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Assessment of lipase levels and lipid profile in chronic hypertensive patients in Kirkuk, Iraq

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Abstract
This study aims to evaluate the levels of lipase enzyme and lipid profile in patients with chronic hypertension, a topic of particular importance due to its role in understanding the risk factors associated with cardiovascular diseases, especially in a society suffering from high rates of hypertension. The study was conducted in Kirkuk Governorate, Iraq, where blood samples were collected from 60 patients with chronic hypertension and compared with 30 samples from healthy individuals (Control group) of the same age group. The analyses included measuring the levels of lipase, cholesterol, triglycerides, and various lipoproteins (HDL, LDL, and VLDL) using standard techniques for blood analysis and determining the components of the lipid profile.
The results showed a significant decrease in the level of lipase enzyme in patients with hypertension compared to the control group. A significant increase in the levels of triglycerides and VLDL was also observed in the patients, which is associated with an increased risk of atherosclerosis and heart disease. The study also showed an association between obesity and increased levels of cholesterol and triglycerides, which enhances the scientific understanding that excess weight increases the risk of hypertension.

Keywords: Lipase, hypertension, lipids profile

1. Introduction
Blood pressure is a vital characteristic of individuals that is characterized by genetic, environmental, and other factors [1]. Blood pressure is defined as the pressure inside the arteries, which maintains the presence of a compressive force that leads to pushing blood through the circulatory system to all parts of the body. Blood pressure is expressed by two numbers, for example, 80/130 mm of mercury. The lower number is called diastolic pressure, and the higher number is systolic pressure. These are measurements that correspond to the movements of expansion and contraction of the heart muscle [2].
Hypertension is a chronic medical condition caused by elevated blood pressure in the arteries above normal levels [3]. If the diastolic pressure is continuously 90 mm Hg or higher and the systolic pressure is continuously 140 mm Hg or higher, the blood pressure is deemed high, but in general, normal blood pressure for adults (≥18 years) is 120/80 mm Hg [4]. Globally, hypertension is the primary cause of cardiovascular diseases (CVD) and early mortality [5]. Over the past few decades, the prevalence of hypertension and cardiovascular disease has not declined, and CVD continues to be responsible for over 33% of all fatalities worldwide [6].
Lipases, which include phospholipases and triacylglycerol, are enzymes that break down fat. Hydrolysis of triacylglycerol to free fatty acids (Mono- and diglycerides) is catalyzed by triacylglycerol lipase, whereas phospholipases catalyze the hydrolysis of phospholipids [7]. Lingual, gastric, preduodenal, extraduodenal, hepatic, lipoprotein, and the newly identified endothelial lipase are examples of human lipases [8]. The intestinal lumen's ability to digest dietary lipids depends on the exocytic enzyme pancreatic lipase, which is generated by pancreatic acinar cells. Both pancreatic and gastric lipases must hydrolyze dietary triacylglycerols in order for intestinal cells to absorb them, which makes it easier for the body to absorb dietary fats. Instead of a single molecule, the substrate for pancreatic lipase is a non-aqueous phase of aggregated lipids made up of micelles, monolayers, or ester molecule aggregates that interact with an aqueous media [9].
Lipids are known as natural compounds that are not soluble in water but dissolve in organic solvents and are of great importance as they enter into the composition of cell walls and are one of the most important means of obtaining and storing energy, in addition to entering into the composition of hormones [10, 11].

Lipids are classified into cholesterol, triglycerides (TG), and lipoproteins. Cholesterol is known as an important steroid and is found in the body in two forms: free, which makes up 30-40%, and esterified, which makes up 60-70% [12]. It is a soft fatty substance, a type of fat, and is transported in the bloodstream via lipoproteins and does not dissolve in water [13]. Triglycerides (TG) are also non-polar fatty molecules consisting of a glycerol molecule linked to three fatty acid molecules, which represent the main form of fat and energy storage in the body [14]. They are mainly produced through the glycerol phosphate pathway and the monoglycerol pathway [15]. Lipoproteins are one of the most important means of transporting fats from the small intestine via the blood to the liver and then from the liver to fat storage sites or transporting them to other organs of the body [16, 17]. One of the functions of lipoproteins is to provide insoluble fats in the tissues to be formed for storage and metabolism. The structural composition of the lipoprotein molecule consists of a hydrophobic inner part consisting of triglycerides and cholesterol esters and is surrounded by a shell of hydrophilic fats such as phospholipids, whose function is similar to the function of regulators for the dissolution of cholesterol, non-esterified fats, and proteins [18]. Lipoproteins can be classified based on their density, which represents the fat content, which ranges between 75-30%, where the higher the fat content, the lower the density of the lipoprotein. The types of lipoproteins are: high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol [19].

