



# Evaluating the Role of Autoimmunity in Endometriosis Patients

Fatemeh Davari Tanha<sup>1</sup>, Zahra Kaveh<sup>2</sup>, Maryam Marghoub<sup>1\*</sup>, Mehrshad Abdoli<sup>3</sup>, Ghazal Sahraeian<sup>1</sup>, Razieh Akbari<sup>4</sup>

## Abstract

**Objectives:** Endometriosis is a chronic inflammatory disease and one of the common causes of infertility in women. Increasing evidence suggests that the pathogenesis of endometriosis is not limited to hormonal changes, but also that immune dysregulation plays a key role. The present study aimed to investigate the presence of autoimmunity in the ovarian reserve of patients with endometriosis.

**Materials and Methods:** In this case-control study, 200 women were selected between April 2024 and April 2025. Based on endometriosis, patients were split into two groups: 120 infertile women without endometriosis and 80 infertile women with endometriosis. All patients had blood samples measured for antibodies to antinuclear antibodies (ANA), to anti-thyroid peroxidase (ATPO), to lupus anticoagulant (LAC), and to antiphospholipid antibodies (APLA). Ovarian reserve was evaluated by measuring anti-Müllerian hormone (AMH). Clinical signs and disease severity were recorded. The data were analyzed using SPSS version 22, with a significance level of 0.05.

**Results:** Patients were 34.95±5.6 years of age and had a body mass index (BMI) of 25.20±3.56 kg/m<sup>2</sup>. The Adhesion ( $P<0.0001$ ), sliding ( $P<0.0001$ ), kissing ovary ( $P<0.001$ ), and uterosacral involvement ( $P<0.0001$ ) were all significantly more frequent in the group with endometriosis. It showed that the prevalence of ANA (26.3% vs. 10.1%;  $P=0.003$ ), LAC (38.8% vs. 6.7%;  $P=0.003$ ), and ATPO (22.5% vs. 10.1%;  $P=0.025$ ) was significantly higher in the endometriosis group, while no difference was observed in APLA ( $P=0.686$ ). In patients with endometriosis, a significant decrease in oocyte count ( $P=0.033$ ) and a significant increase in cycle count ( $P=0.045$ ) were observed.

**Conclusions:** In this study, comparing infertile women with those without endometriosis, a significantly higher prevalence of ANA, LAC, and anti-TPO antibodies was observed in women with endometriosis, along with reduced ovarian reserve indices. These findings indicate systemic immune involvement and suggest that ovarian dysfunction may be linked to autoimmune responses. Identification and follow-up of patients with positive antibody tests may therefore be important for fertility management and the prevention of declines in ovarian reserve.

**Keywords:** Autoimmunity, Endometriosis, Immunology, Ovarian reserve

## Introduction

Endometriosis, which is characterized by the presence of tissue similar to endometrial tissue, affects 3-10% of women of reproductive age and is often associated with infertility and pelvic pain (1-4). Since endometriosis may reduce ovarian reserve, estimates indicate that 25%-50% of patients receiving fertility treatments have the condition (5). However, precise statistics are not available. Although retrograde menstruation is common, occurring in 76-90% of women, only a minority of these women develop endometriosis, suggesting that other factors, such as immune disorders, may be important in the pathogenesis of endometriosis (6,7).

Autoimmune disorders are key contributors to the disease's pathophysiology, and studies have shown that patients often have impaired immune control and altered antibody levels (6,8). Growing scientific evidence suggests

that endometriosis should not be viewed solely as a hormonally driven disorder, but rather as a condition in which chronic inflammation and immune dysregulation play central roles in disease initiation and progression (9,10). Defects in immune surveillance, increased production of autoantibodies, and impaired clearance of ectopic endometrial tissue may provide a strong biological rationale to investigate autoimmunity as a mechanistic contributor to reduced ovarian reserve in endometriosis (10). Systemic inflammation, often associated with endometriosis, reduces the ovarian reserve, a key marker of fertility (11), and there is a reported relationship between anti-Müllerian hormone (AMH) level and inflammatory markers (16). Decreased ovarian reserve is a common problem in women with ovarian endometriosis (11-13).

