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Original Research Article

Estimation of Lipid Profile and ECG Changes in Postmenopausal Women: A Prospective Observational Real-World Study

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Abstract

Background: Cardiometabolic risk rises across the menopausal transition, but real-world Indian data integrating lipid abnormalities with resting electrocardiographic findings are limited.

Aim: To estimate the lipid profile and ECG changes in postmenopausal women and to examine clinical and lipid correlates of ECG abnormalities.

Methods: This prospective observational real-world study analyzed 100 postmenopausal women evaluated at Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India, from 5 January 2019 to 30 March 2021. Fasting total cholesterol, triglycerides, HDL-C, LDL-C, non-HDL-C and lipid ratios were assessed with a standard 12-lead ECG. Women were stratified by ECG status and by <5 versus ≥5 years since menopause. Group comparisons, correlations, and multivariable logistic regression were performed.

Results: Mean age was 57.4 ± 6.1 years and median duration since menopause was 8.6 (IQR 2.8–13.1) years. Total cholesterol was 206.9 ± 30.9 mg/dL, LDL-C 125.8 ± 31.3 mg/dL, HDL-C 49.4 ± 8.3 mg/dL, and median triglycerides 160.6 (126.5–203.2) mg/dL. Any ECG abnormality occurred in 45.0%; ST–T abnormalities were present in 25.0%, an ECG-LVH pattern in 15.0%, axis deviation in 10.0%, conduction abnormalities in 8.0%, ectopy in 7.0%, and QTc ≥465 ms in 5.0%. Women with abnormal ECGs had higher LDL-C (139.5 ± 30.5 vs 114.5 ± 27.3 mg/dL), non-HDL-C (174.6 ± 31.9 vs 143.5 ± 31.8 mg/dL), systolic blood pressure (129.8 ± 10.6 vs 121.8 ± 12.2 mmHg), and QTc (442.6 ± 15.9 vs 426.5 ± 15.5 ms), with lower HDL-C (46.8 ± 6.5 vs 51.4 ± 9.1 mg/dL). Any ECG abnormality was present in 58.0% of women ≥5 years from menopause versus 16.1% within 5 years ($p<0.001$). In adjusted analysis, years since menopause (OR 1.16 per year, 95% CI 1.02–1.33; $p=0.024$) and LDL-C (OR 1.20 per 10 mg/dL, 95% CI 1.00–1.44; $p=0.044$) were independently associated with an abnormal ECG.

Conclusion: Increasing postmenopausal duration was associated with a more atherogenic lipid profile and greater ECG-abnormality burden. Combined assessment of lipids, blood pressure, reproductive history, and a resting ECG may aid pragmatic cardiovascular risk recognition in postmenopausal women.

Keywords: postmenopause; dyslipidemia; electrocardiography; LDL cholesterol; QTc; cardiovascular risk; India.

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Introduction

Cardiovascular disease becomes increasingly important in women after the final menstrual period, and the menopausal transition is now recognized as a clinically meaningful window during which several adverse cardiovascular risk factors accelerate.

The American Heart Association scientific statement on menopause and cardiovascular disease emphasized that the transition is accompanied by changes in body composition, blood pressure, glycemic regulation, vascular function, and lipoprotein metabolism, with implications for earlier preventive assessment in women [1]. This period is therefore not simply a reproductive milestone. It represents a cardiometabolic transition in which loss of ovarian function intersects with chronological aging, lifestyle, adiposity, and genetic susceptibility.

For clinicians working in routine medical settings, two inexpensive tests—the fasting lipid profile and

the standard 12-lead electrocardiogram (ECG)—offer complementary information: the lipid profile characterizes a major modifiable atherosclerotic risk pathway, whereas the ECG can identify electrical, repolarization, conduction, and hypertrophic patterns that may signal accumulated cardiovascular stress before overt clinical disease becomes apparent.

The biological plausibility for a postmenopausal shift toward higher cardiovascular risk is substantial. Declining estradiol alters hepatic lipid handling, adipose-tissue distribution, insulin sensitivity, endothelial signaling, and autonomic balance. Contemporary reviews describe menopause as a cardiometabolic transition characterized by redistribution toward visceral adiposity, a tendency toward insulin resistance, and an increasingly atherogenic lipid phenotype [2]. A recent comprehensive review of lipid metabolism in women further documents that the menopausal transition is commonly associated with increases in total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglycerides, together with changes in high-density lipoprotein cholesterol (HDL-C), HDL functionality, and lipoprotein(a) [3]. These alterations are important because LDL-containing apolipoprotein B particles remain causal mediators of atherosclerotic cardiovascular disease, while hypertriglyceridemia and low HDL-C often cluster with central adiposity and insulin resistance. In a postmenopausal population, therefore, a conventional lipid panel may reflect both reproductive aging and the broader metabolic milieu.