This study aims to evaluate the level of lipase enzyme and lipid classes in detail in patients with chronic hypertension among hypertensive individuals in Kirkuk Governorate, Iraq.

Materials and Methods

60 blood samples were collected from hypertensive patients, and 30 blood samples were collected from apparently healthy individuals (Control group) at Kirkuk Teaching Hospital, whose ages ranged from 50 to 70 years. Patients and healthy individuals were classified into groups according to gender and body mass index (BMI).

Blood was drawn from the vein using a sterile syringe of 5 ml and placed in new sterile plastic tubes containing gel (Gel tube) and left for (15) minutes, after which the blood serum was separated from the clotted part by centrifugation at a speed of 4000 rpm (Round per minute) for (10) minutes, then the clear blood serum was drawn and stored in the freezer at -20 °C until it was used in conducting various analyses according to the study requirements. The body mass index (BMI) was measured by dividing the weight in kilograms by the height in meters squared, and this method was followed for both patients and the control group. According to the World Health Organization, the body mass index (BMI) is classified into the following values: (<18) underweight, (24-18) normal weight, (29-25) overweight, and (≥30) is classified as obesity [20].

The analysis kit (KIT) from the Italian business GIESS DIAGNOSTICS was used to measure the amount of lipase enzyme in serum. This technique is based on the colorimetric substrate 1,2-O-DILAURYL-RAC-GLYCERO-3-GLUTARIC ACID-(6'METHYL-RESORUFIN)-ESTER, which is hydrolyzed under alkaline test conditions to yield the chromophore methylresorufin after being cleaved by pancreatic lipase. The amount of lipase in the sample is proportional to the photometrically determined amount of methylresorufin that is produced [21, 22].

Cholesterol in serum was estimated using the enzymatic method using a ready-made analysis kit from the French company Biolabo [23]. While the concentration of triglycerides in serum was estimated using the analysis kit from the French company Biolabo, this method relies on the enzymatic analysis of triacylglycerol to glycerol, which passes through a series of reactions to eventually produce a pink complex [24].

A pre-made assay kit was used to estimate the amount of HDL in the blood serum. The method's underlying premise is the enzymatic approach, which precipitates the chylomicrons and lipoproteins of LDL and VLDL by adding phosphotungstic acid and magnesium ions. Only HDL is left in the blood serum following centrifugation [25].

The Friedewald formula was used to calculate serum LDL-C and VLDL-C concentrations. Only triacylglycerol concentrations less than 400 mg/dl were acceptable for the formula:

LDL (mg/dl) = Total cholesterol – (HDL + VLDL)

VLDL concentration (mg/dl) = (Triglycerides/5) [26]

The results were statistically analyzed using SPSS version 20, and the arithmetic mean and standard deviation were extracted. The T-Test was also used to compare two groups, and the Anova test was used to compare more than two groups. The significant differences for these groups were chosen at a probability level of $p<0.05$.

Results and Discussion

The study included 60 cases of patients with hypertension (32 males, 28 females) whose ages ranged from 50 to 70 years. These cases were compared with 30 samples of healthy people (17 males, 13 females) and the same age group of patients.

The results included the statistical values of the variables measured in the current study in the serum of patients with hypertension and the control group, as shown in Table 1.

Table 1: Mean±standard deviation of measured variables

Parameter	Control	Patients	P value ≤0.05
	Mean± SD N=30	Mean± SD N=60	
Lipase (U/L)	67.14±8.36	59.72±13.45	0.006
Cholesterol (mg/dl)	165.5±29.64	174.4±47.22	NS
TG (mg/dl)	112.5±26.8	209.72±87.6	0.001
HDL (mg/dl)	37.8±6.32	34.2±11.26	NS
LDL (mg/dl)	103.09±42.17	98±41.72	NS
VLDL (mg/dl)	23.69±6.08	45.23±28.6	0.001

The results shown in Table (1) showed a significant decrease at the probability level ($p\leq0.05$) regarding the activity of lipase enzyme in the blood serum of patients with hypertension (59.72±13.45) compared to the control group (67.14±8.36) as shown in Figure 1.

