Lipid Profile in Post Menopausal Women Assessing Health Care in Madonna University Teaching Hospital (MUTH)

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Abstract

A significant worldwide health issue, postmenopause is associated with cardiovascular problems and may be impacted by lipid imbalance (dyslipidaemia). The purpose of this study was to compare the serum levels of postmenopausal subjects to premenopausal controls for total cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDL-C), and low density lipoprotein cholesterol (LDL-C). At Madonna University Teaching Hospital (MUTH), a cross-sectional analytical study was carried out with 142 women, 77 of whom were postmenopausal (aged 50–80) and 65 of whom were premenopausal (aged 20–35). The Spectrophotometry Technique was used to measure the serum levels of TC, TG, HDL-C, and LDL-C. Version 25.0 of the IBM Statistical Package for Social Science (IBM-SPSS) was used to analyse the data. According to the data, postmenopausal women's serum TC (mg/dl) was significantly higher (156.75±4.33) than that of premenopausal women (128.98±5.21) at p=0.00. At p=0.00, postmenopausal women had a substantially higher serum TG (mg/dl) (166.00±2.83) than premenopausal women (143.82±7.88). At p=0.000, postmenopausal women's serum HDL-C levels were considerably lower (18.57±3.15) than those of premenopausal women (47.83±3.45). Additionally, it was discovered that postmenopausal women had a substantially higher serum LDL-C level (107.49±2.66) than premenopausal women (51.16±3.48) at p=0.000. Analysis of postmenopausal women by age group (50-59; 60-69; 70-80) showed no discernible differences in TC, TG, HDL-C, and LDL-C levels; TC: p = 0.075; TG: p = 0.614; HDL-C: p = 0.969; and LDL-C: p = 0.645. In conclusion, postmenopausal women evaluating their health care at Madonna University Teaching Hospital (MUTH) had higher serum levels of TC, TG, and LDL-C. This shows that these women are at risk for cardiovascular disease (CVD) because HDL-C has a cardioprotective effect and is decreasing.

Keywords

Lipid Profile, Post Menopausal, Women, Health Care, Madonna University

1. Introduction

A non-pathological condition of persistent amenorrhoea after menopause is a global health concern. In the UK, menopause typically occurs at age 52. Alongside it, oestrogen levels decline, perhaps leading to associated symptoms and the requirement for Hormone Replacement Therapy (HRT). According to studies, 60–86% of women have symptoms that are sufficiently unpleasant that they seek medical attention [1]. Many people later feel misunderstood and let down that their issues were not taken care of. There are safe, well-researched, government-approved medical treatments that work. Globally, there were a projected 1.02 billion postmenopausal women in 2021, and by 2050, 1.65 billion are expected [2].

High density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), and very low density lipoprotein cholesterol (VLDL-C) are all components of total cholesterol, which is the total quantity of cholesterol in the blood. An increased risk of atherosclerosis and coronary heart disease is linked to elevated TC (NCEP, 2002). The body stores a form of fat called triglycerides (TG), which are used as fuel. Atherosclerosis and coronary heart disease are linked to elevated TG levels, especially in postmenopausal women. Often called "good cholesterol," HDL-C aids in the removal of cholesterol from the circulation. Postmenopausal lower HDL-C levels lessen cardiovascular protection [3]. Because high levels of LDL-C encourage the formation of plaque in arteries, it is regarded as "bad" cholesterol. Women who have gone through menopause frequently have elevated LDL-C levels, which increases their risk of cardiovascular disease [4]. By positively regulating cholesterol levels, oestrogen also contributes to cardiovascular health protection. Women usually have higher levels of HDL-C and lower levels of LDL-C before menopause. On the other hand, oestrogen shortage causes adverse lipid profile alterations following menopause, including an increase in TC, TG, and LDL-C and possibly a decrease in HDL-C [5]. The higher prevalence of hypertension, atherosclerosis, and coronary artery disease in postmenopausal women is mostly caused by these lipid changes. For the early identification and treatment of dyslipidaemia in this population, routine evaluation of lipid profiles becomes crucial.

2. Materials and Method

2.1 Study Area

The study was conducted at Madonna University Teaching Hospital, a tertiary healthcare facility located in Madonna University Nigeria, Elele, Rivers State. The teaching hospital was established in 2002, by Rev. Fr. Prof. E.M.P Edeh. It has 250 bed capacity with tertiary health care facilities equipped with modern diagnostic and therapeutic equipment. Their facility caters to a diverse population of patients from Rivers State and surrounding regions, ensuring a wide spectrum of cases for analysis.

