Study of LCAT and some biochemical parameters in cardiovascular patient

Amel Taha Yassein Al-Juraisy[1] , Ameera Aziz Mahmood Al-Juraisy[1] , Nadia Ahmed saleh[2]
[1] *Department of chemistry , College of Education for Pure Science , Mosul University , Mosul , Iraq*
[2] *Department of chemistry , College of Education for Pure Science , Tikrit University , Tikrit , Iraq*
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Corresponding Author:
Name: Amel Taha Yassein
E-mail:
amal2005biochem@gmail.com
Tel:

ABSTRACT

The Clinical study included individuals (m23ales and females aged 25-75y) with cardiovascular disease. The activity of Lecithin: cholesterol acyl transferase (LCAT) and glutathione peroxidase(GPx) was estimated, in addition to the level of apolipoprotein AI (apo AI), apolipoprotein B100 (apoB100), total cholesterol (TC), triglyceride (TG), high density lipoprotein-cholesterol (HDL-c), low density lipoprotein-cholesterol (LDL-c), glutathione (GSH), peroxy nitrate (PN), malondialdehyde (MDA), glucose and certain elements (Zn, Cu, Mg and Co) in blood serum. The results showed that there was a significant decrease in the activity of LCAT and GPx, and the level of apoAI, HDL-c, GSH, Zn and Mg. on the other hand, it was shown that there was a significant increase in the level of apoB100, TC, TG, LDL-c, PN, Glu, Cu and Co but on unsignificant change in the level of MDA for both male and females.

Introduction

Cardiovascular diseases (CVD) in the present century will pose a major challenge in public health, both in industrialized societies and in developing countries. There is therefore an urgent need to find effective preventive and curative strategies to change this fact because of its serious economic and human consequences[1]. The main cause of cardiovascular disease is atherosclerosis. The direct cause of the disease is the nature and level of blood lipids that cause the arterial wall to thicken. It loses its elasticity as a result of the accumulation and aggregation of these fats in the endothelial cells of the wall. Calcium is often deposited with the fatty substances, producing calcified plaques. [2,3]. Since the 1940s, researchers have found that the imbalance in the metabolism or transfusion of blood cholesterol has a significant effect on the bloodstream disease [4,5].Studies also indicate that the amount and nature of food has a clear effect on the level of blood cholesterol, as saturated fatty acids, working to raise the level of cholesterol. While polyunsaturated fatty acids, reduce cholesterol levels, and monoclonal fatty acids did not have a statistically significant effect on cholesterol[6,7]Low density lipoprotiens (LDL) are the main carriers of cholesterol in the blood. This is the direct cause of cardiovascular disease [8,9]. These particles are oxidized in various free radicals. LDL

Oxidation (OX-LDL) can easily penetrate the surfaces of phagocytic cells because it does not require special receptors. This pathway penetrates into the cells, helping to form what is known as the foam cells, which helps in the accumulation of cholesterol in the wall of blood vessels [10,11]. In contrast, the high level of cholesterol in high density lipoprotein cholesterol (HDL-C) is associated with a lower risk of these diseases, and may be due to the role of these particles in the reverse transport of cholesterol, any they the transfer of various tissues of the body To the liver[12]. One of the effective ways to reduce the risk of cardiovascular disease is to reduce the level of cholesterol, especially the free oxidized cholesterol[13].Free cholesterol is converted to cholesterol ester by lecithin: Lecithin: cholesterol acyl transferase(LCAT) Which stimulates the transfer of a basal group of lecithin to cholesterol, this process speeds up the process of forming HDL particles [14]. LCAT is primarily created in the liver and is created in small amounts in both testes and stellar cells in the brain [15]. Although the role of LCAT in the reverse transfer of cholesterol, there are risk factors that may interfere with and affect the protective LCAT role and These factors include heredity [16] and age [17] and the nature of sex [18], since women are less likely to be infected at childbearing age[19]. Alcohol

abuse[20] smoking [21]and the nature of food [22] are low-calorie and rich in fish and vegetables Cardiovascular disease.

