An Physiological Enzymatic Study for Atherosclerosis Patients in the City of Fallujah

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Abstract

Atherosclerosis is a syndrome that affects arterial blood vessels. Arteriosclerosis thickens the walls of blood vessels as a result of accumulation of macrophages and white blood cells, and fat accumulation is more pronounced over time. Blood pressure, smoking, obesity, lifestyle, sugar and age are risk factors. Atherosclerosis. The symptoms and signs of atherosclerosis differ depending on the arteries affected by atherosclerosis.

This study was conducted in the Pharma lab in the city of Fallujah and samples were collected from the Fallujah General Hospital. 50 samples were collected, 30 of which were patients with atherosclerosis and the remaining 20 samples were for healthy people. The samples included both males and females between the ages of 30 to 78 years. A questionnaire was used to collect some information from patients and corrections that included (gender, age, blood pressure, smoking, Weight and blood sugar) and some tests were performed (Cholesterol , TG, HDL, LDL, VLDL, GOT, GPT, ALP, Albumin). The results showed a significant increase in concentrations of Chol, TG, LDL, VLDL, GOT, GPT, ALP) in patients with atherosclerosis and a significant decrease in concentrations of both HDL and ALB in patients with atherosclerosis at the probability level ($P \leq 0.05$). The results also showed that males are more susceptible to arteriosclerosis than Females, and also showed that the greater the age, the greater the risk factors for atherosclerosis. The more risk factors for atherosclerosis increase with increasing body mass. As for smoking, the results indicated that it has an effect on atherosclerosis, as people who smoke have increased risk factors for atherosclerosis, and disease risk factors increase with increased blood pressure and sugar.

Keywords : *Atherosclerosis, physiological study, Enzymatic study, Cholesterol, Triglyceride, LDL-C, HDL-C, VLDL, GPT, GOT, ALK, and Albumin.*

Introduction

Atherosclerosis is a disease that clinically affects all blood vessels, including the cerebral, coronary and peripheral arteries [1]. It is an inflammatory vascular disease caused by the deposits of fats in the walls of blood vessels, and it grows to a level that obstructs blood flow [3]. The accumulation of these deposits leads to narrowing of the artery and causing a clot, which leads to a stroke, dilated blood vessels in the abdomen, gangrene in one of the extremities, heart attack, myocardial infarction (angina pectoris) and ultimately, sudden death [2]. This build-up occurs over a period of years and shows no direct symptoms or signs until blood flow is severely affected, resulting in reduced organ nutrition [1]. Here the symptoms and signs appear, and this depends on the type of affected organ, and in most cases more than one member suffers from a lack of blood nutrition as a result of narrowing of the arteries that feed them due to atherosclerosis, and most cases these symptoms appear after the age of fifty or more, and angina is the first case [3].

Methods

1. Collections of Samples :

This study was conducted from (October 2019 to January 2020) in Pharma laboratory Fallujah city in Anbar governorate. During this period (50) samples (patients = 30, controls= 20) were collected from Males and females, the number of males was (28) and the number of females (22), and their ages ranged between

no Both sexes (30-78) years old.

2. Initial treatment of blood samples :

The blood was drawn intravenously in a volume of 10 ml and some blood was taken

Information for each, as blood samples were placed into test tubes Centrifuge at 3500 rpm for 15 minutes to obtain Blood serum, which was placed in new plastic tubes and recorded to and through all the information Lipid levels were checked in the central laboratory by measuring total cholesterol, triglycerides, and high-density lipoprotein concentration Density lipoprotein and low lipoprotein density.

3. Physiological study :

1.3. Determination of Cholesterol in Blood **:**

The enzymatic method was used to estimate the level of cholesterol in the blood serum by using the diagnostic kit (Cholesterol) provided by the Spanish company Biosystem [4], the light absorption was measured at (λ=500nm).

2.3. Determination of Triglyceride in Blood :

The enzymatic method was used to estimate the level of Triglyceride in the blood serum by using the diagnostic kit (Trigleseraide) provided by the Spanish company Biosystem [4], the light absorption was measured at (λ=500nm).

3.3. Determination of serum HDL-Cholesterol **:**

The enzymatic method was used to estimate the level of Triglyceride in the blood serum by using the diagnostic kit (HDL) provided by the Spanish company Linear [5]. the light absorption was measured at (λ=500nm).

