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ENDOMETRIOSIS: AN UPDATE (THE POSTCRIPT TO THE 13TH CONGRESS OF THE ASIAN SOCIETY OF ENDOMETRIOSIS, COLOMBO, SRI LANKA, OCTOBER 31 - NOVEMBER 3, 2025)

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HIGHLIGHTS

This mini-review is based on an analysis of scientific presentations, program abstracts, and selected recent publications discussed during ACE-2025, with integration of epidemiological, genetic, molecular, diagnostic, and therapeutic evidence. Despite meaningful progress, endometriosis continues to impose a substantial burden on affected women and healthcare systems worldwide. Future advances will depend on interdisciplinary collaboration, integration of genomic and molecular data into clinical practice, and patient-centered models of care that address both symptom control and long-term health outcomes.

Abstract

Endometriosis is a chronic, estrogen-dependent inflammatory disease that affects approximately 10% of women of reproductive age worldwide and is associated with pelvic pain, infertility, and significant comorbidity. Despite more than a century of research, its pathogenesis, optimal diagnostic pathways, and long-term management strategies remain incompletely understood. **Aim.** To summarize and critically appraise recent advances in the understanding, diagnosis, and management of endometriosis, based on key findings presented at the 13th Congress of the Asian Society of Endometriosis (ACE-2025). **Materials and Methods.** This mini-review is based on an analysis of scientific presentations, program abstracts, and selected recent publications discussed during ACE-2025, with integration of epidemiological, genetic, molecular, diagnostic, and therapeutic evidence. **Results**. Recent data reinforce the concept of endometriosis as a systemic, inflammatory, and fibrotic disorder with substantial heterogeneity. Large population studies confirm prolonged diagnostic delay, high multimorbidity, and significant reproductive impact. Advances in genomics, including genome-wide association studies and polygenic risk score analyses, demonstrate shared genetic architecture with several comorbid conditions. Pathophysiological insights highlight the central roles of estrogen dominance, progesterone resistance, immune dysregulation, and fibrosis. Diagnostic progress includes expert-guided ultrasound, MRI, and emerging multimarker approaches. Therapeutically, long-term medical management has become central, with extended GnRH analogue therapy using add-back regimens and dual progestin systems providing effective options for refractory pain. Special considerations in adolescents and infertility management emphasize early intervention and fertility preservation. **Conclusion.** Endometriosis care is undergoing a paradigm shift toward personalized, multidisciplinary, and long-term management. Continued integration of molecular research with clinical practice is essential to improve outcomes and quality of life for affected women.

Keywords: Endometriosis, Chronic pelvic pain, Infertility, Inflammation, Fibrosis, Genetics, Diagnosis, Hormonal therapy, GnRH analogues, Progestins, The 13th Congress of the Asian Society of Endometriosis (ACE-2025)

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Abbreviations:

ACE-2025 – Asian Society of Endometriosis Congress
ARID1A – AT-rich interactive domain-containing protein 1A
CHC – combined hormonal contraception
CI – confidence interval
COX-2 – cyclooxygenases -2
DCs – dendritic cells
DIE – deep infiltrative endometriosis
DPS – drospirenon
E2 – estradiol
ECGRI – Clinical and Genetic Research in India
ER – estrogen receptors
GnRH – gonadotropin releasing hormone agonists
GWAS – genome-wide association study
HOXA – homeobox A
HPG – Hypothalamic-Pituitary-Gonadal axis
HR – hazard ratio
HSD – hydroxysteroidehydrogenase
IL-1 – interleukin-1
IL-6 – interleukin-6
IL-8 – interleukin-8
IVF-ET – in vitro fertilization-embryo transfer
LNG-IUS – levonorgestrel-releasing intrauterine system
MR – Mendelian randomization
miRNA – microRNA
NETA – northiesterone acetate
NSAID – nonsteroidal anti-inflammatory drug
OR – odds ratio
P4 – Progesterone
PGE2 – Prostaglandin E2
PIK3CA – Phosphatidylinositol 3-kinase
PR A – progesterone receptors A
PR B – progesterone receptors B
PRS – polygenic risk score
RGES – Reverse Gene Expression Score algorithm
SPL – superficial peritoneal lesions
TGF-β – transforming growth factor-β
TIAR – Tissue Injury and Repair Theory
TLH – total laparoscopic hysterectomy
TNF-α – tumor necrosis factor alpha
uNK – uterine natural killer

ОБЗОРНАЯ СТАТЬЯ
АКУШЕРСТВО И ГИНЕКОЛОГИЯ

НОВОЕ ОБ ЭНДОМЕТРИОЗЕ (ПОСТСКРИПТУМ 13-ГО КОНГРЕССА АЗИАТСКОГО ОБЩЕСТВА ЭНДОМЕТРИОЗА, КОЛОМБО, ШРИ-ЛАНКА, 31 ОКТЯБРЯ - 3 НОЯБРЯ 2025 Г.)

