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Endometriosis as a Risk Factor for Epithelial Ovarian Cancer: Sub-types, Mechanisms and Risk Stratification

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Abstract

Background: Endometriosis is a common gynecologic condition that may increase the risk of certain ovarian cancers, particularly clear-cell and endometrioid subtypes.

Objective: To review subtype-specific risks, molecular mechanisms, and clinical risk stratification of endometriosis-associated ovarian cancer.

Method: An integrative literature review was conducted, covering studies published between 2003 and 2025. Databases searched included PubMed, Scopus, Web of Science, EMBASE, and the Cochrane Library. Keywords included "endometriosis," "ovarian cancer," "endometrioma," "malignant transformation," "risk stratification," and "molecular pathways." Both epidemiologic and molecular studies reporting on endometriosis and epithelial ovarian cancer were included.

Results: Ovarian endometriomas showed the highest potential for malignant transformation. Chronic inflammation, hormonal imbalances, oxidative stress, and mutations in ARID1A, PIK3CA, and PTEN contribute to oncogenic progression. Clinical and imaging features, including cyst size, growth rate, and solid components, help identify high-risk patients.

Conclusion: Risk-stratified monitoring and early intervention can improve detection and outcomes, and future research should integrate molecular and clinical data for personalized management of endometriosis.

Keywords: Endometriosis, Risk Factor, Epithelial Ovarian Cancer, Endometrioma, Clear cell carcinoma.

1. Introduction

Endometriosis is a prolonged, estrogen-dependent, inflammatory condition characterized by the formation of ectopic endometrial glandular and stromal cells outside the uterus [1]. Approximately 5% to 15% of reproductive-age women are affected by endometriosis, which is linked with dysmenorrhea, chronic pelvic pain, dyspareunia and infertility [2]. Endometriosis, a benign gynaecological disorder, has similar pathophysiological similarities to cancer. Furthermore, histopathological and epidemiological results imply that ovarian endometriosis may sometimes lead to the formation of epithelial ovarian cancer, also called endometriosis-associated ovarian cancer (EAOC) [3].

Ovarian cancer is grouped based on the type of cell it originates from, which can be epithelial, germ cell, or stromal cell [3]. In most cases, about 85%, develop from the epithelial layer covering the ovaries. EAOCs form a rare subgroup within these epithelial cancers. Endometriosis is found in 30%-55% of ovarian clear cell carcinoma (CCC) cases and in 30%-40% of ovarian endometrioid carcinoma (EC) cases; therefore, EAOCs consist of clear cell and endometriosis types [1, 3, 4]. Epithelial ovarian cancer remains one of the most lethal forms of gynecologic malignancy due to its late detection and vague early symptoms. EOC is categorized into various subtypes such as high-grade serous, low-grade serous, endometrioid, clear-cell, mucinous, and transitional cell carcinoma.

Although the total lifetime risk for ovarian cancer in women with endometriosis remains low, the literature reveals that the risk is marginal but significantly greater than in those without the illness [5]. Several demographic cohort studies and case-control analyses have validated this link during the previous two decades [6-8]. Although only a small number of women experience this malignant transformation, there is increasing interest in understanding why benign endometriosis might develop into ovarian cancer due to the potential long-term consequences [9]. Despite years of scientific research, the pathophysiology of endometriosis is still not fully understood, with numerous mechanisms

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proposed. The Sampson theory of retrograde menstruation proposes that endometrial cells can reflux via the fallopian tubes and implant in pelvic tissues, although this does not completely explain why endometriosis affects only a fraction of women despite broad retrograde flow [10, 11]. Alternative ideas include coelomic metaplasia, hereditary vulnerability, immunological dysfunction, and hormonal abnormalities [12-14]. Endometriotic lesions require estrogen for growth and are resistant to progesterone [15]. Inflammation caused by cytokines, adipokines, and angiogenic agents, including VEGF, IL-1β, and TNF-α, leads to lesion creation, pain, fibrosis, and infertility [16]. Deep penetrating endometriosis, ovarian endometriomas, and superficial peritoneal lesions are clinically different subgroups with varied symptom severity and cancer risk [17, 18]. Endometriosis and endometriosis-associated ovarian cancer share genetic and epigenetic changes, including mutations in ARID1A, PIK3CA, and PTEN, as well as dysregulation of Wnt, NOTCH, and TGFβ pathways. Non-coding RNAs, miRNAs, and ubiquitin pathway regulators, including ITGB3 and ITCH, also influence lesion invasiveness and proliferation [19]. These molecular and cellular alterations, along with oxidative stress and chronic inflammation, provide a probable mechanistic relationship to malignant transformation, emphasizing the importance of subtype-specific risk assessment and close clinical monitoring.