Longitudinal evidence has helped separate changes related to menopause from those attributable to aging alone. In the Study of Women's Health Across the Nation (SWAN), Matthews and colleagues showed that total cholesterol, LDL-C, and apolipoprotein B rose particularly around the late perimenopausal and early postmenopausal phases, supporting an effect of the menopausal transition beyond simple chronological aging [4]. More recent trajectory analyses have reinforced the concept that lipid patterns are heterogeneous across midlife, but that the final menstrual period remains an informative anchor for adverse changes in several lipid measures [5]. These observations are clinically relevant because a single postmenopausal lipid value is the visible consequence of a trajectory that may have accelerated over the preceding years. Duration since menopause may therefore serve as a useful, readily obtainable marker when interpreting lipid abnormalities in routine practice.

Importantly, LDL-C concentration alone does not fully describe the postmenopausal atherogenic phenotype. Changes in LDL particle distribution and composition have also been linked to subclinical atherosclerosis. El Khoudary and colleagues reported that LDL subclasses evolving across the menopausal transition were associated with later coronary artery calcification and carotid atherosclerosis, providing mechanistic support for a qualitative as well as quantitative change in atherogenic lipoproteins [6]. Clinical cohort work has similarly demonstrated a deterioration in the conventional lipid profile as women move from peri- to postmenopause [7]. This has practical consequences in resource-constrained settings, because direct lipoprotein particle testing is not routinely available; careful use of LDL-C, non-HDL-C, triglycerides, and cholesterol ratios can provide a pragmatic approximation of atherogenic burden.

Indian data are especially important because cardiometabolic risk often emerges at lower levels of generalized adiposity than in many Western populations, and access to advanced cardiovascular testing is uneven. An Indian comparative study reported higher total cholesterol, triglycerides, and LDL-C in postmenopausal women than in premenopausal women, alongside lower estradiol levels [8]. Other Indian clinical literature has highlighted the convergence of dyslipidemia, blood-pressure elevation, central adiposity, and menopause in shaping cardiovascular risk [9]. Yet much of the available regional literature has examined lipids in isolation or has compared premenopausal and postmenopausal groups without simultaneously characterizing resting ECG abnormalities. A combined assessment may be more clinically informative because biochemical and electrical phenotypes need not evolve independently.

Recent cardiovascular reviews have increasingly framed the menopause transition as a period for active risk recognition rather than passive observation. Uddenberg and colleagues summarized the accumulation of adverse cardiovascular risk factors across the transition and stressed the opportunity for preventive intervention [10]. Reproductive timing also appears relevant: pooled individual-participant data have linked earlier natural menopause with higher subsequent cardiovascular disease risk [11]. A 2024 review in Nature Reviews Cardiology similarly emphasized that both ovarian aging and conventional cardiovascular risk factors contribute to the rise in clinical and subclinical cardiovascular risk during the menopausal transition [12]. Together, these findings suggest that years since menopause, lipid abnormalities, blood pressure, and ECG findings should be considered within the same clinical framework rather than as unrelated observations.

The resting ECG provides an inexpensive window into several cardiovascular processes. In asymptomatic postmenopausal women from the Women's Health Initiative, both major and minor

ECG abnormalities were associated with future cardiovascular events and mortality, establishing that apparently modest ECG findings may carry prognostic information [13]. In a separate Women's Health Initiative analysis, repolarization and other ECG abnormalities predicted coronary heart disease events and coronary mortality in postmenopausal women [14]. Although a cross-sectional ECG cannot establish the presence or absence of coronary artery disease, patterns such as ST–T abnormalities, left ventricular hypertrophy (LVH), conduction disturbances, axis deviation, ectopy, and QT prolongation may reflect myocardial remodeling, blood-pressure burden, ischemic substrate, autonomic influences, or drug and electrolyte effects. Their presence in a population with an unfavorable lipid profile may therefore identify a subgroup deserving more detailed cardiovascular evaluation.

Menopause may also influence atrial and ventricular electrical behavior. Dogan and colleagues reported differences in P-wave parameters and cardiac repolarization indices according to menopausal status, suggesting that reproductive hormonal state can influence electrophysiologic measurements [15]. The QT interval is particularly relevant because it is modulated by sex hormones and autonomic tone. In postmenopausal women, Kadish and colleagues observed associations between estrogen/progestin use and QT interval characteristics [16]. A randomized trial evaluating vasomotor symptoms and repolarization further illustrated that cardiac repolarization in postmenopausal women is dynamic and potentially hormone-sensitive [17].