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Основные положения

Данный краткий обзор основан на анализе научных презентаций, программных тезисов и отдельных последних публикаций, обсуждавшихся на конгрессе ACE-2025, с учетом эпидемиологических, генетических, молекулярных, диагностических и терапевтических данных. Несмотря на значительный прогресс, эндометриоз продолжает представлять собой проблему для женщин и системы здравоохранения во всем мире. Будущие достижения будут зависеть от междисциплинарного сотрудничества, интеграции геномных и молекулярных данных в клиническую практику и пациентоориентированных моделей оказания медицинской помощи, которые предполагают, как контроль симптомов, так и отдаленных последствия для здоровья.

Резюме

Эндометриоз – это хроническое эстрогензависимое воспалительное заболевание, поражающее примерно 10% женщин репродуктивного возраста во всем мире, проявляющееся тазовой болью, бесплодием и другой сопутствующей патологией. Несмотря на более чем столетнюю историю исследований, его патогенез, оптимальные диагностические пути и долгосрочные стратегии лечения остаются недостаточно изученными. **Цель**. Обобщить и критически оценить последние достижения в понимании, диагностике и лечении эндометриоза на основе ключевых результатов, представленных на 13-м Конгрессе Азиатского общества эндометриоза (ACE-2025). **Материалы и методы**. Данный мини-обзор основан на анализе научных презентаций, программных тезисов и отдельных недавних публикаций, обсуждавшихся на ACE-2025, с учетом эпидемиологических, генетических, молекулярных, диагностических и терапевтических данных. **Результаты.** Последние данные подтверждают концепцию эндометриоза как системного, воспалительного и фиброзного заболевания со значительной гетерогенностью. Крупномасштабные популяционные исследования подтверждают длительную задержку в диагностике, высокую множественную сопутствующую патологию и значительное влияние на репродуктивную функцию. Достижения в геномике, включая полногеномные ассоциативные исследования и анализ полигенных показателей риска, демонстрируют общую генетическую архитектуру с рядом сопутствующих заболеваний. Патофизиологические данные подчеркивают центральную роль доминирования эстрогена, резистентности к прогестерону, иммунной дисрегуляции и фиброза. Прогресс в диагностике включает ультразвуковое экспертное исследование, МРТ и использование новых биомаркеров. В терапевтическом плане центральное место занимает долгосрочное медикаментозное лечение, при этом длительная терапия аналогами ГнРГ с использованием схем заместительной терапии и двойных прогестиновых систем обеспечивает эффективные варианты лечения рефрактерной боли. Особое внимание в лечении подростков и бесплодия уделяется раннему вмешательству и сохранению фертильности. **Заключение.** В лечении эндометриоза происходит смена парадигмы в сторону персонализированного, междисциплинарного и долгосрочного подхода. Дальнейшая интеграция молекулярных исследований с клинической практикой имеет важное значение для улучшения результатов лечения и качества жизни женщин, страдающих этим заболеванием.

Ключевые слова: эндометриоз, хроническая тазовая, бесплодие, воспаление, фиброз, генетика, диагностика, гормональная терапия, аналоги ГнРГ, прогестины, 13-й Конгресс Азиатского общества эндометриоза (ACE-2025)

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Introduction

The 13th Congress of the Asian Society of Endometriosis (ACE-2025) was held in Colombo, Sri Lanka, from October 31 to November 3, 2025.

This event brought together clinicians, surgeons, scientists, allied health professionals, patient advocates, and thought leaders from around the world—united by a common purpose of transforming the future of care for endometriosis and adenomyosis. All specialists gathered here for more than just a congress to shape the future. The theme of this congress, "Empowering Women, Advancing Knowledge: The Future of Endometriosis and Adenomyosis," reflects a dual responsibility. First, to empower women by making them partners in treatment processes and recognizing that women's voices must remain at the center of our efforts. And second, to advance a knowledge revolution based on science, innovation, and interdisciplinary and cross-border collaboration, ensuring our clinical practice evolves in line with rapidly evolving scientific evidence. The ACE 2025 conference showcased cutting-edge research, state-of-the-art surgical and medical strategies, emerging diagnostic tools, and real-world implementation frameworks.

The aim

The aim of this study was to analyze new data on endometriosis from the perspective of the materials of the13th Congress of the Asian Society of Endometriosis (ACE-2025).

Materials and methods

An analysis of materials of the13th Congress of the Asian Society of Endometriosis (ACE-2025).

Results

It is well-known that Endometriosis is the presence of endometrial-like tissue outside of the uterine cavity, is a non-malignant disorder associated with inflammation, chronic pelvic pain, and infertility, affecting 1 in 10 women of reproductive age (over 190 million women worldwide) [1] and an estimated 50 million in India [2]. Endometriosis is currently defined as the presence of endometrial epithelial and stromal cells at ectopic sites; however, advances in research on endometriosis have some authors believing that endometriosis should be re-defined as “a fibrotic condition in which endometrial stroma and epithelium can be identified”. There are several theories on the etiology of the disease, but the origin of endometriosis remains unclear [3, 4] .

