
**"A STUDY OF LIPID PROFILE AMONG THE PRE
AND POST-MENOPAUSAL WOMEN AT A
TERTIARY CARE CENTRE - A CROSS
SECTIONAL STUDY"**

BY

REG NO: BG0122008

Dissertation

Submitted to

KAHER, Belagavi, Karnataka

**In partial fulfilment
of the requirements for the degree of**

M.D.

IN

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE

J. N. MEDICAL COLLEGE

BELAGAVI - 590010. KARNATAKA

SEPTEMBER/OCTOBER 2025

**KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,
BELAGAVI**

**Endorsement by the HOD/ Principal/
Head of the Institution**

This is to certify that the dissertation entitled “A STUDY OF LIPID PROFILE AMONG THE PRE AND POST-MENOPAUSAL WOMEN AT A TERTIARY CARE CENTRE - A CROSS SECTIONAL STUDY” is a bonafide research work done by **REG NO: BG0122008**.



Dr. REKHA S PATIL MD

Professor and Head,
Department of General Medicine,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Professor & HOD
Dept. of Medicine
J. N. Medical College, Belagavi

Date : 27/08/2025

Place : Belagavi



Dr.(Mrs.) N.S. MAHANTSHETTI MD

Principal **PRINCIPAL**
Jawaharlal Nehru Medical College
BELAGAVI
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date : 27/08/2025

Place : Belagavi

UNDERTAKING

I, **REG. NO: BG0122008**, hereby declare that the information and the data mentioned in my dissertation “**A STUDY OF LIPID PROFILE AMONG THE PRE AND POST-MENOPAUSAL WOMEN AT A TERTIARY CARE CENTRE - A CROSS SECTIONAL STUDY**” belongs to me and is original. I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorization and the representation of that author’s work as one’s own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another’s words, thoughts, or ideas as one’s own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the University.

Date:28-03-2025

Place: Belagavi



REG NO: BG0122008

PLAGIARISM ACCEPTANCE CERTIFICATE



JAWAHARLAL NEHRU MEDICAL COLLEGE

(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be-University)

(Recognized by National Medical Commission, New Delhi)



Accredited 'A+' Grade by NAAC (3rd Cycle)

Placed in Category 'A' by MoE (GoI)

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

☎ 0831 - 2471350

☎ 0831 - 2470759

🌐 www.jnmc.edu

✉ principal@jnmc.edu

Ref No: MDC/PG/


Date: 24-03-2025

"ACCEPTANCE LETTER"

The softcopy of thesis entitled: "A STUDY OF LIPID PROFILE AMONG THE PRE AND POST-MENOPAUSAL WOMEN AT A TERTIARY CARE CENTRE - A CROSS SECTIONAL STUDY" has been submitted for anti-plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of 10% which is within the acceptable limits of 10% as per the guidelines given by UGC.


Guide.




Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BG0122008
Postgraduate Student,
2022-23 Batch,
Department of General Medicine
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE CERTIFICATE



K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed - to- be- University)

Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)

JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website: <http://www.jnmc.edu>
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 - 2470759

Ref No.MDC/JNMCIEC/ 83

Date: 18/03/2023

To,

PG Student in General Medicine
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled "A STUDY OF LIPID PROFILE AMONG THE PRE-MENOPAUSAL AND POST-MENOPAUSAL WOMEN A CROSS SECTION STUDY IN A TERTIARY CARE CENTER", is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

LIST OF ABBREVIATIONS

1. BMI - Body Mass Index
2. BP - Blood Pressure
3. PR - Pulse Rate
4. RR - Respiratory Rate
5. SBP - Systolic Blood Pressure
6. DBP - Diastolic Blood Pressure
7. TC - Total Cholesterol
8. TGL - Triglycerides
9. LDL - Low-Density Lipoprotein
10. HDL - High-Density Lipoprotein
11. LDL_c - Low-Density Lipoprotein Cholesterol
12. HDL_c - High-Density Lipoprotein Cholesterol
13. AIP - Atherogenic Index of Plasma
14. MS Excel - Microsoft Excel
15. SPSS - Statistical Package for the Social Sciences
16. CI - Confidence Interval

17. ICMR - Indian Council of Medical Research
18. AHA - American Heart Association
19. ACC - American College of Cardiology
20. ADA - American Diabetes Association
21. SD - Standard Deviation

ABSTRACT

Introduction

Cardiovascular disease (CVD) is a leading cause of mortality among women, with lipid profile alterations playing a crucial role in determining cardiovascular risk. Menopause significantly impacts lipid metabolism, increasing susceptibility to dyslipidemia and CVD. This study aims to compare the lipid profiles of pre-menopausal and post-menopausal women, focusing on total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides (TG) to assess cardiovascular risk.

Materials and Methods

A hospital-based, cross-sectional study was conducted at a tertiary care center in Belagavi, India, over 12 months. A total of 158 women (pre-menopausal and post-menopausal) were selected based on inclusion and exclusion criteria. Lipid profiles were assessed using enzymatic methods on a fully automated analyzer after overnight fasting. Statistical analyses, including Student's t-test and Chi-square tests, were performed using SPSS software, with significance set at $p < 0.05$.

Results

The study found that post-menopausal women exhibited significantly higher levels of TC, LDL, and TG, along with lower HDL levels, compared to pre-menopausal women. The atherogenic index of plasma (AIP) was also notably elevated in post-menopausal women, indicating an increased risk of cardiovascular diseases. These lipid alterations suggest that estrogen depletion during menopause contributes to unfavorable metabolic changes, increasing CVD risk.

Conclusion

Post-menopausal women experience significant dyslipidemia, characterized by increased LDL, TG, and decreased HDL levels, predisposing them to CVD. Regular lipid profile screening and early preventive measures, including lifestyle modifications and pharmacological interventions when necessary, are crucial for reducing cardiovascular risk. Further longitudinal studies are needed to establish causal relationships and explore additional metabolic markers for a more comprehensive understanding of CVD risk in post-menopausal women.

TABLE OF CONTENTS

S. NO.	CONTENT	PAGE NO.
1.	INTRODUCTION	1-2
2.	OBJECTIVE	3
3.	REVIEW OF LITERATURE	4-20
4.	METHODOLOGY	21-25
5.	RESULTS	26-40
6.	DISCUSSION	41-55
7.	CONCLUSION	56-57
8.	BIBLIOGRAPHY	58-65
9.	ANNEXURES	66-74

TABLE OF TABLES

S. NO.	TABLES	PAGE NO.
1.	Age distribution of subjects	26
2.	Distribution of subjects according to their mean age	27
3.	Distribution of subjects according to Body mass index (BMI)	28
4.	Distribution of subjects as per their mean BMI	29
5.	Distribution of subjects as per their mean BMI	30
6.	Distribution of subjects according to lab parameters	31
7.	Distribution of subjects according to their mean lab parameters between the pre-menopausal group and the post-menopausal group	32
8.	Distribution of subjects according to atherogenic index of plasma (AIP)	34
9.	Comparison of AIP between the pre-menopausal group and the post-menopausal group among subjects	35
10.	Distribution of subjects according to Total Cholesterol (TC) and atherogenic index of plasma (AIP)	36
11.	Distribution of subjects according to Triglycerides (TGL) and atherogenic index of plasma (AIP)	37
12.	Distribution of subjects according to Low Density Cholesterol (LDL_c) and atherogenic index of plasma (AIP)	38
13.	Distribution of subjects according to High Density Cholesterol (HDL_c) and atherogenic index of plasma (AIP)	39

14.	Comparison of Dyslipidemia between the pre-menopausal group and the post-menopausal group among subjects	40
15.	Baseline characteristics of the study participants	43-44
16.	Lipid profile of the study participants	50
17.	AIP of the study participants	51
18.	Dyslipidemia	54

TABLE OF FIGURES

S. NO.	FIGURE	PAGE NO.
1.	Considerations for Atherosclerotic cardiovascular disease risk among women over their lifespan	14
2.	Pie chart showing age distribution among subjects	26
3.	Line graph showing mean age distribution among subjects	27
4.	Pie chart showing BMI distribution among subjects	28
5.	Line graph showing mean BMI distribution among subjects	29
6.	Line graph showing mean vitals distribution among subjects	30
7.	Line graph showing mean values of lab parameters among subjects	31
8.	Bar diagram showing mean values of lab parameters among the pre-menopausal group and the post-menopausal group	32
9.	Pie chart showing atherogenic index of plasma distribution among subjects	34
10.	Line graph showing mean values of AIP between the pre-menopausal group and the post-menopausal group among subjects	35

INTRODUCTION

Cardiovascular disease remains the leading cause of death among women [1]. Research indicates that nearly one in every two women dies from a heart-related condition—a figure that surpasses the combined death toll from cancer, chronic lung disease, and accidents [2]. Even though there is a 10-year difference in overall mortality between men and women, the annual death rate from cardiovascular disease is comparable for both sexes [3]. Additionally, women experience the same risk factors for heart disease as men, such as high blood pressure and smoking, and imbalances in cholesterol levels—specifically, elevated low-density lipoprotein (LDL-C) or reduced high-density lipoprotein (HDL-C)—are well recognized as modifiable risks for cardiovascular disease in both genders [4, 5].

According to few studies conducted in United States, up to 20% of women have hypercholesterolaemia, which is increased total-cholesterol levels of 240 mg/dL, with a greater proportion requiring treatment.[6] Several randomised controlled studies completed over the last 25 years have found that statin treatment of hypercholesterolaemia lowers cardiovascular events among women who already have cardiovascular disease.[7] While men and the women are similar according to the hazards related to aberrant lipo protein levels, women have some significant biological differences that affect their understanding and management of lipid illnesses.

The goal of our study was to investigate at lipid profile and the occurrence of dyslipidaemia in postmenopausal women, as blood lipid levels have a significant role in risk towards developing cardiovascular disease among adult women. In postmenopausal women, a positive result with LDL-C levels was statistically

significant. Their HDL-C levels were much lower. The computed atherogenic index of plasma (AIP) in postmenopausal women was substantially higher than when compared to that in the premenopausal women.

AIMS AND OBJECTIVES

AIM:

To estimate the lipid profile among the pre-menopausal and post-menopausal women and to estimate the Atherogenic index of plasma (AIP) in assessing the cardiovascular risk.

OBJECTIVES:

1. To estimate the lipid profile in the pre-menopausal and post-menopausal women.
2. To estimate the Atherogenic index of plasma (AIP) in assessing the cardiovascular risk.

REVIEW OF LITERATURE

Dyslipidaemia is a known risk factor for cardiovascular disease (CVD), the leading cause of morbidity and mortality in women worldwide. Dyslipidemia becomes more common as a woman ages, with negative effects occurring around menopause. Menopause, as well as the years preceding the final menstrual period, is characterised by oestrogen fluctuation and, eventually, oestrogen shortage, which has been linked to proatherogenic changes in the lipid profile. Menopause is related with increases in blood total cholesterol, LDL cholesterol, apolipoproteins, and triglycerides, as well as decreases in HDL cholesterol (HDL-C), regardless of age.

Emerging evidence also reveals that functional HDL cardioprotective qualities decline after menopause. Early beginning of menopausal hormone therapy (MHT) improves lipid profiles, but this does not transfer into better CVD outcomes, therefore guidelines do not recommend it for primary or secondary prevention of CVD. Women with CVD-related disorders, such as polycystic ovarian syndrome, premature menopause, early menopause, premature ovarian insufficiency, and familial hypercholesterolaemia, should get special treatment during menopause. Statins remain the basis of dyslipidaemia treatment, but new lipid-lowering medicines are emerging.

The aetiology and the pathophysiology of dyslipidaemia among women

Lipid and lipoprotein primer

LDL-C as a Key Target in Dyslipidemia Management

Global guidelines identify LDL-C as the primary target for therapy in patients with dyslipidemia [8-10]. The recommended targets for LDL-C and non-HDL-C

(calculated as total cholesterol minus HDL-C, serving as an indicator of total atherogenic lipoprotein burden in the blood) are stratified based on an individual's risk level. HDL-C levels have an inverse association with coronary heart disease (CHD) risk in both men and women, meaning that lower HDL-C levels correspond to a higher risk. Although HDL-C is recognized as a significant predictor of CHD, it is not a direct therapeutic target. Additionally, lipoprotein(a) [Lp(a)] has emerged as a crucial risk factor due to its strong correlation with CHD risk [11,12]. Apoprotein B100 serves as the principal apoprotein in atherogenic lipoproteins, while HDL particles primarily contain apolipoproteins A-I and A-II.

Lipoprotein Metabolism and the Role of Insulin Resistance

The liver produces triglyceride-rich very low-density lipoproteins (VLDLs), which facilitate the transport of oxidizable substrates, such as fatty acids, throughout the body. Lipoprotein lipase gradually hydrolyzes VLDL triglycerides, generating intermediate-density lipoproteins and, eventually, LDL particles. The liver removes LDL particles from circulation by expressing LDL receptors; however, LDL can also infiltrate the subendothelial space, promoting atherosclerosis.

Insulin resistance and diabetes lead to a decline in lipoprotein lipase activity. This impairment results in incomplete VLDL metabolism, increasing circulating VLDL remnants and reducing the production of intermediate-density lipoproteins and LDLs. Accumulated VLDLs and triglycerides in the bloodstream activate cholesterol ester transfer protein, which facilitates the exchange of cholesterol esters from HDL and LDL particles with triglycerides from VLDLs and their remnants. Consequently, HDL and LDL particles become enriched in triglycerides, making them more susceptible to

hepatic lipase degradation. This process contributes to reduced serum HDL-C levels as the kidneys rapidly clear HDL particles.

Several mechanisms contribute to declining HDL-C levels in insulin resistance. The gene encoding apolipoprotein A-I contains three insulin response elements, and reduced hepatic insulin sensitivity leads to lower apolipoprotein A-I production and decreased HDL formation. Furthermore, insulin-resistant adipocytes generate fewer HDL particles. Chylomicrons from the jejunum, which contain substantial amounts of apolipoprotein A-I, fail to release surface components such as apolipoprotein A-I when lipoprotein lipase activity is diminished [13]. Under normal conditions, apolipoprotein A-I molecules lipidize to form HDL in circulation.

Triglyceride-rich LDL particles undergo degradation, resulting in smaller, denser, and more numerous LDL particles. Some studies indicate that these smaller LDL particles are more atherogenic than larger, less dense LDL particles.

Lipid Profile Variations in Women Across Different Life Stages

Dyslipidemia in women differs not only from that in men but also varies throughout different phases of life, including the menstrual cycle, pregnancy, and menopause.

Menstrual Cycle and Lipid Fluctuations

Lipid levels fluctuate throughout the menstrual cycle in reproductive-age women, indicating that hormonal regulation plays a role. Total cholesterol and LDL-C levels decline during the luteal phase following their peak in the follicular phase. These levels rise sharply after menstruation, reaching their highest point in the follicular phase before gradually decreasing throughout the luteal phase [14]. The rise in total cholesterol and LDL-C occurs just before the estrogen peak in the follicular phase,

followed by a decline as progesterone levels increase towards the end of the cycle [14].

HDL-C levels peak around ovulation, coinciding with the highest estrogen levels. Therefore, lipid profiles measured at different phases of the menstrual cycle may show slight variations. Additionally, numerous studies, including a large meta-analysis of 82 trials, indicate that women using oral contraceptives tend to have higher total cholesterol and triglyceride levels compared to those who do not, with varying effects on LDL-C [15-17].