These studies do not imply that menopause alone causes clinically important arrhythmia, but they support systematic measurement of QT and corrected QT (QTc) when characterizing postmenopausal ECG patterns. Despite these advances, there remains a practical evidence gap in everyday Indian hospital populations. Many women undergo lipid testing and ECG recording during the same clinical encounter, yet the relationship between these two readily available assessments is often not formally analyzed. A real-world study that quantifies lipid abnormalities, describes the spectrum of resting ECG changes, compares women with and without ECG abnormalities, and examines whether years since menopause and lipid measures independently relate to an abnormal ECG can generate clinically actionable information. Such an approach is particularly relevant in tertiary centers serving mixed urban, semi-urban, and rural populations, where inexpensive risk stratification tools are valuable. The present prospective observational real-world study therefore evaluated the lipid profile and resting ECG findings in 100 postmenopausal women attending Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India, during 5 January 2019 to 30 March 2021. The objectives were to estimate the burden of conventional dyslipidemia and ECG abnormalities, compare biochemical and clinical characteristics according to ECG status, examine differences according to duration since menopause, and identify independent clinical and lipid correlates of a composite ECG abnormality. The underlying clinical question was whether an inexpensive combined lipid–ECG assessment could reveal a higher-risk postmenopausal phenotype that may otherwise remain unrecognized during routine care.

Materials and Methods

This prospective observational real-world study included 100 postmenopausal women evaluated at Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India, between 5 January 2019 and 30 March 2021. Postmenopause was defined clinically as at least 12 consecutive months of spontaneous amenorrhea not attributable to pregnancy, lactation, medication, or another obvious pathological cause. Women were evaluated consecutively in routine clinical care.

For the analytical framework, women with acute coronary syndrome or acute systemic illness at the index encounter, known persistent tachyarrhythmia preventing standard interval measurement, severe hepatic or renal dysfunction likely to materially alter lipid metabolism, untreated overt thyroid dysfunction, current menopausal hormone therapy, or triglycerides ≥400 mg/dL were excluded from lipid-ratio and calculated LDL analyses.

Demographic history included age, age at final menstrual period, and duration since menopause. Height and weight were used to calculate body mass index (BMI, kg/m²), waist circumference was measured at the midpoint between the lower rib and iliac crest, and seated blood pressure was recorded after rest using standard clinical technique. Venous blood was obtained after an overnight fast of approximately 8–12 hours.

Serum total cholesterol and triglycerides were measured by standard enzymatic colorimetric methods and HDL-C by a selective enzymatic method used in the institutional laboratory. Very-low-density lipoprotein cholesterol (VLDL-C) was estimated as triglycerides/5 when triglycerides were below 400 mg/dL, and LDL-C was estimated using the Friedewald relationship. Non-HDL-C was calculated as total cholesterol minus HDL-C. The total cholesterol/HDL-C and LDL-C/HDL-C ratios were calculated for every participant.

The atherogenic index of plasma was calculated as log10 of the molar triglyceride-to-HDL-C ratio. For descriptive categorization, total cholesterol ≥200

mg/dL, triglycerides ≥150 mg/dL, LDL-C ≥130 mg/dL, HDL-C <50 mg/dL, and non-HDL-C ≥160 mg/dL were treated as adverse lipid thresholds. These thresholds were used to describe the burden of dyslipidemia rather than to substitute for individualized guideline-based treatment decisions.

A resting 12-lead ECG was obtained at the index assessment at a standard paper speed of 25 mm/s and calibration of 10 mm/mV. Heart rate, PR interval, QRS duration, QT interval, and corrected QT interval (QTc) were recorded. QTc was calculated using Bazett correction and QTc ≥465 ms was categorized as prolonged for the descriptive analysis. ECGs were reviewed for ST–T abnormalities, an ECG pattern compatible with LVH, axis deviation, conduction abnormality, and premature atrial or ventricular beats/ectopy. A composite 'any ECG abnormality' outcome was defined as the presence of at least one of these abnormalities or prolonged QTc. The categories were non-mutually exclusive, so a participant could contribute to more than one specific ECG abnormality while being counted only once in the composite endpoint. To explore the temporal relationship with reproductive aging, participants were also stratified into <5 years and ≥5 years since menopause.

Statistical analysis was performed on the complete 100-participant analytical dataset. Continuous variables were summarized as mean ± standard deviation when approximately symmetric and as median with interquartile range (IQR) when skewed; categorical variables were reported as number and percentage. Women with and without any ECG abnormality were compared using Welch's independent-samples t test for continuous variables with approximately normal distributions and the Mann–Whitney U test for skewed variables.

