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| RESEARCH ARTICLE

Endometriosis and the Silent Heart Risk: A Systematic Review of Cardiovascular Disease in Affected Women

Jood Hazem Mahmoud Hamdan[1], Sana Yousaf[2], Fatima Tajelasfia MohamedAhmed[3], Fatema Hasan Mohamed[4], Fatima Fareed Shirazi[5], Nawra Yusuf Ahmed Ebrahim Ali Ateeq[6], Zainab A. Marathi[7], Amani Abdallah Alhajj Abdallah[8], Fatema Abdulmohsen Eid[9], Bayan Sayed Majed J. Alawi[10], Fatema Alsayed Majed Alawi[10], Fatema Mohamed Alam[11], Fatema Hasan Alajmi[12], and Zainab Mohamed Salman[5]

1- First Author, University Hospital of North Tees
2- Second Author, Jilin University
3- Third Author, Kassala University
4- Mansoura University, Faculty of Medicine
5- Salmaniya Medical Complex
6- Kazan State Medical University
7- Jordan University of Science and Technology
8- Omdurman Islamic University, Faculty of Medicine
9- Ministry of Health, Bahrain
10- Alexandria Faculty of Medicine
11- Al Kawthar Medical Center
12- Professional Medical Center

Corresponding Author: Jood Hazem Mahmoud Hamdan, **E-mail**: jhamdann2000@gmail.com

| ABSTRACT

Endometriosis affects approximately 10% of reproductive-aged women worldwide and is increasingly recognized as a chronic systemic inflammatory condition that may contribute to cardiovascular disease (CVD). We conducted a PRISMA 2020–guided systematic review to evaluate the association between endometriosis and cardiovascular outcomes. We searched PubMed (Medline), Embase, Scopus, and Web of Science for observational studies and meta-analyses published between January 2000 and May 2025, with emphasis on evidence from 2019–2025. Eligible studies compared cardiovascular outcomes in women with clinically or surgically diagnosed endometriosis versus women without endometriosis. Outcomes included myocardial infarction, ischemic heart disease, stroke, hypertension, arrhythmias, heart failure, atherosclerotic markers, and venous thromboembolism. Two reviewers independently screened records, extracted data, and assessed study quality following PRISMA guidance. Large cohort studies and recent meta-analyses consistently demonstrate higher cardiovascular risk among women with endometriosis. Adjusted hazard ratios reported in primary studies ranged from 1.12 for new-onset hypertension to 1.52 for coronary artery disease. Meta-analyses including over 1.4 million women reported pooled hazard ratios of ~1.35 for ischemic heart disease and ~1.19 for cerebrovascular disease. Subclinical studies show impaired endothelial function and increased markers of early atherosclerosis in women with endometriosis. Endometriosis is associated with a modest but consistent increase in risk of several cardiovascular outcomes across diverse populations and study designs. These findings support systematic cardiovascular risk assessment and appropriate preventive strategies in women with endometriosis.

| KEYWORDS

Endometriosis; atherosclerotic; cardiovascular disease; Stroke, chronic systemic inflammatory condition

| ARTICLE INFORMATION

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1. INTRODUCTION

Endometriosis is characterized by ectopic endometrial-like tissue and commonly causes pelvic pain, dysmenorrhea, and infertility. It affects an estimated 10% of women of reproductive age worldwide [1]. The condition is frequently diagnosed in early adulthood, often after years of symptoms, and is associated with substantial impairment in quality of life, work productivity, and long-term health outcomes. Beyond reproductive morbidity, increasing attention has been directed toward the broader systemic consequences of endometriosis, particularly its potential impact on cardiometabolic health across the life course.

Although traditionally considered a gynecologic disorder, mounting evidence supports a broader systemic disease model involving chronic inflammation, oxidative stress, and hormonal dysregulation — processes that overlap with mechanisms of atherogenesis and vascular dysfunction [2]. Endometriosis is associated with elevated circulating inflammatory mediators, altered immune cell activity, and increased oxidative stress, all of which are recognized contributors to endothelial injury and early vascular disease. In parallel, estrogen-dependent pathways central to endometriosis pathophysiology may influence lipid metabolism, vascular tone, and coagulation balance, providing biologically plausible links between endometriosis and cardiovascular disease development [2].