Auto-antibodies against endometrial and ovarian antigens have been reported in women with endometriosis

Received 10 October 2025, Accepted 17 January 2026, Available online 13 February 2026

<sup>1</sup>Department of Obstetrics and Gynecology, YAS Hospital, Tehran University of Medical Sciences, Tehran, Iran. <sup>2</sup>Department of Obstetrics & Gynecology & Reproductive Endocrinology of Women Hospital, Tehran University of Medical Sciences, Tehran, Iran. <sup>3</sup>Department of Obstetrics & Gynecology, Ardabil University of Medical Sciences, Ardabil, Iran. <sup>4</sup>Department of Obstetrics and Gynecology, Imam Khomeini Hospital, Tehran University of Medical Sciences, Tehran, Iran

\*Corresponding Author: Maryam Marghoub, Email: ms.dr.mary@gmail.com



since the early 1980s (8), and, like anti-ovarian antibodies, these antibodies may have a direct effect on ovarian function by targeting the functional areas of the pellucid and granulosa cells that are critical for folliculogenesis and fertilization (14). The widespread impact of autoimmune endometriosis extends beyond the ovarian reserve and also affects the outcome of assisted reproductive techniques (ART). In women undergoing in vitro fertilization-embryo transfer (IVF) cycles, the presence of concurrent autoimmune disease in patients with endometriosis is a negative predictor of cumulative clinical pregnancy (15). This suggests that while ovarian stimulation can produce normal numbers of oocytes, the autoimmune environment underlying it may compromise subsequent steps critical to a successful pregnancy.

The level of autoimmunity in patients with endometriosis should be investigated, as it is a known underlying mechanism of endometriosis pathogenesis and may be associated with reduced ovarian reserve. The concurrent presence of autoimmunity in IVF is thought to be a detrimental predictor of pregnancy outcomes in certain studies, but this detrimental effect was not reported in other studies (16). Accurate identification of autoantibodies specific to ovarian and endometrial tissues may indicate more targeted interventions to maintain or enhance ovarian reserve. Based on the above, this study compared levels of autoimmune factors in patients with endometriosis with those in the control group and investigated their potential effects on ovarian reserve.

Therefore, the primary objective of this study was to compare the prevalence of selected autoantibodies (antinuclear antibodies [ANA], antiphospholipid antibodies [APLA], lupus anticoagulant [LAC], and anti-thyroid peroxidase antibodies [ATPO]) between infertile women with and without endometriosis. A secondary objective was to evaluate whether the presence of these autoantibodies is associated with reduced ovarian reserve, as measured by AMH levels, in affected women. We prespecified the hypothesis that women with endometriosis would show higher rates of autoimmunity and lower ovarian reserve than controls.

## Materials and Methods

### Study Design and Selection of Patients

This was a case-control study in which 200 women with endometriosis undergoing IVF were enrolled between April 2024 and April 2025. Patients were studied in two groups. The first group was composed of infertile women with endometriosis of grade III or IV ( $n=80$ ), and the control group was other non-endometriotic infertile patients diagnosed ( $n=120$ ) by surgery ( $n=42$ , 35%) or transvaginal ultrasound (TVUS) ( $n=78$ , 65%). The control group was composed of infertile women with non-endometriosis-related causes of infertility, including unexplained infertility and tubal factor infertility, with no clinical or imaging evidence of endometriosis.

Women with known autoimmune diseases, inflammatory disorders, or other systemic conditions that could affect autoantibody levels were excluded from both groups to minimize potential confounding.

Due to limited prior data on the prevalence of autoantibodies in patients with endometriosis and the exploratory nature of the study, the sample size was not calculated in advance. However, the sample size was considered adequate based on previously published case-control studies reporting approximately 15%–20% differences in the prevalence of autoantibodies between women with endometriosis and controls (17). Based on this expected effect size, a total sample of 200 participants would have more than 80% statistical power to detect a clinically significant difference between groups at a two-sided significance level of 0.05.

The inclusion criteria were age 18–42 years, body mass index (BMI) 18.5–30 kg/m<sup>2</sup>, and a diagnosis of endometriosis grade III or IV, with surgical or TVUS confirmation of endometriosis. The exclusion criteria included any other infertility in the couple other than endometriosis (i.e., male factor infertility), autoimmune or inflammatory disease with poor control, and refusal of treatment.

