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Silent Killer: Dyslipidaemia as a Predictor of Hypertension

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Abstract: Background: Hypertension is not a temporary symptom, but a "silent killer" and one of the major risk factors for cardiovascular disease and atherosclerosis. Obesity and dyslipidaemia have also been largely responsible for structural damage to the artery wall and increased vascular resistance, which forces the heart muscle to work harder and damages other organs. **Aim:** This descriptive cross-sectional study aimed to study the effect of biochemical variables, biological factors, and lifestyle factors on patients with hypertension. **Method:** This study was conducted between December 2025 and February 2026. 112 samples were collected, and participants were divided into 2 groups. The age group of the study group was 40 – 50 years, which is a critical age group. The method was based on data collection through tailor-made questionnaires. Biochemical variables were estimated. All data were statistically analyzed using SPSS and Microsoft Excel. **Results:** Statistically significant differences ($p < 0.001$) between the two groups were observed. In the patient group, there was a sharp rise in triglyceride levels (mean 223.7 mg/dL) and total cholesterol levels (mean 207.8 mg/dL) compared to the control group. The study also indicated that obesity and overweight were the major characteristics in the patients (74%). The patients had low levels of HDL, especially the pre-menopausal women, and were therefore more prone to cardiovascular risks. Mean values of blood glucose were higher in patients with statistically significant differences compared to healthy individuals ($p > 0.05$). This means that lipid metabolic disorders and obesity are the most influential factors in hypertension in this particular sample. **Conclusion:** The fight against high blood pressure must go beyond drugs and adopt a holistic approach, said the study. The focus should be on early detection of lipid disorders and on lifestyle and dietary modifications to lower body mass index. **Future directions:** The association of body fat with hypertension should be monitored in long-term studies in individuals with obesity, metabolic syndrome, and insulin resistance in families and specific regions to find out the effect of genetics or lifestyle.

Keywords: Hypertension, dyslipidaemia, cholesterol, triglycerides, LDL, HDL.

INTRODUCTION

High Blood Pressure (Hypertension) is a condition in which the force of blood on the walls of the arteries is too high. Over time, this pressure can damage your blood vessels and cause health problems. If the systolic blood pressure is higher than 140 mm of mercury, it puts pressure on the cardiovascular system and raises the risk of heart damage, even if there are no symptoms (Davidson, L. J., *et al*., 2024). There are two types of high blood pressure: The first type is primary hypertension, which has no identifiable cause, and affects more than 95% of patients. The second type is secondary hypertension that has a known cause such as kidney disease or endocrine disorders, etc. It only represents less than 5% of patients (Anderson, N. M., 2005; Zhang, Y. *et al*, 2020). Hypertension is a major public health challenge in Iraq affecting more than 40% of Iraqi adults. Hypertension is known as the "silent killer" because it usually has no obvious symptoms and if you ignore it, it can do great damage to the organs of your body. Early detection of hypertension is clinically important because of the potential to reduce the risk of cardiovascular disease and neurological disorders, and to improve heart and eye health and vision. The diagnosis is confirmed by two or more readings during two or more visits following the initial examination (Al-Khuzaiy, A. *et al*., 2013). After diagnosis, adult blood pressure classification is based on the Seventh Report of the Joint National Committee (JNC 7) guidelines of 2003 (Chobanian, A. V. *et al*., 2003). For those aged 40 to 70 years, each 20 mmHg increase in systolic blood pressure or 10 mmHg increase in diastolic blood pressure doubles the risk of cardiovascular disease (Al-Khuzaiy, A. *et al*., 2013).

Fats are a group of heterogeneous organic compounds insoluble in water but soluble in organic solvents. They play important roles including energy storage as they are the most efficient and effective energy source for the body. They are also structural components of cell membranes and act as basic units in the biosynthesis of steroid hormones and fat-soluble vitamins (Vasudevan, D. M. *et al*., 2019). Fats can be divided into saturated fats, trans fats, and unsaturated fats (Hamad Medical Corporation, 2017).

The Fats Used In This Study

Cholesterol and its types: Cholesterol is a vital sterol molecule characterised by its bivalency and rigid steroid nucleus. It is vital for the control of fluidity and stability of animal cell membranes. It acts as an important precursor for the synthesis of

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steroid hormones, bile acids, and vitamin D. Its biosynthesis takes place in the liver, starting from acetyl-CoA, controlled by HMG-CoA reductase, which is the rate-limiting step.

