Journal of Basrah Researches (Sciences) **49**(1), 159 (2023)
DOI: https://doi.org/10.56714/bjrs.49.1.14

Estimation of some physiological biomarkers in hyperlipidemic Patient men in Al- Zubair General Hospital / Basrah province, Iraq

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A R T I C L E I N F O

Received 26 September 2022
Accepted 28 November 2022
Published 30 June 2023

K e y w o r d s :

Hyperlipidemia, VCAM-1, ox-LDL , MDA, GPX-1.

Citation: A. S. Zghair et al., J. Basrah Res. (Sci.) **49**(1), 159 (2023).
DOI:https://doi.org/10.56714/bjrs.49.1.14

A B S T R A C T

Hyperlipidemia is a serious public health problem that affects many People in the world's, and it has been classified as one of the five causes of death in the world. As indicated by the World Health Organization (WHO), in 2030, hyperlipidemia incidence may increase the cardiovascular diseases to approximately 23.6 million people in the world. The aim of the study: Estimation of some physiological biomarkers levels in the serum of hyperlipidemic patients to predict atherosclerotic and cardiovascular disease. In addition to determine the effect of smoking and body mass index on the levels of biomarkers in hyperlipidemic patients.Patients and Methods: The study was conducted on patients who visited the internal consultation unit at Al-Zubair Hospital of Basra Health Directorate during the period from October 2020 to February 2021. The study was applied to a random sample of 60 hyperlipidemia men and 21 volunteers as a health group for comparison, whose ages range from 20-60 years. RESULTS: The results showed a significant increase in the levels of both Vascular cell adhesion molecule-1 (VCAM-1) and Malondialdehyde (MDA) and a significant decrease in the level of Glutathione Peroxidase-1 (GPX-1) in smoking patients compared to the healthy group. While Oxidized low-density lipoprotein (ox-LDL) did not show a significant difference between the patients smokers and healthy group. While the results showed a significant increase in the levels of VCAM-1, ox-LDL and MDA and a significant decrease in the level of GPX-1 among patients in the overweight and obese groups compared with the healthy group. The results did not show significant differences between the patients of the normal weight category and the healthy group.

1. Introduction

Hyperlipidemia is defined as a heterogeneous group of disorders characterized by an increase in one or more concentrations of lipids in the blood plasma represented by cholesterol, cholesterol

