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ORIGINAL ARTICLE

Medicine Science International Medical Journal

Medicine Science 2026;15(3):1156-64

Hematological and inflammatory markers in scar endometriosis compared with ovarian endometriosis without scar formation

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Received 09 January 2026; Accepted 28 June 2026
Available online 13 August 2026 with doi: 10.5455/medscience.2026.01.08

Abstract

This study aimed to evaluate the clinical and demographic characteristics of patients with scar endometriosis and to investigate the association between systemic inflammatory indices and the development of scar endometriosis, using patients with ovarian endometriosis who did not develop scar endometriosis during follow-up as a reference group. This retrospective study included patients diagnosed with scar endometriosis at a tertiary care centre between 2015 and 2022. The study group comprised patients with scar endometriosis. The control group included patients diagnosed with ovarian endometriosis who had undergone surgery and did not develop scar endometriosis during follow-up. Systemic inflammatory indices derived from complete blood counts were compared between groups. The mean age of patients with scar endometriosis was 32.2 ± 6.1 years. Lesions were most commonly located on the left side (49.3%). The study group comprised 72 patients with scar endometriosis, while the control group consisted of 67 patients. Compared with the control group, patients with scar endometriosis had higher lymphocyte counts and lower monocyte counts. Neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), systemic inflammatory response index (SIRI), and monocyte-to-platelet ratio (MPR) were significantly lower, whereas lymphocyte-to-monocyte ratio (LMR) was significantly higher in the scar endometriosis group. After adjustment for age, gravida, and number of caesarean sections, only LMR remained independently associated with scar endometriosis (OR: 1.492, 95% CI: 1.075–2.071, p = 0.017). Scar endometriosis is a clinical condition frequently seen after caesarean section. While surgical excision is the basis of treatment, understanding the clinical presentation is essential for timely diagnosis. These findings suggest that scar and ovarian endometriosis may exhibit distinct characteristics in terms of immune system responses and inflammatory processes. The pathogenesis, immune regulation, and inflammatory pathways of these two conditions may differ.

Keywords: Scar endometriosis, caesarean section, endometriosis, haematological biomarkers, systemic inflammatory indices

Introduction

Endometriosis is a chronic inflammatory disease characterised by the presence of endometrial tissue outside the uterus. This condition most commonly presents as a pelvic problem, but in some cases, it may also occur at surgical sites such as the anterior abdominal wall [1]. This pathology is referred to as "scar endometriosis." It is a relatively rare form of the condition, primarily affecting women who have undergone caesarean section surgery [2]. The typical symptoms are pain, a mass at the surgical site, and symptoms related to menstruation. However, variation in symptom presentation may result in a lengthy period before diagnosis [3]. The incidence of scar endometriosis ranges from 0.03% to 3.5% [3,4].

The main factor in the development of scar endometriosis is the implantation of endometrial cells at the incision site during surgery; however, the immune-inflammatory cascade that enables this process is not yet understood [5-7]. Due to the low incidence of the disease and lack of awareness among healthcare providers, there are limited studies establishing its clinical factors and pathophysiological principles. Available information

CITATION
Yildiz AG, Kurt A, Taskin EA, et al. Hematological and inflammatory markers in scar endometriosis compared with ovarian endometriosis without scar formation. Med Science. 2026;15(3):1156-64.

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DOI: 10.5455/medscience.2026.01.08

indicates that endometriosis is linked not only to local lesions but also to a systemic immune-inflammatory state [6-8].

Haematological values obtained through complete blood count are expected to play a significant role in the future as practical, inexpensive biomarkers capable of estimating the inflammatory process [9]. Neutrophil-to-lymphocyte ratio (NLR), lymphocyte-to-monocyte ratio (LMR), monocyte-to-lymphocyte ratio (MLR), systemic inflammatory response index (SIRI), and systemic immune-inflammation index (SII) ratios have been shown to provide valuable information regarding the level of inflammation associated with ovarian endometriosis [8,10]. Nevertheless, little attention has been given in the current literature to the role of these values in scar endometriosis. This study aimed to investigate whether routine blood-derived inflammatory indices differ between patients who develop scar endometriosis following caesarean delivery and those with ovarian endometriosis who do not develop scar endometriosis during follow-up.