3.4. Determination of Serum LDL-Cholesterol:

Low density lipoprotein in serum was determined using Fried Wald's equation The famous, it is clear from this equation that the value of VLDL = 5 / TG, then the Fried Wald equation states if : Ch = HDL + LDL + VLDL

LDL = Ch - (HDL + VLDL)

So LDL = Ch - (HDL + TG / 5)

4. Enzymatic study :

1.4. Determination of Glutamat-pyruvic (GPT) transaminase in Blood : The level of effectiveness of GPT was estimated using the diagnostic kit (GPT) provided by the Spanish company Linear [6]. The principle of this method is based on monitoring the reaction kinetically at 340nm at a rate of reduced absorption to the activity of ALT present in the sample.

$$\text{L-Alanine + 2-Oxoglutarate} \xrightarrow{\text{ALT (GPT)}} \text{L-Glutamate + Pyruvate}$$

$$\text{Pyruvate + NADH + H} \xrightarrow{\text{LDH}} \text{Lactate + NAD}$$

2.4. Determination of Glutmate-Oxaloacetate (GOT) transaminase in Blood :

The level of effectiveness of GOT was estimated the diagnostic kit (GOT) provided by the Spanish company Linear [6]. The principle of this method is based on monitoring the reaction kinetically at 340nm at a rate of reduced absorption to the activity of AST present in the sample.

$$\text{L-Aspartate + 2.Oxoglutarate} \xrightarrow{\text{AST (GOT)}} \text{L-Glutamate + Oxaloacetate}$$

$$\text{Oxaloacetate + NADH + H} \xrightarrow{\text{MDH}} \text{L- Malate + NAD}$$

3.4. Determination Alkaline Phosphate (ALP) in Blood :

The level of effectiveness of was estimated the diagnostic kit (ALP) provided by the Spanish company Bio system [7]. The concentration of (ALP) in the sample is calculated using the general formula :

$\Delta A/min \times Vt \times 106 / E \times I \times Vs = U/L$

The molar absorbance (E) of 4-nitrophenal at 405 nm is 18450, the light path (I) is 1cm, the total reaction volume (Vt) is 1.20, the sample volume (Vs) is 0.02, and U/L are 0.0166 μkat/L.

5. Determination Albumin in Blood :

The level of effectiveness of was estimated the diagnostic kit Albumin provided by the Spanish company Bio system [7]. the light absorption was measured at (λ=630nm).

6. Determination Glucose Test in Blood :

The level of effectiveness of was estimated the diagnostic kit (Glucose) provided by the Spanish company Linear [5]. the light absorption was measured at (λ=500nm).

Statistical Analysis

The Duncan test for statistics was used at the significance level ($P \leq 0.05$) for the studied variables in the blood serum of patients with atherosclerosis and compared them with controls.