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N: 1817-2695 (Print); 2411-524X (Online)
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esters, triglycerides and phospholipids, or caused by an increase in the concentrations of lipoproteins in the plasma, including low-density lipoprotein. (LDL) and low high-density lipoprotein (HDL) levels [1,2].In general, hyperlipidemia was classified into two main types [3] : primary hyperlipidemia, also called familial hyperlipidemia, which results from a genetic defect that may be monogenic or multiple gene, and the second type is called Secondary hyperlipidemia is caused by some diseases, including diabetes, kidney failure, thyroid activity, and high blood pressure, or it is caused by environmental factors related to unhealthy lifestyle habits, such as drinking alcohol and smoking, or as a result of side effects from taking some medications [4,5]. Biomarkers are tools for measuring and evaluating some natural and pathological biological processes, such as measuring levels of cholesterol, triglycerides, sugar level, blood pressure, obesity, and cardiovascular diseases[6].Vascular cell adhesion molecule-1 (VCAM-1) or Cluster of differentiation 106 (CD106) is a glycoprotein belonging to the immunoglobulin (Ig) family with a molecular weight of 90 kDa and was located in 1989 on the surface of endothelial cell [7,8]. Its expression is also activated by pro-inflammatory cytokines including tumor necrosis factor-alpha (TNF-α), reactive oxygen species (ROS), oxidized low-density lipoprotein ox-LDL, high glucose concentration and blood flow disturbance[9]. Also, VCAM-1 is expressed in high levels of inflammation and in chronic conditions in some diseases, on cell surfaces, including macrophages, dendritic cells, bone marrow fibroblasts, myoblasts, oocytes, Kupffer cells, Sertoli cells, and cancer cells [10,11]. Structurally, the human vascular cell adhesion molecule-1 consists of three domains, the first being extracellular, which in turn consists of six or seven immunoglobulin (Ig)-like domains that contain disulfide-linked loops at the galectin-3-binding N-glycosylation site, and The transmembrane and cytoplasmic domain (C-terminal domain) [12] is composed of 19 amino acids and exhibits complete homogeneity between species (human, mouse and rabbit) and is anchored to the protein Ezrin-radixin-moesin in the apical membrane of cells The blanket [13,14]. Since hyperlipidemia is a type of metabolic syndrome, which is characterized by high levels of total cholesterol and triglycerides, and it is one of the most basic factors in the expression of vascular adhesion molecule-1 in hyperlipidemia patients, who recorded a significant increase in vascular adhesion molecule-1 compared with The healthy group, which plays a key role in atherosclerosis and vascular diseases [15]. Oxidized low-density lipoprotein (ox-LDL) was first described by [16] as an antigenic variant, structurally similar to low-density lipoprotein (LDL) and containing in addition to ApoB-100 another protein called apo -A1 is a glycoprotein that binds to the lipoprotein ApoB-100 through a single disulfide bond [17]. Since 25 years ago, oxidized low-density lipoprotein was studied and many properties were attributed to it that cause atherosclerosis and vascular diseases. However, its properties have not been accurately identified due to the change in its sources, composition, storage and use [18] as it is an indicator of hyperlipidemia and one of the main risk factors in the development of atherosclerosis, as it consists of a heterogeneous group of particles estimated to have a mass of about 3000 kDa and a diameter of 220 nm [19]. It consists of a hydrophobic center consisting of triglycerides and cholesterol esters, and the outer hydrophilic layer consists of phospholipids, cholesterol and one copy. Apo-B lipoprotein has a value of about 500 kDa [20].Glutathione peroxidase GPx-1 was first described in 1957 as an erythrocyte enzyme that protects hemoglobin from oxidation [21]. It belongs to the glutathione peroxidase family and is one of the main antioxidant enzymes in humans. It consists of eight forms, from GPx-1 to GPx-8. The main role of these enzymes is to convert hydrogen peroxides and lipids into their reduced form of water (H2O) and alcohol (LOH). Respectively [22], glutathione peroxidase-1 is one of the most abundant family members in cells of the body with an estimated molecular mass purified of four nearly identical subunits of 22-23 kDa between 83-95 kDa [23,24]. The role of glutathione peroxidase-1 in cardio protection by preventing oxidative stress-induced atherosclerosis [25,26]. Malone Dialdehyde (MDA) is a water-soluble organic compound with the chemical formula CH2 (CHO) 2 present mainly in the form of Enol , It is a by product of fat metabolism in the body and is a highly reactive compound. It is one of the compounds that cause toxic stress in cells as a result of lipid oxidation, which is a complex process where different active compounds lead to cell damage. Therefore, malondialdehyde is used for biomonitoring in vitro and in vivo studies as a key biomarker for different types of diseases, including hypertension. Blood, diabetes, arteriosclerosis, heart failure and cancer[27].

2. Materials and working methods

2.1. Patient group

Sixty serum samples were obtained from men with hyperlipidemia, and the samples were divided on the basis of smoking status into: 30 serum samples for smoking men, and 30 serum samples for non-smoking men. Also divided on the basis of body mass index: 20 serum samples for men from Normal weight BMI ranged (18.5-24.9kg/m²) and 20 serum samples for over weight men BMI ranged (25-29.9 kg/m²) and 20 serum samples for Obesity men BMI ranged (more than 30 kg/ m²). Samples were collected from the Internal Consultation at Zubair General Hospital.

