The Medical Journal of Tikrit University (2025) 31 (2): 378-388
DOI: http://dx.doi.org/10.25130/mjotu.31.2.39

IRAQI Academic Scientific Journals

ISSN: 1813-1638

The Medical Journal of Tikrit University

Available online at:www.mjotu.com

MJTU

The Medical Journal of Tikrit University

Dyslipidemia and Metabolic Risk Factors in Postmenopausal Iraqi Women with Chronic Heart Disease: A Case-Control Study

Asen Hussein Salih [1]; Nihad Nejres Hilal [1]

[1] Department of Clinical Biochemistry, College of Medicine, University of Tikrit, Iraq.

Keywords: *Postmenopausal women, Chronic heart disease (CHD), Lipid profile, Dyslipidemia, total cholesterol (TC), High-density lipoprotein (HDL), Triglycerides (TG), Low-density lipoprotein (LDL), Very-low-density lipoprotein (VLDL)*

ARTICLE INFO

Article history:

Received 01 Jul 2025
Accepted 01 Sep 2025
Available online 31 Dec 2025

Citation:

Corresponding Email:
AH240009me@st.tu.edu.iq

ABSTRACT

Background: The main reason postmenopausal women die is cardiovascular disease; dyslipidemia is a major contributing factor. This study evaluates postmenopausal women with chronic heart disease (CHD) to clarify how changes in lipid profile affect disease progression.

Methods: Case-control research is conducted on ninety 50–60-year-old women from Kirkuk, Iraq. 30 age-matched healthy controls were matched against 60 postmenopausal women diagnosed with CHD. Serum total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL) were determined using enzymatic spectrophotometry.

Results: Postmenopausal CHD patients exhibited significantly elevated levels of total cholesterol (201.2 ± 40.08 mg/dL), TG (154.34 ± 44.90 mg/dL), LDL (129.62 ± 39.95 mg/dL), and VLDL (30.86 ± 8.98 mg/dL) compared to controls. HDL levels were significantly reduced (40.73 ± 9.28 mg/dL vs. 51.33 ± 6.26 mg/dL; $p < 0.05$).

Conclusion: In postmenopausal women, dyslipidemia is significantly correlated with CHD, which underlines the importance of lipid monitoring and early treatment in this high-risk population.

INTRODUCTION

Women after menopause particularly at increased risk with cardiovascular diseases (CVDs) which is remain the primary cause of morbidity and death worldwide. This is mostly related to hormonal changes—especially the drop in estrogen—which is very important for controlling lipid metabolism, blood flow, and inflammatory responses[1]. Decreasing (LDL) cholesterol, increasing (HDL) cholesterol, and preserving endothelial integrity are among estrogen's cardioprotective actions [2].

Dyslipidemia, increased oxidative stress, metabolic syndrome, and endothelial dysfunction all contribute to postmenopausal women's higher susceptibility to cardiovascular issues[3, 4]. Studies have found that the change to menopause is linked to raised total cholesterol, LDL, triglycerides (TG), and lowered HDL, all of which support atherosclerotic processes and the development of coronary heart disease (CHD) [5].

Despite such risks, unusual symptoms and poor public knowledge cause women's cardiovascular diseases to be often underdiagnosed or misattributed. Women who experience delayed detection of cardiovascular symptoms can have later-stage diagnosis and less-than-ideal outcomes from treatment.[6] In areas like Iraq, where there is little gender-specific cardiovascular screening and health education.[7] This study is to assess, in Kirkuk, Iraq, postmenopausal women diagnosed with CHD's lipid profile in comparison to age-matched healthy controls. Improving early diagnosis, preventive policies, and the general management of cardiovascular health in postmenopausal women depends on an awareness of these lipid abnormalities.

Objectives of the Study:

1- To Compare the lipid profile (total cholesterol, LDL, HDL, triglycerides, and VLDL) of postmenopausal women with coronary heart disease versus healthy controls.

2- To examine associations between blood pressure, BMI, and lipid abnormalities in CHD patients.

MATERIALS AND METHODS

Study Design and Participants:

A case control study which was conducted in Kirkuk Governorate from late December 2024 to the end of April 2025 and involved 90 participants aged 50 to 60 years, including 60 patients with chronic heart disease, who were under the supervision of a specialist physician for treatment and clinical observation. A control group of 30 individuals without chronic heart disease was selected. Laboratory analyses were performed on both groups to assess the concentrations of lipid profile using a spectrophotometer.

