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Comparison of lipid profile parameters in hypertension patients versus healthy individuals in Babylon province

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Abstract
This research aimed to assess the correlation between parameters lipids in male and female hypertension patients, and compare with control healthy persons, the study was done retrospectively for investigations conducted in governmental hospitals and private laboratories in the city of Babylon. The verbal accord was gained from all study applicants. This study intricate 235 human participants in total, ages ranging from 20 to 80. One hundred male and female volunteers with normotension were chosen as the control group out of the 235 subjects. Group 2 consisted of the remaining individuals, both male and female, who were classified as hypertensive. The results revealed a major optimistic association between LDL and HDL in the male group also optimistic connection was revealed between them in the female group, also results showed a positive correlation between LDL and HDL in hypertension of female groups, and the results revealed a significant increase (p<0.01) of LDL in female compare with the male in both patient and healthy groups, but no significant differences (p>0.01) between male and female in both patient and control in the level of HDL.

Keywords: Low-density lipoprotein, hypertensive, lipoprotein, high-density lipoprotein, cholesterol

Introduction
It has been determined that hypertensive, distinct as a blood burden equal to or larger than 140/90 mmHg [1], is the maximum prevalent circulatory illness [2] and the primary reason for disease and death in both advanced and emergent states. It has also been identified as one of the top ten reported causes of death, with hypertensive complications accounting for approximately 4% of these deaths. When the diastolic plasma pressure is less than 80 mm Hg and the systolic blood pressure is less than 120 mm Hg, blood pressure is considered optimal. Chronic hypertension, or elevated arterial blood pressure, is a widespread health issue with ongoing global increase in the incidence. Because essential hypertension typically goes undiagnosed and asymptomatic, it has been aptly dubbed the silent killer. Uncontrolled hypertension has the potential to harm every organ in the body [3]. The most prevalent risk factors for coronary artery disease (CAD) are dyslipidemia and hypertension [4].
Equally a significant danger influence for cardiac disease, arterial hypertension ranks third in terms of disability and is single of the foremost reasons for death [5]. The co-administration of anti-hypertension and lipid-lowering medicines is frequently suitable due to the fact that increased pressure of blood frequently co-exists with dyslipidemia, and is another risk factor for cardiovascular disease. According to current research, there may not be a significant difference in the lipid profiles that hospital laboratories routinely report between patients with cardiovascular disease and healthy individuals. According to recent research, evaluating the different lipoproteins' subtractions or subpopulations is probably going to be much more important [6].
It is well known that the particles that comprise high-concentration lipoprotein (HDL) and low-density lipoprotein (LDL) are heterogeneous, with the size and density of the particles determining the subfractions.
LDL subdivisions have been divided hooked on seven subfractions (LDL 1-7) [7] based on gel electrophoresis. There are at least 10 subfractions of HDL (HDL 1-10; while the most modern approaches permit the distinction. The different degrees of bioactivity exhibited by LDL and HDL particles are also linked to their heterogeneity.

Large LDL particles, or LDL subfractions 1 and 2, have only been linked to a moderate risk of CVD, while LDL subfractions that are small dense LDL (sdLDL) (LDL 3-7) raise the risk by up to four times [8]. The therapeutically favorable benefits of HDL cholesterol have generally been linked to the subfraction of bigger HDL particles (HDL 2) [9].

However, there are still many conflicting findings about this matter. On the other hand, the HDL 3 subfraction and the majority of other subpopulations of smaller HDL particles may have an undesired atherogenic effect without reducing inflammation [10]. The biological differences that have been reported are probably caused by the fact that small dense HDL particles (sdLDLs; subpopulations 3-7) have an easier time passing through vascular walls, oxidize, and have a decreased affinity for the LDL receptor. However, apolipoproteins (Apo) AI and AII are largely absent from small dense HDL particles, which makes them less efficient in the reverse transport of cholesterol from peripheral tissues to the liver. Additionally, these particles may catabolize more quickly and lose their endothelium-protective properties.

