

Review Article

Serum Lipid Profile Changes and Cardiovascular Risk Across Premenopausal and Postmenopausal Women: A Review

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ABSTRACT:

Cardiovascular disease remains a leading cause of morbidity and mortality among women, with risk rising markedly after menopause. This review examines changes in serum lipid profiles across premenopausal and postmenopausal stages and their implications for cardiovascular risk. During the reproductive years, women generally exhibit a more favorable lipid pattern, characterized by lower low-density lipoprotein cholesterol and relatively higher high-density lipoprotein cholesterol. However, the menopause transition is associated with significant hormonal alterations, particularly a decline in estrogen levels, which influence hepatic lipid metabolism, body fat distribution, and insulin sensitivity. These changes contribute to increases in total cholesterol, low-density lipoprotein cholesterol, triglycerides, and other atherogenic lipoprotein fractions, while the functional cardioprotective role of high-density lipoprotein may be diminished despite stable or mildly increased concentrations. Evidence suggests that many of these lipid changes occur around the final menstrual period rather than progressing gradually with age alone, indicating that menopause itself acts as a metabolic turning point. The resultant dyslipidemia, often accompanied by central adiposity and features of metabolic syndrome, accelerates atherosclerotic processes and elevates long-term cardiovascular risk in postmenopausal women.

Keywords: Menopause; Premenopausal women; Postmenopausal women; Serum lipid profile; Cardiovascular risk

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INTRODUCTION

Cardiovascular disease (CVD) remains the most common cause of death in women, but the way risk develops across a woman's life course is often simplified into a single axis of chronological aging. Menopause challenges this simplification because it represents a relatively abrupt biological transition that overlaps with major shifts in endocrine signaling, body composition, and cardiometabolic physiology. The menopause transition (perimenopause through early postmenopause) is characterized by fluctuating and then persistently lower ovarian estradiol production, rising gonadotropins, and changes in progesterone exposure. These hormonal changes do not occur in isolation: sleep quality can decline, vasomotor symptoms may disrupt daily function, physical activity patterns may change, and

psychological stress can increase. Together, these factors create a "metabolic inflection period" in which traditional risk factors may worsen quickly, sometimes faster than clinicians anticipate based on age alone. A major scientific statement has emphasized that the menopause transition is associated with adverse alterations in lipids and lipoproteins alongside changes in vascular health and fat distribution, strengthening the concept that midlife is a pivotal window for prevention rather than simply a late stage for risk documentation.¹ Among cardiometabolic biomarkers, the serum lipid profile is especially important because it is widely available, strongly predictive of atherosclerotic cardiovascular disease (ASCVD), and responsive to both lifestyle and pharmacologic intervention. The classic lipid panel—total cholesterol, LDL cholesterol (LDL-C),

HDL cholesterol (HDL-C), and triglycerides—captures key aspects of lipoprotein transport, but it is also a proxy for deeper biology: the number of atherogenic particles, the size and composition of those particles, and the balance between lipid delivery to tissues versus hepatic clearance. During reproductive years, many women display a relatively favorable lipid profile compared with age-matched men, yet this advantage narrows with the menopause transition. Epidemiologic and mechanistic work suggests that declining estrogen can reduce hepatic LDL receptor activity and modify hepatic lipoprotein production, contributing to higher LDL-C and other atherogenic markers. Importantly, the observed lipid changes are not always gradual; multiple longitudinal studies indicate that some of the steepest increases occur in proximity to the final menstrual period (FMP), meaning that the timing of lipid worsening may matter for risk counseling and screening cadence. A key question is how much lipid change is attributable to menopause itself rather than aging, since both progress over similar years. Longitudinal studies that anchor measurements to reproductive stage help separate these effects. One influential analysis concluded that several risk factors shift with age, but total cholesterol, LDL-C, and apolipoprotein B (apoB) show particularly notable increases clustered around the FMP, supporting a menopause-linked acceleration rather than a purely age-driven drift.² This distinction matters clinically: if lipid trajectories steepen during a short window, then a single “normal” lipid test earlier in the 40s may provide false reassurance for what will happen across late perimenopause and early postmenopause. Beyond magnitude, the *pattern* of lipid change is central to understanding ASCVD risk. Menopause is frequently associated with higher total cholesterol and LDL-C, but cardiometabolic risk is not determined by LDL-C alone. Atherosclerosis is driven by apoB-containing particles (including LDL and triglyceride-rich remnants), and risk can rise even when LDL-C appears only modestly elevated if apoB particle number, remnant cholesterol, or small dense LDL fractions increase. HDL-C can also be misleading: HDL quantity may remain stable—or even rise in some cohorts—while HDL function (such as cholesterol efflux capacity) changes in ways that alter its cardioprotective associations. In SWAN-related work, lipid changes around the FMP have been linked to later measures of subclinical vascular disease, suggesting that menopause may alter both lipid levels and the biological “meaning” of those levels for arterial health.³ Clinical guidelines increasingly acknowledge that female reproductive factors influence ASCVD risk estimation. In primary prevention, clinicians are encouraged to consider sex-specific risk enhancers (for example, premature menopause) when deciding whether lifestyle change alone is sufficient or whether earlier lipid-lowering therapy is warranted—especially when traditional 10-