Due to its hydrophobic nature, cholesterol is carried in the bloodstream by lipoproteins (LDL and HDL) to maintain a balance between the liver and surrounding tissues (Nelson, D. L., & Cox, M. M., 2013). Living organisms store energy primarily in the form of triglycerides . These molecules are composed of three fatty acids attached to a glycerol molecule by ester bonds . They are completely hydrophobic and nonpolar . They can be stored in high concentrations inside cells without interacting with water molecules . Triglycerides are a far more efficient source of energy than carbohydrates and oxidation of fatty acids yields a great deal of ATP. In mammals these fats are stored primarily as oil droplets in adipocytes (fat cells). The subcutaneous fat layers not only provide a store of fuel, but also provide thermal insulation and protect animals in cold environments, highlighting their vital importance not only for metabolism, but also for environmental adaptation (Nelson, D. L., & Cox, M. M., 2013). Plasma triglycerides come from the diet and from synthesis in the liver; In the intestine or liver, these fats are packed into chylomicrons and used for the synthesis of triglyceride-rich VLDL molecules (Harchaoui, K. E. L. *et al*., 2009).

Lipids and High Blood Pressure

Lipids are a major risk factor for high blood pressure, and therefore heart disease. High blood pressure and heart disease tend to occur together due to common pathophysiological mechanisms like obesity, insulin resistance and vascular dysfunction (Chen, S., & Cheng, W., 2022). Lipids are important in the variability of blood pressure . Dyslipidemias and high blood pressure are closely and complexly related . The explanation of this relation is given in the following mechanisms: Atherosclerosis - high cholesterol leads to deposition of fatty plaques in the walls of the arteries narrowing the vessels and reducing their elasticity. This causes the heart to pump blood more powerfully . This causes high blood pressure . Endothelial dysfunction High levels of cholesterol reduce the production of nitric oxide, a substance that helps to relax and widen blood vessels. This leads to vasoconstriction and an increase in blood pressure. Blood viscosity: high triglyceride levels increase the viscosity of the blood, which forces the heart to work harder to pump blood through the capillaries, thus contributing to high blood pressure. Insulin resistance: The body's cells do not respond to insulin. This leads to the sugar not being able to get into the cells and the level of sugar in the blood increases. This forces the pancreas to secrete large and increased amounts of insulin, subsequently leading to weight gain, constant hunger, fatigue, and increased appetite (Hall JE. & Hall ME., 2020; Jeppesen, J. *et al*., 2000). Free fatty acids in the blood stream increase and disrupt the insulin signaling, leading to hyperinsulinemia (Kasper D *et al*., 2015).

As a result, the activity of the sympathetic nervous system increases, especially the sympathetic nerves located in the kidneys, which reduces their ability to secrete salt and water, thereby increasing blood pressure (Hall JE. & Hall ME., 2020).

The Relationship between Fats and Blood Pressure

Hyperlipidemia is a widespread disorder in the world, affecting both men and women (Chen, S., & Cheng, W., 2022; Yeasmin, N.*et al*., 2019), and it leads to high blood pressure (Sari, N., 2023). Research has shown that high total cholesterol levels cause atherosclerosis, which reduces the ability of blood vessels to expand, thus increasing resistance and raising blood pressure (Safar, M. E. *et al*., 2018). Research has also shown that patients with high cholesterol have greater difficulty controlling their blood pressure readings than those with normal levels (Borghi, C. *et al*., 2016). Triglycerides are an energy reserve and are involved in the development of atherosclerosis and heart disease, and high blood pressure (Chen, S., & Cheng, W, 2022). All studies have shown that high triglyceride levels lead to high blood pressure (Chen, S., & Cheng, W, 2022; Kasper, D. *et al*., 2015; Yeasmin, N.*et al*., 2019). The pivotal role of triglycerides lies in their association with increased blood viscosity and sympathetic nervous system activation, which lead to an increase in systolic blood pressure (Harchaoui, K. E. L. *et al*., 2009; Yeasmin, N. *et al*., 2019). In urban societies this association is further reinforced by unhealthy lifestyle (Ekanem, U. S. *et al*., 2013).

The Relationship of High Blood Pressure to Other Variables in the Study

Smoking: it leads to a sudden increase in blood pressure, as a result of the stimulation of the sympathetic nervous system by nicotine and the release of catecholamines that cause vasoconstriction and an increase in heart rate

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(Virdis, A. *et al*., 2010; Groppelli, A. *et al*., 1992). It has been shown in studies that the effects of smoking are not only limited to temporary hypertension but also include damage to the inner lining of arteries and increased arterial stiffness leading to chronic atherosclerosis (Virdis, A. *et al*., 2010; Leone, A., 2011). It was found that smoking causes the condition "masked hypertension", when the blood pressure is normal in a visit to the doctor, but increases significantly in daily activities (Mann, S. J. *et al*., 1991; Omvik, P., 1996). **Age:** The association between age and high blood pressure is direct and strong as the arteries become stiffer (Kandpal, V. *et al*., 2016). This results in a gradual increase of systolic blood pressure due to structural changes in the walls of blood vessels (Smith, A., 2021). Hormonal changes are also a major factor in high blood pressure among the elderly (Brown, C., 2023). Research shows that the likelihood of acquiring high blood pressure is more than 60% in people over 60 years of age (WHO, 2024).