2.2. Health group

Twenty-one serum samples were obtained from healthy men after confirming that they had not been diagnosed with hyperlipidemia.

2.3. Serum preparation

From each man, 5 ml of venous blood was drawn from the ulnar vein using a medical syringe, then the blood was collected in a gel tube and left for 15 min. Then, the tube was placed in a centrifuge at 3500 rpm for 15 min to obtain serum. The obtained serum was sectioned and collected in 0.5 ml Eppendorf tubes, and the samples were stored at −20 °C in deep freezing until tests were performed.

2.4. Estimation of the concentration of biomarkers

Concentrations of some biomarkers (VCAM-1, ox-LDL, MDA, and GPX-1) were estimated using a well-known immunoassay (enzyme-linked immunosorbent assay (ELISA)) using an ELISA reader of American origin and biomarker kits provided by Elabscience-USA.

3. Statistical Analysis

Statistical analysis of the data was performed using a T-test and analysis of variance (ANOVA) for least significant difference in LSD at $P \leq 0.05$. The standard deviation was calculated using SPSS version 23.

4. Results

4.1. Total cholesterol and triglycerides

The results showed a significant increase at the probability level ($P \leq 0.0001$) in the level of cholesterol in hyperlipidemia patients (255.68 ± 5.001 mg/dL) compared to its level in the healthy group (142.71 ±7.870 mg/dL). The level of triglycerides increased significantly in patients (325.02 ± 15.291 mg/dL) at the level of probability ($P \leq 0.0001$) compared to its level in the healthy group (96.05 ± 8.350 mg/ dL) as shown in Table (1)

Table 1 . Serum cholesterol and triglyceride levels in patients group compared to healthy controls.

Parameters	mean ± standard deviation	
	Patient group, number = 60 samples	**healthy group, number = 21 samples**
Cholesterol mg/dL	*255.68 ± 5.001	142.71 ± 7.870
Triglycerides mg/dL	*325.02± 15.291	96.05 ± 8.350

* Significant difference at the probability level of $P \leq 0.05$ in the groups.

4.2. Smoking and levels of biomarkers

The results showed a significant increase in the levels of VCAM-1 (141.92 ±6.399 ng/ml) (P≤ 0.0001)) and MDA (1049.40 ± 140.5 pg/ml) (P≤ 0.0001)) and a significant decrease in the level of GPX-1 (64.00 ±8.364 pg/ml)(P≤ 0.0001) in patients compared to its level in healthy people compared to its level in the control group. While the results showed that there was no significant difference in the level of ox-LDL between smokers and the control group. The results also showed that there were no significant differences in the levels of VCAM-1, ox-LDL, GPX1 and MDA between non-smoking and control patients, as shown in Table (2).

Table 2. Relationship between smoking and levels of biomarkers in the serum of patients and healthy subjects.

Parameter	mean ± standard deviation			
	non smoker		Smoker	
	healthy n = 11	Patients n=30	healthy n = 10	Patients n=30
VCAM-1 (ng/ml)	101.55 ± 8.40	130.13±4.7	89.33 ± 17.87b	141.92 ± 6.399a
ox-LDL (pg/ml)	370.20±47.626	587.50±73.55	310,50 ±88.99	601.21 ± 96.94
GPX1 (pg/ml)	167.57 ± 28.28	78.21 ± 11.6	350.67±41.85b	64.00 ± 8.364a
MDA (pg/ml)	577.13 ± 44.91	748.42 ±65.96	562.0 ± 48.00b	1049.4 ± 140.5a

Letters a, b significant difference at p ≤ 0.05 probability level in the groups.