A number of processes could be responsible for the structural and functional alterations in HDL and LDL. As an atherosclerotic CVD frequently coexists with inflammation and oxidative stress, these factors appear to be especially significant [11].

In addition to hypertension, an imbalance favoring one atherogenic subfraction of HDL or LDL may play a role in the development of obesity, metabolic syndrome, insulin resistance, and eventually diabetes mellitus. Evaluation of the lipid profile in normotensive persons would enable early targeted pharmacological intervention in vulnerable patients if dyslipidemia is directly associated with the onset of hypertension [12].

High blood pressure and cholesterol have a close relationship on many levels, as high blood pressure often occurs due to cholesterol, and their high, individually, is a risk factor that increases the chance of heart disease, and the presence of these two factors together at the same time doubles this risk, even if cholesterol and pressure levels are relatively slightly high because the presence of both causes damage to blood vessels and weakens the heart muscle faster [13].

When blood cholesterol levels rise, cholesterol particles begin to accumulate on the walls of blood vessels and arteries, causing them to narrow and obstruct their blood flow [14]. With the passage of time and the accumulation of cholesterol more and more, the arteries narrow more, and the blood vessels become narrow and stiff, which makes it difficult for the blood to pass normally, which puts an effort on the heart by the need to increase its effort to pump blood with greater force to ensure that the necessary oxygen and food reach the rest of the body's organs. And with the effort on the heart, and the continuous rise in cholesterol levels, the pressure on the walls of the blood vessels increases, and high blood pressure ends up [15]. Hence the importance of continuous pressure monitoring for all those who suffer from high cholesterol. Both high blood pressure and cholesterol pose a threat to the arteries and veins, as not controlling each of them leads to damage and destruction of the arteries, and weakens the heart muscle. With the passage of time, it is difficult for the blood vessels to withstand the pressure placed on them, which causes damage to their tissues, and the formation of what is known as weak points on the walls [16]. On the other hand, with the blood flow in these blood vessels that have become weak, cholesterol takes advantage of the weak points and their homes in the vessels to accumulate more and more.

Events are still going on in an endless loop, as high pressure leads to cholesterol deposition on the vessel walls, and cholesterol deposition leads to narrowing and hardening of the arteries, which makes high pressure worse [17]. In turn, the heart tries to cope with the narrowing of the vessels and works harder to pump blood, which puts extra pressure on the heart muscle and eventually weakens it. The complications of high blood pressure and cholesterol are not limited to heart and artery diseases only, as both may cause problems in vision, kidneys, and the brain, so it is important to control the concentration of cholesterol and monitor pressure by committing to taking medications and conducting the necessary periodic examinations [18].

Materials and Methods

Specimens collection

This study was done retrospectively for investigations conducted in governmental hospitals and private laboratories in the city of Babylon and its suburbs. Verbal consent was received from all study participants. This study included a total of 235 human participants, with ages ranging from 20 to 80. One hundred male and female volunteers with normotension were selected as the control group from the 235 subjects. Group 2 consisted of the remaining individuals, both male and female, who were classified as hypertensive. After a 12-hour fast, five milliliters of venous blood were drawn and placed into EDTA bottles between the hours of 8:00 and 10:00 AM. The device used for sample analysis is the Mindray BS-230.

Determination of lipids profile

Measurement of Total Cholesterol (TC) in Serum

The following reactions demonstrate the enzymatic approach used to assess cholesterol concentration:

$$\text{Cholesterol Esters} + H_2O \xrightarrow{\text{Cholesterol Esterase}} \text{Cholesterol} + \text{Fatty Acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{Cholester Oloxidase}} H_2O_2 + \text{Cholest} - 4 - \text{en} - 3 - \text{one}$$

$$H_2O_2 + \text{Phenol} + \text{PAP} \xrightarrow{\text{Peroxidase}} 4\,H_2O + \text{Quinonimine (Pink)}$$

Table 1 shows the reagents used for the measurement of total cholesterol in serum.

