

REVISÃO DE LITERATURA IDENTIFICADO

MENOPAUSA E RISCO CARDIOVASCULAR: MECANISMOS, AVALIAÇÃO CLÍNICA E ESTRATÉGIAS PREVENTIVAS - REVISÃO INTEGRATIVA

MENOPAUSE AND CARDIOVASCULAR RISK: MECHANISMS, CLINICAL ASSESSMENT AND PREVENTIVE STRATEGIES - AN INTEGRATIVE REVIEW

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Eixo Temático: 01

RESUMO

Introdução: A transição menopausal associa-se a alterações hormonais, metabólicas e vasculares que podem acelerar o risco cardiovascular feminino durante a meia-idade. **Objetivo:** Analisar os mecanismos que relacionam menopausa e risco cardiovascular, os marcadores clínicos relevantes e as estratégias de prevenção. **Método:** Realizou-se revisão integrativa nas bases PubMed/MEDLINE, Scientific Electronic Library Online e Literatura Latino-Americana e do Caribe em Ciências da Saúde, além de documentos institucionais, com busca em agosto de 2026. Foram priorizadas publicações entre 2020 e 2026, mantendo-se estudos anteriores quando fundamentais. Nove fontes foram incluídas. **Resultados:** A queda estrogênica e o envelhecimento ovariano relacionam-se ao aumento de adiposidade visceral, perfil lipídico aterogênico, resistência à insulina, hipertensão, disfunção endotelial e rigidez arterial. Menopausa precoce e sintomas vasomotores frequentes ou persistentes identificam grupos de maior vulnerabilidade. A prevenção exige avaliação individual, controle de fatores de risco, atividade física, alimentação adequada e cessação do tabagismo. A terapia hormonal alivia sintomas e deve ser individualizada, não sendo indicada exclusivamente para prevenção cardiovascular. **Conclusão:** A menopausa constitui uma janela estratégica para rastreamento e intervenção cardiovascular precoce, com decisões terapêuticas baseadas no risco global e nas preferências da paciente.

Palavras-chave: Menopausa. Doenças Cardiovasculares. Fatores de Risco. Terapia Hormonal. Prevenção Primária.

ABSTRACT

Introduction: The menopause transition is associated with hormonal, metabolic, and vascular changes that may accelerate cardiovascular risk in women during midlife. **Objective:** To analyze the mechanisms linking menopause and cardiovascular risk, relevant clinical markers, and preventive strategies. **Method:** An integrative review was conducted in PubMed/MEDLINE, Scientific Electronic Library Online, and Latin American and Caribbean Health Sciences Literature, together with institutional documents, in August 2026. Publications from 2020 to 2026 were

prioritized, while earlier seminal studies were retained. Nine sources were included. **Results:** Estrogen decline and ovarian aging are associated with increased visceral adiposity, an atherogenic lipid profile, insulin resistance, hypertension, endothelial dysfunction, and arterial stiffness. Early menopause and frequent or persistent vasomotor symptoms identify groups with greater vulnerability. Prevention requires individualized assessment, risk-factor control, physical activity, an appropriate diet, and smoking cessation. Hormone therapy relieves symptoms and should be individualized; it is not indicated solely for cardiovascular prevention. **Conclusion:** Menopause is a strategic window for early cardiovascular screening and intervention, with therapeutic decisions based on global risk and patient preferences.

Keywords: Menopause. Cardiovascular Diseases. Risk Factors. Hormone Therapy. Primary Prevention.

INTRODUCTION

Menopause is a natural biological transition defined retrospectively after 12 consecutive months without menstruation, in the absence of another physiological or pathological cause. Natural menopause generally occurs between 45 and 55 years of age and is preceded by a variable transition marked by menstrual irregularity and fluctuating ovarian hormone concentrations. Although it is not a disease, the associated symptoms and long-term health changes may substantially affect quality of life and healthy aging.¹

Cardiovascular disease is a leading cause of illness and death among women, and risk increases notably across midlife. Longitudinal evidence indicates that the menopause transition is not merely a marker of chronological aging: it coincides with adverse changes in body composition, lipids and lipoproteins, blood pressure, glucose homeostasis, and vascular health. This period therefore represents a clinically important window for identifying risk that may otherwise remain unrecognized until after menopause.²⁻³

Estrogen decline contributes to impaired endothelial signaling, increased arterial stiffness, inflammation, and redistribution of fat toward the abdominal visceral compartment. At the same time, sleep disruption, vasomotor symptoms, reduced physical activity, and weight gain may interact with hypertension, dyslipidemia, insulin resistance, and smoking. Premature ovarian insufficiency and early menopause are particularly relevant because longer exposure to the postmenopausal metabolic environment is associated with greater cardiovascular vulnerability.²⁻⁵

Menopause care should extend beyond symptom relief. This review analyzed mechanisms linking the transition to cardiovascular risk, clinically relevant markers, and preventive and therapeutic strategies for midlife care.

METHOD

This integrative literature review addressed the following question: which mechanisms, clinical markers, and preventive strategies are most relevant to cardiovascular risk during and after the menopause transition? Searches were conducted in August 2026 in PubMed/MEDLINE, Scientific Electronic Library Online (SciELO), and Latin American and Caribbean Health Sciences Literature (LILACS), complemented by documents from international scientific organizations.

The descriptors Menopause, Cardiovascular Diseases, Estrogens, Dyslipidemias, Hypertension, Insulin Resistance, Vasomotor Symptoms, Exercise, and Hormone Replacement Therapy were combined with AND and OR. The principal strategy was: Menopause[MeSH] AND (Cardiovascular Diseases[MeSH] OR Cardiovascular Risk OR Dyslipidemia OR Hypertension OR Insulin Resistance) AND (Prevention OR Exercise OR Hormone Therapy). Portuguese equivalents were also used.

