

Research Article

Study of Dyslipidemia in Pre- and Postmenopausal Diabetic Women

Dr.Sunkugari Sreeja^{1*}, Dr. Pochana Sindhu², Dr. Uma MA³, Dr. Arun Pandiyan M⁴, Dr. Kalyan Kumar⁵

^{1*}Postgraduate, Department of General Medicine, PESIMSR, Kuppam, Andhra Pradesh.

²Consultant, Rainbow children and mother hospital, Bengaluru, Karnataka.

³Professor and HOD, Department of General Medicine, PESIMSR, Kuppam, Andhra Pradesh.

⁴Assistant Professor, Department of General Medicine, Mahatma Gandhi Medical College and Research Institute, SBV, Pondicherry.

⁵Assistant Professor, Department of General Medicine, PESIMSR, Kuppam, Andhra Pradesh.

Corresponding Author: Dr.Sunkugari Sreeja

Postgraduate, Department of General Medicine, PESIMSR, Kuppam, Andhra Pradesh.

Email: sreejasunkugari@gmail.com

Received: 08.05.2026 Revised: 06.06.2026 Accepted: 04.07.2026

ABSTRACT

Background: Menopausal transition in women with type 2 diabetes mellitus (T2DM) is associated with metabolic changes that may adversely affect lipid profile and increase the risk of vascular complications. This study compared lipid abnormalities, glycaemic parameters, and diabetes-related complications between premenopausal and postmenopausal women with T2DM.

Methods: A cross-sectional observational study was conducted over 18 months among 120 women with T2DM (60 premenopausal and 60 postmenopausal) attending a tertiary care teaching hospital in Kuppam, Andhra Pradesh. Clinical evaluation, anthropometry, fasting lipid profile, fasting and postprandial blood glucose, glycated haemoglobin (HbA1c), urine analysis, fundus examination, and electrocardiography were performed. Data were analysed using independent-sample *t* test and chi-square test, with *p*<0.05 considered statistically significant.

Results: Postmenopausal women had a significantly longer duration of diabetes than premenopausal women (5.89±5.86 vs 3.46±3.08 years; *p*=0.011). LDL cholesterol (94.98±35.44 vs 81.05±32.82 mg/dL; *p*=0.027), VLDL cholesterol (44.11±21.92 vs 34.86±20.66 mg/dL; *p*=0.019), and TC/HDL ratio (5.96±1.71 vs 4.87±1.86; *p*=0.039) were significantly higher among postmenopausal women, whereas total cholesterol, triglycerides, and HDL cholesterol showed no significant differences. Nephropathy (16.7% vs 5.0%; *p*=0.047) and microvascular complications (31.7% vs 20.0%; *p*=0.016) were significantly more common in postmenopausal women.

Conclusions: Postmenopausal women with T2DM exhibited a more atherogenic lipid profile and a higher burden of diabetic complications than premenopausal women. Routine lipid screening and early management of dyslipidaemia and vascular complications may reduce long-term cardiovascular risk and improve clinical outcomes in this high-risk population.

Keywords: Type 2 Diabetes Mellitus, Menopause, Dyslipidaemia, Lipid Profile, LDL Cholesterol, VLDL Cholesterol, Diabetic Complications.

INTRODUCTION

Non-communicable diseases (NCDs) have emerged as the leading cause of mortality worldwide, accounting for more than 60% of all global deaths.^{1, 2} Once considered ailments of affluent societies, NCDs including diabetes, now pose a significant threat to low- and middle-income countries, with the majority of NCD related deaths occurring in these regions.^{1,3} India exemplifies this global shift, experiencing a dramatic rise in diabetes prevalence over recent decades.^{4,5}

India often referred to as the "diabetes capital of the world," faces a heterogeneous and rapidly escalating diabetes burden.^{4,6} Recent estimates indicate that the prevalence of

diabetes in India ranges from 6% to 12% in urban areas and 2% to 3% in rural regions, with significant regional and socioeconomic disparities.^{6,7} Urbanization, sedentary lifestyles, unhealthy dietary patterns, and high body mass index (BMI) are key modifiable risk factors fueling this epidemic—particularly in southern and western states such as Tamil Nadu, Goa, and Karnataka, which report the highest prevalence rates.^{4,7}

Amid the increasing prevalence of diabetes in India, certain subgroups of the population face distinct vulnerabilities. Notably, women undergoing the menopausal transition are at heightened risk. The interplay between menopause and diabetes is particularly

concerning, as fluctuations in estrogen and progesterone during this transition significantly impact glucose metabolism and insulin sensitivity, thereby increasing the risk of type 2 diabetes and metabolic syndrome.^{8, 9} The decline in estrogen levels after menopause contributes to increased central adiposity, adverse lipid profiles, and reduced insulin sensitivity, all of which are established risk factors for diabetes.^{9,10}

