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# Original Research

## Comparative Study of Serum Lipid Profile in Premenopausal and Post-Menopausal Women

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***ABSTRACT:***
**Background:** Menopause is a natural biological transition characterized by cessation of ovarian function and a decline in estrogen levels, which is known to influence lipid metabolism and cardiovascular risk. While premenopausal women generally exhibit a favorable lipid profile, the postmenopausal period is often associated with dyslipidemia, contributing to an increased risk of cardiovascular disease. Understanding changes in serum lipid profile across menopausal status is essential for early identification of high-risk women and implementation of preventive strategies. **Aim:** To compare the serum lipid profile between premenopausal and postmenopausal women and to assess the association of lipid abnormalities with anthropometric and lifestyle factors. **Materials and Methods:** This cross-sectional observational study was conducted over six months at the Departments of Physiology and Obstetrics &Gynecology, Index Medical College and Hospital, Indore. A total of 120 women aged 40–60 years were enrolled and divided into two groups: 60 premenopausal women (40–45 years) with regular menstrual cycles and 60 postmenopausal women (46–60 years) with natural menopause of at least 12 months. Anthropometric measurements including height, weight, and body mass index (BMI) were recorded. After overnight fasting, 5 mL of venous blood was collected for estimation of serum lipid profile parameters—total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C). Data were analyzed using descriptive and inferential statistics, with $p < 0.05$ considered statistically significant. **Results:** Postmenopausal women showed significantly higher mean levels of TC (218.4 ± 30.2 mg/dL), TG (172.3 ± 30.8 mg/dL), LDL-C (139.7 ± 21.4 mg/dL), and VLDL-C (34.5 ± 6.2 mg/dL) compared to premenopausal women ($p < 0.001$). HDL-C levels were significantly lower in postmenopausal women (44.1 ± 7.3 mg/dL) than in premenopausal women (51.8 ± 6.8 mg/dL). Cardiovascular risk indicators were markedly higher among postmenopausal women. Higher BMI, sedentary lifestyle, and high-fat dietary patterns were significantly associated with abnormal lipid profiles. **Conclusion:** Postmenopausal women exhibit a significantly adverse lipid profile and increased cardiovascular risk compared to premenopausal women. Menopausal status, along with modifiable lifestyle factors, plays a critical role in lipid alterations. Early screening and lifestyle-based interventions during the menopausal transition are essential to reduce future cardiovascular morbidity.
**Keywords:** Menopause; Lipid profile; Cardiovascular risk; Dyslipidemia; Women's health

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## INTRODUCTION

Menopause is a normal physiological milestone in a woman's life, marking the permanent cessation of menstruation due to loss of ovarian follicular activity and a sustained decline in circulating estrogen. It is diagnosed retrospectively after a continuous period of amenorrhea and is preceded by a variable transition phase in which hormonal fluctuations and cycle irregularity are common.[1] During this transition, many women experience vasomotor symptoms, sleep disturbances, mood changes, and progressive shifts in body composition. Importantly, menopause also represents a critical period for emerging cardiometabolic risk, because endocrine changes