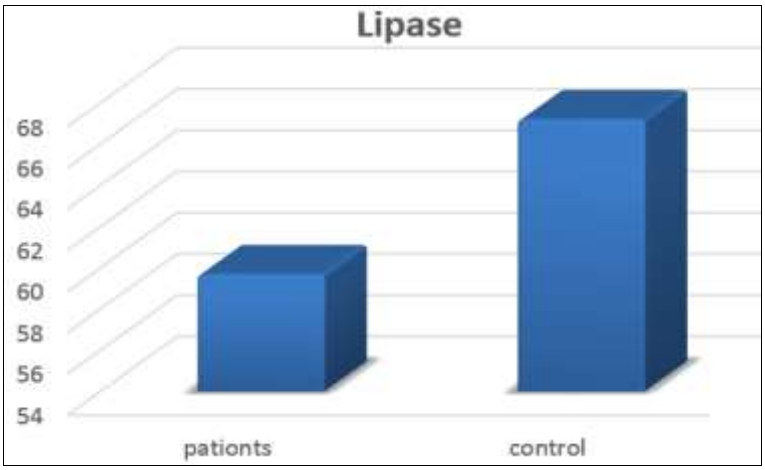

Fig 1: Lipase enzyme levels in patients and control group

Through the results of the current study, we note a significant decrease in the activity of the lipase enzyme between hypertensive patients and healthy people. Previous studies [27, 28] found a relationship between the pancreatic lipase enzyme and blood pressure and hypertension, as the hydrolysis of dietary triglycerides by both pancreatic lipase and lipase is necessary for their absorption by intestinal cells to facilitate the absorption of dietary fats in the body, and high triglycerides are a major risk factor for cardiovascular diseases.

The results shown in Table 1. also showed a non-significant increase at the probability level ($p>0.05$) regarding the level of cholesterol in the blood serum of patients with hypertension (174.4±47.22) compared to the control group (165.5±29.64) as shown in Figure 2.

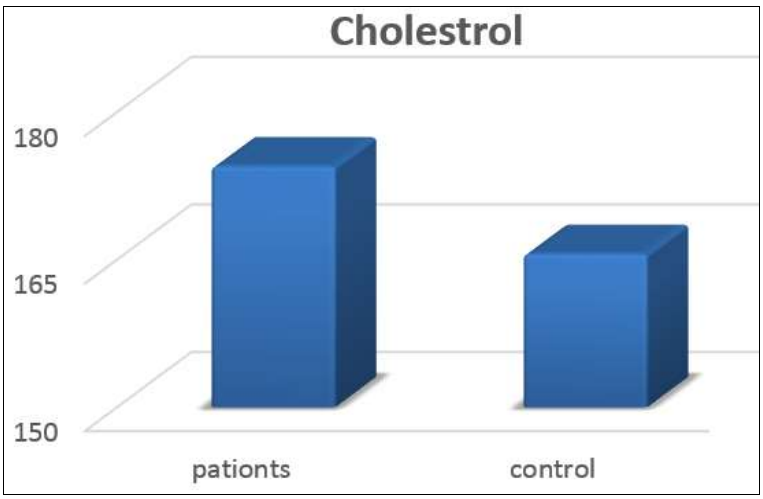

Fig 2: Cholesterol levels in patients and control group

However, the results showed a significant increase at the probability level ($p\leq0.05$) regarding the level of triglycerides in the blood serum of patients with hypertension (209.72±87.6) compared to the control group (112.5±26.8) as shown in Figure 3.