Materials and methods

The chemicals used in this study are equipped with international companies: Sigma, Fluka and Aldrich. The standard kit are prepared by the French company Biolabo for measuring Glucose, total cholesterol, triglycerides, (TG) and high-density lipoprotein cholesterol (HDL-C). Clutathione peroxidase has been analyzed by the French company Szakits. For apoB100, apo AI, it has been measured using several analyzes from RayBio. The samples were divided into two groups, a control group containing 100 samples of blood for healthy people aged 25-75 years for both sexes, divided into 23 females, 76 males.The group of patients included 100 blood samples of people with cardiovascular disease ranging in age from 25-75 years for both sexes. These included 45 females and 55 males. The activity of lecithin-cholesterol Acyl transferase (LCAT) in the serum using Manabe and his group (1976) [23]. The apo AI Enzyme-linked immunoassay and the protein fraction apo B100 is also estimated in Enzyme-Linked Immunoassay using several Elisa Kits[24,25], Based on the method used by Pistorius and Axelrod (1973) [26], zinc, copper, magnesium and cobalt were estimated in serum after dilution using an atomic absorption spectrophotometer, The activity of Clutathione Pyroxidase (GPX) was estimated using the kit[27]. The total cholesterol level [28], the level of high-density lipoprotein cholesterol in the serum was measured by enzymatic method using the kit[29],The concentration of LDL cholesterol and LDL was calculated according to

LDL-Cholesterol (Conc.)=Conc. of Total Cholesterol - Conc. HDL-C [30]

The level of glucose in the serum was estimated enzymatically using the kit[28]. In addition, the GSH level was assessed in serum using the method used by Sedlak and Lindsay (1986) [31]. The level of peroxy nitrite was estimated using the method used by Vanuffelen and his group (1998) [32]. The level of Malondialdehyde (MDA) was also assessed in the serum[33]

Results and discussion

The results in table (1) and table (2) showed a significant decrease in the level of LCAT in serum cardiovascular and both sexes. These results suggest that these conditions may be related to a reduction in the level of the LCAT enzyme responsible for transferring the basal group from lecithin to cholesterol and converting it into cholesterol ester in high density lipoprotein (HDL-C) particles. This is consistent with the findings of Manabe and his group (1987) [23] as well as Ossoli and his group 2016[34]. The low level of the LCAT enzyme is an indicator of the low level of HDL-C, which plays an important role in preventing or reducing the possibility of cardiovascular disease. In the last decade, there has

been much interest in strategies to raise the level of HDL-C to prevent this lesion [35,36]. The results shown in Table 1 and table 2 Showed a significant decrease in the level of apoAI-associated protein (at the probability level P≤0.01) in serum cardiovascular and cardiovascular patients for males and females (at the probability level P≤0.001). ApoAI is created in the liver and in the endothelial cells of the intestines and is mainly present in HDL particles and its blood level is inversely proportional to the risk of cardiovascular disease[37,38], which explains its low level of patients This study was addressed. Estimation of the level of apoAI in the blood is considered as a more important indicator than HDL-C in assessing the risk of cardiovascular disease[39]. The estimated level of the associated protein (apoB100) B100 in the serum of patients with vascular disease was found to be significantly higher At the probability level(P≤0.01, P≤0.001) at the male and female levels respectively, as shown in Table 1 and Table 2, this result is consistent with what Ooi and his group (2017) [40] and Ogedegbe (2002)[39] Plasma apoB100 is directly related to the risk of cardiovascular disease. Apo B is an essential component of LDL It is responsible for binding these particles to their receptors, then transferring the cholesterol to different tissue cells, and apoB level reflects the amount of LDL particles in the blood. Increasing the level of apoB100 is directly related to increased risk of cardiovascular disease [37]. On the other hand, females show a higher level of apo AI than males, and vice versa for apoB100[39]. The results shown in Table (1) and Table (2) indicate that the total cholesterol level in serum cardiovascular patients increased significantly (P≤0.001) for both males and females. This may be due to free radicals due to low activity of peroxidase enzymes . These roots lead to the formation of oxy sterol, which in turn inhibits the effectiveness of LDL receptors in the liver and then increases their levels and levels of cholesterol in the blood[41]. The reason for the moral rise in cholesterol may be due to the nature of nutrition, Food that contains high saturated fat content with low content of essential unsaturated fatty acids leads to elevated cholesterol. The basic unsaturated fatty acids inhibit the enzyme hydroxyl methyl glutaryl-CoA reductase (HMG-CoA reductase), the structured enzyme for building cholesterol [42]. The results shown in Table (1) and Table(2) show a rise significant in triglyceride at (P≤0.013) in male and vascular serum at (P≤0.008) for females. Elevation in the level may be due to increased activity of L-enzyme or reduced activity of lipoprotein lipase Removal of triglycerides from cylomicrone or lipoprotein particles[4,42] The results shown in Table (1) and Table(2) indicate that the level of HDL cholesterol in the serum for cardiovascular patients Was significantly reduced (P≤0.001) for both sexes. This may be attributed to the low activity of lipoprotein lipase, which acts to

