

Prevalence of Dyslipidemia in Patients Diagnosed with Ischemic Heart Disease in Najaf

Amjed Mohammed Sahib

^{1,2,3} Directorate of Education in A-Najaf, Ministry of Education, Iraq.

*Corresponding Author Email: amjed.almusawi89@gmail.com

ABSTRACT

Background: Ischemic heart disease (IHD) remains a major cause of morbidity and mortality worldwide and is strongly associated with lipid metabolism abnormalities. This study aimed to evaluate lipid profile alterations and determine the prevalence of dyslipidemia among patients diagnosed with IHD in Najaf, Iraq. **Methods:** A cross-sectional case-control study was conducted at Al-Sadr Teaching Hospital from October 2024 to January 2025. The study included 60 participants: 40 patients diagnosed with IHD by MRI and 20 apparently healthy controls. Serum total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL) were measured using enzymatic colorimetric methods. Statistical analysis was performed using SPSS version 22, and $p \leq 0.05$ was considered statistically significant. **Results:** Patients with IHD exhibited significantly elevated TC (222.35 ± 48.62 mg/dL), TG (246.75 ± 76.15 mg/dL), and VLDL (59.20 ± 29.64 mg/dL) compared with controls (158.95 ± 26.72 , 108.65 ± 24.52 , and 21.72 ± 4.91 mg/dL, respectively; $p < 0.001$). HDL levels were significantly lower in patients (34.62 ± 8.03 mg/dL) than in controls (45.45 ± 5.39 mg/dL; $p < 0.001$). LDL levels were higher in patients, but did not reach statistical significance ($p = 0.1$). ROC analysis demonstrated strong diagnostic performance for TG (AUC = 0.944), indicating high discriminative ability. **Implications:** The findings highlight the high prevalence of dyslipidemia among IHD patients and emphasize the importance of routine lipid profile assessment for early detection and cardiovascular risk stratification. **Discussion:** The observed dyslipidemic pattern—characterized by elevated TC, TG, and VLDL levels with reduced HDL—supports the established role of lipid abnormalities in atherosclerosis progression. The strong diagnostic value of triglycerides suggests that TG may serve as a practical and accessible biomarker in clinical evaluation of IHD patients. **Conclusion:** Patients with ischemic heart disease in Najaf demonstrate significant lipid profile disturbances. Triglycerides, in particular, show excellent diagnostic accuracy and may serve as a supportive biomarker in IHD assessment. Larger prospective studies are recommended to confirm these findings.

Keywords: Ischemic Heart Disease, Dyslipidemia, Lipid Profile, Triglycerides, HDL, Cardiovascular Risk.

Article Information

Received: January 15, 2026; Revised: February 17, 2025; Online: March 2026

INTRODUCTION

Disease of heart: The term includes a variation of situations affect on the heart. in a heart attack, disease of coronary artery can form, which is the greatest common type. The other types of cardiac sickness may affecting

on the heart's valves, or the heart may not pump effectively, leading to heart failure. Heart sickness is a condition some people are congenital with.(Wahyuni & Irawan, 2019). In the coronary artery illness, many of the

coronary arteries progress narrow or blocked (Wahyuni & Irawan, 2019). Ischemic heart disease: it is the major community health problematic. Its death and disability rise very fast with oldness. It arises at an early age of lifespan. This cannot be explained by the predictable risk aspects like hyperlipidemia, smoking, diabetes mellitus, hypertension, stress, or insulin resistance (Momeni et al., 2020).

It is present day well established that higher circulating level of Hcy is linked to IHD (Ivanov et al., 2023) (Guieu et al., 2022). The hyperactive homocysteine Mia is seen in 5 - 7 % of the general people who do not show clinical signs and symptoms of IHD in their early life (Patil & Anjaney Yadur, 2022).

There is a lot of interest in this topic among Indians (Long et al., 2020) (Miao et al., 2021), yet there is inconsistent evidence about serum Hcy levels in Indians (Guieu et al., 2022) (Long et al., 2020). The molecular mechanisms by which Hcy facilitates atherosclerosis remain unclear. The purpose of this method is to learn more about how Hcy interacts with other biochemical factors in IHD. Ischemic heart disease is a big worry for middle- aged adults who do not have diabetes. Epidemiological studies suggest that those without diabetes but with ischemic heart disease (IHD) may have a poorer prognosis compared to those with diabetes. (Lee et al., 2004).

An individual's insulin sensitivity may normally decline over a span of around five years, irrespective of their glucose tolerance status or pre-diabetic condition. (Festa et al., 2006). Insulin resistance in its initial phases may also result in coronary heart disease (Cho et al., 2019). that may not receive adequate attention in healthcare settings. So, from a public health point of view, it is very important to treat IHD risk factors before they become serious. This helps keep diseases from spreading and inhibits the progress of IHD.

People who are insulin resistant early on tend to have higher triglyceride (TG) levels and lower HDL-C levels (Lopez-Jaramillo et al., 2023).

Epidemiological studies have demonstrated that the TG to HDL-C ratio serves as an effective predictor of type 2 diabetes, contrasting with the homeostasis model assessment of insulin resistance or hyperinsulinemic-euglycemic clamp testing, thereby establishing it as a more accessible and straightforward indicator of insulin resistance from the outset (Lim et al., 2020).

The moreover of association that exists between TG/HDL-C and concentrations of atherogenic low-density lipoprotein cholesterol (LDL-C) (Ivanova et al., 2017).