Table 1: Reagents of measurement of total cholesterol in serum

The Reagents	The Composition	Concentration
The buffer Reagent	Preservative	5 mmol/L
	Phosphate buffer	100 mmol/L
	Sodium chloride	2.3 mmol/L
	Chloro-4-phenol	5.0 mmol/L
The enzyme Reagent 2	Peroxidase	1200 IU/L
	Cholesterol oxidase	100 IU/L
	Polyethylene glycol 6000	167 µmol/L
	Cholesterol esterase	170 IU/L
	Phenol aminophenazone (PAP)	0.25 mmol/L
The standard Reagent 3	Cholesterol	200 mg/dL or 5.17 mmol/L

To create the working reagent, the contents of vial reagent 2 (Enzymes) were added to vial reagent 1 (Buffer) and swirled gently for two to five minutes, or until the contents were completely dissolved. The procedure for the determination of serum total cholesterol is illustrated in Table 2.

Table 2: Procedure of estimation of serum total cholesterol

The Reagents	The Blank	The Standard	The Sample
The Reagent	1mL	1mL	1mL
Deionized water (DW)	0.01mL	-	-
Std.	-	0.01mL	-
The Sample	-	-	0.01mL

After mixing the tubes, they were allowed to stand at 37 °C for five minutes. The absorbance was then estimated at 500 nm in comparison to a blank. The coloration remained consistent for one hour. The following formula was utilized to determine the TC concentration:

$$\text{Cholesterol (mmol/L)} = \frac{\text{Sample Absorbance}}{\text{Standard Absorbance}} * 5.17$$

Measurement of high and low-density lipoprotein cholesterol in serum

Magnesium chloride and phosphotungstic acid precipitated HDL and LDL. After centrifugation, HDL and LDL were extracted from the supernatant, which was subsequently measured using the total cholesterol reagent. The Reagents of determination of serum HDL-LDL cholesterol as seen in Table 3.

Table 3: Reagents of determination of serum HDL-LDL cholesterol

The Reagents	The Composition	Concentration
The Precipitant Reagent 1	Magnesium chloride pH 6.2	490 mmol/L
	Phosphotungstic acid	13.9 mmol/L
The Standard Reagent 2	Cholesterol	100 mg/dl or 2.58 mmol/L

The steps below are the process for estimating HDL and LDL

1. Plain tubes were filled with 0.5 mL of samples (serum).
2. A volume of 50µL precipitant was added.
3. After thoroughly mixing the tubes, they were let to remain at room temperature for ten minutes.
4. Centrifuge for 15 minutes at 2000 ×g. Measuring the cholesterol content of the supernatant was the next stage.

The procedure for the estimation of serum HDL-LDL cholesterol is illustrated in Table 4.

Table 4: Procedure of determination of serum HDL-LDL cholesterol

The Reagents	The Blank	The Standard	The Sample
Reagent	1 ml	1 ml	1 ml
Distilled water	0.025 ml	-	-
The Standard	-	0.025 mL	-
Supernatant	-	-	0.025 ml

After mixing the tubes, they were kept at 37˚C for five minutes. The absorbance was measured at 500 nm in comparison to the blank. The hue remained constant for one hour. The HDL concentration was determined using the following formula:

$$\text{HDL} - \text{Cholesterol (mmol/L)} = \frac{\text{Sample Absorbance}}{\text{Standard Absorbance}} * 2.58$$

(Standard concentration = 2.58 mmol/L)

Estimation of Triglycerides (T.G.) in Serum

Triglyceride level was estimated by the enzymatic method by the Trinder reaction's fundamental approach, as demonstrated by the following reactions, and the Fossati method:

$$\text{Triglycerides} \xrightarrow{\text{Lipase}} \text{Free fatty acids} + \text{Glycerol}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow[+\ \text{ADP}]{\text{Glycerol Kinase}} \text{Glycerol} - 3 - \text{phosphate}$$

$$\text{Glycerol} - 3 - \text{P} + \text{O}_2 \xrightarrow{\text{Glycerol-3-phosphate oxidase}} \text{Dihydroxacetone} - \text{p} + \text{H}_2\text{O}_2$$

$$4 - \text{Chlorophenol} + \text{H}_2\text{O}_2 + \text{Phenol aminophenazone} \xrightarrow{\text{Peroxidase}} \text{Quinonimine (Pink)} + \text{H}_2\text{O}_2$$

The concentration of TGs in the sample is directly correlated with the absorbance of the colored complex (quinonimine). The usage of the reagents is indicated in Table 5.

