

**RISK OF CARDIOVASCULAR DISEASE DEVELOPMENT DURING
MENOPAUSE**

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Abstract

Menopause is a natural biological transition characterized by the permanent cessation of menstruation and a marked decline in ovarian estrogen production. Although menopause itself is not a disease, it is associated with significant cardiovascular, metabolic, and vascular changes that may increase the risk of hypertension, dyslipidemia, insulin resistance, arterial stiffness, atherosclerosis, coronary artery disease, stroke, and heart failure. Cardiovascular disease remains the leading cause of death among women globally, and the risk rises noticeably after the menopausal transition. According to the World Health Organization, cardiovascular diseases caused an estimated **19.8 million deaths in 2022**, representing about **32% of all global deaths**.

The increase in cardiovascular risk during menopause is not explained by age alone. Estrogen deficiency contributes to endothelial dysfunction, unfavorable lipid changes, increased vascular stiffness, visceral adiposity, systemic inflammation, and impaired glucose metabolism. Recent studies emphasize that menopause should be viewed as a critical window for cardiovascular risk assessment and prevention rather than only as a reproductive milestone. This article discusses the mechanisms, clinical

significance, diagnostic evaluation, prevention strategies, and limitations of current approaches to cardiovascular disease risk management in menopausal women.

Keywords: menopause, cardiovascular disease, estrogen deficiency, atherosclerosis, hypertension, dyslipidemia, endothelial dysfunction, arterial stiffness, women's health.

Introduction

Menopause usually occurs between the ages of 45 and 55 and marks the end of ovarian reproductive function. From a physiological perspective, it is defined by a decline in estrogen and progesterone production, leading to menstrual cessation and a wide range of systemic changes. While hot flashes, sleep disturbance, mood changes, and urogenital symptoms are commonly associated with menopause, one of the most clinically important consequences is the increase in cardiovascular risk.

Before menopause, women generally have a lower incidence of coronary artery disease than men of similar age, partly because estrogen has protective effects on the vascular endothelium, lipid metabolism, inflammatory regulation, and arterial elasticity. After menopause, this advantage gradually decreases. The European Society of Cardiology notes that cardiovascular disease is the biggest killer of women and that CVD risk in women rises after menopause, while adverse lipid changes during perimenopause may contribute to this acceleration.

The American Heart Association scientific statement on the menopause transition emphasizes that this period is associated with changes in body composition, lipids, vascular health, and metabolic risk. These changes may accelerate atherosclerotic cardiovascular disease risk, especially when menopause occurs early or when traditional risk factors such as hypertension, diabetes, obesity, smoking, or dyslipidemia are also present.

This topic is clinically important because women are often underdiagnosed or undertreated for cardiovascular risk. Symptoms of ischemic heart disease in women may be atypical, and cardiovascular prevention is sometimes delayed because

menopause-related symptoms are viewed as purely hormonal rather than cardiometabolic. Therefore, menopause should be considered an important period for screening, lifestyle intervention, and individualized cardiovascular prevention.

Materials and Methods

This article was prepared as a narrative scientific review. Current and authoritative sources were reviewed, including World Health Organization materials, American Heart Association statements, European Society of Cardiology updates, and recent peer-reviewed reviews on menopause and cardiovascular disease. The analysis focused on the following domains: hormonal changes during menopause, endothelial and vascular dysfunction, lipid and glucose metabolism, body composition, hypertension, clinical consequences, prevention, and modern approaches to risk assessment.

The review used a clinical-biological framework. First, the physiological role of estrogen in cardiovascular protection was summarized. Second, the effects of menopause on major cardiovascular risk factors were examined. Third, clinical implications were discussed, including coronary artery disease, stroke, heart failure, and hypertension. Finally, prevention strategies and limitations of current clinical practice were analyzed.

Results

The reviewed data show that menopause is associated with a cluster of cardiovascular changes. The most important mechanisms include estrogen deficiency, endothelial dysfunction, increased arterial stiffness, dyslipidemia, visceral fat accumulation, insulin resistance, low-grade inflammation, and autonomic changes. A 2025 review reported that estrogen deficiency is central in menopause-related cardiovascular risk because it negatively affects vascular endothelial function, arterial stiffness, and lipid profile.

One of the earliest and most important vascular changes is endothelial dysfunction. The endothelium regulates vascular tone by producing vasodilators such

as nitric oxide and prostacyclin. Estrogen normally supports endothelial function and helps maintain vascular elasticity. When estrogen levels fall, nitric oxide bioavailability may decrease, oxidative stress may increase, and vasoconstrictive pathways may become more active. This creates favorable conditions for hypertension, arterial stiffness, and atherosclerosis.

Another major change is lipid deterioration. During the menopausal transition, many women experience increases in total cholesterol, low-density lipoprotein cholesterol, triglycerides, and sometimes reductions or functional changes in high-density lipoprotein cholesterol. These lipid changes contribute to atherosclerotic plaque formation. The European Society of Cardiology highlighted that adverse blood fat profile changes during perimenopause are one possible mechanism linking menopause to increased cardiovascular risk.

Body composition also changes substantially. Even when total body weight does not increase dramatically, fat distribution often shifts toward visceral and abdominal adiposity. Visceral fat is metabolically active and promotes insulin resistance, chronic inflammation, endothelial dysfunction, and dyslipidemia. This helps explain why some women develop metabolic syndrome during or after menopause.

Blood pressure commonly increases with age, but menopause may accelerate vascular stiffening and sympathetic activation. Reduced estrogen levels can alter vascular smooth muscle function and increase arterial resistance. Hypertension is especially important because it is a major driver of stroke, heart failure, chronic kidney disease, and coronary artery disease.