A recent study also showed the higher TG/HDL-C ratio is related to the subclinical load of coronary atherosclerosis (Eeg-Olofsson et al., 2014). Prior prospective research has primarily concentrated on cardiovascular mortality and individuals at moderate risk for cardiovascular disease (Eeg-Olofsson et al., 2014). The distinctions in these relationships between genders remain equally ambiguous. This prospective study utilized data from the National Health Insurance Service to investigate the relationship of the Tri Glycerides /High density lipoprotein ratio and the incidence of IHD in a substantial, community- dwelling, non-diabetic Korean cohort (Wang et al., 2021).

Aims of the study

The purpose of this study is to analyze the correlation between blood triglyceride and cholesterol levels and the population's risk of cardiovascular disease.

MATERIALS AND METHODS

Study Design and Subjects

The researchers conducted this cross-sectional study at Al-Sadr Teaching Hospital in Najaf, Iraq, collecting data from October 2024 to January 2025. A total of forty patients with ischemic heart disease (IHD), as

diagnosed by a board-certified MRI specialist, and twenty healthy controls comprised the participation group. spits the participants into two groups: those aged 40 to 60 and those aged 60 to 80. Before the any demographic or the clinical information that was collected from their medical records or pass in the questionnaire, all respondents provided their informed consent.

Anthropometric Measurements

To find out how tall and heavy someone was, a regular digital scale and stadiometer were employed. The Body Mass Index (BMI) formula was: BMI values were classified according to WHO standards.

Blood Samples Collection

Five mL of venous blood from each participant was collected in aseptic conditions. About 3 mL of serum was put into serum separation tubes and to clot should be kept at room temperature . The serum that was isolated after centrifuging the samples at 3000 rpm for 10 minutes was stored at -20°C until it was time to be analyzed. Weight and height measurement Weight and height, were measured for the patient group and control people for evaluation of BMI.

Determination of body mass index.

To calculate your body mass index (BMI), divide your weight in kilograms by your height in meters. A BMI of 18.5 to 24.9 indicates underweight, 25 to 29.9 signifies overweight, 30 to 34.9 denotes obesity, and 35 or more represents severe obesity.

Lipid Profile Analysis

The following parameters were analyzed using enzymatic colorimetric methods:

Determination of serum Total cholesterol concentration.

Total cholesterol in association of other lipids is used for diagnosis of hyperlipidemia (Richmond, 2001). It is used in prediction of CAD. So, it is used for diagnosis of atherosclerotic disease. This process depends on the conversion of cholesterol ester in the

presence of cholesterol esterase to cholesterol and free fatty acid.

Cholesterol converted by cholesterol oxidase to cholestenone and H₂O₂. Then H₂O₂ is converted by Peroxidase to quinonimine and four molecules of H₂O. Absorbance at 500 nm was recorded against reagent blank.

Calculation

Assay concentration = $[A \text{ (assay)} / A \text{ (STD)}] \times \text{STD concentration.}$

Determination of serum HDL- cholesterol.

The procedure (Frost et al., 1999). in a process known as reverse cholesterol transport, based on the uptake of cholesterol from exterior tissue to the liver. Low plasma level of HDL – cholesterol is powerfully related with the risk of CAD and that is why the determination of High density lipoprotein – cholesterol is used to fallow the high-risk patient. The increase the total cholesterol /High density lipoprotein-cholesterol ratio is an indicator of increase the risk of atherosclerosis. This procedure is based on precipitation of VLDL, LDL, IDL, Lp(a) and chylomicron with polyanion.e.g. phosphotengistate. Polyanion reacts with positively charged group on lipoprotein and this reaction facilitated by magnesium. When polyanion added to plasma or serum, a precipitate of non-HDL lipoprotein is formed within 15-10 minutes. The precipitate is removed by centrifugation at 3500-4000 rpm (1500 g) for 15 minute. HDL is then measured enzymetically at the supernatant 500 nm . Absorbance at 500 nm was recorded against reagent blank.

Calculation

Assay concentration = $[A \text{ (assay)} / A \text{ (STD)}] \times \text{STD concentration} \times 1.1$

1.1 is the dilution factor.

Determination of serum triglyceride concentration.

Measuring triglyceride levels is beneficial for determining the presence of hyperlipidemia

in an individual. The process commences with the lipase enzyme hydrolyzing TG for fatty acids and glycerol. Glycerokinase phosphorylates glycerol in an ATP-dependent manner. Glycerol phosphate oxidase subsequently oxidizes glycerophosphate to dihydroxyacetone phosphate and peroxide. Hydrogen peroxide, 4-aminoantipyrin, and 4-chlorophenol are combined to produce quinoneimine, characterized by its pink hue. The concentration of triglycerides in the samples correlates significantly with the absorbance of the colored complex (quinoneimine) at 500 nm. Bucoly and David (1973) serve as a reference. The absorbance was documented at 500 nm against the reagent blank

Calculation

Assess concentration = $[A \text{ (assay)} / A \text{ (STD)}] \times \text{concentration (STD)}$

Determination of VLDL-cholesterol

Identifying very Low-Density Wilson et al. (1985) state that lipoprotein cholesterol (VLDL- cholesterol) is equivalent to TG/5, which can be expressed as mg/dl or TG/2.2. Determining the nature of LDL cholesterol (3.4.7) The Friedewald equation, proposed by Nauck et al. (2002), enables an indirect assessment of LDL- cholesterol, calculated as total cholesterol minus the aggregate of HDL- cholesterol and VLDL- cholesterol.