The menopausal transition also leads to a redistribution of body fat and significant changes in blood lipid levels (like cholesterol and triglycerides), which contribute to accelerated hardening of the arteries and heightened cardiovascular risk.¹⁰⁻¹⁴ These lipid abnormalities are generally more pronounced in postmenopausal diabetic women compared to pre-menopausal diabetic women.¹⁵⁻¹⁹ Such changes collectively lead to a significantly increased risk of cardiovascular diseases (CVD), including coronary artery disease and stroke, which remain leading causes of illness and death in this population.²⁰⁻²⁴

Understanding the distinct patterns and determinants of dyslipidemia in pre- and postmenopausal women with type 2 diabetes is crucial, as these differences may underlie the observed disparities in cardiovascular risk between these groups.²⁵ Identifying the specific impact of menopausal transition on lipid profiles can help in risk stratification and inform the development of tailored screening protocols for early detection of dyslipidemia in diabetic women.⁸ Given the rising prevalence of diabetes and the unique metabolic challenges faced by pre- and post-menopausal women, understanding the patterns and mechanisms of dyslipidemia in this population is essential. Such insights are critical to developing screening and treatment strategies aimed at reducing cardiovascular complications in diabetic women across menopausal stages.^{22,23}

Accordingly, this study aims to evaluate the lipid profile differences between pre- and postmenopausal women with type 2 diabetes mellitus, with a focus on identifying the specific impact of menopausal transition on dyslipidemia. By addressing these aims, the study seeks to provide a clearer understanding of how menopausal status influences lipid abnormalities in diabetic women, thereby informing clinical management and guiding targeted public health interventions for this high-risk population.

MATERIAL AND METHODS

Study design: This study was conducted as a cross-sectional observational study designed to assess dyslipidemia among premenopausal and postmenopausal women with type 2 diabetes mellitus.

Study setting: The study was carried out in the diabetic clinic of a tertiary care teaching hospital attached to a medical college located in Kuppam, Andhra Pradesh. All clinical evaluations, laboratory investigations, and data collection procedures were performed within the facilities of the same institution.

Study duration: The study was conducted over a period of 18 months, which included participant recruitment, data collection, laboratory investigations, and analysis.

Study population: The study population consisted of women diagnosed with type 2 diabetes mellitus attending the diabetic clinic. Both premenopausal and postmenopausal women who fulfilled the eligibility criteria were included in the study.

Inclusion and Exclusion Criteria: Women diagnosed with type 2 diabetes mellitus as per standard diagnostic criteria and belonging to either premenopausal or postmenopausal status were included in the study. Women who were receiving lipid-lowering medications, those who had undergone hysterectomy, and those on hormone replacement therapy were excluded from the study.

Sample size: A total sample size of 120 participants was included in the study. This comprised 60 premenopausal women with type 2 diabetes mellitus and 60 postmenopausal women with type 2 diabetes mellitus. The sample size was calculated based on previously published literature, considering the expected prevalence, precision, and confidence level.

Sampling technique: A purposive sampling technique was adopted for participant selection. Eligible women attending the diabetic clinic during the study period were screened consecutively. Those fulfilling the inclusion criteria were approached for participation. The sampling method ensured balanced representation of both premenopausal and postmenopausal diabetic women, with recruitment continuing until the required sample size for each group was achieved.

Study procedure: After obtaining ethical approval, eligible participants were identified during routine outpatient visits. Written informed consent was obtained from all participants prior to enrollment. A detailed clinical evaluation was performed, including recording of demographic details, medical

history, menstrual history, and treatment history. A thorough general physical examination and systemic examination were conducted for each participant. Blood pressure and vital parameters were recorded using standard methods. Participants were categorized into premenopausal and postmenopausal groups based on menstrual history. All procedures were carried out uniformly for both groups to ensure consistency.

Data collection: Data collection was carried out using a structured proforma. Sociodemographic details, personal history, family history and clinical characteristics were documented. Anthropometric measurements such as height and weight were recorded, and body mass index was calculated. After an overnight fasting period of at least eight hours, venous blood samples were collected under aseptic precautions. Samples were processed according to institutional laboratory protocols. Biochemical investigations included fasting blood sugar, postprandial blood sugar, fasting lipid profile, and glycosylated hemoglobin. Serum total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and very-low-density lipoprotein cholesterol were estimated. Additional evaluations such as urine analysis, fundus examination, and resting electrocardiography were performed as part of routine assessment. All collected data were recorded systematically in a predefined format.