Material and Methods

This retrospective study examined patients diagnosed with scar endometriosis at the Reproductive Endocrinology and Gynaecology Clinics of a tertiary centre and research hospital between 2015 and 2022. As this was a retrospective archive-based study, ethical approval was obtained retrospectively before data extraction and statistical analysis commenced. None of the patients had a history of ovarian endometrioma, and all had undergone caesarean section, episiotomy, or gynaecological surgery. Cases were managed through follow-up, medical, or surgical treatments. Of the 76 patients initially identified in the scar endometriosis group, 4 were excluded due to active pelvic inflammatory disease (PID) at the time of blood sampling. Therefore, 72 patients were included in the final analysis.

All patients were diagnosed with scar endometriosis based on ultrasonographic findings and/or histopathological confirmation at our institution. The 72 eligible cases included patients with complete clinical and laboratory data, including complete blood count (CBC) parameters obtained at the time of diagnosis. Only patients aged between 18 and 50 years were included in the study.

Control group patients were identified from the clinic database and consisted of individuals diagnosed with ovarian endometriosis who had undergone caesarean delivery and did not develop scar endometriosis during follow-up. Among 170 patients with a history of ovarian endometriosis who gave birth at our institution, 79 had caesarean deliveries. After excluding 5 patients due to missing data and 7 who lacked adequate postnatal follow-up, 67 patients remained eligible. None of these individuals showed clinical or radiological signs of scar endometriosis during the two-year follow-up. The results of blood tests performed on both groups during diagnosis or routine clinical follow-up were obtained retrospectively from the hospital information system. All analyses used 10 ml of peripheral venous blood collected in EDTA tubes and analysed with a Mindray CS6000 haematology analyser. No additional procedures or sampling were performed outside routine clinical practice.

The inflammatory indices were calculated using CBC parameters as follows:

- Neutrophil-to-Lymphocyte Ratio (NLR) = Neutrophil count / Lymphocyte count
- Platelet-to-Lymphocyte Ratio (PLR) = Platelet count / Lymphocyte count
- Monocyte-to-Lymphocyte Ratio (MLR) = Monocyte count / Lymphocyte count
- Monocyte-to-Platelet Ratio (MPR) = Monocyte count / Platelet count
- Lymphocyte-to-Monocyte Ratio (LMR) = Lymphocyte count / Monocyte count
- Systemic Immune-Inflammation Index (SII) = (Neutrophil × Platelet) / Lymphocyte
- Systemic Inflammatory Response Index (SIRI) = (Neutrophil × Monocyte) / Lymphocyte
- Pan-Immune-Inflammation Value (PIV) = (Neutrophil × Platelet × Monocyte) / Lymphocyte

Cell counts were expressed as $\times 10^3/\mu L$.

The delta neutrophil index (DNI) is a calculated parameter that reflects the proportion of circulating immature granulocytes. It is derived from the difference between myeloperoxidase-reactive leukocyte fractions and mature polymorphonuclear leukocytes, as measured by automated haematology analysers. Due to this subtraction-based, algorithm-derived calculation, DNI values may occasionally be negative depending on leukocyte distribution patterns; such values do not represent analytical or measurement errors [11].

Clinical and laboratory data were obtained using a standardised data collection form created specifically for this study. Data were collected from patient files, discharge summaries (epicrises), and hospital information system records. The variables examined included demographic characteristics such as age, height, weight, smoking status, and obstetric history, as well as comorbidities, previous surgical procedures, and medication use. Inflammatory indices were calculated based on current haematology test results and compared between the two groups. These calculations were based solely on routine clinical tests.

Ethical approach

Ethical approval for the study was obtained from the local ethics committee (Approval No: 2024-025, dated 6 November 2024). The study was conducted in accordance with the principles set out in the Declaration of Helsinki.

Statistical analysis

The primary outcome of the study was the comparison of inflammatory indices between the study groups. All statistical evaluations were carried out using the RStudio software environment for statistical analysis (Affero General Public

DOI: 10.5455/medscience.2026.01.08

License v3; initial release 2011. RStudio for Linux, released on 19 September 2022; developed by Posit, PBC). To determine whether the data followed a normal distribution, both visual methods (such as histograms and probability plots) and analytical tests (Kolmogorov–Smirnov and Shapiro–Wilk) were used. The homogeneity of variances between groups was assessed using Levene's test. For variables showing a normal distribution, results were expressed as mean ± standard deviation, and comparisons between groups were made with the independent samples t-test. When the data were not normally distributed, descriptive statistics were presented as median and interquartile range (Q1–Q3), and group differences were evaluated using the Mann–Whitney U test. Categorical data were summarised as numbers and percentages, and associations between these variables were analysed using the chi-square test or Fisher's exact test, depending on the validity of the chi-square assumptions. Spearman correlation analysis was performed to evaluate the association between continuous variables, including pathological lesion size and relevant clinical or laboratory parameters. Univariate analyses were performed to identify variables associated with the study groups. Variables with potential clinical or statistical relevance were further evaluated using multivariable logistic regression analysis to adjust for potential confounding factors, including age, number of caesarean sections, and gravida. Results were reported as odds ratios (ORs) with 95% confidence intervals (CIs). A Type I error rate of 5% was used to determine statistical significance, and p-values < 0.05 were considered indicative of statistically significant results.