The reason the 40-50 age group was chosen for this study is that it is a crucial period when high blood pressure starts to enlarge the heart muscle and thicken the walls of the arteries (Williams, B., 2023). At this age, not treating hastens the coming of many diseases that could lead to stroke and kidney failure in the future (Miller, J., 2021). However, in this age group, the cortisol level increases as a result of stress at work, which further aggravates insulin resistance and increases blood pressure (Stress & Health Lab, 2023). **Sex and Blood Pressure:** Biological and hormonal factors are important in the differences between the two sexes in blood pressure. Before menopause, the effect of oestrogen leads to lower blood pressure in women than men of the same age (Reckelhoff JF., 2021). With age, women's blood pressure rises faster than men's, and after menopause, they are more likely to develop atherosclerosis (Ji, H. *et al*., 2020). Testosterone is an important and effective factor in increasing blood pressure levels in men, acting through different mechanisms (Gillis, E. E., & Sullivan, J. C., 2016). **Glucose and high blood pressure:** Glucose is the main source of energy, but chronic high levels cause metabolic imbalances and promote oxidative stress, leading to various types of damage, including to blood vessels and nerves (Wang, G. *et al*., 2024). Several physiological mechanisms mediate the effect of blood glucose on blood pressure, especially during fasting. High blood glucose increases the osmolality of the blood, drawing fluid from the walls of blood vessels, which increases blood volume and pressure (Brown, A., and Smith, J., 2023). This leads to the formation of advanced glycation end products (AGEs), which play a role in atherosclerosis and hardening of arteries (Meng, H. *et al*., 2024). Higher blood glucose also increases insulin production. This will stimulate the sympathetic nervous system. This increases sodium reabsorption in the kidneys. This causes increased blood pressure (Miller, G., 2024). This condition also damages the blood vessel endothelium by reducing nitric oxide, which plays a role in vasodilation (Khan, M., 2021). Studies prove that stable fasting blood glucose lowers the risk of atherosclerosis, heart disease, and high blood pressure in the future (Roberts, S., and Thompson, D., 2025).

MATERIAL AND METHODS

Design of the study: A descriptive cross-sectional study design was used to examine the variables and their relationships. This design was used to achieve the objectives of the current study, which investigated the relationships between triglycerides, cholesterol, obesity, and certain biochemical variables and hypertension during the period from December 2025 to February 2026, in Baquba City, Diyala Province, Iraq.

Data Collection: The researcher used the questionnaire described in the appendix to collect data and information from the study participants. The study included 112 cases (57 patients and 55 healthy individuals) aged 40 to 50 years. Of the 55 healthy individuals, 40 were female, and 15 were male; among the patients, 43 were female, and 14 were male.

Materials: Laboratory materials from Mindray Bio-Medical Electronics Co., Ltd., simply known as Mindray, were used to perform biochemical tests (blood sugar, triglycerides, total cholesterol, LDL-cholesterol, and HDL-cholesterol) and calculate the LDL from equation (Kannan, S. *et al*., 2014):

LDL = Total Cholesterol - HDL - (Triglycerides / 5)

Blood pressure was measured using a GD-Rossmax pneumatic device. The scale was used to measure weight and the measuring tape to measure height, to calculate BMI, and to divide the study subjects into four groups (Nuttall, F. Q., 2015) .

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Statistical Data Analysis: Data were examined for missing values or duplicate records, and none were found. All continuous variables were screened for implausible / outlier values by checking against clinically plausible physiological ranges for adults aged 40-50 years: no value was implausible or suggestive of data-entry error, and no outliers were detected. Moreover, Body Mass Index (BMI = weight (kg) / height (m^2)) and World Health Organization (WHO) (WHO, 2024; Nuttall, F. Q., 2015) BMI category (Normal (18.5-24.9), Overweight (25-29.9), Obese (≥ 30); no subject was underweight (< 18.5)) were calculated for each subject (WHO, 2024; Nuttall, F. Q., 2015). The Shapiro-Wilk test was used to test normality for every continuous variable, separately within each group. A variable was considered normally distributed only if both groups passed ($p > 0.05$); if either group failed, the variable was considered non-normally distributed. Continuous variables are presented as mean ± standard deviation (SD) when normally distributed and as median and interquartile range (IQR) when not. Categorical variables are presented as numbers (n) and percentages.