year risk calculators may underestimate lifetime risk in women.⁴ Complementing guideline approaches, lipid societies have emphasized that women’s lipid biology varies across the life course and that sex-specific contexts—including menopause—should be integrated into interpretation of lipid markers and treatment decisions.⁵ This review synthesizes evidence on serum lipid profile changes and cardiovascular risk across premenopausal and postmenopausal women. We focus on (1) hormonal and hepatic mechanisms that drive lipid remodeling, (2) the timing and magnitude of shifts in total cholesterol, LDL-C, triglycerides, and apoB, (3) changes in LDL subclasses and HDL functionality, and (4) implications for clinical risk stratification and prevention across midlife. By organizing the evidence around menopause staging rather than age alone, the goal is to support more precise screening, earlier recognition of rapid lipid deterioration, and better-targeted interventions to reduce ASCVD risk in women transitioning into postmenopause.

MATERIAL AND METHODS

This narrative review was conducted over six months at the Departments of Physiology and Obstetrics & Gynecology, Index Medical College and Hospital, Indore, to synthesize evidence on serum lipid profile changes and cardiovascular risk in premenopausal versus postmenopausal women. A structured literature search was performed in PubMed/MEDLINE, Scopus, and Google Scholar using combinations of keywords such as “premenopausal,” “postmenopausal,” “menopause transition,” “lipid profile,” “LDL,” “HDL,” “triglycerides,” and “cardiovascular risk.” Eligible articles included observational studies, clinical trials, and systematic reviews published in English that reported lipid parameters and/or cardiovascular outcomes or validated surrogate markers in women stratified by menopausal status. Studies focused exclusively on men, pregnancy, surgical menopause without subgroup analysis, or lacking clear menopausal classification were excluded. Data were extracted on participant characteristics, menopausal definition, lipid measures (total cholesterol, LDL-C, HDL-C, triglycerides, and where available non-HDL/apoB), and cardiovascular endpoints or risk indicators. Evidence was narratively synthesized with emphasis on consistency across study designs, temporality around the final menstrual period, and clinically relevant risk interpretation, and key themes were summarized to guide preventive screening and management.

Endocrine drivers of lipid remodeling

Menopause alters lipid metabolism largely through changes in estrogen signaling, with the liver acting as a central “processing hub” for circulating lipoproteins. Estradiol influences hepatic LDL receptor expression and cholesterol clearance; when estrogen levels decline, LDL particle residence time can increase,

raising circulating LDL-C. Menopause also appears to affect VLDL production and triglyceride handling, partly through changes in adipose tissue lipolysis, insulin sensitivity, and hepatic de novo lipogenesis. These shifts can produce a profile that is not only higher in LDL-C but also richer in triglyceride-containing particles, which are increasingly recognized as atherogenic when they persist as remnants. Another key point is that menopause often coincides with changes in dietary patterns, activity levels, and sleep, all of which interact with hepatic lipid processing. Thus, lipid changes during menopause are best understood as a combined hormonal–metabolic transition rather than a single-hormone effect. Importantly, women do not experience menopause uniformly. Baseline metabolic health, ethnicity, body mass index, and lifestyle can influence the direction and magnitude of lipid changes. Women entering midlife with insulin resistance, central adiposity, or pre-existing dyslipidemia may show more pronounced increases in triglycerides and non-HDL cholesterol, while those with healthier metabolic profiles may show relatively larger changes confined to LDL-related measures. This variability helps explain why cross-sectional comparisons sometimes yield mixed findings: the same menopausal stage can produce different lipid “signatures” depending on the underlying metabolic context. From a prevention standpoint, the implication is that the menopause transition may be a period when individualized lipid assessment becomes more valuable than relying on population averages, and when clinicians should consider expanding beyond the basic lipid panel if discordance between LDL-C and overall risk is suspected.⁶