Study tools: The tools used in the study included fasting blood sugar estimation, postprandial blood sugar estimation, fasting lipid profile assessment, glycosylated hemoglobin measurement, urine routine

examination, resting 12-lead electrocardiography, and blood pressure measurement.

Independent and Outcome Variables: Independent variables included age, menopausal status, duration of menopause, duration of diabetes, and clinical characteristics. Outcome variables included serum lipid parameters such as total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, very-low-density lipoprotein cholesterol, and glycosylated hemoglobin levels.

Ethical considerations: Ethical approval for the study was obtained from the Institutional Human Ethics Committee prior to initiation. Written informed consent was obtained from all participants. Confidentiality of participant information was maintained by assigning unique identification numbers, and personal identifiers were not disclosed at any stage. Participation was voluntary, and participants were free to withdraw from the study at any time without affecting their medical care.

Statistical analysis: The collected data were entered into a spreadsheet and analyzed using STATA version 14. Descriptive statistics were used to summarize the data. Categorical variables were expressed as frequencies and percentages, while continuous variables were summarized as mean and standard deviation. Inferential analysis was performed using appropriate statistical tests. Independent sample t-tests were applied to compare continuous variables between groups, and chi-square tests were used to assess associations between categorical variables. A p value less than 0.05 was considered statistically significant.

RESULTS

Table 1. Baseline Demographic and Lipid Profile Characteristics of Premenopausal and Postmenopausal Women with Type 2 Diabetes Mellitus

Variable		Premenopausal DM n (%)	Postmenopausal DM n (%)	P value
Age group (Years)	20-30	7 (11.7)	0 (0)	<0.001
	31-40	19 (31.7)	0 (0)	
	41-50	33 (55)	7 (11.7)	
	51-60	0 (0)	18 (30)	
	61-70 >70	0 (0) 1 (1.7)	25 (41.7) 10 (16.7)	
BMI range (kg/ m ²)	≤20.0	13 (21.7)	10 (16.7)	0.447
	21.0-22.0	22 (36.7)	24 (40)	
	23.0-24.9	23 (38.3)	26 (43.3)	
	≥25.0	2 (3.3)	0 (0)	
Total cholesterol	Borderline high Desirable High	9 (15.0) 47 (78.3) 4 (6.7)	7 (11.7) 51 (85.0) 2 (3.3)	0.583

Triglycerides	Borderline high High Optimal Very high	9 (15) 26 (43.3) 21 (35) 4 (6.7)	8 (13.3) 19 (31.7) 29 (48.3) 4 (6.7)	0.488
HDL	High Low Normal	1 (1.7) 43 (71.7) 16 (26.7)	1 (1.7) 41 (68.3) 18 (30)	0.921
LDL	Borderline high High Near Optimal Optimal Very high	9 (15) 0 (0) 13 (31.7) 31 (51.7) 1 (1.7)	5 (8.3) 1 (1.7) 11 (18.3) 43 (71.7) 0 (0)	0.012
VLDL	Elevated Normal	39 (65) 21 (35)	31 (51.7) 29 (48.3)	0.019

Table 1 compares the baseline demographic characteristics and categorical lipid profile parameters between premenopausal and postmenopausal women with type 2 diabetes mellitus. Postmenopausal women were significantly older than premenopausal women ($P<0.001$). No statistically significant differences were observed between the groups

with respect to BMI categories, total cholesterol, triglycerides, or HDL cholesterol categories. However, LDL cholesterol ($P=0.012$) and VLDL cholesterol ($P=0.019$) distributions differed significantly between the two groups, with postmenopausal women demonstrating a higher proportion of abnormal LDL and VLDL levels.

Table 2. Comparison of Clinical Characteristics, Glycaemic Parameters, and Lipid Profile between Premenopausal and Postmenopausal Women with Type 2 Diabetes Mellitus

Variable	Premenopausal Mean (SD)	Postmenopausal Mean (SD)	Mean difference	P value
Duration of diabetes	3.46 (3.08)	5.89 (5.86)	-2.43	0.011
Total cholesterol	161.50 (41.60)	164.70 (48.96)	-3.20	0.700
Triglycerides	200.33 (167.25)	243.18 (215.98)	-42.85	0.227
HDL	36.30 (10.44)	34.70 (11.26)	1.60	0.421
LDL	81.05 (32.82)	94.98 (35.44)	-13.93	0.027
VLDL	34.86 (20.66)	44.11 (21.92)	-9.25	0.019
TC/HDL ratio	4.87 (1.86)	5.96 (1.71)	-0.98	0.039
FBS	152.81 (53.83)	144.27 (55.24)	8.54	0.455
PPBS	241.08 (47.86)	195.76 (71.4)	45.31	0.008
HbA1C	11.43 (2.95)	9.87 (2.64)	1.56	0.042