Results

Demographic and Clinical Characteristics of the Scar Endometriosis Group

Demographic and reproductive characteristics were evaluated in 76 patients diagnosed with scar endometriosis. The mean age was 32.2 ± 6.1 years (median: 32, range: 20–48), and the mean age at menarche was 13.7 ± 1.73 years. Obstetric history showed a median gravida of 2 (range: 0–6) and parity of 2 (range: 0–5). Caesarean section was the predominant mode of delivery, with a mean of 1.64 ± 0.81, compared to 0.26 ± 0.71 for vaginal births, indicating a primarily surgical obstetric profile. The median duration of lactation after the most recent delivery was 12 months (range: 0–36), and menstruation resumed after a median of 1 month postpartum (range: 1–25). In 37 patients with available data, the mean serum CA 125 level was 32.6 ± 25.3 U/mL, consistent with normal values (Table 1).

Among patients with scar endometriosis, the mean ultrasonographic lesion size (longest axis) was 2.48 ± 1.81 cm (median: 2.2 cm), and the mean pathological volume of excised tissue was 22.49 ± 37.53 cm³ (median: 8.9 cm³) (Table 1). No statistically significant correlation was found between ultrasonographic lesion size and pathological volume (Figure 1). A weak positive correlation was observed between patient age and pathological lesion volume, whereas no significant correlations were identified between lesion volume and age at menarche, number of pregnancies (gravida), number of caesarean sections, or CA 125 levels (Table 2).

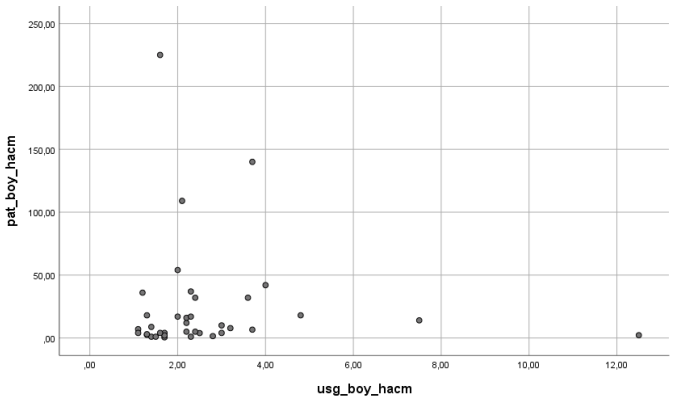

Figure 1. Simple dot scatter plot comparing ultrasound lesion size and pathological volume

All 76 patients were included in the analysis of demographic and clinical characteristics, but four were excluded from the comparison of inflammatory markers due to active pelvic inflammatory disease. Therefore, 72 patients were included in the final analysis of systemic inflammatory indices. The patient selection process and group allocation are illustrated in Figure 2.

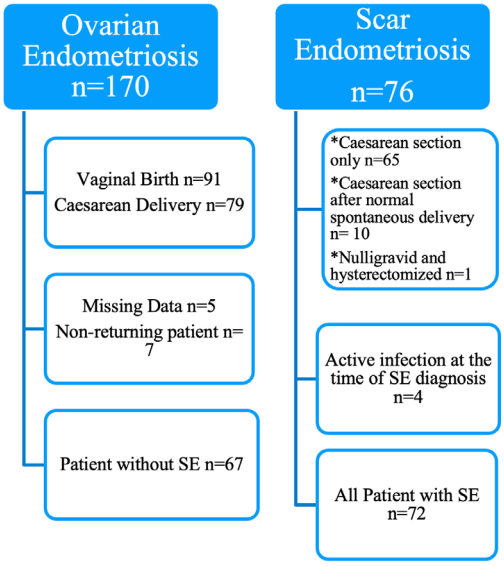

Figure 2. Flow-chart for selection of patients

As presented in Table 3, lesion localisation among patients with scar endometriosis was most commonly on the left side (49.3%), followed by the right side (30.7%), with fewer cases involving the midline (5.3%) or episiotomy scar (1.3%). Regarding anatomical depth, lesions were primarily situated in the subcutaneous tissue (44.0%), followed by fascia (26.7%) and rectus muscle (9.3%), while involvement of the umbilicus (1.3%), episiotomy scar (2.7%), or previous laparoscopy scars (1.3%) was rare. Surgical excision was the most common

DOI: 10.5455/medscience.2026.01.08

treatment approach, applied in 76.0% of patients. Among those who received non-surgical management, dienogest was used in 6.7%, oral contraceptives in 2.7%, and 14.6% did not receive any pharmacological treatment.