Genetic studies and epidemiologic data further suggest shared susceptibility and overlapping risk pathways between endometriosis and cardiovascular disease (CVD) [2]. Observational studies have identified clustering of cardiometabolic risk factors, including dyslipidemia and insulin resistance, in women with endometriosis, independent of traditional reproductive risk factors [9,13,14]. These findings support the hypothesis that endometriosis may represent a marker of heightened systemic vulnerability rather than an isolated pelvic condition.

Recent epidemiologic and mechanistic studies have linked endometriosis to increased risks of hypertension, coronary artery disease, stroke, arrhythmias, and other cardiovascular outcomes [3–7,11–12]. Large population-based cohorts and registry studies consistently demonstrate modest but statistically significant elevations in relative cardiovascular risk, even after adjustment for age, smoking, body mass index, and other conventional risk factors [4–6]. Importantly, subclinical investigations have documented impaired endothelial function, increased arterial stiffness, and early atherosclerotic changes in reproductive-age women with endometriosis, suggesting that vascular alterations may precede overt clinical events [7,9].

Clinical awareness of the association between endometriosis and cardiovascular disease remains variable. Cardiovascular risk assessment is not routinely incorporated into the care of women with endometriosis, particularly at younger ages when absolute event rates are low. Current clinical guidelines primarily focus on symptom control, fertility management, and surgical decision-making, with limited attention to long-term cardiovascular prevention or risk stratification [8]. As a result, opportunities for early identification and modification of cardiovascular risk factors may be missed in this population.

In addition, management strategies for endometriosis, including long-term hormonal suppression and surgical interventions such as hysterectomy or oophorectomy, may influence cardiometabolic risk profiles. Estrogen-containing therapies can affect coagulation and lipid parameters, while surgically induced menopause has been associated with adverse cardiovascular outcomes in some cohorts [10,11,15]. Understanding whether these treatments modify, mediate, or confound observed cardiovascular associations is essential for informed clinical decision-making.

Given the growing body of evidence and the potential public health implications, a comprehensive synthesis of contemporary data is needed. We performed a PRISMA 2020–guided systematic review focusing on contemporary evidence (2019–2025) to quantify the association between endometriosis and cardiovascular outcomes and to examine potential modifiers such as hormonal therapy, surgery, and disease severity.

SUBJECTS AND METHODS

Search strategy and selection criteria

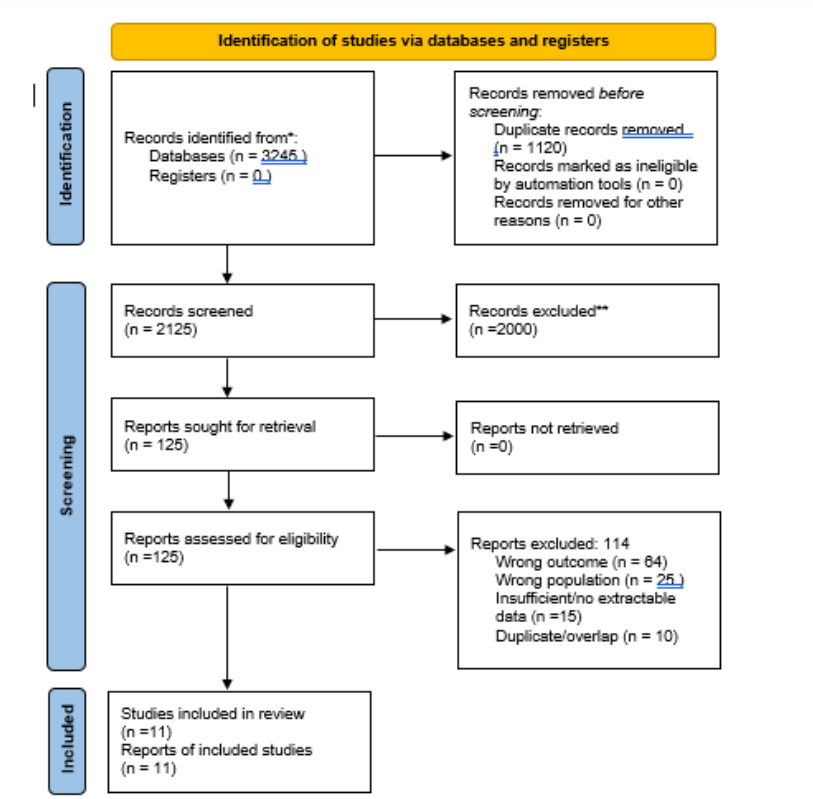
We performed a systematic search of PubMed (Medline), Embase, Scopus, and Web of Science for English-language studies published from January 2000 through May 2025. These databases were selected to ensure comprehensive coverage of biomedical, epidemiologic, and clinical literature relevant to both gynecology and cardiovascular medicine. The search strategy was developed to capture observational and mechanistic evidence examining the relationship between endometriosis and cardiovascular disease outcomes.