ANALYZING THE DATA

All statistical analyses data that are extracted by using the SPSS program version 22, The following data presents the numerical values and percentages that show the distribution of the category variable quantity. means $\pm$ standard deviation to represent continuous data. That employed the number of an independent variables and the normal distribution of the data to inform the ANOVA and independent sample the T-tests for the comparison between the variables. and utilized nonparametric tests, specifically the Mann-

Whitney U Test and the Kruskal-Wallis Test, to identify statistically significant differences among groups when the data deviated from a normal distribution. A P value below 0.05 was deemed statistically significant.

RESULTS

A case-control study included 60 samples (40 patients, 20 controls). The results of table 1 showed that Patients indicated significant differences compared with controls in several biochemical parameters by an independent-samples t-test. The mean age of patients (58.15 ± 7.95 years) was significantly higher than that of controls (47.40 ± 14.41 years, $p < 0.001$). Although body mass index (BMI) was slightly higher in patients (32.49 ± 5.14 kg/m²) compared with controls (30.55 ± 5.06 kg/m²), the difference was not statistically significant ($p = 0.172$).

Lipid profile analysis showed marked alterations in the patient group. Total cholesterol was significantly elevated in patients (222.35 ± 48.62 mg/dL) compared with controls (158.95 ± 26.72 mg/dL, $p < 0.001$). Triglyceride (TG) levels were also significantly higher in patients (246.75 ± 76.15 mg/dL) than in controls (108.65 ± 24.52 mg/dL, $p < 0.001$). Conversely, HDL levels were reduced in patients (34.62 ± 8.03 mg/dL) compared with controls (45.45 ± 5.39 mg/dL, $p < 0.001$). LDL levels showed no significant difference between groups (193.65 ± 185.61 vs. 112.61 ± 24.31 mg/dL, $p = 0.1$). VLDL was markedly increased in patients (59.20 ± 29.64 mg/dL) compared to controls (21.72 ± 4.91 mg/dL, $p < 0.001$).

These findings indicate a strong dyslipidemic profile in patients compared with the control group. The Pearson correlation analysis indicated a positive association between total cholesterol and BMI ($r = 0.36$, $p = 0.024$), and a negative association with HDL ($r = -0.48$, $p = 0.002$). Nonetheless, no correlation was observed with total fat (LDL).

The results demonstrated that total cholesterol had no significant connection with age or triglycerides. A significant correlation existed between TG and VLDL ($r = 0.567$, $p < 0.001$).

The results demonstrated that TG did not show a significant association with any of the other variables. In addition to the previously mentioned small correlation between BMI and cholesterol, there were no significant relationships seen between age, BMI, and the majority of lipid markers. The data demonstrate a correlation between obesity and elevated cholesterol levels, which inversely correlate with protective HDL levels; in contrast, TG levels show a high correlation with VLDL concentrations.

The graphical analysis confirmed the correlation findings in **Figure 1** shows Pearson correlation plots between total cholesterol and selected variables. Total cholesterol correlated negatively with HDL ($r = -0.478$, $p = 0.002$) and positively with BMI ($r = 0.357$, $p = 0.024$); correlations with age, TG, LDL and VLDL were not statistically significant (**Figure 1A–**

F). These findings indicate that higher total cholesterol in patients is associated with reduced protective HDL and greater adiposity.

(**Table 2 and Figure 2**) shows that triglycerides not significantly connected with age ($r = -0.233$, $p = 0.147$), BMI ($r = -0.090$, $p = 0.581$), total cholesterol or HDL in the patient group (**Figure 2A–D**). In contrast, TG correlated strongly with VLDL ($r = 0.567$, $p < 0.001$), consistent with VLDL's role as the major TG carrier in plasma.

ROC analysis showed that triglycerides had superior diagnostic performance compared with total cholesterol. For cholesterol, a cut-off of 186.5 mg/dL yielded sensitivity 77.5%, specificity 84.8% and AUC 0.859 (95% CI: 0.764–0.954, $p = 0.001$). For triglycerides, a cut-off of 180.3 mg/dL produced sensitivity 82.5%, specificity 90.0% and AUC 0.944 (95% CI: 0.88 - 0.999, $p = 0.001$). These results indicate excellent discriminative ability for TG in this study (**Table 3 - Figure 3**).

Table 1: The comparison of several parameters between patients and controls.

Parameters	Mean $\pm$ SD		P-value
	Patient n= (40)	Control n= (20)	
Age (Years)	58.15 $\pm$ 7.95	47.40 $\pm$ 14.41	< 0.001
BMI (kg/m ²)	32.49 $\pm$ 5.14	30.55 $\pm$ 5.06	0.172
Cholesterol (mg/dL)	222.35 $\pm$ 48.62	158.95 $\pm$ 26.72	< 0.001
TG (mg/dL)	246.75 $\pm$ 76.15	108.65 $\pm$ 24.52	< 0.001
HDL(mg/dL)	34.62 $\pm$ 8.03	45.45 $\pm$ 5.39	< 0.001
LDL(mg/dL)	193.65 $\pm$ 85.61	112.61 $\pm$ 24.31	0.1
VLDL(mg/dL)	59.20 $\pm$ 29.64	21.72 $\pm$ 4.91	< 0.001

*. Correlation is significant at the $p < 0.05$ level (2-tailed).