Contraceptive use following the last delivery showed that 66.7% of patients reported no contraception, while condoms (21.3%), intrauterine devices (6.7%), and tubal ligation (5.3%) were used less frequently. Regular menstrual cycles were observed in 78% of patients, while 16% reported irregular menses and 3% had frequent menstruation. Active smoking was present in 18.6% of patients.

Demographic and Inflammatory parameters comparative results of two groups

The study included two groups: Group I consisted of patients diagnosed with ovarian endometriosis without scar involvement (n = 67), while Group II consisted of patients with histopathologically and/or ultrasonographically confirmed scar endometriosis (n = 72).

When comparing mean age, the median age of patients in Group II was significantly higher (32 [IQR: 27–36] vs 26 [IQR: 24–30]; $p < 0.001$). Additionally, the number of pregnancies (gravida) and births (parity) was significantly higher in the scar endometriosis group ($p = 0.001$ and $p = 0.003$, respectively). Smoking prevalence, hypothyroidism, and diabetes mellitus (DM) prevalence were similar between groups ($p = 0.086$, $p = 0.482$, and $p = 0.109$, respectively; Table 4).

Lymphocyte levels were significantly higher in Group II and lower in Group I ($p = 0.049$). Conversely, monocyte levels were significantly lower in Group II compared to Group I ($p = 0.036$).

Significant intergroup differences were observed in several systemic inflammatory indices. NLR was significantly higher in Group I [2.60; IQR: 1.72–3.61] compared to Group II [2.22; IQR: 1.65–3.05] ($p = 0.049$). Similarly, MLR was higher in Group I [0.216; IQR: 0.172–0.295] than in Group II [0.182; IQR: 0.134–0.231] ($p = 0.001$). In contrast, LMR was significantly higher in Group II [5.48; IQR: 4.34–7.47] than in Group I [4.62; IQR: 3.38–5.80] ($p = 0.001$). Furthermore, SIRI and MPR were both significantly greater in Group I compared to Group II ($p = 0.024$ for both). These findings indicate a heightened systemic inflammatory response in ovarian endometriosis without scar endometriosis compared to scar endometriosis (Table 5).

Multivariable logistic regression analysis was performed to adjust for potential confounding factors, including age, number of caesarean sections, and gravida, as shown in Table 6. After adjustment, LMR (OR: 1.492, 95% CI: 1.075–2.071, $p = 0.017$) remained significantly associated with the study groups. However, the significance of SIRI and MPR was no longer observed after adjustment.

Table 1. Main characteristics of the scar endometriosis group

Variable (n = 76)	Mean ± SD	Median (Range)
Age (years)	32.2 ± 6.10	32 (20 – 48)
Age at menarche (years)	13.7 ± 1.73	14 (10 – 17)
Gravida	2.3 ± 1.34	2 (0 – 6)
Parity	1.9 ± 0.90	2 (0 – 5)
Normal vaginal deliveries	0.26 ± 0.71	0 (0 – 4)
Caesarean sections	1.64 ± 0.81	2 (0 – 4)
Lactation duration after last birth (months)	14.7 ± 10.94	12 (0 – 36)
Time to first menses postpartum (months)	4.6 ± 6.79	1 (1 – 25)
Serum CA125 (U/mL) (n = 37)	32.6 ± 25.3	25 (7 – 99)
Ultrasound-based lesion size (The longest axis of the lesion, cm) (n = 62)	2.48 ± 1.81	2.2 (0.7 – 12.5)
The pathological volume of the excised tissue (volume = length × width × 0.52, cm^3) (n = 58)	22.49 ± 37.53 cm^3	8.9 (0.26 – 200)

Table 2. Correlation analysis between pathological lesion volume and other variables

Variable	*r*	*p*
Ultrasound-based lesion size (The longest axis of the lesion, cm)	0.196	0.245
Age (years)	0.329	0.021
Age at menarche (years)	0.195	0.410
Gravida	0.193	0.188
Caesarean sections	0.084	0.570
Serum CA125 (U/mL)	0.247	0.224

r: Spearman correlation coefficient, p: p-value (significance level); $p < 0.05$ was considered statistically significant