transfer VLDL components to HDL and inhibition of Lecithin: LCAT cholesterol, which is necessary for the construction of HDL[43] Decrease in the production of early HDL particles from liver and small intestine cells [44]. The results shown in Table (1) and Table (2) showed a significant increase in serum and low density lipoprotein lipase in vascular serum and both sexes at the probability level (P00.001). The increase in LDL-C may be due to a decrease in the potential for LDL-C to be removed from the thrombocytopenic cells or liver cells or due to a defect in the apoB100 synthesis on the surface of the LDL particle or its receptors on the surface of hepatocytes and phagocytes[45,46]. As shown in Tables (1) and (2), the activity of Clutathione Peroxidase was significantly reduced (P $\leq$0.01) in serum cardiovascular patients for both males and females, and Clutathione Pyroxidase is an important enzyme that contributes to the removal of free radical Which are usually associated with vascular disease due to high oxidative stress and this affects the effectiveness of the enzyme that reduces these negative effects [47,48]. The high oxidative stress and the low activity of clutathione peroxidase is attributable to Ench The level of the nuclear factor erythroid 2-related factor 2 (Nrf2), which is responsible for regulating the cloning of genes responsible for the synthesis of protein-based antioxidants, scavenger receptors, and ATP- binding cassett transporters, the Nrf2 factor is directly related to controlling the levels of components that contribute to the process of oxidation and reduction in the body, including Clutathione Peroxidase. The incidence of atherosclerosis or vascular disease in general is associated with lower level of the factor Nrf2 and then lead to a decrease in the level of protein components that contribute to the reactions of oxidation and reduction, including the enzyme Clutathione peroxidase [40,49]. The results shown in Table (1) and Table (2) indicate a significant decrease (P$\leq$0.01) In the level of clutathione in the serum of patients with vascular disease for both sexes and the reason may be due to the condition of oxidative stress usually associated with vascular disease. In the case of oxidative stress, free radicals such as ROS (Reactive Oxygen Species), which are usually produced as by-products of oxidative metabolic processes, are enhanced by various environmental factors such as UV radiation, ionic radiation, pollutants and heavy metals[50,51] GSH, along with other antioxidants, works to remove free radicals by reducing these roots, which in turn is oxidized to GSSG. This process leads to lower serum levels of patients suffering from oxidative stress [52]. Peroxy nitrite is one of the high-effective free radicals that have been associated with vascular disease [53]. This is in line with what was reached in this study as shown in Table (1) and Table (2) (P$\leq$0.001) of peroxy nitrite in the serum of patients with vascular disease, male or female. Because of the low activity of

antioxidant enzymes that act to remove free radicals from blood vessel patients such as Clutathione Peroxidase (as mentioned in a previous paragraph), this condition makes this and other free radicals more available than normal [54]. The level of serum malondialdehyde reflects the level of lipid peroxides produced by the oxidation of unsaturated fatty acids and is often associated with vascular disease [55]. The results indicated in Table (1) indicate that there was non a significant increase in (P = 0.46) in the serum of male patients, while the results in Table (2) showed that there was a significant increase (P$\leq$0.05) in female serum The patients. The high level of malondialdehyde may be due to increased peroxidation of fat due to reduced antioxidants in cardiovascular patients, especially clutathione and clutathione peroxidase [56]. Malondialdehyde is one of the products of lipid oxidation and lipid oxidation .The process of peroxidate fat is effective in tissues rich in unsaturated fatty acids. In this process, unsaturated fatty acids are analyzed through a series of free radicals reactions to form lipid hydro peroxide [57].The results shown in Table (1) and Table (2) indicate a significant increase (P$\leq$0.001) in the serum glucose level of patients with cardiovascular disease for both sexes. This may be due to the case of oxidative stress which leads to an increase in active oxygen [58]. The human body contains 2-3 grams of zinc, and is found mainly in skeletal muscle (about 57% of the body)), Bone (about 29%) and a small percentage of the heart (about 0.4%) and plasma blood (about 0.1%) [59], and zinc deficiency in the body has serious consequences such as delayed growth, anemia, dysfunction in the nervous system and cardiovascular disease[60]. This is consistent with the level of zinc in vascular patient (P$\leq$0.001) low in its level as shown in Table (1) and Table (2). This may be due to oxidative stress which leads to damage to the endothelial cells of the blood vessels and disturbance In the process of NO formation and NF-KB factor and low-density lipoprotein oxidation. The presence of zinc in its normal level helps to prevent these events because of its effectiveness as an anti-oxidant and anti-inflammatory function that make it structured to prevent atherosclerosis and deficiency is a key factor in the incidence of infection [61,62]. The results shown in Table (1) and Table (2) show a significant increase (P$\leq$0.001) in the serum copper level of vascular patients of both sexes. This result is consistent with Chowdhury and his group 2018[63] that copper helps increase the risk of vascular disease by promoting the LDL oxidation process, which in turn contributes to atherosclerosis, that is, its low level is positive towards the prevention of this injury. Overall, the high level of copper Has a harmful role in promoting and increasing the condition of oxidative stress by increasing the number of active oxygen in the case of free and not associated with protein or otherwise, as in this case has the ability to produce free radicals and the middle