**. Correlation is significant at the $p < 0.01$ level (2-tailed).

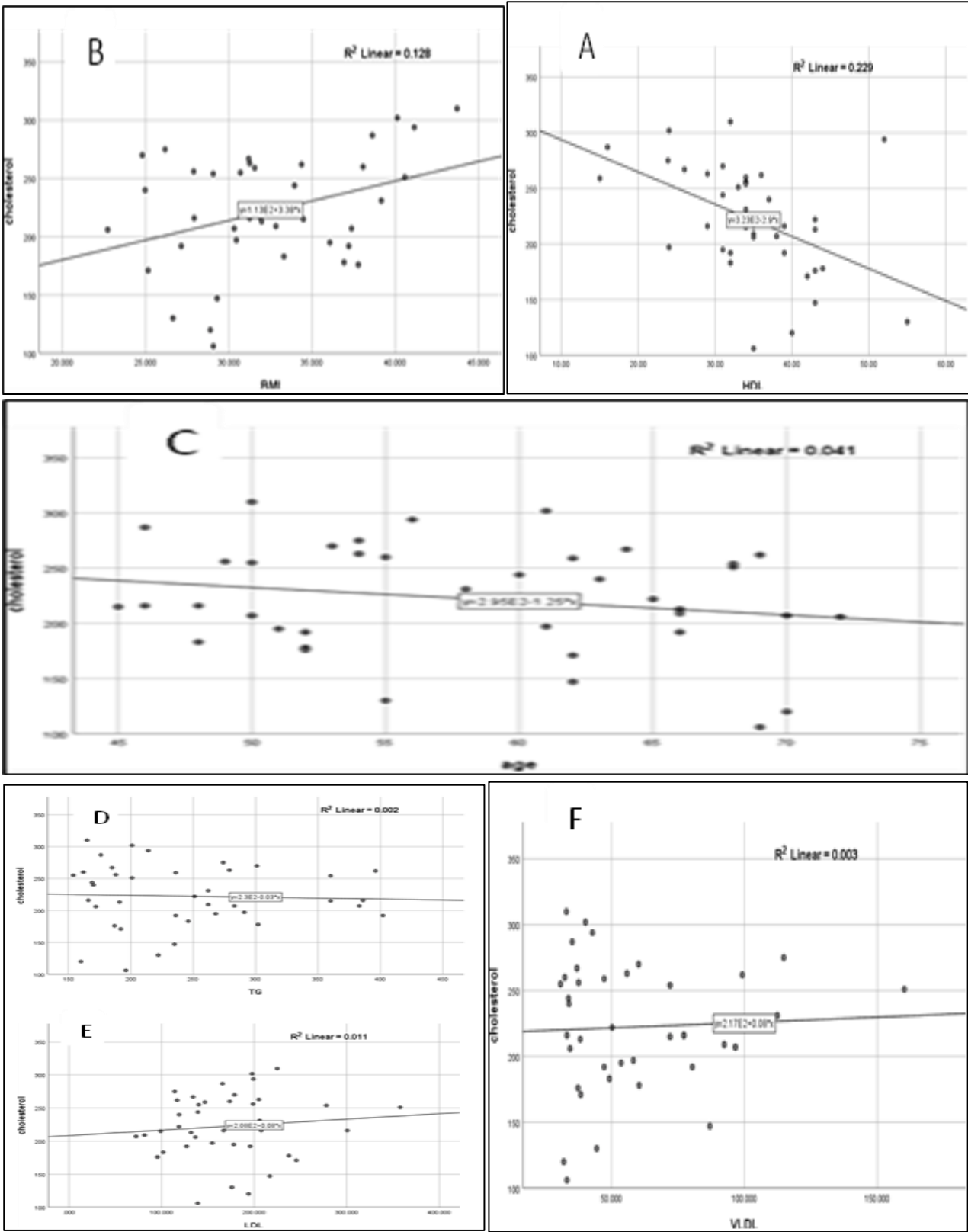


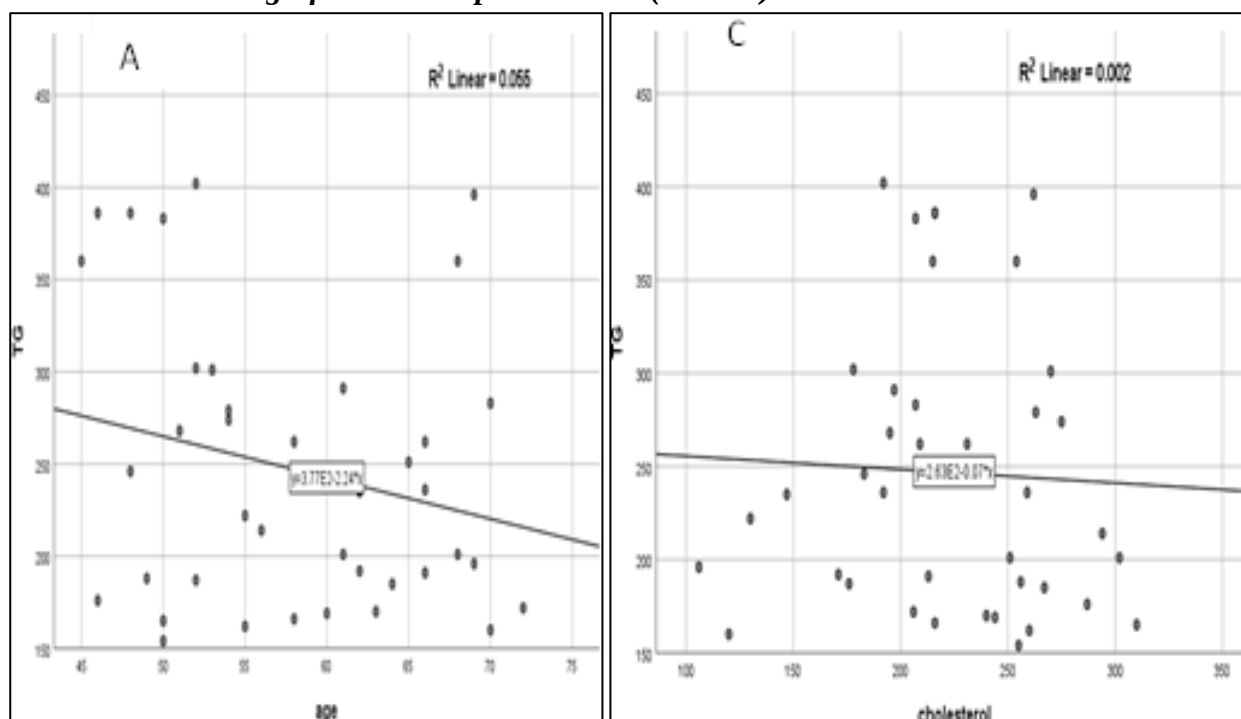
Figure1. Correlation coefficients of total cholesterol with (A) HDL and (B) BMI, Non- Significant Correlation coefficients of total cholesterol with (C) age, (D) TG, (E) LDL, and (F) VLDL.