Results & Discussion

1. Serum Test:

The mean concentrations (cholesterol, TG, -HDL-C, LDL-C, VLDL) and enzymes (GPT, GOT and ALK) and albumin were measured in patients with atherosclerosis compared to healthy controls, the study was divided into seven tables: the first for the level of lipid and enzyme concentrations for total patients (30) and total control (20). Second : classification of atherosclerosis patients and controls according to gender. Third : classification of atherosclerosis patients and controls according to age group. Fourth : the effect of body mass index (kg / cm) on the studied variants of atherosclerosis patients and controls. Fifth : The effect of smoking on the variables studied for atherosclerosis patients and controls. Sixth : the effect of pressure on the variables studied for atherosclerosis patients and controls. Seventh : the effect of blood glucose concentration on the variables studied for atherosclerosis patients and controls. Measuring a rate serum of concentration (Cholesterol, TG, HDL-C, LDL-C, VLDL, GPT, GOT, ALK and ALB) for patients with Atherosclerosis and controls, the results are shown as shown in table (1) where significant increase was observed in the mean at ($P \leq 0.05$) in the levels of concentration (Cholesterol, TG, LDL, VLDL, GPT, GOT and ALK) in patients with results (263.9±21.136, 321.2±20.095, 175.933±8.469, 33.6±4.952, 47.967±6.835, 41.767±6.452 and 161.3±8.074) respectively**.** This is accordance with a study (Chikkanna *et al.*) [8] and (Karajibani *et al.*) [9], where they notice a significant higher in levels (Cholesterol, TG) , the results also accordance with the study [10]. Where they observed a significant decreased for HDL-C in atherosclerosis patients and higher in concentration of (Cholesterol, TG) , Cholesterol increases above average normal when suffering from some disease, including atherosclerosis and heart disease [11]. This increase was caused for several reasons including nutrition, which causes an increase in the concentration of lipids in the plasma [12]. That increase cholesterol in the blood is one of the main causes of heart disease and atherosclerosis and plays an important role in the development of the injury in coronary heart disease, cholesterol deposits on the walls of blood vessels, causing them to narrow and blockage, and the increase in the concentration of triglyceride is due to an increase in fat-rich nutrition which leads to an increase in the production of chylomicrons in the intestine, which is then broken down it causes fatty acids to be released, so the liver cells will receive large amounts of fatty acids, causing increase in the release of triglycerides [13]. The high LDL-C level may be attributed to the degradation that occurs binding of (LDL-C) to receptor in the liver, which plays a large role in reducing the transformation of LDL-C to the liver tissue and then increase its concentration in the blood serum, and this leads to an increase LDL-C [14]. While the decrease in (HDL-C) concentration was significantly decreased at likelihood level ($P \leq 0.05$) for patients with atherosclerosis were (3.408± 33.067) compared to controls were (48.65 ± 8.592) the reason for decrease in (HDL-C) levels is to decrease in the activity of the enzyme lipoprotein LPL which leads to the degradation of (TG) to fatty acids and glycerol [15]. The results as shown in table (1) showed a significant increase in the level of probability ($P \leq 0.05$) in the level of (GPT, GOT) concentrations in patients with atherosclerosis, and the results were (6.452±41.767,6.835±47.967) compared with controls were (6.612±22.15, 5.906±22.4) respectively, and these results were accordance with a study (Alicia & Debabrata) [10]. Where noticed an increase in the levels of (GPT, GOT) that the increase in enzyme activity it can lead to abnormal structural and functional changes that the occur of hepatic cells, and these changes may increase the necrosis of hepatocytes (Necrosis). Thus, the enzymes are released into the bloodstream [16]. The results of the albumin concentration showed a significant decrease, and the result was (3.004±0.456) this result was accordance with the result of (Savini *et al*.), as this study that appears confirmed low serum albumin is associated with an increased risk developing cardiovascular disease in adults age.

Table (1) : A rate serum of concentration (Cholesterol, TG, HDL, LDL, VLDL, GPT, GOT, ALK and ALB) for patients with Atherosclerosis and controls.

Parameters Factors	Patients ± SD	Controls ± SD
Cholesterol	*263.9±21.136	177.174± .051
TG	*321.2±20.095	124.391±12.578
HDL-C	33.067±3.403	48.65±8.592
LDL-C	*175.933±8.469	99.1±11.248
VLDL	*33.6±4.952	26.615±4.214
GPT	*47.967±6.835	22.15±6.612
GOT	*41.767±6.452	22.4±5.906
ALK	*161.3±8.074	174.55±13.567
Albumin	3.004±0.456	4.268±0.302

* Significant differences according to the Duncan test ($P \leq 0.05$)

2. Effect of personal factors on Atherosclerosis:

1.2. Sex:

Table (2) shows the classification of the infected group according to sex, as the level of concentrations of each of (Cholesterol, TG, HDL-C, LDL-C, VLDL, GPT, GOT and Albumin) in the blood serum of patients with atherosclerosis (male-female) was measured compared to the healthy ones, as males notice more injury than females, and this is consistent with [17] it confirmed that males are more susceptible to cardiovascular disease. Testosterone increases the activity of the enzyme lipoprotein LPL, which increases the breakdown of (HDL-C) and thus high cholesterol and increases the amount of fat carried in the blood (VLDL, LDL-C and TG) which has a direct effect on heart disease [18]. In females, the female hormone estrogen has a role in the protection and prevention of heart diseases [19], as it helps in converting carbohydrates into fats in the process of storage under the skin and at the same time reduces the level of cholesterol in the blood and obesity and increases the level of (HDL-C), but in the case of Reducing or discontinuation of the hormone occurs an increase in the level of cholesterol and an increase in exposure to vascular and cardiac diseases, and this is what we observe after menopause, which leads to the occurrence of obesity, joint pain and osteonecrosis [20]. The results in table (2) showed a significant decrease at the probability level ($P \leq 0.05$) in the levels of (HDL and Albumin) concentrations in patients with atherosclerosis (male, female) compared with the controls, there are international studies that have demonstrated that the risk of atherosclerosis for both sexes is inversely related to levels of (HDL-C) in the blood, the higher the levels of (HDL-C) the oxidative damage is implicated in the development of atherosclerosis [21]. The results also showed that GPT enzyme increased in males when compared with women, and this study contradicts the study (Hussain) [22]. While the results showed that the concentration of ALK is high in healthy people compared with patients and this is due to the level of the ALK enzyme that varies in different people depending on blood types and is associated with an antigen present on the membranes of red blood cells [23].