The IVF-ET cycle protocols followed the clinical care standard, which is described in detail elsewhere (18). Ovarian stimulation was initiated with recombinant follicle-stimulating hormone or human menopausal gonadotropin, with doses individualized based on age, body mass index, and ovarian reserve. Pituitary suppression was performed using a gonadotropin-releasing hormone antagonist. When the follicles reached 18 mm in diameter, oocyte maturation was initiated with human chorionic gonadotropin, and oocyte retrieval was performed 34–36 hours later. In vitro fertilization or intracytoplasmic sperm injection was then performed, depending on sperm quality. Embryo transfer was performed under ultrasound guidance and based on embryo quality.

### Data Collection and Study Results

For all women in both groups, age, BMI, parity, pregnancy, live births, and underlying conditions were collected at the baseline. Assessment of basal ovarian reserve included measuring AMH in both groups. Clinical indicators and disease severity, including preoperative symptom involvement and deep infiltrating endometriosis (DIE), surgery, and related interventions, were recorded for both groups. ANA, APLA, LAC, and ATPO were also noted.

Autoantibodies were measured using enzyme-linked immunosorbent assay (ELISA) kits. The positivity cutoff values were defined according to the manufacturer's recommendations.

### Statistical Analysis

SPSS version 22 was used for statistical analysis. Values and percentages (%) were used to display categorical variables.

We used Fisher's exact test or Pearson's chi-square test to compare qualitative variables and Student's independent t-test for quantitative variables. Given the evaluation of multiple autoantibodies, multiple comparisons were adjusted using the Bonferroni correction where appropriate. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to estimate the association between autoantibody prevalence and endometriosis status. All tests were two-sided, and statistical significance was defined as  $P < 0.05$ .

## Results

Two groups of patients with endometriosis ( $n=80$ ) and without endometriosis ( $n=120$ ) were enrolled in this study. Patients were aged  $34.95 \pm 5.6$  years and had a BMI of  $25.20 \pm 3.56$  kg/m<sup>2</sup>. There were no significant differences in baseline and demographic patient data between the two groups, as shown in Table 1 (all  $P < 0.05$ ).

Table 2 presents the results comparing surgical findings and fertility indices between the two groups. Adhesion ( $P < 0.0001$ ), sliding ( $P < 0.0001$ ), kissing ovary ( $P < 0.001$ ), and uterosacral involvement ( $P < 0.0001$ ) were all significantly more common in the endometriosis group, according to the results. Patients with endometriosis also had a higher incidence of surgical complications (11.3% vs. 1.9%,  $P=0.011$ ). There were significant differences

between the two groups in fertility indices for the number of oocytes and cycles ( $P=0.033$  and  $P=0.045$ , respectively). The number of cycles increased, while the number of oocytes decreased, in patients with endometriosis.

Table 3 displays the autoimmune index results for both groups. Patients with endometriosis had a significantly higher percentage of positive ANA (26.3% vs. 10.1%,  $P=0.003$ , OR=3.17, 95% CI: 1.46–6.90), positive LAC (38.8% vs. 6.7%,  $P=0.003$ , OR=8.77, 95% CI: 3.98–20.41) and positive ATPO (22.5% vs. 10.1%,  $P=0.016$ , OR=2.59, 95% CI: 1.17–5.71). On the other hand, there was no significant difference in APLA ( $P=0.686$ ) between the two groups ( $P=0.686$ ).

## Discussion

In the present study, the overall relationship between immune-inflammatory indices was confirmed in two groups of patients with and without endometriosis. The ANA positivity rate was significantly higher in the endometriosis group (26.3%) than in the control group (10.1%). The peritoneal cavity in women with endometriosis is important for triggering and maintaining an autoimmune response (19–24). This environment contains abnormal levels of cytokines, chemokines, and growth factors, as well as altered immune cell activity, including macrophages, natural killer cells, lymphocytes,