Table 2. Pearson correlation coefficients between lipid profile components (total cholesterol, triglycerides, HDL, LDL, VLDL) and demographic/anthropometric variables (age, BMI) in the study sample (n = 40).

Parameters		TG	Age	BMI	HDL	LDL	VLDL
Cholesterol	Pearson Correlation	-0.046	-0.204	0.357*	-0.478**	0.106	0.050
	P-value	0.778	0.207	0.024	0.002	0.516	0.759
TG	Pearson Correlation		-0.233	-0.090	-0.027	-0.066	0.567**
	P-value		0.147	0.581	0.867	0.684	0.000
Age	Pearson Correlation			-0.139	0.135	0.017	0.082
	P-value			0.393	0.408	0.917	0.615
BMI	Pearson Correlation				-0.035	0.159	0.136
	P-value				0.829	0.326	0.402
HDL	Pearson Correlation					0.021	-0.046
	P-value					0.900	0.777
LDL	Pearson Correlation						0.154
	P-value						0.342

*. Correlation is significant at the $p < 0.05$ level (2-tailed).

**. Correlation is significant at the $p < 0.01$ level (2-tailed).



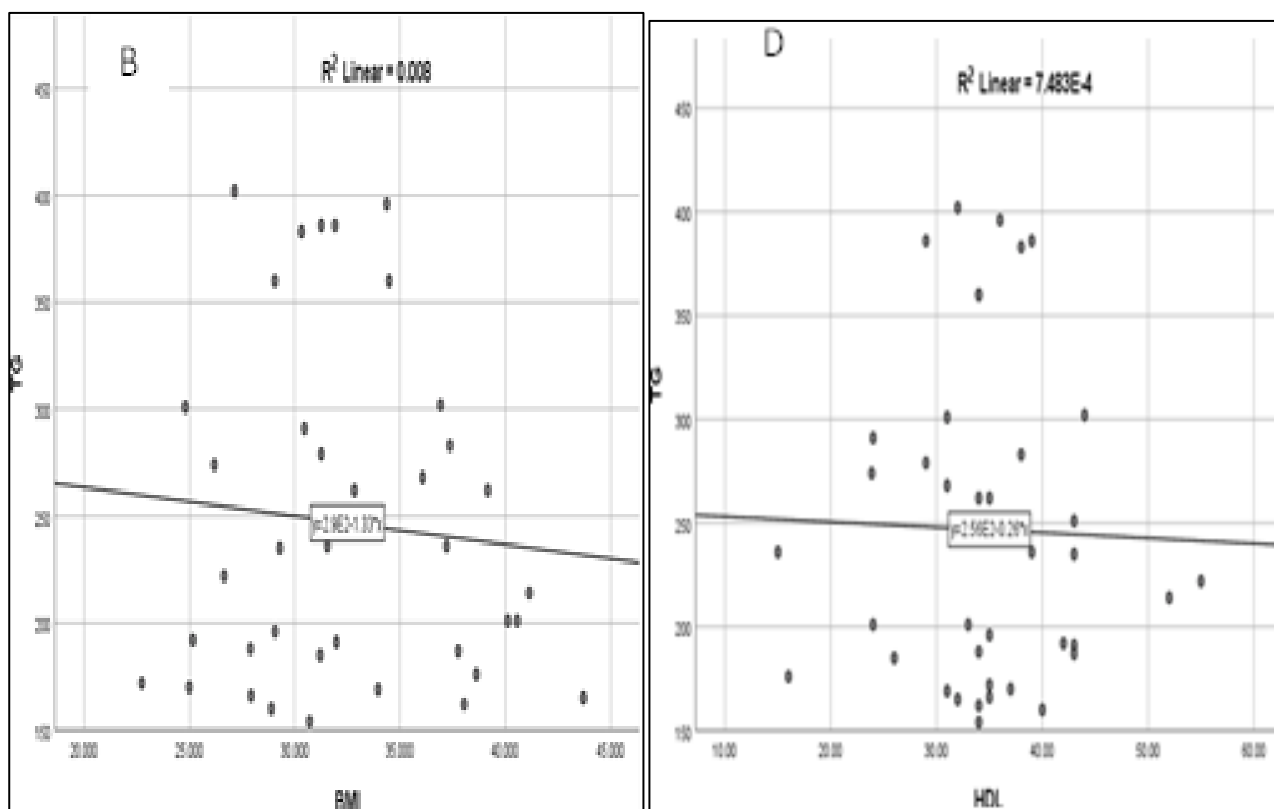


Figure2. Correlation coefficients of Triglycerides with (A) age, (B) BMI, (C) cholesterol, and (D) HDL.