Categorical proportions were compared with the chi-square test or Fisher's exact test when expected cell counts were small. Pearson correlation coefficients were calculated for selected continuous lipid, menopause-duration, and QTc relationships. A parsimonious binary logistic regression model evaluated independent correlates of any ECG abnormality; years since menopause, LDL-C (per 10 mg/dL), triglycerides (per 25 mg/dL), systolic blood pressure (per 10 mmHg), and BMI were entered simultaneously. Odds ratios (ORs) with 95% confidence intervals (CIs) are reported. Model fit was summarized using the likelihood-ratio test and McFadden pseudo-R^2. A two-sided p value <0.05 was considered statistically significant.

Results

The analytical cohort comprised 100 postmenopausal women. Mean age was 57.4 ± 6.1 years and mean age at menopause was 48.7 ± 2.6 years; the median duration since menopause was 8.6 (2.8–13.1) years. Mean BMI was 26.7 ± 3.6 kg/m², mean waist circumference 88.5 ± 9.0 cm, mean systolic blood pressure 125.4 ± 12.1 mmHg, and mean diastolic blood pressure 77.9 ± 7.5 mmHg. The lipid profile was atherogenic overall: total cholesterol averaged 206.9 ± 30.9 mg/dL, triglycerides had a median of 160.6 (126.5–203.2) mg/dL, HDL-C averaged 49.4 ± 8.3 mg/dL, LDL-C 125.8 ± 31.3 mg/dL, and non-HDL-C 157.5 ± 35.3 mg/dL. Mean TC/HDL-C and LDL-C/HDL-C ratios were 4.4 ± 1.2 and 2.7 ± 1.0, respectively. Mean QTc was 433.7 ± 17.5 ms. Any ECG abnormality was present in 45 (45.0%) of the cohort. The most frequent specific pattern was an ST–T abnormality (25 (25.0%)), followed by an ECG-LVH pattern (15 (15.0%)), axis deviation (10 (10.0%)), conduction abnormality (8 (8.0%)), premature beats/ectopy (7 (7.0%)), and QTc ≥465 ms (5 (5.0%)). Compared with women with a normal ECG, those with an abnormal ECG were older (61.0 ± 5.0 vs 54.5 ± 5.3 years), had a longer duration since menopause (median 11.9 vs 5.4 years), higher systolic blood pressure (129.8 ± 10.6 vs 121.8 ± 12.2 mmHg), higher total cholesterol (221.4 ± 29.4 vs 195.0 ± 27.0 mg/dL), higher LDL-C (139.5 ± 30.5 vs 114.5 ± 27.3 mg/dL), higher non-HDL-C (174.6 ± 31.9 vs 143.5 ± 31.8 mg/dL), and higher QTc (442.6 ± 15.9 vs 426.5 ± 15.5 ms). HDL-C was lower in the abnormal-ECG group (46.8 ± 6.5 vs 51.4 ± 9.1 mg/dL). The age at menopause, BMI, waist circumference, and diastolic blood pressure did not differ significantly between ECG groups (Table 1).

Table 1: Clinical, lipid and ECG characteristics according to ECG status

Variable	Overall (n=100)	Normal ECG (n=55)	Abnormal ECG (n=45)	p value
Age (years)	57.4 ± 6.1	54.5 ± 5.3	61.0 ± 5.0	<0.001
Age at menopause (years)	48.7 ± 2.6	48.6 ± 2.5	48.9 ± 2.8	0.562
Years since menopause	8.6 (2.8–13.1)	5.4 (1.0–9.0)	11.9 (9.6–16.0)	<0.001
BMI (kg/m²)	26.7 ± 3.6	26.3 ± 3.8	27.1 ± 3.3	0.259
Waist circumference (cm)	88.5 ± 9.0	88.1 ± 8.3	89.0 ± 9.8	0.616
Systolic BP (mmHg)	125.4 ± 12.1	121.8 ± 12.2	129.8 ± 10.6	<0.001
Diastolic BP (mmHg)	77.9 ± 7.5	77.1 ± 7.6	78.7 ± 7.3	0.290
Total cholesterol (mg/dL)	206.9 ± 30.9	195.0 ± 27.0	221.4 ± 29.4	<0.001

Triglycerides (mg/dL)	160.6 (126.5–203.2)	136.4 (106.7–182.1)	185.8 (152.2–219.9)	<0.001
HDL-C (mg/dL)	49.4 ± 8.3	51.4 ± 9.1	46.8 ± 6.5	0.004
LDL-C (mg/dL)	125.8 ± 31.3	114.5 ± 27.3	139.5 ± 30.5	<0.001
Non-HDL-C (mg/dL)	157.5 ± 35.3	143.5 ± 31.8	174.6 ± 31.9	<0.001
TC/HDL-C ratio	4.4 ± 1.2	4.0 ± 1.1	4.9 ± 1.2	<0.001
LDL-C/HDL-C ratio	2.7 ± 1.0	2.3 ± 0.8	3.1 ± 1.0	<0.001
QTc interval (ms)	433.7 ± 17.5	426.5 ± 15.5	442.6 ± 15.9	<0.001

Data are mean ± SD unless otherwise indicated; years since menopause and triglycerides are median (IQR). p values were obtained using Welch t test or Mann–Whitney U test, as appropriate.