The economic impact of this disease in the U.S. is profound, with an estimated annual cost for diagnosis and treatment exceeding $60 billion. However, the existence of endometriosis has been known for over 100 years, our current knowledge of the pathogenesis of the disease remains minimal. Most women with endometriosis report that their pain symptoms emerged during adolescence and young adulthood and approximately 80% of these women have superficial peritoneal lesions (SPL). Further, even with surgical removal 50% re-present with persistent, or recurrent, pain within 5 years [1].

There is emerging evidence that suggests an association between endometriosis and certain ovarian cancer subtypes, mainly clear cell and endometrioid ovarian cancers. The mechanisms underlying this association are not fully understood but may involve chronic inflammation, oxidative stress and hormonal imbalances that can promote malignant transformation in endometriotic tissue. Genetic mutations such as ARID1A loss of function and PIK3CA mutations have been identified in both endometrioses associated ovarian cancers and benign endometriosis lesions suggesting a possible progression pathway [5].

Women with endometriosis had a 7-fold increased risk of surgical menopause (hazard ratio (HR) 7.54, 95% confidence interval (CI) 6.84, 8.32) and were less likely to experience natural menopause (HR 0.40, 95% CI 0.33, 0.49) than women without the condition. On average, surgical menopause occurred 19 months earlier in women with endometriosis. Among women who experienced natural menopause, it was 5 months earlier for those with endometriosis. Women with endometriosis were also twice as likely to experience premature surgical menopause (<40 years) (odds ratio (OR) 2.11, 95% CI 2.02, 2.20) or 1.4 times more likely to develop spontaneous ovarian polycystic ovary syndrome (OR 1.36, 95% CI 1.17, 1.59). They were also at increased odds of early surgical and natural menopause (40-44 years) [6].

Epidemiology of Endometriosis. The Endometriosis Clinical and Genetic Research in India (ECGRI) study, conducted from 2020 to 2024 across 18 centers in nine states included 1,775 surgically and/or histologically confirmed cases of endometriosis and 1,775 unrelated controls were enrolled using standardized instruments adapted from the WERF-EPHect EPQ and SSF. This study showed that in Indian population endometrioma was the most frequent phenotype (59%), with notable geographic variation in lesion types. Advanced-stage disease was more prevalent in public health facilities (73.1%), reflecting delayed access to care. The

mean diagnostic delay was 6.9 years, longest in the Central and Southern zones. Pain (73%) and infertility (46%) were the predominant presentations. Women with endometriosis showed a 7.5-fold higher burden of multimorbidity, most commonly fibroid uterus (34.3%), thyroid disease (17.6%), and adenomyosis (10.6%) [2].

Etiology and pathophysiology of Endometriosis. The etiology of endometriosis is complex and there are several contributing factors leading to the development of this disorder. There are numerous theories that have been put forward to explain the origin of endometriosis: Sampson's Theory (Retrograde Menstruation), Coelomic Metaplasia, Embryonic Rest Theory, Vascular and Lymphatic Metastasis, Tissue Injury and Repair Theory (TIAR), Quinn's "Denervation–Reinnervation" Theory, Stem Cell Theory, Genetic/Epigenetic Theory [3, 4] .

Pathophysiology of Endometriosis includes hormonal dysregulation, estrogen dominance, progesterone resistance, inflammatory response and immune dysregulation, proinflammatory environment: immune cells, T-cells, Macrophages, dendritic cells (DCs), uterine natural killer (uNK) cells, cytokines and growth factors, interleukin-6 (IL-6), interleukin-8 (IL-8), interleukin-1 (IL-1), tumor necrosis factor alpha (TNF-α), transforming growth factor-β (TGF-β) and the Fibrotic Component [3, 4]

Recently, the Endometriosis Initiative Group called for the development of new theories about the pathogenesis and pathophysiology of endometriosis [7].These include genome-wide association studies [8], investigations into microRNA (miRNA) [9] and the microbiome [10] and the molecular assessment of endometrial aberrations [11].

Almost one century after Sampson proposed his retrograde menstruation theory [12, 13], few know he also demonstrated the presence of "bits of endometrium" in uterine vessels during menses, already suggesting that a singular mechanism was not able to explain the variable clinical diseases associated with ectopic endometrium. Many alternative hypotheses are proposed [3, 4, 14−18].