Original studies, reviews, scientific statements, guidelines, and institutional documents in English, Portuguese, or Spanish were eligible. Publications from 2020 through August 2026 were prioritized, with earlier seminal evidence retained when necessary. Duplicates, case reports, studies without separable menopause data, and publications without cardiovascular information were excluded. Titles, abstracts, and full texts were screened; nine sources were synthesized thematically.

RESULTS

The evidence comprised an international fact sheet, a cardiovascular scientific statement, clinical reviews, a longitudinal cohort, a systematic review with meta-analysis, and a hormone-therapy position statement. Findings were organized into mechanisms, cardiometabolic markers, female-specific indicators, and preventive strategies.

Hormonal and vascular mechanisms

Estradiol influences nitric oxide availability, vasomotor tone, vascular inflammation, oxidative stress, and lipid metabolism. During the menopause transition, declining and fluctuating estrogen exposure may reduce endothelial responsiveness and favor arterial stiffening. These changes occur alongside ovarian aging and may not be fully explained by chronological age, supporting the interpretation of menopause as a period of accelerated cardiovascular risk rather than a single isolated event.²⁻⁵

The transition is also accompanied by redistribution of adipose tissue from gluteofemoral to visceral depots. Visceral fat is metabolically active and is linked to proinflammatory signaling, insulin resistance, elevated triglycerides, lower-quality high-density lipoprotein particles, and a greater burden of atherogenic apolipoprotein B-containing lipoproteins. Loss of lean mass and lower energy expenditure may further facilitate weight gain and metabolic deterioration.²⁻⁴

Cardiometabolic phenotype and risk assessment

Clinically, the most consistent changes include increases in total cholesterol, low-density lipoprotein cholesterol, triglycerides, central adiposity, blood pressure, and glycemic dysfunction. High-density lipoprotein cholesterol may remain stable or increase, but particle composition and function can change, so an apparently favorable concentration should not be interpreted in isolation. Endothelial dysfunction and subclinical atherosclerosis may progress before a cardiovascular event becomes apparent.²⁻⁵

Assessment should include smoking status, blood pressure, body mass index and waist circumference, fasting lipid profile, glucose or glycated hemoglobin when indicated, physical activity, diet, sleep, family history, and previous pregnancy-related complications. The age at menopause should be documented. Menopause before 40 years, whether natural or surgical, and menopause between 40 and 44 years warrant particular attention because they are associated with higher long-term risk.²⁻⁵

Risk calculators may underestimate lifetime vulnerability in younger midlife women. Interpretation should integrate traditional factors with female-specific history, symptoms, and the timing and cause of menopause. Established hypertension, diabetes, dyslipidemia, or cardiovascular disease requires guideline-based management rather than attribution to menopause alone.²⁻⁵

Vasomotor symptoms and female-specific indicators

Hot flashes and night sweats are common manifestations of thermoregulatory instability. Their importance may extend beyond discomfort: in the Study of Women's Health Across the Nation, frequent vasomotor symptoms at baseline were associated with a higher incidence of cardiovascular events, and persistent frequent symptoms across follow-up showed an even stronger association after adjustment for conventional risk factors. These findings support symptom history as a prompt for cardiovascular assessment, although it is not a stand-alone diagnostic test.^{3,6}

Sleep disturbance, depression, and anxiety may indirectly worsen cardiovascular health through inactivity, altered eating, reduced adherence, and sympathetic activation. Menopause consultations should therefore assess mental health, sleep, sexual health, and social context alongside metabolic variables.^{1,3,6}

Palpitations, chest pain, dyspnea, syncope, or reduced exercise tolerance should not automatically be labeled menopausal. Clinical assessment must exclude arrhythmia, ischemia, heart failure, thyroid disease, anemia, and other conditions.

Prevention and hormone therapy

Prevention centers on lifestyle and risk-factor management. Aerobic and resistance exercise, tobacco avoidance, adequate sleep, limited alcohol, and a diet rich in plant foods, fish, and unsaturated fats support metabolic control. A systematic review found that aerobic training improved flow-mediated dilation after menopause. Hypertension, diabetes, and dyslipidemia should be treated according to individual risk and current guidelines.^{2,4-5,7}

Menopausal hormone therapy is the most effective treatment for bothersome vasomotor symptoms and prevents bone loss, but it is not indicated for cardiovascular prevention. In healthy symptomatic women younger than 60 years or within 10 years of menopause onset, without contraindications, the benefit-risk profile is generally more favorable. Route, dose, formulation, uterine status, thromboembolic and breast-cancer risks, and patient preferences must guide shared decisions and reassessment. Later initiation carries greater absolute vascular risk; nonhormonal options are appropriate when therapy is contraindicated or not desired.^{5,8-9}

CONCLUSION

Evidence indicates that the menopause transition is a period of accelerated cardiovascular vulnerability characterized by visceral adiposity, a more atherogenic metabolic profile, rising blood pressure, insulin resistance, and adverse vascular remodeling. Early menopause, premature ovarian insufficiency, and frequent or persistent vasomotor symptoms strengthen the need for individualized assessment, but they should complement rather than replace established cardiovascular evaluation.

Menopause care enables prevention before clinical disease emerges through lifestyle counseling, treatment of conventional risk factors, attention to sleep and mental health, and shared decisions. Hormone therapy may be appropriate for symptoms after risk assessment, but not solely for cardiovascular prevention. Future studies should refine the incorporation of female-specific markers into primary care.

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