2.2 Study Population

A total number of 142 premenopausal and post menopausal women, aged 20-35 years and 50-80 years respectively, was randomly selected and recruited for the study. The post menopausal women consist of apparently **77** healthy women who have attained the post menopausal age (50-80 years), assessing health care in Madonna University Teaching Hospital, Elele, Rivers State. The premenopausal women consist of apparently **65** healthy women who have attained the pre menopausal age (20-35 years).

2.3 Selection Criteria

2.3.1 Inclusion Criteria

- The post menopausal women used were at the age range of 50-80 years (post menopausal age).
- The premenopausal women used were at the age range of 20-38 years.
- The post menopausal women whom were randomly selected were apparently healthy.
- The post menopausal women do not have any past or present record of cardiovascular diseases.
- The premenopausal women were apparently healthy and were experiencing their monthly menstrual flow regularly.

2.3.2 Exclusion Criteria

- Those undergoing any form of medical treatment using Steroids
- Those that are not healthy
- Those with past or present records of cardiovascular diseases
- Those with irregular menstrual flow

2.4 Sample Size Calculation

Formula; $n = \frac{Z^2 p.(1-p)}{d^2}$ Where,

n= Required Sample Size

Z = Confidence Interval (i.e., 95% = 1.96)

p= Prevalence Rate (10.3%)

d= Margin error = 0.05

$n = \frac{1.96^2 \times 0.103\ (1-0.103)}{0.05^2}$

n= 141.88 ≅142 participants

2.5 Ethical Clearance

Approval was obtained from the Ethical Committee of Madonna University Teaching Hospital (MUTH).

2.6 Informed Consent

Participants were informed about the study's purpose, risks, and benefits, and written informed consent was obtained before sample collection. Confidentiality was maintained throughout the study.

2.7 Reagent

Randox kit Grove for Total Cholesterol (TC), Triglyceride (TG) and High Density Lipoprotein Cholesterol (HDL-C).

2.8 Research Design

The study was a cross sectional study involving post menopausal and pre menopausal women. Five millimeter (5ml) of fasting venous blood was collected from both subjects and was dispensed in a plain container which was allowed to coagulate at room temperature and centrifuged at 3000rpm for 10minute to separate the serum present in the supernatant

from the infranatant. The serum was extracted and stored at -20^0 C and analyzed within 7 days for lipid profile determination.

2.8.1 Sample Collection

For the purpose of this study, blood samples was collected from both premenopausal and post menopausal women assessing health care in Madonna University Teaching Hospital. All specimens were randomly sampled and blood collection was properly collected by venipuncture technique. No dietary restrictions and regulation was recommended for the subjects. All subjects were sampled in a sitting room. The prominent vein was selected and the tourniquet was tied but not too tight. The site of puncture was sterilized thoroughly with a cotton wool soaked with methylated spirit. A sterile hypodermic needle and a syringe was unsealed and fitted well, after checking for absent of any blockage in the needle and syringe by pushing and drawing the syringe position ensuring dryness. The vein was punctured and 5ml of blood was drawn. The time between the application of tourniquet and vein puncture was made as short as possible to prevent venous stasis. The blood sample was emptied into a plain container.

2.8.2 Laboratory Procedures / Analytical Method

The enzymatic end point method was used for the estimation serum total cholesterol, triglyceride, and high density lipoprotein cholesterol

The low Density lipoprotein cholesterol (LDL-C) was estimated according to the Friedwald's formula, LDL-C= Total Cholesterol - Triglycerides - High density lipoprotein

2.5

3. Statistical Analysis/Data Analysis

Data were analyzed using SPSS version 25.0. Descriptive statistics (mean, standard deviation) were used to summarize TC, TG HDL-C and LDL-C levels. Independent t-tests were used to compare post menopausal and premenopausal groups. Statistical significance was set at $p < 0.05$ at 95% confidence interval.