Lipid Changes During Pregnancy

Pregnancy represents a unique physiological state characterized by increased cholesterol levels and insulin resistance [18,19]. Rising estrogen and progesterone levels in the first trimester stimulate pancreatic beta-cell hyperplasia, leading to increased insulin production and reduced insulin sensitivity [20]. Lipid synthesis and storage also increase to support future fetal needs [21,22]. Alterations in hepatic lipid metabolism can be detected as early as 10 weeks of gestation, along with maternal adipocyte hypertrophy [23].

Later in pregnancy, high estrogen levels in the third trimester promote lipogenesis and VLDL synthesis in the liver. Estrogen also reduces hepatic lipase activity, impairing lipoprotein clearance [20]. Consequently, hormonal shifts during pregnancy contribute to both increased lipid synthesis and decreased clearance [24]. As a result, plasma cholesterol levels rise by approximately 50% compared to pre-pregnancy levels, and triglyceride levels can nearly double [25].

Studies suggest that pregnant women with elevated LDL cholesterol levels face a higher risk of gestational diabetes, preeclampsia, Cesarean delivery, and preterm birth [22,26,27]. These complications also translate into a 1.8 to 4-fold increased risk of cardiovascular disease for affected mothers later in life [25,28]. Given the impact of pregnancy on lipid metabolism and its associated health risks, the ideal time for dyslipidemia screening is before conception [29].

The American Heart Association and the American College of Cardiology recommend establishing a baseline lipid profile in early adulthood [30]. However, current data show that early screening for lipid disorders is uncommon, with approximately 80% of women of reproductive age never having had their cholesterol levels checked [31]. The National Lipid Association advises that if pre-pregnancy lipid assessments were not performed, lipid levels should be evaluated during the first obstetric visit [29].

Tailored Approaches to Dyslipidemia in Women

Given the unique lipid profile changes throughout a woman's life, clinicians should consider establishing a baseline lipid profile for young women of reproductive age to assess cardiovascular risk before pregnancy.

After childbirth, women tend to maintain higher triglyceride and total cholesterol levels, which support lactation by facilitating cholesterol and triglyceride utilization. Studies indicate that women who breastfeed for extended periods tend to have higher HDL-C levels, although there is no consistent evidence linking LDL-C, total cholesterol, or triglyceride levels to breastfeeding duration [32,33]. Despite persistently elevated lipid levels during pregnancy and postpartum, some research suggests that breastfeeding offers long-term cardiovascular benefits [34].

Impact of Menopause on Lipid Levels and Cardiovascular Risk

The risk of cardiovascular disease in women increases significantly after the age of 50, coinciding with the onset of menopause [35]. Prior to this stage, women's lipid levels tend to be lower than men's, but after menopause, total cholesterol levels rise disproportionately in women compared to men. This shift is thought to be driven by estrogen deficiency, which contributes to metabolic changes such as increased visceral fat accumulation, higher triglyceride and LDL-C levels, and elevated lipoprotein(a) [35]. Conversely, HDL-C levels decline [35].

Postmenopausal women also experience heightened insulin resistance, endothelial dysfunction, and elevated blood pressure, alongside increased sympathetic nervous system activity [36]. Additionally, menopause alters drug metabolism, slowing the clearance of certain medications and affecting treatment efficacy [37]. Due to these hormonal and metabolic shifts, postmenopausal women often require more intensive lipid management. The latest guidelines from the American College of Cardiology (ACC) and the American Heart Association (AHA) recognize menopause and pregnancy-related complications as risk factors for cardiovascular disease.

Researchers have explored hormone replacement therapy (HRT) as a potential intervention for cardiovascular risk reduction in postmenopausal women [38]. While the decline in estrogen levels is linked to an increased risk of heart disease, clinical trials have shown mixed results regarding the effectiveness of HRT in cardiovascular protection [39-41]. For certain women with elevated lipoprotein(a) levels, aspirin has been identified as a beneficial option for primary prevention [42]. A retrospective study involving 12,815 patients (54% women) found that among 406 individuals with a genotype associated with elevated Lp(a), those taking aspirin had a significantly

lower incidence of major adverse cardiovascular events (MACE), with an absolute risk reduction of 11.4 per 1,000 person-years compared to 1.7 in the general population ($p < 0.008$) [42].

Additional Cardiovascular Risk Factors in Women

Certain medical conditions further contribute to cardiovascular risk in women. Polycystic ovarian syndrome (PCOS), characterized by hormonal imbalances that result in elevated androgen levels, is associated with obesity, insulin resistance, hypertension, and dyslipidemia [43]. A meta-analysis found that women with PCOS have twice the cardiovascular risk of the general population [44, 45]. Early screening and proactive lipid management through lifestyle modifications and pharmacological treatments are essential for this high-risk group [44].

Similarly, familial hypercholesterolemia (FH) necessitates careful monitoring of cardiovascular health. Heterozygous FH, estimated to affect approximately 1 in 250 individuals, is likely underdiagnosed due to low screening rates [46, 47]. This inherited disorder increases the likelihood of developing atherosclerotic cardiovascular disease (ASCVD) by approximately 20-fold [48]. Although FH can be detected as early as childhood through lipid screening, many cases remain unrecognized until cardiovascular complications arise in young adulthood, particularly among women in their 20s and 30s [46]. Unlike the general population, women with FH develop cardiovascular disease at the same young age as men [49].

Homozygous FH, though rarer, presents with more severe disease and an earlier onset of ASCVD. Without treatment, about 30% of untreated women with FH experience myocardial infarction before the age of 60, underscoring the importance of early diagnosis and intervention [49]. In all patients, lifestyle modifications—including

smoking cessation, dietary adjustments, and regular exercise—should be emphasized to reduce overall risk. Women with FH who are pregnant or planning pregnancy should consult a lipid specialist to carefully weigh the risks and benefits of medication and, in some cases, lipoprotein apheresis.

Dyslipidemia and Cardiovascular Disease Risk in Women

Research has consistently demonstrated a strong link between abnormal lipoprotein levels and increased CHD risk. Elevated LDL-C and triglycerides, along with low HDL-C levels, independently contribute to atherosclerotic cardiovascular disease. However, the degree of risk associated with each lipoprotein abnormality differs between men and women.

On average, women have HDL-C levels that are about 10 mg/dL higher than men. The National Cholesterol Education Program's Adult Treatment Panel identifies HDL-C levels below 50 mg/dL as a CHD risk factor in women—10 mg/dL higher than the threshold for men [8].

Young women with a high concentration of small, dense LDL-C particles face a significantly greater risk of premature myocardial infarction. Beyond LDL particle size, lipoprotein(a) has been recognized as an independent predictor of CHD risk in women. The Heart and Estrogen/Progestin Replacement Study found that postmenopausal women with Lp(a) levels in the highest quartile had a 54% increased risk of CHD events compared to those in the lowest quartile [50].

Management Strategies for Dyslipidemia in Women

Current guidelines for preventing cardiovascular disease prioritize lifestyle modifications as the primary intervention for women with dyslipidemia. The

INTERHEART study evaluated the impact of modifiable risk factors, including smoking cessation, daily consumption of fruits and vegetables, and regular physical activity. Findings revealed that these lifestyle changes reduced the risk of myocardial infarction by over 80% [51].

According to data from the World Health Organization (WHO), eight modifiable risk factors—alcohol consumption, smoking, hypertension, obesity, hypercholesterolemia, diabetes mellitus, inadequate fruit and vegetable intake, and physical inactivity—account for 61% of cardiovascular deaths and over 75% of coronary heart disease cases. Although lifestyle modifications effectively lower cardiovascular risk, they appear to benefit men more than women.

Statins are recommended as the first-line treatment for patients with hypercholesterolemia to lower cardiovascular disease risk. Over the past two decades, multiple clinical trials have demonstrated that statin therapy significantly reduces morbidity and mortality in both primary and secondary prevention settings. The JUPITER trial specifically evaluated the benefits of statins in primary prevention and found a 44% reduction in the composite endpoint of myocardial infarction, stroke, revascularization, unstable angina, or CHD-related death [52].

To improve risk assessment for statin therapy initiation, clinicians should consider additional factors such as coronary artery calcium (CAC) score, history of pregnancy complications, elevated Lp(a), inflammatory conditions like rheumatoid arthritis, and comorbid fatty liver disease. The latest ACC/AHA guidelines encourage the incorporation of these risk-enhancing factors into clinical decision-making [53].

For patients who cannot tolerate statins or require additional LDL-C reduction, alternative lipid-lowering therapies—including ezetimibe, bile acid sequestrants,

PCSK9 siRNA, and PCSK9 monoclonal antibodies—may be considered. Ezetimibe lowers LDL-C by approximately 13% to 20% and is generally well tolerated [30]. The 2018 ACC/AHA cholesterol guidelines recommend the use of non-statin medications in combination with statins in select cases to achieve optimal LDL-C reduction [30].

To ensure effective cardiovascular risk management, clinicians should prioritize routine screening and evidence-based treatment for female patients. A baseline lipid profile is recommended for all young women of reproductive age. Additionally, healthcare providers should be vigilant in identifying conditions such as FH and PCOS, as women with these disorders require early intervention and comprehensive risk reduction strategies. Preventive measures, including aspirin and statins, should be initiated early, particularly for high-risk populations, including minority women who are frequently underrepresented in clinical research.

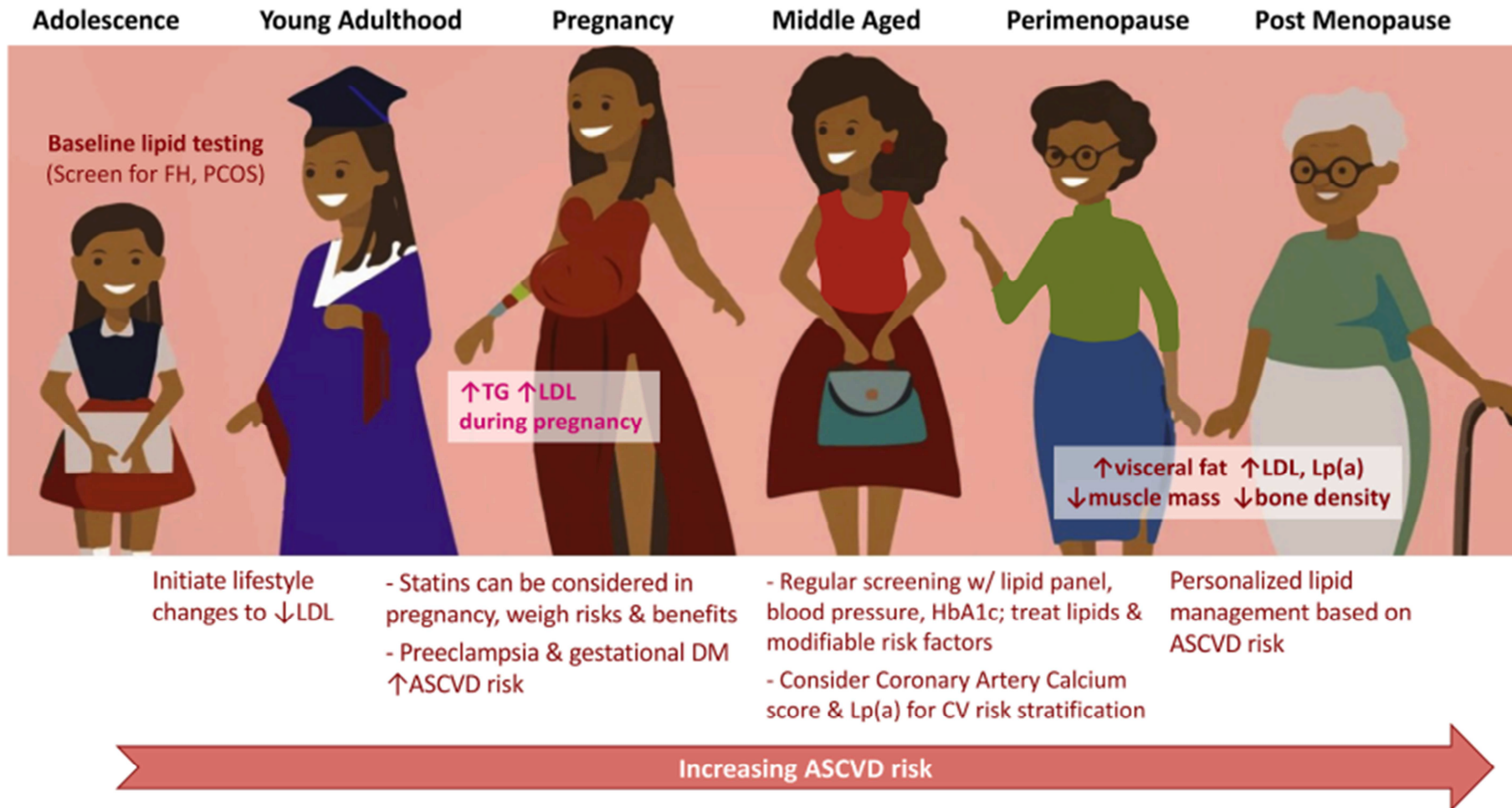


Figure 1: Considerations for Atherosclerotic cardiovascular disease risk among women over their lifespan

Heart disease is the leading cause of mortality among women. Dyslipidaemia is very commonly seen in women. Women undergo various levels of hormonal disturbances throughout their lifetimes, which have a considerable impact on the lipo-protein metabolism. Women's lipid profiles worsen with menopause, transitioning to dyslipidemia that cause increased atherogenicity. Hormone replacement therapy usually produces a better lipid profile, however it was not seen playing a role in lowering the usual risk of CHD. Even though women usually have a greater baseline HDL-C than men of the same age and weight, a physician should not presume that a high level of HDL-C will disguise the risk associated with high or non high levels of HDL. Women in whom primary prevention can be done, they should have their risk assessment done along with starting statins according to the guidelines. Women who have atherosclerotic - cardiovascular disease should consider taking statins. Statin therapy is proven to be effective in these women. Statins and drugs like lipid-modifying agents should be administered according to regional guidelines.

EVIDENCE FROM PREVIOUS LITERATURE

- 1. Dipak Kumar Adak, et al.,** conducted a study amongst pre-menopausal and the post-menopausal Kshatriya women in Sonarpur, South 24 Parganas, West Bengal, India in order to determine the prevalence of dyslipidemia. 142 adult Paundra Kshatriya women (n pre-menopausal women = 96; n post-menopausal women = 46) were selected from a peri-urban setting of Sonarpur, South 24 Parganas, West Bengal. The results showed that 19.79% of pre-menopausal women and 17.39% of post-menopausal women had normal levels of lipids. Prevalence of dyslipidemia was slightly higher (82.7%) among the post-menopausal women compared to pre-menopausal women (80.2%). While high LDL cholesterol emerged as one of the prime causes for dyslipidemia among pre-menopausal

women, hypercholesterolemia emerged as one of the prime causes for dyslipidemia among post-menopausal women. Regression analysis revealed a significant impact of 3 factors in pre-menopausal women and impact of 6 factors in post-menopausal women. Though Paundra Kshatriya women maintain a relatively less stressful and high physical activity lifestyle, they exhibited high levels of lipid abnormalities. The peri-urban population is undergoing lifestyle and dietary changes due to a close proximity to the urban centre, Kolkata [54].