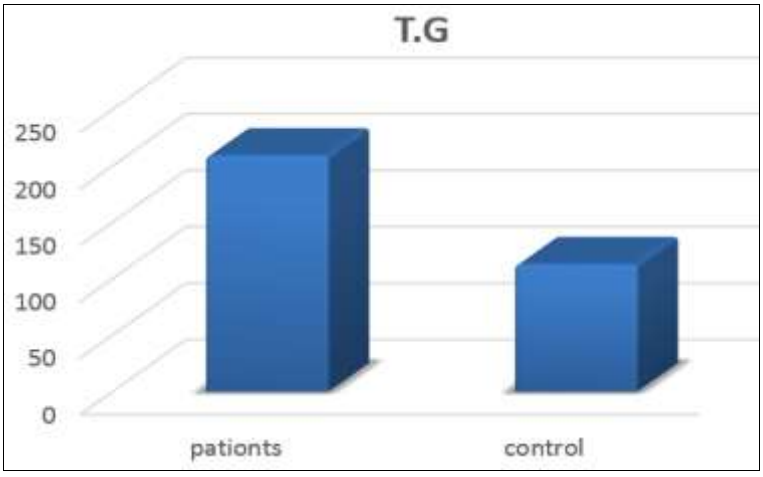

Fig 3: Triglyceride levels in patients and control group

We note a highly significant increase in triglycerides, as their increase in blood pressure patients is due to increased oxidation in the body, which leads to a decrease in the lipoprotein's effectiveness lipase (LPL) enzyme found in various body tissues. This decrease leads to an imbalance in fat levels and an increase in the level of TG in the blood. The percentage of triglycerides in (VLDL) is a large percentage, and therefore the mechanism of the increase in (VLDL) can be explained by the same mechanism of the increase in TG [29]. Numerous pathophysiological diseases and disorders, including diabetes, obesity, hypertension, and non-alcoholic fatty liver disease, can result in hypertriglyceridemia [30, 31]. Elevated TG readings are linked to atherosclerosis and raise the risk of cardiovascular disease even when there are no high TC or LDL-C levels present [32, 33].

The results shown in Table 1. showed a non-significant decrease at the probability level (p>0.05) regarding the level of high-density lipoproteins (HDL) in the blood serum of hypertensive patients (34.2±11.26) compared to the control group (37.8±6.32), as shown in Figure 4.

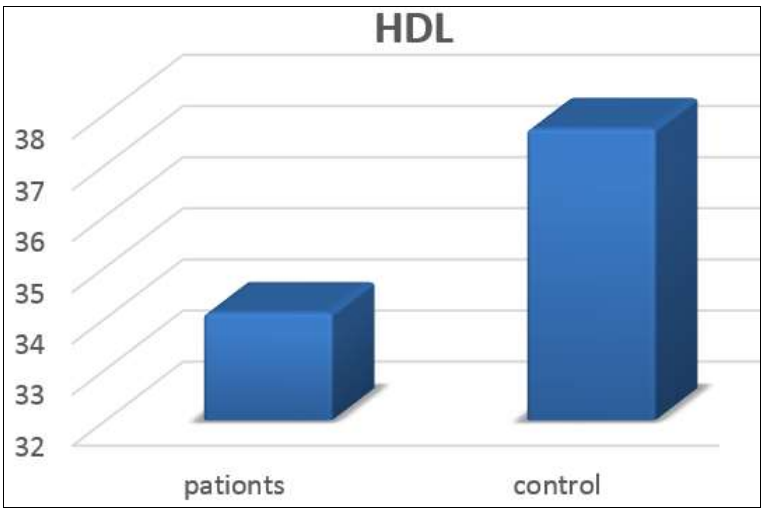

Fig 4: HDL levels in patients and control group

HDL stimulates the production of nitric oxide, a powerful vasodilator. Nitric oxide causes vasodilation, which improves blood circulation and helps maintain normal blood pressure. HDL helps protect endothelial cells from damage and dysfunction [34].

The results also showed a non-significant decrease at the probability level (p>0.05) regarding the level of low-density lipoproteins (LDL) in the blood serum of hypertensive patients (98±41.72) compared to the control group (103.09±42.17) as shown in Figure 5.