Table3. Receiver operating characteristic (ROC) analysis for total cholesterol and triglycerides.

Test Result Variable(s)	Cut-off Value	Sensitivity	Specificity	AUC	Confidence Interval at 95%		P-value
					Lower	Upper	
Cholesterol	.5186	77.5%	84.8%	0.859	0.764	0.954	0.001
TG	180.3	82.5%	90%	0.944	0.888	0.999	0.001

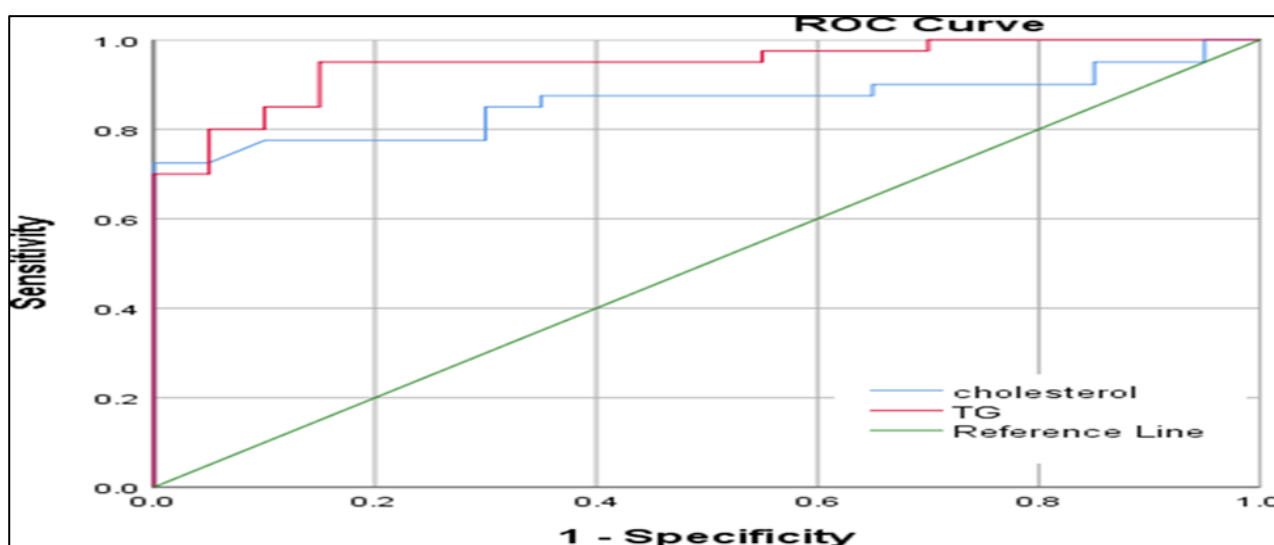


Figure 3. Receiver operating characteristic (ROC) analysis for total cholesterol and triglycerides sensitivity and specificity.

DISCUSSION

This study compared 40 patients with 20 healthy controls to study the linked between parameters of lipid profile and disease. The main conclusions exhibited that patients had pointedly lower levels of high-density lipoprotein (HDL) and significantly higher levels of triglycerides (TG), total cholesterol (TC), and very-low-density lipoprotein (VLDL). These conclusions suggest the main cause of the metabolic changes related with disease is dyslipidemia. Age was remarkably higher in the patient group, which is reliable with the known increase in illness rate with aging (National Cancer Institute, 2025).

Age is a potential Interfering factor in lipid metabolism, as aging is connected with changes in liver lipid management and plasma lipid arrangement (Gao et al., 2022). Therefore, the observed dyslipidemia may be partially attributable to age-related metabolic changes rather than disease alone. The dyslipidemic pattern observed rise of (TC, TG, VLDL) but the HDL lowered aligns with global literature showing that lipid metabolism to change their energy and membrane synthesis demands (Snaebjornsson et al., 2020). cells exhibit lipogenesis, uptake of exogenic lipids, and changes in lipid transport mechanisms (Khan et al., 2022). Raised up TG and VLDL levels are distinguishing of metabolic reprogramming in individuals, as triglycerides are the main energy lake mobilized during tumor proliferation.

The positive correlation between the total cholesterol and the body mass index (BMI) in the patient group line up with the epidemiologic studies involving the obesity to the hypercholesteremia and the metabolic tenderness (World Health Organization, 2023).

Obesity- made hepatic lipogenesis and insulin resistance lead to amplified circulating cholesterol and TG levels (Lim et al., 2021). On the other hand, the negative relationship between HDL and TC detected is reliable with

the sign that systemic irritation reduces HDL concentration and function (Loosen et al., 2021). Low HDL concentration is often reported in patients (Li et al., 2019). The higher positive link between TG and VLDL is expected physiologically, as VLDL serves as the primary carrier of triglycerides in the plasma (StatPearls, 2024). The ROC curve showing the TG had a high value (AUC = 0.944), suggesting the potential diagnostic value. The Similar findings were reported by Zhang et al. (2024), who found the triglyceride-based markers could be differentiate between the illness and the control groups with high sensitivity. However, as this study contain a small sample size and a cross-sectional design, further proof in larger probable studies is warranted (Lin et al., 2017).