Body composition shifts as a lipid amplifier

Although estrogen decline can directly influence hepatic lipid handling, menopause is also associated with changes in body composition that indirectly amplify dyslipidemia. Many women gain fat mass during the menopause transition, with a tendency toward greater central or visceral adiposity. Visceral fat is metabolically active: it promotes free fatty acid flux to the liver, worsens insulin resistance, and increases inflammatory signaling. These pathways encourage hepatic VLDL secretion and impair triglyceride clearance, contributing to higher triglycerides and a more atherogenic remnant burden. In addition, loss of lean mass and reduced physical activity (sometimes linked to symptoms or life-stage demands) can lower metabolic rate and worsen glucose handling, which further pushes lipid metabolism toward a pro-atherogenic direction. The interaction between adiposity and lipid biology is clinically important because lipid levels may rise even when weight change appears modest. A small increase in waist circumference can reflect a meaningful shift toward visceral fat, which is more strongly tied to triglycerides, low HDL-C, and insulin resistance than

subcutaneous fat. In practice, this means clinicians should not assume “stable weight” implies stable lipid risk during midlife. Rather, tracking waist measures, blood pressure, glycemia, and triglycerides can reveal evolving cardiometabolic risk even before LDL-C crosses a conventional threshold. From a mechanistic viewpoint, this also clarifies why lifestyle interventions during perimenopause—especially resistance training, aerobic activity, and dietary patterns that reduce postprandial lipemia—can have outsized benefit: they target not only LDL-C but also the visceral fat–liver axis driving triglyceride-rich lipoproteins. Finally, body-composition change provides a bridge between menopause and cardiometabolic clustering. When central adiposity rises, women are more likely to meet criteria for metabolic syndrome, which combines high triglycerides, low HDL-C, elevated blood pressure, and dysglycemia. This clustering substantially increases ASCVD risk and can make the lipid profile appear “mixed” rather than purely LDL-driven, emphasizing the need for comprehensive risk assessment across the transition.⁷

Timing of lipid changes around the final menstrual period

One of the most clinically actionable insights from recent cohort work is that lipid changes are not evenly distributed across midlife. Rather than a slow linear progression, some markers begin rising several years before menopause and accelerate as women approach the final menstrual period. This pattern matters because it suggests there may be a predictable “pre-menopause ramp” during which earlier counseling and monitoring could prevent women from entering postmenopause with a markedly atherogenic profile. For example, if LDL-C and apoB begin rising meaningfully five years before menopause, then the late 40s may be a particularly strategic time to intensify lifestyle interventions and decide whether pharmacotherapy is appropriate for those with additional risk factors. The timing insight also helps interpret why two lipid tests separated by only a few years can show surprising divergence. A woman with an LDL-C that is acceptable at age 45 may experience a sharper-than-expected increase by age 50, even if her habits feel unchanged. Menopause-associated sleep disruption and symptom burden may contribute, but endocrine changes appear to play a role in shaping these trajectories. Clinically, this argues for a more stage-sensitive approach: lipid testing frequency might be increased in late perimenopause, especially when other risk factors are present (family history, hypertension, diabetes, smoking history, or prior pregnancy-related complications). Another practical implication is patient communication. Women often seek explanations for “why my cholesterol suddenly changed,” and menopause staging can provide a biologically coherent narrative that encourages engagement with prevention rather than resignation.