Exclusion Criteria:

Women under 50 (premenopausal), those within six-month acute cardiovascular events (such as stroke or myocardial infarction), or those on lipid-lowering or antioxidant drugs were not included. Those with thyroid problems, chronic inflammatory or autoimmune illnesses, cancer, prior six-month alcohol usage or smoking were also eliminated.

Data Collection:

Structured questionnaires were used to obtain primary data through face-to-face interviews. The questionnaire collected personal and demographic information (age, date of birth, gender), medical history (metabolic disorders, hypertension, diabetes), family history, and drug consumption.

Samples collection and handling:

Five ml of venous blood were drawn from a suitable vein in each patient and control participant. Fasting samples were obtained between 8 and 9 A.M. The blood was let to coagulate for 20 minutes in gel tubes at room temperature (25 °C). The blood serum was extracted using centrifugation at 3000 rpm along 10 minutes. 1 ml of serum was partitioned into two equal aliquots and put into 0.5 ml Eppendorf tubes. The residual serum was transferred to a distinct sterile 0.5 ml tube for preservation at -20°C in a deep freezer until analysis.

Ethical approval

Ethical approval for this investigation was obtained from Kirkuk General Hospital in Kirkuk city. The Scientific Council of the Faculty of Medicine at Tikrit University granted official approval for the research protocol, which had previously accepted the technique. All participants in the research were informed of its purpose, methodology, associated risks and benefits, and objectives. Prior to data and sample collection, each participant provided written consent. The research meticulously maintained the confidentiality and identity of participants.

Biochemical Analysis

- **TC and HDL**: Measured using enzymatic CHOD-PAP method.
- **TG**: Measured using GPO-PAP enzymatic method.
- **LDL**: Calculated using the Martin-Hopkins formula.
- **VLDL**: Derived from triglyceride values (TG/5).

Determination of total Cholesterol[8]

Principles of the tests

Cholesterol esterase degrades total cholesterol into free cholesterol and fatty acids by enzymatic hydrolysis. Cholesterol oxidase subsequently oxidizes free cholesterol to produce cholest-4-en-3-one and hydrogen peroxide (H_2O_2). In the presence of peroxidase, H_2O_2 reacts with a chromogen (often 4-aminoantipyrine and phenol) to produce a colored quinoneimine dye. The dye is identified at 500 nm via a spectrophotometer.

Cholesterol esterase

Cholesteryl esters ⟶ Free cholesterol+Fatty acids

Cholesterol oxidase

Cholesterol+O2 ⟶ Cholest-4-en-3-one+H2O2

Peroxidase

H2O2+Phenol+4-Aminoantipyrine ⟶ Quinoneimine +4H2O

Cholesteryl esters constitute the majority of cholesterol present in the bloodstream. Cholesterol esterase facilitates the hydrolysis of these compounds into free cholesterol and fatty acids. Cholesterol oxidase and oxygen (O_2) are introduced to free cholesterol, resulting in its oxidation. This produces cholestenone (cholest-4-en-3-one) and hydrogen peroxide (H_2O_2).

The produced hydrogen peroxide subsequently reacts with a mixture of phenol and 4-aminoantipyrine in the presence of the

enzyme peroxidase (POD). This produces a colored quinoneimine dye and water. The intensity of the pink / red color formed is proportional to the cholesterol concentration.

Calculation

$$C = \left(\frac{A_s - A_b}{A_{std} - A_b}\right) \times C_{std}$$

C = The cholesterol concentration in the test specimen (mg/dL)
A s = The absorbance (optical density) of the blood sample
A b = Absorbance of the reagent blank
A std= A standard deviation (std) represents the quantity of light absorbed by the standard solution.
C std = the concentration of the standard in a solution (often 200 mg/dL)

Determination of Triglyceride (9)

Principle of the tests

The GPO-PAP (glycerol-3-phosphate oxidase-phenol aminophenazone) enzymatic method was employed to measure triglycerides. Lipase facilitates the breakdown of triglycerides in the assay, producing glycerol and free fatty acids. Glycerol kinase facilitates the phosphorylation of glycerol to produce glycerol-3-phosphate, which is then oxidized by glycerol-3-phosphate oxidase to generate hydrogen peroxide. In the presence of peroxidase, hydrogen peroxide reacts with 4-aminophenazone and phenol to produce a vibrant hue. We assessed the absorbance within the range of 520 to 550 nm and using a standard curve to determine the triglyceride concentration in the sample.