For between-group comparisons, normally distributed continuous variables were first tested for homogeneity of variance using Levine's test. Student's t-test was used when Levine's $p > 0.05$, and Welch's t-test was used when Levine's $p \leq 0.05$, and the effect size was calculated using Cohen's d (pooled SD). For non-normally distributed continuous variables, the Mann-Whitney U test was used, with rank-biserial correlation r being used for effect size calculation. Pearson's chi-square test of independence was utilised for categorical variables, with Fisher's exact test substituted for 2×2 tables whenever more than 20% of expected cell counts were < 5 or any expected count was < 1.

For effect size calculation, Cramer's V (and the odds ratio for 2×2 tables) were used. A two-tailed significance threshold of 0.05 was used throughout. The relationship between the directly assayed LDL-C and Friedewald-calculated LDL value (LDL = Total Cholesterol − HDL − Triglycerides/5) was quantified using Pearson's r and Spearman's ρ (for association), Lin's concordance correlation coefficient (for agreement), and a Bland-Altman analysis ''for mean bias and 95% limits of agreement'' (Giavarina, D., 2015). The normality of the paired differences was evaluated (Shapiro-Wilk) to select an appropriate paired test (paired t-test versus Wilcoxon signed-rank test) for a systematic (non-random) difference between methods. A multivariable binary logistic regression was used to identify biochemical and clinical variables independently associated with hypertension status after adjustment, with the participant group (hypertensive = 1, control = 0) being the outcome.

Possible predictors (age, BMI, sex, smoking, family history, triglycerides, total cholesterol, direct LDL-C, HDL, and fasting glucose) were first evaluated using separate univariate logistic regression models, and variables with $p < 0.20$ were used in the final model. Calculated LDL was excluded beforehand from the possible predictors because it is an exact linear function of total cholesterol, HDL, and triglycerides, and including it would produce multicollinearity. Moreover, multicollinearity among the selected predictors was evaluated using the variance inflation factor (VIF; VIF > 5 flagged for concern). Additionally, model fit was assessed using the likelihood-ratio test against the null (intercept-only) model, McFadden's pseudo-R^2, and the Hosmer–Lemeshow goodness-of-fit test ($p > 0.05$ indicates no evidence of lack of fit). Discrimination was also quantified with the area under the receiver-operating-characteristic curve (AUC), and coefficients were reported as adjusted odds ratios (AOR) with 95% Wald confidence intervals (CI). All analyses were performed using SPSS (Statistical Package for the Social Sciences) version 26 (IBM Corp., Armonk, NY, USA).

RESULTS

Distribution of Study Cases: The classification of the study population by age, sex, family history, smoking status, and body mass index (BMI) is shown in Table 1. Subjects in the age range of 40 to 50 years were included in the study sample. Table 1 presents the percentage frequencies of the study variables: age, gender, family history, smoking, and body mass index. Table 2 shows demographic, anthropometric, and risk-factor characteristics by group.

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Table 1: Frequency and percentage for study subjects.

Parameters		**Patient Group n= 57**		**Control Group n= 55**	
		F*	**%***	**F***	**%***
Age (years)	**40 - 43**	19	33	20	36
	44 - 47	22	39	17	31
	48 - 50	16	28	18	33
Sex	**Female**	43	75	40	73
	Male	14	25	15	27
Smoking	**Yes**	18	32	12	22
	No	39	68	43	78
Family History	**Yes**	31	54	18	33
	No	26	46	37	67
BMI	**≤ 18.4**	0	0	0	0
	18.5 – 24.9	11	19	9	16
	25.0 – 29.9	42	74	40	73
	≥ 30	4	7	6	11
**F: Frequency, %: percentage.*					

Table 2: Demographic, anthropometric, and risk-factor characteristics by group.