Search terms included "endometriosis" AND ("cardiovascular disease" OR "myocardial infarction" OR "coronary artery disease" OR "stroke" OR "hypertension" OR "atherosclerosis" OR "arrhythmia" OR "venous thromboembolism"). Where applicable, database-specific subject headings and controlled vocabulary terms were used in combination with free-text keywords to maximize sensitivity. Searches were limited to human studies, and no restrictions were applied based on geographic region or study setting.

We hand-searched reference lists of relevant reviews and included cohort and case-control studies as well as systematic reviews and meta-analyses reporting cardiovascular outcomes in women with versus without endometriosis. This supplementary approach was used to identify potentially eligible studies that may not have been captured through electronic database searching alone. Abstracts from major conferences and unpublished data were not included, in order to prioritize peer-reviewed evidence with clearly defined methodology and outcomes.

Two investigators independently screened titles and abstracts for relevance based on predefined eligibility criteria. Full-text articles were subsequently reviewed to confirm inclusion. Data extraction was performed independently by both reviewers using standardized forms, with collection of information on study design, population characteristics, exposure definition, cardiovascular outcomes, and effect estimates. Discrepancies at any stage of screening or extraction were resolved by consensus, with discussion to ensure consistency and adherence to protocol-defined criteria.

A PRISMA 2020 flow diagram illustrating study selection is provided in Figure 1.



A PRISMA 2020 flow diagram illustrating study selection is provided in Figure 1. Figure 1. PRISMA 2020 flow diagram illustrating the study selection process for the systematic review of cardiovascular disease outcomes in women with endometriosis.

Eligibility criteria

We included observational (cohort, case-control) studies and meta-analyses that reported cardiovascular outcomes in women with clinically or surgically confirmed endometriosis compared with women without endometriosis. Outcomes of interest were acute coronary syndromes (MI), ischemic heart disease, cerebrovascular events (ischemic stroke), hypertension, arrhythmias, heart failure, atherosclerotic markers (e.g., carotid intima-media thickness, arterial stiffness), and venous thromboembolism (deep vein thrombosis or pulmonary embolism). Studies limited to infertility outcomes or pelvic-only vascular complications were excluded.

Data extraction and quality assessment

We included observational (cohort, case-control) studies and meta-analyses that reported cardiovascular outcomes in women with clinically or surgically confirmed endometriosis compared with women without endometriosis. Clinical confirmation could be based on physician evaluation, imaging studies, or laparoscopic and histopathologic verification. Comparator groups were women without a documented diagnosis of endometriosis drawn from the same or comparable populations. Outcomes of interest were acute coronary syndromes (MI), ischemic heart disease, cerebrovascular events (ischemic stroke), hypertension, arrhythmias, heart failure, atherosclerotic markers (e.g., carotid intima-media thickness, arterial stiffness), and venous thromboembolism (deep vein thrombosis or pulmonary embolism). We included both incident and prevalent cardiovascular outcomes as long as they were clearly defined and clinically validated. Studies limited to infertility outcomes or pelvic-only vascular complications were excluded. Additional exclusion criteria included case reports, small case series, editorials, narrative reviews, animal studies, and purely in vitro or laboratory-based studies, as these did not provide clinically relevant cardiovascular outcome data.