**Table 1.** Baseline Information and Demographic Characteristics in Two Groups

Variable		Total (n=200)	Without endometriosis (n=120)	With endometriosis (n=80)	P value
Age	-	34.95±5.68	35.16±5.68	34.63±5.71	0.517 <sup>a</sup>
BMI (kg/m <sup>2</sup> )	-	25.20±3.56	25.19±3.60	25.20±3.54	0.997 <sup>a</sup>
AMH levels (ng/mL)	-	1.90±1.56	1.88±1.59	1.94±1.52	0.777 <sup>a</sup>
Parity	0	106 (53)	62 (51.7)	44 (55)	0.884 <sup>b</sup>
	1	56 (28)	35 (29.2)	21 (26.3)	
	2	34 (17)	20 (16.7)	14 (17.5)	
	3	4 (2)	3 (2.5)	1 (1.3)	
Gravidity	0	67 (33.5)	35 (29.2)	32 (40)	0.268 <sup>c</sup>
	1-3	124 (62)	80 (66.7)	44 (55)	
	3≥	9 (4.5)	5 (4.2)	4 (5)	
Live births	0	108 (54)	63 (52.5)	45 (56.3)	0.860 <sup>b</sup>
	1	54 (27)	34 (28.3)	20 (25)	
	2	34 (17)	20 (16.7)	14 (17.5)	
	3	4 (2)	3 (2.5)	1 (1.3)	
Underlying diseases	None	129 (64.5)	78 (65)	51 (63.8)	0.833 <sup>b</sup>
	Thyroid	23 (11.5)	11 (9.2)	12 (15)	
	Diabetes	6 (3)	4 (3.3)	2 (2.5)	
	Hypertension	12 (6)	9 (7.5)	3 (3.8)	
	Anemia	4 (2)	2 (1.7)	2 (2.5)	
	Depression and anxiety	7 (3.5)	4 (3.3)	3 (3.8)	
Other	19 (9.5)	12 (10)	7 (8.8)		

<sup>a</sup> t-test; <sup>b</sup> Fisher's exact test; <sup>c</sup> Chi-square test.

**Table 2.** Surgical Findings and Fertility Indices in the Two Groups

Variable		Total (n=200)	Without endometriosis (n=120)	With endometriosis (n=80)	P value
<b>Involvement</b>					
DIE size (mm)	-	7.90±12.24	-	7.90±12.24	-
Size of endometriosis right ovary (mm)	-	60.56±27.79	67.27±22.59	59.01±28.79	0.303 <sup>a</sup>
Size of the endometriosis in the left ovary (mm)	-	55.87±24.50	52.31±18.43	56.67±25.73	0.565 <sup>a</sup>
Adhesion	No	101 (50.5)	101 (84.2)	0 (0)	<0.0001 <sup>b</sup>
	Yes	99 (49.5)	19 (15.8)	80 (100)	
Sliding	No	103 (51.5)	101 (84.2)	2 (2.5)	<0.0001 <sup>b</sup>
	Yes	97 (48.5)	19 (15.8)	78 (97.5)	
Kissing ovary	No	143 (71.5)	113 (94.2)	30 (37.5)	<0.0001 <sup>c</sup>
	Yes	57 (28.5)	7 (5.8)	50 (62.5)	
Uterosacral involvement	No	117 (58.5)	110 (91.7)	7 (8.8)	<0.0001 <sup>c</sup>
	Yes	83 (41.5)	10 (8.3)	73 (91.3)	
Müllerian	No	194 (97)	117 (97.5)	77 (96.3)	0.685 <sup>b</sup>
	Yes	6 (3)	3 (2.5)	3 (3.8)	
<b>DIE Surgery and Related Interventions</b>					
Surgery DIE size (mm)	-	17.97±11.45	-	17.97±11.45	-
Surgical right ovarian endometrioma size (mm)	-	49.68±25.37	56.87±30.27	47.91±23.95	0.207 <sup>a</sup>
Surgical left ovarian endometrioma size (mm)	-	51.86±22.92	52.50±23.79	51.72±22.95	0.916 <sup>a</sup>
Surgery complication	No	172 (94)	101 (98.1)	71 (88.8)	<b>0.011<sup>b</sup></b>
	Yes	11 (6)	2 (1.9)	9 (11.3)	
<b>Fertility and Fertility Outcomes</b>					
Number of oocytes	-	4.63±6.36	5.42±7.25	3.46±4.54	<b>0.033<sup>a</sup></b>
Number of cycles	-	0.47±1.02	0.35±0.91	0.65±1.16	<b>0.045<sup>a</sup></b>
Number of embryos transferred	-	0.53±0.88	0.47±0.86	0.61±0.92	0.269 <sup>a</sup>

<sup>a</sup> t-test; <sup>b</sup> Fisher's exact test; <sup>c</sup> Chi-square test.