Table 5: Reagents for the determination of triglycerides in serum.

The Reagents	Composition	Concentration
The Buffer Reagent 1	Magnesium chloride	9.8 mmol/L
	PIPES	100 mmol/L
	Chloro-4-phenol Preservative	3.5 mmol/L
The Enzyme Reagent 2	Glycerol kinase	660 IU/L
	Lipase	1000 IU/L
	Glycerol-3-p-oxidase	3000 IU/L
	Peroxidase	1700 IU/L
	Phenol aminophenazone adenosine	0.5 mmol/L
	Triphosphate	1.3 mmol/L
Standard Reagent 3	Glycerol equivalent to TGs	200 mg/dl or 2.28 mmol/L

The contents of vial reagent 2 (enzymes) were added to vial reagent 1 (buffer) to create the work reagent. The mixture was then gently swirled until it was completely dissolved, which took around two minutes.

Statistical analysis

With SPSS software, the statistical analysis was carried out. The study's results are reported as the mean±standard deviation (SD). Using the student's t-test, the statistical significance of the difference between the various groups was ascertained; a p-value of less than 0.05 and less than 0.01 is deemed significant.

Results and Discussion

The findings showed a strong positive relationship between HDL and LDL in the male group as shown in Figure 1, and also positive correlation was revealed in the female group as shown in Figure 2**.**

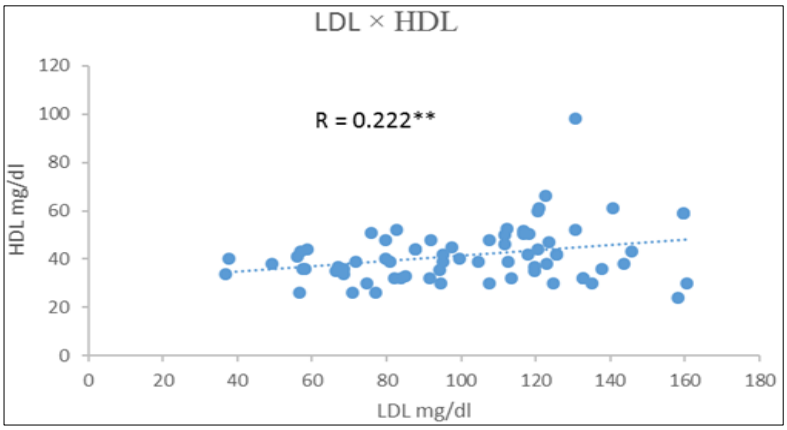

Fig 1: Correlation between LDL and HDL in hypertension male (Patient group)

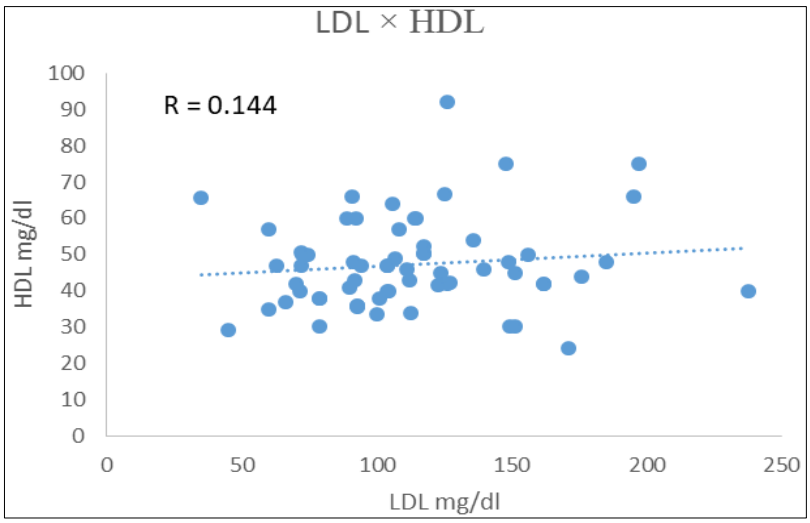

Fig 2: Correlation between LDL and HDL in hypertension females (Patient group)