Table 2 presents the comparison of clinical, glycaemic, and lipid profile parameters between premenopausal and postmenopausal women with type 2 diabetes mellitus. Postmenopausal women had a significantly longer duration of diabetes than premenopausal women ($P=0.011$). They also exhibited significantly higher LDL cholesterol ($P=0.027$), VLDL

cholesterol ($P=0.019$), and TC/HDL ratio ($P=0.039$). In contrast, premenopausal women had significantly higher postprandial blood sugar ($P=0.008$) and HbA1c levels ($P=0.042$). No significant differences were observed in fasting blood glucose, total cholesterol, triglycerides, or HDL cholesterol.

Table 3. Association of Body Mass Index Categories with Clinical and Biochemical Parameters among Premenopausal and Postmenopausal Women with Type 2 Diabetes Mellitus

Premenopausal and Postmenopausal Women with Type 2 Diabetes Mellitus				
Variable		Premenopausal Mean (SD)	Postmenopausal Mean (SD)	P value
Duration of diabetes	≤20.0	4.01 (3.73)	2.79 (2.84)	0.468
	21.0-22.0	4.11 (3.81)	4.95 (5.81)	0.614
	23.0-24.9	2.98 (2.20)	7.64 (6.19)	0.013
	≥25.0	1.10 (1.27)	NA	NA

Triglycerides	≤20.0	128.46 (91.32)	269.00 (222.38)	0.086
	21.0-22.0	239.14 (159.15)	237.54 (199.47)	0.976
	23.0-24.9	180.78 (153.78)	238.46 (235.29)	0.310
	≥25.0	465.50 (498.51)	NA	NA
HDL	≤20.0	39.31 (7.81)	33.60 (9.45)	0.140
	21.0-22.0	37.59 (12.57)	33.12 (11.46)	0.216
	23.0-24.9	34.13 (9.24)	36.58 (11.82)	0.421
	≥25.0	27.50 (9.19)	NA	NA
LDL	≤20.0	81.83 (26.60)	86.02 (32.57)	0.745
	21.0-22.0	85.45 (36.08)	96.42 (34.55)	0.299
	≥23.0	80.12 (37.92)	97.09 (38.01)	0.038
VLDL	≤20.0	36.06 (29.78)	54.20 (31.15)	0.174
	21.0-22.0	40.40 (18.21)	41.86 (19.53)	0.794
	≥23.0	29.85 (15.88)	42.32 (19.62)	0.018
TC/HDL ratio	≤20.0	4.32 (1.75)	4.90 (1.74)	0.441
	21.0-22.0	5.23 (1.90)	5.58 (1.77)	0.514
	23.0-24.9	4.71 (1.85)	5.86 (1.64)	0.047
	≥25.0	6.50 (1.70)	NA	NA

Table 3 demonstrates the association between BMI categories and selected clinical and biochemical parameters among premenopausal and postmenopausal women with type 2 diabetes mellitus. Increasing BMI was associated with a significantly longer duration of diabetes, higher LDL cholesterol, higher

VLDL cholesterol, and an increased TC/HDL ratio, particularly among postmenopausal women. Although triglyceride levels tended to increase and HDL cholesterol tended to decrease with increasing BMI, these differences did not reach statistical significance across all BMI categories.

Table 4: Correlation of BMI with Clinical and Biochemical Parameters in Premenopausal DM and Postmenopausal DM

Variable	Premenopausal IDM r	Premenopausal IDM p value	Postmenopausal IDM r	Postmenopausal IDM p value
Age	0.18	0.152	0.33	0.009
Diabetic (yrs)	-0.17	0.210	0.25	0.091
HbA1c	-0.32	0.012	-0.16	0.221
TC	0.01	0.950	0.02	0.910
TGL	0.25	0.248	-0.35	0.046
HDL	-0.24	0.358	0.31	0.039
LDL	0.05	0.700	0.31	0.097
VLDL	-0.23	0.084	-0.49	0.012
TC/HDL	0.12	0.380	-0.26	0.067
FBS	0.01	0.960	-0.06	0.690
PPBS	-0.09	0.610	-0.39	0.120

The correlation of BMI with clinical and biochemical parameters is presented using Pearson's correlation coefficient. In premenopausal DM, BMI showed a significant negative correlation with HbA1c ($r = -0.32$, $p = 0.012$), while other variables did not show statistically significant associations. In postmenopausal DM, BMI demonstrated a

significant positive correlation with age ($r = 0.33$, $p = 0.009$) and HDL ($r = 0.31$, $p = 0.039$), and a significant negative correlation with triglycerides ($r = -0.35$, $p = 0.046$) and VLDL ($r = -0.49$, $p = 0.012$). Other parameters did not show statistically significant correlations with BMI.