- 2. B Sudhakar Babu, et al.,** did a cross sectional study at Viswabharathi medical College and General Hospital from June-2019 to July 2019 in 100 healthy female attendants who comprised 50 Premenopausal women, aged between 20-45 years and 50 post-menopausal women aged women between 46-70 years with a history of natural menopause, and stoppage of menstruation for a minimum of one year accompanying with the patients attending OPD of General Medicine and OBG department. The results showed that the mean of serum total cholesterol was 187.54 mg/dl in test group and 171.98 mg/dl in control group with p value of <0.01. The mean of serum triglycerides was 160.36 mg/dl in test group and 117.7 mg/dl in control group with p value of <0.0001. The mean of serum LDL-C was 118.3mg/dl mg/dl in test group and 103.54 mg/dl in control group with p value of <0.003. The mean of serum VLDL-C was 118.3mg/dl in test group and 103.54 mg/dl in control group with p value of <1.48. The mean of serum HDL-C was 37.1mg/dl in test group and 42.14 mg/dl in control group with p value of <0.0001 [55].

- 3. Nkeunen Gerard, et al.,** did a cross-sectional study at Yaounde Military Hospital between November 2016 to July 2017 to determine the plasma lipid profile and dyslipidemia prevalence of some Cameroonian women. The enrolled participants

were grouped into postmenopausal (105) and premenopausal (127) apparently normal women. The results showed that there was no significant difference in the mean values of total cholesterol, HDL-C, LDL-C as well as triglyceride between premenopausal and postmenopausal women. The mean HDL-C concentration was stable in the age subgroups while the mean total cholesterol and LDL-C were slightly increasing as postmenopausal participants got older. LDL-C level was significantly higher in women who had been in menopause for more than 15 years. The overall dyslipidemia prevalence was 53%. Dyslipidemia was significantly higher in the subgroup of women who had freshly entered menopause. Even though there was no change in the mean concentration of the plasma lipid parameters, of pre- and postmenopausal participants there was a general need to improve their lifestyle in order to reduce dyslipidemia prevalence [56].

- 4. Manafa P.O., et al.,** conducted a study aimed to determine the impact of menopause over lipid profile in total of 100 apparently healthy subjects who comprised 50 menopausal women aged 45 – 77 years and 50 pre-menopausal women between the aged between 20-52 years. The findings reveal that the mean levels of serum triglycerides (TG) and low density lipoprotein cholesterol (LDL cholesterol) showed a statistically significant increase in menopausal women compared with the premenopausal subjects ($P < 0.05$). There was a progressive increase in the mean levels of total cholesterol, triglycerides, LDL-cholesterol and VLDLcholesterol with duration of menopause while the levels of HDL-cholesterol decreased with duration of menopause. There were no significant variations among the various age categories of the menopausal subjects and the levels of the lipid parameters studied ($p > 0.05$). The findings suggest that

premenopausal women have less proatherogenic lipid profile than their menopausal counterparts [57].

5. Shilpa Joshi, et al., did a case control study at a Medical College Hospital & Research centre Pimpri, Pune from January 2023 to December 2023 on patients attending the OPD of Dr D Y Patil Medical College Hospital Pimpri, Pune to evaluate lipid profile status in post-menopausal women and compare with premenopausal women. The results showed that mean of serum total cholesterol in post menopausal group was 185.28 mg/dl and 158.28 mg/dl in pre menopausal group. The mean of serum Triglycerides was 145.59 mg/dl in post menopausal group as compared to 116.34 mg/dl in pre menopausal group. Mean of serum VLDL was 29.12 mg/dl in post menopausal grp as compared to 23.24 mg/dl in pre menopausal group. Mean of serum LDL was 116.96 mg/dl in post menopausal grp as compared to 98.86 mg/dl in pre menopausal females. Lipid profile determinations were done by Enzymatic methods on Alinity C – fully Automated Analyzer. Dyslipidemia is an important risk factor for arteriosclerotic cardiovascular disease (ASCVD). Due to decreased estrogen in post-Menopausal women and increased levels of cholesterol and LDL levels, chances of cardiovascular diseases are increased. It is necessary to educate Women about Menopause and Lipid Profile should be done to assess the risk of cardiovascular diseases [58].

6. S Khanduker, et al., did a cross-sectional study in Bangladesh medical college hospital dhanmondi, Dhaka from the period of April to July 2017 to estimate the serum lipid profile and the atherogenic index of plasma among the pre and post-menopausal women from a group of 339 women, 140 premenopausal aged between 25-50 years and 199 postmenopausal aged between 51-70 years. The

results showed that there were statistically significant increase in serum TC (191.21±45.50 mg/dl), TG (185.83± 111.83 mg/dl) and LDL-C (118.71±38.48 mg/dl) in post-menopausal women. Their HDL-C level (38.67±10.00mg/dl) was significantly decreased. The calculated atherogenic index of plasma (AIP) was significantly higher (0.63±0.27) in post-menopausal women as compared to that in premenopausal women (0.50±0.29). Menopause leads to changes in lipid profile. By elevating LDL and the reduction of cardioprotective HDL is an indication that menopause is an independent risk factor for developing cardiovascular disease. These changes are caused by loss of cardio-protective effect of oestrogen [59].

7. Ranjit Kumar conducted a study to December 2023 on fifty pre-menopausal women and fifty postmenopausal women to assess lipid profile in pre and post-menopausal women. The postmenopausal women who were studied were those with a history of natural menopause, who had cessation of menstruation for a minimum of one year, and premenopausal women who were studied were those who had regular menstruation. The results showed that TC, TGL, VLDL and LDL were significantly elevated in post-menopausal women as compared to premenopausal women. Similarly, the HDL level in premenopausal level was higher as compared to postmenopausal women. The post-menopausal women are at more risk of cardiovascular diseases as compared to premenopausal women [60].

8. Tiwari J, Naagar JK conducted a cross sectional study in the Medicine Department Bundelkhand Medial College and associated hospital Sagar, MP during the period of March 2013 to March 2014 among 100 patients each in the premenopausal and post menopausal group to evaluate risk factors for coronary artery disease in post menopausal women. This includes study and comparison of the serum lipid profile in premenopausal and postmenopausal women with

reference to body mass index. The study found that postmenopausal women have significantly higher serum levels of total cholesterol, LDL, VLDL, and triglycerides and lower serum HDL levels than their premenopausal counterparts, regardless of BMI level ($p \leq 0.05$). Postmenopausal women's lipid profiles change, increasing their risk of cardiovascular disease [61].

MATERIALS AND METHODS

STUDY DESIGN:

This study was hospital-based single centered, cross-sectional study.

STUDY SETTING:

This hospital based study was carried out in Medical Intensive Care Unit in the Tertiary Care Centre, Belagavi, India.

STUDY PERIOD:

The study was for duration of 12 months i.e., August 2023 to July 2024 upon getting approval from IRB (Ethical and Scientific Clearance).

STUDY POPULATION:

The study population comprised of patients admitted to Tertiary Care Centre, Dr Prabhakar Kore Hospital Belagavi, India and attenders of patients, hospital working staff.

Inclusion criteria:

- The participants who had physiological menopause with amenorrhea for at least 1 year were enrolled.
- Pre-menopausal women without co morbidities like HTN, T2DM.

Exclusion criteria:

- Exclusion criteria for the study include those with fat abnormalities, diabetes, high blood pressure, cardiovascular illness, acute and chronic liver diseases, and inflammation.

- Women who had menopause owing to hysterectomy or any other causes except for natural causes were also not included.
- The study excluded participants who were receiving exogenous hormones, hormone replacement treatment, or lipid-lowering medications.

SAMPLE SIZE:

Formula used for sample size calculation is,

$$n = \frac{(1 - Prevalence) Z_{\alpha/2}^2 \sigma^2}{d^2}$$

where σ is the expected standard deviation of the population, d is acceptable margin of error and for 95% confidence level, $Z_{\alpha/2}$ value is 1.96.

The mean triglyceride (TG) in pre-menopausal women is 100.87 with standard deviation of 32.11. Considering same result at 95% CI and 5% maximum error, the sample size is given by,

$$n = \frac{1.96 * 32.11^2}{d^2}$$

$$n = 158.4356 \approx 158$$

Hence, the minimum sample size required was 158 subjects.

SAMPLING TECHNIQUE:

A total of 158 consecutive patients who were at the Tertiary Care Centre, Belagavi, India satisfying the inclusion criteria and exclusion criteria were selected by convenient sampling and were included.

METHOD OF DATA COLLECTION:

- This hospital-based single centered, cross-sectional study; was conducted in KLE Prabhakar Kore Hospital Belgavi healthy women postmenopause and premenopause.
- Subjects were randomly selected from healthy women who were visitors or patient's escort in KLE hospital, and hospital staff.
- After taking Ethical clearance, an informed written consent was obtained.
- The patients or their attenders were explained about the implication and outcome of study in local language. They were told that they may choose whether or not to participate in the study, and that doing so would have no effect on the therapy procedure.

Assessment of parameters:

- Detailed history was taken from the patients or attendants including demographic details and past medical history.
- A complete physical examination with monitoring of vitals (temperature, PR, RR, and BP) was done.
- Then the collection of samples was taken from them.
- Blood samples were tested for analytical purposes at KLE Hospital's specialised laboratory. A total of 158 female samples were selected.

Methodology:

- Patients provided informed consent.
- Data was collected using a pre-structured and tested proforma.
- Baseline data, including socio-demographics and medical history, were gathered.

- Questionnaire sheets were used to capture detailed information for each sample.
- Then they were sub divided into premenopausal and post-menopausal women based on the inclusion criteria.
- The survey collected data on sociodemographic variables, age, weight, height, lifestyle, and menopausal status.
- All women in the study provided written informed consent.
- Lipid measurement. Cholesterol, LDL, HDL, and triglycerides will be measured by using the same approach and an equipment, and the details are as follows: Blood was obtained hours after an overnight fast in both groups before and after menopausal women. Blood tubes were centrifuged, and the results were recorded.

STATISTICAL ANALYSIS

- Data had been imported into MS Excel and analysed with Statistical Package for Social Sciences software version 23.
- Descriptive statistics were used to summarise quantitative factors in clinical data. The standard deviation was determined as a measure of variability.
- Qualitative factors were reported as percentages with 95% CI.
- Statistically significant differences in mean values was seen in pre and postmenopausal women were examined using student's t test or Mann Whitney test for non-normal distributions.
- Chi-square and Fisher's exact tests were used to compare the proportions of pre- and postmenopausal women.
- The level of significance (P-Value) was set at $P < 0.05$.

ETHICAL CONSIDERATIONS

All patients participating in the study followed the rules provided by ICMR (1994) and the Helsinki Declaration (modified 2000).

- Patients who enrolled were informed.
- Each patient was adequately informed of the aims, methods, sources of funding, any possible conflicts of interest, and institutional affiliations of the researcher, the anticipated benefits and potential risks of the study and the discomfort it may entail to him/her and the remedies thereof.
- The study was conducted with utmost respect for the patient's privacy, confidentiality, and little influence on their emotional and physical well-being.
- All the possible treatment options were given and none were withheld.
- There was no discrimination of patients and all were treated in the best interest of the patient.
- Patients were given the option to withdraw from the research at any time without consequences.
- At all stages of the research, care and caution were used to minimise patient risk, prevent irreversible bad effects, and maximise benefit.
- All patients enrolled in the study provided informed consent.
- The management for the participants was as percurrent standard departmental protocol.

RESULTS

Table 1: Age distribution of subjects

Age	Frequency	Percentage
≤ 20 years	3	1.9%
21 – 40 years	43	27.2%
41 – 60 years	78	49.4%
> 60 years	34	21.5%
Total	158	100%
Mean (years)	48.4 ± 13.84	

Figure 1: Pie chart showing age distribution among subjects

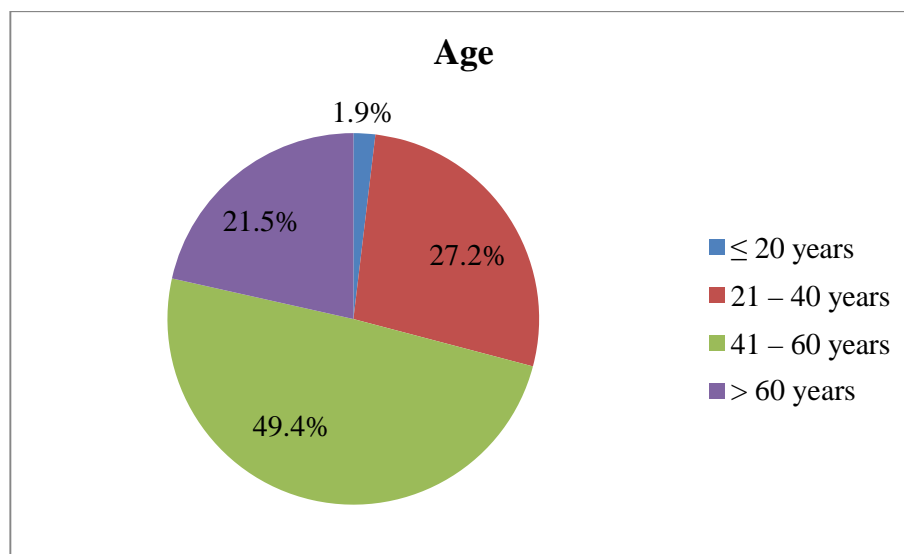


Table 1 shows the age distribution of the subjects. 3 were of age ≤ 20 years, 43 were 21 - 40 years, 78 between 41 and 60 years and 34 of age > 60 years. Mean age was 48.4 ± 13.84 years. Majority were of age 41 - 60 years (49.4%).

Table 2: Distribution of subjects according to their mean age

Age (in years)	Pre-menopausal group	Post-menopausal group
Mean	36.9	59.8
SD	8.66	6.66
p-value	0.003	

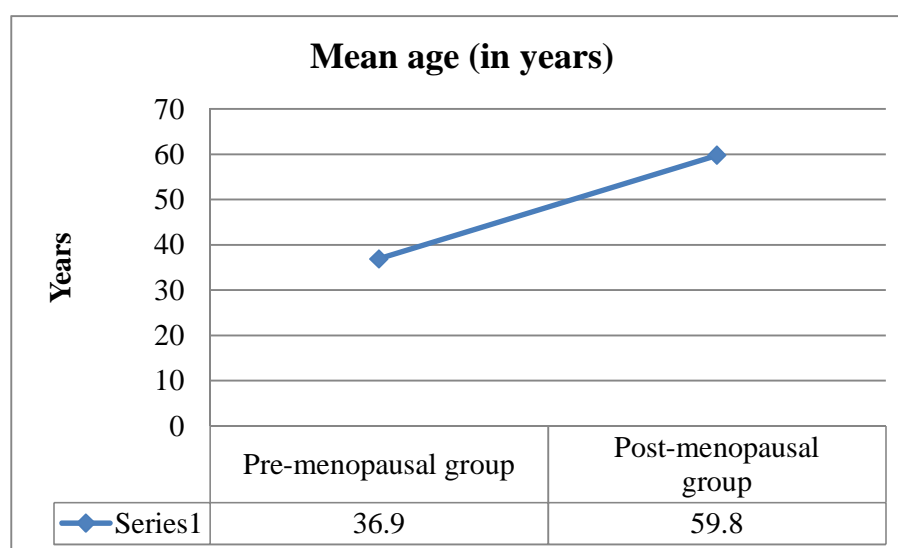
Figure 2: Line graph showing mean age distribution among subjects

Table 2 shows the mean age distribution of the subjects between the pre-menopausal group and the post-menopausal group. The mean age in pre-menopausal group was 36.9 years and mean age in the post -menopausal group was 59.8 years. We observed a good amount of difference in the mean age between pre-menopausal group and post-menopausal group (p-value = 0.003).