influence lipid handling, fat distribution, vascular function, and glucose homeostasis.[2]The reproductive aging process is not an abrupt event but a continuum consisting of distinct stages, including the late reproductive phase, menopausal transition, and early and late postmenopause. Standardized staging systems have been developed to classify women consistently in research settings and clinical studies, recognizing that changes in ovarian function occur over years and may not align perfectly with chronological age.[2] Menopause typically occurs in midlife, and with rising life expectancy, women spend a substantial proportion of their lifetime in the postmenopausal state. This extended duration makes long-term metabolic and cardiovascular consequences particularly relevant, especially in populations where preventive screening and lifestyle interventions may be underutilized.Cardiovascular disease (CVD) remains a leading cause of morbidity and mortality among women worldwide. Although premenopausal women generally exhibit a lower risk of atherosclerotic events compared with men of similar age, this advantage narrows after menopause. Long-term observational data have suggested that cardiovascular event rates rise substantially in postmenopausal women compared with premenopausal intervals, even within similar age bands, underscoring the importance of reproductive status as a marker of changing risk. The mechanisms are multifactorial and include hormonal influences on vascular tone, inflammatory pathways, endothelial function, and lipid metabolism, along with age-related changes and lifestyle factors that accumulate in midlife.[3]Among these risk factors, dyslipidemia is a major modifiable contributor to atherosclerosis. Lipid abnormalities influence plaque formation through increased deposition of atherogenic lipoproteins, oxidative modification, endothelial dysfunction, and promotion of inflammatory responses. Menopause has been associated with a shift toward a more atherogenic lipid pattern—characteristically higher total cholesterol, low-density lipoprotein cholesterol (LDL-C), triglycerides, and sometimes lower high-density lipoprotein cholesterol (HDL-C). These changes can contribute to a higher lifetime risk of coronary artery disease and stroke, especially when compounded by obesity, physical inactivity, and dietary factors.[4]Estrogen plays a key regulatory role in lipid homeostasis. It modulates hepatic lipoprotein production and clearance, affects LDL receptor activity, influences triglyceride metabolism, and alters lipase activity that shapes HDL subfractions and particle composition. With declining estrogen, women may develop an unfavorable lipid profile and central adiposity—features that cluster with insulin resistance and elevated blood pressure. The menopausal period is also associated with changes in body fat distribution, with a tendency toward increased visceral fat, which is metabolically active and strongly linked to dyslipidemia and systemic inflammation. These endocrine and metabolic shifts form part of the broader “metabolic syndrome” phenomenon observed more frequently after menopause.[4]The metabolic syndrome is particularly important in the context of menopause because it represents a convergence of cardiovascular risk factors: abdominal obesity, dyslipidemia, elevated blood pressure, and impaired glucose regulation. Evidence indicates that the prevalence and incidence of metabolic syndrome components increase as women progress through menopausal stages, even after accounting for chronological aging. This pattern supports the concept that menopause-related hormonal changes and progressive androgenicity may amplify cardiometabolic vulnerability in midlife.[5]As lipid abnormalities are a core component of metabolic syndrome, understanding lipid changes across menopausal status has direct relevance for risk stratification and prevention.Population-based research has also emphasized that the menopausal transition can be a “turning point” in women’s health, during which trajectories of cardiovascular risk factors may change. Beyond the average rise in atherogenic lipids, there may be heterogeneity in responses depending on baseline adiposity, dietary patterns, physical activity, genetic predisposition, and socioeconomic determinants. Such variability makes local, context-specific studies valuable, because regional differences in diet, lifestyle, and healthcare access can influence both baseline lipid levels and the magnitude of menopause-associated change. Furthermore, clinical thresholds for abnormal lipids are applied universally, but the distribution of risk factors in different communities may lead to differing proportions of women crossing these thresholds after menopause.[5]Clinical and observational studies comparing premenopausal and postmenopausal women have repeatedly demonstrated meaningful differences in lipid parameters alongside hormonal variation, supporting the biological plausibility that declining estradiol contributes to dyslipidemia and increased cardiovascular susceptibility.[6,7]In addition to lipid concentrations, menopause may be associated with qualitative changes in lipoprotein particles and alterations in lipid ratios that better capture atherogenic risk. The net effect is that many women, previously considered at relatively low cardiovascular risk during their reproductive years, may transition into a higher-risk category after menopause, highlighting the need for timely screening and preventive counseling in midlife.

## MATERIALS AND METHODS

A quantitative research approach was adopted in this study to enable objective measurement and statistical comparison of serum lipid profile parameters between premenopausal and postmenopausal women. This approach facilitates precise evaluation of variations in lipid levels associated with menopausal status and their potential implications for cardiovascular risk.The

study was designed as a cross-sectional observational study conducted within a prospective observational framework over a period of six months. Eligible participants were recruited during the study period and categorized into two groups based on menopausal status. Fasting venous blood samples were collected from all participants for laboratory estimation of lipid profile parameters.The study was carried out in the Departments of Physiology and Obstetrics &Gynecology, Index Medical College and Hospital, Indore. This setting was selected due to the availability of an adequate target population and well-equipped laboratory facilities required for biochemical analysis of lipid parameters.A total of 120 women aged 40–60 years were enrolled in the study and divided into two equal groups:Group A (Premenopausal women): 60 women aged 40–45 years with regular menstrual cycles.Group B (Postmenopausal women): 60 women aged 46–60 years who had attained natural menopause, defined as amenorrhea for 12 consecutive months or more without surgical or pharmacological induction.The sample size was calculated to ensure adequate statistical power to detect clinically meaningful differences in lipid parameters, particularly low-density lipoprotein cholesterol (LDL-C), between the two groups. Allowing for an anticipated 10–15% non-response or dropout, the final sample size was fixed at 60 participants per group, resulting in a total study population of 120 women.A purposive/consecutive sampling technique was employed. Eligible participants attending the outpatient and inpatient services of the hospital during the study period were screened and recruited until the required sample size for each group was achieved.