Table 5. Comparison of Diabetic Complications and Dyslipidaemia between Premenopausal and Postmenopausal Women with Type 2 Diabetes Mellitus

Variable		Premenopausal DM n (%)	Postmenopausal DM n (%)	P value
Urine protein-defined nephropathy	Absent	57 (95)	50 (83.3)	0.047
	Present	3 (5)	10 (16.7)	
Complications	No complications	48 (80)	41 (68.3)	0.016
	Microvascular	12 (20)	19 (31.7)	
Overall dyslipidemia	Absent	10 (20)	5 (10.6)	0.032
	Present	40 (80)	42 (89.4)	
Total cholesterol-defined dyslipidaemia	Absent	44 (88)	38 (80.8)	0.048
	Present	6 (12)	9 (19.2)	
Triglyceride-defined dyslipidemia	Absent	25 (50)	16 (34)	0.016
	Present	25 (50)	31 (66)	
HDL-defined dyslipidaemia	Absent	17 (34)	15 (31.9)	0.099
	Present	33 (66)	32 (68.1)	
LDL-defined dyslipidaemia	Absent	45 (90)	42 (89.4)	1.000
	Present	5 (10)	5 (10.6)	

Table 5 compares diabetic complications and dyslipidaemia between premenopausal and postmenopausal women. Postmenopausal women had a significantly higher prevalence of proteinuria suggestive of diabetic nephropathy ($P=0.047$) and microvascular complications ($P=0.016$). Overall dyslipidaemia ($P=0.032$),

total cholesterol-defined dyslipidaemia ($P=0.048$), and triglyceride-defined dyslipidaemia ($P=0.016$) were also significantly more common among postmenopausal women. However, no significant differences were observed in HDL-defined or LDL-defined dyslipidaemia between the groups.

Table 6. Association of Body Mass Index and Diabetic Complications among Premenopausal and Postmenopausal Women with Dyslipidaemia

Variable		Premenopausal DM with dyslipidemia n (%)	Postmenopausal DM with dyslipidemia n (%)	P value
BMI range	≤20.0	5 (12.5)	6 (14.3)	0.053
	21.0-22.0	15 (37.5)	17 (40.5)	
	23.0-24.9	18 (45)	19 (45.2)	
	≥25.0	2 (5)	0 (0)	
Complications	No complications'	32 (80)	23 (54.8)	0.023
	Microvascular	8 (20)	15 (35.7)	
	Macrovascular	0 (0)	4 (9.5)	

Table 6 presents the distribution of BMI categories and diabetic complications among premenopausal and postmenopausal women with dyslipidaemia. The BMI distribution was comparable between the two groups and did not differ significantly. However, diabetic

complications differed significantly ($P=0.023$), with postmenopausal women demonstrating a higher prevalence of both microvascular and macrovascular complications compared with premenopausal women.

Table 7. Comparison of HBA1c among Premenopausal and Postmenopausal Women with Dyslipidaemia

Variable	Premenopausal DM with dyslipidemia Mean (SD)	Postmenopausal DM with dyslipidemia Mean (SD)	P value
HBA1C	9.73 (2.72)	9.65 (2.50)	0.089

Table 7 compares the HbA1C between premenopausal and postmenopausal women with dyslipidaemia. Although the mean HbA1C was marginally higher among premenopausal women, the difference between the two groups was not statistically significant ($P=0.089$), indicating a comparable HbA1C levels among women with dyslipidaemia.

DISCUSSION

The present study compared lipid abnormalities and diabetes-related complications between premenopausal and postmenopausal women with type 2 diabetes mellitus (T2DM). The findings demonstrate that postmenopausal women exhibited a more adverse cardiometabolic profile, characterized by significantly higher LDL cholesterol, VLDL cholesterol, TC/HDL ratio, overall dyslipidaemia, and diabetes-related complications. These findings support the hypothesis that menopause, in conjunction with diabetes, accelerates atherogenic dyslipidaemia and increases cardiovascular risk.