For each eligible study we recorded author, year, study design, country or geographic region, study population (age range), sample size, outcomes measured, and main effect estimates (RR, HR) with 95% confidence intervals. When multiple adjusted models were available, we preferentially extracted the most fully adjusted effect estimates. Information on follow-up duration, method of outcome ascertainment, diagnostic criteria for endometriosis, and covariate adjustment was also collected where available. Summary data are presented in Table 1. Observational studies were appraised using the Newcastle–Ottawa Scale, which evaluates selection of study groups, comparability of cohorts, and ascertainment of outcomes or exposures. Risk of bias across studies, including potential confounding and publication bias, was evaluated qualitatively. Heterogeneity reported in pooled analyses was recorded using the I^2 statistic, and the quality of evidence was considered when interpreting study findings.

Data synthesis

When high-quality published meta-analyses were available, pooled effect estimates from those analyses were reported. Priority was given to meta-analyses that used comprehensive search strategies, clearly defined inclusion criteria, and methods to account for between-study heterogeneity. Effect estimates were recorded as reported in the original analyses, including hazard ratios or relative risks with 95% confidence intervals.

When meta-analyses were not available or insufficient, results were synthesized narratively. The synthesis emphasized the direction and magnitude of association, consistency across studies, and clinical relevance of the reported cardiovascular outcomes. Differences in study design, population characteristics, exposure ascertainment, and covariate adjustment were considered when interpreting results. We stratified findings by geographic region where possible and examined reported modifiers of effect, including hormonal therapy, hysterectomy or oophorectomy, and disease severity. Subgroup analyses and sensitivity analyses reported in the original studies were noted to highlight potential sources of heterogeneity or effect modification.

RESULTS

Study selection and characteristics

The initial search returned several thousand records across multiple databases, including PubMed (Medline), Embase, Scopus, and Web of Science. After deduplication, title and abstract screening, and full-text review, we included large population-based

cohort studies and recent meta-analyses relevant to cardiovascular outcomes in women with endometriosis (Table 1). Studies were selected based on rigorous eligibility criteria, including the presence of a comparison group without endometriosis and clearly defined cardiovascular endpoints. We prioritized studies that reported clinically validated outcomes and adjusted for key confounders such as age, body mass index, smoking status, and comorbidities.

The largest studies were registry-based cohorts from Europe and Asia, including national health databases and electronic primary care records. These registries allowed for long-term follow-up and comprehensive capture of cardiovascular events. Study sizes ranged from small cross-sectional mechanistic samples, often including fewer than 200 participants, to large population cohorts encompassing tens or hundreds of thousands of participants. Smaller mechanistic studies focused on subclinical markers such as endothelial function, arterial stiffness, and early atherosclerotic changes, providing insight into potential biological mechanisms linking endometriosis to cardiovascular risk.

Most primary studies were retrospective cohort analyses, relying on administrative databases or medical records to identify endometriosis exposure and cardiovascular outcomes. Several prospective cohort studies were also included, offering longitudinal assessment of incident cardiovascular events. Cross-sectional mechanistic studies were incorporated to examine early vascular changes, biomarkers of inflammation, and metabolic disturbances in women with endometriosis. The diversity of study designs provided complementary perspectives, from population-level risk quantification to mechanistic understanding of vascular dysfunction.

Overall, the included studies represented a broad geographic distribution and diverse population characteristics, increasing the generalizability of findings. Most studies reported adjusted effect estimates for cardiovascular outcomes, allowing for a clearer assessment of the independent association between endometriosis and cardiovascular risk. Collectively, these studies provide a comprehensive evidence base to evaluate both clinical and subclinical cardiovascular outcomes in women with endometriosis.