of the process of lipid peroxidation [64,65]. Results of serum magnesium level showed a significant decrease (P≤0.01) in both male and female levels as shown in Table (1) and Table (2). This is in line with what Cunha and his group 2018 [66] have pointed out that the magnesium level is inversely proportional to the risk of cardiovascular disease in various forms. The low level of this metal promotes oxidative stress. Magnesium deficiency also leads to calcification in soft tissues such as heart, liver and muscle. This deficiency leads to kidney damage because this deficiency helps in depositing apatite crystals in the Henley helix and renal tubules. Magnesium was found to improve circulatory function in patients with vascular disease [67]. The results shown in Table (1) and Table (2) showed a significant increase (P≤0.01) in serum cobalt Cardiovascular patients studied by both sexes. This corresponds to the fact of the linear relationship between cobalt level and the risk of vascular disease, which helps precipitate calcium and then thickens the inner walls of the arteries [68]. The effect of high cobalt level in the body simulates the effect of hypoxia. This condition is diagnosed by increasing the reactive sp oxygen species (ROS), which leads to a decrease in the level of antioxidants, especially clutathione, as well as low antioxidant enzymes such as clutathione peroxidase, catalase and superoxide dismutase. These conditionspromote vascular disease [69,70].

Table 1:comparison of the level of activity of the LCAT Enzyme with some biochemical variables between male cardiovascular patients(mean±SD)

Parameters	Patients/55	Control/76	*P*
Lecithin-cholesterol acyl transferase (U/ml)x10^{-3}	240.35±1.98.78	402.46±170.60	0.001
Apo AI (ng/ml)	1.68±0.18	2.74±0.16	0.01
ApoB100 (ng/ml)	103.28 ± 335.48	61.43 ± 224.35	0.01
Total cholesterol (mmol/l)	6.10±2.16	3.97±0.95	0.001
Triglyceride (mmol/l)	1.96±0.81	1.51±0.69	0.013
HDL-C (mmol/l)	0.81±0.31	1.62±0.76	0.001
LDL-C (mmol/l)	5.13±1.90	3.24±1.68	0.001
Glutathion peroxidase (U/l)	1.24±0.17	1.91±0.48	0.001
Glutathion (Mmol/l)	1.56±0.83	2.63±1.16	0.01
Peroxy nitrate (Mmol/l)	53.18±25.87	25.25±16.14	0.001
Malondialdehyde (Mmol/l)	1.80±0.12	1.77±0.11	0.46
Glucose (mmol/l)	7.61±3.50	4.41±0.98	0.001
Zn (Mg/100ml)	64.94±19.45	83.0±9.14	0.001
Cu (Mg/100ml)	117.70±40.41	97.74±16.21	0.001
Mg (mg/100ml)	1.42±0.13	2.34±0.86	0.01
Co (Mg/l)	0.34±0.02	0.22±0.01	0.01

Table 2:comparison of the level of activity of the LCAT Enzyme with some biochemical variables between female cardiovascular patients (mean±SD)