Reaction sequence:

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

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

$$\text{Glycerol – 3- phosphate + } O_2 \xrightarrow[\text{oxidase}]{\text{Glycerol-3-phosphate}} \text{ADP + (Dihydroxyaceton phosphate) + } H_2O_2$$

$$2H_2O_2 + \text{parachlorophenol + 4-aminoantipyrine} \xrightarrow{\text{Peroxidase}} \text{Quinoneimine + } 4H_2O$$

Calculation

$$TG = \left(\frac{A_s - A_b}{A_{std} - A_b}\right) \times C_{std}$$

G = The concentration of triglycerides in mg/dL
A_s = The absorbance of the sample (optical density)
Ab = The absorbance of the blank
Astd = The absorbance of the standard
Cstd = The quantity of the standard (often 200 mg/dL

Estimation of HDL – Cholesterol[10]

Principle of the tests

We employed a two-step precipitation enzymatic method to quantify HDL-C. Initially, blood samples were subjected to treatment with a precipitation reagent containing phosphotungstate and magnesium chloride, or alternatively, dextran sulfate-Mg^{2+}. This reagent exclusively precipitated lipoproteins containing apolipoprotein B, namely VLDL and LDL. The mixture was subsequently centrifuged at 1500×g for 10 minutes to isolate the precipitate. The supernatant containing HDL particles was collected and analyzed for cholesterol content using the CHOD-PAP enzymatic method, identical to that employed for total cholesterol measurement. Standard

calibrators were employed to determine the concentration of HDL-C in the sample by measuring the absorbance at 500 nm.[9]

Calculation

$$HDL = \left(\frac{A_s}{A_{std}}\right) \times n \times 2$$

High-Density Lipoprotein Cholesterol (mg/dL)
A_s: The absorbance of the sample
A_{std}: The absorbance of the standard
n: The quantity of the standard (mg/dL)
2: Dilution factor (if a constant volume factor or reagent-specific multiplier is utilized)

Estimation of Low-Density Lipoprotein Cholesterol (LDL-C)[11]

The Martin-Hopkins formula was employed to calculate LDL-C. This method is more precise than the Friedewald equation, particularly in samples with low LDL or elevated triglycerides. The Martin-Hopkins equation employs a patient-specific component to calculate very low-density lipoprotein cholesterol (VLDL-C). This method determines LDL and VLDL utilizing the measured values of Total Cholesterol (TC), HDL-Cholesterol, and Triglycerides (TG). It appears as follows:

$$LDL = \frac{\text{Total cholesterol} - \text{HDL}}{\text{Triglycerides}/5}$$

$$VLDL = \frac{\text{Triglycerides}}{5}$$

Anthropometric Data: We assessed the height and weight of all individuals in both groups to calculate their Body Mass Index (BMI) with the standard formula:

$$\text{BMI} = \frac{\text{Weight (kg)}}{\text{Height (m)}^2}$$

These findings clarify the nutritional and metabolic conditions of people, identified risk factors for cardiovascular disease and oxidative stress in postmenopausal women.

Classification of the World Health Organization of BMI (WHO)[12]

BMI(kg/m2)	Classification	Risk of obesity co- morbidity
< 18.5	Under weigh	None
18.5 - 24.9	Normal range	Negligible
25.0 – 29.9	Overweight	Mildly increased
>30.0 30.0 – 34.9 35.0 – 39.9 >40.0	Obese Class I Class II Class III	Moderate Severe Very Severe

- **Blood Pressure**: A certified sphygmomanometer was used to measure both systolic and diastolic blood pressures.
- **Statistical Analysis:**

All data were examined with Minitab software employing ANOVA. Continuous variables were reported as mean ± standard deviation (SD), whereas categorical data were conveyed as percentages. Duncan's multiple range test was employed for comparisons between the patient and control groups, while Pearson correlation coefficients were computed to evaluate the correlations among biochemical markers, lipid profiles, BMI, and blood pressure. A p-value less than 0.05 was deemed statistically significant.