DOI: 10.5455/medscience.2026.01.08

Table 3. Clinical characteristics of the scar endometriosis group

Clinical category (n = 76)		n (%)
Scar side	Left (n, %)	37 (49.3)
	Right (n, %)	23 (30.7)
	Midline (n, %)	4 (5.3)
	Episiotomy (n, %)	1 (1.3)
	Unspecified (n, %)	11 (13.3)
Anatomical layer	Subcutaneous tissue (n, %)	33 (44.0)
	Fascia (n, %)	20 (26.7)
	Rectus muscle (n, %)	7 (9.3)
	Umbilicus (n, %)	1 (1.3)
	Episiotomy scar (n, %)	2 (2.7)
	Previous laparoscopy scar (n, %)	1 (1.3)
	Unspecified (n, %)	11 (14.7)
Procedure	Caesarean Section (n, %)	71 (93.5)
	Hysterectomy (n, %)	2 (2.6)
	Episiotomy (n, %)	2 (2.6)
	Laparoscopy (n, %)	1 (1.3)
Surgical treatment	Surgical excision (%)	76.0
Non-surgical treatment	Dienogest (%)	6.7
	Oral contraceptives (%)	2.7
	None (%)	14.6
Contraception after birth	None (%)	66.7
	Condom (%)	21.3
	Intrauterine device (%)	6.7
	Tubal ligation (%)	5.3
Menstrual pattern	Regular (%)	78
	Irregular (%)	16
	Frequent (%)	3
Smoking (%)		18.6
Palpable mass at presentation (%)		41
Cyclical pain present (%)		32
Analgesic use (%)		74
Recurrence (%)		16

Table 4. Demographic comparative results of two groups

Variable	Group IOvarian endometriosis without scar formation (n = 67)	Group IIScar endometriosis (n = 72)	*p*
Age (years)	26 (24 – 30)	32 (27 – 36)	**< 0.001**
Number of vaginal births	0 (0 – 1)	0 (0 – 1)	0.204
Number of caesarean sections	1 (1–2)	2 (1 – 2)	**< 0.001**
Gravida (n)	1 (1 – 2)	2 (1 – 3)	**0.001**
Parity (n)	1 (1 – 2)	2 (1 – 2)	**0.003**
Smoking (n, %)	6 (9)	15 (20.8)	0.086
Hypothyroidism (n, %)	5 (7.5)	3 (4.2)	0.482
Diabetes Mellitus (n, %)	3 (4.5)	0 (0)	0.109

Data are expressed as mean ± SD, median and quartiles (Q1-Q3), or number (percentage) where appropriate; A p value of < 0.05 indicates a significant difference; Statistically significant p-values are in bold

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Table 5. Comparative inflammatory parameters between two groups

Variable	Group IOvarian endometriosis without scar formation (n = 67)	Group IIScar endometriosis (n = 72)	*p*
WBC (10^3/μL)	7310 (5900 – 9130)	7175 (5685 – 8583)	0.538
PLT (10^3/mm^3)	265 (227 – 291)	275 (216 – 335)	0.421
Neutrophils (10^3/μL)	4400 (3560 – 6350)	4335 (3490 – 5485)	0.301
Lymphocytes (10^3/μL)	1740 (1450 – 2240)	2005 (1570 – 2463)	**0.049**
Monocytes (10^3/μL)	410 (320 – 480)	360 (270 – 428)	**0.036**
DNI	-3.1 (-4.9; -1.6)	-4.1 (-6.4; -1.9)	0.070
SII (10^3/μL)	654 (459 – 945)	585 (401 – 821)	0.114
SIRI (10^3/μL)	1.005 (0.611 – 1.705)	0.744 (0.486 – 1.158)	**0.024**
PIV (10^6/μL^2)	234 (160 – 494)	218 (125 – 328)	0.068
NLR	2.60 (1.72 – 3.61)	2.22 (1.65 – 3.05)	**0.049**
PLR	146 (115 – 186)	141 (110 – 166)	0.241
MLR	0.216 (0.172 – 0.295)	0.182 (0.134 – 0.231)	**0.001**
MPR	1.54 (1.15 – 1.86)	1.29 (0.95 – 1.68)	**0.024**
LMR	4.62 (3.38 – 5.80)	5.48 (4.34 – 7.47)	**0.001**