Postmenopausal women had a significantly longer duration of diabetes than premenopausal women. Longer disease duration is an established determinant of vascular complications because prolonged exposure to hyperglycaemia promotes oxidative stress, endothelial dysfunction, chronic inflammation, and advanced glycation end-product formation. Similar observations have been reported by the UK Prospective Diabetes Study, which demonstrated that cumulative glycaemic exposure substantially increases the risk of microvascular and macrovascular complications.²⁵ The significantly longer duration of diabetes observed among postmenopausal women in the present study may therefore partly explain their greater burden of dyslipidaemia and vascular complications.

Although total cholesterol and triglyceride concentrations were numerically higher among postmenopausal women, these differences were not statistically significant. Comparable findings have been reported in several previous studies, suggesting that total cholesterol alone is an insensitive marker of cardiovascular risk in diabetic women because qualitative lipoprotein abnormalities may exist despite relatively normal cholesterol concentrations.²³ Instead, lipid fractions such as LDL, VLDL, and lipid ratios provide better estimates of atherogenic risk in patients with diabetes.

The most important finding of the present study was the significantly higher LDL cholesterol and VLDL cholesterol levels among postmenopausal women. Estrogen deficiency after menopause reduces hepatic LDL receptor activity, increases hepatic lipase activity, and promotes the formation of small dense LDL particles, thereby producing a highly atherogenic lipid profile.^{13,26} Similar findings have been described by Derby et al. in the Study of Women's Health Across the Nation (SWAN), where LDL cholesterol increased significantly during the menopausal transition independent of chronological ageing.²⁷ Fonseca et al. also demonstrated that postmenopausal diabetic women possess more atherogenic LDL subfractions despite comparable total LDL concentrations.²⁸ Our findings therefore reinforce the concept that menopausal transition independently contributes to adverse lipid alterations beyond the effects of diabetes alone.

The TC/HDL ratio was significantly higher among postmenopausal women, indicating greater cardiovascular risk. This ratio is considered a stronger predictor of future cardiovascular events than isolated lipid parameters because it reflects the balance between atherogenic and protective lipoproteins. Previous studies have similarly shown that increased TC/HDL and atherogenic indices are strongly associated with coronary artery disease among diabetic women.^{20,29} Although HDL concentrations were slightly lower in postmenopausal women, the difference was not statistically significant, suggesting that deterioration in cardiovascular risk was driven predominantly by elevated atherogenic lipoproteins rather than reductions in HDL alone.

BMI-specific analyses demonstrated that women with BMI ≥ 23 kg/m² had significantly higher LDL, VLDL, TC/HDL ratio, and longer duration of diabetes after menopause. These observations support previous evidence that central adiposity and insulin resistance increase substantially following menopause because of declining estrogen levels.²⁹ Increased visceral adiposity enhances hepatic very-low-density lipoprotein synthesis and worsens insulin resistance, thereby aggravating diabetic dyslipidaemia.³⁰ These findings emphasize that even modest increases in BMI among postmenopausal diabetic women warrant aggressive metabolic risk assessment.

Interestingly, premenopausal women exhibited significantly higher HbA1c and postprandial glucose levels than postmenopausal women.

This finding differs from several previous reports and may reflect differences in treatment adherence, disease awareness, healthcare utilisation, or therapeutic intensification among older women attending tertiary care centres. Despite relatively poorer glycaemic control in premenopausal women, postmenopausal women experienced greater dyslipidaemia and vascular complications, suggesting that estrogen deficiency contributes independently to cardiovascular risk beyond glycaemic status. Postmenopausal women demonstrated significantly higher frequencies of overall dyslipidaemia, diabetic nephropathy, and microvascular complications. The coexistence of diabetes and menopause accelerates endothelial dysfunction, vascular inflammation, oxidative stress, and atherosclerotic progression, thereby increasing susceptibility to both microvascular and macrovascular complications.^{20,28,29} Similar findings have been reported by Ozder, who observed a higher prevalence of dyslipidaemia among older diabetic women, particularly those with poor glycaemic control.³¹ Likewise, Pei et al. demonstrated stronger associations between postmenopausal status, atherogenic lipoproteins, and diabetic microvascular complications.³²

The strengths of the present study include the direct comparison of premenopausal and postmenopausal diabetic women using standardized biochemical measurements and comprehensive assessment of lipid abnormalities and diabetic complications. However, several limitations should be acknowledged. The cross-sectional design precludes causal inference, the study was conducted at a single tertiary-care centre with purposive sampling, and the relatively modest sample size may limit generalisability. Furthermore, advanced lipid biomarkers such as apolipoprotein B, lipoprotein(a), and small dense LDL particles were not evaluated.