**Table 3.** Immune-inflammatory INDICES in the Two Groups

Variable		Total (n=200)	Without endometriosis (n=120)	With endometriosis (n=80)	ORs (95% CIs)	P value
ANA	Negative	166 (83.4)	107 (89.9)	59 (73.8)	3.17 (1.46-6.90)	<b>0.003<sup>a</sup></b>
	Positive	33 (16.6)	12 (10.1)	21 (26.3)		
LAC	Negative	160 (80.4)	111 (93.3)	49 (61.3)	8.77 (3.98-20.41)	<b>0.003<sup>a</sup></b>
	Positive	39 (19.6)	8 (6.7)	31 (38.8)		
ATPO	Negative	169 (84.9)	107 (89.9)	62 (77.5)	2.59 (1.17-5.71)	<b>0.016<sup>a</sup></b>
	Positive	30 (15.1)	12 (10.1)	18 (22.5)		
APLA	Negative	193 (97)	116 (97.5)	77 (96.3)	0.664 (0.131-3.37)	0.686 <sup>b</sup>
	Positive	6 (3)	3 (2.5)	3 (3.8)		

<sup>a</sup> Chi-square test; <sup>b</sup> Fisher's exact test.

and T lymphocytes (20-27). This immunoregulatory dysregulation may lead to loss of immune tolerance and cause the immune system to attack itself and nuclear components, leading to the formation of ANA (19-22). This finding aligns with those of other studies. Boas et al also reported that 21.2% of endometriosis patients tested positive for ANA, compared with 5.4% in the control group (17). Taylor et al's study of patients with endometriosis found a 27.9% prevalence of ANA positivity and reported that ANA titers increased with disease progression (28). In the study by Malinowski et al, ANA titers of 1:40 or higher were observed in 63% of women with endometriosis,

70.4% of women with idiopathic infertility, and 5.6% of healthy non-pregnant women (29).

The current study also found that 38.8% of women with endometriosis had positive LAC, compared to 6.7% of women without the disease. Lupus antibodies are a type of anti-phospholipid antibody, a heterogeneous group of circulating autoantibodies, which is associated with an increased risk of thrombosis and adverse outcomes in pregnancy, including recurrent abortion and stillbirth (30,31). Although anti-phospholipid antibodies, including LACs, have been associated with reproductive failure such as recurrent spontaneous abortion and failure of IVE,

their specific association with infertility associated with endometriosis needs further investigation.

Additionally, the ATPO positivity rate in the endometriosis group (22.5%) was significantly higher than in the controls (10.1%). According to some recent research, endometriosis patients may be more likely to have autoimmune thyroid conditions and ATPO (32). Meena et al showed a possible link between an elevated risk of infertility and thyroid dysfunction, as indicated by elevated ATPO antibody levels (33). Although thyroid conditions (hypothyroidism, hyperthyroidism) were the most prevalent underlying disease in both groups of infertile women in our study, there was no discernible difference. As a result, additional research that removes confounding and interfering variables is necessary to definitively confirm a higher prevalence of thyroid disorders and ATPO in patients with endometriosis.

There was no significant difference in AMH levels between groups. Although AMH is widely accepted as a reliable marker of ovarian reserve, previous studies have reported inconsistent results regarding its association with endometriosis. While some investigations have demonstrated reduced AMH levels in women with endometriosis—particularly in advanced stages of the disease or following surgical treatment of ovarian endometriomas—others have found no significant difference, especially in non-surgically treated patients or those with mild disease (34,35). These findings indicate that the impact of endometriosis on ovarian reserve is multifactorial and may depend on disease severity, patient characteristics, and treatment history. Therefore, suggests that endometriosis alone may not universally impair ovarian reserve.

Comparison of the number of oocytes in the two groups revealed significant differences, likely due to immune dysregulation that reduced ovarian reserve by damaging ovarian tissue and follicles. Antibodies such as ATPO and anti-ovarian antibodies are implicated in reducing ovarian function and lead to reduced oocyte yield during IVF cycles (36). Similarly, studies have confirmed reduced ovarian reserve in endometriosis patients with concomitant autoimmune diseases (15,37).