The rationale for this review is to synthesize current evidence regarding the association between endometriosis and EOC in a clinically meaningful framework. This review aimed to synthesize current knowledge on the oncogenic potential of endometriosis and propose a clinical risk-stratification approach.

2. Methodology

2.1. Research Design

This review used an integrative review method to gather and summarize information about the link between endometriosis and EOC. The process followed five major steps described by Whittemore and Knafl in 2005 [20]: identification of the main problem, searching, checking the quality of information, analysis, and the presentation of results. This strategy allowed the inclusion of several types of studies in this review, including those that use numbers, interviews, or mixed approaches. This approach was adopted to clearly outline a comprehensive description of how endometriosis may be associated with ovarian cancer, including biological factors, clinical patterns, and management-related insights.

2.2. Search Strategy

This review collected information from peer-reviewed journals and important search databases, such as PubMed, Scopus, Web of Science, Medline, EMBASE, the Cochrane Library, and Google Scholar. Studies published between 2003 and 2025 were included. It focused on identifying studies discussing endometriosis, its biological behavior, and its potential progression to epithelial ovarian cancer. Keywords included, but were not limited to, the following: “endometriosis,” “ovarian carcinoma,” “endometrioid carcinoma,” “clear cell carcinoma,” “malignant transformation,” “risk stratification,” and “molecular pathways.” Boolean operators like AND, OR, and NOT have been used to enhance precision in the search for highly relevant studies. The sources used in the search and the keywords are outlined in Table 1.

Table 1. Data Selection Strategy

Years	Search Engines	Keywords
2003- 2025	✓ Google Scholar	✓ Endometriosis
	✓ Scopus	✓ Ovarian carcinoma
	✓ Web of Science	✓ Endometrioid carcinoma
	✓ PubMed	✓ Clear cell carcinoma
	✓ MDPI	✓ Risk Factor
	✓ Medline	✓ Malignant transformation
	✓ Medscape	✓ Carcinogenesis
	✓ Cochrane Library	✓ Epithelial ovarian carcinoma
	✓ EMBASE	

2.3. Search Strings

The following sample search strings were used to search the databases and ensure a thorough and efficient literature retrieval process:

"Endometriosis" AND "ovarian cancer" AND "malignant transformation"

"Endometrioma" OR "epithelial ovarian carcinoma" AND "risk factors" AND "clear cell carcinoma"

"Endometriosis" AND "molecular pathways" OR "ARID1A mutation" AND "carcinogenesis"

"Endometriosis subtypes" AND "oncogenic potential" AND "endometrioid carcinoma" OR "deep infiltrating endometriosis"

2.4. Eligibility Criteria

2.4.1. Inclusion and Exclusion Criteria

Full-text research articles in English that reported on the association between endometriosis and EOC were included in this review. Acceptable studies were those related to epidemiology, molecular alterations, or clinical characteristics that linked the two diseases. Articles were also excluded if they did not include primary data or if they were not subject to peer review. Case reports, editorials, letters, conference abstracts, and articles limited to animal studies were excluded. Articles related to cancers other than EOC were excluded. These criteria guarantee that only reliable, relevant, and high-quality studies were used to support the aims of this review.

2.5. Study Selection and Evaluation

Titles and abstracts of all the articles retrieved from the search were first screened for relevance. Full texts of relevant studies were then read. The approach described by Whittemore and Knafl (2005) was adopted to determine the quality of each study and the clarity and reliability of the information provided. References in selected papers were also consulted (snowballing) to identify other useful studies.

2.6. Data Extraction and Analysis

Information on the study design, participants, type of endometriosis, subtypes of ovarian cancer, molecular alterations, and clinical outcome of each study was collected. The results are then described in simple narrative form to show what is known and what gaps still exist.

3. Subtypes of Endometriosis and Their Oncogenic Potential

Endometriosis may manifest differently, and the risk of progressing to cancer appears to vary by each subtype. Ovarian endometriomas (OMAs) are the cystic form of the ovary and are the most strongly related to clear-cell and endometrioid ovarian cancer [21]. One large, population-based study discovered that women with ovarian endometrioma had a strongly increased standardized incidence ratio for clear-cell (5.17) and endometrioid carcinoma (3.12) [22]. Deep infiltrating endometriosis (DIE), which deeply infiltrates pelvic tissues, similarly demonstrates a very high adjusted hazard ratio (aHR) for ovarian cancer-aHR up to 18.76 [5]. The aHR is lower but still meaningful for superficial peritoneal endometriosis-aHR ~ 2.82. Again, these data emphasize that not all endometriosis is alike [5]. The risk of ovarian cancer depends greatly on the subtype, suggesting that subtype-specific risk models may be warranted for better clinical monitoring.