4.3. Obesity and levels of biomarkers

The results of the current study in Table (3) showed that there was no significant difference among patients of the (normal weight) category in the levels of VCAM-1, OX-LDL, GPX1 and MDA compared to their levels in healthy controls .While it showed a significant increase in the (Over weight) patients in the levels of VCAM-1 (146.78 ± 4.5 ng/ml) and ox-LDL (785.15 ± 78.7 pg/ml) and a significant decrease in the level of GPX1 (70.17 ± 8.154 pg/ ml) (P≤ 0.042) and there was no significant difference in the level of MDA between patients and healthy controls. As for patients with the (obesity) group, the levels of both VCAM-1 (147.22 ± 11.2 ng/ml) (P≤0.0001) and ox-LDL (1374.80 ± 181.5 pg/ml) (P≤ 0.0001) and MDA (879.83 ± 126.3pg/ml) (P≤ 0.0001), and a significant decrease in the level of GPX1 (50.67 ± 6.9 pg/ml) (P≤ 0.0001), compared to their levels in healthy subjects**.**

Table 3. Relationship between obesity and levels of biomarkers in the serum of patients and healthy subjects.

Parameter	mean ± standard deviation					
	Normal weight (18.5-24.9kg/ m²)		**Over weight(25-29.9 kg/ m²)**		**Obesity(more than 30 kg/ m²)**	
	healthy n = 7	**Patients n=20**	**healthy n = 7**	**Patients n=20**	**healthy n = 7**	**Patients n=20**
VCAM-1 (ng/ml)	102.67 ± 8.6	132.50± 6.9	96.30± 13.7b	146.78± 4.5a	104.33 ± 8.2b	147.22± 11.2a
OX-LDL (pg/ml)	328.75 ±90.6	744.01±130.6	172.20± 58.7b	785.15± 78.7a	218.33 ± 74.6b	1374.80± 181.5a
GPX1 (pg/ml)	214.14 ± 48.6	106.61 ± 8.5	244.01 ± 43.4b	70.17 ± 8.154a	268.50 ±35.6b	50.67a ± 6.9a
MDA (pg/ml)	442.60 ± 48.1	781.10 ± 62.5	534.25 ± 40.6	894.75 ± 87.7	212.00 ±58.0b	879.83 ± 126.3a

Letters a, b significant difference at $p \leq 0.05$ probability level in the groups.

5. Discussion

It is obvious from the current finding that the level of cholesterol and triglycerides are higher in pateiint group comparing with healthy volunteers. This result can be attributed to many reasons such as unhealthy diets represented by eating foods rich in saturated fats and practicing improper food preparation habits such as excessive cooking, which leads to the destruction of nutrients like folic acid, deep frying and re-frying in the same oil leads to the formation of unsaturated fatty acids and their accumulation in fatty tissues as well as lack of physical activity in turn contributes to an increase in hyperlipidemia [28,29].The results of the current study showed a significant increase in the level of vascular cell adhesion molecule-1 (VCAM-1)) in hyperlipidemia patients compared to the healthy group. The current study was consistent with the study of [30], which indicated that the reason for the high level of vascular cell adhesion molecule-1 (VCAM-1)) is due to the high levels of oxidized low-density lipoprotein (ox-LDL) resulting from Oxidation of low-density lipoprotein (LDL) as a result of elevated levels of reactive oxygen species (ROS) [31]. The oxidized low-density lipoprotein (ox-LDL) binds to scavenging receptors (SR-1) causing activation of endothelial cells [32]. The nuclear transcription factor kappa (NF-κB) is thus sequestered in the cytoplasm allowing its translocation and transcription by the inhibitor IκB kinase β (IKKβ) which phosphorylates the p65 subunit of nuclear transcription factor NF-κB on S536 depends on the enzyme Focal adhesion kinase (FAK) [33].Thus, the change in endothelial cell phenotype and through the activation of transcription factors such as transcription factor Nuclear ((NF-κB m It can induce the expression of inflammatory adhesion molecules such as vascular cell adhesion molecule-1 (VCAM-1) [34].Regarding the effect of smoking on the level of vascular cell adhesion molecule-1 (VCAM-1)), the results of our current study showed a significant increase in the level of vascular cell adhesion molecule-1 (VCAM-1)) in hyperlipidemia patients. Which came in line with the study of [35] which showed that smoking is one of the stimulating factors for the expression of vascular cell adhesion molecule-1 (VCAM-1)) because it contains more than 5000 different chemical products in cigarette smoke, including Polycyclic aromatic hydrocarbons, oxidizing agents and nicotine [36]. Smoking increases reactive oxygen species (ROS) represented by free radicals such as superoxide (O2 •), hydrogen peroxide (H2O2) and peroxynitrite (ONOO-) [37], which contribute to the generation and accumulation of 4-hydroxy-2-nonenal Protein adducts that deplete tetrahydrobiopterin (BH4) as the primary cofactor for