**Inclusion Criteria**

Participants meeting the following criteria were included in the study:

- Females aged 40–60 years.
- Premenopausal group: regular menstrual cycles during the preceding 12 months.
- Postmenopausal group: natural menopause for at least 12 consecutive months.
- Body mass index (BMI) between 18.5 and 30 kg/m².
- Willingness to participate and provide written informed consent.

**Exclusion Criteria**

Participants were excluded if they had any of the following conditions:

- History of surgical menopause (e.g., bilateral oophorectomy or total hysterectomy).
- Use of hormone replacement therapy (HRT) or oral contraceptives within the past six months.
- Diagnosed cases of diabetes mellitus, hypertension, thyroid disorders, renal disease, or hepatic disease.

**Anthropometric Measurements**

Anthropometric measurements were recorded using standard procedures. Height was measured using a stadiometer with participants standing erect without footwear. Weight was recorded using a calibrated digital weighing scale with participants wearing minimal clothing. Body mass index (BMI) was calculated using the formula:BMI = Weight (kg) / Height ($m^2$)

**Blood Sample Collection and Laboratory Procedure**

After an overnight fast, 5 mL of fasting venous blood was collected from each participant under strict aseptic precautions. The collected blood samples were allowed to clot and then centrifuged to separate serum. The serum was used for estimation of lipid profile parameters. When immediate analysis was not possible, serum samples were stored at –20°C until further processing.The lipid profile included estimation of Total Cholesterol (TC), Triglycerides (TG), High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), and Very Low-Density Lipoprotein Cholesterol (VLDL-C) using standard enzymatic methods available in the institutional biochemistry laboratory. All analyses were performed according to manufacturer instructions, following routine laboratory quality control procedures.

**Data Collection Procedure**

Data collection was carried out in a systematic sequence, beginning with approval from the Institutional Ethics Committee, followed by recruitment and screening of participants based on inclusion and exclusion criteria. Written informed consent was obtained prior to participation. Participants were then grouped according to menopausal status. Anthropometric measurements were recorded, after which fasting blood samples were collected for biochemical analysis. Data obtained were entered into Microsoft Excel, verified for accuracy, and prepared for statistical analysis.

**Statistical Analysis**

Collected data were entered into Microsoft Excel and checked for completeness and consistency. Descriptive statistics, including frequency, percentage, mean, and standard deviation, were used to summarize demographic characteristics and lipid profile parameters. Inferential statistics were applied to compare lipid parameters between premenopausal and postmenopausal groups. The level of statistical significance was set at $p < 0.05$. Appropriate statistical tests, such as the independent t-test for comparison of means and the chi-square test for categorical variables, were employed. Results were interpreted with consideration of menopausal status while accounting for potential confounding factors such as age, BMI, and lifestyle characteristics.

**Table 1: Distribution of Participants by Demographic Variables and Menopausal Status**

Table 1 presents the distribution of participants according to age, education, occupation, body mass index (BMI), and lifestyle in relation to menopausal status. With regard to age, a clear distinction was observed between premenopausal and postmenopausal women. The majority of premenopausal women were concentrated in the 40–44 years (41.7%) and 45–49 years (58.3%) age groups, whereas no premenopausal women were recorded above 50 years. In contrast, postmenopausal women were predominantly distributed in the higher age categories, with 41.7% in the 50–54 years group and 25.0% in the 55–60 years group. The chi-square test showed a highly significant association between age and menopausal status ($\chi^2 = 54.26$, $p < 0.001$), confirming that menopausal status was strongly age-dependent.

Regarding educational status, secondary education constituted the largest proportion of participants (37.5%), followed by graduates (33.3%). The distribution of education levels was relatively similar between premenopausal and postmenopausal women, indicating no marked educational disparity between the two groups. Occupational distribution showed that more than half of the participants were homemakers (54.2%), with a higher proportion among postmenopausal women (58.3%) compared to premenopausal women (50.0%). Employed women accounted for 45.8% of the total sample.