Our results may indicate a connection between these parameters in both males and females, as several conditions are influenced by both genetic and environmental factors. There are many cardiovascular risk factors, such as metabolic syndrome (MetS), which is primarily associated with patients who have hypertension. The metabolic

syndrome, which includes visceral obesity, dyslipidemia, hyperglycemia, and hypertension, has emerged as one of the world's biggest public health concerns [19]. Also, the result showed a positive correlation between LDL and HDL in hypertension of female groups as shown in Figures 3, and 4.

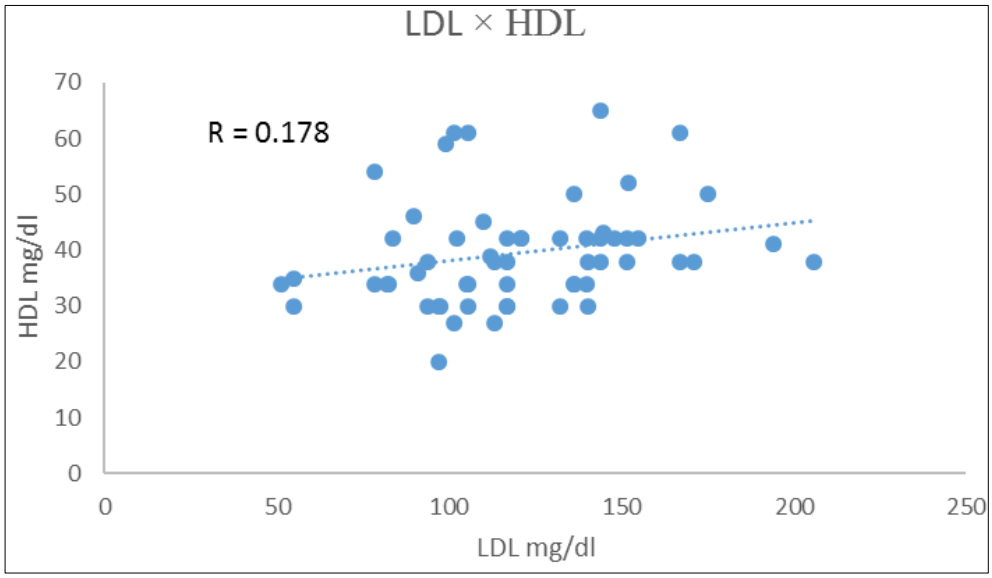

Fig 3: Correlation between LDL and HDL in hypertension of male control group

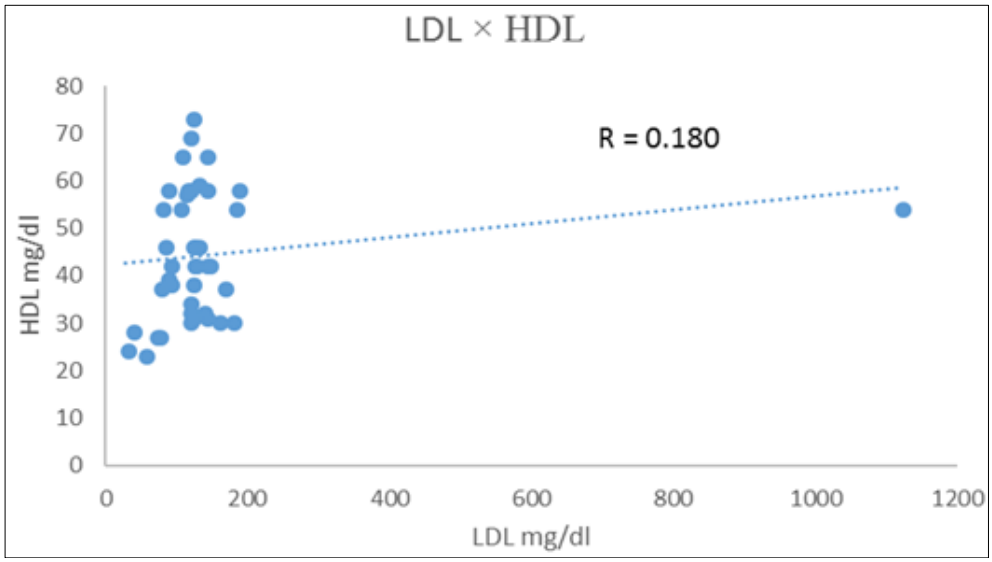

Fig 4: Correlation between LDL and HDL in hypertension of female control group

That may refer to the accumulation of lipid profile in blood vessels and lead to a decreased diameter, which is already associated with hypertension, and increase in women because of variation of blood volume during menstruation, Fluctuations in lipoprotein cholesterol levels during the menstrual cycle are linked to changes in reproductive hormone levels. It is common for TC and LDL-C to peak in the follicular phase and to decrease in the luteal phase. During the late follicular and periovulatory stages, HDL-C is often at its maximum. These results suggest that when assessing lipoprotein cholesterol levels in women of reproductive age, the menstrual cycle phase should be considered [20].

The results revealed a significant increase (p<0.01) of LDL in females compared with males in both patient (hypertensive) and control groups, but no significant differences (p>0.01) between males and females in both patient (hypertensive) and control in the level of HDL, as shown in Tables 7 and 8.

Table 7: Comparison between males and females in low-density lipoprotein in patient group.

Groups Parameters	Male	Female	SIG	P value
LDL	100.5±30.66	112.25±39.74	0.08	0.01*
HDL	41.44±11.4	47.66±12.53	0.21	

* Significant differences between male and female patient groups

Table 8: comparison between male and female in low density lipoprotein in control group

Groups Parameters	Male	female	SIG	P value
LDL	119.21±22.4	137.31±39.53	0.04	0.01*
HDL	39.66±4.72	44.166±13.64	0.37	

*Significant differences between male and female of control groups

Our results may refer to negative effect of LDL in healthy of female hypertensive because changed volume blood during