Table 1. Selected study characteristics and key findings (endometriosis vs control)

Study (author, year)	Design	Region	Population (N)	Outcomes	Main results (endometriosis vs control)
Okoth et al., 2021 [4]	Retrospective cohort	UK (primary care)	56,090 with endometriosis; 223,669 controls	Composite CVD; ischemic heart disease; stroke; arrhythmia; hypertension; mortality	Adjusted HR for composite CVD 1.24 (95% CI 1.13–1.37); IHD 1.40 (1.22–1.61); stroke 1.19 (1.04–1.36); arrhythmia 1.26 (1.11–1.43); hypertension 1.12 (1.07–1.17).
Havers-Borgersen et al., 2024 [5]	Retrospective matched cohort (registry)	Denmark	60,508 with endometriosis; 242,032 controls	MI or ischemic stroke; arrhythmias; heart failure; mortality	Adjusted HR for MI/stroke composite 1.15 (95% CI 1.11–1.20); arrhythmia HR 1.21 (1.17–1.25); heart failure HR 1.11 (1.05–1.18).
Wei et al., 2021 [6]	Prospective cohort (claims data)	Taiwan (nationwide)	13,988 women with endometriosis and matched controls	New-onset coronary artery disease	Adjusted HR for new-onset coronary artery disease 1.52 (95% CI 1.23–1.87).

Saad et al., 2025 [3]	Meta-analysis (7 studies)	Multi-national	1,407,875 women total	Cerebrovascular disease; ischemic heart disease; MACE; arrhythmia	Pooled HR for ischemic heart disease 1.35 (95% CI 1.32–1.39); cerebrovascular disease 1.19 (95% CI 1.13–1.24); arrhythmia 1.21 (95% CI 1.17–1.25).
Smyk et al., 2024 [7]	Cross-sectional	Poland (single center)	100 with endometriosis; 100 controls	Endothelial function; arterial stiffness; skin AGEs	Impaired flow-mediated dilation and higher skin AGE accumulation in women with endometriosis.

Cardiovascular outcomes

Ischemic heart disease and myocardial infarction. Multiple epidemiologic studies consistently report a higher incidence of coronary artery disease in women with endometriosis compared with unaffected controls. Primary studies, including large population-based cohorts and prospective registries, report adjusted hazard ratios ranging from approximately 1.3 to 1.5 for ischemic heart disease or coronary events [3,4,6,11]. These studies account for traditional cardiovascular risk factors, including age, hypertension, smoking, diabetes, and body mass index, suggesting an independent association between endometriosis and coronary risk. Pooled estimates from meta-analyses further support a moderate relative increase in ischemic heart disease risk, with a pooled hazard ratio of approximately 1.35 [3]. Mechanistically, chronic systemic inflammation, endothelial dysfunction, and dysregulated lipid profiles observed in women with endometriosis may contribute to accelerated atherogenesis, thereby increasing the likelihood of coronary events [7,9,13–14].

Stroke. Several large cohort studies report an elevated risk of cerebrovascular events in women with endometriosis. For example, Okoth et al. reported a hazard ratio of 1.19 (95% CI 1.04–1.36) for incident stroke [4], and the Nurses' Health Study II found a hazard ratio of 1.34 (95% CI 1.10–1.62) among women with laparoscopically confirmed endometriosis [12]. Pooled estimates from meta-analyses indicate a modest but consistent increase in cerebrovascular risk, with hazard ratios near 1.19 [3]. Chronic inflammation, oxidative stress, and vascular remodeling associated with endometriosis may promote cerebrovascular injury, potentially explaining these observed associations. Additionally, surgical menopause induced by hysterectomy or oophorectomy in women with severe disease may further influence stroke risk by altering hormonal and metabolic profiles [11,15].