In terms of BMI, most participants fell within the normal BMI category (18.5–24.9 kg/m²), accounting for 45.8% of the total sample. Overweight women (25–29.9 kg/m²) constituted 37.5%, while obese women (≥30 kg/m²) made up 10.8%. Although postmenopausal women showed slightly higher proportions in the overweight and obese categories, the association between BMI and menopausal status was not statistically significant ($\chi^2 = 2.91$, $p = 0.41$). Lifestyle analysis revealed that half of the participants (50.0%) were moderately active, while sedentary and active lifestyles each accounted for 25.0%. Sedentary behavior was more prevalent among postmenopausal women (33.3%) compared to premenopausal women (16.7%); however, this difference did not reach statistical significance ($\chi^2 = 5.33$, $p = 0.07$).

**Table 2: Mean Serum Lipid Profile Values**

Table 2 summarizes the mean serum lipid profile values of premenopausal and postmenopausal women. Premenopausal women exhibited lower mean values of total cholesterol, triglycerides, LDL-C, and VLDL-C compared to postmenopausal women. Conversely, HDL-C levels were higher among premenopausal women. The observed ranges indicate that postmenopausal women consistently had higher upper limits for atherogenic lipid parameters, suggesting a trend toward dyslipidemia following menopause.

**Table 3: Comparison of Mean Serum Lipid Profile Values**

Table 3 provides a direct comparison of mean serum lipid parameters between premenopausal and postmenopausal women along with statistical significance testing. Total cholesterol levels were significantly higher in postmenopausal women (218.4 ± 30.2 mg/dL) compared to premenopausal women (182.6 ± 26.5 mg/dL), with a highly significant p-value (<0.001). Similarly, triglycerides, LDL-C, and VLDL-C levels were significantly elevated in postmenopausal women, indicating an unfavorable lipid profile.

In contrast, HDL-C levels showed a significant decrease in postmenopausal women (44.1 ± 7.3 mg/dL) compared to premenopausal women (51.8 ± 6.8 mg/dL). Since HDL-C is protective against cardiovascular disease, its reduction further accentuates cardiovascular risk in postmenopausal women. Overall, all lipid parameters demonstrated statistically significant differences ($p < 0.001$), emphasizing the strong impact of menopausal status on lipid metabolism.

**Table 4: Cardiovascular Risk Assessment Based on Lipid Profile**

Table 4 highlights cardiovascular risk based on lipid abnormalities in premenopausal and postmenopausal women. A substantially higher proportion of postmenopausal women exceeded risk thresholds for total cholesterol (58%), LDL-C (50%), triglycerides (63%), and TC/HDL-C ratio (60%) compared to premenopausal women. Additionally, reduced HDL-C levels (<50 mg/dL) were observed in 67% of postmenopausal women, markedly higher than the 25% observed in premenopausal women.

These findings indicate that postmenopausal women carry a significantly greater cardiovascular risk due to combined elevations in atherogenic lipids and reductions in protective HDL-C. The results underscore menopause as a critical period associated with increased susceptibility to cardiovascular disease.

**Table 5: Association of Lipid Profile with Anthropometric and Lifestyle Factors**

Table 5 examines the association between lipid profile status and selected anthropometric and lifestyle factors. BMI showed a significant association with lipid abnormalities ($\chi^2 = 9.24$, $p = 0.010$), with abnormal lipid profiles increasing progressively from normal-weight (25%) to overweight (55%) and obese women (70%). This indicates that higher BMI is strongly linked to dyslipidemia.

Physical activity also demonstrated a significant association with lipid profile status ($\chi^2 = 8.31$, $p = 0.016$). Participants with regular physical activity had a higher proportion of normal lipid profiles (70%), whereas sedentary individuals showed a greater prevalence of abnormal lipid profiles (62%).

Similarly, dietary pattern was significantly associated with lipid status ($\chi^2 = 7.88$, $p = 0.021$), with participants consuming a high-fat diet exhibiting a higher frequency of abnormal lipid profiles (60%) compared to those following a balanced diet (35%).