Glucose metabolism may also worsen. Menopause-associated visceral adiposity and reduced insulin sensitivity increase the risk of type 2 diabetes. Diabetes in women is particularly dangerous from a cardiovascular perspective because it tends to reduce the premenopausal cardiovascular advantage and markedly increases risk of ischemic heart disease and stroke.

Inflammation and oxidative stress are additional contributors. After menopause, increased inflammatory mediators and oxidative stress may accelerate endothelial injury and plaque progression. These processes are not isolated; they interact with obesity, dyslipidemia, hypertension, and glucose intolerance.

Clinically, these mechanisms increase the likelihood of coronary artery disease, myocardial infarction, stroke, peripheral artery disease, atrial fibrillation, and heart failure. Importantly, cardiovascular risk may be higher in women who experience early menopause, premature ovarian insufficiency, surgical menopause, severe vasomotor symptoms, or menopause combined with prior adverse pregnancy outcomes such as preeclampsia or gestational diabetes.

Discussion

Menopause should be understood as a cardiovascular transition, not only a reproductive event. The decline in estrogen affects multiple systems at once: blood vessels become less flexible, lipids become more atherogenic, visceral fat increases, insulin sensitivity worsens, and inflammatory activity may rise. These changes do not automatically cause disease in every woman, but they create a biological environment in which traditional risk factors become more harmful.

One clinically important point is that chronological age and menopause are closely linked but not identical. Aging itself increases cardiovascular risk, but menopause adds specific hormonal and metabolic changes. This distinction matters because women with early menopause or surgical menopause may develop cardiovascular risk earlier than expected. Therefore, clinicians should ask about menstrual history, age at menopause, ovarian surgery, vasomotor symptoms, pregnancy complications, and family history when assessing cardiovascular risk.

The cardiovascular presentation in women may also differ from classic textbook descriptions. Coronary artery disease may present with chest discomfort, but women may also report shortness of breath, fatigue, epigastric discomfort, nausea, jaw pain,

back pain, or exercise intolerance. This can delay diagnosis if symptoms are attributed only to menopause, anxiety, anemia, or gastrointestinal problems.

Another important issue is hormone therapy. Menopausal hormone therapy can be effective for vasomotor and genitourinary symptoms, but it should not be used routinely as a primary strategy to prevent cardiovascular disease. Its risk-benefit profile depends on age, time since menopause, baseline cardiovascular risk, history of thrombosis, stroke risk, breast cancer risk, route of administration, dose, and whether estrogen is combined with progestogen. Current clinical thinking increasingly supports individualized decision-making rather than a single rule for all women. However, lifestyle modification and standard cardiovascular prevention remain the foundation.

Prevention should begin early. A woman entering menopause should have blood pressure measured, lipid profile assessed, glucose or HbA1c checked, body mass index and waist circumference evaluated, smoking status reviewed, and physical activity and diet discussed. Women with hypertension, diabetes, chronic kidney disease, autoimmune disease, obesity, history of preeclampsia, gestational diabetes, premature menopause, or strong family history require closer follow-up.

Lifestyle intervention is highly effective and should be presented as medical treatment, not only advice. A cardioprotective diet rich in vegetables, fruits, legumes, whole grains, fish, nuts, and unsaturated fats can improve lipid and glucose metabolism. Regular aerobic and resistance exercise improves endothelial function, blood pressure, insulin sensitivity, body composition, and mental health. Smoking cessation is essential because smoking accelerates endothelial injury and thrombosis. Sleep quality and stress management are also important, especially because menopausal symptoms can worsen sleep and autonomic balance.

Pharmacological prevention should be based on risk level. Antihypertensive treatment, statins, antidiabetic therapy, antiplatelet therapy when indicated, and weight-management strategies may be needed. The key is not to treat menopause itself as a

disease, but to identify and treat the cardiometabolic changes that become more common during this period.

A limitation in current practice is that many cardiovascular risk calculators do not fully capture female-specific risk enhancers, including early menopause, premature ovarian insufficiency, preeclampsia, gestational diabetes, and autoimmune disease. Therefore, risk assessment should combine standard tools with clinical judgment and women-specific history.

Conclusion

Menopause is a major biological transition associated with increased cardiovascular disease risk. The decline in estrogen contributes to endothelial dysfunction, arterial stiffness, dyslipidemia, visceral adiposity, insulin resistance, inflammation, and blood pressure elevation. These changes increase the risk of coronary artery disease, stroke, heart failure, and other cardiovascular disorders, particularly in women with early menopause, surgical menopause, metabolic syndrome, diabetes, hypertension, obesity, or adverse pregnancy history.

Cardiovascular prevention during menopause should be proactive, individualized, and long-term. Every menopausal woman should be assessed for blood pressure, lipid profile, glucose metabolism, body weight, lifestyle factors, family history, and female-specific risk enhancers. Lifestyle intervention, early diagnosis, risk-based pharmacotherapy, and regular monitoring are the most important strategies for reducing cardiovascular complications.

In clinical practice, menopause should be treated as a key opportunity for prevention. If cardiovascular risk is recognized early, many complications can be delayed or prevented, improving both life expectancy and quality of life for women.

References

1. World Health Organization. Cardiovascular diseases. WHO Fact Sheet, 2025.

2. El Khoudary SR, et al. Menopause Transition and Cardiovascular Disease Risk: Implications for Timing of Early Prevention. *Circulation*. 2020.
3. European Society of Cardiology. Menopause potentially linked to adverse cardiovascular health through blood fat profile changes. ESC Press Release, 2024.
4. Fasero M, et al. Cardiovascular Disease Risk in Women with Menopause. 2025.
5. Centers for Disease Control and Prevention. About Women and Heart Disease. 2024.
6. World Heart Federation. Women and cardiovascular disease.
7. Vallée A. Menopause and risk of atherosclerotic cardiovascular disease. 2025.