Data are expressed as mean ± SD, median and quartiles (Q1 - Q3), or number (percentage) where appropriate; A p value of <0.05 indicates a significant difference; Statistically significant p-values are in bold; WBC: white blood cell count, PLT: platelet count, DNI: delta neutrophil index, SII: systemic immune-inflammation index, SIRI: systemic inflammation response index, PIV: pan-immune-inflammation value, NLR: neutrophil-to-lymphocyte ratio, PLR: platelet-to-lymphocyte ratio, MLR: monocyte-to-lymphocyte ratio, MPR: mean platelet volume-to-platelet ratio, LMR: lymphocyte-to-monocyte ratio

Table 6. Multivariable logistic regression analysis

Variable	OR	CI 95%	p
Age	1.181	1.098 – 1.271	**< 0.001**
Number of caesarean sections	11.672	5.440 – 25.043	**< 0.001**
Gravida	1.598	1.158 – 2.205	**0.004**
Parity	1.861	1.191 – 2.908	**0.006**
Lymphocytes	1.001	1.000 – 1.001	0.057
Monocytes	0.997	0.994 – 1.000	**0.028**
SIRI	0.999	0.999 – 1.000	**0.022**
MPR	0.525	0.290 – 0.951	**0.034**
LMR	1.415	1.159 – 1.729	**0.001**
SIRI*	0.999	0.999 – 1.000	0.093
MPR*	0.416	0.138 – 1.247	0.117
LMR*	1.492	1.075 – 2.071	**0.017**

* Age, number of caesarean sections, gravida adjusted; SIRI: systemic inflammation response index, MPR: mean platelet volume-to-platelet ratio, LMR: lymphocyte-to-monocyte ratio

Discussion

Several theories have been proposed to explain the pathogenesis of endometriosis. The first is the implantation theory, proposed by Sampson. According to this view, during menstruation, endometrial cells can pass backwards through the fallopian tubes and attach to pelvic structures [12]. This mechanism is also used to explain the development of abdominal wall endometriosis, where endometrial cells that escape through the uterine incision during a caesarean section may implant on the abdominal wall. However, this theory does not adequately explain the rare locations of endometriosis, such as in the lungs, kidneys, or brain. To address this limitation, Halban proposed the vascular spread theory [13]. According to this theory, endometrial cells can be transported outside the uterine structures via the lymphatic system or blood vessels, enter the peripheral circulation, and reach distant organs where they can implant ectopically. A third theory involves metaplasia, in which cells of the abdominal wall transform into endometrial-

DOI: 10.5455/medscience.2026.01.08

like tissue. This metaplastic process may be triggered by cell mimicry or hormonal stimulation. Some researchers have suggested that the pathogenesis of endometriosis may be best explained by a combination of these theories [14].

Although scar endometriosis is classified as an iatrogenic subtype of endometriosis, there is no clear explanation for why this condition develops in some individuals following caesarean section. Therefore, beyond technical aspects and preventive measures, it is believed that the underlying pathogenic mechanism may be more complex, potentially involving endocrine, immunological, and inflammatory pathways. Intraoperative implantation is not a plausible mechanism for non-surgical (or endogenous) endometriosis, and although retrograde menstruation, or Sampson's hypothesis, resembles the proposed process in post-caesarean scar endometriosis, it is not considered to play a direct role, as it does in pelvic endometriosis [15–17]. Only a few studies have demonstrated the presence of pre-existing pelvic endometriosis in patients with scar endometriosis [18,19]. It is believed that in women with multiple caesarean sections, the likelihood of endometrial cells being implanted into the incision site during surgery increases. Although caesarean delivery is the most commonly associated procedure, scar endometriosis has also been reported following vaginal deliveries, particularly in episiotomy scars [14].

In nearly half of the patients, lesions were located on the left side, with the subcutaneous tissue and fascia being the most frequently affected anatomical layers. A predominance of left-sided localisation has also been reported in some studies, suggesting that this pattern may be related to the surgeon's dominant hand, the direction of the incision, or suture techniques [3,4]. Lesions extending into the fascia and rectus muscle indicate the potential for progressive infiltration into deeper tissues. In more than 50% of cases in the literature, scar endometriosis has been reported to be localised in the subcutaneous layer [20], and the findings of this study are consistent with these rates.