Table (2): The effect of gender on the studied variants of atherosclerotic patients and control.

Parameters / Factors	Patients		Control	
	Male	Female	Male	Female
Cholesterol	*214.215±40.628	*243.05±47.509	165.57±9.65	167.66±16.13
TG	*222.917±97.041	*260.532±99.05	107.81±11.51	109.03±12.33
HDL-C	*38.5±8.511	*40.038±10.876	54.125±6.23	57.40±8.65
LDL-C	*140.083±41.182	*149.923±37.474	129.44±17.88	145.21±3.34
VLDL-C	*29.158±5.557	*32.32±5.652	27.122±3.23	22.15±6.5
GPT	37.792±15.322	37.5±13.831	38.287±16.81	33.22±12.4
GOT	33.333±11.846	34.654±11.17	32.45±12.5	31.24±10.13
ALK	*170.25±9.289	*163.231±13.99	166.45±7.33	160.33±10.9
ALB	3.5054±0.807	3.513±0.692	3.99±0.88	3.59±0.65

* Significant differences according to the Duncan test ($P \leq 0.05$)

2.2. Age:

Table (3) shows the classification of the group of people with atherosclerosis according to the age group, as it is noticed that people of late age groups (more than 50 years) are more susceptible to developing atherosclerosis to measure the level of concentrations of each of (TG, HDL-C, LDL-C, VLDL, GPT ALK and Albumin) and this is in accordance with (Costa *et al*.) [18] and those who indicated that progress Age is one of the factors that cause heart disease, as the incidence increases after the fifth decade and in both sexes [23]. Table (3) showed a significant decrease at the probability level ($P \leq 0.05$) in the concentrations of (HDL, albumin) for patients with atherosclerosis for all age groups compared to the controls, where the concentration of (HDL-C) decreased significantly in the age groups (40-50). Our results are not in accordance with (Attyha *et al.*) [24] which showed that it is positively correlated with GPT and negatively correlated with (GOT, Albumin), but it is in agreement with their study which showed that low serum albumin is associated with cardiovascular disease [22].

Table (3): The effect of age group on the variables studied for atherosclerosis patients and Controls

Parameters / Factors	Patients			Controls		
	40Less of ±SD	50-40 ±SD	50 More of ±SD	40Less of ±SD	50-40 ±SD	50 More of ±SD
Cholesterol	* 203.291±36.67	* 228.805±44.76	* 252.394±45.60	151.44±9.44	169.11±2.99	100.01±3.99
TG	* 193±92.515	*245.97±99.085	*281.401±90.53	99.04±2.88	106.11±23.87	113.34±8.99
HDL-C	* 40±6.725	* 39.75±11.525	* 38.125±10.02	56.98±5.88	54.10±4.88	53.65±6.00
LDL-C	*131.786±43.199	* 144.8±38.845	* 157.438±34.13	128.98±20.8	133.9±13.65	144.00±15.39
VLDL	* 26.914±4.875	* 30.425±4.833	* 34.688±5.326	24.98±2.67	26.33±5.6	30.11±5.87

Cont... **Table (3): The effect of age group on the variables studied for atherosclerosis patients and Controls**

GPT	* 33.214±14.61	*39.25±15.583	39.5±12.675	30.34±12.76	35.00±14.98	37.89±13.66
GOT	* 30.071±10.637	* 34.2±11.428	*37.25±11.642	28.65±9.55	30.23±10.8	36.22±11.80
ALK	* 157.688±11.965	*170.8±11.399	* 157.688±11.965	166.44±6.90	165.77±3.99	152.61±10.55
ALB	* 3.252±0.62	*3.606±0.775	* 3.252±0.62	2.88±0.23	2.98±0.54	3.00±0.45

* Significant differences according to the Duncan test ($P \leq 0.05$)