Characteristic	**Hypertensive (n = 57)**	**Control (n = 55)**	**Test statistic**	**p-value**
Age (years), mean ± SD	45.1 ± 3.2	45.2 ± 3.4	U = 1554	0.939
BMI (kg/m²), mean ± SD	26.8 ± 2.5	26.9 ± 2.4	t = -0.142	0.887
Weight (kg), mean ± SD	77.8 ± 9.0	77.4 ± 9.0	t = 0.261	0.795
Height (cm), median (IQR)	170 (166.0-175.0)	167 (164.5-175.5)	U = 1722.5	0.368
Sex (female), n (%)	43 (75.4%)	40 (72.7%)	χ^2 = 0.012	0.911
Smoker (yes), n (%)	18 (31.6%)	12 (21.8%)	χ^2 = 0.908	0.341
Family history of hypertension (positive), n (%)	31 (54.4%)	18 (32.7%)	χ^2 = 4.492	0.034*
BMI category (Overweight), n (%)	42 (73.7%)	40 (72.7%)	χ^2 = 0.613	0.736
BMI category (Obese), n (%)	4 (7%)	6 (10.9%)		

**Significant difference at 0.05 level.*

Abbreviations: U, Mann-Whitney U test; t, Student's t-test; χ^2, Pearson's chi-square test.

Table 3 and Figure 1 compare and distribute the results obtained from the fasting biochemical examination between hypertensive patients and healthy individuals. The directly measured LDL-C value was also determined, rather than the calculated value, as a predictor of LDL in the study sample, as shown in Figure 2.

Table 3: Comparison of the fasting biochemical panel between hypertensive patients and controls.

Variable	**Hypertensive (n = 57)**	**Control (n = 55)**	**Statistic (p-value)**	**Effect size**
Triglycerides (mg/dL)	223.7 ± 105.9	131.8 ± 52.3	t' = 5.857 (< 0.001)*	d = 1.095
Total cholesterol (mg/dL)	207.8 ± 52.0	166.2 ± 33.0	t' = 5.073 (< 0.001)*	d = 0.952
LDL-C, direct assay (mg/dL)	131.8 (106.7-157.6)	94.5 (88.0-103.7)	U = 2493.5 (< 0.001)*	r = -0.591
LDL-C, Friedewald-calculated (mg/dL)	118.9 ± 35.1	90.3 ± 24.8	t' = 4.987 (< 0.001)*	d = 0.937
HDL-C (mg/dL)	39.7 (34.4-43.1)	43.7 (40.7-47.7)	U = 954 (< 0.001)*	r = 0.391
Fasting glucose (mg/dL)	109.5 (99.4-136.2)	98.3 (93.4-114.2)	U = 2035 (0.007)*	r = -0.298

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**Significant difference at the 0.05 level.*
Abbreviations: t', Welch's t-test; U, Mann-Whitney U test; d, Cohen's d; r, rank-biserial r.

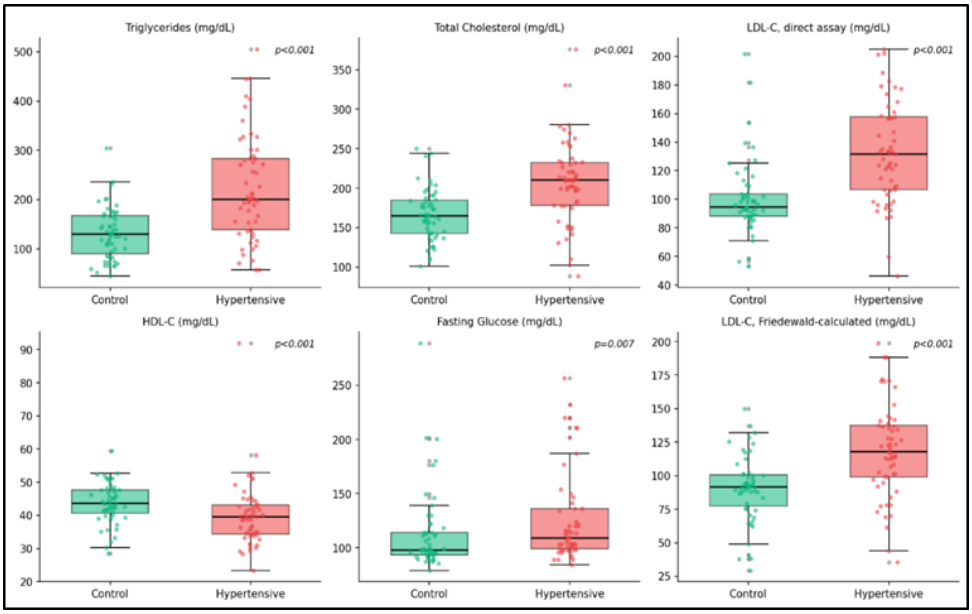

Figure 1: Distribution of triglycerides, total cholesterol, direct LDL-C, HDL-C, fasting glucose, and calculated LDL-C by group. Boxes show the median and IQR; whiskers extend to 1.5×IQR; points are individual subjects. All six comparisons were statistically significant ($p \leq 0.007$; see Table 3).