In the systematic review of scar endometriosis cases, pain and the presence of a palpable lesion at the surgical site were consistently reported as the most frequent clinical features, reinforcing their diagnostic relevance in patients with a history of caesarean section or other pelvic surgeries [21]. Similarly, in our study, a palpable mass was detected in 41% of patients, 32% reported cyclical pain, and 74% reported analgesic use. These findings are consistent with data reported in the literature; despite the frequent occurrence of classic findings such as cyclic pain and masses, in some patients, the absence of cyclic symptoms or the limitation of symptoms to analgesic use alone may delay diagnosis. Some studies have reported that the average delay in diagnosis ranges from one to three years [15]. In addition, some cases are diagnosed incidentally when endometriosis foci are detected along the incision line during surgical procedures performed for unrelated reasons (e.g., cosmetic interventions) [18]. In this study, 76% of patients underwent surgery, while approximately 10% received medical treatment. Although surgical excision generally provides favourable results, the limited number of patients treated with medical therapy makes it difficult to make a valid comparison of effectiveness between the two approaches. The risk of recurrence increases when excision is incomplete or when lesion margins are left behind. A recurrence rate of 16% was observed in this study, consistent with previously published data reporting recurrence rates ranging from 4% to 25% with long-term follow-up [2,3].

Medical treatments such as dienogest or other oral contraceptives may be considered to alleviate symptoms in patients who are not suitable for surgery [17]. Moreover, two-thirds of our patients (66.7%) who did not use any contraceptive method in the postpartum period may have indirectly facilitated the clinical manifestation of scar endometriosis. The absence of contraception – or the use of non-hormonal methods such as condoms or copper intrauterine devices – allows menstruation to resume early and the oestrogen–progesterone cycle to normalise quickly, potentially accelerating the proliferation of endometrial cells implanted in the incision line. Conversely, progestin-based methods (e.g., oral contraceptives or levonorgestrel-releasing IUDs) and hypoestrogenic conditions such as prolonged lactation can suppress lesion growth and delay the onset of symptoms.

Furthermore, analyses comparing the groups demonstrated significant differences in certain inflammatory parameters between patients with scar endometriosis (Group II) and patients with ovarian endometriosis who did not develop scar endometriosis during the post-caesarean follow-up period (Group I). In Group II, lymphocyte levels were higher and monocyte levels were lower, while LMR was significantly elevated. In contrast, NLR and MLR were higher in Group I. However, after adjustment for potential confounding factors, including age, number of caesarean sections, and gravidity, only some inflammatory parameters retained statistical significance, while others lost significance. This finding suggests that part of the initially observed differences may be attributable to baseline differences between the groups.

Pelvic endometriosis is generally associated with an active inflammatory process characterised by increased neutrophil and monocyte activity. In contrast, the relative lymphocyte predominance observed in the scar endometriosis group may indicate a more localised and organised inflammatory response. Previous studies have also suggested that the immunological characteristics of iatrogenic (scar-associated) endometriosis may differ from those of spontaneous pelvic disease [15,22].

These results suggest that patients with scar endometriosis exhibit relatively milder inflammatory activity and a distinct pattern of immune regulation. Pelvic endometriosis typically reflects an ongoing and active inflammatory process characterised by increased neutrophil and monocyte counts. In contrast, the lymphocyte predominance observed in the scar endometriosis group suggests a more chronic, organised, and localised type of inflammation. Previous reports have also suggested that the

DOI: 10.5455/medscience.2026.01.08

immunoregulatory mechanisms in iatrogenic (scar-associated) endometriosis may not fully resemble those in spontaneous pelvic disease [15,22]. Some studies have shown that systemic indices, including NLR, MLR, and SIRI, tend to increase in pelvic endometriosis and other chronic inflammatory conditions, consistent with the concept of stronger systemic inflammatory activity [8,10,23]. In these results, SIRI and MPR values are also significantly elevated in patients with ovarian endometriosis. In the scar endometriosis group, high lymphocyte counts and low monocyte counts indicate a more localised and controlled inflammatory response. When evaluated together, the lower levels of systemic inflammation markers such as NLR, MLR, and SIRI in scar endometriosis compared to systemic endometriosis support the view that the pathophysiological mechanisms underlying scar and pelvic endometriosis are not entirely the same. In summary, the inflammatory profiles of patients with scar endometriosis differ from those with ovarian endometriosis. The differences in inflammatory markers observed between scar and ovarian endometriosis cases suggest that there may be distinct differences in immune response and disease progression between these two subgroups.

Previous studies have demonstrated that systemic inflammatory markers may vary across different phases of the menstrual cycle under the influence of sex steroid fluctuations. For example, hs-CRP concentrations have been reported to be highest during the early follicular phase and inversely associated with estradiol levels, suggesting a close interaction between hormonal status and systemic inflammatory responses. Therefore, some variability in haematological inflammatory indices in the present study may have been related to physiological cycle-dependent changes in addition to disease-specific inflammatory mechanisms [24].