Parameters	Patients/45	Control/23	*P*
Lecithin-cholesterol acyl transferase (U/ml)x10^{-3}	162.13 ± 243.94	403.02±162.42	0.001
Apo AI (ng/ml)	1.63±0.14	0.32 ± 3.38	0.001
ApoB100 (ng/ml)	332.50±194.97	210.46±54.56	0.001
Total cholesterol (mmol/l)	6.23±2.39	4.19±0.55	0.001
Triglyceride (mmol/l)	2.05±1.0	1.53±0.71	0.008
HDL-C (mmol/l)	0.75±0.26	1.46±0.59	0.001
LDL-C (mmol/l)	5.25±1.62	3.36±1.46	0.001
Glutathion peroxidase (U/l)	1.27±0.16	1.65±0.27	0.01
Glutathion (Mmol/l)	1.50±0.65	2.93±1.47	0.01
Peroxy nitrate (Mmol/l)	53.43±22.34	28.66±15.25	0.001
Malondialdehyde (Mmol/l)	2.73±1.03	1.52±0.21	0.05
Glucose (mmol/l)	8.10±2.44	5.32±0.82	0.001
Zn (Mg/100ml)	59.78±16.15	80.39±8.49	0.001
Cu (Mg/100ml)	131.29±17.86	98.22±37.26	0.001
Mg (mg/100ml)	1.42±0.74	2.19±0.14	0.01
Co (Mg/l)	0.36±0.03	0.22±0.01	0.012

References

[1] Mahato B., Chaudhary B.L. and Singh B.N. 2016 .Study of Lipid profil, enzymatic antioxidant and oxidative stress in cardiovascular diseases. Int J of Bio Assay.5(8):4759-4762.

[2] Fox S. 2002. Human physiology, 7th ed., McGraw Hill, Boston, pp. 604-606.

[3] Lenihan D.J., Hartlage G. and Decara J. 2016. Cardio-oncology training : aproposal from international cardioncology society and canadian cardiac oncology. *J Card Fail*., 22:465-471.

[4] Montgomery R., Conway T. and Speator A. 1996. Biochemistry, 6th ed., Mosby Publication, New York, pp. 357-384.

[5] Blum A. and Cannon R.O. 1998. Effects of oestrogens and selective oestrogen recepters modulators on serum lipoproteins and vascular function. *Lipidology*., 9(6): 575-586.

[6] Al-Juraisy A.A.M. 2002. Lipid components and fatty acid composition of human serum subjects consuming dairy products and of human mother colostrum and Mature milk, Ph.D.Thesis. Univ. Of Mosul, Iraq.

[7] Bahador F., Pouyan A., Elahe A. and Sofoura K. 2017. Study of lipid profiles in the serum of cardiovascular patients suffering from diabetes. *Bio Med Res*., 28(8): 3397-3401.

[8] Brenna J.Th. 2002. Efficiency of conversion of alpha linolenic acid to long chain n-3 fatty acids in man .*Clin Nutr and Metab*., 52:127-132.

[9] Ettehad D., Emdin C.A., Kiran A. and Anderson S.G. 2016. Blood pressure lowering for prevention of cardiovascular disease and death . *Lancet*, 387: 957-967.

[10] Muclean J., Wionk K., Drayna D., Fielding C. and Lawn R. 1986. Human Lecithin cholesterol acyltransferase gen: complete gene sequence and sites of expression . *Nucl acid Res*., 14(23): 9397-9406.

[11] Laxmi S.M., Karol E.W., Ana Barac M.J. 2018. Cardiovascular disease and breast cancer where these entities intersect. *Circulation*., 137: e30-e66.

[12] Sanchez-Muniz, F.J. Merinero M.C., Rodriguez Gil S., Ordovas J.M., Rodenas S. and Cuesta C. 2002. Dietary fat saturation affects apolipoprotien AII levels and HDL composition in postmenopausal women. *Am Soc Nutr Sc*., 132: 50-54.

[13] Ikeda I. and Sugano M. 1998. Inhibition of Cholesterol absorption by plant sterols for mass intervention. *Current Opinion in Lipidology*., 9: 527-531.

[14] Ossoli A., Pavancllo Ch. and Calabresi L.2016a. High Density lipoprotein Lecithine - cholesterol acyltransferase and atherosclerosis. *Endo metab*., PI 2093-596X, 1-7.

[15] Clay, F.S. and Ira J.G., 2016. Textbook of endocrinology. 13th ed., Elsevier B.V., UK.354.

[16] Ahmad SB., Miller M. and Hanish S., 2016. Sequential kidny-Liver transplantation from the same living donor for LCAT . *Clin Trans plant*., 30(10): 1370-1374.

[17] Samarghandian S., Azimi-Nezhad M., Bolji A., and Farkhoudeh T. 2016. Effect of Crocin on aged rat kidney through inhibition of oxidative stress and pro inflammatory state. *Phytother Res* . 30(8): 1345-1353.

[18] Virtanen J.K., Mursu J., Virtanen H.E. and Fogelholm M. 2016. Associations of egg and cholesterol intakes with carotid intima – media thickness and risk of incident coronary artery disease according to apolipoprotein E phenotype in men. *Am J Clin Nutr*., 103:895-901.