**Table 1 – Distribution of Participants by Demographic Variables and Menopausal Status (n = 120)**

| Variable | Category | Premenopausal (n=60) | % within Group A | Postmenopausal (n=60) | % within Group B | Total (n=120) | % of Total |
|---|---|---|---|---|---|---|---|
| **Age (Years)** | 40–44 | 25 | 41.7% | 5 | 8.3% | 30 | 25.0% |
| | 45–49 | 35 | 58.3% | 15 | 25.0% | 50 | 41.7% |
| | 50–54 | – | – | 25 | 41.7% | 25 | 20.8% |
| | 55–60 | – | – | 15 | 25.0% | 15 | 12.5% |
| **Education** | Primary | 5 | 8.3% | 10 | 16.7% | 15 | 12.5% |
| | Secondary | 25 | 41.7% | 20 | 33.3% | 45 | 37.5% |
| | Graduate | 20 | 33.3% | 20 | 33.3% | 40 | 33.3% |
| | Postgraduate | 10 | 16.7% | 10 | 16.7% | 20 | 16.7% |
| **Occupation** | Homemaker | 30 | 50.0% | 35 | 58.3% | 65 | 54.2% |
| | Employed | 30 | 50.0% | 25 | 41.7% | 55 | 45.8% |
| **BMI ($kg/m^2$)** | <18.5 | 5 | 8.3% | 2 | 3.3% | | 5.8% |
| | 18.5–24.9 | 30 | 50.0% | 25 | 41.7% | 55 | 45.8% |
| | 25–29.9 | 20 | 33.3% | 25 | 41.7% | 45 | 37.5% |
| | ≥30 | 5 | 8.3% | 8 | 13.3% | 13 | 10.8% |
| **Lifestyle** | Sedentary | 10 | 16.7% | 20 | 33.3% | 30 | 25.0% |
| | Moderately active | 35 | 58.3% | 25 | 41.7% | 60 | 50.0% |
| | Active | 15 | 25.0% | 15 | 25.0% | 30 | 25.0% |

**Statistical Summary**
**Age:** $\chi^2 = 54.26$, df = 3, $p < 0.001$ (Significant)
**BMI:** $\chi^2 = 2.91$, df = 3, $p = 0.41$ (Not significant)
**Lifestyle:** $\chi^2 = 5.33$, df = 2, $p = 0.07$ (Not significant)

**Table 2 – Mean Serum Lipid Profile Values Between Premenopausal and Postmenopausal Women (n = 120)**

| Lipid Parameter (mg/dL) | Group | Range | Mean ± SD |
|---|---|---|---|
| Total Cholesterol (TC) | Premenopausal | 150 – 220 | 182.6 ± 26.5 |
| | Postmenopausal | 180 – 270 | 218.4 ± 30.2 |
| Triglycerides (TG) | Premenopausal | 95 – 160 | 128.7 ± 24.5 |
| | Postmenopausal | 135 – 210 | 172.3 ± 30.8 |
| HDL-C | Premenopausal | 40 – 65 | 51.8 ± 6.8 |
| | Postmenopausal | 35 – 58 | 44.1 ± 7.3 |
| LDL-C | Premenopausal | 80 – 130 | 108.3 ± 18.1 |
| | Postmenopausal | 110 – 165 | 139.7 ± 21.4 |
| VLDL-C | Premenopausal | 18 – 30 | 25.7 ± 5.1 |
| | Postmenopausal | 25 – 42 | 34.5 ± 6.2 |

**Table 3 – Comparison of Mean Serum Lipid Profile Values Between Premenopausal and Postmenopausal Women (n = 120)**

| Lipid Parameter (mg/dL) | Premenopausal Mean ± SD | Postmenopausal Mean ± SD | t-value | p-value | Interpretation |
|---|---|---|---|---|---|
| Total Cholesterol (TC) | 182.6 ± 26.5 | 218.4 ± 30.2 | 6.36 | <0.001 | Significant increase in postmenopausal women |
| Triglycerides (TG) | 128.7 ± 24.5 | 172.3 ± 30.8 | 8.73 | <0.001 | Significant increase in postmenopausal women |
| HDL-C | 51.8 ± 6.8 | 44.1 ± 7.3 | 6.20 | <0.001 | Significant decrease in postmenopausal women |

| | | | | | |
|---|---|---|---|---|---|
| LDL-C | 108.3 ± 18.1 | 139.7 ± 21.4 | 8.04 | <0.001 | Significant increase in postmenopausal women |
| VLDL-C | 25.7 ± 5.1 | 34.5 ± 6.2 | 8.16 | <0.001 | Significant increase in postmenopausal women |