3. The effect of obesity with atherosclerosis:

Atherosclerosis is associated with obesity, as Table (4) shows the body mass index values (BMI_1, BMI_2, BMI_3) for a group of people with the disease compared to a group of Controls, as it is noticed that people who suffer from obesity are more likely to develop arteriosclerosis disease and this is confirmed [25]. The presence of high body mass index in Iraqi patients with atherosclerosis. The levels of lipid concentrations (Cholesterol, TG, LDL-C and VLDL) were significantly increased at the ($P \leq 0.05$) probability level with an increase in BMI in patients with atherosclerosis when compared with the Controls, The relationship of obesity, the rate of occurrence of heart attacks and arteriosclerosis is a direct relationship, because obesity is associated with the disturbance in the pattern of distribution of fat in the body and the deposition of excess fat under the skin, abdomen and muscles, as it represents an additional burden on the heart leading to a decrease in HDL-C and a high level of (Cholesterol, TG) As a result, it is a risk factor for developing heart disease [26]. Table (4) also shows that there are significant differences in the concentration of (GPT and ALK) for patients with atherosclerosis compared with the Controls, and this study came in line with (Hirakawa *et al.*) [27] when an increase in BMI was observed in people who suffer from obesity, which is a risk factor. In the development of oxidative stress associated with many diseases, including atherosclerosis in terms of an increase in oxidative products and a decrease in levels of antioxidants [27]. The study also showed a significant increase in albumin concentrations at the probability level ($P \leq 0.05$) compared with the controls. The activity of enzymes (GPT, ALK) in addition to albumin is an important diagnostic marker to infer hepatopathies [22].

Table (4): The effect of BMI (kg / cm2) on the studied variants of atherosclerosis patients

Parameters / Factors	Patients			Controls
	BMI1 (n=10)	BMI2 (n=10)	BMI3 (n=10)	BMI Controls (n=20)
Cholesterol	* 209.271±39.67	269.4±11.908	278.9±21.074	166.10±13.04
TG	* 205.252±96.287	* 329.4±19.126	* 329.3±11.441	107.90±10.20
HDL-C	* 41.629±10.606	34.4±4.45	33.6±3.565	55.266±7.77
LDL-C	132.143±39.884	174.2±9.094	* 176.4±10.554	130.34±11.04
VLDL	* 28.923±5.012	36±3.391	34.8±5.922	32.6±4.99
GPT	* 34.057±15.262	* 51.4±7.369	* 43.3±6.056	33.09±5.87
GOT	* 30.686±11.427	41.2±8.319	42.1±6.28	32.66±6.65
ALK	* 170.771±11.7	* 162.4±8.792	* 154.1±4.977	155.23±3.66
ALB	3.688±0.778	3.366±0.434	* 2.953±0.377	3.782±0.32

* Significant differences according to the Duncan test ($P \leq 0.05$), BMI_1= Normal weight, BMI_2= Above normal weight, BMI_3= Obese, $BMI_{Controls}$= Normal weight for Controls.

4. Effect of Clinical Factors on Atherosclerosis:

1.4. Blood Pressure:

Table (5) shows the effect of the blood pressure level on patients with atherosclerosis, as the results showed a significant increase in the concentrations of (Cholesterol, TG, LDL-C, VLDL, GPT, GOT and ALK) in both groups of patients, which are less than (10/15) and a group more than (10/15) at the probability level ($P \leq 0.05$) when Compare it with the controls group, This accordance with (Coresh *et al.*) [28], which showed that people with high blood pressure have a double risk of coronary heart disease at a rate of (2-3) times more than individuals with normal pressure. Our study also accordance with the study of people with high blood pressure and cholesterol relationship. In Nigeria, epidemiological studies indicated increased heart disease due to blood cholesterol [31]. High concentrations of cholesterol increase stress and are attributed to a combination of factors as diverse as genetics and lack of exercise [29]. The British Heart Foundation indicated that a decrease in pressure of (5) mmHg leads to a reduction in the risk of developing cardiovascular disease [29]. Studies have shown that angiotensin II, which is often elevated in hypertensive patients, has a role in narrowing the arteries, as it contributes to the establishment of stiffness by stimulating the growth of smooth muscles through its association with special receptors on the surface of smooth muscles, causing a stimulation of the phospholipase enzyme, which leads to the accumulation of calcium outside Cells, causing those muscles to contract further [30]. Increased angiotensin II stimulates increased activity of Lipoxygenase in smooth muscles, which in turn increases oxidation of (LDL-C) molecules it is thus a primary cause of inflammation by increasing H2O2 hydrogen peroxide and free radicals in the plasma [29]. The results showed a significant decrease in the concentrations of (HDL and Albumin) in both groups of patients at the probability level ($P \leq 0.05$) when compared to the controls, that (HDL-C) works to transport cholesterol from the peripheral tissues to the liver [28]. Our results showed that there was an increase in liver enzymes (GPT, GOT and ALK), It was found that negative hepatic congestion causes increased central vein pressure that may cause elevated liver enzymes [30].