[19] Levy L. and Tedstone A. 2017. UK dietary policy for the prevention cardiovasular disease. *Health Care*., 5(1):9.

[20] Azimini M., Farkhondeh T. and Samar S. 2016. The effect of zataria multiflora on blood glucose, lipid profile and oxidative stress parameters in adult mice during exposure to bisphenol A. *Cardio vas Hematol*., 16(1): 41-46.

[21] Damen J.A.A., Hooft L., Schuit E. and Debray T. 2017.Prediction models for cardiovasclar disease risk in the general population. *B M J*., 353: 212-222.

[22] Norris G.H, Poter CM. and Jiang C. 2016. Dietary Sphingonyelin attenuates hepatic steatosis and adipose tissue inflammation in high fat diet induce obese mice. *J Nutr Biochem*., 40: 36-43.

[23] Manabe M., Abe T., Nozawa M., Maki A., Hirata M.Z. and Itakura, H. 1987. New substrate for determination of serum lecithin: cholesterol acyltransferase . *J of Lip Res*., (28): 1206-1215.

[24] Png K., Halberg N., Yoshida M., Tavazoie S.A.(2011). Micro RNA regulon that mediates endothelial recuitment and metastastasis by cancer cells. *Nature*., 481:7380.

[25] Hoo S.P., Loh, Q.L. and Yue Z .2013. Preparation of asoft and interconnected macroporous hydroxy propyl cellulose mathacrylate scaffold for adipose tissue engineering. *J Mater Chem.,*13:1-12.

[26] Pistorius K. and Axelrod B. 1973. Iron an essential component of Lipoxygenase .Th*e Journal of Biological Chemistry*,249 (10): 3183-3186.

[27] Rotruck J.T., Pope A.L., Gaanther H.E., and Swanson A.B. 1984. Selenium biochemical roles as a component of glutathione peroxidase. *Science*., 17: 588-590.

[28] Burtis C.A, Ashwood E.R. and Bruns D.E. 2012. Tietz textbook of clinical chemistry and molecular diagnostics. By saunders, an imprint of Elsevier Inc.USA.

[29] Kostner G.M. 1976 . Enzymatic determination of cholesterol in high density lipoprotein fraction prepered by polyanion precipitation. *Clin Chem*., 22(5): 698.

[30] Fischbach F. 2000 . A manual of laboratory and diagnostic tests. 6th ed., Lippincott Williams and Wilkins, USA., 472.

[31] Sedlak J. and Lindsay R.H. 1986. Analytical biochemistry.P.192.cited by Al-Zamle O.M., Al-Nimer M.S, and AL-Muslih R.K, 2001. Detection

the level of level of peroxynitrite and related with antioxidant status in the serum of patients with acute myocardial infraction. *Nat J Chem*., 4: 625-637.

[32] Vanuffelen B.E., Van Derzo J. and Dekoster B.M. 1998. Biochem J. 330. 719. Cited by Al-Zamely *et al* .(2001) .

[33] Muslih R.K., AL-Nimer, M.S. and AL-Zamely O.M.Y. 2002. The level of malondialdehyde after activation with (H2O and CuSO4) and inhibition by disferoxamine myocardial infraction. *Nat J of Chem.,* 5: 139-148.

[34] Ossoli A., Simonelli S., Vitali C., and Caabresi L. 2016. Role of LCAT in Atherosclerosis. *J Athero Theromb*., 23(2): 119-120.

[35] Navab M., Reddy S.T., Meriwether D., Fogelman S. and Fogelman A.M. 2015. Apo AI Mimetic peptides A review of the present status , in apolipoprotein mimetics in the management of human disease. *SPA Int Pub*., 2: 15-27.

[36] Casteleijn M.G., Parkkila P., Vijtala T. and Kovuniemi A.2017. Interaction of lecithin-cholesterol acyltransferase with lipid surfaces and apolipoprotein A-I derived peptides implication for the cofactor mechanisim of apolipoprotein A-I.

[37] Cordon S.M. and Davidson W.S. 2012. Apolipoproteins A-I.mimetics and high-density lipoprotien function. *Curr Opin End Diab Obes*., 19(2): 109-114.

[38] Ding X., Li B., Tian F., Zhou SH. and Chen Y. 2017.Effect of blood lipids stability on progression of caroted atherosclerosis. *Pak J Med Sci*., 33(3): 599-602.