RESULTS

The table of Demographic and clinical characteristics shows the variations in demographic and clinical factors between the control group (n = 30) and the patient group (n = 60) of postmenopausal women. The distinctions are significant for chronic heart disease (CHD):

The mean age of the patients (56.4 ± 2.9) was slightly greater than that of the control group (55.1 ± 3.2), although the difference was not statistically significant (p = 0.071). Patients had significantly increased weight and BMI relative to controls (p = 0.002 and 0.001, respectively). Patients had a higher prevalence of familial history of both diabetes mellitus (DM) and hypertension (HTN) at 30%, in contrast to controls at 10%. Both systolic and diastolic blood pressure were significantly elevated in patients (p < 0.001), substantiating hypertension as a critical risk factor.

This study aimed to evaluate the prevalence of chronic heart disease (CHD) in postmenopausal women aged 50 to 60 years. The findings show that most CHD occurrences were in the 50–55 age group, including 51.7% of patients, whereas 48.3% were in the 55–60 age range. while in control group 60% represent the 50–55 age group whereas 40% were in in the 55–60 age range.

The table show a strong significant correlation between BMI and chronic heart disease. The women with CHD in this research show that 11.67% of postmenopausal women with CHD are of normal weight; the great majority (68.33%), fall into the obese categories (Class I 33.33% and class II 35.00%). This distribution These findings show that body weight plays important role in cardiovascular risk in postmenopausal women and emphasizes the clear correlation in this population between high BMI and the frequency of chronic heart disease.

This study observed significant alterations in lipid profile features between healthy controls and postmenopausal women with chronic heart disease. The patient cohort exhibited a substantial decrease in high-density lipoprotein (HDL) concomitant with significantly increased levels of total cholesterol, (TG), (LDL), and (VLDL). P < 0.05.

As shown in the table total cholesterol (TC) level was notably elevated in patient group (201.20±40.08 mg/dl) compared to control group. (170.33±26.68 mg/dl) with P-value: < 0.05. Triglycerides (TG) in the patient group (154.34 ± 44.90 mg/dl) exhibited markedly elevated triglyceride levels than control group (94.86 ± 15.86 mg/dl). P-value: < 0.05. HDL cholesterol, was much lower in the sick group (40.73 ± 9.28 mg/dl) compared to the control group (51.33 ± 6.26 mg/dl), with a P-value of < 0.05. Low-Density Lipoprotein (LDL) was markedly elevated in the patient group (129.62 ± 39.95 mg/dl) compared to the control group (100.02 ± 24.73 mg/dl). P-value: less than 0.05. Very Low-Density Lipoprotein (VLDL) levels were markedly higher in patients at 30.86 ± 8.98 mg/dl compared to the control group at 18.97 ± 3.17 mg/dl. P-value< 0.05

DISCUSSION

Age alone may not be a determinant of coronary heart disease (CHD); rather, age-associated oxidative and metabolic alterations persist in affecting

cardiovascular risk.[13]. In postmenopausal women, an elevated BMI and weight are significantly associated with an increased risk of cardiovascular disease, metabolic syndrome, and oxidative stress.[14] Hypertension is an acknowledged contributor to endothelial dysfunction and oxidative injury in postmenopausal women. A significant association was seen between the history of diabetes and hypertension($p=0.003$).[15] A strong family history of metabolic problems may predispose individuals to coronary heart disease by genetic and epigenetic processes that increase lipid dysregulation and oxidative stress.[16]

The results revealed that whereas 48.3% of CHD cases occurred in the 55–60 age range (Table 4.2), most instances—51.7%—were found in the 50–55 age group. These results imply that the risk of cardiovascular disorders starts to increase fast soon after menopause and stays high in the next years. A research in (2024) observed similar age-based cardiovascular patterns whereby women in the early postmenopausal stage had raised blood pressure and oxidative stress indicators compared to healthy controls.[17]

Furthermore, an extensive study covering Middle Eastern communities revealed that women in the first few years after menopause had very high rates of metabolic syndrome, a substantial risk factor for cardiovascular disease; which rates 60% among Iranian populations. This is in agreement with our results indicating early rising cardiovascular load in Iraqi postmenopausal women.[18] Among widely recognized major modifiable risk factor for cardiovascular disease is obesity. After menopause, the drop in estrogen helps to cause an undesirable redistribution of fat, usually central or abdominal, which is strongly associated with rising cardiovascular events. Our investigation found that the patient group definitely showed this pattern.[19]