menstrual cycle and association of this parameter with decrease and increase blood vessels diameters, clinical study showed high levels of LDL cholesterol raise the risk for heart disease and stroke, Studies on the connection and underlying mechanism between dyslipidemia and hypertension are proliferating. In addition to sharing internal contributing variables and metabolic abnormalities, dyslipidemia and hypertension also affect one another through a variety of methods. First, dyslipidemia raises blood pressure and destroys the vascular endothelium [21]. The most likely connection between hypertension and hyperlipidemia is vascular endothelial function. Vascular endothelial function has a major role in controlling arterial contractility. Abnormal lipids may indirectly impact arterial elasticity by disrupting vascular endothelial control, ultimately leading to hypertension [22]. Second, through altering receptor function and cell membrane structure, dyslipidemia raises blood pressure. Research has demonstrated that patients with hypertension have a variety of aberrant ion transport patterns in their cell membranes, leading experts to believe that hypertension is a cell membrane disease [23].

Conclusion

1. There is a highly significant correlation between two types of lipids in healthy individuals, making it essential to include these tests in every comprehensive health survey.
2. All types of lipids in lipid profile parameters are significantly associated with cardiovascular disease.
3. The levels of lipid profile parameters in males are generally lower than those in females.

Recommendations

1. Examine the problem at the tissue level more broadly.
2. Expanding the side of the studies in terms of genetics and linking susceptibility to blood pressure in terms of genetics.
3. Studying the effect of some sports practices on preventing blood pressure and its impact on lipid profile.
4. Study of participants between female and male patients during specific age groups.

Ethical approval

Before any medical procedures can begin, ethical committees, patients, and their supporters must all provide their approval. Furthermore, before sample collection, all participants were informed about the research and gave their consent for both the study and the publication of the results. The human study received approval from the Review Board of Al-Qasim Green University in Babylon Province, Iraq.

Conflict of Interest

The authors disclose no conflicts of interest.

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