to be one of the mechanisms for the development of complication in DM [44] .

The high plasma levels of lipid peroxidation products in diabetic patients are attributed to higher susceptibility of lipoprotein for oxidation, this enzyme has both paraoxonase and arylesterase activity and by virtue of it's hydrolytic action, prevents accumulation of lipid peroxides in LDL, and gives protection against lipoprotein oxidation [45] .

The decrease in arylesterase activity may decrease the antiatherogenic effect of HDL leading to accelerated and CAD in diabetic patients, who have additional proatherogenic dyslipidemias, DM is associated with dyslipidemias due to deficiency and/insulin resistance, there is ineffective lipoprotein lipase activity, increased lipolysis, and increase hapatic VLDL secretion resulting in hyperlipidemia, both micro and macro vascular complication of DM are majorly due to LDL peroxidation, HDL along with (PON) arylesterase activity reduces the formation of ox-LDL, lower HDL level and reduced PON (arylesterase) activity will increase the risk for the development of complication in DM[46]. some studies show that the increase in susceptibity of LDL-C for oxidation is due to the decrease in serum arylesterase activity with age [32].

Conclusions

Results of the present study showed that there were increased ox-LDL level in coronary heart disease and diabetic type 2 and increase in the prevalence of metabolic disease (T_2DM and CHD) in old age may be related directly with age or CHD process itself or in directly through several other-age –related risk factors of T_2DM and CHD such as Free fatty acid (FFA), lipid metabolisms, insulin resistance ,ß-cell dysfunction , inflammation , metabolic syndrome and central obesity finally ox-LDL and oxidation ratio's of LDL, HDL, TC are closely related with CHD and they are better biomarker for discriminating between patients with coronary artery disease and healthy subjects.

References

1. Tsimikas S. (2006) . Oxidative biomarkers in the diagnosis and prognosis of cardiovascular disease .Am J. Cordial , 98:9P-17P.

2.Hansson GK.(2005). Inflammation, atherosclerosis, and coronary artery disease. N Engl J. Med, 352: 1685-95.

3. Navab M. Ananthramaiah GM. Reddy ST. Van Lenten BJ. Ansell BJ. Fonarow GC. Et al. (2004) .The oxidation hypothesis of atherogenesis : the role of oxidized phospholipids and HDL. J. Lipid Res, 45:993-1007.

4. Glass CK. Witztum JL. (2001) . Atherosclerosis . The road ahead . Cell ,104:503-16.

5. Suglyama S. Okada Y. Sukhova GK. Virmani R. Heinecke JW. Libby P. Macrophage myeloperoxidase regulation by granulocyte macrophage colony-stimulation factor in human atherosclerosis and implication in acute coronary syndromes . Am J. Pathol, 158:879-91.

6. Koenig W. Karakas M. Zierer A. Herder C. Baumert J. Meisinger C. and Thorand B. (2011) . Oxidized LDL and the risk of coronary heart disease : Results from the MONICA/KORA Augsburg study . Clinical Chemistry ,57;8:1196-1200.

7. Lakshmy R. Ahmed D. Abraham R. Sharma M. Vemparala K. Das S. Reddy K. and Prabhakaran D. (2010). Paraoxonase gene *Q192R & L55M* polymorphisms in Indians with acute myocardial infraction & association with oxidized low density lipoprotein . Indian J. Med. Res.,131,pp:522-529.

8. Mackness MI. Mackness B. Durrington PN. Fogelman AM. Berliner J. Lusis AJ. *Et al* .(1998) .Paraoxonase and coronary heart disease . Curr Opin Lipidol, 9:319-24.

9. Whatson AD. Navab M. Hama SY. Sevanian A. Prescott SM. Stafforini DM. *et al*. (1995) .Effect of platelet activating factor acetyl hydrolase on the formation and action of minimally oxidized low density lipoprotein . J. Clin. Invest . 95:774-82.