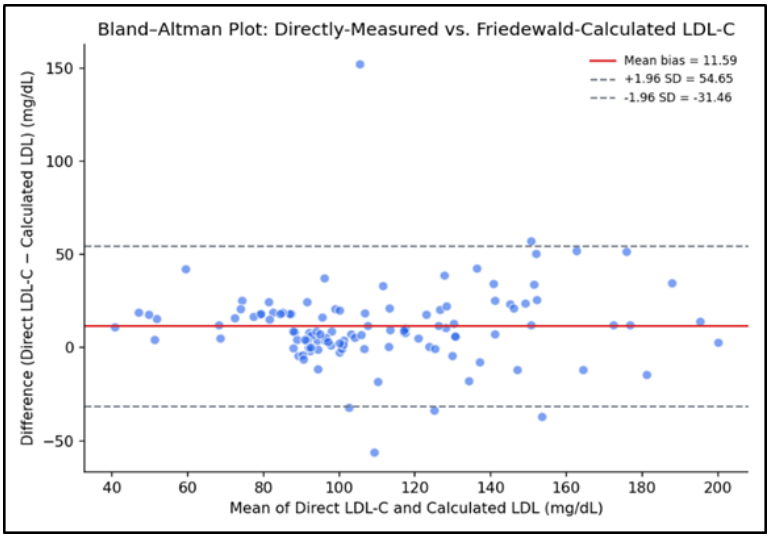

Figure 2: Bland-Altman plot comparing directly-measured LDL-C against Friedewald-calculated LDL across all 112 subjects. The solid red line is the mean bias; dashed grey lines are the 95% limits of agreement.

Univariable assessments were performed for family history, triglycerides, total cholesterol, direct low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and fasting blood glucose (Table 4 and Figure 3). They were used in the final multivariate model. Age, body mass index (BMI), sex, and smoking did not show statistical significance in this model and were therefore not included.

Table 4: Multivariable logistic regression with adjusted odds ratios for hypertension status.

Predictor	AOR	95% CI	Statistic	p-value
Family history, positive (ref: negative)	2.422	0.957-6.131	z = 1.87	0.062
Triglycerides (per 1 mg/dL)	1.014	1.004-1.023	z = 2.76	0.006*
Total cholesterol (per 1 mg/dL)	1.003	0.984-1.022	z = 0.29	0.769
LDL-C, direct (per 1 mg/dL)	1.020	0.995-1.047	z = 1.56	0.118
HDL-C (per 1 mg/dL)	1.043	0.978-1.112	z = 1.29	0.197

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Fasting glucose (per 1 mg/dL)	1.010	0.997-1.023	z = 1.51	0.131

**Significant association at 0.05 level.*

AOR = adjusted odds ratio per unit increase (or for the presence vs. absence of family history), holding the other five predictors constant. Model likelihood-ratio χ^2 (6) = 45.15, p <0.001; McFadden pseudo-R^2 = 0.291. Hosmer-Lemeshow goodness-of-fit χ^2 (8) = 6.71, p = 0.568 (non-significant, indicating no evidence of lack of fit). AUC = 0.835 (95% bootstrap CI 0.753-0.906); classification accuracy = 80.4%, sensitivity = 75.4%, specificity = 85.5% at a 0.5 probability threshold.

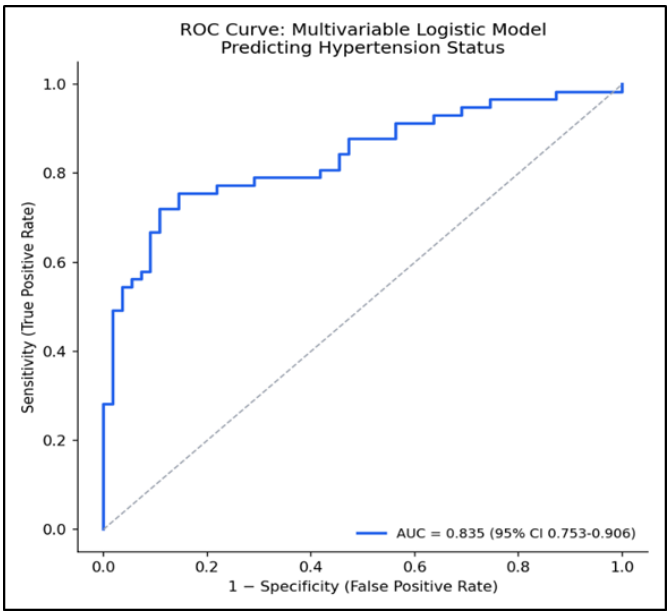

Figure 3: Receiver-operating-characteristic (ROC) curve for the multivariable logistic regression model predicting hypertension status from family history and the biochemical panel.