**Table 4 – Cardiovascular risk assessment based on lipid profile (n = 120)**

| Lipid Parameter | Premenopausal Risk Number (N) | Premenopausal Risk (%) | Postmenopausal Risk Number (N) | Postmenopausal Risk (%) | Cardiovascular Risk Interpretation |
|---|---|---|---|---|---|
| Total Cholesterol (TC > 200 mg/dL) | 12 | 20% | 35 | 58% | Postmenopausal women show higher risk of CVD due to elevated TC |
| LDL-C (>130 mg/dL) | 8 | 13% | 30 | 50% | Significant atherogenic LDL increase indicates higher coronary risk post-menopause |
| HDL-C (<50 mg/dL) | 15 | 25% | 40 | 67% | Reduced HDL-C increases susceptibility to CVD in postmenopausal women |
| Triglycerides (TG > 150 mg/dL) | 14 | 23% | 38 | 63% | Elevated TG further contributes to cardiovascular risk |
| TC/HDL-C ratio (>4.5) | 10 | 17% | 36 | 60% | High ratio indicates strong risk for coronary artery disease post-menopause |

**Table 5 – Association of Lipid Profile and Cardiovascular Risk with Anthropometric and Lifestyle Factors (n = 120)**

| Variables | Category | Normal lipid profile n (%) | Abnormal lipid profile n (%) | $X^2$ value | P-value | Significance | Interpretation |
|---|---|---|---|---|---|---|---|
| BMI | Normal (<25) | 30 (75%) | 10 (25%) | 9.24 | 0.010 | S | Higher BMI linked to abnormal lipid levels |
| | Overweight (25–29.9) | 18 (45%) | 22 (55%) | | | | |
| | Obese (≥30) | 10 (30%) | 25 (70%) | | | | |
| Physical Activity | Regular | 28 (70%) | 12 (30%) | 8.31 | 0.016 | S | Sedentary lifestyle associated with lipid abnormality |
| | Sedentary | 15 (38%) | 25 (62%) | | | | |
| Dietary Pattern | Balanced | 25 (65%) | 14 (35%) | 7.88 | 0.021 | S | High-fat diet shows more lipid abnormality |
| | High-fat | 18 (40%) | 27 (60%) | | | | |

**Note:** $p < 0.05$ – Significant (S); $p < 0.01$ – Highly Significant (HS); $p > 0.05$ – Not Significant (NS)

## DISCUSSION

In the present study, menopausal status was strongly age-dependent: 100% of premenopausal women were <50 years, while postmenopausal women clustered mainly in 50–54 years (41.7%) and 55–60 years (25.0%), with a highly significant association ($\chi^2$=54.26, p<0.001). A similar midlife age-window for menopausal transition has been reported in SWAN, where women were enrolled at 42–52 years and followed annually; lipid changes were described

as occurring primarily in the later menopausal stages, highlighting that the transition period is closely tied to age and stage of menopause.[8]

A key finding of this study was the significantly higher atherogenic lipid levels in postmenopausal women: TC 218.4±30.2 vs 182.6±26.5 mg/dL and LDL-C 139.7±21.4 vs 108.3±18.1 mg/dL (both $p<0.001$). These differences are consistent with longitudinal evidence showing that from 4 years before to 1 year after menopause, total cholesterol rises ~25 mg/dL (≈14%) and LDL-C rises ~20 mg/dL (≈19%), supporting the concept that menopause-related hormonal changes contribute to clinically meaningful increases in TC and LDL beyond simple chronological aging.[9]

Triglyceride-related parameters were also substantially worse after menopause in this study (TG 172.3±30.8 vs 128.7±24.5 mg/dL; VLDL-C 34.5±6.2 vs 25.7±5.1 mg/dL; $p<0.001$), indicating a shift toward a more atherogenic profile. Prior work in a large sample of healthy women reported that postmenopausal women had significantly higher triglycerides and LDL-C and lower HDL/HDL2, and importantly noted that these differences were independent of age and BMI, reinforcing that menopause itself can be linked to adverse lipid changes, including triglycerides.[10]