The effect of stress on the studied variants of atherosclerosis patients and controls

Parameters Factors	Patients		Controls	
	10/15 نم لقا ±SD	10/15 نم رثكأ ±SD	10/15 نم لقا ±SD	10/15 نم رثكأ ±SD
Cholesterol	*205.59±13.984	* 275.059±51.458	188.05±12.09	198.67±11.09
TG	*197.085±20.839	* 330.588±104.98	190.56±14.19	199.89±10.89
HDL-C	* 42.242±3.097	* 33.588±10.13	38.12±8.09	30.88±12.09
LDL-C	* 129.485±6.36	* 175.706±40.1	119.78±7.90	170.78±6.87
VLDL	* 29.009±4.28	* 34.294±6.12	26.73±4.05	32.89±5.19
GPT	* 33.33±5.517	* 46±13.84	29.67±7.54	33.55±6.66
GOT	* 30.33±6.489	*41.176±11.293	27.03±4.54	38.32±10.42
ALK	* 171.091±5.732	* 157.882±14.031	160.56±8.37	150.88±6.11
ALB	*3.716±0.474	*3.109±0.686	2.089±0.567	3.001±0.231

* Significant differences according to the Duncan test ($P \leq 0.05$).

2.4. Concentration of Glucose in Blood:

The results showed as shown in Table (6), that there was a significant increase in the concentrations of (Cholesterol, TG, LDL-C, VLDL, GPT, GPT and ALK) in each of (less than 180 and 200-250 and more than 250) compared to healthy subjects, and a significant decrease in the concentrations of (HDL-C and Albumin). This is in accordance with Marks [31], who confirmed that individuals with diabetes have higher rates of cardiovascular disease compared to non-diabetics, and as it has been shown that people with type 2 diabetes have dangerously high levels of (LDL-C) and triglycerides that circulate in the bloodstream, and these high levels are associated with an increased risk of cardiovascular disease. The high level of triglycerides and cholesterol may be due to the body's use of fat as a source of energy instead of glucose sugar, as it consumes stored fats and then increases its level in the blood, and this increase leads to cholesterol deposition in the blood vessels to be a cause of atherosclerosis and heart disease [32]. Another study showed that there is a slight correlation between sugar and total cholesterol concentration in both atherosclerosis and type 2 diabetes, and this causes a long chain of fatty acids that inhibit the enzymes of the control points in the glucose analysis, which leads to an increase in the concentration of glucose and on the other hand, high glucose provides energy or adenosine. Triphosphate, which provides cholesterol metabolism [31]. The cause of high concentrations of (GPT, GOT and ALK) in patients with atherosclerosis in all groups, less than (180 and 200-250), and more than 250 result from the occurrence of abnormal structural and functional changes that may occur to hepatocytes and these changes may increase the laceration Or the hepatocyte necrosis (Necrosis) and thus the release of enzymes into the bloodstream [32]. The significant increase is also attributed to the increase in glycogen build-up in hepatocytes, which leads to Hepatic cirrhosis, which occurs as a result of the accumulation of fats inside hepatocytes [33]. The increase in the level of the enzyme (ALK) is also due to the release of the enzyme from the system, as diabetes may be associated with many bone diseases, especially osteoporosis, as a result of an imbalance in the secretion of the parathyroid gland (PTH), which plays a major role in maintaining the level of calcium in the blood [33].

Table (6): The effect of blood glucose concentration on the variables studied in atherosclerosis patients and controls.