Table 3: Distribution of subjects according to Body mass index (BMI)

BMI (kg/m ²)	Frequency	Percentage
< 18.5 Underweight	0	0%
18.5 – 22.9 Normal	97	61.4%
23 – 24.9 Overweight	26	16.5%
25 – 29.9 Pre-Obese	29	18.4%
≥ 30 Obese	6	3.8%
Total	158	100%
Mean (kg/m²)	22.7 ± 2.72	

Figure 3: Pie chart showing BMI distribution among subjects

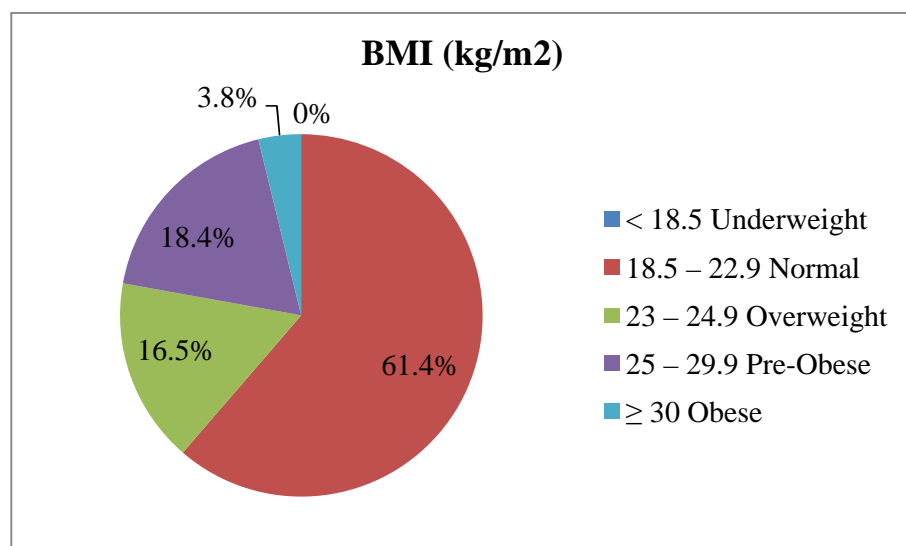


Table 3 shows the BMI distribution of the participants. None were underweight, 97 had normal BMI, 26 were of overweight, 29 were pre-obese and 6 were obese. Mean BMI was 22.7 ± 2.72 kg/m². Majority had normal BMI (61.4%).

Table 4: Distribution of subjects as per their mean BMI

BMI (kg/m ²)	Pre-menopausal group	Post-menopausal group
Mean	22.7	22.6
SD	2.71	2.73
p-value	0.948	

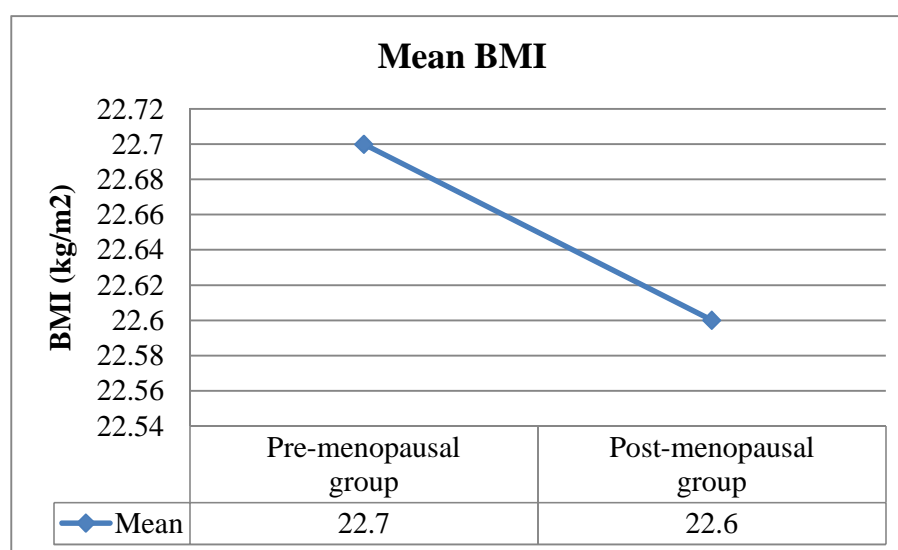
Figure 4: Line graph showing mean BMI distribution among subjects

Table 2 shows the mean BMI distribution of the subjects between the pre-menopausal group and the post-menopausal group. The mean BMI in the pre-menopausal group was 22.7 ± 2.71 kg/m² and that in the post-menopausal group was 22.6 ± 2.73 kg/m². There was no significant difference in the mean BMI between the pre-menopausal group and the post-menopausal group (p-value = 0.948).

Table 5: Distribution of subjects according to general physical examination findings

General Physical Examination	Mean	SD
Pulse Rate (bpm)	72.1	10.31
SBP (mmHg)	123.4	12.07
DBP (mmHg)	76.3	6.46

Figure 5: Line graph showing mean vitals distribution among subjects

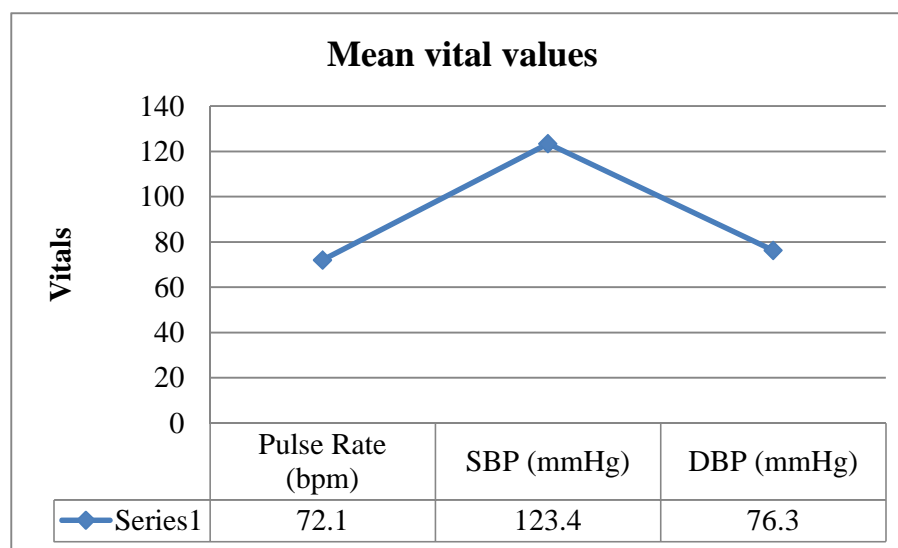


Table 5 shows the distribution of subjects according to their general physical examination findings. The mean values of PR were 72.1 ± 10.31 bpm, SBP and DBP were 123.4 ± 12.07 and 76.3 ± 6.46 mmHg respectively.

Table 6: Distribution of subjects according to lab parameters

Lab parameters	Mean \pm SD
Hb (g/dl)	10.5 \pm 1.15
Total Cholesterol (mg/dL)	179.3 \pm 40.42
Triglycerides (mg/dL)	135.6 \pm 75.63
LDL-CHOLESTROL (mg/dL)	125.5 \pm 38.36
HDL-CHOLESTROL (mg/dL)	47.0 \pm 12.44

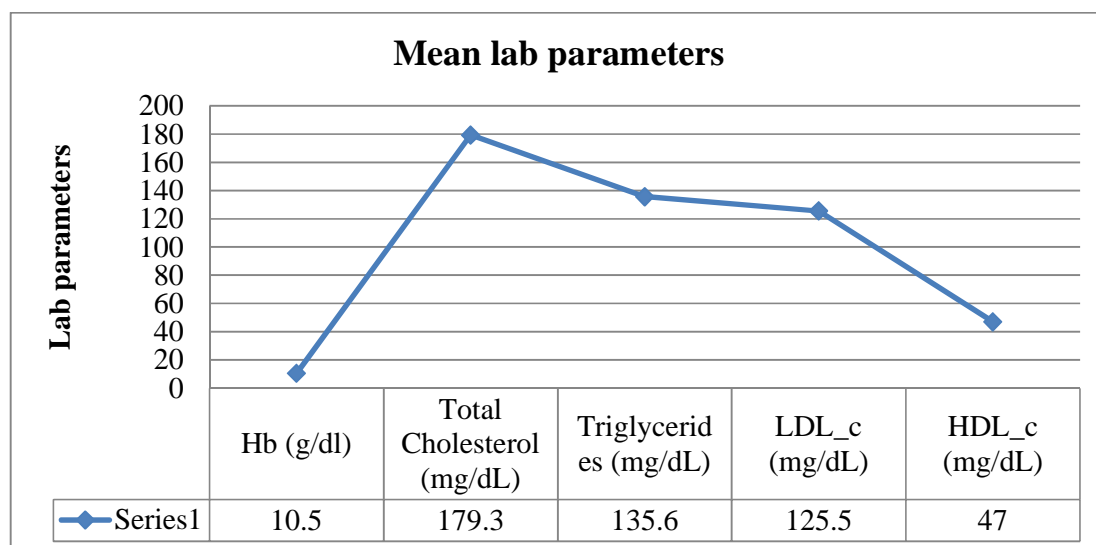
Figure 6: Line graph showing mean values of lab parameters among subjects

Table 6 shows distribution of subjects according to their lab parameters. The mean Hb was 10.5 \pm 1.15 g/dL, mean total cholesterol (TC) was 179.3 \pm 40.42 mg/dl, mean Triglycerides (TGLs) was 135.6 \pm 75.63 mg/dL, mean Low Density Cholesterol (LDL) was 125.5 \pm 38.36 mg/dL and mean High Density Cholesterol (HDL) was 47.0 \pm 12.44 mg/dL.

Table 7: Distribution of subjects according to their mean lab parameters between the pre-menopausal group and the post-menopausal group

Mean lab parameters	Pre-menopausal group	Post-menopausal group	p-value
Hb (g/dl)	10.8 ± 1.19	10.1 ± 1.02	0.319
Total Cholesterol (mg/dL)	173.0 ± 34.33	185.5 ± 45.07	0.047
Triglycerides (mg/dL)	122.7 ± 63.87	148.5 ± 84.23	0.021
LDL_c (mg/dL)	119.3 ± 30.48	131.7 ± 44.22	0.009
HDL_c (mg/dL)	47.6 ± 13.10	46.5 ± 11.80	0.041

Figure 7: Bar diagram showing mean values of lab parameters among the pre-menopausal group and the post-menopausal group

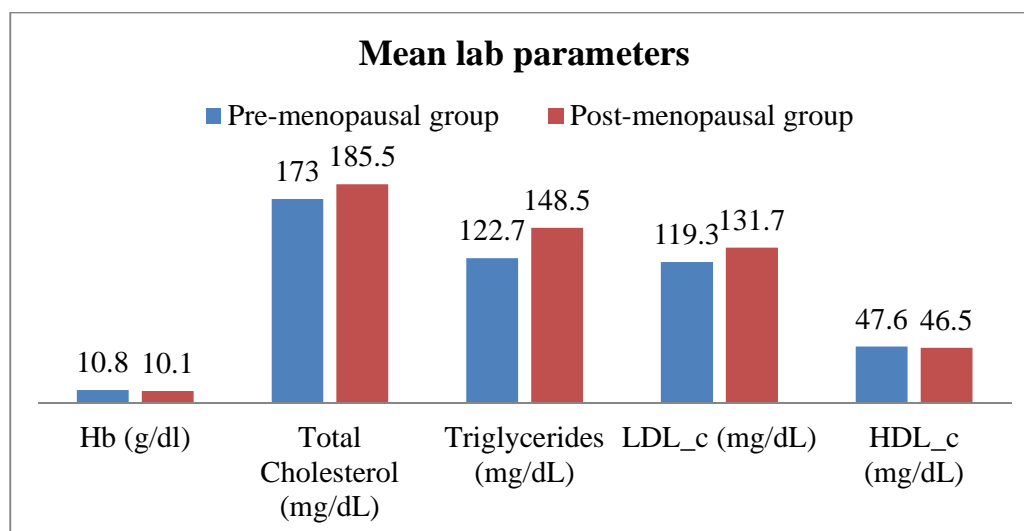


Table 7 displays the mean values of lab parameters for the pre-menopausal and post-menopausal groups. The pre-menopausal group's mean Hb was 10.8 ± 1.19 g/dL, while the post-menopausal group's was 10.1 ± 1.02 g/dL. There was no statistically significant difference in mean Hb levels between the pre- and post-menopausal groups (p-value = 0.319).

In the pre-menopausal group, the mean total cholesterol was 173.0 ± 34.33 mg/dL, while the post-menopausal group had an average of 185.5 ± 45.07 mg/dL. Both pre-menopausal and post-menopausal groups had significantly different mean Total Cholesterol levels (p-value = 0.047). It was much higher after menopause than before menopause.

In the pre menopausal group, the mean triglyceride level was 122.7 ± 63.87 mg/dL, while post menopausal group, the mean was 148.5 ± 84.23 . We observed a difference in mean Triglycerides between the pre-menopausal and post-menopausal groups (p=0.021) which was statistically significant. It was much higher after menopause than before menopause.

The pre-menopausal group's mean LDL_c was 119.3 ± 30.48 mg/dL, while the post-menopausal group's was 131.7 ± 44.22 mg/dL. There was a statistically significant difference in mean LDL_c between the pre- and post-menopausal groups (p-value = 0.009). It was much higher after menopause than before menopause.

The pre-menopausal mean HDL_c was 47.6 ± 13.10 mg/dL, while the post-menopausal mean was 46.5 ± 11.80 mg/dL. The pre-menopausal and post-menopausal groups had significantly different mean HDL_c levels (p-value = 0.041). It was much lower in the post-menopausal compared to the pre-menopausal group.

Table 8: Distribution of subjects according to atherogenic index of plasma (AIP)

Risk of cardiovascular disease (AIP)	Frequency	Percentage
Low (< 0.10)	16	10.1%
Medium (0.10–0.24)	20	12.7%
High (> 0.24)	122	77.2%
Total	158	100%
Mean AIP	0.43 ± 0.251	

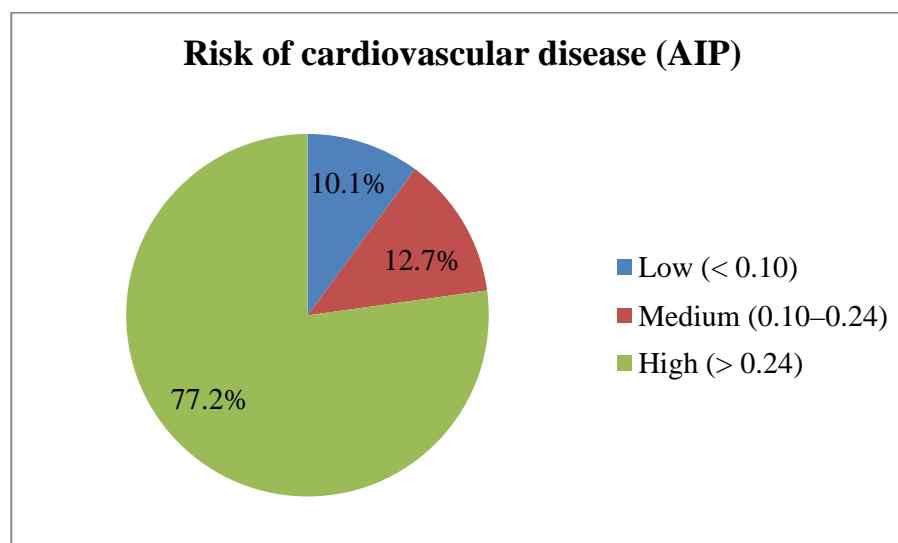
Figure 8: Pie chart showing atherogenic index of plasma distribution among subjects

Table 8 shows the risk of CVD and the atherogenic index of plasma distribution among subjects where out of 158, 16 had low risk of CVD with AIP being < 0.10, 20 had medium risk of CVD with AIP being 0.10 – 0.24 and 122 had high risk of cardiovascular disease with AIP being > 0.24. The mean AIP was 0.43 ± 0.251 . Majority had high risk of cardiovascular disease (77.2%).