4. Pathophysiological Links Between Endometriosis and EOC

Some of the pathophysiological mechanisms linking endometriosis with EOC concern malignant transformation. Inflammation is at the core of these processes, given that ectopic endometrial tissue is known to secrete proinflammatory cytokines like IL-1β, TNF-α, and IL-6, stimulating local immune cells and leading to the proliferation of epithelial and stromal cells in the endometriotic lesion [23-25]. The oxidative stress, which is triggered by the rise in the levels of ROS, is particularly high in ovarian endometrioma, where iron is deposited as a result of repeated hemorrhages [26]. Overload of iron enhances hydroxyl radical activation by the Fenton reaction, which causes oxidative DNA damage and increases mutational burden, thus promoting oncogenesis [27, 28]. The contributions of hormonal imbalancing, both enhanced local estrogen synthesis in endometriotic tissue by aromatase upregulation and inhibited effects of progesterone on normal apoptotic regulation of abnormal endometrial growth and survival, and compromised immune mechanisms, such as natural killer cells, allow the proliferation and survival of abnormal endometrial cells [23, 29, 30]. There is evidence supporting a stepwise model of transformation whereby benign endometriosis first develops atypical cytologic features-nuclear enlargement and architectural atypia-followed by development of an endometriosis-associated ovarian carcinoma, usually clear-cell or endometrioid subtypes [6, 31]. The knowledge

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of such mechanisms highlights the need to focus on identifying individuals with high-risk lesions and possibly guide preventive measures, early diagnosis, and specific treatment to interrupt the inflammatory, oxidative, and hormonal processes in endometriosis to lower the risk of EOC.

5. Mechanistic Pathways Linking Endometriosis to Ovarian Carcinogenesis

Endometriosis can also play a role in the formation of ovarian carcinogenesis by a variety of interrelated mechanisms and pathways. The persistent inflammation in the endometriotic lesions forms a cytokine-filled microenvironment, which comprises interleukin-1B, tumor necrosis factor-alpha, and interleukin-6, which enhance epithelial growth and tissue regeneration [23]. This process is also aggravated by oxidative stress, which causes iron build-up and the formation of reactive oxygen species (ROS) with repeated hemorrhage in ovarian endometriomas, thus resulting in DNA damage and increasing the risk of mutations [32].

Hormonal factors play a central role. Endometriotic tissues overexpress aromatase, producing elevated local estrogen levels, while progesterone resistance disrupts normal apoptotic pathways, favoring persistent growth of atypical epithelial cells [33]. The genetic and epigenetic variations play a crucial role in the malignant transformation. Activating mutations of PIK3CA and evidence of microsatellite instability have often been found to accompany mutations in tumor suppressor genes (ARID1A and PTEN) in endometriosis-associated ovarian cancers, especially the clear-cell and endometrioid types [34, 35].

The development of tumors is also achieved through immune evasion and angiogenesis. The natural killer cells and cytotoxic T-cells are found to be defective in the endometriotic lesions, enabling the proliferation of abnormal cells to escape immune control. Simultaneously, angiogenesis necessitates the vascular endothelial growth factor (VEGF) that facilitates the longevity and proliferation of lesions, due to the provision of the necessary vascular supply to tumorigenic progression [36].

6. Clinical and Imaging Features Suggestive of Malignant Transformation

The rare but clinical impact of malignant transformation of endometriosis into EAOC that is especially evident in clear-cell and endometrioid adenocarcinoma requires careful consideration of suspicious clinical and radiographic features to enable early diagnoses of the complication [37, 38]. The rapid growth of a cyst, its persistent presence in a postmenopausal patient, and reappearance of a cyst or pelvic symptoms following a certain treatment are important warning signs that require further investigation [37, 39]. Early diagnosis is most important, because EAOC can occur in women with long-term benign endometriosis two to three decades prior to the development of de novo ovarian carcinoma [37, 39]. Radiologic characteristics that indicate the development of malignant transformation in an endometrioma are mostly linked to the development of solid masses in ultrasound (US) and magnetic resonance imaging (MRI). Major alarming observations are papillary projections into the cyst lumen, an enhancing solid mural nodule in the cyst, and the change or loss of the typical “shading sign” or T2-shortening that normally signifies a benign endometrioma [40]. MRI is regarded as the most selective layer in evaluating EAOC, and some of the features they focus on include constriction of diffusion, nodular septations, and solid enhancing nodules (pivotal to the subtraction imaging) [38]. The high predictive power of the modern imaging methods, including MR relaxometry, also helps to support early detection [37]. General gynecologists should have profound knowledge of these clinical and imaging signs because they will help to identify the possible malignant process in time and refer patients to gynecologic oncology specialists to obtain the definitive histopathologic results and timely treatment [41].