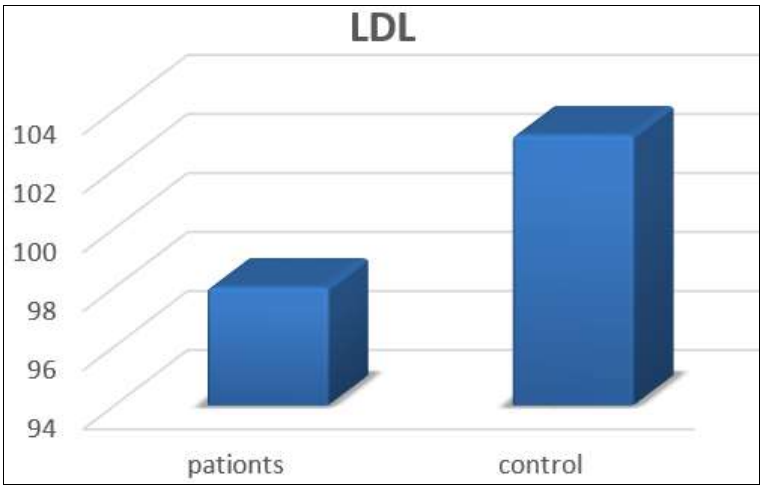

Fig 5: LDL levels in patients and control group

The results shown a significant increase at the probability level ($p \leq 0.05$) regarding the level of very low-density lipoproteins (VLDL) in the blood serum of patients with hypertension (45.23±28.6) compared to the control group (23.69±6.08) as shown in Figure 6. High VLDL levels are a greater risk factor than high LDL because of the binding of VLDL receptors to form a complex with Very Low Density Lipoprotein Receptor (VLDLR) and stimulating the development of atherosclerosis [35]. The percentage of TG in VLDL is high and therefore the reason for its increased concentration is due to increased oxidation in the body, which reduces the effectiveness of the enzyme (LPL) found in various tissues of the body, which causes an imbalance in lipid levels and an increase in the level of TG and thus an increase in the percentage of VLDL in the blood [36]. The results in Table 2. show that there is a relationship between higher BMI categories and hypertension, showing that overweight and obesity may be associated with an increased risk of hypertension in the people studied. This is consistent with what was mentioned in previous studies [37, 38] that showed a relationship between the cause of hypertension and those who are overweight. This is also consistent with [39, 40].

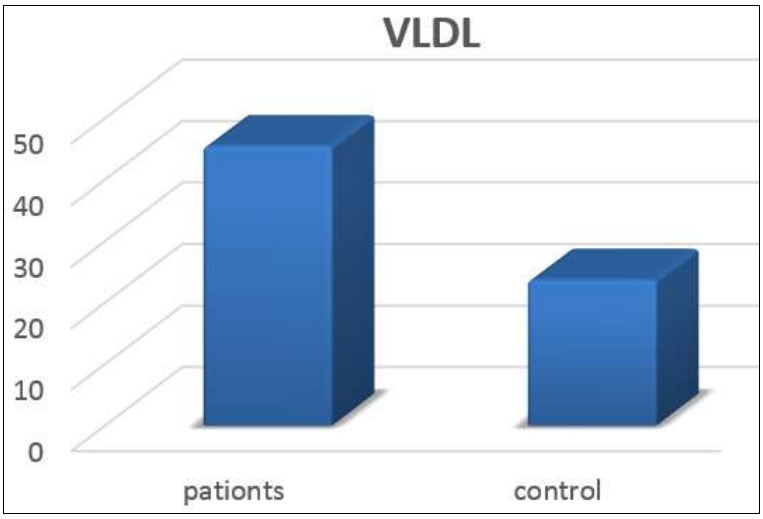

Fig 6: VLDL levels in patients and control group

Table 2: Mean±standard deviation of BMI

		N	Mean±SD G (18-24)	N	Mean±SD G (25-29)	N	Mean±SD G (≥30)
BMI (Kg/m2)	Patients	5	21.5±3	18	27.26±1.75	37	34±3.08
	Control	6	23±1	17	27.25±1.57	7	30±0.786

In this study, BMI was scientifically associated with the prevalence of hypertension, with more obese patients having hypertension compared to the control group. Old and new studies have concluded that obesity is a major risk factor for hypertension [41].

Hypertensive patients' samples were divided into three groups according to the body mass index (BMI) and these groups were compared with the control group as shown in Table 3.

Table 3: Mean±standard deviation of variables according to BMI

Parameters	Mean±SD		
	A Normal weight	B Over weight	C Obese
Lipase	55.50±8.496 b	64.60±14.78a	57.99±11.493b
Cholesterol	170.5±48.21 a	180.3±54.34 a	169.5±41.35 a
TG	186.0±77.94b	230.1±94.57 a	215.9±90.03 a
HDL	40.75±10.66 a	34.32±13.21 b	30.94±11.02 b
LDL	96.55±39.28 a	99.972±44.51 a	97.429±40.56 a
VLDL	37.20±19.963 b	49.98±28.93 a	47.19±27.17 a

The results in Table 3. showed a significant increase in the activity of lipase enzyme in the second category at the probability level ($p \leq 0.05$) when compared with the first and third categories and no significant difference between the first and third categories. The results did not show any significant difference between the three categories in the level of cholesterol and the level of low-density lipoproteins LDL. While the results showed a significant decrease in the level of triglycerides TG and the level of VLDL in the first category at the probability level ($p \leq 0.05$) when compared with the second and third categories. It also showed a significant increase in the level of high-density lipoproteins HDL at the probability level ($p \leq 0.05$) when compared with the second and third categories.

Table 4. shows the effect of the gender factor for hypertensive patients on the level of variables compared to the control group.