It is proved that genetic, hormonal, immunological, environmental, anatomical factors take place in the natural history of endometriosis. Endometrial stromal cells carry specific epigenetic abnormalities altering expression of key transcription factors which cause estrogen dependent inflammation and deficient expression of progesterone receptor causing P4 resistance. Implants reveal reduced ER-α and upregulated ER-b. Furthermore, loss of PR-B and ability to induce HSD17b2 leads to P4 resistance, PR-A overexpression and higher estrogen activity. Cumulative effect of sequential genetic (low HOXA 10 gene expression) and epigenetic incidents causes higher susceptibility for endometriosis. While DNA hypomethylation ends up with increased COX-2, STAR, CYP19, SF1, ER-b, therefore higher PGE2 and E2, hypermethylation cause suppressed PR-B and HSD17b. Hypothalamic pituitary adrenal axis and stress hormones are also involved in endocrine pathogenesis of endometriosis. Corticotropin releasing hormone and urocortin levels are twice as high in women with endometrioma than in the peritoneal fluid and plasma. Autoimmune thyroid disorders are frequently found in women with endometriosis. Thyroxine triggers ROS production by ectopic endometrial cells, leading to endometriotic cell proliferation [18].

Currently the analysis of large genetic studies across multiple conditions has provided new molecular insights and greater understanding of the relationships between endometriosis and these comorbid conditions. Multiple traits are genetically correlated with endometriosis, suggesting a shared genetic background [19].

Mendelian randomization (MR) uses genetic data to assess causal relationships between traits and has revealed causal relationships for depression and gastrointestinal disorders and effects of genetic risk factors for endometriosis on ovarian cancer and uterine fibroids. However, causality is not responsible for most comorbid relationships. Genetic liability to endometriosis summarized into a quantitative score, known as a polygenic risk score (PRS), is associated with multiple health conditions, blood and urine biomarkers, and reproductive factors, showing that many endometriosis comorbidities are not dependent on disease development [20].

The PRS can be assessed in both males and females and differences in the associated traits also demonstrate the importance of sex-specific pathways in the overlap of endometriosis with many other traits. Result in the UK and Estonian biobanks show the comorbidity burden is significantly higher in endometriosis cases and demonstrate significant interactions between polygenic risk for endometriosis and some comorbidities in the susceptibility to endometriosis [21].

The shared genetic risk factors and potential target genes suggest a role for diverse biological systems. Genetic evidence supports the view that endometriosis is a multisystem disorder and highlights the need for multidisciplinary care [21].

To explore genetic underpinnings, Gajbhiye R.K.

(2025) conducted the first genome-wide association study (GWAS) of endometriosis in an Indian cohort (n=2,578). Analysis identified 17 suggestive genomic loci, with the strongest associations on chromosomes 13 (SHISA2) and 2 (NBAS, DDX1). Replication with European and Asian datasets revealed shared risk loci near WNT4 (chr1) and CDKN2B-AS1 (chr9), confirming cross-population genetic overlap while highlighting novel Indian-specific signals [2].

Fazleabas A. (2025) at the Congress of the ACE-2025 propose that the endometriotic epithelium promotes a local inflammatory environment that sustains peritoneal lesions. Identify conserved inflammatory signaling networks that promote early lesion development. To target inflammatory pathways, we employed the Reverse Gene Expression Score algorithm (RGES) alongside RNA-sequencing data obtained from baboon and human lesions to predict FDA-approved compounds targeting inflammation-related pathways that are altered within the epithelial cells in lesions. Initial studies with selective compounds were tested for efficacy screening using an endometriotic spheroid model. The mechanism of action i.e., proliferation and invasion without inducing cytotoxicity in endometrial spheroids were also determined by RNA-sequencing [1].

According to the opinion, H.Taylor (USA) endometriosis involves the inappropriate growth of endometrium outside of the uterus generated primarily by retrograde menstruation. While most menstrual debris is cleared from the peritoneal cavity by the immune system, endometriosis is specifically sequestered from immune clearance. There is an apparent immune privilege. Simultaneously there is a tremendous inflammatory reaction, with release of multiple inflammatory cytokines. Understanding the immune cell composition of endometriosis is essential to develop new treatments. Targeting inflammatory pathways and well as enabling immune surveillance and clearance will allow for more precise therapy of endometriosis. Understanding endometriosis as an inflammatory disease will also give perspective on the systemic impacts of the disease [22].

It is well recognized that endometriosis is a systemic disease [23] Endometriosis is now considered a systemic disease rather than a disease predominantly affecting the pelvis. Endometriosis affects metabolism in liver and adipose tissue, leads to systemic inflammation, and alters gene expression in the brain that causes pain sensitization and mood disorders. The full effect of the disease is not fully recognized and goes far beyond the pelvis. Recognition of the full scope of the disease will facilitate clinical diagnosis and allow for more comprehensive treatment than currently available.