7. Risk Stratification Framework

Clinicians can determine who needs early surgery or extended monitoring by using risk stratification. Research indicates that ovarian endometriomas in younger women (less than 40 years old) with small cysts (less than 5 cm) and no solid areas have a minimal immediate risk and can be treated promptly with routine follow-up [41-43]. On the other hand, patients are classified as intermediate or high-risk if they have larger cysts (≥5–8 cm), are older (perimenopausal or postmenopausal), expand fast, or have new papillary or solid mural nodules. According to large cohort data, endometriomas with suspicious characteristics and severe forms, including deep infiltrating disease, have a much greater relative risk [5]. For instance, age and cyst size (often ≥8 cm) were found to be independent predictors of malignant transformation in another study [37], whereas one population study indicated significantly higher risk

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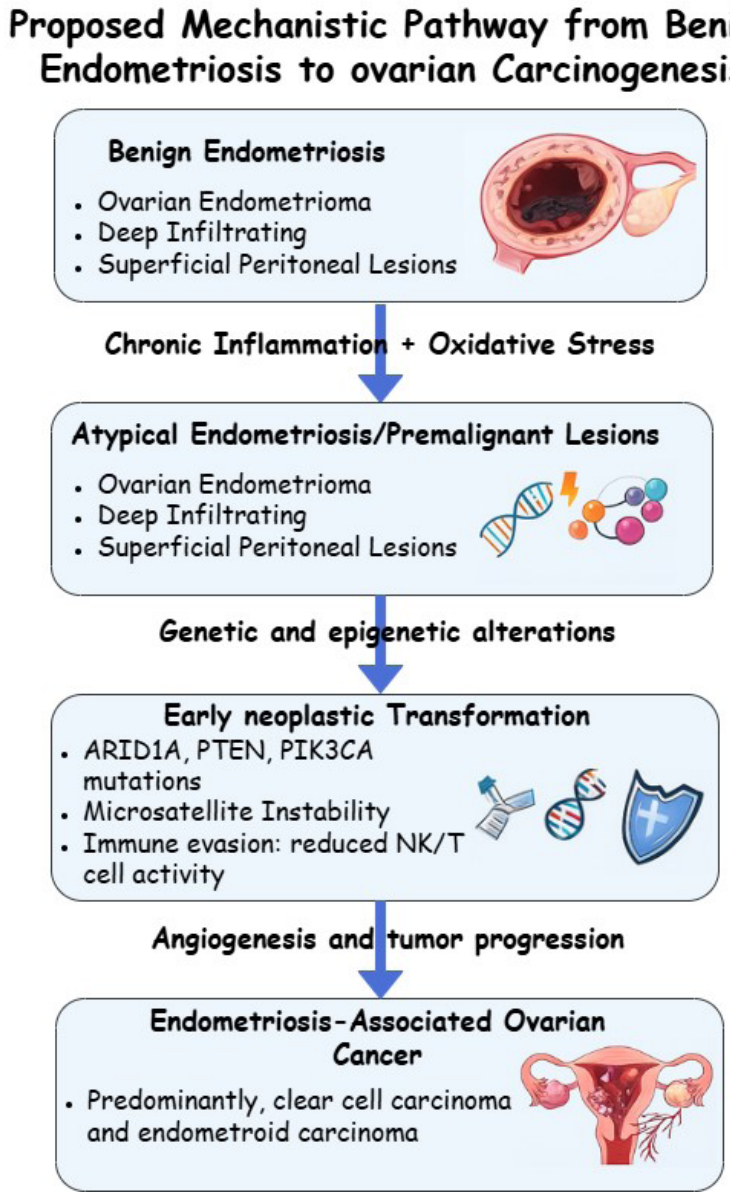

Figure 1. Sequence of events from benign endometriosis to epithelial ovarian cancer

ratios for malignant outcomes in severe endometriosis [44].

Thus, a practical risk stratification model could be:

- Low risk: young women (< 40 years), small cysts (< 5 cm), no solid or papillary features, periodic follow-up;
- Moderate risk: perimenopausal, cysts ≥ 5–8 cm, recurrent lesions, annual imaging;
- High risk: postmenopausal, rapid cyst growth, papillary or solid changes, surgical referral and closer evaluation.