Duration since menopause showed a strong gradient with both dyslipidemia and ECG abnormalities. Among 31 women <5 years from menopause and 69 women ≥5 years from menopause, the prevalence of total cholesterol ≥200 mg/dL was 25.8% versus 65.2%, triglycerides ≥150 mg/dL 22.6% versus 76.8%, LDL-C ≥130 mg/dL 22.6% versus 53.6%, HDL-C <50 mg/dL 32.3% versus 71.0%, and non-HDL-C ≥160 mg/dL 12.9% versus 63.8%, respectively. Any ECG abnormality was observed in 16.1% of women <5 years from menopause compared with 58.0% of those ≥5 years from menopause (p<0.001). ST–T abnormalities and ECG-LVH were also more common in the longer-duration group (Table 2; Figure 1).

Table 2: Prevalence of dyslipidemia and ECG abnormalities by duration since menopause

Variable	Overall (n=100)	<5 years (n=31)	≥5 years (n=69)	p value
Total cholesterol ≥200 mg/dL	53 (53.0%)	8 (25.8%)	45 (65.2%)	<0.001
Triglycerides ≥150 mg/dL	60 (60.0%)	7 (22.6%)	53 (76.8%)	<0.001
LDL-C ≥130 mg/dL	44 (44.0%)	7 (22.6%)	37 (53.6%)	0.004
HDL-C <50 mg/dL	59 (59.0%)	10 (32.3%)	49 (71.0%)	<0.001
Non-HDL-C ≥160 mg/dL	48 (48.0%)	4 (12.9%)	44 (63.8%)	<0.001
Any ECG abnormality	45 (45.0%)	5 (16.1%)	40 (58.0%)	<0.001
ST–T abnormality	25 (25.0%)	2 (6.5%)	23 (33.3%)	0.004
ECG-LVH pattern	15 (15.0%)	1 (3.2%)	14 (20.3%)	0.033
Axis deviation	10 (10.0%)	1 (3.2%)	9 (13.0%)	0.167
Conduction abnormality	8 (8.0%)	2 (6.5%)	6 (8.7%)	1.000
Premature beats/ectopy	7 (7.0%)	1 (3.2%)	6 (8.7%)	0.431
QTc ≥465 ms	5 (5.0%)	0 (0.0%)	5 (7.2%)	0.320

Values are n (%). Categories of specific ECG abnormalities are non-mutually exclusive. p values were obtained using chi-square or Fisher exact tests as appropriate.

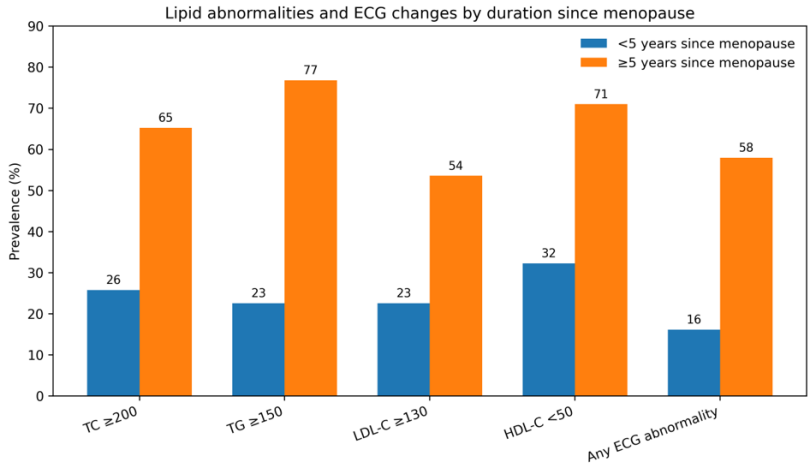


Figure 1: Prevalence of selected lipid abnormalities and any ECG abnormality according to duration since menopause

Continuous-variable analysis supported these group differences. Years since menopause correlated positively with non-HDL-C (r=0.59, p<0.001) and QTc (r=0.67, p<0.001). LDL-C was moderately correlated with QTc (r=0.46, p<0.001). In multivariable logistic regression, each additional

year since menopause remained independently associated with any ECG abnormality (adjusted OR 1.16, 95% CI 1.02–1.33; p=0.024), and each 10 mg/dL higher LDL-C was also independently associated with the outcome (adjusted OR 1.20, 95% CI 1.00–1.44; p=0.044). Triglycerides, systolic blood pressure, and BMI were not independently significant after simultaneous adjustment.