Moreover, endometriosis at least in a certain proportion of women may behave as other disease characterized by an intrauterine programming alteration. In these diseases, intrauterine exposure can serve as ‘prenatal hit’ able to program the fetal metabolism. After birth, some ‘postnatal hits may favor the development of the disease. Indeed, the early-life environment has been suggested as an important window for endometriosis development as intrauterine exposure to diethylstilbesterol, the synthetic estrogen with the highest affinity for estrogen receptors, and low birth weight have been associated with endometriosis risk. Moreover, a short anogenital distance has been demonstrated in women affected, a positive association was found with maternal smoking during pregnancy, and a positive association was found with preterm birth. In a recent case-control study, we have demonstrated that estradiol levels were significantly higher in samples of umbilical cord blood of endometriosis cases compared to non-affected controls both before and after a propensity score matching. To the best of our knowledge, this was the first study on the levels of steroid hormones in cord blood of women with endometriosis. Estrogenic exposures during the critical timeframe of fetal development promotes the premature activation of HPG axis inducing early age at menarche and early age at menopause, interferes with estrogen-related metabolic enzymes such as aromatase and affect the transcription of estrogen receptors to regulate their number and sensitivity and all these phenomena do occur in women with endometriosis. The ‘post-natal hit’ would be represented by the increased amount of retrograde menstruation due to either the development of adenomyosis or a genetic predisposition to heavy menstrual bleeding. This would be favored by the dramatic changes of the reproductive pattern characteristic of the post-industrial period [24].

Symptoms and diagnosis of endometriosis. Endometriosis is an underdiagnosed disease that is associated with a delay from the onset of symptoms that can take up to 7–8 years before diagnosis [3], and the symptoms can vary widely. Women may be asymptomatic or present with a single symptom or a combination of symptoms with different intensities that can easily be attributed to other conditions [25].

Some of the symptoms that are associated with endometriosis include painful menstruation (dysmenorrhea), cyclical or non-cyclical abdominal

pain, recurrent painful urination (dysuria), pain during and after sexual intercourse (dyspareunia), painful defecation (dyschezia), gastrointestinal discomfort and decreased libido [26]. Currently, there are no reliable biomarkers available to diagnose this disease. The gold standard for the diagnosis of endometriosis has been surgical assessment by laparoscopic visualization [3].

It is well-known that infertility affects one in six individuals globally, with endometriosis contributing to 25–50% of female infertility. Prof. Shaw-Jenq (Sean) Tsai (Taiwan) showed that defective oocytes, unfavorable fertilization environments, and reduced uterine receptivity have been proposed to be associated with low fertility in endometriosis women. However, oocyte donation and in vitro fertilization did not improve much of the pregnancy rate in women with endometriosis, which highlight that uterine receptibility may be the critical factor causing low fertility in endometriosis women. Decidualization is a process of differentiation of endometrial cells into decidual cells, which is a prerequisite for the successful implantation of an embryo in the uterus. We found that women with endometriosis had defective decidualization markers in their endometria, indicating an impairment in decidualization. In depth investigation identified that endometriosis women have less and shorter primary cilia in the endometrial stromal cells. Inhibition of primary cilium formation by ciliogenic genes knockdown or pharmacological disruption hampers endometrial stromal cell decidualization. So, he summarized how primary cilia and uterine receptivity are compromised in women with endometriosis and those who fail to conceive after in vitro fertilization-embryo transfer (IVF-ET) [27].

Laparoscopic investigation of the pelvis is considered the gold standard for endometriosis diagnosis; however, surgical procedures carry risks and are not suitable for all patients. Advances in imaging technologies, including ultrasound and MRI, have improved non-invasive diagnosis, particularly for deep infiltrating disease [28].

The basic method for diagnosing endometriosis is ultrasound. Alborzi S. (Iran) demonstrated that TVS and TRS have appropriate diagnostic accuracy in diagnosis of DIE comparable to MRI [29]. Sopova Y. (Russia) demonstrated a step by step in ultrasound examination deep infiltrating endometriosis [30].

Recently, much attention has been paid to proteomic and metabolomic biomarkers, which are powerful tools for assessing the molecular heterogeneity of endometriosis and may lead to the discovery of new diagnostic and prognostic biomarkers for endometriosis [31].

A plethora of potential biomarkers for the diagnosis of endometriosis have been proposed, including proteins, nucleic acids, metabolites and hormones. Of note, a salivary signature of 109 microRNA molecules has entered a multicenter external validation study, with promising preliminary results. It is likely that future diagnostic tests will embrace multimarker approaches that also integrate clinicodemographic features and other diagnostic outputs with the aid of machine learning. To achieve this, harmonization of sample collection and processing procedures across countries and societies is imperative to create robust diagnostic tests that are accurate and translatable. Such a test could revolutionize endometriosis care and significantly improve patient's quality of life, whilst also reducing the diagnostic burden on healthcare providers [28].

Prof. Guo S.W. (China) posit that the degree of lesional fibrosis, which can be quantitatively assessed via elastography as lesional stiffness, represents a strong candidate biomarker for measuring progression. This metric is compelling due to its established associations with aberrant cellular histology, molecular alterations, symptom severity, and clinical prognosis [32].