Results from national research on Korean women similarly showed a linear rise in cardiovascular disease risk when BMI rose among postmenopausal women.[20] This study recognized that obesity is the most important risk factor for cardiovascular disorders. The drop in estrogen's levels in postmenopausal women helps to explain negative changes in body fat distribution, lipid profiles, and insulin sensitivity, all of which could increase cardiovascular risk. Recent research has shown how obesity affects cardiac condition in postmenopausal women. For women who experience late menopause (age 55 or older), for example, Many studies have demonstrated that obesity is correlated with a delayed start of menopause.[21]

Moreover, research in the European Heart Journal underlined that, even in women with a normal BMI, regional body fat distribution—especially greater trunk fat is linked to a higher risk of cardiovascular disease.[22] These results align with the data we collected for our study, where higher BMI categories match higher prevalence of chronic heart disease. According to the data, particular fat distribution patterns as well as general obesity help to determine cardiovascular risk in postmenopausal women.[23]

The study shows postmenopausal women with CHD had significant dyslipidemia relative to healthy controls. Research done in Baghdad found comparable findings regionally: postmenopausal women had greater triglyceride and LDL-C levels and lower HDL-C levels than premenopausal women. These data highlight how

consistently lipid profile alterations during menopause vary across various groups.[24] Because their estrogen is lower, postmenopausal women have greater TC levels; usually, this increases hepatic LDL receptor activation and helps to remove cholesterol. In prior research, revealing a significant elevation in total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C) with prolonged menopause duration, indicating that alterations in lipid profiles among postmenopausal women are likely attributable to hormonal fluctuations. The lack of estrogen post-menopause highlights its essential role in lipid metabolism and distribution, hence increasing the risk of cardiovascular morbidity and death.[9]

Elevations in HDL-C levels during and after menopause were linked to the advancement of atherosclerosis. This discovery aligns with previous results from an independent cohort of postmenopausal women suggesting that heightened HDL-C is associated with an elevated risk of carotid plaque formation.[25] A recent study indicated that postmenopausal hyperlipidemia is directly caused by metabolic syndrome resulting from hypoestrogenism, which leads to increased central adiposity and insulin resistance. Elevated visceral adiposity leads to adipose tissue hypertrophy, diminishing fatty acid sequestration and hence enhancing hepatic absorption of free fatty acids. This leads to increased triglyceride production, hence raising VLDL secretion in the liver. The rise in visceral adiposity among postmenopausal women mostly stems from decreased endogenous estrogen levels.[26]

Hormonal changes—most notably a drop in estrogen levels—that accompany the menopause modify lipid metabolism. While HDL levels drop, this hormonal change causes raised levels of total cholesterol, LDL, and TG. With HDL levels essentially unaltered, a meta-analysis included over 114,000 women found that postmenopausal women show noticeably higher levels of total cholesterol, LDL, and TG than premenopausal women.[27] These results make regular lipid profile tests for postmenopausal women—especially those with established cardiovascular diseases—even more important. Early dyslipidemia diagnosis and management by medication and lifestyle changes helps to lower the increased risk of cardiovascular events in this population.

CONCLUSION

This study underlines the notable changes in lipid profile characteristics between postmenopausal women with chronic heart disease and healthy controls. The results suggest that dyslipidemia is a common and significant risk factor in this group, thereby maybe helping to aggravate cardiovascular problems.

RECOMMENDATION

These findings highlight the need of focused lipid control for postmenopausal women within a whole cardiovascular risk evaluation.

Preventive policies are crucial considering the higher cardiovascular risk in postmenopausal women with dyslipidemia. Early dyslipidemia detection made possible by routine lipid profile screening will enable quick intervention and assist to lower the burden of chronic heart disease in this sensitive population.

REFERENCES

1. Phan BAP, Toth PP. Dyslipidemia in women: etiology and management. International journal of women's health. 2014:185-94.

2. Mendelsohn ME. Protective effects of estrogen on the cardiovascular system. The American journal of cardiology. 2002;89(12):12-7.