The overall model was significant (likelihood-ratio p<0.001) with McFadden pseudo-R^2=0.259 (Table 3; Figure 2).

Table 3: Logistic regression for predictors of any ECG abnormality

Predictor	Unadjusted OR (95% CI)	p value	Adjusted OR (95% CI)	p value
Years since menopause (per 1 year)	1.24 (1.13–1.36)	<0.001	1.16 (1.02–1.33)	0.024
LDL-C (per 10 mg/dL)	1.34 (1.15–1.56)	<0.001	1.20 (1.00–1.44)	0.044
Triglycerides (per 25 mg/dL)	1.52 (1.21–1.91)	<0.001	1.11 (0.79–1.58)	0.542
Systolic BP (per 10 mmHg)	1.85 (1.26–2.71)	0.002	1.14 (0.71–1.81)	0.593
BMI (per 1 kg/m²)	1.07 (0.95–1.19)	0.264	1.01 (0.87–1.17)	0.908

The adjusted model included years since menopause, LDL-C, triglycerides, systolic BP and BMI simultaneously. Likelihood-ratio p<0.001; McFadden pseudo-R^2=0.259. OR, odds ratio; CI, confidence interval.

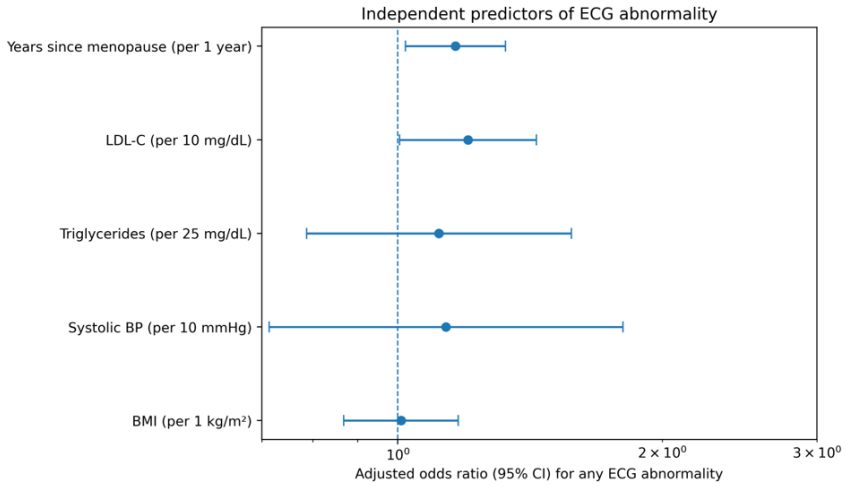


Figure 2: Forest plot of adjusted odds ratios for any ECG abnormality

Discussion

The present study describes a clinically coherent postmenopausal phenotype in which adverse lipid measures and resting ECG abnormalities clustered with increasing time since menopause. Nearly one half of the analytical cohort had at least one predefined ECG abnormality, and women with an abnormal ECG had substantially higher total cholesterol, LDL-C, non-HDL-C, triglycerides, cholesterol ratios, systolic blood pressure, and QTc, together with lower HDL-C. The strongest descriptive gradient was seen with duration since menopause: women at least five years beyond menopause had markedly higher prevalences of hypercholesterolemia, hypertriglyceridemia, elevated LDL-C, low HDL-C, elevated non-HDL-C, and any ECG abnormality than women within five years of menopause. In the multivariable model, years since menopause and LDL-C retained independent associations with the composite ECG outcome. These results do not establish causality, but they support the practical value of considering lipid burden, reproductive aging, blood pressure, and the resting ECG together when assessing postmenopausal women. The lipid findings are directionally consistent with the established biology and longitudinal epidemiology of the menopause transition. The American Heart Association statement identifies menopause as a phase during which atherogenic lipids, central adiposity, and other cardiovascular risk factors worsen and argues that midlife should be treated as a prevention opportunity [1]. The mean total cholesterol of 206.9 mg/dL and LDL-C of 125.8 mg/dL in this cohort, together with the high prevalence of triglycerides ≥150 mg/dL and HDL-C <50 mg/dL, fit the pattern described in contemporary reviews [2,3].