Table 9: Comparison of AIP between the pre-menopausal group and the post-menopausal group among subjects

Mean \pm SD	Pre-menopausal group	Post-menopausal group	p-value
AIP	0.21 \pm 0.139	0.47 \pm 0.256	0.025

Figure 9: Line graph showing mean values of AIP between the pre-menopausal group and the post-menopausal group among subjects

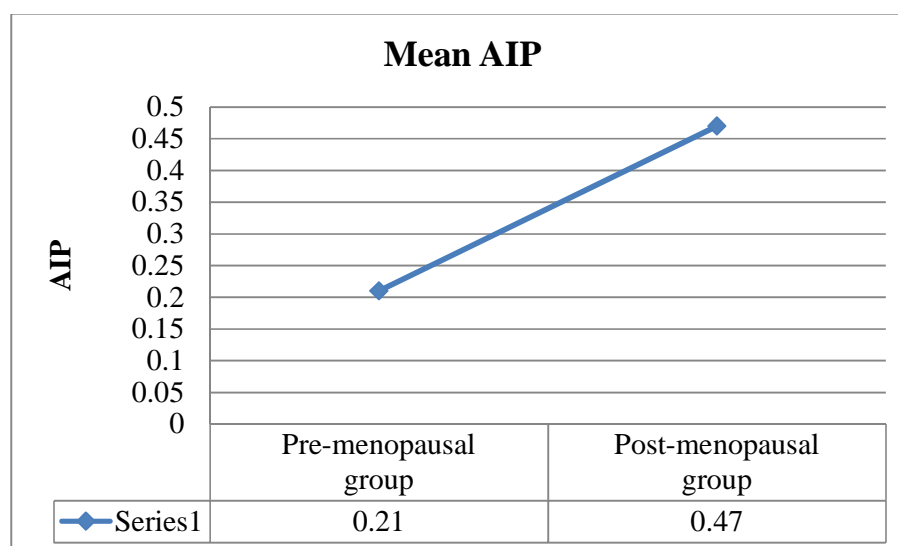


Table 9 compares AIP in the pre-menopausal and post-menopausal groups among participants. The average AIP of the pre menopausal group was 0.21 ± 0.139 , and the post menopausal group it was 0.47 ± 0.256 . The mean AIP between the pre-menopausal and post-menopausal groups ($p=0.025$) was statistically significant. It was substantially elevated in the post-menopausal group than in the pre-menopausal group, indicating a risk of cardiovascular disease.

Table 10: Distribution of subjects according to Total Cholesterol (TC) and atherogenic index of plasma (AIP)

Risk of cardiovascular disease (AIP)	TC Mean (mg/dL)	SD (mg/dL)
Low (< 0.10)	171.5	25.55
Medium (0.10–0.24)	177.9	33.56
High (> 0.24)	180.2	42.52
Total	179.3	40.42
p-value	0.783	

Table 10 displays the risk of cardiovascular disease and total cholesterol among participants. The average TC level was 171.5 ± 25.55 mg/dL in those at low risk, 177.9 ± 33.56 for medium risk, 180.2 ± 42.52 mg/dL for high risk. There was no statistically significant difference in mean TC among those with low, medium, high risk of CVD (p-value = 0.783).

Table 11: Distribution of subjects according to Triglycerides (TGL) and atherogenic index of plasma (AIP)

Risk of cardiovascular disease (AIP)	TGL Mean (mg/dL)	SD (mg/dL)
Low (< 0.10)	71	9.83
Medium (0.10–0.24)	84.1	15.74
High (> 0.24)	149.3	78.14
Total	135.6	75.63
p-value	< 0.001	

Table 11 displays the risk of cardiovascular disease and triglycerides among participants. TGL levels ranged from 71 ± 9.83 mg/dL in those at low risk to 84.1 ± 15.74 mg/dL for medium risk, 149.3 ± 78.14 mg/dL for high risk. There was a significant difference in mean TGL between those with low, medium, and high risk of cardiovascular disease (p-value < 0.001). It was much higher among those with a high risk of CVD than in those with a low or moderate risk.

Table 12: Distribution of subjects according to Low Density Cholesterol (LDL_c) and atherogenic index of plasma (AIP)

Risk of cardiovascular disease (AIP)	LDL_c Mean (mg/dL)	SD (mg/dL)
Low (< 0.10)	115.1	24.49
Medium (0.10–0.24)	120.4	28.85
High (> 0.24)	127.2	40.55
Total	125.5	38.36
p-value	0.497	

Table 12 displays the risk of cardiovascular disease and Low Density Cholesterol among participants. The average LDL_c level was 115.1 ± 24.49 mg/dL in those at low risk of cardiovascular disease, 120.4 ± 28.85 mg/dL for medium risk, 127.2 ± 40.55 mg/dL for high risk. There was no statistically significant difference in mean LDL_c levels among those at low, medium, and high risk of cardiovascular disease (p-value = 0.497).

Table 13: Distribution of subjects according to High Density Cholesterol (HDL_c) and atherogenic index of plasma (AIP)

Risk of cardiovascular disease (AIP)	HDL-C Mean (mg/dL)	SD (mg/dL)
Low (< 0.10)	61.5	8.09
Medium (0.10–0.24)	53.9	8.52
High (> 0.24)	44.7	12.07
Total	47.0	12.44
p-value	< 0.001	

Table 13 displays the risk of cardiovascular disease and High Density Cholesterol among participants. The average HDL_c level was 61.5 ± 8.09 mg/dL in those at low risk, 53.9 ± 8.52 mg/dl for medium risk, 44.7 ± 12.07 mg/dL for high risk. There was a significant difference in mean HDL_c between those with low, medium, and high risk for cardiovascular disease (p-value < 0.001). It was much lower among those with a high risk of cardiovascular disease than in those with a low or moderate risk.

Table 14: Comparison of Dyslipidemia between the pre-menopausal group and the post-menopausal group among subjects

Dyslipidemia	Pre-menopausal group	Post-menopausal group	Total
Yes	51 (64.6%)	64 (81.0%)	115 (72.8%)
No	28 (35.4%)	15 (19.0%)	43 (27.2%)
Total	79 (100%)	79 (100%)	158 (100%)
p-value	0.020		

Table 14 compares dyslipidaemia in pre menopausal and post menopausal groups of patients, where 115 had dyslipidaemia and 43 did not. The prevalence of dyslipidaemia was substantially greater in the post-menopausal group (81%), compared to the pre-menopausal group (64.4%), with a p-value of 0.020.

DISCUSSION

The present study aimed to conduct a comprehensive analysis in lipid analysis variations between pre-menopausal and post-menopause women at tertiary care center and to compare these findings with previous research to identify significant trends and deviations. Our results indicate substantial alterations in lipid metabolism post-menopause, characterized by increased levels of TC, LDL-C, and TG, accompanied by a decline in HDL-C. These changes are chiefly endorsed to hormonal fluctuations, mainly the turn down in estrogen levels, which has a vital role in lipid homeostasis. This observation aligns with multiple prior studies, reinforcing the idea that menopause is a key factor influencing lipid profile deterioration and elevating cardiovascular risk among aging women.

Baseline characteristics of the study participants:

Majority (49.4%) were aged between 41 and 60 years, 1.9% were ≤ 20 years, 27.2% were between 21- 40 years and 21.5% were between >60 years. The mean age was 48.4 ± 13.84 years. The mean age in the pre-menopausal group was 36.9 years and that in the post -menopausal group was 59.8 years.

Dipak Kumar Adak et al. [54] conducted a cross-sectional study to investigate the occurrence of dyslipidaemia among pre- and postmenopause women at Sonarpur. There were substantial age differences ($p < 0.001$) between pre- and post-menopause women. These baseline characteristics as per the age of the subjects were similar to that in the current study.

B Sudhakar Babu et al. [55] undertook a cross-sectional study to compare lipid characteristics between pre and postmenopause women, because cardiovascular

disease is prevalent in postmenopause. The mean age of pre-menopause women was 33.7 years, whereas age of post-menopause women was 55.9 years. Participants in this study had similar baseline age characteristics to those in the current investigation.

Nkeunen Gerard et al. [56] conducted a cross-sectional study to determine the lipid characteristics and occurrence of dyslipidaemia in women. The first group consisted of 105 postmenopause women with a mean age being 57.8 years. The second group consisted of 127 premenopause women. The average age - 31.9 years. The participants in this study had similar baseline characteristics in terms of age to those in the current investigation.

Manafa P.O., et al., [57] conducted a retrospective study to determine the influence of menopause on lipid profile in 100 apparently healthy patients, including 50 menopausal women aged 45 - 77 years and 50 pre-menopausal women aged 20 - 52 years. There were substantial age differences ($p < 0.001$) among pre and postmenopause women. The participants had similar baseline characteristics in terms of age to those in the current investigation.

Shilpa Joshi et al. [58] conducted a case control study to assess the lipid profile status of postmenopausal women and compare them to premenopausal women. There were substantial age differences ($p < 0.001$) among pre and postmenopause women. The participants had similar baseline characteristics in terms of age to those in the current investigation.

S Khanduker et al. [59] conducted a cross-sectional study to estimate the serum lipid profile and plasma atherogenic index among 339 women, 140 premenopausal aged 25-50 years and 199 postmenopausal aged 51-70 years. There were substantial age

differences ($p < 0.001$) among pre and postmenopause women. The participants had similar baseline characteristics in terms of age to those in the current investigation.

Ranjit Kumar [60] conducted a cross-sectional study to analyse the lipid profile of pre- and post-menopausal women. There were substantial age differences ($p < 0.001$) among pre and post-menopausal women. The participants had similar baseline characteristics in terms of age to those in the current investigation.

Tiwari J, Naagar JK [61] conducted a cross-sectional study to investigate risks to CAD in postmenopause. Premenopausal women had a mean of 30.4 years, whereas postmenopausal women range in age from 45 to 55, with an average of 44.3 years. There were substantial age differences ($p < 0.001$) among pre and postmenopause women. The participants had similar baseline characteristics in terms of age to those in the current investigation.

Table 15: Baseline characteristics of the study participants

Current study	Majority (49.4%) were aged between 41 and 60 years Mean age: pre-menopausal group was 36.9 years and in post -menopausal group was 59.8 years
Dipak Kumar Adak, et al. [54]	Significant differences in age ($p < 0.001$) among pre- and post-menopausal women
B Sudhakar Babu, et al. [55]	Mean age: pre-menopausal women: 33.66 ± 7.89 years Post-menopausal women: 55.88 ± 6.72 years
Nkeunen Gerard, et al., [56]	Mean age: pre-menopausal women: 31.9 ± 10.0 years Post-menopausal women: 57.8 ± 7.4 years

Manafa P.O., et al., [57]	Significant differences in age ($p < 0.001$) among pre- and post-menopausal women
Shilpa Joshi, et al., [58]	Significant differences in age ($p < 0.001$) among pre- and post-menopausal women
S Khanduker, et al., [59]	Significant differences in age ($p < 0.001$) among pre- and post-menopausal women
Ranjit Kumar [60]	Significant differences in age ($p < 0.001$) among pre- and post-menopausal women
Tiwari J, Naagar JK [61]	Mean age: pre-menopausal women: 30.38 years Post-menopausal women: 44.25 years

Clinical characteristics:

Majority (61.4%) had a normal BMI. The pre-menopausal group's mean BMI was 22.7 ± 2.71 kg/m², while the post-menopausal group's was 22.6 ± 2.73 kg/m². There was no significant difference in mean BMI between the pre- and post-menopausal groups (p -value = 0.948).

PR averaged 72.1 ± 10.31 bpm, with SBP and DBP at 123.4 ± 12.07 and 76.3 ± 6.46 mmHg, respectively. The mean haemoglobin level was 10.5 ± 1.15 g/dL.

Dipak Kumar Adak et al. [54] discovered that among pre-menopausal women, BMI was significantly associated with total cholesterol and HDL-c. Furthermore, DBP was strongly associated with LDL-c. The WHR was associated with TG in postmenopausal women, DBP was associated with LDL-c, FPG was associated to total cholesterol and HDL-c, and SBP was associated with LDL-c and HDL-c. The features of the participants in this study were similar to those of the current study participants.

B Sudhakar Babu et al. [55] conducted a cross-sectional investigation and discovered that the features of the participants in this study were similar to those of the current study participants.

In a cross-sectional study by Nkeunen Gerard et al. [56], 105 (45.3%) postmenopausal women aged 45 to 81 had a mean WC of 100 cms and mean BMI of 30.4 Kg/m². The other group comprised 127 premenopause women. The average WC and BMI were 80 cms and 27.3 kg/m². Postmenopausal participants had considerably larger waist circumference and BMI than premenopausal participants. The features of the participants in this study were similar to those of the current study participants.

Manafa P.O., et al., [57], in a retrospective analysis, found that the features of the participants in this study were similar to those of the current study participants.

Shilpa Joshi et al., [58] conducted a case control research and found that the features of the participants in this study were similar to those of the current study participants.

The study by S Khanduker et al., [59], which was a cross-sectional study, found that the features of the participants in this study were similar to the current study participants.

Ranjit Kumar's [60] cross-sectional study found that the features of the study participants were identical to those of the current study participants.

Tiwari J, Naagar JK [61] conducted a cross-sectional study and found that all women had no abnormal general and systemic examinations. There is a significant disparity in weight and Body Mass Index between the normal and other groups. The features of the participants in this study were similar to those of the current study participants.

Lipid Profile:

Mean total cholesterol (TC): 179.3 ± 40.42 mg/dl, mean Triglycerides (TGLs) was 135.6 ± 75.63 mg/dL, mean Low Density Cholesterol (LDL) was 125.5 ± 38.36 mg/dL, and mean High Density Cholesterol (HDL) was 47.0 ± 12.44 mg/dL.

The post-menopausal group had significantly higher mean Total Cholesterol levels than the pre-menopausal group (p-value = 0.047). The mean Triglyceride level was substantially greater among post-menopause group than pre-menopause group (p-value = 0.021).

The mean LDL_c level was considerably greater in post-menopause group than pre-menopausal group (p-value = 0.009). The post-menopausal group had significantly lower mean HDL_c levels than the pre-menopausal group (p-value = 0.041).