Management of endometriosis. Endometriosis management involves managing its various symptoms such as pain and infertility and includes medical and surgical treatments. The management of endometriosis-related pain remains controversial—balancing the surgical "knife" and medical "pill" approaches requires careful patient-centered decision-making. Prof. M. Al-Jefout (UAE) demonstrated that surgical excision effectively relieves pain and restores anatomy but carries a recurrence risk of 40–50% at five years without postoperative hormonal suppression. Medical therapy using combined oral contraceptives, progestins, and GnRH antagonists achieves substantial pain reduction in most patients. The DPS provided sustained relief in 85% of women with refractory disease, while extended GnRH agonist therapy with NETA add-back maintained efficacy and bone health for 24 months. Combining surgery with postoperative hormonal therapy offers the best durability of results. So, endometriosis-related pain management demands individualized, multidisciplinary strategies. Integrating precise surgery with prolonged hormonal suppression, or employing novel dual progestin systems, offers a pragmatic and effective long-term pathway for women

with refractory or recurrent pain [33, 34, 35].

Non-hormonal treatment options are urgently needed to manage endometriosis-associated pain. Prof. Andrew Horne (UK) demonstrated how can we use a combination of ex-vivo, in-vitro and in-vivo models of endometriosis to identify promising new drug candidates and advance them into early-phase clinical trials. He showed innovative therapeutic strategies that target altered cellular metabolism within the peritoneal microenvironment, modulate the endocannabinoid system, and reprogram endometriosis-associated macrophages [35].

Recently, the understanding of endometriosis has undergone a dramatic transformation. Hormonal therapies and assisted reproductive technology have emerged as first-line treatments, dethroning the once-central role of surgery. Non-invasive diagnosis of the disease has spread. This shift marks a notable evolution in how the disease is managed. However, high-throughput technologies have failed to deliver transformative insights, and the root causes of the disease remain as elusive as ever. Despite the setbacks, the progress made offers hope and direction [36].

The results of the study by A. Popov et al., which included more than 2,999 surgical procedures for deep endometriosis, demonstrated that performing surgical interventions on the colon is associated with a number of possible perioperative complications and postoperative functional disorders, which are leveled out after 6-12 months. Functional disorders after the surgery, such as complete emptying of the bladder, difficult urinating, rapid stools, constipation and dyschezia were the highest in the first 2 weeks of the postoperative period and lasted up to 2 months, the complete relief of functional disorders occurred 12 months after the surgery. Intraoperative complications occurred in 14 (5.9%) cases with laparoscopic access and in 8 (3.3%) cases with robot-assisted access. A recurrence of colorectal endometriosis, which required the repeated surgical intervention, occurred in two patients (8%) [37].

The most critical challenge in TLH is the frozen pelvis, characterized by dense adhesions, distorted anatomy, and a high risk of complications. There is no single standardized technique for TLH in a frozen pelvis. Successful surgery depends on sound anatomical knowledge, blunt and sharp dissection techniques, and the application of practical tips and tricks to navigate altered pelvic anatomy. When adhesolysis cannot be achieved with sharp or blunt dissection, a retroperitoneal approach becomes essential to secure the ureters bilaterally and carefully separate the sigmoid colon from the posterior uterus and uterosacral ligaments. The pararectal, paravesical, and vesico-uterine spaces serve as key avascular entry points that facilitate safer dissection. The use of vasopressin injected into the posterior uterine wall helps to minimize oozing, maintain a clear operative field, and enable hydro dissection [37].

Management of Endometriosis in adolescents. There are three specific treatment goals for adolescents with endometriosis: control of symptoms, prevention of further progression of the disease, preservation of reproductive function [*39*]. Pathogenic and clinical goals of menstrual suppression in the period from symptom onset to conception seeking [40]:

1.Restore physiological amenorrhoea.

2. Stop cyclic, reiterative uterine auto-traumatization.

3. Limit pelvic exposure to refluxing endometrial glands.

4. Reduce pelvic iron overload and oxidative stress by reducing transtubal retrograde menstruation.

5. Stop repeated inflammatory events both at the endometrial–myometrial junction and on the peritoneal surface of pelvic structures.

6. Decrease the oestrogenic pro-inflammatory effect and increase the progestogenic anti-inflammatory effect.

7. Relieve dysmenorrhoea and improve health-related quality of life.

8. Limit the potential progression of clinically diagnosed superficial peritoneal endometriosis towards infiltrating, fibrotic lesions.

9. Avoid premature surgery and ovarian damage.

10. Preserve reproductive potential.

11. Limit the potential transition from repetitive acute pelvic pain events to chronic pelvic pain through the development of central sensitization.