DISCUSSION

The results obtained in the Results section (Table 1) indicate that the 40-47 age group had the highest percentage of hypertension cases (Chen, S., & Cheng, W., 2022), and the majority were women. According to studies, women in this age group are more prone to hypertension because of hormonal changes due to perimenopause and menopause (Zanchetti, A. *et al*., 2005). Lifestyle, family history and smoking were found to be important contributory factors of hypertension (Al-Khuzaiy, A. *et al*., 2013). Data showed that 54% of the patients had a family history of the disease, confirming the previous studies that heredity significantly increases the risk due to shared genetics, dietary habits, and environmental factors (Ranasinghe, P. *et al*., 2015). Overweight (BMI) in 74% of patients. There is a strong, direct correlation between excess weight and blood pressure because increased fat tissue increases vascular resistance, narrows the vessels, increases the workload of the heart and decreases blood flow to other organs, eventually causing hypertension (Hall, J. E. *et al*., 2015). 32% of the patients were smokers and 68% of the patients were non-smokers during smoking. However, nicotine contributes to high blood pressure as it causes immediate vasoconstriction and increases the rate of the heart leading to acute and chronic hypertension (Nuttall, F. Q., 2015).

Table 2 presents very similar average weights for both groups and the body mass index (BMI) is almost identical. Both groups are above age. The gender distribution of the two groups shows that the majority is females in both groups. All variables (age, weight, height, gender, smoking, and BMI) gave a p-value of more than 0.05, indicating no statistically significant difference between the two groups. This confirms the efficacy and reliability of the matching process. Family history was the only variable that showed statistical significance (p = 0.034) in association with the disease. For most of the control group, total cholesterol levels were within the ideal range. However, the largest group among the patient group was outside the ideal range and thus in the risk category. This implies that patients with hypertension also have high total cholesterol levels (Borghi, C. *et al*., 2016). As shown in Table 3 and Figure 1, the p-values of the comparison between the patient and control groups are statistically significant because they are much less than 0.001.

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This proves that high total cholesterol in hypertensive patients is not by coincidence but a feature of the disease state of study subjects . These results are contradictory to another study (Sari, N., 2023) and are consistent with a second study (Borghi, C. *et al*., 2016).

Direct LDL-C assay and the Friedewald-calculated LDL value were strongly correlated (Pearson r = 0.794, $p < 0.001$; Spearman $\rho = 0.816$, $p < 0.001$) but showed only moderate agreement (Lin's concordance correlation coefficient = 0.749), indicating that correlation alone overstates interchangeability between the two methods. Another remarkable finding was the significantly lower levels of HDL cholesterol observed in patients, especially among premenopausal women, because the HDL cholesterol values differ according to gender (Hamad Medical Corporation, 2017), increasing their sensitivity to cardiovascular risks. This indicates that high blood pressure in women is closely related to deteriorating good cholesterol levels in the lower categories, which increases the likelihood of developing cardiovascular diseases (Liu, R., & Cheng, W., 2024).

The Bland-Altman analysis (Figure 2) showed a systematic positive bias: directly assayed LDL-C averaged 11.59 mg/dL higher than the calculated value (95% limits of agreement: -31.46 to 54.65 mg/dL). Because the paired differences were non-normally distributed (Shapiro-Wilk $p < 0.001$), this systematic difference was confirmed with the Wilcoxon signed-rank test rather than a paired t-test (statistic = 972, $p < 0.001$). Therefore, the two LDL measures should not be used interchangeably, and analyses or clinical decisions based on the calculated value may understate LDL-C burden relative to direct assay. Hence, the directly assayed LDL-C, rather than the calculated value, was used as the LDL predictor in the multivariable model. In univariable evaluation, family history, triglycerides, total cholesterol, direct LDL-C, HDL, and fasting glucose reached the $p < 0.20$ threshold and were used in the final multivariable model; age, BMI, sex, and smoking did not meet the threshold and were not included. Variance inflation factors for the six selected predictors ranged from 1.05 to 2.82, well below the threshold of concern (VIF = 5), indicating multicollinearity was not an issue for this model. After mutual adjustment, as shown in Table 4, triglycerides remained an independent, statistically significant correlate of hypertension status (AOR = 1.014 per mg/dL, 95% CI 1.004-1.023, $p = 0.006$), equivalent to around a 15% increase in the odds of hypertension per 10 mg/dL increase in triglycerides. Family history showed a positive association that approached but did not reach statistical significance after adjustment (AOR = 2.422, 95% CI 0.957-6.131, $p = 0.062$), while the rest of the predictors did not achieve statistical significance. The overall model discriminated hypertensive from control subjects well (AUC = 0.84, Figure 3) and showed good calibration (Hosmer-Lemeshow $p = 0.57$).