When framed well, this can improve adherence to lifestyle changes and shared decision-making about lipid-lowering therapy. It also supports the idea that menopause is a window for risk prevention rather than a point at which risk becomes fixed. Evidence describing these characteristic trajectories provides a foundation for building stage-aligned screening and intervention strategies.⁸

ApoB and LDL subclass remodeling

LDL-C is a useful clinical measure, but it does not always capture the number of atherogenic particles circulating in blood. ApoB reflects the particle count because each atherogenic particle contains one apoB molecule. During the menopause transition, apoB may rise alongside LDL-C, but discordance can occur: LDL-C may change modestly while apoB rises more substantially, implying more particles carrying less cholesterol per particle. This matters because particle number is more directly tied to the probability of arterial wall retention events that initiate atherosclerosis. Menopause-associated insulin resistance and triglyceride elevations can also shift LDL toward smaller, denser particles, which are more prone to oxidative modification and may have different arterial wall interactions. LDL subclass profiling provides insight into how menopause may change lipid risk without dramatic changes in the standard lipid panel. When triglycerides rise, lipid exchange processes remodel LDL particles, often increasing small dense LDL fractions. This pattern can coincide with lower HDL-C and higher remnant cholesterol, producing the classic “atherogenic dyslipidemia” phenotype that is common in insulin-resistant states. For clinicians, the practical takeaway is that menopause can move some women toward a mixed dyslipidemia pattern even if LDL-C is only mildly elevated. In such settings, non-HDL cholesterol and apoB can provide more complete information about atherogenic burden, and targeting triglyceride-rich pathways through lifestyle (and, when indicated, pharmacotherapy) may be needed in addition to LDL-C lowering. Understanding LDL remodeling also clarifies why women may experience rising ASCVD risk after menopause even when HDL-C appears acceptable. If the atherogenic particle pool expands and becomes more triglyceride-influenced, the overall risk environment can worsen despite a superficially “balanced” cholesterol profile. Detailed SWAN analyses of LDL subclasses across the menopause transition support the presence of these compositional shifts and reinforce the value of particle-focused metrics during midlife risk assessment.⁹

HDL quantity versus HDL function

HDL-C has long been considered protective, but evidence increasingly shows that HDL *function* can matter more than HDL cholesterol concentration. HDL particles participate in reverse cholesterol

transport, antioxidant activity, and modulation of inflammation. During the menopause transition, HDL-C does not always decline dramatically; in some women it stays stable or rises. However, shifts in HDL composition (protein and lipid cargo) and particle distribution can change how effectively HDL performs key functions such as cholesterol efflux from macrophages. This helps explain why the relationship between HDL-C and atherosclerosis can weaken after menopause: the same HDL-C value may not reflect the same functional capacity. From a clinical perspective, this complexity matters because it discourages overreliance on HDL-C as a “buffer” against other risk factors. A woman with rising LDL-C and triglycerides should not be reassured solely because HDL-C is high. Menopause may be a period when HDL particles become less efficient per particle or when increases in HDL-C reflect larger, cholesterol-rich HDL that does not translate into better efflux. Lifestyle interventions—physical activity, weight management, smoking cessation, and dietary quality—can improve HDL function even when HDL-C changes little, which provides a more actionable framework than chasing HDL-C numbers. Research measuring cholesterol efflux capacity and HDL subclasses across the transition supports the idea that midlife is a period of dynamic HDL remodeling. These functional approaches are not yet routine in most clinical settings, but they influence interpretation: a “normal” HDL-C should not be used to dismiss menopause-associated rises in apoB-containing particles. Instead, the emphasis should remain on lowering atherogenic particle burden and improving global metabolic health. Studies linking changes in HDL particle characteristics and efflux measures during the menopause transition provide a mechanistic underpinning for this updated view of HDL in postmenopausal risk.¹⁰

Lipids and subclinical atherosclerosis in midlife women

Understanding lipid change is clinically valuable only if it meaningfully connects to vascular outcomes. Subclinical atherosclerosis measures—such as carotid intima-media thickness, plaque assessment, and coronary artery calcium—offer a window into arterial disease before clinical events occur. In midlife women, menopause has been associated with adverse changes in vascular structure and function, and lipid profiles play a central role in that relationship. When LDL-C and apoB rise around menopause, the arterial wall is exposed to a higher burden of atherogenic particles during a period when vascular stiffness and endothelial function may also be worsening, creating a “double hit” for plaque initiation and progression. The relationship between lipids and subclinical disease can also change across menopause. For example, the protective association of HDL metrics may weaken, while the predictive value of apoB-containing particles remains strong. This