Parameters / Factors	Patients			Controls		
	180 نم لقا ±SD	250-200 ±SD	250 نم رثكأ ±SD	180 نم لقا ±SD	250-200 ±SD	250 نم رثكأ ±SD
Cholesterol	*** 199.98±34.24**	*** 267.72±14.30**	*** 279.55±22.24**	**191.22±15.09**	**200.41±20.13**	**230.45±19.61**
TG	*** 182.99±85.24**	331.091±17.751	332.44±6.002	178.87±23.89	290.12±12.89	299.58±10.77
HDL-C	* 43.26±10.56	33.364±3.557	33.33±3.674	33.89±11.90	29.50±4.44	21.34±2.99
LDL-C	* 124.76±18.17	174.90±8.642	177±11.011	120.34±10.66	155.45±6.89	170.45±9.21
VLDL	*28.743±5.296	33.727±4.777	34.11±5.841	25.76±5.01	29.41±3.79	30.42±4.98
GPT	*30.867±13.788	* 52.182±7.040	* 42.44±5.747	28.91±10.76	33.81±6.87	37.90±6.90
GOT	* 28.4±10.11	43.455±8.513	41.22±5.975	21.86±6.33	35.81±5.00	34.84±7.22
ALK	171±12.407	164.72±9.001	* 154.22±5.26	161.80±5.64	157.51±5.87	144.90±4.82
ALB	* 3.79±0.781	3.12±0.47	3.025±0.317	2.75±0.22	3.801±0.23	3.98±0.239

* Significant differences according to the Duncan test ($P \leq 0.05$).

Conclusions

There was a significant increase in the concentrations of cholesterol and TG in patients with atherosclerosis compared to controls, and the subjects had a significant decrease in the concentration of (HDL-C) with a significant increase in the concentration of (LDL-C) in the blood serum of patients compared to controls. It was also observed that the level of the enzyme (ALK) varies in different people depending on the blood types and is associated with an antigen present on the membranes of red blood cells and the decrease in serum albumin within the normal range related to cardiovascular disease.

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Ethical Clearance: This study is ethically approved by the institutional ethical Committee.

References

1. Clemmons, D. R. ‘’Atherosclerosis: Risks, Mechanisms. And Therapies’’.(2015). P:555-565.
2. Lüscher, T. F. ‘’’Atherosclerosis and CAD’’. European heart journal . (2015). p:457-459.
3. Rubin, S.; David, S.; Rubin, R. E. ‘’Clinicopathoiogy Foundation of Medicine’’. Lippincott Williams & Wilkins. (2011). ISBN . p: 44-53.
4. Young, D. S. Effects of drugs on clinical laboratory tests. 5th ed AACC Press. (2000). P :122-129.
5. Buccolo, G. & David, H. Cin Chim.(1973). 476.
6. Winn-Deen, E. S. ; David, H. ; Sigler, G. Chaves, R. Cin Chim. (2005).p: 1988-1999.

39. Nugen S., Baeumner H. J. Trends and opportunities in food pathogen detection. Anal Bioanal Chem. (2008). 391:451-454.

8. Chikkanna, D., Thammanna, G., Dinesha, R. B., Harsha, R., Chethan, K. M. ‘’Effects with Coronary Heart Disease’’. Adichunchanagiri Institute of Medical Sciences and Research, Pharmacologyonline .(2010). p: 385- 392. 9. Karajibani M., Montazerifar F., Hashemi M., Bolouri A., Dikshit M. “A Study on Oxidative Stress in Patients with Angina Pectoris Admitted to Coronary Care Unit.” Zahedan Journal of Research in Medical Sciences.(2013). 15.3 : 20-25.
10. Alicia, M. A., & Debabrata M. Liver Abnormalities in Cardiac Diseases and Heart Failure International Journal of Angiology .(2011). 20(3): 135–142.
11. Veiraiah, A. Hyperglycemia , lipoprotein glycation, and vascular disease. Angiology. (2005).56 (4) : 431-8.
12. Balakumar P., & Babbar L. ‘’ Preconditioning the hyperlipidemia myocardium : fact or fantasy ‘’ . Cell Signal .(2012). 24 : 589 – 595. 13. Akuyam, S.A. ; Aghogho, U. B. ; Bakari, A.G. Serum total cholesterol in hypertensive Northern Nigerians . International Jornal of Medicine and Medical Science .(2009). 1(3) : 73-78.
14. Block, J.H. & Beale, J.M. “Wilson and Gisvolds Text book of organic medical and pharmaceutical chemistry”. 6thed. Lippincott williams & wilkins. (2004). 657-660.
15. Salih, A. M., Kaleel, F. M., Mubder, N.S. Determination of testosterone inflammatory proteins and several biochemical parameters in sera of patients with atherosclerosis. Iraqi J. Biotech .(2012). 11:121-132.
16. Savini, I., Valeria G., and Maria V. C. ‘’Oxidative Stress and Obesity’’. Springer International Publishing. (2016). 65-86.
17. Sias, B. , Ferrato, F. , Pillcer, R. M. , Forgerty, Y. , Guillouet, P.H. , Leboeuf, B. , Carriere, F. Cloning and seasonal secretion of the pancreatic lipase-related protein 2 present in goat seminal plasma. Biochim. Biophys. Acta .(2012). vol. 1686: 169-180.
18. Costa, P., Sutin, A., Deiana, B., Terracciano, A., Lakatta, E., Schlessinger, D., Uda, M. “Cholesterol, Triglycerides, and the Five Factor Model of Personality.” Biological Psychology.(2016). 84.2.
19. Ahmad V., H., Shahsavari, G., Tavafi, M., Bagheri, S., Moradkhani, M. R., Mohammad R., K. R., Khosravi, P., Jafari, M., Zahabi, K., Eftekhar, R., Soleimaninejad, M., Moghadam, S. ‘’Protective effects of european against renal injury oxidative damage in alloxaninduced diabetic rats; a histological and biochemical study’’. J of Nephropathy. (2017). 6(3), p :204-209.
20. Feingold, K. R., & Carl G. ‘’Obesity and Dyslipidemia’’.(2015). p:122-135. 21. David, G. G., Asha, J., Dolores, S. ‘’Green span’s Basic and clinical Endocrinology’’ . Yale J Biol. Med.(2012). 85(4) , p: 559-570.