The magnitude of the difference by menopause duration is also compatible with SWAN observations that total cholesterol, LDL-C, and apolipoprotein B rise around the menopausal transition [4]. Although our study used a cross-sectional lipid measurement within a prospective observational enrollment framework rather than repeated pre-to-postmenopause measurements, the strong positive correlation between years since menopause and non-HDL-C provides a similar

clinical signal: longer reproductive aging was accompanied by greater atherogenic cholesterol burden. The finding that women ≥5 years beyond menopause had substantially more adverse lipid thresholds deserves specific attention. Wu and colleagues showed that midlife women follow distinct lipid trajectories and that menopause contributes to the evolution of these patterns [5]. Our results are not directly comparable to a longitudinal trajectory model, but the higher prevalence of elevated total cholesterol, LDL-C, triglycerides, low HDL-C, and elevated non-HDL-C in the longer-duration group is concordant with the concept of cumulative postmenopausal lipid exposure. The observation that non-HDL-C rose strongly with years since menopause is clinically useful because non-HDL-C integrates cholesterol carried in LDL and triglyceride-rich atherogenic particles. It can therefore be informative in a population where hypertriglyceridemia is common. Likewise, the LDL subclass work of El Khoudary and colleagues indicates that changes across menopause extend beyond LDL-C concentration to particle characteristics associated with later coronary calcification and carotid disease [6]. Our conventional lipid panel cannot measure those subclasses, but the higher LDL-C and cholesterol ratios in women with ECG abnormalities are compatible with a broader adverse lipoprotein phenotype.

The present results also align with previous clinical comparisons of lipid status around menopause. Inaraja and colleagues documented unfavorable lipid changes during the menopausal transition [7], while the Indian study by Kilim and Chandala reported higher total cholesterol, triglycerides, and LDL-C in postmenopausal than premenopausal women [8]. Dosi and colleagues similarly emphasized the cardiovascular implications of menopause in Indian women [9]. Direct numerical comparison across these studies must be cautious because of differences in age distribution, laboratory methods, adiposity, medication use, urban-rural mix, and definitions of menopause. Nevertheless, the consistency in direction is important. In our cohort, the dyslipidemic pattern was not limited to one analyte: several measures moved together, and women with abnormal ECGs had higher non-HDL-C and cholesterol ratios as well as higher LDL-C. This multidimensional pattern argues against interpreting a single lipid component in isolation. The ECG findings add a second layer of risk characterization. Denes and colleagues demonstrated in asymptomatic postmenopausal women that both major and minor ECG abnormalities were associated with subsequent cardiovascular events and mortality [13]. Rautaharju and colleagues further showed that electrocardiographic abnormalities, including repolarization-related measures, predicted coronary heart disease outcomes in a large Women's Health Initiative cohort [14]. The 45% composite abnormality prevalence in our analytical cohort should not be equated with a 45% prevalence of structural heart disease or ischemic heart disease, because the composite included minor as well as major patterns and categories could overlap. However, the concentration of ST–T changes, LVH patterns, and longer QTc among women with a worse lipid profile is clinically relevant. It identifies a subset in whom the ECG may be capturing the accumulated influence of age, blood pressure, myocardial remodeling, autonomic factors, and atherosclerotic risk.

Repolarization deserves separate consideration. Mean QTc was approximately 16 ms longer in women with any ECG abnormality, and years since menopause correlated with QTc. Prior work indicates that menopausal status and sex-hormone exposure can influence cardiac repolarization. Dogan and colleagues found differences in repolarization indices according to menopausal status [15], and Kadish and colleagues reported that estrogen and progestin use were associated with QT interval differences in postmenopausal women [16]. Lantto and colleagues also explored cardiac repolarization in the context of vasomotor symptoms and hormone therapy [17]. Our study was not designed to determine a hormonal mechanism and did not measure estradiol; therefore, the QTc association should be viewed as phenotypic rather than mechanistic. Even so, the coexistence of longer postmenopausal duration, higher LDL-C, and longer QTc provides a rationale for attention to repolarization when routine ECGs are already available. The multivariable analysis is particularly informative because it distinguishes crude clustering from independent associations. Years since menopause was associated with a 16% increase in the odds of an abnormal ECG per additional year after simultaneous adjustment for LDL-C, triglycerides, systolic blood pressure, and BMI. LDL-C remained independently associated as well, with approximately 20% higher odds per 10 mg/dL increment. By contrast, triglycerides and systolic blood pressure, although strongly associated in unadjusted analyses, were attenuated after adjustment. This pattern is plausible in a small cohort with correlated cardiometabolic exposures: postmenopausal duration, lipid variables, blood pressure, and age-related physiology share substantial variance. The model should therefore be interpreted as an exploratory risk-phenotyping model rather than a causal model or a clinical prediction tool. The confidence interval for LDL-C was also close to unity, underscoring the need for confirmation in larger datasets. From a preventive perspective, the findings support current thinking that the menopausal transition is a strategic time for cardiovascular risk assessment [10,12]. Earlier