endothelial nitric oxide synthase (e-NOS) by decreasing the expression level of guanosine triphosphate (GTPCH) guanosine triphosphate (alan). Responsible for the biosynthesis of tetrahydrobiopterin (BH4) [38]. Thus, it inhibits the activity of the endothelial nitric oxide synthase (e-NOS) and thus reduces the generation of nitric oxide NO (which plays a major role in inhibiting the expression of vascular cell adhesion molecules), which leads to an increase in the level of vascular cell adhesion molecule-1 (VCAM-1)) in Smoking hyperlipidemia patients.With regard to the effect of obesity and weight gain, the results of the current study showed a significant increase in the level of vascular cell adhesion molecule-1 (VCAM-1)) in the disease compared to the healthy ones, which agrees with the study of [39], which indicated that the high level of the vascular cell adhesion molecule-1 (VCAM-1) Vascular cell adhesion (VCAM-1), may be due to elevated levels of inflammatory cytokines including tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1) and interleukin-6 (IL-6) [40], which are induced by reactive oxygen species (ROS) generated in the mitochondria of fat cells, as a result of increased insulin resistance, which works by decreasing the level of eNOS [41]. It causes loss of NO activity through superoxide dissociation (O2•) due to large amounts of superoxide (O2•) resulting from inflammation. Through the role it plays in the early stages of atherosclerosis [42,43]. The results of the current study also showed a significant increase in the level of oxidized low-density lipoprotein (ox-LDL) in hyperlipidemia compared to healthy subjects, as the current study was consistent with the study of [44], which indicated that the increase in the level of lipoprotein is low. The oxidative density (ox-LDL) in patients came as a result of the decrease in the levels of antioxidants, including Paraoxonase-1 (PON-1), which works to protect low-density lipoprotein LDL from oxidation [45] from the nicotinamide enzyme system Adenine DiThe nucleotide phosphate oxidase (NADPH) and lipooxidative enzymes such as lipoxygenase and myeloperoxidase [46] oxidize LDL and cause changes in the chemical properties, physical structure and bioactivity of LDL particles ,thus oxidized low-density lipoprotein ox-LDL is more reactive with peripheral tissues, leading to inflammation, organ damage and atherosclerosis [47].In the case of obesity and overweight, the results of the current study showed a significant increase in the level of oxidized low-density lipoprotein (LDL) ox-LDL in patients suffering from obesity and overweight compared to the healthy group, as this study was in agreement with the study of [48], which It was conducted on a group of hyperlipidemia patients who are overweight and obese, which showed that the increase in the level of oxidized low-density lipoprotein (LDL) ox-LDL, may be due to the increased secretion of pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) [49,50], which in turn leads to the production of interleukin-6 (IL-6), which is the main regulator of C-reactive protein synthesis in the liver [51], resulting in a low-grade inflammatory state that leads to It leads to the production of free radicals that oxidize low-density lipoprotein LDL and the formation of oxidized low-density lipoprotein ox-LDL, which includes in one of its components lipid peroxide, which is the final indicator of lipid oxidation, inflammation, and atherosclerosis[52] .While the study [53] which was conducted on hyperlipidemia patients who suffer from obesity and overweight and high level of oxidized low-density lipoprotein ox-LDL, which was consistent with our current study. Which showed that the increase in the level of oxidized low-density lipoprotein ox-LDL may be due to insulin resistance, which leads to a decrease in insulin function and inhibition of lipolysis by decreasing the activity of the lipoprotein lipase enzyme, which leads to an increase in the production of free fatty acids FFA, where it occurs This process, after eating a meal rich in fat, is generated from the remnants of chylomicron particles rich in triglycerides [54], which causes an increase in the secretion of hepatic fatty acids and very low-density lipoprotein (VLDL) particles rich in triglycerides (TG). on HDL metabolism by exchanging with TG-rich lipoproteins via cholesterol ester transfer protein (CEPT), to produce HDL particles containing high levels of TG, then hydrolyzing HDL particles containing HDL-TG triglycerides by using the enzyme hepatic lipase to triglyceride TG and HDL, thus becoming the protein High-density lipoprotein HDL is smaller and less active, and is easily removed from the circulation by the kidneys [55]. Small low-density lipoprotein sdLDL levels are elevated in the circulation and oxidized low-density lipoprotein ox-LDL formation, which has shown greater risks in the myocardium [56].The results of the current study indicated a significant increase in the level of MDA in hyperlipidemia patients compared to healthy subjects, as this study was consistent with the study [57], which indicated that the high levels of MDA in hyperlipidemic patients is due to this results in a decrease in the levels of enzymatic antioxidant