[39] Ogedegbe H.O. 2002. Apolipoprotein AIU/B, ratios may be useful in coronary hearted disease risk assessment. *Lab Med*., 33(10): 789-793.

[40] Ooi B.K., Goh B.H., and Yap W.H. 2017. Oxidative stress in cardiovascular disease : involvement of Nrf2 antioxidant redox signaling in Macrophage foam cells formation. *Int J Mol Sci*., 18(2336): 1-16.

[41] Conway JP. and Kinter M. 2006. Dual role of peroxiredoxin in macrophage-derived foam cells. *J Biol Chem*., 281: 27991-28001.

[42] Hu F.B., Mason J.E. and Willett A.J. 2001. Types of dietary fat and risk of cornary heart diseas. *J Am Coll Nutritiuon*., 20(1): 5-19.

[43] Rifkin M.R.1978. Identification of tryponoicdal factor in normal human serum : high density lipoprotein. *Proc Nat Acta Sci*. 75:3450.

[44] Harvey R.A., Clark M.A., Finkel R., Rey J.A. and Whalen K., 2017, lippin cott's illustrated reviews: Biochemistry. 6th ed., lippincott williams and wilkins philadelphia, USA, p.230.

[45] Chen B., Lu Y., Chen Y. and Cheng J. 2015. Therole of NrF2 in oxidative stress-induced endothelial injuries . *J Endocrinol*., 225: R83-R99.

[46] Bansal M., Condra S.K., Nair T., Lyenger S., Gupta R. and Manchanda S. 2016. Consensus statement on the management of dyslipidemia in Indian subjects: our perspective. *Ind Hear J*., 68(2): 238-241.

[47] Pignatelli P., Menichelli D., Pastori D. and Violi F. 2018.Oxidative stress and cardiovascular disease : new insights. *Nat Inst Of Heal*., 76(4): 713-722.

[48] Moldogazieva N.T., Mokhosoev I.M., Feldman N.B. and Lutsenko S.V. 2018. ROS and RNS Signalling: adaptive redox switches through oxidative /nitrosative protein modifications. *Free Rad Res*., 52(5): 507-543.

[49] Zhang M., Zhang B.H., Chen L. and An W. 2002. Overexpression of hem oxygenase-1 protects smooth muscle cells against oxidative injury inhibits cell proliferation. *Cell Res*., 12: 123-132.

[50] Zuzak E., Horecka A., Kietczykowska M., Dudek A., Musik I., Kurzepa J. and Kurzepa J. 2017. Glutathion level and glutathione reductase activity in serum of coronary heart disease patients. *J Pre Clin and Clin Res*., 11(2): 103-105.

[51] Pizzino G., Irrera N., Gucinotta M., Pallio G., Mannino F., and Arcoraci V. 2017. Oxidative stress Harms and Benifits for Human Health. *Oxid Med Cell Long*., 2017: 8416763.

[52] Suleyman D., Mustafa Y., Mchmet K., Natan A., Divler A. and Ahmet A. 2003. Role of free radicals in peptic ulcer and gastristies. *Turk J Gastroenterol*., 14(1). 39-43.

[53] Forstermann V., Xia N. and Li H. 2017. Role of oxidative stress and oxide in the pathogenesis of atherosclerosis. *Circ Res*., 120(4): 713-735.

[54] Kattor A.J., Pathineni N.V.K., Palagiri D. and Mehta J.L. 2017. Oxidative stress in atherosclerosis , *Curr Atheroscler Rep*.,19(11): 42.

[55] Thammi TH.J., Rana Md. M, and Uiislan M. 2017. Lipid peroxidation and antioxidant status in Bangladeshi myocardial infraction patients. *Int J Res in Phar and Bios*., 4:1-6.

[56] Wolonciej M., Milewska E. and Roszkowska Jakimiec W. 2016. Trace elements as an activator of antioxidant enzymes. *Post High 1Med Dosw (onlin)*., 70(0): 1483-1498.

[57] Sabhakumari K.N., Reshmy G.S. and Sajitha K.P. 2015. Evaluation of antioxidant status in myocardial infraction in diabetic and non-diabetic subjects: Acomparative study. *Adv Diab and Metabolism.*, 3(1): 1-6.

[58] Edwards C.R.W., and Bouchier I.A.D. 1999. Davidson's principles and practice medicine 18th ed., Churchill livingstone, London. 724-764.

[59] King J.c., Shames D.M. and Woodhouse L.R. 2000. Zinc homeostasis in humans .*J Nutr*.,130: 1360s-1366s.