Limitations

The current study has several limitations. The cross-sectional case-control design renders causal or temporal inferences unattainable from the current data. The events-per-variable (EPV) ratio for the multivariable model (9.2) was marginally below the conventional minimum (10), so the model coefficients, while informative, should be treated as hypothesis-generating and confirmed in a larger sample. Both groups were restricted to a narrow age range (40-50 years); findings may not generalise to younger or older populations. Information regarding medication use (e.g., antihypertensive or lipid-lowering therapy), diet, and physical activity, all of which can markedly affect the biochemical panel, was not collected and could not be adjusted for. The Friedewald equation used to derive calculated LDL is known to be less accurate at triglyceride concentrations above ~400 mg/dL, several of which were observed in this study; this likely contributes to the disagreement documented in the Results section.

CONCLUSION

In Iraq, particularly in the Diyala Governorate, this study makes a scientific contribution to our understanding of the intricate link between adult hypertension and biochemical and physiological factors. The core idea of the study is that hypertension is a "silent killer" and a significant factor in atherosclerosis and cardiovascular disease instead of being a temporary symptom. It has been confirmed that obesity and dyslipidaemia are major factors in structural damage to arterial walls and an increase in vascular resistance, which ultimately puts pressure on the cardiac muscle and results in serious consequences like stroke and kidney failure.

The aim of this cross-sectional descriptive study was to analyse the effects of biological features such as age, gender, and obesity, and biochemical variables such as triglycerides and cholesterol in patients with hypertension. The study was

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conducted between December 2025 and February 2026. The sample consisted of 112 participants divided into two groups: a control group of healthy individuals (55 cases) and a patient group (57 cases). Special attention was paid to the critical age group of 40 to 50 years. Data were obtained by specially designed questionnaires and body mass index (BMI) was calculated from accurate height and weight measurements. Laboratory tests for total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, computed and measured low-density lipoprotein (LDL) cholesterol, and fasting blood glucose (FBG) using Mindray reagents were also performed. All data were analysed in detail using Microsoft Excel and SPSS v.26 to ensure the accuracy of the findings.

The results showed significant differences ($p < 0.001$) between the patient and control groups, with the patient group having significantly higher triglyceride (mean 223.7 mg/dL) and total cholesterol (mean 207.8 mg/dL) levels than the control group. The study also found that 74% of the patients were obese or overweight, supporting the hypothesis that the accumulation of fat tissue causes the production of hormones that intensify insulin resistance and atherosclerosis, eventually causing hypertension. Another remarkable finding was the significantly lower levels of HDL cholesterol observed in patients, especially among premenopausal women, because HDL cholesterol values differ according to gender, increasing their sensitivity to cardiovascular risks. The mean fasting blood sugar levels of the patients were also found to be high compared to healthy individuals with statistical significance ($p < 0.05$), indicating that obesity and abnormalities in lipid metabolism are the main factors contributing to hypertension in this population. Age and smoking status analyses also showed that these variables worsen the condition and make it more difficult to use antihypertensive medications to control and regulate blood pressure levels. The study also showed that the management of hypertension requires a comprehensive approach, not only medication therapy. Early detection of lipid abnormalities and lifestyle and diet modifications to decrease body mass index are important.

In conclusion, the study recommended that patients with high blood pressure should have a healthy diet and participate in regular physical activity along with their medications to improve community health and reduce the huge pressure on healthcare facilities. It also recommended to minimize stress and its provoking factors.

RECOMMENDATIONS

First: Recommendations for Improving Public Health and Prevention: Regular comprehensive check-ups, changes in behaviour and nutrition, regular and systematic physical activity, and public awareness: launching educational campaigns explaining the relationship between obesity, metabolic syndrome, and hypertension. **Second:** Recommendations for Researchers and Future Studies: Longitudinal Studies: Conduct longitudinal studies to follow up on the effects of low triglyceride levels on blood pressure values in obese individuals or patients with metabolic syndrome and insulin resistance. Genetic factors: Study the interaction of genetic and environmental factors in the blood pressure response to high cholesterol in different ethnic groups and study the intergenerational transmission of these genes and the risks they carry. Molecular effects: Extend the study of the effect of visceral fat tissue on the hormonal system (renin-angiotensin system) that regulates blood pressure. Investigate if there is a relationship between technological advancement and obesity and hypertension.

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