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Endometriosis and Women's Reproductive Health: Pathophysiology and Emerging Treatments

Rubena Angeles*

Division of Reproductive Endocrinology and Infertility, North Shore University Hospital, Manhasset, New York, USA

Corresponding author: Rubena Angeles, Division of Reproductive Endocrinology and Infertility, North Shore University Hospital, Manhasset, New York, USA, **E-mail:** angles.ruby@nsu.hospital

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Introduction

Endometriosis is a chronic gynecological disorder characterized by the presence of endometrial-like tissue outside the uterine cavity, most commonly on the ovaries, pelvic peritoneum and other reproductive structures. Affecting approximately 10% of women of reproductive age, it is one of the leading causes of chronic pelvic pain, dysmenorrhea, dyspareunia and infertility. Despite its high prevalence and significant impact on quality of life, endometriosis often remains underdiagnosed and mismanaged due to its heterogeneous clinical presentation and lack of definitive non-invasive diagnostic tools. The disorder is not only a gynecological condition but also a systemic inflammatory disease with endocrine, immune and neurological components. The relationship between endometriosis and women's reproductive health is profound, as it disrupts fertility, compromises ovarian reserve and complicates Assisted Reproductive Technologies (ART). While surgical removal of endometriotic lesions and hormonal therapies remain mainstays of management, recurrence rates are high and side effects often limit long-term use. This article examines the underlying mechanisms linking endometriosis to reproductive dysfunction and explores emerging treatment strategies aimed at improving outcomes and quality of life [1].

Description

The pathophysiology of endometriosis is multifactorial, involving hormonal, genetic, immunological and environmental factors. The most widely accepted theory is Sampson's concept of retrograde menstruation, where endometrial tissue refluxes into the peritoneal cavity and implants on pelvic structures. However, this mechanism alone does not explain the selective survival and proliferation of ectopic endometrial tissue. Aberrant immune responses, such as impaired Natural Killer (NK) cell activity and increased production of pro-inflammatory cytokines, allow ectopic lesions to evade immune clearance. Hormonal dysregulation, particularly estrogen dominance and progesterone resistance, promotes lesion survival and angiogenesis, fueling chronic inflammation and pain. Endometriotic lesions also secrete prostaglandins and growth factors that enhance local nerve fiber proliferation, explaining the severe pain symptoms disproportionate to lesion size in many women. Additionally, oxidative stress and genetic predispositions contribute to altered cellular signaling and lesion persistence. Molecular studies have identified involvement of signaling pathways such as PI3K/AKT, MAPK and Wnt, linking endometriosis to oncogenic potential and endometriosis-associated ovarian cancer. The chronic inflammatory microenvironment not only exacerbates pelvic pain but also interferes with ovulatory function, tubal motility and implantation, significantly impairing fertility. Endometriosis profoundly affects women's reproductive health through both mechanical and biochemical pathways. Ovarian endometriomas also damage ovarian reserve by destroying healthy follicles and triggering premature ovarian failure in severe cases. On a biochemical level, endometriotic lesions alter the peritoneal environment by releasing inflammatory mediators, reactive oxygen species and proteolytic enzymes that compromise sperm motility, oocyte quality and embryo development [2,3].

Implantation failure is another hallmark of endometriosis-related infertility, as endometrial receptivity is impaired by progesterone resistance, altered expression of adhesion molecules and epigenetic modifications. Assisted reproductive technologies (ART), particularly in vitro fertilization (IVF), offer a viable option, but success rates in women with advanced endometriosis are often lower compared to those without the disease. Thus, its implications extend beyond reproduction, underscoring the need for holistic approaches to management. Recent advances in molecular and immunological research have unveiled promising therapeutic targets. Selective progesterone receptor modulators (SPRMs) and aromatase inhibitors offer more tailored hormonal suppression, while oral GnRH antagonists provide effective pain relief with improved tolerability compared to traditional agonists [4].

Beyond hormonal therapies, research is focusing on immunomodulatory and anti-inflammatory agents to counteract the chronic immune activation in endometriosis. Agents targeting angiogenesis, such as anti-VEGF therapies, are under investigation to limit lesion vascularization. Neuromodulators, including drugs targeting nerve growth factor (NGF), show potential in alleviating chronic pain. Non-pharmacological approaches, such as dietary modifications, physical therapy and mindfulness-based interventions, are also gaining recognition for their role in improving quality of life. In reproductive medicine, refinement of ART protocols and use of fertility preservation techniques, such as oocyte or ovarian tissue cryopreservation, provide hope for women struggling with infertility. Future perspectives emphasize precision medicine approaches, integrating genetic, molecular and epigenetic profiling to deliver individualized therapies that minimize recurrence and optimize reproductive outcomes [5].

Conclusion

Endometriosis is a complex disorder with significant implications for women's reproductive health, fertility and quality of life. Its multifactorial pathophysiology involves hormonal, immune and genetic disruptions that promote lesion survival, chronic inflammation and impaired fertility. Traditional treatments offer symptomatic relief but are limited by recurrence and side effects, necessitating the development of innovative therapies. Advances in hormonal agents, immunomodulation, angiogenesis inhibitors and neuromodulatory treatments offer promising directions for more effective and personalized care. Integrating medical, surgical and supportive lifestyle interventions remains essential to managing the diverse impacts of the disease. Continued research and awareness will be pivotal in addressing the unmet needs of women with endometriosis, ultimately improving reproductive outcomes and long-term health.

Acknowledgment

None.

Conflict of Interest

None.

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