4. Results

Table 1. Lipid Profile Parameters between Premenopausal (Control) and Postmenopausal (Test) women accessing health care in Madonna University Teaching Hospital (MUTH)

Parameter	Premenopausal Mean ± SD	Postmenopausal Mean ± SD	t-Value	p-Value
Total Cholesterol (mg/dL)	128.98 ± 5.21	156.75 ± 4.33	-30.08	0.000
Triglycerides (mg/dL)	143.82 ± 7.88	166.00 ± 2.83	-21.15	0.000
High-Density Lipoprotein (mg/dL)	47.83 ± 3.45	18.57 ± 3.15	49.58	0.000
Low-Density Lipoprotein (mg/dL)	51.16 ± 3.48	107.49 ± 2.66	-97.45	0.000

In the table 1, The results show a statistically significant difference in all lipid profile parameters between premenopausal and postmenopausal women ($p = 0.000$ for all). Total cholesterol levels were markedly higher in postmenopausal women (156.75 ± 4.33 mg/dL) compared to their premenopausal counterparts (128.98 ± 5.21 mg/dL), with a large t-value of -30.08 indicating strong evidence of difference. Similarly, triglycerides increased from 143.82 ± 7.88 mg/dL in the control group to 166.00 ± 2.83 mg/dL in the test group. High-density lipoprotein (HDL), a protective lipid, was significantly lower in postmenopausal women (18.57 ± 3.15 mg/dL) versus premenopausal women (47.83 ± 3.45 mg/dL), showing a dramatic decline with a t-value of 49.58. Conversely, low-density lipoprotein (LDL) nearly doubled from 51.16 ± 3.48 mg/dL in premenopausal women to 107.49 ± 2.66 mg/dL in postmenopausal women. These significant alterations suggest that menopause has a profound impact on lipid metabolism, predisposing postmenopausal women to increased cardiovascular risk.

Table 2. Lipid Profile levels Across Age Groups in Postmenopausal Women

Age Group	TC (mg/dL)	TG (mg/dL)	HDL-C (mg/dL)	LDL-C (mg/dL)
50–59	157.28 ± 3.46	165.81 ± 3.73	18.58 ± 3.56	107.65 ± 2.70
60–69	158.73 ± 3.47	166.24 ± 2.85	18.59 ± 3.30	107.04 ± 2.79
70–80	156.55 ± 3.68	165.37 ± 3.17	18.81 ± 3.68	107.65 ± 2.76
F-value	2.67	0.49	0.03	0.44
p-value	0.075	0.614	0.969	0.645

KEYS:

TC (mg/dL): Total Cholesterol

TG (mg/dL): Triglycerides

HDL-C (mg/dL): High-Density Lipoprotein Cholesterol

LDL-C (mg/dL): Low-Density Lipoprotein Cholesterol

Table 2 presents the lipid profile of postmenopausal women across three age groups (50–59, 60–69, and 70–80 years). Total cholesterol (TC) levels were slightly higher in women aged 60–69 years (158.73 ± 3.47 mg/dL) compared to the 50–59 (157.28 ± 3.46 mg/dL) and 70–80 groups (156.55 ± 3.68 mg/dL), although the difference was not statistically significant ($F = 2.67$, $p = 0.075$). Triglyceride (TG) levels remained relatively stable across all age groups, ranging from 165.37 ± 3.17 mg/dL to 166.24 ± 2.85 mg/dL ($F = 0.49$, $p = 0.614$). HDL-C values were nearly identical across age groups, with the highest in the 70–80 group (18.81 ± 3.68 mg/dL), yet the variation was not significant ($F = 0.03$, $p = 0.969$). Similarly, LDL-C levels showed minor fluctuations, with values between 107.04 ± 2.79 mg/dL and 107.65 ± 2.70 mg/dL ($F = 0.44$, $p = 0.645$). Overall, the data indicate no statistically significant differences in lipid parameters across the postmenopausal age groups, suggesting that age may not have a strong impact on lipid levels during this stage of life.

Table 3. Lipid Profile levels Across Age Groups in Premenopausal Women

Age Group	TC (mg/dL)	TG (mg/dL)	HDL-C (mg/dL)	LDL-C (mg/dL)
20–25	128.78 ± 5.55	145.31 ± 9.07	47.32 ± 3.61	51.23 ± 3.91
26–30	129.12 ± 5.58	143.54 ± 7.92	48.34 ± 3.45	50.59 ± 3.59
31–35	128.31 ± 5.62	145.52 ± 9.67	47.75 ± 4.02	50.75 ± 3.53
F-value	0.12	0.26	0.31	0.11
p-value	0.888	0.771	0.735	0.897

KEYS:

TC (mg/dL): Total Cholesterol

TG (mg/dL): Triglycerides

HDL-C (mg/dL): High-Density Lipoprotein Cholesterol

LDL-C (mg/dL): Low-Density Lipoprotein Cholesterol

Table 3 shows the lipid profile distribution among premenopausal women aged 20–35 years, categorized into three age brackets: 20–25, 26–30, and 31–35 years. Total cholesterol (TC) levels were consistently low and similar across the groups, with the highest seen in the 26–30 age group (129.12 ± 5.58 mg/dL) and the lowest in the 31–35 group (128.31 ± 5.62 mg/dL), with no statistical significance ($F = 0.12$, $p = 0.888$). Triglyceride (TG) levels ranged narrowly from 143.54 ± 7.92 mg/dL to 145.52 ± 9.67 mg/dL, and this difference was also not significant ($F = 0.26$, $p = 0.771$). HDL-C levels, which are protective, were fairly high in all groups, peaking at 48.34 ± 3.45 mg/dL in the 26–30 age group, yet with no significant variation ($F = 0.31$, $p = 0.735$). LDL-C values were similarly stable, ranging from 50.59 ± 3.59 mg/dL to 51.23 ± 3.91 mg/dL ($F = 0.11$, $p = 0.897$). These findings suggest a steady lipid profile across premenopausal age groups, indicating that age within this range has little influence on lipid metabolism in healthy women.

5. Discussion

The results of this study show that premenopausal and postmenopausal women who visited Madonna University Teaching Hospital had statistically significant variances in lipid profile parameters. Following menopause, each lipid marker displayed notable changes that were in line with recognised metabolic changes that occur during this physiological transition.

Table 1 shows that postmenopausal women had significantly higher levels of total cholesterol than their premenopausal counterparts. This is consistent with other research showing that lower oestrogen levels after menopause lead to higher TC because they usually increase hepatic LDL receptor function and cholesterol clearance [6,7]. A lack of oestrogen promotes cholesterol buildup and causes disruption of lipid metabolism. Similar findings were reported by [8], who found that postmenopausal Nigerian women had greater TC levels. However, a study conducted in South East Nigeria by [9] found no evidence of a substantial increase in TC after menopause, which could be due to differences in lifestyle, diet, or genetics. Variations in sample sizes and age distributions may also be responsible for the discrepancy in results between research.

The postmenopausal group's triglyceride levels were noticeably greater than those of the premenopausal group. This conclusion confirms previous findings by [10], who observed that postmenopausal women typically have higher triglyceride levels because of decreased lipoprotein lipase activity due to oestrogen shortage and increased hepatic lipogenesis. Hypertriglyceridemia may be made worse by elderly women's increased insulin resistance and lack of physical activity. However, a study conducted in a separate Nigerian cohort by [4] found just a slight, non-significant rise, indicating that regional dietary practices can mitigate the effects of menopause on lipid metabolism.

Compared to premenopausal women, postmenopausal women had considerably decreased levels of HDL-C, which is renowned for its beneficial cardiovascular role. This sharp drop is consistent with research by [11,12], who linked the decline to the loss of estrogen's up-regulatory influence on HDL production and apolipoprotein A1. Postmenopausal women's lower HDL indicates a higher atherogenic risk, which may be a factor in the higher prevalence of cardiovascular disease in this population. Nonetheless, a study conducted in the West [13] discovered that HDL barely changed during menopause, which might have been brought on by lifestyle changes or the early start of hormone replacement treatment. This study's extremely low HDL readings could possibly be the result of undernutrition, restricted physical activity, or regional dietary variables.

In line with the documented correlation between menopause and elevated LDL levels, postmenopausal women's LDL-C levels virtually doubled when compared to premenopausal women. It has been demonstrated that oestrogen increases the expression of the hepatic LDL receptor, which facilitates the removal of LDL from the blood . LDL builds up when oestrogen levels fall, raising the risk of cardiovascular disease. This result is consistent with research by [14]. Nevertheless, a study by [15] did not find such dramatic increases in LDL, which may have been because of differences in BMI or the usage of lipid-lowering drugs in their group. Our findings support the idea that LDL metabolism in Nigerian women is considerably impacted by the menopausal transition.

The lipid profiles that were examined across age groups within the postmenopausal and premenopausal cohorts are displayed in Tables 4.2 and 4.3; the differences found were not statistically significant ($p > 0.05$). This suggests that age groups within each reproductive phase do not substantially alter lipid status, even though menopause itself has a considerable impact on lipid markers. This finding is consistent with research by [16], which indicates that lipid dysregulation in this cohort is mostly caused by hormonal changes rather than ageing.

6. Conclusion

This study provides compelling evidence that post menopause significantly alters lipid metabolism in women, increasing total cholesterol, triglycerides, and LDL-C while reducing HDL-C. These changes were statistically significant and suggest a heightened risk of cardiovascular disease among postmenopausal women. However, age group variations within both premenopausal and postmenopausal categories did not yield significant differences, indicating that hormonal status rather than age is the key determinant of lipid profile changes.

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