The results showed that autoimmunity was associated with an increased number of IVF cycles in women with endometriosis. Dysregulation of immune regulation at the maternal-fetal interface. Altered immune regulation at the maternal-fetal interface has been associated with reduced endometrial receptivity. It may affect embryo implantation, necessitating the need for potentially leading to the need for additional cycles to achieve pregnancy. One study showed that the onset or activity of autoimmune diseases has been associated with impairment of embryo implantation (38). Autoimmunity disrupts autoimmune conditions have been reported to alter the maternal endometrial immune-receptive profile by creating an abnormal inflammatory microenvironment at the

implantation site (39). A recent multicenter study also confirmed that the presence of concurrent autoimmunity in endometriosis has a significant additive negative impact on embryo implantation outcomes (40).

This study, like many studies, has limitations. First, some immunological tests (e.g., LAC) require a repeat interval and the use of international standard criteria for a definitive diagnosis; failing to repeat the test or using other methods may lead to inaccurate results. Second, our data lacks specific information on the nature of preoperative treatments, the duration of the illness, and other factors that might be effective. Third, as this study included only IVF patients, there may be selection bias, and the findings may not be generalizable to the broader population outside Iran.

## Conclusions

The results of our study suggest that patients who have imaging findings such as adhesions, sliding, kissing ovary, and uterosacral involvement are at high risk for infiltrative endometriosis. Additionally, it has been confirmed that endometriosis is linked to higher rates of ANA, LAC, and ATPO, and autoimmunity reduces ovarian reserve and implantation in patients with endometriosis. These results support the hypothesis that immunologic disorders contribute to endometriosis from a pathophysiologic perspective.

## Authors' Contribution

**Conceptualization:** Fatemeh Davari Tanha, Maryam Marghoub.

**Data curation:** Fatemeh Davari Tanha, Zahra Kaveh, Mehrshad Abdoli.

**Formal analysis:** Fatemeh Davari Tanha, Ghazal Sahraeian, Razieh Akbari.

**Funding acquisition:** Maryam Marghoub.

**Investigation:** Fatemeh Davari Tanha, Zahra Kaveh, Mehrshad Abdoli, Razieh Akbari.

**Methodology:** Fatemeh Davari Tanha, Zahra Kaveh, Razieh Akbari.

**Project administration:** Maryam Marghoub.

**Resources:** Zahra Kaveh, Maryam Marghoub, Mehrshad Abdoli.

**Software:** Fatemeh Davari Tanha, Mehrshad Abdoli.

**Supervision:** Maryam Marghoub.

**Validation:** Fatemeh Davari Tanha, Ghazal Sahraeian, Razieh Akbari.

**Visualization:** Fatemeh Davari Tanha, Ghazal Sahraeian.

**Writing—original draft:** Fatemeh Davari Tanha, Zahra Kaveh.

**Writing—review & editing:** Maryam Marghoub, Mehrshad Abdoli, Ghazal Sahraeian, Razieh Akbari.

## Conflict of Interests

None to be declared.

## Ethical Issues

The study was approved by the ethical committee of Tehran University of Medical Sciences (Ethics approval number: IR.TUMS. MEDICINE.REC.1402.601). All IVF patients gave informed consent to their data being used for research purposes.

## Financial Support

This is a self-funded study. The authors received no financial support for the research, authorship, and/or publication of this article.