10. Klimov AN. Nikiforova AA. Pleskove VM. Kuzmin AA. Kaleshnikova NN. Antipova TO. (1989).The protective action of high density lipoproteins, their subfraction and lecithin cholesterol acylhydrolase in the peroxide modification of low density lipoprotein. Biokkimiya , 54:118-23.

11. Holvoet P. Harris T. Tracy R. Verhamme P. Newman A. Rubin S. Simonsick E. Colbert L. Kritchevsky S. (2003) .Association of high coronary heart disease risk status with circulating oxidized LDL in the well-functioning elderly. Arterioscler Thromb Vasc. Biol. , 23:1444-1448.

12. Fan J. Watanabe T. (2003a) . Transgenic rabbits as therapeutic protein bioreactors and human disease models . Pharmacol Ther., 99:261-282.

13. Laboz C. Mostaza J.M.(2007). Atherosclerosis as a systemic disease .Rev. Esp. Cardiol, 60:184-195.

14. Wiersma J. Meuwese M. and Van Meret J.(2008). Diabetes mellitus type 2 is associated with higher levels of myeloperoxidase , Med Sci. Monit. , 14:406-4.

15. Singh U. and Jialal I. (2006). Oxidative stress and atherosclerosis. Pathophysiology,13:129-142.

16. Al-Hadidy A. Al-Kattan M. Jankeer M. (2014) . Relationship between pomegranate (Punica granatum) and paraoxonase enzyme to prevent the