supports a prevention strategy that prioritizes lowering atherogenic burden rather than focusing on HDL-C elevation. It also strengthens the rationale for identifying rapid lipid worsening: if a sharp LDL-C increase occurs in late perimenopause, that may be an opportunity to intervene before calcification or plaque burden accumulates. Clinically, subclinical imaging is not required for most women, but it can be useful when treatment decisions are uncertain—such as when LDL-C is borderline and 10-year risk appears modest, yet there is a strong family history or multiple risk enhancers. In such cases, imaging may help align patient understanding with the need for earlier therapy. Importantly, the lipid changes that accompany menopause should be interpreted in the context of vascular aging, symptom burden, and metabolic clustering. Evidence from SWAN-related imaging work supports the view that menopause is a period when lipid shifts and subclinical arterial changes can occur in parallel, reinforcing midlife as a critical prevention window.¹¹

Hormone therapy: lipid effects are not the same as CVD outcome effects

Menopausal hormone therapy (MHT) often improves certain lipid parameters—particularly lowering LDL-C and raising HDL-C with oral estrogen—but lipid improvements do not automatically translate into lower coronary event risk. Large randomized trials evaluating estrogen plus progestin demonstrated reductions in total cholesterol and LDL-C compared with placebo, yet did not show an overall reduction in coronary heart disease risk and, in some analyses, suggested early harm in certain groups. This apparent contradiction highlights a key principle: CVD outcomes depend on more than the lipid panel alone, including effects on thrombosis, inflammation, blood pressure, glucose metabolism, and plaque stability, as well as timing of initiation relative to menopause. Timing remains a dominant hypothesis for reconciling biology and trial outcomes. Initiation of MHT closer to menopause may have different vascular effects than initiation later, when atherosclerosis is more established. Even so, MHT is not primarily prescribed for CVD prevention; it is used for symptom relief and other approved indications, with individualized risk–benefit discussions. From a lipid perspective, clinicians should recognize that MHT can change triglycerides differently depending on route (oral estrogens can increase triglycerides through hepatic first-pass effects), and progestin type can modify lipid responses. Therefore, lipid monitoring during MHT is reasonable, but lipid changes should be interpreted within the broader cardiovascular and thrombotic risk profile. The key takeaway is that menopause-related lipid worsening does not necessarily imply that replacing estrogen is the best lipid-management strategy. Instead, standard preventive approaches—dietary quality, weight management, exercise, and

evidence-based lipid-lowering therapies—remain central. Trial evidence documenting lipid changes alongside cardiovascular outcomes in women assigned to estrogen plus progestin provides an important caution against assuming lipid improvements guarantee event reduction.¹²

Meta-analytic evidence on MHT and lipid profiles

When considering MHT specifically for symptom management, clinicians and patients often ask what to expect on lipid labs. Meta-analyses of randomized trials provide a more systematic view than single studies. Overall, MHT tends to reduce LDL-C and total cholesterol, while effects on triglycerides vary by route and formulation; oral regimens more consistently raise triglycerides than transdermal approaches. Combined estrogen–progestin regimens can blunt some estrogen-associated lipid benefits compared with estrogen alone. These patterns reinforce the clinical importance of selecting route and regimen based on the full risk profile, not lipid targets alone. From a prevention viewpoint, the most useful role of meta-analytic lipid evidence is in planning monitoring and counseling. For example, if a woman already has elevated triglycerides or features of insulin resistance, an oral regimen that increases triglycerides may be less desirable, and transdermal options may be considered when clinically appropriate. Similarly, if LDL-C is rising rapidly around menopause, MHT-associated LDL reductions may be observed, but they should not replace guideline-based lipid-lowering decisions when ASCVD risk warrants statins or other therapies. The lipid changes from MHT are also best interpreted as part of a broader cardiometabolic landscape: a woman might see improved LDL-C yet still develop increased waist circumference or worsening glucose tolerance during midlife, which would still raise overall risk. Finally, meta-analytic findings underscore that lipid response is not identical across individuals. Baseline lipid status, BMI, smoking, dietary patterns, and genetic factors can influence observed changes. The practical approach is therefore to measure, reassess, and tailor—rather than assume a uniform “MHT improves lipids” effect. A systematic review and meta-analysis of MHT trials provides consolidated evidence for these lipid patterns and highlights the route-dependent trade-offs that are particularly relevant for postmenopausal lipid monitoring.¹³