[60] Pan Z., Choi S., Ouadid – Achidouch H., Yang J.M., Beattie J.H. and Korichnerva I. 2017. Zinc transporters and dysregulated channels in cancers. *Front Bio Sci*., 22: 623-624.

[61] Freltas E.P., Cunha A.T., Aquino S.L., Predosa L.F., Lima S.C. and Lima J.G. 2017. Zinc status blomarkers and cardiometabolic risk factors in

metabolic syndrome acase control study. *Nutrient.*, 9: E175.

[62] Choi S., Liu X. and Pan Z. 2018. Zinc defeicincy and cellular oxidative stress: Prognostic implication in cardiovascular disease. *Acta Phar Sinica.*, 39: 1120-1132.

[63] Chowdhury R., Ramond A., Okeeffe L.M., Shahzad S., Kuntsor S. and Muka T. 2018. Environmental toxic metal contaminats risk of cardiovascular disease: systematic review and meta – analysis. *Research.*, 362: 1-10.

[64] Jamova K. and Valko M. 2011. Advances in metal induced oxidative stress and human disease. *Toxicology.*, 283:65-87.

[65] Manto M. 2014. Abnormal copper homeostasis mechanisim and roles in neurodegeneration. *Toxics.*, 2:327-345.

[66] Cunha A.R., Marques B.C., d'El-Rei J. and Neves M.F. 2018. Magnesium and cardiovascular

disease a mini review. *J Cariol and cardiovasc Ther.*, 10(5): 1-2.

[67] Dinicolantonio J.J., Liu J. and O'keefe J.H. 2018. Magnesium for the prevention and treatment of cardiovascular disease. *ofen heart.*, 5:1-10.

[68] Kaya Y., Demir H., Asicioglu E. and Keskin S. 2011. The correlation of serum trace elements and heavy metals with carotide artery atherosclerosis in maintenance hemodialysis patients. *Biol Trac Elem Res.*,144:351-359.

[69] Baskaran R., Kalaiselvi P., Huang Ch.Y. and Padma V.V. 2015. Neferine, abisbenzylisoquinoline alkaloid, offers protection against cobalt chloride mediated hypoxia-induced oxidative stress in muscle cells. *Integr Med Res.*, 4:231-241.

[70] Moniz S., Hodgkinson S. and Yates P. 2017.Cardiac transplant due to metal toxicity associated with hip arthroplasty. *Arth Tod.*, 3:151-153.

دراسة انزيم LCAT وبعض المتغيرات الكيموحيوية في مصل دم مرضى القلب والاوعية الدموية

امل طه ياسين الجريسي[1] , اميرة عزيز محمود الجريسي[1] , نادية احمد صالح[2]

[1] قسم الكيمياء ، كلية التربية للعلوم الصرفة ، جامعة الموصل ، الموصل ، العراق

[2] قسم الكيمياء ، كلية التربية للعلوم الصرفة ، جامعة تكريت ، تكريت ، العراق

الملخص

تناولت الدراسة السريرية لأشخاص(ذكور واناث) مصابين بأمراض القلب والاوعية الدموية بأعمار تراوحت بين 25–75 سنة، فقد تم تقدير فعالية انزيم ليسيثين : كوليستيرول أسايل ترانسفريس (LCAT) وفعالية انزيم كلوتاثايون بيروكسيديز (GPx), كما تم تقدير مستوى كل من البروتين المرافق AI (apo AI) والبروتين المرافق B100 (apo B100) والكوليستيرول الكلي (TC) والكليسيريدات الثلاثية (TG) وكوليستيرول البروتين الدهني عالي الكثافة (HDL-C)، كوليستيرول البروتين الدهني واطيء الكثافة (LDL-C) والكلوتاثايون (GSH) والبيروكسي نايتريت (PN) والمالوندايالديهايد (MDA) والكلوكوز (Glu), كذلك تم تقدير مستوى كل من العناصر Zn و Cu و Co و Mg في مصل الدم. بينت النتائج التي تم التوصل إليها وجود انخفاض معنوي في فعالية LCAT و GPx وفي مستوى كل من apo AI و HDL-C و GSH و Zn و Mg ، في حين كان هناك ارتفاع معنوي في مستوى كل من apoB100 و TC و TG و LDL-C و PN و Glu و Cu و Co لكلا الجنسين. أما بالنسبة لـ MDA فتشير النتائج الى وجود ارتفاع معنوي لدى إلاناث في حين لم يكن الارتفاع معنويا لدى الذكور.