This study suggests that inflammatory indices may differ between patients who develop scar-site endometriosis and those who do not develop scar-site endometriosis during post-caesarean follow-up but have ovarian endometriosis. In this context, inflammatory markers may be considered a potential variable associated with scar formation in individuals with similar surgical exposure. However, given potential confounding factors – including the retrospective design and differences in baseline characteristics – these findings should be interpreted with caution. Therefore, the results should be evaluated as hypothesis-generating rather than definitive evidence of an immunological mechanism. Further prospective and controlled studies are needed to validate these observations.

Strengths and Limitations of the Study

This study has several strengths. First, the inclusion of a relatively high number of cases for a rare clinical condition such as scar endometriosis is an important feature. Additionally, the selection of a control group consisting of patients who had undergone caesarean delivery but did not develop scar endometriosis during follow-up allowed comparison among individuals with similar surgical exposure, enhancing the methodological strength of the study. Furthermore, the inflammatory indices evaluated are routinely obtainable, low-cost, and easily applicable in clinical practice, increasing the study's potential clinical relevance. Finally, this study contributes to the limited literature investigating inflammatory profiles potentially associated with scar endometriosis development.

This study also has several limitations. First, its retrospective single-centre design and baseline differences between groups, including age and obstetric characteristics, may have introduced selection bias and residual confounding despite multivariable adjustment. Second, the control group consisted of women with ovarian endometriosis rather than women without endometriosis, and the absence of scar endometriosis was defined within a two-year follow-up period; given the potentially long interval between surgery and clinical presentation, delayed, clinically silent, or unrecognised cases cannot be completely excluded. In addition, several potentially relevant clinical variables, including endometriosis stage, endometrioma rupture, concomitant adenomyosis, lesion burden, and other inflammatory or systemic conditions, were not uniformly available, particularly in the control group. Therefore, the findings should be interpreted as a comparison between two clinical phenotypes rather than definitive evidence of factors associated with scar endometriosis development. Third, complete blood count parameters were obtained from routine clinical practice without standardised timing according to menstrual cycle phase, postpartum status, lactation, or hormonal treatment, all of which may have influenced inflammatory indices. Hormonal fluctuations occurring during the different phases of the menstrual cycle may alter leukocyte distribution and systemic inflammatory responses. Therefore, the fact that blood samples were collected at different times during the menstrual cycle may have influenced haematological indices such as NLR, MLR, and LMR. Due to the retrospective nature of the study, it was not possible to standardise blood sampling times according to the phase of the cycle, and this may have contributed to interindividual variability in inflammatory markers. Finally, the relatively limited sample size and the simultaneous evaluation of multiple interrelated ratio-based haematological markers increase the possibility of imprecise estimates and chance findings; accordingly, the results should be regarded as exploratory and hypothesis-generating.

Conclusion

In conclusion, this study demonstrates that complete blood count–derived inflammatory indices differ between patients with scar endometriosis and those with ovarian endometriosis without scar involvement, suggesting distinct systemic inflammatory profiles between these clinical phenotypes. Notably, the persistence of LMR as an independent variable after multivariable adjustment highlights its potential relevance in reflecting underlying immunological differences. These findings support the concept that scar endometriosis, despite sharing histopathological characteristics with pelvic disease, may involve a more localised and relatively controlled inflammatory response rather than a pronounced systemic inflammatory state.

DOI: 10.5455/medscience.2026.01.08

From a clinical perspective, routinely available haematological indices may offer additional, non-invasive insights into disease characterisation and could contribute to a more nuanced understanding of endometriosis subtypes. However, given the retrospective design, potential residual confounding, and the exploratory nature of the analyses, these results should be interpreted cautiously and primarily as hypothesis-generating.

Ethical Approval

This study received ethical clearance from the Ethics Committee of Etlik Zübeyde Hanım Training and Research Hospital (Approval No: 2024-025, dated 6 November 2024). All procedures adhered to the principles outlined in the Declaration of Helsinki. To maintain confidentiality, patient data were anonymised before any analysis was performed.

Patient Consent

Individual informed consent was not obtained due to the retrospective nature of the study, as all data were collected from existing medical records and no additional procedures or interventions were performed for research purposes.

Conflict of Interest

The authors confirm the absence of any conflicts associated with this work.

Funding

The authors declare that they have received no financial support for the study.

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