Translating lipid patterns into cardiovascular risk assessment

Atherogenic lipid changes after menopause occur alongside rising absolute CVD risk because age increases event probability, but the menopause transition adds a clinically useful interpretive layer. Risk calculators often prioritize short-term (10-year) event risk and may underweight life-course exposures or sex-specific factors. As a result, some women in

their early 50s with significant lipid worsening and multiple metabolic changes may still appear “low risk” by short-term estimates, despite having decades of future exposure to higher apoB burden. Recognizing menopause-related acceleration in lipids helps clinicians justify earlier intervention, especially when additional enhancers are present (family history, autoimmune disease, pregnancy-related complications, or premature menopause). In practical terms, risk assessment in postmenopausal women benefits from emphasizing non-HDL cholesterol or apoB when triglycerides are elevated or when LDL-C appears discordant with the broader risk profile. It also benefits from assessing metabolic syndrome components, because triglycerides and low HDL-C frequently travel with hypertension and dysglycemia. When these factors cluster after menopause, cardiovascular risk rises more sharply than any single lipid parameter would suggest. This is also a point where patient education can be powerful: framing cholesterol change as part of a menopause-associated cardiometabolic transition can increase adherence to diet and activity changes and reduce stigma or self-blame. Evidence syntheses on menopause-associated CVD risk emphasize routine assessment of lipid profiles and other cardiometabolic factors in postmenopausal women, reflecting both the frequency of adverse shifts and the availability of effective preventive therapies. The overarching clinical message is to treat menopause as a prompt to reassess risk comprehensively, not merely as a demographic label. Reviews consolidating this evidence support integrating lipid changes into a broader, stage-aware approach to cardiovascular prevention in women.¹⁴

Management implications: beyond “check a lipid panel”

Because menopause can accelerate atherogenic lipid trajectories, management should be proactive and structured. Lifestyle interventions remain foundational: dietary patterns that reduce saturated fat and refined carbohydrates, increased fiber intake, regular aerobic activity plus resistance training, and weight/waist management can improve LDL-C, triglycerides, insulin sensitivity, and blood pressure simultaneously. However, for many women—especially those with elevated LDL-C, diabetes, hypertension, or multiple risk enhancers—lifestyle alone may be insufficient, and guideline-directed lipid-lowering therapy is appropriate. Modern cholesterol guidelines emphasize matching treatment intensity to overall ASCVD risk and LDL-C level, while also recognizing risk-enhancing factors that can shift decisions toward earlier statin use. For women, premature menopause is explicitly included among risk enhancers, reinforcing the principle that reproductive history informs lipid management thresholds. In midlife practice, this means that a postmenopausal woman with borderline LDL-C but several enhancers may reasonably start statin therapy

after shared decision-making. Non-statin therapies may be considered based on LDL-C response and risk category, and non-HDL or apoB targets can be useful when triglycerides are elevated. Monitoring should also be stage-sensitive. A reasonable approach is to recheck lipids during late perimenopause or early postmenopause if prior values were borderline or if symptoms and body composition are changing rapidly. Clinicians should also consider secondary contributors that become more common in midlife (hypothyroidism, medication effects, liver steatosis) when lipid changes seem disproportionate. The core idea is that menopause is not merely a time to *detect* dyslipidemia but a time to *anticipate* it and intervene earlier to reduce cumulative exposure to atherogenic particles. Cholesterol management guidance that formalizes risk enhancers and life-course prevention provides the framework for this approach.¹⁵

CONCLUSION

In conclusion, the menopause transition represents a critical period marked by adverse changes in serum lipid profiles that contribute to a heightened cardiovascular risk in postmenopausal women. The rise in atherogenic lipids, particularly LDL cholesterol and triglycerides, along with qualitative changes in lipoproteins, underscores the loss of cardioprotective effects seen in premenopausal women. These alterations highlight the importance of timely lipid screening and risk assessment during midlife. Early lifestyle modification and appropriate therapeutic interventions can play a key role in reducing long-term cardiovascular morbidity in women across the menopausal transition.

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