## References

1. Feng D. Vascularization in the Pathogenesis and Treatment of Endometriosis [thesis]. Saarland University; 2013.

2. Laschke MW, Menger MD. Basic mechanisms of vascularization in endometriosis and their clinical implications. *Hum Reprod Update*. 2018;24(2):207-224. doi:10.1093/humupd/dmy001
3. McKinnon BD, Bertschi D, Bersinger NA, Mueller MD. Inflammation and nerve fiber interaction in endometriotic pain. *Trends Endocrinol Metab*. 2015;26(1):1-10. doi:10.1016/j.tem.2014.10.003
4. Sampson JA. Peritoneal endometriosis due to the menstrual dissemination of endometrial tissue into the peritoneal cavity. *Am J Obstet Gynecol*. 1927;14(4):422-469.
5. Máté G, Bernstein LR, Török AL. Endometriosis is a cause of infertility. Does reactive oxygen damage to gametes and embryos play a key role in the pathogenesis of infertility caused by endometriosis? *Front Endocrinol (Lausanne)*. 2018;9:725. doi:10.3389/fendo.2018.00725
6. Giudice LC, Evers JLH, Healy DL, eds. *Endometriosis: Science and Practice*. Wiley-Blackwell; 2012.
7. Dai W, Liang J, Guo R, et al. Bioengineering approaches for the endometrial research and application. *Mater Today Bio*. 2024;26:101045. doi:10.1016/j.mtbio.2024.101045
8. Mathur SP. Autoimmunity in endometriosis: relevance to infertility. *Am J Reprod Immunol*. 2000;44(2):89-95. doi:10.1111/j.8755-8920.2000.440206.x
9. Burney RO, Giudice LC. Pathogenesis and pathophysiology of endometriosis. *Fertil Steril*. 2012;98(3):511-519. doi:10.1016/j.fertnstert.2012.06.029
10. Lebovic DI, Mueller MD, Taylor RN. Immunobiology of endometriosis. *Fertil Steril*. 2001;75(1):1-10. doi:10.1016/s0015-0282(00)01630-7
11. Kitajima M, Masuzaki H. Ovarian reserve in patients with endometriosis. In: *Endometriosis: Pathogenesis and Treatment*. Springer; 2014:419-429.
12. Tan Z, Gong X, Wang CC, Zhang T, Huang J. Diminished ovarian reserve in endometriosis: insights from in vitro, in vivo, and human studies—a systematic review. *Int J Mol Sci*. 2023;24(21):15967. doi:10.3390/ijms242115967
13. Seyhan A, Ata B, Uncu G, eds. The impact of endometriosis and its treatment on ovarian reserve. *Semin Reprod Med*. 2015;33(4):237-244. doi:10.1055/s-0035-1554920
14. Emin A, Konova E, Lichev D, Aivazova N, Popov I. The study of ovarian autoimmunity and ovarian reserve in women with uncertain sterility. *Akush Ginekol (Sofia)*. 2008;47(2):20-23.
15. Salmeri N, Vanni V, Ottolina J, et al. O-192 Concomitant autoimmunity in endometriosis-affected women and in vitro fertilization (IVF) outcomes: a cohort study. *Hum Reprod*. 2022;37:deac106.192. doi:10.1093/humrep/deac106.192
16. AlKudmani B, Gat I, Buell D, et al. In vitro fertilization success rates after surgically treated endometriosis and effect of time interval between surgery and in vitro fertilization. *J Minim Invasive Gynecol*. 2018;25(1):99-104. doi:10.1016/j.jmig.2017.07.021
17. Boas LV, Sobrinho CB, Rahal D, et al. Antinuclear antibodies in patients with endometriosis: a cross-sectional study in 94 patients. *Hum Immunol*. 2022;83(1):70-73. doi:10.1016/j.humimm.2021.10.003
18. Papaleo E, Corti L, Vanni VS, et al. Basal progesterone level as the main determinant of progesterone elevation on the day of hCG triggering in controlled ovarian stimulation cycles. *Arch Gynecol Obstet*. 2014;290(1):169-176. doi:10.1007/s00404-014-3175-0
19. Eisenberg VH, Zolti M, Soriano D. Is there an association between autoimmunity and endometriosis? *Autoimmun Rev*. 2012;11(11):806-814. doi:10.1016/j.autrev.2012.01.006
20. Zhang T, De Carolis C, Man GCW, Wang CC. The link between immunity, autoimmunity and endometriosis: a literature update. *Autoimmun Rev*. 2018;17(10):945-955. doi:10.1016/j.autrev.2018.03.025
21. Zhang T, Li TC, Wang CC. Auto-immunity and endometriosis: evidence, mechanism and therapeutic potential. In: *Immunology of Endometriosis*. Elsevier; 2022:85-104.