Table 4: Mean±standard deviation of variables according to gender

Parameter		Male	P value	Female	P value
		Mean ±SD N=24		Mean ±SD N=36	
Lipase	Patients	60.7±13.8	NS	57.3±12.75	0.04
	Control	67.67±7.38		66±8.4	
Cholesterol	Patients	176.1±51.2	NS	171.7±46.23	NS
	Control	153.6±30.61		179.5±27.65	
TG	Patients	224.8±92.9	0.003	195.3±83.7	0.001
	Control	112.26±35.8		108±19.7	
HDL	Patients	34.2±13.3	NS	35.6±11.6	NS
	Control	37.3±8.9		38.36±4.32	
LDL	Patients	97.9±43.8	NS	98±39.4	NS
	Control	89.26±39.6		115.98±42.87	
VLDL	Patients	47.8±32.58	0.001	39.89±23.7	0.001
	Control	23±7.67		22±3.9	

Through the results of our current study shown in Table 4. we notice a significant decrease in the lipase's activity enzyme in the blood serum of female patients with high blood pressure compared to the control group. We also notice a non-significant decrease in the blood serum of male patients with high blood pressure compared to the control group. This is consistent with the results of some studies [42]. Male patients' blood serum cholesterol levels were non-significantly higher than those of the control group, whereas female patients' blood serum cholesterol levels were non-significantly lower than those of the control group. This runs counter to earlier research [43, 44], which showed a highly significant increase in patients with hypertension compared with the control group. Our current study shows a highly significant increase in the level of triglycerides (TG) in the blood serum of both male and female patients compared to the control group, and it was a show that there was a slight increase in the level of triglycerides in the blood serum of males compared to females. The results of the study agreed with the increase in the level of triglycerides showed what in the blood serum of males and females with hypertension compared to the control group [45].

Hypertriglyceridemia is one factor leading to the development of oxidative stress, which leads to many diseases, including endothelial dysfunction and atherosclerosis. The weakness of the vascular endothelium is associated with increased production of adhesion molecules, reduced endothelial-dependent vasodilation, and nitric oxide production. These are important factors in the development of heart disease and high blood pressure [46]. As shown in our current study, Table 4. shows a non-significant decrease in the level of high-density lipoproteins HDL in the serum of hypertensive patients of both sexes, males and females, compared to the control group. In addition, there was a slight non-significant increase in the level of low-density lipoproteins LDL for male hypertensive patients compared to the control group. We also note a non-significant decrease in the level of low-density lipoproteins for female hypertensive patients compared to the control group.

The results of the current study, Table 4. show a highly significant increase in the level of very-low-density lipoproteins (VLDL) in hypertensive patients for both males and females compared to the control group. The results of the current study agree with the results of Zaid [45]. These results are also consistent with previous studies [47, 48], which showed that dyslipidemia, characterized by high levels of VLDL, is a major causative factor for cardiovascular diseases. Increased VLDL is associated with the risk of hypertension, and very-low-density lipoprotein particles are converted to IDLs (intermediate-density lipoproteins) when they deliver triglycerides to tissues [49].

Conclusion

This study showed a clear relationship between chronic hypertension and changes in lipase levels and lipid profile in patients. The results showed a significant decrease in lipase levels in patients, which supports the hypothesis that decreased activity of this enzyme may be associated with increased fat absorption and accumulation in the body, which may contribute to the increased risk of cardiovascular disease. The study also showed an increase in triglyceride and VLDL levels in the blood of patients. Obesity is also associated with high lipid levels and low HDL levels, showing that excess weight is a major risk factor for hypertension and its worsening health consequences.