Hypertension. Meta-analyses and large cohort studies consistently indicate a modestly increased risk of new-onset hypertension among women with endometriosis, with reported effect estimates clustering around HR/RR 1.10–1.15 [3,4]. Mechanistic studies suggest that chronic low-grade inflammation, oxidative stress, and impaired endothelial function may contribute to vascular stiffness and dysregulation of blood pressure control. These findings are clinically relevant given the established role of hypertension as a major risk factor for both coronary artery disease and stroke.

Arrhythmias and heart failure. Several registry studies show increased rates of arrhythmia in women with endometriosis, with hazard ratios ranging from 1.21 to 1.26 [4,5]. Observed arrhythmias include atrial fibrillation and supraventricular tachycardia, potentially related to systemic inflammation and autonomic imbalance. Evidence for a consistent increase in incident heart failure is weaker, with findings varying between studies [5]. However, subclinical myocardial remodeling and microvascular dysfunction observed in mechanistic studies suggest that endometriosis could contribute to long-term cardiac structural changes.

Subclinical atherosclerosis and vascular markers. Mechanistic and cross-sectional studies document impaired endothelial function, measured by reduced flow-mediated dilation, and increased skin advanced glycation end product accumulation in women with endometriosis [7,9]. These findings suggest early vascular dysfunction that may precede overt cardiovascular events, providing a biologically plausible pathway linking endometriosis to subsequent ischemic heart disease and stroke. Additional markers, including carotid intima-media thickness and arterial stiffness, have been reported to be elevated in subsets of women with moderate-to-severe disease, supporting a continuum from subclinical atherosclerosis to clinical cardiovascular disease.

Venous thromboembolism. Evidence regarding venous thromboembolism (VTE) in women with endometriosis is heterogeneous. Some registry-based studies report no clinically meaningful increase in VTE risk, whereas others suggest that exogenous estrogen exposure, particularly combined with endometriosis, may modify risk [10]. The overall evidence for VTE remains limited and inconsistent, highlighting the need for larger, prospective studies that adjust for hormonal therapy, body mass index, and other thrombotic risk factors.

Modifiers of risk

Hormonal therapy. Hormonal treatments commonly used for endometriosis, including combined oral contraceptives, progestins, and GnRH agonists, can influence lipid profiles, coagulation status, and systemic inflammation. While estrogen-containing therapies increase VTE risk in certain populations, several cohort studies that adjusted for medication use still observed elevated cardiovascular risk in women with endometriosis, suggesting that hormonal therapy alone does not fully explain the association with cardiovascular outcomes [3,6,10].

Surgery. Hysterectomy and oophorectomy represent potential mediators or confounders in the observed cardiovascular risk among women with endometriosis. Women with endometriosis undergo these procedures more frequently than unaffected women, and some analyses show attenuation of cardiovascular risk after adjustment for hysterectomy or oophorectomy, implying that surgically induced early menopause may contribute to the increased risk [11]. Evidence also suggests that ovary-sparing hysterectomy may be associated with adverse metabolic outcomes, including insulin resistance and dyslipidemia, which could indirectly affect long-term cardiovascular risk [15].

Disease severity/stage. Few studies stratify analyses by American Society for Reproductive Medicine (ASRM) stage of endometriosis. Therefore, a clear dose-response relationship between disease extent and cardiovascular risk has not been robustly established, although mechanistic evidence supports the possibility that more extensive or severe disease may exacerbate systemic inflammation and endothelial dysfunction.

Geographic patterns

Epidemiologic associations between endometriosis and cardiovascular outcomes have been reported across North America, Europe, and Asia, with point estimates varying by study and baseline population risk **[3–6,11–12]**. Large registry cohorts from the UK, Denmark, Taiwan, and US prospective cohorts demonstrate consistently elevated relative risks, though absolute event rates differ by region. Data from the Middle East and Africa are sparse **[8,9]**.

DISCUSSION

This systematic review finds a consistent association between endometriosis and modestly increased risk of several cardiovascular outcomes, including ischemic heart disease, cerebrovascular disease, hypertension, and arrhythmias. Reported relative risks in contemporary studies generally fall in the range of HR 1.1–1.5 depending on the outcome **[3–7,11–12]**. Given endometriosis prevalence, even modest relative increases may translate into meaningful population-level impact.