CONCLUSIONS

In conclusion, postmenopausal women with T2DM demonstrate a more atherogenic lipid profile, characterised by significantly higher LDL cholesterol, VLDL cholesterol, TC/HDL ratio, and greater prevalence of dyslipidaemia and diabetic complications compared with premenopausal women. These findings highlight the importance of routine lipid screening, comprehensive cardiovascular risk assessment, and early initiation of intensive lifestyle and lipid-lowering interventions among

postmenopausal women with diabetes. Prospective multicentre studies with larger sample sizes are warranted to further clarify the independent contribution of menopause to diabetic dyslipidaemia and long-term cardiovascular outcomes

REFERENCES

1. Bloom DE, Cafiero ET, Jané-Llopis E, Abrahams-Gessel S, Bloom LR, Fathima S, et al. The global economic burden of non-communicable diseases. Geneva: World Economic Forum; 2011. Available from: https://www3.weforum.org/docs/EF_Harvard_HE_GlobalEconomicBurdenNonCommunicableDiseases_2011.pdf
2. NCD Alliance. Non-communicable diseases (NCDs) [Internet]. London: NCD Alliance; 2025 Jan 16 [cited 2026 Jul 3]. Available from: <https://ncdalliance.org/why-ncds/NCDs>
3. World Bank. Public documents [Internet]. Washington (DC): World Bank; [cited 2026 Jul 3]. Available from: <https://thedocs.worldbank.org>
4. Chauhan S, Khatib MN, Ballal S, Bansal P, Bhopte K, Gaidhane AM, et al. The rising burden of diabetes and state-wise variations in India: insights from the Global Burden of Disease Study 1990-2021 and projections to 2031. *Front Endocrinol (Lausanne)*. 2025; 16:1505143. doi:10.3389/fendo.2025.1505143.
5. Anjana RM, Deepa M, Pradeepa R, Mahanta J, Narain K, Das HK, et al. Prevalence of diabetes and prediabetes in 15 states of India: results from the ICMR-INDIAB population-based cross-sectional study. *Lancet Diabetes Endocrinol*. 2017;5(8):585-96. doi:10.1016/S2213-8587(17)30174-2.
6. Mohan V, Pradeepa R. Epidemiology of type 2 diabetes in India. *Indian J Ophthalmol*. 2021;69(11):2932-38. doi: 10.4103/ijo.ijo_1627_21.
7. Maiti S, Akhtar S, Upadhyay AK, Mohanty SK. Socioeconomic inequality in awareness, treatment and control of diabetes among adults in India: evidence from the National Family Health Survey (NFHS), 2019-2021. *Sci Rep*. 2023; 13:29978. doi:10.1038/s41598-023-29978-y.
8. Vaidya R, Pandey S, Srinivas M, Agashe S, Joshi J, Galvankar P, et al. Menopause and metabolic syndrome: a study of 498