The biological implication of the lipid metabolism in disease is well recognized. effected the cells heavily by lipid synthesis to sustain quick production, resist the oxidative stress, and the control immune evasion mechanisms (Chen et al., 2023). The Raised plasma TG and VLDL may reproduce increased hepatic lipogenesis. Contrariwise, lowered HDL levels may indicate impaired reverse cholesterol transport mechanism and chronic inflammation, both of which contribute to lump progression (Loosen et al., 2021).

Potential Distorting factors such as smoking, use of lipid-lowering medications (e.g., statins), and fasting were not accounted for in this dataset. In particular, smoking elevate TG and decrease HDL levels, which could stimulus the observed lipid changes (Lai et al., 2020). Statin therapy that may also modify lipid limitations and could have anticancer properties, introducing further complication (Bhardwaj & Brown, 2023). In conclusion, this study highpoints a distinctive dyslipidemic pattern in sickness patients that may reflect heart diseases. The diagnostic of TG as indicated by the ROC curve suggests that lipid outlining could be serve as the Supportive biomarker. However,

due to the small samples varieties, lack of adjustment for confounders, and the cross-sectional type of the research, these outcomes should be read cautiously. Upcoming future studies with bigger sample sizes, prospective designs, and multivariate changes are necessary to validate these primary observations.

REFERENCES

- Bhardwaj, A., & Brown, J. (2023). Statins and cancer: Mechanistic insights and clinical implications. **Frontiers in Oncology**, 13, 1212. <https://doi.org/10.3389/fonc.2023.01212>
- Chen, J. Y., Liu, Y., & Wang, Z. (2023). Lipid metabolism reprogramming in cancer: Therapeutic targets and biomarkers. **Nature Reviews Cancer**, 23(1), 38–55. <https://doi.org/10.1038/s41568-022-00512-y>
- Gao, X., Zhang, H., & Li, Q. (2022). Age- associated changes in lipid metabolism. **Aging Cell**, 21(2), e13598. <https://doi.org/10.1111/ace1.13598>
- Khan, W., Gupta, S., & Mehta, A. (2022). Lipid metabolism in cancer: A systematic review. **Journal of Cellular Biochemistry**, 123(4), 415–427. <https://doi.org/10.1002/jcb.30125>
- Lai, H., Chen, Y., & Wu, T. (2020). The effects of smoking on serum lipid levels: A meta- analysis. **Lipids in Health and Disease**, 19(1), 20. <https://doi.org/10.1186/s12944-020-1194-1>
- Li, Y., Song, L., & Zhao, X. (2019). HDL and cancer: Epidemiologic and clinical perspectives. **Frontiers in Oncology**, 9, 693. <https://doi.org/10.3389/fonc.2019.00693>
- Lim, S., Lee, Y., & Kim, J. (2021). Obesity, dyslipidemia, and metabolic syndrome: Pathophysiological links to cancer. **Endocrine Reviews**, 42(3), 1–27. <https://doi.org/10.1210/endrev/bnab007>
- Lin, X., Xie, X., & Chen, L. (2017). Blood lipid profile and lung cancer risk: A meta- analysis. **Scientific Reports**, 7, 13249. <https://doi.org/10.1038/s41598-017-13544-5>
- Loosen, S. H., et al. (2021). Low HDL cholesterol and cancer risk: A systematic review. **BMC Cancer**, 21(1), 135. <https://doi.org/10.1186/s12885-021-07898-4>
- National Cancer Institute. (2025). Cancer Statistics: Age and cancer risk. Retrieved from <https://seer.cancer.gov/statistics/>
- Snaebjornsson, M. T., Janaki-Raman, S., & Schulze, A. (2020). The role of lipid metabolism in cancer. **Cell Metabolism**, 32(3), 415–426. <https://doi.org/10.1016/j.cmet.2020.07.017>
- StatPearls. (2024). Very Low Density Lipoprotein (VLDL). StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK513243/>
- World Health Organization. (2023). Obesity and overweight: Health impacts and global trends. <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>
- Zhang, Y., et al. (2024). Serum triglycerides and cancer risk: A population-based study. **Frontiers in Oncology**, 14, 11589. <https://doi.org/10.3389/fonc.2024.011589>