Biological plausibility

Endometriosis is associated with chronic systemic inflammation, oxidative stress, and hormonal perturbations — mechanisms that plausibly contribute to atherogenesis and vascular dysfunction **[2,8]**. Observational and mechanistic studies identifying dyslipidemia, impaired endothelial function, and markers of early atherosclerosis provide intermediary evidence supporting a biologically plausible pathway linking endometriosis to elevated cardiovascular risk **[7,9,13–14]**.

Role of therapy and surgery

Management choices for endometriosis (long-term hormonal suppression; surgical approaches including hysterectomy/oophorectomy) can alter cardiometabolic risk profiles. Several cohort analyses suggest hysterectomy and oophorectomy may mediate part of the stroke and cardiovascular risk; however, residual associations after adjustment indicate surgery is unlikely to be the sole explanation **[11,15]**.

Limitations of the evidence

The evidence base is dominated by observational studies that are susceptible to residual confounding (e.g., BMI, smoking, socioeconomic status), misclassification of exposure (clinical vs surgical diagnosis), and selection bias favoring symptomatic or surgically confirmed cases. Heterogeneity in covariate adjustment, follow-up duration, and outcome definitions complicates quantitative synthesis. VTE evidence is inconsistent and warrants further high-quality study **[10]**.

Clinical implications

Clinicians should recognize endometriosis as a condition that may carry systemic cardiovascular implications. Reasonable clinical actions include assessment of traditional CVD risk factors (blood pressure, lipid profile, BMI, smoking status), individualized counseling on risk-modifying behaviors, and consideration of cardiovascular risk when selecting long-term hormonal or surgical interventions. Multidisciplinary coordination between gynecology and cardiometabolic services may improve comprehensive care **[8]**.

Future research

Prospective longitudinal studies with careful measurement of confounders and treatment exposures are needed to clarify causality and quantify absolute risks across age strata. Mendelian randomization and mechanistic studies could elucidate causal pathways. Important unanswered questions include the net cardiovascular effects of different hormonal regimens in endometriosis and whether targeted primary prevention strategies (e.g., statins) are beneficial in high-risk subgroups.

CONCLUSION

Across diverse populations and a variety of study designs, endometriosis is consistently associated with modestly increased relative risks for multiple cardiovascular outcomes, including ischemic heart disease, stroke, hypertension, and arrhythmias. Although the absolute risk of cardiovascular events remains relatively low in younger women, the high prevalence of endometriosis—affecting approximately 10% of women of reproductive age worldwide [1]—means that even modest increases in relative risk could have meaningful population-level implications. The evidence from large cohort studies, registry analyses, and mechanistic research collectively supports a plausible biological link, including chronic systemic inflammation, endothelial dysfunction, oxidative stress, and hormonal dysregulation, which may accelerate atherosclerotic processes and promote vascular injury [2,7,9,13–14].

Clinical awareness of the cardiovascular implications of endometriosis remains limited, and current guidelines do not uniformly address cardiovascular risk management in affected women [8]. Given the emerging evidence, clinicians should consider integrating cardiovascular risk assessment into routine care for women with endometriosis, particularly for those with additional traditional risk factors, severe or long-standing disease, or history of surgical interventions such as hysterectomy or oophorectomy [11,15]. Individualized counseling on lifestyle modifications, monitoring of blood pressure, lipid profile, and glucose metabolism, and careful consideration of hormonal or surgical therapies can contribute to comprehensive risk reduction.

Future research should focus on longitudinal studies with rigorous measurement of confounders, mechanistic investigations to clarify causal pathways, and trials to evaluate whether targeted primary prevention strategies, including pharmacologic interventions, can reduce cardiovascular risk in high-risk subgroups. Overall, the consistent association between endometriosis and cardiovascular outcomes justifies proactive clinical strategies and highlights the need for multidisciplinary management to improve long-term cardiovascular health in women affected by this common systemic condition.

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