3. Cole J, Zubirán R, Wolska A, Jialal I, Remaley AT. Use of apolipoprotein B in the era of precision medicine: time for a paradigm change? Journal of Clinical Medicine. 2023;12(17):5737.

4. Allami M. A Cross-Sectional Study on the Epidemiology and Risk Factors of Acute Coronary Syndrome in Northern Iraq. Cureus. 2024;16(6).

5. Mohajan D, Mohajan HK. Body mass index (BMI) is a popular anthropometric tool to measure obesity among adults. Journal of Innovations in Medical Research. 2023;2(4):25-33.

6. Singh S. Examining the simultaneous influence of individual and neighborhood determinants on cardiovascular health: a multilevel study in Canadian adults: The University of Western Ontario (Canada); 2020.

7. Sinha T, Bakht D, Bokhari SFH, Amir M, Fatima R, Bakht K, et al. Gender Matters: A Multidimensional Approach to Optimizing Cardiovascular Health in Women. Cureus. 2024;16(6).

8. Leth KW, Dalgård C, Gerke O, Lindholt JS, Lambrechtsen J, Frost L, et al. Sex-specific associations between total cholesterol and non–high-density lipoprotein cholesterol and the presence and extent of coronary artery calcifications. European Journal of Preventive Cardiology. 2025:zwaf047.

9. Hamza A, Abbas M, Elfaki E, Ibrahim M, Abdalhabib EK, Karar T. Assessment of plasma lipid profile among Sudanese menopausal women in Khartoum State-Sudan. Biomedical and Pharmacology Journal. 2019;12(04):2037-41.

10. Ambikairajah A, Walsh E, Cherbuin N. Lipid profile differences during menopause: a review with meta-analysis. Menopause. 2019;26(11):1327-33.

11. Drobnik S, Scharnagl H, Samani NJ, Braund PS, Nelson CP, Hollstein T, et al. Evaluation of current indirect methods for measuring LDL-cholesterol. Clinical Chemistry and Laboratory Medicine (CCLM). 2025;63(6):1099-108.

12. Hilleary RS, Jabusch SM, Zheng B, Jiroutek MR, Carter CA. Gender disparities in patient education provided during patient visits with a diagnosis of coronary heart disease. Women's Health. 2019;15:1745506519845591.

13. Bordoni L, Fedeli D, Piangerelli M, Pelikant-Malecka I, Radulska A, Samulak JJ, et al. Gender-related differences in trimethylamine and oxidative blood biomarkers in cardiovascular disease patients. Biomedicines. 2020;8(8):238.

14. Cignarella A, Bolego C, Barton M. Sex and sex steroids as determinants of cardiovascular risk. Steroids. 2024;206:109423.

15. Day AL, Singh JA. Cardiovascular disease risk in older adults and elderly patients with rheumatoid arthritis: what

role can disease-modifying antirheumatic drugs play in cardiovascular risk reduction? Drugs & Aging. 2019;36(6):493-510.

16. Fardoun M, Dehaini H, Shaito A, Mesmar J, El-Yazbi A, Badran A, et al. The hypertensive potential of estrogen: An untold story. Vascular Pharmacology. 2020;124:106600.

17. Jassim AM, Hussain ZK. Estimation of Some Antioxidant Factors in Postmenopausal Iraqi Women with Hypertension. Iraqi Journal of Science. 2024.

18. Tabatabaei-Malazy O, Djalalinia S, Asayesh H, Shakori Y, Esmaeili Abdar M, Mansourian M, et al. Menopause and metabolic syndrome in the Middle East countries; a systematic review and meta-analysis study. Journal of diabetes & metabolic disorders. 2018;17:357-64.

19. Singar S, Suminski R, Arjmandi B, Hooshmand S, Chai S. Body Mass Index Predicts Cardiovascular Risk Factors and Inflammatory Status in Postmenopausal Women. Current Developments in Nutrition. 2021;5:1249.

20. Koo BK, Park S-H, Han K, Moon MK. Cardiovascular outcomes of obesity according to menopausal status: a nationwide population-based study. Endocrinology and Metabolism. 2021;36(5):1029-41.

21. Opoku AA, Abushama M, Konje JC. Obesity and menopause. Best Practice & Research Clinical Obstetrics & Gynaecology. 2023;88:102348.