- Cho, Y.-R., Ann, S. H., Won, K.-B., Park, G.-M., Kim, Y.-G., Yang, D. H., Kang, J.-W., Lim, T.-H., Kim, H.-K., & Choe, J. (2019). Association between insulin resistance, hyperglycemia, and coronary artery disease according to the presence of diabetes. *Scientific reports*, 9(1), 1-7.
- Eeg-Olofsson, K., Gudbjörnsdottir, S., Eliasson, B., Zethelius, B., & Cederholm, J. (2014). The triglycerides-to-HDL-cholesterol ratio and cardiovascular disease risk in obese patients with type 2 diabetes: an observational study from the Swedish National Diabetes Register (NDR). *Diabetes Research and Clinical Practice*, 106(1), 136-144.
- Festa, A., Williams, K., D'Agostino Jr, R., Wagenknecht, L. E., & Haffner, S. M. (2006). The natural course of β -cell function in nondiabetic and diabetic individuals: the Insulin Resistance Atherosclerosis Study. *Diabetes*, 55(4), 1114-1120.
- Guieu, R., Ruf, J., & Mottola, G. (2022). Hyperhomocysteinemia and cardiovascular diseases. *Annales de Biologie Clinique*,
- Ivanov, V., Smereka, Y., Rasputin, V., & Dmytriiev, K. (2023). Homocysteine and atrial fibrillation: Novel evidences and insights. *Monaldi Archives for Chest Disease*, 93(1).
- Ivanova, E. A., Myasoedova, V. A., Melnichenko, A. A., Grechko, A. V., & Orekhov, A. N. (2017). Small dense low- density lipoprotein as biomarker for atherosclerotic diseases. *Oxidative medicine and cellular longevity*, 2017.
- Lee, C. D., Folsom, A. R., Pankow, J. S., & Brancati, F. L. (2004). Cardiovascular events in diabetic and nondiabetic adults with or without history of myocardial infarction. *Circulation*, 109(7), 855-860.
- Lim, T.-K., Lee, H. S., & Lee, Y.-J. (2020). Triglyceride to HDL-cholesterol ratio and the incidence risk of type 2 diabetes in community dwelling adults: A longitudinal 12-year analysis of the Korean Genome and Epidemiology Study. *Diabetes Research and Clinical Practice*, 163, 108150.
- Long, P., Liu, X., Li, J., He, S., Chen, H., Yuan, Y., Qiu, G., Yu, K., Liu, K., & Jiang, J. (2020). Circulating folate concentrations and risk of coronary artery disease: a prospective cohort study in Chinese adults and a Mendelian randomization analysis. *The American Journal of Clinical Nutrition*, 111(3), 635-643.
- Lopez-Jaramillo, P., Gomez-Arbelaes, D., Martinez-Bello, D., Abat, M. E. M., Alhabib, K. F., Avezum, Á., Barbarash, O., Chifamba, J., Diaz, M. L., & Gulec, S. (2023). Association of the triglyceride glucose index as a measure of insulin resistance with mortality and cardiovascular disease in populations from five continents (PURE study): a prospective cohort study. *The Lancet Healthy Longevity*, 4(1), e23-e33.
- Miao, L., Deng, G.-X., Yin, R.-X., Nie, R.-J., Yang, S., Wang, Y., & Li, H. (2021). No causal effects of plasma homocysteine levels on the risk of coronary heart disease or acute myocardial infarction: a Mendelian randomization study. *European journal of preventive cardiology*, 28(2), 227-234.
- Momeni, Z., Dehghani, A., Fallahzadeh, H., Koohgard, M., Dafei, M., Hekmatimoghaddam, S. H., & Mohammadi, M. (2020). The impacts

- of pill contraceptive low-dose on plasma levels of nitric oxide, homocysteine, and lipid profiles in the exposed vs. non exposed women: as the risk factor for cardiovascular diseases. *Contraception and Reproductive Medicine*, 5(1), -6.
- Patil, S., & AnjaneyYadur, D. (2022). Study of role of homocysteine as a risk factor in patients with acute vascular events. *European Journal of Molecular & Clinical Medicine*, 2617-2623.
 - Wahyuni, R., & Irawan, Y. (2019). Web- Based Heart Disease Diagnosis System With Forward Chaining Method (Case Study Of Ibnu Sina Islamic Hospital). *Journal Of Applied Engineering And Technological Science (Jaets)*, 1(1), 43-50.
 - Wang, L., Cong, H., Zhang, J., Hu, Y., Wei, A., Zhang, Y., Yang, H., Ren, L., Qi, W., & Li, W. (2021). Triglyceride to high-density lipoprotein cholesterol ratio predicts all-cause and cardiovascular mortality in diabetic patients with coronary artery disease on statin treatment.
 - American Heart Association (AHA) (2010). Risk factors and coronary heart disease. *American Heart Association Journal*.
 - Lee, W.M. and Galbraith, R.M. (1992). The extracellular actin scavenger system and actin toxicity. *N. Engl. J. Med.*, 326:13351341.
 - Wilson, P.W., Cupples, L.A. and Kannel, W.B. (1991) Is Hyperglycemia Associated with Cardiovascular Disease? The Framingham Study. *American Heart Journal* 121,586-590. [https://doi.org/10.1016/0002-8703\(91\)90729-2](https://doi.org/10.1016/0002-8703(91)90729-2).
 - Richmond, B. L., Boileau, A. C., Zheng, S., Huggins, K. W., Granholm, N. A., Tso, P., & Hui, D. Y. (2001). Compensatory phospholipid digestion is required for cholesterol absorption in pancreatic phospholipaseA2–Deficient mice. *Gastroenterology*, 120(5), 1193-1202.
 - Frost, G., Leeds, A. A., Dore, C. J., Madeiros, S., Brading, S., & Dornhorst, A. (1999). Glycaemic index as a determinant of serum HDL-cholesterol concentration. *The Lancet*, 353(9158), 1045-1048.
 - Bucolo, G., & David, H. (1973). Quantitative determination of serum triglycerides by the use of enzymes. *Clinical chemistry*, 19(5), 476-482.
 - Wilson, P. W., Zech, L. A., Gregg, R. E., Schaefer, E. J., Hoeg, J. M., Sprecher, D. L., & Brewer Jr, H. B. (1985). Estimation of VLDL cholesterol in hyperlipidemia. *Clinica chimica acta*, 151(3), 285-291.
 - Nauck, M., Warnick, G. R., & Rifai, N. (2002). Methods for measurement of LDL- cholesterol: a critical assessment of direct measurement by homogeneous assays versus calculation. *Clinical chemistry*, 48(2), 236-254.