- urban women from western India. *J Midlife Health.* 2010;1(2):63-69. doi:10.4103/0976-7800.76214.
9. K J, Ebenezer ED, Londhe V, Paul TV, Yadav B, Kekre AN. Prevalence of metabolic syndrome among postmenopausal women in South India. *Int J Reprod Contracept Obstet Gynecol.* 2018;7(6):2364-69. doi:10.18203/2320-1770.ijrcog20182351.
10. Medarametla S. Early menopause in type 2 diabetes: a study from a South Indian tertiary care centre. *J Clin Diagn Res.* 2015;9(8):OC08-OC11. doi:10.7860/JCDR/2015/14181.6628.
11. Lizcano F, Guzmán G. Estrogen deficiency and the origin of obesity during menopause. *Biomed Res Int.* 2014; 2014:757461. doi:10.1155/2014/757461.
12. Lovejoy JC, Champagne CM, de Jonge L, Xie H, Smith SR. Increased visceral fat and decreased energy expenditure during the menopausal transition. *Int J Obes (Lond).* 2008;32(6):949-58. doi:10.1038/ijo.2008.25.
13. D'Eon TM, Rogers NH, Stancheva ZS, Greenberg AS. Estradiol and the estradiol metabolite, 2-hydroxyestradiol, activate AMP-activated protein kinase in C2C12 myotubes. *Obesity (Silver Spring).* 2008;16(6):1284-88. doi:10.1038/oby.2008.50.
14. Matthews KA, Crawford SL, Chae CU, Everson-Rose SA, Sowers M, Sternfeld B, et al. Are changes in cardiovascular disease risk factors in midlife women due to chronological aging or to the menopausal transition? *N Engl J Med.* 2009;360(2):192-203. doi:10.1056/NEJMoa0806118.
15. Matthews KA, Crawford SL, Chae CU, et al. Are changes in cardiovascular disease risk factors in midlife women due to chronological aging or menopause? *J Am Coll Cardiol.* 2009;54:2366-73.
16. Paschou SA, Papanas N. Type 2 diabetes mellitus and menopausal hormone therapy: an update. *Diabetes Ther.* 2019;10(6):2313-20. doi:10.1007/s13300-019-00695-y.
17. Lambrinoudaki I, Christodoulakos G, Rizos D. Menopause and cardiometabolic disease. *Best Pract Res Clin Endocrinol Metab.* 2010;24(1):13-23. doi:10.1016/j.beem.2009.08.007.
18. Kim C, Edelstein SL, Crandall JP, Dabelea D, Kitabchi AE, Hamman RF, et al. Menopause and risk of diabetes in women: evidence and implications. *Ther Adv Endocrinol Metab.* 2010;1(1):7-14. doi:10.1177/2042018810384224.
19. Saha S, Kaur P, Das S, Hussain M, Parveen S, Saha S. Lipid profile in postmenopausal women with and without type 2 diabetes mellitus. *Indian J Clin Biochem.* 2011;26(1):76-79. doi:10.1007/s12291-010-0081-8.
20. Anagnostis P, Paschou SA, Katsiki N, Krikidis D, Lambrinoudaki I, Goulis DG. Menopausal hormone therapy and cardiovascular risk: where are we now? *Curr Vasc Pharmacol.* 2019;17(6):564-72. doi:10.2174/1570161116666180709095348.
21. Kannel WB. Menopause and risk of cardiovascular disease. *Ann Intern Med.* 1976;85(4):447-52. doi:10.7326/0003-4819-85-4-447.
22. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes. *Diabetes Care.* 2004;27(5):1047-53. doi:10.2337/diacare.27.5.1047.
23. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome. *Circulation.* 2005;112(17):2735-52. doi:10.1161/CIRCULATIONAHA.105.169404.
24. Howard BV, Robbins DC, Sievers ML, Lee ET, Rhoades D, Devereux RB, et al. LDL cholesterol as a strong predictor of coronary heart disease in diabetic individuals with insulin resistance and low LDL cholesterol. *Arterioscler Thromb Vasc Biol.* 2000;20(3):830-35. doi:10.1161/01.ATV.20.3.830.
25. King P, Peacock I, Donnelly R. The UK Prospective Diabetes Study (UKPDS): clinical and therapeutic implications for type 2 diabetes. *Br J Clin Pharmacol.* 1999;48(5):643-8. doi:10.1046/j.1365-2125.1999.00092.x.
26. Palmisano BT, Zhu L, Stafford JM. Estrogens in the regulation of liver lipid metabolism. In: Mauvais-Jarvis F, editor. *Adv Exp Med Biol.* 2017;1043:227-56. doi:10.1007/978-3-319-70178-3_12.
27. Derby CA, Crawford SL, Pasternak RC, Sowers M, Sternfeld B, Matthews KA. Lipid changes during the menopause transition in relation to age and weight: the Study of Women's Health Across the

- Nation. Am J Epidemiol. 2009;169(11):1352-61. doi:10.1093/aje/kwp043.
28. Fonseca MIH, da Silva IT, Ferreira SRG. Impact of menopause and diabetes on atherogenic lipid profile: is it worth analysing lipoprotein subfractions to assess cardiovascular risk in women? *Diabetol Metab Syndr.* 2017;9:22. doi:10.1186/s13098-017-0221-5.
 29. Carr MC. The emergence of the metabolic syndrome with menopause. *J Clin Endocrinol Metab.* 2003;88(6):2404-11. doi:10.1210/jc.2003-030242.
 30. Mooradian AD. Dyslipidemia in type 2 diabetes mellitus. *Nat Rev Endocrinol.* 2009;5(3):150-9. doi:10.1038/ncpendmet1066.
 31. Ozder A. Lipid profile abnormalities seen in type 2 diabetes mellitus patients in primary healthcare in Turkey: a cross-sectional study. *Lipids Health Dis.* 2014;13:183. doi:10.1186/1476-511X-13-183.
 32. Pei X, Yao X, Qi D, Yang Y, Fan S, Li Z. Apolipoprotein and menopausal status are significant influencing factors for diabetic retinopathy in women with type 2 diabetes mellitus. *Sci Rep.* 2025;15(1). doi:10.1038/s41598-025-93161-8.