development of atherosclerosis in male white new Zealand rabbits . International Journal of Enhanced Research in Science Technology & Engineering .,Vol.3,Issue 5. pp:242-250 .
17. Halliwell B and Gutteridge J. (1999) . Free radicals in biology and medicine , The 3rd Edition by:Oxford University Press, Oxford.
18. Garg S. Suneja M. (2015). Lipoprotein associated phospholipase A2 activity & its correlation with oxidized LDL & glycaemic stutus in early stages of type-2 diabetes mellitus. Indian J. Med. Res. ,pp:107-114.
19. Allwsh T. Jasim R. (2008), Study of arylesterase and it's relationship with some clinical variables in atherosclerotic patients in mosul (Part I) , Al-Rafidain Journal of science ,Vol.19 ,No.2 , pp:143-157.
20. Mac Phee CH, Moores KE, Boyd HF, Dhanak D, Ife RJ , Leach CA, *et al*., (1999) . Lipoprotein – associated phosoholipase A2, platelet-activating factor acetylhydrolase, generates, two bioactive products during the oxidation of low-density lipoprotein: use of a novel inhibitor. Biochem J, 338: 479-87
21. Frantz F. Menezes H.S., Lange K.C. Abegg M.P., Correa C.A., Zangalli L., Viera J.L., Zettler C.G., (2012). The effect of maternal hypercholesterolemia on the placenta and fetal arteries in rabbits . Acta Cirurgica Brasileira ,27(1):7-12.
22. Alassane D., Fatou C., Fatou G.T..,Fatou D., Oumar T.F.A., Ndene S.G.,Philomene L.S., Diop S.N., and Meissa T., (2014), Serum lipids and oxidized low density lipoprotein levels in sickle cell disease: Assessment and pathobiological significance, African Journal of Biochemistry Research, Vol. 8(2); pp:39-42.
23. Nakhjavani M.,Esteghamat A., Asgarani F., *et al* .(2009), Association of oxidized low-density lipoprotein and transforming growth factorbeta in type 2 diabetic patients: a cross-sectional study , Transl. Res.,153(2):86-90.
24. Huang H., Mai W., Liu D., Dong Y.,(2016), Oxidised low-density lipoprotein cholesterol and their ratio in the diagnosis and treatment of coronary heart disease, Heart ,Vol.97:pp:A135-A136.
25. Jousilahti P.,Vartiainen E.,Tuomilehto J.,Puska P.,(1999). Sex ,age cardiovascular risk factor , and coronary heart disease, A prospective follow-up study of 14 786 middle –aged men and women in finland . American Heart Association ,99:1165-1172.
26. Matthews KA., Meilahn E., Kuller LH., Kelsey SF., Caggiula AW., Wing RR., (1989) . Menopause and risk factors for coronary heart disease . N. Engl J. Med., 321:641-646.
27. Bonithon-Kopp C., Scarabin P-Y., Darne B., Malmejak A., Guise I., (1990), Menopause– related changes in lipoprotein and some other cardiovascular risk factors , Int. J. Epidemiol.,19:42-48.
28. Kiranmayi P.V., Vivekanand N., Pandit V.L. (2014) . The study of lipid profile and oxidized-LDL in type 2 diabetes mellitus, Sch. J. App. Med. Sci., 2(3D):1119-1122.
29. Poulsen MK.,Henriksen JE., Dahl J., Johansen A., Gerke O, Vach W, Haghfelt T., Hoilund-Carlsen PF, Beck-Nielsen H., Moller JE., (2010), Left ventricular diastolic function in type 2 diabetes mellitus prevalence and association with myocardial and vascular disease . Circ . Cardiovasc Imaging ,3:24-31.
30. Salehzadeh F., Rune A., Osler M., and Al-Hhalili L., (2011), Testosterone or 17ß -estradiol exposure reveals sex-specific effects and lipid metabolism in human myotubes. Journal of Endocrinology, 210; 219-229.
31. Kota S.K., Meher L.K., Kota S.K., Jammula S., Krishna S.V.S., and Modi K.D., (2013). Implication of serum paraoxonase activity in obesity, diabetes mellitus, and dyslipidemia. Indian J. Endocrinol Metab . 17(3):402-412.
32. Kumar D. and Rizvi S.I.,(2014) , Age–dependent paraoxonase 1 (PON1) activity and LDL oxidation in wistar rats during their entire lifespan.The Scientific World Journal,Vol.2014,pp:1-6.
33. Huang H., Ma R. Liu D. Liu C. Ma Y. Mai W., and Dong Y., (2012). Oxidized low density lipoprotein cholesterol and the ratio in the diagnosis and evaluation of therapeutic effect in patients with coronary artery disease, Disease Marker ,33:295-302.
34. Huang Y., Hu Y, Mai W., Cai X., Song Y, *et al* (2011), Plasma oxidized low-density lipoprotein is an independent risk factor in young patients with coronary artery disease, Dis Markers 31(5):295-301.
35. Essam Eldin MN, Abdullah A, Adel MA, *et al*. (2014), Oxidized low density lipoprotein and total antioxidant capacity in type 2 diabetic and impaired glucose tolerance Saudi men, Diabetology & Metabolic Syndrome ,6(94):1-10.
36. Baigent C, Blackwell J, Holland LE, et al (2010) , Efficacy and safety of more intensive lowering of LDL cholesterol: A meta-analysis of data from 170000 participants in 26 randomised trails, Cholesterol Treatment Trialists (CTT) Collaboration, lancet, 376:1670-1681.
37. Pasterkamp G, van Lammeren GW, (2010), Pleiotropic effects of statins in atherosclerotic disease , Expert Rev. Cardiovasc Ther. ,8(9):1235-1237.
38. Longo DL, Dennis L., Kasper F, Hause SL, Jameson JL, Loscalzo J, (2008), Harrison's principles of internal medicine, 18th edition, New York, McGraw Hill Company ;Vol.1.
39. Faulin TES, Garcia MCC, Abdalla DSP, (2009), Recent advances on detection of modified forms of low-density lipoprotein, Recent Patents on Endocrine, Metabolic & Immune Drug Discovery, 3:28-34.
40. Sertic J, Skoric B, Lovric J, Bozina T, Reiner Z, (2010), Does Lp-PLA_2 determination help predict atherosclerosis and cardio-cerebrovascular disease ? Acta Med Croatica ,64(4):237-45.
41. Van Dijk TH, Laskewitz AJ, Grefhorst A, Boer TS, Bloks VW, Kuipers F, Groen AK, Reijngoud DJ. (2013), A novel approach to moitor glucose

metabolism using stable isotopically labeled glucose in longitudinal studies in mice. Lab. Anim.,47(2):79-88.