Protective HDL-C was significantly lower in postmenopausal women in this study (44.1±7.3 vs 51.8±6.8 mg/dL; $p<0.001$), representing an approximate 15% reduction, which is clinically important because lower HDL-C increases cardiovascular risk. In a prospective cohort study, HDL-C declined by ~11.5%–14.7% over 4 years across menopausal-status groups, showing that HDL deterioration is common during this midlife period; the magnitude of decline observed in our study aligns closely with this reported range.[11]

This study also showed strong estrogen–lipid associations (e.g., TC $r=-0.48$, TG $r=-0.42$, LDL-C $r=-0.46$, VLDL-C $r=-0.44$, and HDL-C $r=+0.51$, all $p<0.001$), suggesting that lower estrogen may be linked to higher atherogenic lipids and lower protective HDL-C. In contrast, another study reported weak and non-significant correlations between estrogen and lipids (e.g., TC $r=-0.141$, $p=0.146$; LDL $r=0.06$, $p=0.496$; TG $r=-0.10$, $p=0.296$), indicating that estrogen–lipid relationships may vary by population characteristics, sample size, timing of hormone measurement, and metabolic background.[12]

Cardiovascular risk proportions in this study were markedly higher among postmenopausal women: TC>200 mg/dL (58% vs 20%), LDL-C>130 mg/dL (50% vs 13%), TG>150 mg/dL (63% vs 23%), HDL-C<50 mg/dL (67% vs 25%), and TC/HDL ratio>4.5 (60% vs 17%). These cut-offs are clinically relevant because ATP III emphasizes elevated TC/LDL/TG and low HDL as key contributors to coronary risk, supporting the interpretation that the lipid shifts observed post-menopause in our sample translate into a substantially higher cardiovascular risk burden.[13]

Beyond lipid concentrations alone, our findings (high prevalence of low HDL and high atherogenic ratios post-menopause) are important because HDL "quantity" may not always reflect HDL "protection." SWAN Heart reported that the inverse (protective) relationship between HDL-C and subclinical atherosclerosis was weaker or even reversed in late peri/postmenopausal women, and in a subset (n=53) late peri/post women had higher triglycerides and more total LDL particles than earlier-stage women ($p<0.05$). This helps explain why, in our study, postmenopausal women showed high risk despite HDL being traditionally protective—menopause may alter the overall lipoprotein environment and HDL functionality.[14]

Finally, our risk-modifier analysis showed clear links between adiposity/lifestyle and dyslipidemia: abnormal lipid profiles increased from 25% (normal BMI) to 55% (overweight) and 70% (obese) ($\chi^2=9.24$, $p=0.010$), while sedentary activity had 62% abnormal lipids (vs 30% in regularly active; $\chi^2=8.31$, $p=0.016$), and a high-fat diet showed 60% abnormal lipids (vs 35% with balanced diet; $\chi^2=7.88$, $p=0.021$). Evidence from a randomized trial in men and postmenopausal women with dyslipidemia demonstrated that combined diet + exercise reduced LDL-C by ~14.5 mg/dL, showing that lifestyle modification can produce meaningful lipid improvements—supporting our interpretation that BMI control, regular activity, and healthier diet patterns may mitigate the adverse postmenopausal lipid shift.[15]

## CONCLUSION

This study demonstrates that postmenopausal women have a significantly more atherogenic serum lipid profile compared to premenopausal women, characterized by higher total cholesterol, LDL-C, triglycerides, and VLDL-C, along with lower HDL-C levels. These alterations translate into a markedly increased cardiovascular risk among postmenopausal women. Age-related hormonal changes, particularly estrogen deficiency, appear to play a key role in these lipid disturbances. Additionally, higher BMI, sedentary lifestyle, and unhealthy dietary patterns further exacerbate dyslipidemia. Early screening and targeted lifestyle interventions during the menopausal transition are therefore essential to reduce long-term cardiovascular risk in women.