References

1. Bencivenga L, *et al*. Blood pressure variability: A potential marker of aging. Ageing Res Rev. 2022;80:101677.
2. Duckitt K, Harrington D. Risk factors for pre-eclampsia at antenatal booking: systematic review of controlled studies. BMJ. 2005;330(7491):565.
3. Brouwers S, *et al*. Arterial hypertension. The Lancet. 2021;398(10296):249-261.
4. Perangin-angin IH, *et al*. Cholesterol and hypertension examination of cholesterol levels and education on hypertension risk control in elderly in Tanjung Anov Village, Deli Serdang District Year 2022. Jurnal Pengabdian Kesehatan (JUPKes). 2023;2(2):48-54.
5. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. Nat Rev Nephrol. 2020;16(4):223-237.
6. Jagannathan R, *et al*. Global updates on cardiovascular disease mortality trends and attribution of traditional risk factors. Curr Diabetes Rep. 2019;19:1-12.
7. Lim SY, Steiner JM, Cridge H. Lipases: it's not just pancreatic lipase! Am J Vet Res. 2022, 83(8).
8. Singh A. Hyperlipidemia in cardiovascular health and digestion. In: Nutrition and Functional Foods in Boosting Digestion, Metabolism and Immune Health; c2022. p. 141-50.
9. Qian D, *et al*. Clinical efficacy and ultrasound inspection of the treatment of hypertensive heart patients with valsarian combined with hydrochlorothiazide. Pak J Pharm Sci. 2018;31(4 SI):1659-1663.
10. Huang B, Song B-L, Xu C. Cholesterol metabolism in cancer: mechanisms and therapeutic opportunities. Nat Metab. 2020;2(2):132-141.
11. Ouweneel AB, Thomas MJ, Sorci-Thomas MG. The ins and outs of lipid rafts: Functions in intracellular cholesterol homeostasis, microparticles, and cell membranes: thematic review series: Biology of lipid rafts. J Lipid Res. 2020;61(5):676-686.
12. Iman A-B, Al Faisal A-H. Association of CYP19A1 rs743572 polymorphism with breast cancer risk factor in Iraqi women-case control study. Cancer. 2020, 66.
13. Nakhaei P, *et al*. Liposomes: structure, biomedical applications, and stability parameters with emphasis on cholesterol. Front Bioeng Biotechnol. 2021;9:705886.
14. Vourakis M, Mayer G, Rousseau G. The role of gut microbiota on cholesterol metabolism in atherosclerosis. Int J Mol Sci. 2021;22(15):8074.
15. Kim SH. Cardiovascular risk factors and obesity in adolescents. Korean Circ J. 2020;50(8):733-735.
16. Björnson E, *et al*. Apolipoprotein B48 metabolism in chylomicrons and very low-density lipoproteins and its role in triglyceride transport in normo- and hypertriglyceridemic human subjects. J Intern Med. 2020;288(4):422-438.
17. Li H, *et al*. Hepatic cholesterol transport and its role in non-alcoholic fatty liver disease and atherosclerosis. Prog Lipid Res. 2021;83:101109.
18. Lepedda AJ, *et al*. Proteomic analysis of plasma-purified VLDL, LDL, and HDL fractions from atherosclerotic patients undergoing carotid endarterectomy: identification of serum amyloid A as a potential marker. Oxid Med Cell Longev. 2013;2013.
19. Moosaie F, *et al*. Lipoprotein (a) and apolipoproteins as predictors for diabetic retinopathy and its severity in adults with type 2 diabetes: A case-cohort study. Can J Diabetes. 2020;44(5):414-421.