According to Russian clinical guidelines on endometriosis (2024), treatment of endometriosis in adolescents should be carried out in consultation with a pediatrician. Adolescents with DIE should be treated in expert centers. Medical treatment for endometriosis in adolescents may include NSAIDs for the pain management as first-line therapy, progestogens, combined hormonal contraceptives (CHC). GnRH agonists is not recommended for patients under 18 years of age. If they are prescribed, add-back therapy, monitoring of vitamin D and calcium in the blood serum, and densitometry are mandatory. CHC and dienogest seem to be suitable, safe, and well-accepted treatments. Indications for surgical treat-

ment of endometriosis in adolescents are refractory pain syndrome and ovarian mass. Hormonal therapy should not be used alone in the following cases: suspected obstructive anomaly, endometrioma or deep nodular endometriosis, acute complication (e.g. torsion), patient\parental preference for diagnosis conformation. So, medical therapy is the first choice for symptomatic endometriosis in adolescent population, considering the surgical approach only for selected cases or for patients unresponsive to medical treatment. Probably, the early diagnosis and the use of adequate medical therapies should result in less extensive surgery in adult life [41].

According to the opinion dr. Yu-Chen H. (China) adenomyosis leads to abnormal uterine peristalsis and decreased endometrial receptivity, contributing to infertility. Atosiban, an oxytocin and vasopressin receptor antagonist, may have therapeutic potential by reducing uterine contractions, improving uterine blood flow, and enhancing endometrial receptivity. Studies have shown that Atosiban infusion one hour before embryo transfer can decrease uterine contractions, and meta-analyses suggest it may improve clinical pregnancy rates in both observational and randomized trials [42]. It is therefore of interest to investigate whether Atosiban could improve pregnancy outcomes during embryo transfer in patients with adenomyosis. Given that adenomyosis is linked to poorer IVF outcomes in Taiwanese studies [43], the use of Atosiban during embryo transfer may reduce early miscarriage rates and improve pregnancy outcomes, particularly in patients with diffuse adenomyosis [44].

Recurrence after endometriosis surgery affects up to 40% of patients within five years. Hormonal suppression remains the mainstay of postoperative management, with the levonorgestrel-releasing intrauterine system (LNG-IUS), dienogest, and continuous oral contraceptives providing the strongest evidence for reducing recurrence and pain. Long-term therapy (≥ 12 months) outperforms short-term regimens, and sequential GnRH-based strategies offer additional benefit. No non-hormonal interventions are currently proven to prevent recurrence. Perioperative β-blockers and COX-2 inhibitors show biological plausibility but lack clinical validation. Non-pharmacologic measures such as acupuncture or dietary modification may improve symptoms but not recurrence risk [45].

Despite the high incidence of infertility in endometriosis and the obvious successes in its treatment, the characteristics of the course of pregnancy and childbirth in these patients remain incompletely understood. Patients treated for endometriosis-associated infertility were characterized by complicated pregnancy and childbirth with a higher incidence of placenta previa, weakness of labor, postpartum hemorrhage, retained placenta, and cesarean delivery. Hence, the development of respective therapeutic and preventive measures is required to prevent these complications [46, 47, 48].

Discussion

This mini-review synthesizes key advances in endometriosis research and clinical practice presented at the 13th Congress of the Asian Society of Endometriosis (ACE-2025), reinforcing the evolving concept of endometriosis as a chronic, systemic, inflammatory, and fibrotic disease rather than a condition confined to the pelvis. Across epidemiological, molecular, diagnostic, and therapeutic domains, the data collectively highlight the heterogeneity of disease phenotypes and the need for personalized, multidisciplinary care.

Epidemiological data, particularly from large, well-characterized cohorts such as the ECGRI study, confirm the substantial diagnostic delay, high burden of pain and infertility, and significant multimorbidity associated with endometriosis. Geographic variation in phenotypes and disease stage underscores disparities in healthcare access and emphasizes the importance of standardized diagnostic pathways and early intervention strategies. These findings align with global observations and reinforce that delayed diagnosis remains a major contributor to disease progression and reduced quality of life.

Advances in genetics and molecular biology presented at ACE-2025 provide compelling evidence for a strong heritable component and shared genetic architecture between endometriosis and several comorbid conditions. Genome-wide association studies, polygenic risk score analyses, and Mendelian randomization approaches collectively demonstrate that endometriosis shares biological pathways with ovarian cancer, fibroids, depression, and gastrointestinal disorders. These findings support the concept of endometriosis as a multisystem disorder and offer potential for future risk stratification, early identification, and targeted prevention strategies.

From a pathophysiological perspective, congress data further consolidates the central role of estrogen dominance, progesterone resistance, immune dysfunction, and fibrosis in disease establishment and persistence. Emerging models integrating epigenetic dysregulation, aberrant inflammatory signaling, and impaired immune clearance help reconcile classical

theories such as retrograde menstruation with contemporary molecular insights. The increasing recognition of lesional fibrosis as a driver of pain, progression, and treatment resistance represents a particularly important conceptual advance, with elastography-based stiffness measurements emerging as a promising biomarker of disease severity and prognosis.