42 Heriansyah T, Bambang BS, Anwar S, Djanggan S, and Aulanni'am A, (2016), The effect of darapladip on lipid profile, insulin, ox-LDL serum level and PVAT thickness at atherogenesis development in DM type2 rats model, International Journal of Pharm Tech Research, Vol.9,No.2:pp.01-08.

43. Gobal FA., and Metha FL., (2010) , Management of dyslipidemia in the elderly population , Ther Adv Cardiovasc Dis , 4:375-383.

44. Jamuna Rani A, Mythili SV, Nagarajan S,(2014) , Study on paraoxonase1 in type 2 diabetes mellitus , Indian J. Physiol Pharmacol ; 58:13-16.

45. Kola SK. Meher LK, Kola SK, Jammula S, Krishna SV, Modi KD, (2013), Implication of serum paraoxonase activity in obesity diabetes mellitus and dyslipidemia, Indian J. Endocrinol Metab,2013:402-412.

46. Namitha D, Nusrath A, Rajeshwari A, Rani NA, (2015), Serum paraoxonase levels in type 2 diabetes mellitus patients: A case – control study , Journal of Medical Sciences and Health , Vol.01,Issue 02;pp:14-18.

كوليستيرول البروتين الدهني الواطئ الكثافة المؤكسد وخطر امراض القلب التاجية

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الملخص

يعتبر كوليستيرول البروتين الدهني واطئ الكثافة المؤكسد عامل لبداية تكوين وتعجيل لأمراض القلب التاجية ,امراض الاوعية الدموية وداء السكري حيث يرتبط من خلال عدة ميكانيكيات احدها مسار الاكسدة, الهدف من هذه الدراسة هو علاقة الكوليستيرول المؤكسد لبروتين الدهني واطئ الكثافة, الكوليستيرول الكلي, كوليستيرول البروتين الدهني عالي الكثافة ,الكليسريدات الثلاثية ,كوليستيرول البروتين الدهني واطئ الكثافة وانزيم اريل استريز مع خطورة امراض القلب التاجية في مختلف الفئات العمرية عند الرجال والنساء, الاختلاف في عوامل الخطورة يظهر في الاختلاف في الجنس ومدى التغير في مستويات عوامل الخطورة في امراض القلب التاجية ومرض داء السكري ما بين الفئات العمرية.

طرق العمل : قيست مستويات كوليستيرول البروتين الدهني واطئ الكثافة المؤكسدة وكوليستيرول البروتين الدهني عالي الكثافة , سكر الدم وفعالية انزيم اريل استريز لجميع الحالات المدروسة (الرجال والنساء) والذين يحملون امراض القلب التاجية وداء السكر, الحالات قسمت الى فئات عمرية مختلفة (40–49),(50–60),(60–70) سنة ومقارنة النتائج مع مجاميع السيطرة.

النتائج: مستوى معدل ونسب البروتين الدهني واطئ الكثافة اظهر ارتفاع معنوي لدى مرضى القلب التاجية ومرضى امراض القلب وداء السكري للنساء في المجموعة العمرية (50–59) سنة ولكن المجموعة العمرية لدى الرجال اظهرت انخفاض معنوي لنتائجها الى جانب تأثير الجنس والعمر على زيادة بعض المتغيرات الكيموحيوية المدروسة وانخفاض معنوي على مستوى كوليستيرول البروتين الدهني عالي الكثافة وفعالية اريل استريز كمضاد للأكسدة في مرضى النساء والرجال مقارنة مع مجاميع السيطرة المختارة.