20. Addis P, Shecterle LM, St. Cyr JA. Cellular protection during oxidative stress: a potential role for D-ribose and antioxidants. J Diet Suppl. 2012;9(3):178-182.
21. Tietz NW, Shuey DF. Lipase in serum--the elusive enzyme: an overview. Clin Chem. 1993;39(5):746-756.
22. Jung K. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics. Burtis CA, Ashwood ER, Bruns DE, editors. St. Louis, MO: Elsevier Saunders; 2006. 2448 pp. ISBN: 0-7216-0189-8. Clin Chem. 2006;52(6):1214.
23. Wojnarowska-Nowak R, Polit J, Sheregii EM. Interaction of gold nanoparticles with cholesterol oxidase enzyme in bionanocomplex-determination of the protein structure by Fourier transform infrared spectroscopy. J Nanopart Res. 2020;22(5):107.
24. Van Heerden C, Magwete A, Mabuza D. Evaluating the need for free glycerol blanking for serum triglyceride measurements at Charlotte Maxeke Johannesburg Academic Hospital. Clin Chem Lab Med (CCLM). 2020;58(8):1257-1264.
25. Silva VR, *et al*. HDL-particles separation employing different precipitating agents: functional properties of the impact of chemical precipitation on lipoprotein particle size and paraoxonase-1 activity. Quim Nova. 2019;42:859-865.
26. Noreen S, *et al*. Secoisolariciresinol diglucoside and anethole ameliorate lipid abnormalities, oxidative injury, hypercholesterolemia, heart, and liver conditions. Food Sci Nutr. 2023;11(6):2620-2630.
27. Yang W, *et al*. Linkage and linkage disequilibrium analysis of the lipoprotein lipase gene with lipid profiles in Chinese hypertensive families. Clin Sci. 2005;108(2):137-142.
28. Henderson HE, *et al*. Lipoprotein lipase activity is decreased in a large cohort of patients with coronary artery disease and is associated with changes in lipids and lipoproteins. J Lipid Res. 1999;40(4):735-7543.
29. Al Obaidi ATIT, Rzoqi QA. Effect of high-fat diet on the kidneys of albino rats and the protective role of beetroot juice compared with orlistat. Ann Rom Soc Cell Biol. 2023;27(1):120-137.
30. Tomita Y, *et al*. Relationship between casual serum triglyceride levels and the development of hypertension in Japanese. J Hypertens. 2021;39(4):677-682.
31. Bjelakovic B, *et al*. Lipid profile and left ventricular geometry pattern in obese children. Lipids Health Dis. 2020;19:1-7.
32. Klisić A, Kavarić N, Ninić A. Are liver function biomarkers independently associated with Framingham risk score in women? Srpski arhiv za celokupno lekarstvo. 2020;148(7-8):423-429.
33. Raposeiras-Roubin S, Abu-Assi E, Murphy SA, *et al*. Triglycerides and residual atherosclerotic risk. Journal of the American College of Cardiology. 2021;77(24):3031-3041.
34. Robert J, Osto E, von Eckardstein A. The endothelium is both a target and a barrier of HDL's protective functions. Cells. 2021;10(5):1041.
35. Gianturco SH, Bradley WA. Lipoprotein-mediated cellular mechanisms for atherogenesis in hypertriglyceridemia. In: Seminars in thrombosis and hemostasis. 1988. Copyright© 1988 by Thieme Medical Publishers, Inc.
36. Edwards CR, Bouchier IA, Haslett C, Chilvers ER. Davidson's principles and practice of medicine. Edinburgh: Churchill Livingstone; c1991.
37. Shariq OA, McKenzie TJ. Obesity-related hypertension: a review of pathophysiology, management, and the role of metabolic surgery. Gland Surgery. 2020;9(1):80.
38. Qiao W, Pan Y, Li R, *et al*. Hypertension, BMI, and cardiovascular and cerebrovascular diseases. Open Medicine. 2021;16(1):149-155.
39. Lee C-Y, Chen SC, Lai CC, *et al*. Association of parental overweight and cardiometabolic diseases and pediatric adiposity and lifestyle factors with cardiovascular risk factor clustering in adolescents. Nutrients. 2016;8(9):567.
40. Crawford AG, Cote C, Couto J, *et al*. Prevalence of obesity, type II diabetes mellitus, hyperlipidemia, and hypertension in the United States: findings from the GE Centricity Electronic Medical Record database. Population Health Management. 2010;13(3):151-161.
41. Rahman M, Williams G, Al Mamun A. Hypertension and diabetes prevalence among adults with moderately increased BMI (23.0–24.9 kg/m²): findings from a nationwide survey in Bangladesh. Public Health Nutrition. 2017;20(8):1343-1350.
42. Bernhard A. Serum lipase (Tributyrinase) in hypertension and arteriosclerosis. Proceedings of the Society for Experimental Biology and Medicine. 1951;78(2):533-535.
43. Choudhury KN, Mainuddin AKM, Wahiduzzaman M, Islam SM. Serum lipid profile and its association with hypertension in Bangladesh. Vascular Health and Risk Management. 2014;10:327-332.
44. Bamrara P, Mittal Y, Mathur A. Evaluation of lipid profile of North Indian hypertensive subjects. Asian Journal of Biomedical and Pharmaceutical Sciences. 2013;3(18):38.
45. Zaid HK, Al-Fartosi KG. Assessment of atrial natriuretic peptide (ANP) and lipid profile for hypertension patients in Thi-Qar province, Iraq. University of Thi-Qar Journal of Science. 2021;8(1):48-52.
46. Zhang W, Zhao Y, Zhang F, *et al*. Adenovirus-mediated leptin expression normalizes hypertension associated with diet-induced obesity. Journal of Neuroendocrinology. 2010;22(3):175-180.
47. TV HP, BV S. Assessment of serum lipid profile in hypertensive patients: a case-control study. 2020.
48. Nayak P, Rajput R, Bhatia V, *et al*. Evaluation of lipid profile and apolipoproteins in essential hypertensive patients. Journal of Clinical and Diagnostic Research. 2016;10(10):BC01-BC03.
49. Lawler PR, Akinkuolie AO, Chu AY, *et al*. Atherogenic lipoprotein determinants of cardiovascular disease and residual risk among individuals with low low-density lipoprotein cholesterol. Journal of the American Heart Association. 2017;6(7):e005549.