22. Chen G-C, Arthur R, Iyengar NM, Kamensky V, Xue X, Wassertheil-Smoller S, et al. Association between regional body fat and cardiovascular disease risk among postmenopausal women with normal body mass index. European heart journal. 2019;40(34):2849-55.

23. Calling S, Johansson S-E, Nymberg VM, Sundquist J, Sundquist K. Trajectories of body mass index and risk for coronary heart disease: a 38-year follow-up study. PloS one. 2021;16(10):e0258395.

24. Al AA, Ibrahim HS, Al-Nuaimee AKA-R. Comparison of serum lipid profile between pre and postmenopausal healthy women.

25. Osman AA, Fadlalla AM. Dyslipidemia is the hallmark of the metabolic syndrome in postmenopausal women: Dyslipidemia in postmenopausal women. Annals of Medical Physiology. 2020;4(2):18-21.

26. Jeong HG, Park H. Metabolic disorders in menopause. Metabolites. 2022;12(10):954.

27. Nie G, Yang X, Wang Y, Liang W, Li X, Luo Q, et al. The effects of menopause hormone therapy on lipid profile in postmenopausal women: a systematic review and meta-analysis. Frontiers in pharmacology. 2022;13:850815.

TABLES

Table1: Demographic table and clinical characteristics of the study

Parameter	Control Group (n = 30)	Patient Group (n = 60)	p-value
Age (years)	55.1 ± 3.2	56.4 ± 2.9	0.071
Height (cm)	160.2 ± 5.6	158.9± 6.1	0.218
Weight (kg)	68.7 ± 7.5	74.8 ± 8.2	0.002*
BMI (kg/m²)	26.8 ± 2.9	29.6 ± 3.5	0.001*
Systolic BP (mmHg)	122.4 ± 6.8	144.2±11.7	<0.001*

Diastolic BP (mmHg)	78.3 ± 5.5	91.5 ± 7.4	<0.001*
No Family History (%)	18 (60%)	14 (23.3%)	
Diabetes Mellitus Only (%)	4 (13.3%)	12 (20%)	
Hypertension Only (%)	5 (16.7%)	16 (26.7%)	
Both DM and HTN (%)	3 (10%)	18 (30%)	0.003*†

Table 2: Age Distribution of Study Participants

Age Group (years)	Patients (n = 60)	% (Patients)	Controls (n = 30)	% (Controls)
50–55	31	51.7%	18	60.0%
55–60	29	48.3%	12	40.0%
Total	60	100%	30	100%

Table 3: BMI Among Postmenopausal Women with Chronic Heart Disease

MI Group	No.	Percentage (%)
Normal weight	7	11.67%
Overweight	12	20.00%
Obesity Class I	20	33.33%
Obesity Class II	21	35.00%
Total	60	100%

Table 4: Comparison of lipid profile (cholesterol, TG, HDL, LDL, VLDL) Levels Between Control and Patient Groups

Parameters	Groups		
	Control (n 30)	Patients (n 60)	P. V
	Mean ±S. D	Mean ±S. D	
Cholesterol mg/dl	170.33±26.68	201.20±40.08	<0.05
TG mg/dl	94.86±15.86	154.34±44.90	<0.05
HDL mg/dl	51.33±6.26	40.73±9.28	<0.05
LDL mg/dl	100.02±24.73	129.62±39.95	<0.05
VLDL mg/dl	18.97±3.17	30.86±8.98	<0.05

FIGURES

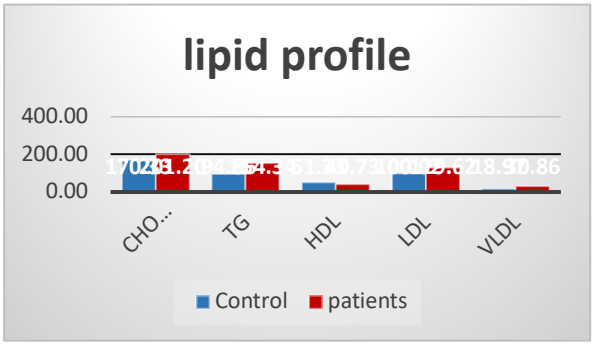

Figure 1: Comparison of Lipid Profile Parameters Between Control and Postmenopausal Women with Chronic Heart Disease