Ultrasound and MRI scans that show blood flow in solid parts or small bumps called papillary projections clearly indicate the need for surgical evaluation [45]. Current evidence is used to create a practical approach, the stratification of patients based on the three risks levels, in which low risk is characterized by younger women with small, stable cysts that lack solid constituents, and which thus only require routine surveillance with ultrasound examination and clinical assessment [46]; moderate risk is characterized by perimenopausal women with cysts 5 cm and above or recurrent lesions and high risk is characterized by postmenopausal women or those with cysts showing rapid growth, solid or papillary changes with annual ultrasounds or MRI scans [47]; high risk includes postmenopausal

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women, or those with cysts that grow quickly, show solid or papillary changes on imaging, or have elevated tumor markers, who need prompt surgical evaluation and referral to specialists in gynecologic oncology [48]. The presented stepwise approach is consistent with the current reviews and imaging investigations, where the size of lesions, patient age, and the appearance of suspicious radiologic findings are the main factors dictating clinical decisions [40, 49].

8. Preventive and Surveillance Strategies

Reducing symptoms, limiting recurrence, and identifying lesions at increased risk of malignant transformation are the major objectives of endometriosis prevention and surveillance techniques [50, 51]. In order to manage symptoms and perhaps lower lesion activity, medical suppression with combined oral contraceptives or progestins is frequently employed; progestins, including dienogest, are useful for managing pain and have received strong support from recent reviews [52-54]. After surgery, gonadotropin-releasing hormone (GnRH) analogs are helpful as short-term adjuncts, but due to side effects (such as bone loss), they must be used carefully and with add-back therapy [54, 55]. When maintaining fertility and long-term control are top priorities, surgical treatment for ovarian endometriomas suggests cystectomy (complete excision) over simple drainage or surgical ablation [56-58]. This is because cystectomy can result in lower recurrence and better pregnancy outcomes, though it can affect ovarian reserve and should be done with caution by skilled surgeons [58]. Long-term follow-up with regular clinical assessment and targeted imaging is advised following therapy, particularly for individuals with moderate to high risk factors (big cysts, advanced age, suspicious imaging) [59]. In addition, the development of regional registries and uniform data collection would enhance surveillance, aid in the improvement of risk models, and direct population-based preventative measures.

9. Research Gaps and Future Directions

To enhance patient care, there are still major gaps in this field of study that need to be filled. First, prospective cohort data are required to validate risk thresholds found in retrospective investigations and to measure the malignant potential of particular endometriosis subtypes over time [5]. Second, well-known somatic changes such as ARID1A, PIK3CA, and PTEN exhibit potential as biomarkers; however, to identify their practical predictive value, they must be incorporated and examined within clinical risk models [60, 61]. Third, there aren't many affordable, useful imaging-based scoring systems that function in low- and middle-income nations. Converting structured MRI/ultrasound tools (such as #Enzian/Endo-MRI frameworks) into streamlined, resource-sensitive scores could allow for more extensive screening and triage [62, 63]. In order to bridge these gaps, more collaborative efforts between clinical gynecology, oncology, radiology, molecular biology, and public health registries will be needed to develop prospective studies, validate biomarkers, and create regional data platforms that support customized surveillance initiatives [64].

10. Conclusion

Endometriosis, especially when it forms ovarian endometriomas, carries a measurable risk of turning into ovarian cancer, mainly the clear-cell and endometrioid types. Although the overall risk is low, it is higher than in women without endometriosis. This transformation seems to result from a combination of chronic inflammation, hormonal imbalances, and genetic changes in the affected tissue. Different subtypes of endometriosis, such as ovarian endometriomas or deep infiltrating lesions, may have varying levels of risk, highlighting the need for individualized monitoring. Clinicians can use a risk-stratified approach, considering factors like age, cyst size, growth rate, and imaging features, to guide follow-up or early intervention. Regular clinical assessments and imaging, combined with careful management of symptoms, can help detect potential malignancy early and improve outcomes. Future research should focus on integrating molecular and clinical data to provide personalized care for women with endometriosis.

Strengths and Limitations

This review provides a comprehensive synthesis of the association between endometriosis and ovarian cancer, integrating epidemiologic, molecular, and clinical evidence. It highlights subtype-specific risks, mechanistic pathways, and practical risk-stratification approaches that can guide early detection and management. The integrative method allowed combining diverse study types for a broader perspective. However, reliance on published studies, many of which are retrospective, may introduce bias. Limited data on certain endometriosis subtypes, molecular markers, and imaging-based assessments, as well as heterogeneity across populations and methodologies, may affect the

generalizability and applicability of the findings.

Statements and Declarations

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Conflict of Interest

The author(s) declare that they have no potential conflict of interest.

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