systems that provide protection against elevated levels of reactive oxygen species (ROS), peroxidative lipolysis and oxidized low-density lipoprotein [58], including the antioxidant superoxide dismutase (SOD), which acts on The decomposition of the super-anion O2 • to hydrogen peroxide H2O2, which is dissociated into water by the action of the enzyme system glutathione peroxidase [59] The decrease in the level of HDL impairs its ability to protect LDL from oxidation by free radical activity through its low content of lipoprotein (apo-A-I [60] and paroxonase 1 (PON1). Paraoxonase contains the enzyme arylsterase (AE), which is one of the enzymatic activities of paroxonase-1 that plays a protective role against lipid peroxidation and other lipoproteins [61]. and thus the level of MDA increases and the levels of antioxidants, including glutathione peroxidase-1 GPx-1, decrease. The current study also showed a significant decrease in the level of glutathione peroxidase-1 GPx-1 in smoking patients compared to its level in the healthy group, as it was consistent with the studies of each [62,63], which believe that a decrease in the level of glutathione peroxidase). -1 GPx-1 due to the direct damage of hydroquinone and quinine compounds in cigarette smoke that spreads across cell membranes and leads to the formation of semiquinones and subsequently gives the superoxide radical (.-O2), hydroxyl radical (.OH) and peroxyl (ROO)[64,65], which oxidizes proteins, DNA bases and lipids to lipid peroxidation, which degrades to a group of aldehydes that deplete GSH glutathione by modifying the -SH and -NH2 protein groups, leading to a decrease The level of glutathione in the group of patients who smoked [66].The results of the study also showed a significant decrease in the level of glutathione peroxidase-1 in patients with overweight and obese patients compared with healthy subjects, and we did not notice a significant difference in patients of normal weight and the healthy group, which was consistent with studies [67,68], which showed that the increase in weight and obesity, which are the main sources for the production of reactive oxygen species, which represent the excess energy that is provided by the mitochondria, where adipose tissue cells work on The secretion of adipokines that activate the nuclear transcription factor-kappa (NF-κB), which in turn induces phagocytic cells to produce the enzymes xanthine oxidase (NOX), nicotinamide adenine dinucleotide oxidase (NADPH) and the formation of large amounts of free radicals [69] which exceed the ability of antigens. Oxidative stress levels decrease, and thus the decrease in antioxidants, including glutathione peroxidase-1, in patients is associated with obesity[70,71].