The article by Dipak Kumar Adak [54], cross-sectional study, showed that while hypercholesterolaemia (pre-menopause-46.9%,post-menopause-69.6%) and hypertriglyceridemia (pre-menopause-11.5%; post-menopause-21.7%) were more common in post-menopause, high LDL-c (pre-menopause-50%; post-menopause-47.8%) and low HDL-c. However, when dyslipidaemia was considered, it was discovered that premenopause had the more frequency of high LDL-c, whereas postmenopause had more occurrence of hypercholesterolaemia. The study indicated a significant difference in the proportions of hypercholesterolaemia (0.22 ± 0.09) and hyperglyceridemia (0.11 ± 0.05), but not in occurrence of more LDL-c and less HDL-c. Pre-menopause women and post-menopause women had three lipid abnormalities (hypercholesterolaemia, hypertriglyceridemia, and high LDL-c). Approximately 19.8% of premenopause and 17.4% of postmenopause had no abnormal lipid levels. But, 4.2% of premenopause and 6.5% of postmenopause women had 4 lipid

disparities. The features in participants in this study did not correspond to those of the present study participants.

In a cross-sectional study by B Sudhakar Babu et al. [55], TC levels averaged 187.54 mg/dl among first group and 171.98 in second group, with a statistically significant $p < 0.014$. Test group had a mean serum triglyceride level of 160.36, while the control had 117.7 mg/dl ($p < 0.0001$). Test group had mean serum LDL of 118.3, while the control had a mean of 103.54 mg/dl, with a statistically significant difference ($p < 0.003$). Mean VLDL levels were 35.88mg/dl in the first group and 21.37mg/dl in the control, ($p < 1.48$), indicating no statistical significance. The test group had a mean HDL of 37.1mg/dl, while the control had a mean of 42.14 mg/dl, indicating statistical significance ($p\text{-value} < 0.0001$). In postmenopausal women, mean TC, TGs, and LDL-c increased significantly, while mean HDL-c decreased significantly when compared to premenopausal women. The features of the participants in this study were similar to those of the current study participants.

Nkeunen Gerard et al. [56], established no dissimilarity in mean TC, HDL-C, LDL-C, triglycerides among premenopause and postmenopause subjects. Mean HDL-C and LDL-C in premenopause participants remained steady across age groups. Mean HDL-C level remained steady in postmenopause, but mean TC and LDL-C increased somewhat upon age. According to detailed investigation, no triglyceride fluctuation pattern was detected in both pre and postmenopause across ages. Participants who had been postmenopausal for over 15 years had a substantially more ($P = 0.02$) mean LDL-C (1.4 ± 0.2 g/l). Women with YSM > 15 had a higher mean total cholesterol levels (2.1g/l) compared to others. There was no significant dissimilarity among them ($P = 0.05$). HDL-C and TGs differed across groupings. The features of the participants in this study did not correspond to those of the present study participants.

A retrospective study by Manafa P.O. et al. [57] found that menopausal women had significantly higher mean levels of serum triglycerides and LDL compared to premenopausal women. However, no significant dissimilarity in mean TC, HDL, and VLDL-C among premenopause and menopause was observed. The mean TC, TGs, LDL-C, VLDL-C increased over time during menopause, but HDL cholesterol levels declined. There were no significant differences in lipid parameters amongst menopausal subjects of different ages ($p>0.05$). The features of the participants in this study did not correspond to those of the present study participants.

Shilpa Joshi et al. [58] conducted a case control study and found that the mean serum total cholesterol in the postmenopausal group was 185.28 mg/dl and in the premenopausal group was 158.28 mg/dl. The mean blood triglyceride level was 145.59 mg/dL in the postmenopausal group, compared to 116.34 mg/dL in the premenopausal group. The mean serum VLDL level was 29.12 mg/dL in the postmenopausal group, compared to 23.24 mg/dL in the premenopausal group. The mean serum LDL level in the postmenopausal group was 116.96 mg/dL, compared to 98.86 mg/dL in the premenopausal females. The values of the aforesaid parameters were considerably more in post-menopause 50 years than pre-menopause under 50. Features of the participants in this study were similar to those of the current study participants.

S Khanduker et al. [59], in a cross-sectional study, found a substantial rise in total cholesterol, triglycerides, and LDL cholesterol, as well as a decrease in HDL cholesterol, among women in the menopausal transition period compared to women of a younger age group. Postmenopausal women had increased TC, TG, LDL-C, and decreased HDL-C levels. Compared to the two groups of women, postmenopausal

women had significantly higher TG levels ($p < 0.05$). The features of the participants in this study were similar to those of the current study participants.

Ranjit Kumar's [60] cross-sectional study revealed that TC, TGL, VLDL, and LDL levels were considerably higher in postmenopausal women than in premenopausal women. Similarly, premenopausal women's HDL levels were higher than postmenopause. The findings were significant. Postmenopausal women have a more harmful lipid profile than premenopausal women. The features of the participants in this study were similar to those of the current study participants.

A cross-sectional study by Tiwari J, Naagar JK [61] found that postmenopausal women had significantly greater levels of blood TC and TG than premenopausal women with the same BMI ($P < 0.05$). In this study group, postmenopausal women have considerably lower HDL c levels than premenopausal women ($p < 0.001$). The post-menopausal group had considerably greater LDL c levels than pre-menopause. VLDL values are modestly elevated among postmenopause. The features of the participants in this study were similar to those of the current study participants.

Table 16: Lipid profile of the study participants

Current study	The post-menopausal group had significantly higher mean total cholesterol, triglycerides, and LDL _c levels compared to the pre-menopausal group (p-value < 0.05). The post-menopausal group had significantly lower mean HDL _c levels than the pre-menopausal group (p-value = 0.041).
Dipak Kumar Adak, et al. [54]	Hypercholesterolaemia (pre-menopausal: 46.87%; post-menopausal: 69.56%) and hypertriglyceridemia (pre-menopausal: 11.46%; post-menopausal: 21.74%) were also shown to be more common in post-menopausal women. Pre-menopausal women had higher rates of high LDL cholesterol (50.00% vs. 47.83%) and lower HDL cholesterol (24.17% vs. 15.22%).
B Sudhakar Babu, et al. [55]	In postmenopausal women, the mean levels of serum total cholesterol (TC), triglycerides (TG), and low density lipoprotein cholesterol (LDL cholesterol) increased significantly, while the mean levels of serum high density lipoprotein cholesterol (HDL cholesterol) decreased significantly when compared to premenopausal women.
Nkeunen Gerard, et al., [56]	There was no significant difference in mean total cholesterol, HDL-C, LDL-C, or triglyceride levels between premenopausal and postmenopausal patients.
Manafa P.O., et al., [57]	Menopausal women had significantly higher mean serum triglyceride and LDL levels compared to premenopausal women (p<0.05). However, there was no significant difference in mean total cholesterol, HDL,
	and VLDL cholesterol values between premenopausal and menopausal women (p>0.05).
Shilpa Joshi, et al., [58]	Post-menopausal women over 50 years had considerably higher lipid profile values compared to pre-menopausal women under 50.
S Khanduker, et al., [59]	Postmenopausal women had increased TC, TG, LDL-C, and decreased HDL-C levels. Compared to the two groups of women, postmenopausal women had significantly higher TG levels (p<0.05).
Ranjit Kumar [60]	TC, TGL, VLDL, and LDL levels were considerably higher in post-menopausal women than in pre-menopausal women. Similarly, premenopausal women's HDL levels were higher than postmenopausal women.
Tiwari J, Naagar JK [61]	In postmenopausal women, the mean levels of serum total cholesterol (TC), triglycerides (TG), and low density lipoprotein cholesterol (LDL cholesterol) increased significantly, while the mean levels of serum high density lipoprotein cholesterol (HDL cholesterol) decreased significantly when compared to premenopausal women.

Atherogenic index of plasma (AIP) of the study subjects:

In this investigation, the average AIP was 0.43 ± 0.251 . The majority had a high risk of cardiovascular disease (77.2%).

The average AIP in pre-menopause : 0.21 ± 0.139 , in post-menopause group it was 0.47 ± 0.256 . There was significant dissimilarity in mean AIP among 2 groups ($p=0.025$). It was substantially high in post-menopause group than pre-menopause group, indicating risk of cardiovascular disease.

In a cross-sectional study by S Khanduker et al. [59], the mean AIP in the pre-menopausal group was 0.50 ± 0.29 , whereas in the post-menopausal group was 0.63 ± 0.27 . The pre-menopausal and post-menopausal groups had significantly different mean AIPs (p -value < 0.001). It was substantially high in post-menopause than pre-menopause, indicating a risk of cardiovascular disease. The features of the participants in this study were similar to those of the current study participants.

Table 17: AIP of the study participants

<p>Current study</p>	<p>The mean AIP in pre-menopause was 0.21 ± 0.139, post-menopause group it was 0.47 ± 0.256. It was substantially high in post-menopause than pre-menopause, indicating a risk of cardiovascular disease (p-value = 0.025).</p>
<p>S Khanduker, et al., [59]</p>	<p>The mean AIP for the pre-menopausal group was 0.50 ± 0.29. Post-menopausal group: 0.63 ± 0.27. The pre-menopausal and post-menopausal groups had significantly different mean AIPs (p-value < 0.001).</p>

Cholesterol levels and atherogenic index of plasma (AIP) of the study subjects:

In this investigation, there was no statistically significant difference in mean TC and LDL_c levels between those at low, medium, and high risk of cardiovascular disease (p-values = 0.783 and 0.497, respectively).

The mean TGL and HDL_c levels differed significantly among those with low, medium, and high risk to cardiovascular disease (p-value < 0.001). The mean TGL was substantially higher in those with a high risk of cardiovascular disease than in those at low and medium risk. The mean HDL_c level was substantially lower in those with a high risk of cardiovascular disease than in those with a low or medium risk.

Dipak Kumar Adak et al. [54] conducted a cross-sectional study and discovered that post-menopause had higher TC, TGs, LDL-C, HDL-C, VLDL-C than pre-menopause. The findings revealed a dissimilarity in TC among 2 (t=5.03; p<0.05). The features of the participants in this study were similar to those of the current study participants.

S Khanduker et al. [59], in a cross-sectional investigation, found that postmenopausal women had a higher atherogenic index. The features of the participants in this study were similar to those of the current study participants.

Tiwari J, Naagar JK [61] conducted a cross-sectional investigation and discovered that the features of the participants in this study were similar to those of the current study participants.

Dyslipidaemia in 2 groups:

Prevalence of dyslipidaemia was substantially greater in the post-menopausal group (81%), compared to the pre-menopausal group (64.4%), with a p-value of 0.020.

Dipak Kumar Adak et al. [54] conducted a cross-sectional study and discovered that more than 82% were with dyslipidaemia. Both may attest to this. Furthermore, postmenopause were with more dyslipidaemia than premenopausal women (80.2%). The features of the participants in this study were similar to those of the current study participants.

B Sudhakar Babu et al. [55] conducted a cross-sectional investigation and discovered that the features of the participants in this study were similar to those of the current study participants.

Nkeunen Gerard et al. [56] conducted a cross-sectional investigation and found that 123 (53%) of the 232 pre- and postmenopausal women had dyslipidaemia. It was more common in postmenopause than premenopause, with significant dissimilarity. The total dyslipidemic pattern in 2 was 37.9% hypercholesterolaemia, 33.6% LDL hypercholesterolaemia, 22.4% HDL hypocholesterolemia, 4.7% hypertriglyceridemia. The prevalence of HDL hypocholesterolemia was more in postmenopause 26.7% than in premenopause 18.9%, although the difference was not significant. The occurrence of hypercholesterolaemia, LDL hypercholesterolaemia, and hypertriglyceridemia was statistically same across 2 groups. The features of the participants in this study were similar to those of the current study participants.

Ranjit Kumar's [60] cross-sectional study found that menopausal women had significantly higher TC and LDL-C than other. Postmenopause had more but nonsignificant TGs than others. The postmenopausal group showed significantly lower HDL levels ($p < 0.001$) compared to the premenopausal group. The features of the participants in this study were similar to those of the current study participants.

A cross-sectional study by Tiwari J, Naagar JK [61] found that post-menopausal women have considerably increased levels of total cholesterol, LDL, and triglycerides ($p < 0.001$). The features of the participants in this study were similar to those of the current study participants.

Table 18: Dyslipidemia

Current study	Dyslipidaemia was substantially greater in post-menopause (81%), than pre-menopause (64.4%) ($p = 0.020$).
Dipak Kumar Adak, et al. [54]	Postmenopausal women (82.7%) showed a greater prevalence of dyslipidaemia than premenopausal women (80.2%).
Nkeunen Gerard, et al., [56]	It was more common in postmenopause 61% Pre-menopause 45.6% $p\text{-value} = 0.03$

Strengths of the study:

1. The study addresses an important health issue, as lipid profile changes during menopause are connected to more risk of CVD.
2. By comparing pre- and post-menopausal women, the study provides insights into hormonal influences on lipid metabolism.
3. The study captures data at specific spot in time, allowing for an efficient comparison between the two groups.
4. Conducted at tertiary center, the study benefits from access to a diverse patient population and standardized diagnostic facilities.

5. The use of lipid profile measurements (e.g., total cholesterol, LDL, HDL, triglycerides) ensures an objective evaluation of metabolic changes.
6. Findings from the study could help in early recognition of women at risk for dyslipidemia and CVD, leading to better preventive care.
7. The study adds to existing literature on menopause-related lipid changes and may guide future research and clinical guidelines.

Limitations of the study:

1. This design confines its capacity in not showing causal links between menopause and lipid profile changes.
2. The study, done at a tertiary care centre, may not be representative of the general community due to the inclusion of patients with pre-existing health issues.
3. The study does not track lipid profile changes over time, making it difficult to assess long-term trends and variations.
4. As the study has a limited number of participants, the findings may lack statistical power and generalizability.
5. As menopausal status was determined through self-report rather than hormonal assessment, there is a possibility of misclassification.
6. Lipid profiles can vary based on ethnicity, geographic location, and lifestyle factors, which might frontier the applicability of the findings to a larger population.
7. The study focused only on lipid profile and did not consider other metabolic markers such as insulin resistance, inflammatory markers, or blood pressure, which are also relevant in post-menopausal health.

CONCLUSION

This cross-sectional investigation in lipid characteristics across pre and postmenopause at tertiary care centre reveals significant alterations in lipid metabolism related with the menopause transition. The data show that postmenopausal women have greater levels of total cholesterol, low-density lipoprotein (LDL), and triglycerides, as well as lower levels of high-density lipoprotein (HDL) than their pre-menopausal counterparts. These changes in lipid markers indicate an increased risk of atherosclerosis, cardiovascular disease (CVD), and metabolic problems in postmenopausal women.

The observed changes can be accredited to **hormonal fluctuations, chiefly the turn down in estrogen levels** during menopause, which plays a crucial role in lipid metabolism regulation. Estrogen is known to have cardioprotective effects by enhancing HDL levels and promoting the clearance of LDL and triglycerides. With its reduction, there is an increased predisposition to **dyslipidemia, obesity, insulin resistance, and other metabolic abnormalities**, further exacerbating cardiovascular risk.

Given the growing prevalence of CVD among postmenopausal women, this study emphasises the importance of early screening, frequent lipid profile monitoring, and timely therapies to reduce the related risks. Adopting lifestyle changes such as a heart-healthy diet, frequent exercise, weight control, and quitting smoking is critical in preventing dyslipidaemia. In high-risk patients, pharmaceutical therapies, such as cholesterol-lowering medications, may be required to maintain appropriate lipid levels.