menopause has been associated with higher subsequent cardiovascular disease risk in large pooled analyses [11], suggesting that reproductive history provides information not fully captured by age alone. In everyday practice, recording age at menopause and years since menopause costs nothing. When this information is paired with blood pressure, fasting lipids, and a resting ECG, clinicians may recognize women who warrant intensified lifestyle management, guideline-based lipid-risk assessment, evaluation for hypertension, or further cardiovascular testing based on symptoms and overall risk. The resting ECG should not be used as a screening substitute for formal atherosclerotic risk estimation, but an unexpected ST–T abnormality, LVH pattern, conduction defect, ectopy, or marked QT prolongation can appropriately trigger a more focused clinical review.

Our observations also have prognostic context. Schröder and colleagues reported that the resting ECG predicted mortality in postmenopausal women with coronary heart disease or coronary risk factors, emphasizing that ECG information may add clinically meaningful risk information even when obtained without advanced imaging [18]. The relationship between reproductive aging and blood pressure may contribute to this signal: a systematic review and meta-analysis found that early menopause is associated with an increased risk of arterial hypertension [19]. In our cohort, systolic blood pressure was significantly higher among women with an abnormal ECG, and an ECG-LVH pattern was more frequent in those further from menopause. Although systolic pressure lost statistical significance in the adjusted model, this likely reflects shared variation and limited sample size rather than an absence of clinical relevance.

Blood pressure remains a fundamental modifiable determinant of LVH, vascular disease, and cardiovascular outcomes. Several features strengthen the analytical approach. The study examined lipid variables and ECG phenotypes in the same women during a defined clinical period, used both continuous and clinically interpretable categorical lipid measures, reported non-HDL-C and lipid ratios in addition to the conventional panel, and prespecified a composite ECG endpoint to avoid fragmenting a modest sample across many sparse outcomes. The analysis also moved beyond simple prevalence estimates by comparing ECG strata, examining correlations, and fitting a parsimonious multivariable model. These elements make the findings more informative than a purely descriptive report of mean cholesterol values. The limitations are equally important. First, the sample size was 100, so estimates for less common ECG abnormalities are imprecise and the regression model must be regarded as exploratory. Second, the study was conducted at a single tertiary center in Bihar and may not represent all postmenopausal women in India.

Third, a single fasting lipid profile and resting ECG cannot establish temporal causation; serial measurements would better characterize trajectories. Fourth, echocardiography, ambulatory ECG monitoring, coronary imaging, apolipoprotein B, lipoprotein(a), sex hormones, thyroid indices, glycemic markers, and inflammatory biomarkers were not incorporated into the present analysis, limiting mechanistic inference and adjudication of ECG findings.

Fifth, Bazett correction can over- or under-correct QT depending on heart rate. Finally, and crucially for the present manuscript-development package, the patient-level master chart supplied with this draft is a synthetic, statistically coherent dataset created because verified source patient records were not provided.

The numerical results are therefore suitable for statistical/manuscript development and layout testing but must be replaced or reconciled against the actual source dataset before scientific submission, publication, or any claim that these exact patient-level observations were collected in clinical practice. Taken together, the data illustrate a clinically plausible pattern in which atherogenic lipids and ECG abnormalities become more frequent with increasing postmenopausal duration.

The association of LDL-C and years since menopause with an abnormal ECG persisted after adjustment for several competing cardiometabolic variables, while the strong differences in non-HDL-C, cholesterol ratios, and QTc suggest that risk is multidimensional. Larger prospective studies with verified longitudinal patient data, repeated lipid measurements, adjudicated ECG interpretation, and cardiovascular follow-up are required to determine whether combined lipid–ECG phenotyping meaningfully improves event prediction beyond established risk scores.

Conclusion

In this 100-woman analytical cohort, postmenopausal duration was accompanied by a progressively more atherogenic lipid profile and a higher frequency of resting ECG abnormalities. Women with abnormal ECGs had higher total cholesterol, triglycerides, LDL-C, non-HDL-C, cholesterol ratios, systolic blood pressure, and QTc, with lower HDL-C. Years since menopause and LDL-C remained independently associated with the composite ECG abnormality. In routine clinical care, simultaneous attention to reproductive history, conventional lipids, blood pressure, and the resting ECG may provide a practical framework for

identifying postmenopausal women who merit more intensive cardiovascular risk assessment.

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