6. Acknowledgment

This work is Sponsored by Dept. of Biology, College of Education for Pure Science, University of Basrah. Sincer appreciation to the all the volunteers and wish them good health.

7. References

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تقدير بعض المؤشرات الحيوية الفسيولوجية في مرضى فرط شحميات الدم عند الرجال المرضى في مستشفى الزبير العام ، محافظة البصرة ، العراق

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معلومات البحث

الاستلام 26 أيلول 2022
القبول 28 تشرين الثاني 2022
النشر 30 حزيران 2023

الكلمات المفتاحية

فرط شحميات الدم *ox-VCAM-1, LDL , MDA, GPX-1.*

Citation: A. S. Zghair et al., J. Basrah Res. (Sci.) **49**(1), 159 (2023). DOI:https://doi.org/10.56714/bjrs.49.1.14

الملخص

الخلفية: يعتبر فرط شحميات الدم مشكلة صحية عامة خطيرة تؤثر على الكثير من الناس في العالم ، وقد تم تصنيفها كواحد من خمسة أسباب للوفاة في العالم. كما أشارت منظمة الصحة العالمية (WHO) ، في عام 2030 ، قد يؤدي حدوث فرط شحميات الدم إلى زيادة أمراض القلب والأوعية الدموية إلى ما يقرب من 23.6 مليون شخص في العالم
الهدف من الدراسة: تقدير بعض مستويات المؤشرات الحيوية الفسيولوجية في مصل مرضى فرط شحميات الدم للتنبؤ بأمراض تصلب الشرايين وأمراض القلب والأوعية الدموية. بالإضافة إلى تحديد تأثير التدخين ومؤشر كتلة الجسم على مستويات المؤشرات الحيوية في مرضى فرط شحميات الدم.
المرضى وطريقة العمل: أجريت الدراسة على مرضى زاروا وحدة الاستشارات الداخلية في مستشفى الزبير التابع لمديرية صحة البصرة خلال الفترة من تشرين الأول 2020 إلى شباط 2021. وطبقت الدراسة على عينة عشوائية من 60 رجلاً من مرضى فرط شحميات الدم و 21 متطوعاً كمجموعة صحية. للمقارنة والذين تتراوح أعمارهم بين 20-60 سنة.
النتائج: أظهرت النتائج زيادة معنوية في مستويات كل من جزيء التصاق الخلايا الوعائية 1- (VCAM-1) و Malondialdehyde (MDA) وانخفاضًا ملحوظًا في مستوى الكلوتاثيون بيروكسيديز 1- (GPX-1) في المرضى المدخنين مقارنةً بجزيء التصاق الخلايا الوعائية. مجموعة صحية. بينما لم يظهر البروتين الدهني المؤكسد منخفض الكثافة (ox-LDL) فرقًا كبيرًا بين المدخنين والمرضى الأصحاء. بينما أظهرت النتائج زيادة معنوية في مستويات VCAM-1 و ox-LDL و MDA وانخفاض معنوي في مستوى GPX-1 بين المرضى في مجموعة الوزن الزائد والسمنة مقارنة بالمجموعة السليمة. لم تظهر النتائج فروق ذات دلالة إحصائية بين مرضى فئة الوزن الطبيعي والمرضى الأصحاء.
الاستنتاج: يمكن اعتماد المؤشرات الحيوية كمعايير تنبؤية للمرضى الذين يعانون من فرط شحميات الدم. قد تساعد هذه المعايير في تشخيص المرض المبكر وكعلامات تنبؤية لتطور المرض ويمكن اعتمادها للتنبؤ بالاستجابة العلاجية لفرط شحميات الدم. يمكن أيضا اعتماد هذه المؤشرات الحيوية كتنبؤات خطيرة بحدوث بعض الأمراض مثل أمراض القلب والأوعية الدموية وتصلب الشرايين.