While this study provides useful insights, more longitudinal studies with bigger sample numbers and various populations are needed to determine the causal links between menopause and lipid profile changes. Furthermore, investigating additional metabolic indicators, inflammatory markers, and genetic predispositions would provide a more complete picture of cardiovascular risks in postmenopausal women.

Finally, the study emphasises the need of proactive cardiovascular risk management in postmenopausal women, arguing for early detection and preventative methods to promote long-term cardiovascular health and general well-being.

BIBLIOGRAPHY

1. Kochanek KD, Xu JQ, Murphy SL, Miniño AM, Kung HC. Deaths: final data for 2009. *Natl Vital Stat Rep.* 2011;60(3).
2. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart disease and stroke statistics – 2011 update. A report from the American Heart Association. *Circulation.* 2011;123(4):e18–e209.
3. Lerner DJ, Kannel WB. Patterns of coronary heart disease morbidity and mortality in the sexes: a 26-year follow-up of the Framingham population. *Am Heart J.* 1986;111(2):383–390.
4. Keys A, Menotti A, Aravanis C, et al. The seven countries study: 2,289 deaths in 15 years. *Prev Med.* 1984;13(2):141–154.
5. Kannel WB, Castelli WP, Gordon T. Cholesterol in the prediction of atherosclerotic disease. *Ann Intern Med.* 1979;90(1):85.
6. Sempos CT, Cleeman JI, Carroll MD, et al. Prevalence of high blood cholesterol among US adults: an update based on guidelines from the Second Report of the National Cholesterol Education Program Adult Treatment Panel. *JAMA.* 1993;269(23):3009–3014.
7. Mostaghel E, Waters D. Women do benefit from lipid lowering: latest clinical trial data. *Cardiol Rev.* 2003;11(1):4–12.
8. [No authors listed]. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation.* 2002;106(25):3143–3421.

9. Genest J, McPherson R, Frohlich J, et al. 2009 Canadian Cardiovascular Society/Canadian guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in the adult – 2009 recommendations. *Can J Cardiol.* 2009;25(10):567–579.
10. European Association for Cardiovascular Prevention and Rehabilitation, Reiner Z, Catapano AL, De Backer G, et al; ESC Committee for Practice Guidelines (CPG) 2008–2010 and 2010–2012 Committees. ESC/EAS Guidelines for the management of dyslipidaemias: the Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). *Eur Heart J.* 2011;32(14):1769–1818.
11. Clarke R, Peden JF, Hopewell JC, et al. Genetic variants associated with Lp(a) lipoprotein level and coronary disease. *N Engl J Med.* 2009;361(26):2518–2528.
12. Emerging Risk Factors Collaboration, Erqou S, Kaptoge S, Perry PL, et al. Lipoprotein(a) concentration and the risk of coronary heart disease, stroke, and nonvascular mortality. *JAMA.* 2009;302(4):412–423.
13. Bays HE, Toth PP, Kris-Etherton PM, et al. Obesity, adiposity, and dyslipidemia: a consensus statement from the National Lipid Association. *J Clin Lipidol.* 2013;7(4):304–383.
14. Mumford SL, Dasharathy S, Pollack AZ, Schisterman EF. Variations in lipid levels according to menstrual cycle phase: clinical implications. *Clin Lipidol* 2011;6: 225–34.
15. Momeni Z, et al. 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. *Contracept Reprod Med* 2020;5.

16. Hashemi SJ, et al. Association between oral contraceptives with lipid profile: results from Hoveyzeh cohort study (HCS). *BMC Womens Health* 2023;23.
17. Silva-Bermudez LS, et al. Effects of oral contraceptives on metabolic parameters in adult premenopausal women: a meta-analysis. *Endocr Connect* 2020;9:978–98.
18. Kalkhoff RK. Metabolic effects of progesterone. *Am J Obstet Gynecol* 1982;142:735–8.
19. Wiznitzer A, et al. Association of lipid levels during gestation with preeclampsia and gestational diabetes mellitus: a population-based study. *Am J Obstet Gynecol* 2009;201. <https://doi.org/10.1016/j.ajog.2009.05.032>. 482.e1-482.e8 Preprint at.
20. Grimes SB, Wild R. Effect of pregnancy on lipid metabolism and lipoprotein levels. MDText.com, Inc.; 2018.
21. Loke DF, et al. Lipid profiles during and after normal pregnancy. *Gynecol Obstet Invest* 1991;32:144–7.
22. Vrijkotte TGM, et al. Maternal lipid profile during early pregnancy and pregnancy complications and outcomes: the ABCD study. *J Clin Endocrinol Metab* 2012;97:3917–25.
23. Herrera E. Lipid metabolism in pregnancy and its consequences in the fetus and newborn. *Endocrine* 2002;19:43–55.
24. Hadden DR, McLaughlin C. Normal and abnormal maternal metabolism during pregnancy. *Semin Fetal Neonatal Med* 2009;14:66–71.
25. Wild R, Weedin EA, Wilson D. Dyslipidemia in Pregnancy. *Endocrinol Metab Clin North Am* 2016;45:55–63.
26. Magnussen EB, et al. Prepregnancy cardiovascular risk factors as predictors of pre-eclampsia: population based cohort study. *BMJ* 2007;335:978.

27. Egeland GM, et al. Preconception cardiovascular risk factor differences between gestational hypertension and preeclampsia: cohort Norway study. *Hypertension* 2016;67:1173–80.
28. Leslie MS, Briggs LA. Preeclampsia and the risk of future vascular disease and mortality: a review. *J Midwifery Womens Health* 2016;61:315–24.
29. Jacobson TA, et al. National lipid association recommendations for patientcentered management of dyslipidemia: part 1—full report. *J Clin Lipidol* 2015;9: 129–69. <https://doi.org/10.1016/j.jacl.2015.02.003>. Preprint at.
30. Grundy SM, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: a report of the american college of cardiology/american heart association task force on clinical practice guidelines. *Circulation* 2019;139:e1082–143.
31. Gopal DJ, et al. Screening for hyperlipidemia in pregnant women: an underutilized opportunity for early risk assessment. *J Am Coll Cardiol* 2019;73: 14.
32. Ram KT, et al. Duration of lactation is associated with lower prevalence of the metabolic syndrome in midlife—SWAN, the study of women’s health across the nation. *Am J Obstet Gynecol* 2008;198. 268.e1–6.
33. Tørris C, et al. Duration of lactation, maternal metabolic profile, and body composition in the Norwegian EBBA I-study. *Breastfeed Med* 2013;8:8–15.
34. Wiklund P, et al. Prolonged breast-feeding protects mothers from later-life obesity and related cardio-metabolic disorders. *Public Health Nutr* 2012;15:67–74.
35. Jensen J, Nilas L, Christiansen C. Influence of menopause on serum lipids and lipoproteins. *Maturitas* 1990;12:321–31.
36. Coylewright M, Reckelhoff JF, Ouyang P. Menopause and hypertension: an ageold debate. *Hypertension* 2008;51:952–9.

37. Gustavson LE, Benet LZ. Menopause: pharmacodynamics and pharmacokinetics. *Exp Gerontol* 1994;29:437–44.
38. Kafonek SD. Postmenopausal hormone replacement therapy and cardiovascular risk reduction. A review. *Drugs* 1994;47(Suppl 2):16–24.
39. Prabakaran S, Schwartz A, Lundberg G. Cardiovascular risk in menopausal women and our evolving understanding of menopausal hormone therapy: risks, benefits, and current guidelines for use. *Ther Adv Endocrinol Metab* 2021;12:20420188211013916.
40. Manson JE, et al. Estrogen plus progestin and the risk of coronary heart disease. *N Engl J Med* 2003;349:523–34.
41. Mosca L, et al. Effectiveness-based guidelines for the prevention of cardiovascular disease in women—2011 update. *J Am Coll Cardiol* 2011;57:1404–23. <https://doi.org/10.1016/j.jacc.2011.02.005>. Preprint at.
42. Lacaze P, et al. Aspirin for primary prevention of cardiovascular events in relation to lipoprotein(a) genotypes. *J Am Coll Cardiol* 2022;80:1287–98.
43. Azziz R, et al. The prevalence and features of the polycystic ovary syndrome in an unselected population. *J Clin Endocrinol Metab* 2004;89:2745–9.
44. Guan C, et al. Polycystic ovary syndrome: a “risk-enhancing” factor for cardiovascular disease. *Fertil Steril* 2022;117:924–35. <https://doi.org/10.1016/j.fertnstert.2022.03.009>. Preprint at.
45. Calderon-Margalit R, et al. Prospective association of polycystic ovary syndrome with coronary artery calcification and carotid-intima-media thickness: the coronary artery risk development in young adults women’s study. *Arterioscler Thromb Vasc Biol* 2014;34:2688–94.

46. Singh A, et al. Familial hypercholesterolemia among young adults with myocardial infarction. *J Am Coll Cardiol* 2019;73:2439–50. <https://doi.org/10.1016/j.jacc.2019.02.059>. Preprint at.
47. Virani SS, Smith SC, Stone NJ, Grundy SM. Secondary prevention for atherosclerotic cardiovascular disease. *Circulation* 2020;141:1121–3. <https://doi.org/10.1161/circulationaha.119.044282>. Preprint at.
48. Ahmad Z, et al. Premature coronary heart disease and autosomal dominant hypercholesterolemia: increased risk in women with LDLR mutations. *J Clin Lipidol* 2016;10:101–8. <https://doi.org/10.1016/j.jacl.2015.09.003>. Preprint at.
49. Krogh HW, Mundal L, Holven KB, Retterstøl K. Patients with familial hypercholesterolaemia are characterized by presence of cardiovascular disease at the time of death. *Eur Heart J* 2016;37:1398–405.
50. Shlipak MG, Simon JA, Vittinghoff E, et al. Estrogen and progestin, lipoprotein(a), and the risk of recurrent coronary heart disease events after menopause. *JAMA*. 2000;283(14):1845–1852.
51. Yusuf S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet* 2004;364:937–52.
52. Mora S, et al. Statins for the primary prevention of cardiovascular events in women with elevated high-sensitivity C-reactive protein or dyslipidemia: results from the justification for the use of Statins in prevention: an intervention trial evaluating rosuvastatin (JUPITER) and meta-analysis of women from primary prevention trials. *Circulation* 2010;121:1069–77.

53. Committee Writing, et al. 2022 ACC expert consensus decision pathway on the role of nonstatin therapies for LDL-cholesterol lowering in the management of atherosclerotic cardiovascular disease risk: a report of the American college of cardiology solution set oversight committee. *J Am Coll Cardiol* 2022;80: 1366–418.
54. Adak, D. K., Bharali, N., Biswas, S., Bagchi, N. K., Biswas, T. K., & Rao, V. R. (2024). Dyslipidemia in pre- and post-menopausal women: a study of peri-urban Paundra Kshatriya women. *Anthropological Review*, 86(4), 13–23.
55. B Sudhakar Babu, G Aparna, C Niranjan Paul. Comparative assessment of serum lipid profile levels between premenopausal and postmenopausal healthy woman- A case control study. *MedPulse International Journal of Community Medicine*. April 2020; 14(1): 13-16.
56. Gerard, Nkeunen, Tsafack Takadong Julie Judith, Simo Gustave, Bertrand Ayuk Tambe, Ayangma Celestin Roger, and Tume Christopher Bonglavnyuy. 2019. “Lipid Profile and Prevalence of Dyslipidemia in Pre- and Postmenopausal Women in Yaounde, Cameroon”. *International Journal of Biochemistry Research & Review* 25 (4):1-11.
57. Manafa, Patrick & C, Agusi & C, Onyejekwe & Okeke, Chizoba & C, I. (2015). Comparative assessment of lipid profile in pre menopausal and menopausal women in Nnewi, Nigeria. *European Scientific journal*. 11. 1857- 7881.
58. Shilpa Joshi, Pradnya J Phalak, Anita Deshmukh. A comparative study of Lipid profile in pre-menopausal and postmenopausal women to assess the risk factors for Cardiovascular diseases. *European Journal of Cardiovascular Medicine*. 2024;14(2):650-653.

59. Khanduker, S., Ahmed, R., Nazneen, M., Alam, A., & Khondokar, F. (2018). A Comparative Study of Lipid Profile and Atherogenic Index of Plasma Among the Pre and Post-Menopausal Women. *Anwer Khan Modern Medical College Journal*, 9(1), 44–49.
60. Ranjit Kumar. Assessment of lipid profile in pre and post menopausal women *Journal of Advanced Medical and Dental Sciences Research*.2018;6(4):171-173.
61. Tiwari J, Naagar JK. Changes in serum lipid profile in postmenopausal women with reference to Body mass index (BMI). *Int J Med Res Rev* 2015;3(5):456-460.

ANNEXURE – I - INFORMED CONSENT FORM

“A COMPARATIVE STUDY OF LIPID PROFILE AMONG THE PRE AND
POST-MENOPAUSAL WOMEN A CROSS SECTIONAL STUDY ”

Name of Student/Principal Investigator:

Name of Guide/Co Investigators:

1.1 Introduction: Menopause is defined as the cessation of menstruation for more than a year and begins with changes in ovarian function¹.

1. The median age for the final menstrual period is about 51 years, when the ovarian follicular reserve and indeed oestrogen production is significantly reduced.^{2,3}
2. A woman from her intrauterine life till the end experiences different stages of reproductive life under the influence of female hormones, which is a physiological process.⁴
3. The sex hormones which are secreted in minute quantities not only play an important role in woman, reproductive life but also influence metabolism in a significant way. Specially Estrogen plays important role in lipid metabolism. They affect mainly serum cholesterol and lipoproteins and hence has an indirect role in coronary heart disease (CAD).⁵
4. During the menopausal transition, more erratic fluctuations in female reproductive hormones are seen.⁶
5. The hormonal changes associated with menopause are low plasma levels of estrogen and marked increase in leutinizing hormone (LH) 3 to 5 fold and follicle stimulating hormones (FSH) 10-20 folds.⁷

6. Decreased oestrogen during and after menopause causes structural, physiological and biochemical changes that alter the general health status of women. Lack of the protection of estrogen influences the risk of cardiovascular diseases (CVD), such as ageing, increased obesity or android pattern of body fat distribution, decreasing resting metabolic rate and physical activity.⁸
7. Lipid profile consists of a group of biochemical tests often used in predicting diagnosing and treating lipid-related disorders including atherosclerosis.^{9,10}
8. Increased levels of cholesterol, triglycerides, LDL, apolipoprotein B and decreased levels of HDL and apolipoprotein A are characteristics of lipid profile in menopause.¹¹
9. During menopause, concentration of triglyceride also increases, which is related to the increase of the abdominal fat count and insulin resistance. Menopause causes decrease of HDL concentration and changes in HDL structure which is inversely proportional with the abdominal fat level.¹²
10. This group of women is also at high risk of CVD¹³, but it is yet unclear whether increase in risk is caused by increased androgen level or decreased estrogen level.¹⁴
11. Up to 50 years prevalence of CAD is lower in woman than man but due to the hormonal changes it increases after menopause.¹⁵
12. Atherogenic index of plasma (AIP) calculated as $\log(TG/HDL-C)$ has been used to assess the cardiovascular risk.

Dear Mr./Mrs./Dr. _____, you are kindly requested to enrol yourself in a research study titled “ **A Comparative Study of Lipid Profile Among the Pre and Post-Menopausal Women A Cross Sectional Study** being conducted by Dr. _____ a post graduate student in M.D. General Medicine and the study will be carried out under the direct supervision and guidance of Dr. _____, Associate Professor, Department of General Medicine, Jawaharlal Nehru Medical College, Belgaum.