Diagnostic innovation remains a critical focus. While laparoscopy continues to represent the historical gold standard, high-resolution transvaginal and transrectal ultrasound and MRI now allow accurate, non-invasive diagnosis of deep infiltrating disease in expert hands. Parallel advances in proteomics, metabolomics, microRNA profiling, and microbiome research suggest that multimarker, machine-learning–assisted diagnostic tools may soon transform early detection and monitoring, although robust external validation and international harmonization remain essential.

Therapeutically, the studies reviewed illustrate a clear shift toward long-term medical management as the cornerstone of care, with surgery increasingly reserved for selected indications. Hormonal suppression strategies—including continuous progestins, GnRH analogues with add-back therapy, and novel combinations—demonstrate durable pain control with acceptable safety profiles. Notably, extended GnRH agonist use with add-back therapy and the double progestin system offer effective options for women with refractory or recurrent pain, addressing a major unmet clinical need. At the same time, emerging non-hormonal approaches targeting inflammation, immune modulation, and cellular metabolism highlight promising future directions.

Special populations, particularly adolescents and women seeking fertility, require tailored management. Evidence supports early medical therapy to control symptoms, limit progression, and preserve reproductive potential in adolescents, while minimizing premature surgical intervention. In infertility and assisted reproduction, growing data emphasize the importance of uterine receptivity, decidualization, and myometrial function, with adjunctive therapies such as oxytocin receptor antagonists offering potential benefit in selected patients, particularly those with adenomyosis.

Despite these advances, significant knowledge gaps persist. High-throughput technologies have yet to deliver definitive insights into disease origin or a cure, and no non-hormonal strategy has proven effective in preventing postoperative recurrence. The data reviewed nonetheless point toward a future in which integrated genomic, molecular, imaging, and clinical approaches enable earlier diagnosis, individualized treatment, and improved long-term outcomes.

Future Directions for Research

Despite substantial advances in understanding endometriosis, several critical research priorities remain. First, elucidating the precise origins of the disease requires integrative models that combine genetic susceptibility, epigenetic regulation, immune dysfunction, and environmental exposures across the life course. Longitudinal birth-cohort studies and transgenerational research are particularly needed to clarify the role of prenatal and early-life programming in disease development.

Second, future efforts should focus on translating molecular and genomic discoveries into clinically actionable tools. The development and validation of non-invasive diagnostic tests based on multimarker panels—including microRNAs, proteomic and metabolomic signatures, and microbiome profiles—represent a major unmet need. Harmonization of biospecimen collection, analytical platforms, and international data sharing will be essential to enable reproducible and scalable diagnostics.

Third, advancing precision medicine in endometriosis requires stratification of patients according to molecular, genetic, and fibrotic phenotypes. Polygenic risk scores, combined with imaging-based biomarkers such as lesional stiffness, may allow individualized prediction of disease progression, treatment response, and recurrence risk. Such approaches could guide personalized therapeutic strategies and optimize long-term outcomes.

Fourth, there is an urgent need to expand research into non-hormonal therapies targeting inflammation, immune modulation, neurogenic pain pathways, and fibrosis. Preclinical models that better reflect human disease heterogeneity should be prioritized to accelerate translation into early-phase clinical trials.

Finally, future research must incorporate patient-reported outcomes, quality-of-life measures, and long-term safety data, particularly in adolescents and women requiring prolonged treatment. Multidisciplinary, patient-centered research frameworks will be key to transforming endometriosis care from symptom control toward disease modification and prevention.

Concluding Remarks

The evidence reviewed from the 13th Congress of the Asian Society of Endometriosis underscores a fundamental shift in the understanding of endome-

triosis—from a localized gynecological condition to a chronic, systemic, inflammatory, and fibrotic disease with wide-ranging clinical consequences. Advances in epidemiology, genetics, molecular biology, and imaging have clarified the complexity and heterogeneity of the disorder, while simultaneously exposing persistent gaps in knowledge regarding disease origin, progression, and prevention.

Contemporary management strategies increasingly prioritize long-term medical therapy, individualized hormonal suppression, and fertility-preserving approaches, with surgery reserved for selected indications. Innovations such as extended GnRH analogue therapy with add-back regimens and dual progestin systems provide effective and sustainable options for women with refractory symptoms, reflecting a move toward precision and durability in care. Parallel progress in non-invasive diagnostics and biomarker discovery offers promise for earlier detection and improved monitoring, although clinical translation remains incomplete.

Despite meaningful progress, endometriosis continues to impose a substantial burden on affected women and healthcare systems worldwide. Future advances will depend on interdisciplinary collaboration, integration of genomic and molecular data into clinical practice, and patient-centered models of care that address both symptom control and long-term health outcomes. Continued global research efforts, such as those highlighted at ACE-2025, are essential to transforming endometriosis care from symptom management toward prevention, disease modification, and ultimately, cure.

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Эл-Джефут Моамар: разработка концепции и дизайна исследования, редактирование, полная ответственность за содержание.

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Все авторы утвердили окончательную версию статьи.

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Al-Jefout Moamar: development of the concept and design of the study, editing, fully responsible for the content.

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