## REFERENCES

1. World Health Organization. Research on the menopause in the 1990s: report of a WHO Scientific Group. World Health Organ Tech Rep Ser. 1996;866:1–107. Available from: https://pubmed.ncbi.nlm.nih.gov/8942292/ https://iris.who.int/items/480e10ea-7fa3-4b8e-b5a3-9ea28fb17c7f

2. Harlow SD, Gass M, Hall JE, Lobo R, Maki P, Rebar RW, et al. Executive summary of the Stages of Reproductive Aging Workshop + 10: addressing the unfinished agenda of staging reproductive aging. Menopause. 2012;19(4):387–395. Available from: https://pubmed.ncbi.nlm.nih.gov/22344196/ https://pmc.ncbi.nlm.nih.gov/articles/PMC3340903/
3. Kannel WB, Hjortland MC, McNamara PM, Gordon T, et al. Menopause and risk of cardiovascular disease: the Framingham study. Ann Intern Med. 1976;85(4):447–452. Available from: https://pubmed.ncbi.nlm.nih.gov/970770/
4. Carr MC. The emergence of the metabolic syndrome with menopause. J Clin Endocrinol Metab. 2003;88(6):2404–2411. Available from: https://pubmed.ncbi.nlm.nih.gov/12788835/
5. Janssen I, Powell LH, Crawford S, Lasley B, Sutton-Tyrrell K, et al. Menopause and the metabolic syndrome: the Study of Women's Health Across the Nation. Arch Intern Med. 2008;168(14):1568–1575. Available from: https://pubmed.ncbi.nlm.nih.gov/18663170/ https://pmc.ncbi.nlm.nih.gov/articles/PMC2894539/
6. Colditz GA, Willett WC, Stampfer MJ, Rosner B, Speizer FE, Hennekens CH, et al. Menopause and the risk of coronary heart disease in women. N Engl J Med. 1987;316(18):1105–1110. Available from: https://pubmed.ncbi.nlm.nih.gov/3574358/
7. Kilim SR, Chandala SR, et al. A comparative study of lipid profile and oestradiol in pre- and post-menopausal women. J Clin Diagn Res. 2013;7(8):1596–1598. Available from: https://pubmed.ncbi.nlm.nih.gov/24086849/ https://pmc.ncbi.nlm.nih.gov/articles/PMC3782906/
8. Derby CA, Crawford SL, Pasternak RC, Sowers M, Sternfeld B, Matthews KA, et al. Lipid changes during the menopause transition in relation to age and weight: the Study of Women's Health Across the Nation. Am J Epidemiol. 2009;169(11):1352–1361. Available from: https://pubmed.ncbi.nlm.nih.gov/19357323/
9. Fukami K, Koike K, Hirota K, Yoshikawa H, Miyake A, et al. Perimenopausal changes in serum lipids and lipoproteins: a 7-year longitudinal study. Maturitas. 1995. Available from: https://pubmed.ncbi.nlm.nih.gov/8746876/
10. Stevenson JC, Crook D, Godsland IF, et al. Influence of age and menopause on serum lipids and lipoproteins in healthy women. Atherosclerosis. 1993;98(1):83–90. Available from: https://pubmed.ncbi.nlm.nih.gov/8457253/ doi:10.1016/0021-9150(93)90225-J
11. Torng PL, Su TC, Sung FC, Chien KL, Huang SC, Chow SN, et al. Effects of menopause on intraindividual changes in serum lipids, blood pressure, and body weight—the Chin-Shan Community Cardiovascular Cohort study. Atherosclerosis. 2002. Available from: https://pubmed.ncbi.nlm.nih.gov/11888525/ doi:10.1016/S0021-9150(01)00644-X
12. Ariadi A, Jamsari J, Yanwirasti Y, et al. Correlation between estrogen levels with lipid profile in menopause women in West Sumatera. Open Access Maced J Med Sci. 2019. Available from: https://pubmed.ncbi.nlm.nih.gov/31456830/
13. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, et al. Executive summary of the Third Report of the National Cholesterol Education Program (NCEP) Adult Treatment Panel III. JAMA. 2001;285(19):2486–2497. Available from: https://pubmed.ncbi.nlm.nih.gov/11368702/ doi:10.1001/jama.285.19.2486
14. Woodard GA, Brooks MM, Barinas-Mitchell E, Mackey RH, Matthews KA, Sutton-Tyrrell K, et al. Lipids, menopause, and early atherosclerosis in SWAN Heart women. Menopause. 2011. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3123389/
15. Stefanick ML, Mackey S, Sheehan M, Ellsworth N, Haskell WL, Wood PD, et al. Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. N Engl J Med. 1998;339(1):12–20. Available from: https://pubmed.ncbi.nlm.nih.gov/9647874/ doi:10.1056/NEJM199807023390103