You have been requested to participate in this as you fit into the laid out criteria for a study ‘subject’/ participant.

Your participation in study is voluntary. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

Explanation of procedure: If you agree to enrol yourself in my study, you will be interviewed regarding your present, past and family history then you will be clinically examined in detail and investigated accordingly Fasting Lipid Profile, Ppbs, Hba1c.

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: By taking part in this study, clinical features, laboratory findings and **CARDIOVASCULAR RISK** in

POSTMENOPAUSAL WOMEN will be better understood **You will not** get any benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the **principal investigator / Participant.** (Strike out which is not applicable)

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact:

“Name of student/PI DR. _____, mobile number _____, email _____ If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study “**A Comparative Study of Lipid Profile Among the Pre and Post-Menopausal Women**”. My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE – II - PROFORMA

CASE NO	
NAME	
IP NO	
AGE	YEARS
SEX	FEMALE
ADDRESS	
OCCUPATION	

Complaints at presentation	
Past history	
Family history	
Personal history	
Treatment history	
MENSTRUAL HISTORY:	
AGE	
WEIGHT	
HEIGHT	
LIFETSYLE	

Vitals :

Temperature	
Pulse	
Respiratory rate	
Blood pressure	

PHYSICAL EXAMINATION:

	Yes	No
Pallor		
Icterus		
Lymphadenopathy		
Cyanosis		
Clubbing		
Edema		

SYSTEMIC EXAMINATION:

C.V.S	
R.S.	
C.N.S	
PER ABDOMEN	

INVESTIGATIONS:

Hemoglobin		FBS		Albumin	
Total Count		S.TSH		SGOT	
Neutrophils		CHOLESTEROL		SGPT	
Lymphocytes		LDL		S.CREATININE	
Eosinophils		HDL		NA	
Monocytes		TGS		K	
Basophils		T.BILIRUBIN			
		D.BILIRUBIN			
		Total protein			

ANNEXURE – III

MASTER CHART

Serial number	OP number	AGE in Years	CHOLESTEROL (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	TGS (mg/dL)	AIP	MENTRUAL HISTORY	BMI(kg/m2))	SBP (mmHg)	DBP (mmHg)	HB (g/dL)
1	7310905	25	149	95	51	60	0.07	PRE	21	132	82	10
2	7310890	46	198	144	43	85	0.30	PRE	24	100	72	11.2
3	7314619	27	181	121	54	87	0.21	PRE	22	110	70	10.4
4	7315989	36	168	120	46	106	0.36	PRE	20	130	84	9.2
5	7343454	26	215	168	54	122	0.35	PRE	21	118	72	12
6	7338339	43	214	178	43	104	0.38	PRE	21.8	120	70	10.8
7	7343497	35	149	115	35	85	0.39	PRE	24.5	132	74	10.2
8	7377318	48	199	139	50	118	0.37	PRE	19.8	100	68	8
9	7494795	42	246	182	69	115	0.22	PRE	21.9	128	78	11
10	7490774	46	194	134	39	310	0.90	PRE	22	134	80	11.2
11	7492003	40	167	104	42	195	0.67	PRE	26	130	78	10.6
12	7386443	47	206	128	38	388	1.01	PRE	20.7	132	88	10.8
13	7384766	38	121	91	37	82	0.35	PRE	21	128	80	12.9
14	7491960	49	158	102	38	263	0.84	PRE	22.8	90	60	12
15	7398141	45	186	135	43	217	0.70	PRE	21	138	84	11.4
16	7494556	42	150	110	40	72	0.26	PRE	26.7	128	74	10
17	7399505	31	166	124	42	108	0.41	PRE	22	134	86	11
18	6899165	27	158	86	59	102	0.24	PRE	19.2	122	74	9.8
19	7493278	35	170	123	40	140	0.54	PRE	18.8	124	80	12
20	7399447	32	242	169	90	77	-0.07	PRE	23.4	128	82	10.8
21	7398218	45	233	185	53	169	0.50	PRE	25	112	70	10.8
22	7402245	40	173	119	36	288	0.90	PRE	29.6	120	80	10.9
23	7402345	38	128	98	38	56	0.17	PRE	24.8	130	80	12.9
24	7416744	26	147	99	48	81	0.23	PRE	22.5	132	70	11.4
25	7479516	28	159	109	44	133	0.48	PRE	23	134	72	11.2
26	7420217	27	170	126	44	133	0.48	PRE	19.8	110	70	9.8
27	7381542	20	140	93	50	56	0.05	PRE	25	90	60	8
28	7502761	32	166	102	51	104	0.31	PRE	22.4	100	72	10.2
29	7387764	34	231	185	39	152	0.59	PRE	27.8	112	70	8.7

30	7402271	39	228	181	53	127	0.38	PRE	23.6	134	72	10.4
31	7420959	43	203	169	33	148	0.65	PRE	21.9	120	70	10.8
32	7421041	35	152	108	45	87	0.29	PRE	19.6	124	80	9.2
33	7414931	43	147	103	51	80	0.20	PRE	27	100	68	10.4
34	7414830	50	152	112	39	119	0.48	PRE	20.7	130	78	10.8
35	7413345	37	198	126	71	81	0.06	PRE	20	128	80	11.4
36	7409606	24	174	135	47	103	0.34	PRE	19	128	82	10
37	6957000	26	133	86	30	92	0.49	PRE	21	118	72	11.4
38	7407697	45	185	114	64	168	0.42	PRE	24	134	86	12
39	7407737	46	144	81	67	82	0.09	PRE	22	138	84	13.4
40	7407920	30	164	124	53	63	0.08	PRE	20	130	84	11
41	7333160	36	150	100	43	90	0.32	PRE	21	132	88	11
42	7330211	43	199	130	31	354	1.06	PRE	21.8	132	82	12.4
43	7330263	46	267	155	96	183	0.28	PRE	24.5	120	80	12.1
44	7329246	36	157	111	36	117	0.51	PRE	19.8	122	74	13
45	7305310	43	119	71	37	136	0.57	PRE	21.9	128	74	11
46	7335477	30	127	92	34	97	0.46	PRE	22	128	78	10.8
47	7305323	19	118	70	34	100	0.47	PRE	26	130	80	9.2
48	7312196	33	184	120	37	149	0.60	PRE	20.7	132	74	11.2
49	7316395	27	172	107	55	83	0.18	PRE	21	132	70	11.2
50	7550307	45	232	167	56	161	0.46	PRE	22.8	134	80	12
51	7534519	46	191	145	40	118	0.47	PRE	21	110	70	10.2
52	7528716	46	200	149	45	138	0.49	PRE	26.7	110	70	9.8
53	7330735	33	102	80	44	101	0.36	PRE	22	90	60	10.8
54	7313443	49	200	132	59	144	0.39	PRE	19.2	90	60	10.2
55	7550307	45	140	88	34	150	0.64	PRE	18.8	100	72	12.9
56	7333425	44	181	117	65	71	0.04	PRE	23.4	100	72	13
57	3824674	33	178	90	70	65	-0.03	PRE	25	112	70	12
58	7420959	43	203	169	33	148	0.65	PRE	29.6	112	70	10.6
59	7403530	30	189	143	40	188	0.67	PRE	24.8	134	72	9.2
60	7144026	26	129	69	45	133	0.47	PRE	22.5	134	72	9.8
61	7515121	37	130	83	37	92	0.40	PRE	23	120	70	10.8
62	7552985	50	193	120	61	107	0.24	PRE	19.8	120	70	9
63	7560166	18	176	80	40	130	0.51	PRE	25	124	80	10.4
64	7571769	40	138	94	34	56	0.22	PRE	22.4	124	80	9
65	7568280	42	193	123	49	110	0.35	PRE	27.8	100	68	11.4
66	7571759	50	247	170	56	87	0.19	PRE	23.6	130	78	8
67	10013397	26	154	89	49	64	0.12	PRE	21.9	100	68	12.2
68	734801	26	151	90	60	49	-0.09	PRE	19.6	130	78	13
69	758024	43	203	169	33	148	0.65	PRE	27	128	80	11

70	758096	35	152	108	45	87	0.29	PRE	20.7	128	80	11
71	750825	43	147	103	51	80	0.20	PRE	20	128	82	11
72	729846	50	152	112	39	119	0.48	PRE	19	128	82	10.5
73	759545	37	198	126	71	81	0.06	PRE	18.8	118	72	12.1
74	895485	24	174	135	47	103	0.34	PRE	23.4	134	86	11
75	151514	26	133	86	30	92	0.49	PRE	25	118	72	11
76	968452	45	185	114	64	168	0.42	PRE	29.6	134	86	10.8
77	7598462	46	144	81	67	82	0.09	PRE	24.8	130	80	9.2
78	7598456	30	164	124	53	63	0.08	PRE	22.5	130	80	10.2
79	7859642	26	133	86	30	92	0.49	PRE	23	132	74	11
80	7396510	58	209	170	48	94	0.29	POST	19.8	132	74	10
81	7378949	53	207	152	65	66	0.01	POST	25	132	70	10.2
82	7395484	63	167	112	45	156	0.54	POST	22.4	132	70	9.8
83	7395281	50	131	92	46	44	0.20	POST	27.8	134	80	10.8
84	7392273	57	170	135	31	112	0.56	POST	23.6	134	80	10.2
85	7419354	52	257	213	54	102	0.28	POST	21.9	138	84	12
86	7420990	55	191	151	47	101	0.33	POST	19.6	138	84	10
87	7424077	58	129	92	31	138	0.65	POST	27	130	84	8.8
88	7422601	63	196	154	46	83	0.26	POST	20.7	130	84	8.6
89	7422630	58	270	233	36	189	0.72	POST	20	132	88	10
90	7490498	67	154	78	60	230	0.58	POST	19	132	88	11.2
91	7490828	58	129	79	35	229	0.82	POST	21	132	82	10
92	7492044	63	218	158	57	115	0.30	POST	24	132	82	11.2
93	7426040	60	219	156	62	117	0.28	POST	22	120	80	10.4
94	7425741	65	223	164	40	249	0.79	POST	20	120	80	9.2
95	7400992	55	154	100	47	126	0.43	POST	21	122	74	10
96	7349021	54	194	158	43	88	0.31	POST	21.8	122	74	10.8
97	7383086	60	121	63	50	163	0.51	POST	24.5	128	74	10.2
98	7492018	67	173	122	46	98	0.33	POST	19.8	128	74	8
99	7402233	67	197	147	63	76	0.08	POST	21.9	128	78	11
100	7335469	62	179	131	37	181	0.69	POST	22	128	78	8.2
101	7304198	76	191	134	38	126	0.52	POST	26	134	80	10.6

102	7300845	75	121	54	50	112	0.35	POST	20.7	110	70	10.8
103	7300756	58	192	128	54	92	0.23	POST	21	110	70	10.2
104	7343460	59	181	138	44	142	0.51	POST	22.8	90	60	11
105	7569110	53	189	141	50	76	0.18	POST	21	90	60	11.4
106	7403473	65	149	118	30	114	0.58	POST	26.7	100	72	10
107	7403399	59	157	118	38	121	0.50	POST	22	100	72	12
108	7406049	50	181	120	62	107	0.24	POST	19.2	112	70	9.8
109	7400899	64	131	87	43	100	0.37	POST	29.8	112	70	11
110	7522811	68	165	107	51	68	0.12	POST	21	134	72	9
111	7498614	55	161	89	60	97	0.21	POST	24	134	72	9.2
112	7425753	59	142	90	40	141	0.55	POST	22	120	70	8
113	7507571	57	120	65	20	165	0.92	POST	20	120	70	10.8
114	7498746	54	200	126	53	142	0.43	POST	21	124	80	9.2
115	7492097	53	189	141	50	76	0.18	POST	21.8	124	80	10
116	7396513	51	189	136	49	116	0.37	POST	24.5	100	68	10.9
117	7522803	62	208	147	54	95	0.25	POST	19.8	130	78	11
118	7501303	73	145	86	46	93	0.31	POST	21.9	100	68	10.2
119	7396530	50	171	120	51	97	0.28	POST	22	130	78	9.8
120	7497361	54	196	142	49	98	0.30	POST	26	128	80	10.8
121	7496119	61	143	90	35	209	0.78	POST	20.7	128	80	10.2
122	7494936	58	181	141	35	104	0.47	POST	21	128	82	12.2
123	7396772	56	185	143	40	121	0.48	POST	22.8	128	82	9
124	7569212	62	167	103	39	154	0.60	POST	21	118	72	9.8
125	7558048	69	252	164	53	273	0.71	POST	26.7	134	86	8.6
126	7568948	46	158	200	30	270	0.95	POST	22	118	72	8.2
127	7518856	50	199	134	38	228	0.78	POST	19.2	134	86	8.9
128	7384694	69	160	132	27	147	0.74	POST	18.8	130	80	10.8
129	7389118	52	146	112	38	80	0.32	POST	23.4	130	80	10.2
130	7552979	64	205	116	86	62	0.20	POST	25	132	74	10.4
131	7407498	58	222	166	66	98	0.17	POST	29.6	132	74	7.7
132	7501571	57	120	65	20	165	0.92	POST	24.8	132	70	7.8
133	7494950	54	163	105	35	276	0.90	POST	22.5	132	70	8.6
134	7399465	65	159	110	38	162	0.63	POST	23	134	80	8.8
135	7398058	60	156	83	66	133	0.30	POST	19.8	134	80	9
136	7338364	67	281	235	56	123	0.34	POST	25	138	84	10
137	7300799	53	182	115	56	85	0.18	POST	22.4	138	84	12
138	7337125	55	218	183	31	144	0.67	POST	27.8	130	84	11
139	7375668	63	158	107	38	155	0.61	POST	23.6	130	84	10.4
140	7312151	55	170	117	33	153	0.67	POST	21.9	132	88	11.4
141	7515667	66	101	51	35	131	0.57	POST	19.6	132	88	10

142	7372548	67	293	204	44	519	1.07	POST	27	132	82	11
143	7386528	54	261	198	54	173	0.51	POST	20.7	132	82	10.8
144	7416487	72	172	121	47	96	0.31	POST	20	120	80	9.2
145	7337159	64	299	254	60	154	0.41	POST	19	120	80	10.9
146	7497279	71	212	148	64	91	0.15	POST	21	122	74	11.2
147	7522841	57	172	109	53	99	0.27	POST	24	122	74	10
148	7515042	62	207	129	61	144	0.37	POST	22	128	74	10.2
149	7399623	61	253	208	37	175	0.67	POST	20	128	74	9.8
150	7399465	65	159	110	38	162	0.63	POST	21	128	78	10.8
151	7409076	52	163	94	48	336	0.85	POST	21.8	128	78	10.2
152	7413353	54	221	142	68	148	0.34	POST	24.5	132	82	12
153	7568304	52	163	94	48	336	0.85	POST	19.8	100	72	10
154	7569034	56	261	198	54	173	0.51	POST	21.9	110	70	10.2
155	7598462	66	101	51	35	131	0.57	POST	22	130	84	10
156	7594622	67	293	204	44	519	1.07	POST	26	118	72	11
157	7598412	54	261	198	54	173	0.51	POST	20.7	120	70	10.8
158	7198456	72	172	121	47	96	0.31	POST	31	132	74	11