22. Hussain, M. M. ‘’Intestinal lipid absorption and lipoprotein formation’’. Curr Opin Lipidol .(2014). (25), p :200-206.

23. Garelnabi, M., Dmitry L., & Srikanth K. Role of Oxidized Lipids in Atherosclerosis. INTECH Open Access Publisher. Oxidative Stress and Diseases. (2012). p.120-140.

24. Attyha , I. N. , Turki , E. M. , Hassan , J. M. Effect of hyperlipidemia in the atherosclerotic patients on sex hormones. Magazine of Al-Kufa University for Biology. (2013). Vol.5 : 123-129.

25. Jae , W.Y. , Seung , T.H. , Shin, H. S. , Min, K. K. , Jae, S. K. Serum T3 Level can predict Cardiovascular events and All-cause Mortality rates in CKD patients with proteinuria . (2012) . volume (34) , p : 364-372 .

26. Joel , E. , Dimsdale, M. D., La Jolla ,C.. ‘’Psychological stress and Cardiovascular Disease ‘’. Journal of American College of Cardiology. (2009). Vol. , 51 :37-45.

27. Hirakawa, Y., Lam, T. H., Welborn, T., Kim, H. C., Ho., S., Fang, X. ‘’The impact of body mass index on the associations of lipids with the risk of coronary heart disease in Asia Pacific region’’. Preventive Medicine Reports.(2016). p: 33-50.

28. Coresh, J., Wei, L., Mc, G. Prevalence of high blood pressure and elevated serum creatinine level in the United States. Findings from the third National health and Nutrition Examination survey , Arch Intern Med.(2001).161:1207.

29. Ho, L., Yin, W. H., Chuang, S. Y., Tseng, W. K., Wu, Y. W., Hsieh, I. C., Ueng, K. C. ‘’Determinants for achieving the LDL-C target of lipid control for secondary prevention of cardiovascular events in Taiwan’’. PloS . (2015). p: 66-80.

30. Garelnabi, M., Dmitry L., & Srikanth K. Role of Oxidized Lipids in Atherosclerosis. INTECH Open Access Publisher. Oxidative Stress and Diseases. (2012). p.120-140.

31. Marks, J. Nomenclature and Glycemia . Clinical Diabetes. (2004). P:22-34. 32. AL-Fadhli, M. A. Study the effect of various treatments and the suffering of diabetes (Type II) in the Governorate Wasit University.(2008). P: 122-130.

33. Ahmadvand, H., Shahsavari, G., Tavafi, M., Bagheri, S., Moradkhani, M. R., Mohammadrezaei, K. R., Khosravi, P., Jafari, M.; Zahabi, K., Eftekhar, R., Soleimaninejad, M., Moghadam, S. ‘’Protective effects of oleuropein against renal injury oxidative damage in alloxaninduced diabetic rats; a histological and biochemical study’’. J Nephropathol. (2017). 6(3), p :204-209.