22. Stanica CD, Neacsu A, Sima RM, Ioan RG. The role of the immune response in the etiopathogenesis of endometriosis. *Rom Med J*. 2021;68(1):5-12.
23. Miller JE, Ahn SH, Monsanto SP, Khalaj K, Koti M, Tayade C. Implications of immune dysfunction on endometriosis associated infertility. *Oncotarget*. 2017;8(4):7138-7147. doi:10.18632/oncotarget.12587
24. Gajbhiye RK. Endometriosis and inflammatory immune responses: Indian experience. *Am J Reprod Immunol*. 2023;89(2):e13590. doi:10.1111/aji.13590
25. Hey-Cunningham A, Riaz A, Fromm P, et al. Circulating and endometrial regulatory T cell and related populations in endometriosis and infertility: endometriosis is associated with blunting of endometrial cyclical effects and reduced proportions in moderate-severe disease. *Reprod Sci*. 2022;29(1):229-242. doi:10.1007/s43032-021-00700-4
26. Freitag N, Baston-Buest DM, Kruessel JS, Markert UR, Fehm TN, Bielfeld AP. Eutopic endometrial immune profile of infertility-patients with and without endometriosis. *J Reprod Immunol*. 2022;150:103489. doi:10.1016/j.jri.2022.103489
27. Geng R, Huang X, Li L, et al. Gene expression analysis in endometriosis: immunopathology insights, transcription factors and therapeutic targets. *Front Immunol*. 2022;13:1037504. doi:10.3389/fimmu.2022.1037504
28. Taylor PV, Maloney MD, Campbell JM, et al. Autoreactivity in women with endometriosis. *Br J Obstet Gynaecol*. 1991;98(7):680-684. doi:10.1111/j.1471-0528.1991.tb13458.x
29. Malinowski A, Szpakowski M, Wilczyński J, Banasik M, Puchala B. Occurrence of antinuclear antibodies in women with endometriosis and unexplained infertility. *Ginekol Pol*. 1995;66(7):420-424.
30. Barbui T, Cortelazzo S, Galli M, et al. Lupus anticoagulant and repeated abortions: a case-control study. *Thromb Haemost*. 1987;58(5):0856.
31. Lubbe WF, Liggins GC. Lupus anticoagulant and pregnancy. *Am J Obstet Gynecol*. 1985;153(3):322-327. doi:10.1016/s0002-9378(85)80121-4
32. Şerifoğlu H, Arinkan SA, Pasin O, Vural F. Is there an association between endometriosis and thyroid autoimmunity? *Rev Assoc Med Bras (1992)*. 2023;69(2):e20221679. doi:10.1590/1806-9282.20221679
33. Meena M, Chopra S, Jain V, Aggarwal N. Prevalence of antithyroid peroxidase antibodies in pregnant women and the effect on the outcome of pregnancy. *Obstet Gynecol*. 2014;123:157S. doi:10.1097/01.AOG.0000447080.12345.ab
34. Somigliana E, Berlanda N, Benaglia L, Viganò P, Vercellini P, Fedele L. Surgical excision of endometriosis and ovarian reserve: a systematic review on serum antimüllerian hormone level modifications. *Fertil Steril*. 2012;98(6):1531-1538. doi:10.1016/j.fertnstert.2012.08.009
35. Streuli I, de Ziegler D, Gayet V, et al. In women with endometriosis anti-Müllerian hormone levels are decreased only in those with previous endometrioma surgery. *Hum Reprod*. 2012;27(11):3294-3303. doi:10.1093/humrep/des332
36. Beydilli Nacak G, Ozkaya E, Yayla Abide C, et al. The impact of autoimmunity-related early ovarian aging on ICSI cycle outcome. *Gynecol Endocrinol*. 2018;34(11):940-943. doi:10.1080/09513590.2018.1469742
37. Aslan K, Narimanova C, Kasapoglu I, Uncu G. Is coexistence of autoimmunity more ruinous on ovarian reserve in endometriosis? A prospective observational study. *J Endometr Pelvic Pain Disord*. doi:10.1177/22840265251372403
38. Motak-Pochrzest H, Malinowski A. Does autoimmunity play a role in the risk of implantation failures? *Neuro Endocrinol Lett*. 2017;38(suppl 8):105-110.
39. Wang Q, Sun Y, Fan R, et al. Role of inflammatory factors in the etiology and treatment of recurrent implantation failure. *Reprod Biol*. 2022;22(4):100698. doi:10.1016/j.repbio.2022.100698
40. Salmeri N, Gennarelli G, Vanni VS, et al. Concomitant autoimmunity in endometriosis impairs endometrium-embryo crosstalk at the implantation site: a multicenter case-control study. *J Clin Med*. 2023;12(10):3557. doi:10.3390/jcm12103557