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Assessment of the Systemic Inflammatory Indices in Patients with Endometriosis with Long COVID

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ABSTRACT

Background and Aim: Endometriosis is a chronic inflammatory disease, and Long COVID is a syndrome of persistent symptoms following SARS-CoV-2 infection. These two conditions may share common inflammatory pathways. This study aimed to compare systemic inflammatory indices between patients with endometriosis with long COVID and healthy individuals.

Methods: A cross- sectional study involving 40 participants, including 20 patients with laparoscopically confirmed endometriosis and 20 healthy individuals as a control group, was conducted. Each group was further divided into subgroups based on Long COVID, confirmed through RT-PCR and a questionnaire, resulting in four groups: a healthy control group without Long COVID (n = 10), a healthy control group with Long COVID (n = 10), an endometriosis group without Long COVID (n = 10), and an endometriosis group with Long COVID (n = 10). The immune system inflammation index (SII), neutrophil- to- lymphocyte ratio (NLR), platelet- to- lymphocyte ratio (PLR), immune system inflammation amount (PIV), and systemic inflammatory response index (SIRI) were calculated from blood samples. Non- parametric tests were used for the statistical analysis.

Results: The immune system inflammation index (SII) was significantly higher in patients with endometriosis and Long COVID than in patients without Long COVID (995.25 vs. 612.48; p = 0.0193). Similarly, the platelet-to-lymphocyte ratio (PLR) was significantly higher in the Long COVID endometriosis group than in the endometriosis- only group (168.65 vs. 99.24; p = 0.0471). Although not statistically significant, a consistent upward trend was observed in the NLR, PIV, and SIRI levels in the Long COVID endometriosis group. No significant increases in these indices were observed in healthy individuals with Long COVID.

Conclusion: Long COVID is significantly associated with increased SII and PLR in patients with endometriosis, highlighting the unique vulnerability of patients with endometriosis to pre- inflammatory complications of SARS-CoV-2 infection. Readily available inflammatory indices, such as the SII, can serve as useful biomarkers for monitoring the inflammatory burden in this patient population.

INTRODUCTION

Endometriosis is a chronic inflammatory disease characterized by the presence of endometrial-like tissue outside the uterus. Endometriosis has a variety of symptoms, including pelvic pain, dysmenorrhea, and infertility. Epidemiological studies estimate that endometriosis affects around 10% of women in their reproductive years (1). The most common body parts involved in endometriosis include

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the ovaries, uterine ligaments, peritoneum, tubes, rectovaginal septum, and bladder (2). Immune dysregulation is recognized as a key factor in the pathophysiology of endometriosis. Increased levels of pro-inflammatory cytokines and altered immune cell profiles have been reported in patients with endometriosis, which may contribute to pain and other symptoms (3). Understanding these changes is critical for developing targeted treatment strategies. On the other hand, Long COVID, which refers to symptoms or complications that occur after acute infection with SARS-CoV-2 and can persist for weeks or even months after initial recovery from COVID-19 (4), has been associated with immune system disorders, including innate and adaptive immunity (5). Long COVID is characterized by persistent symptoms, such as fatigue and cognitive impairment; however, the underlying mechanisms remain unclear (6). The overlap between Long COVID and endometriosis can exacerbate inflammation and complicate the diagnosis and treatment. One of the markers that show the level of inflammation in diseases is the Systemic Immune- Inflammation Index (SII). The SII is a new biomarker used to assess systemic inflammation and immune responses in cancer (7), cardiovascular diseases (8), and inflammatory diseases (9). This index is calculated using a combination of three common blood count parameters. The SII was calculated using the formula platelet count × neutrophil count / lymphocyte count. In addition to detecting the level of inflammation in diseases, this indicator can help make treatment decisions (10). Recent studies have suggested a potential association between SII indicators and endometriosis (11, 12). Due to the increase in dysmenorrhea after COVID-19 (13), it is possible that COVID-19 and Long COVID may play a role in increasing the symptoms and severity of endometriosis. The impact of Long COVID on inflammation in endometriosis remains unexplored. The pan- immune- inflammation value (PIV) and systemic inflammatory response index (SIRI) are now recognized as crucial biomarkers for assessing immune activation and systemic inflammation. These indices are increasingly recognized for their significant roles in predicting patient prognosis and treatment outcomes across various diseases (14). This study examined whether Long COVID worsens systemic inflammation in endometriosis.

MATERIALS and METHODS

Study data and population

A single- center, cross- sectional study was conducted at the Payvand Clinical Laboratory between August 2024 and March 2025. We enrolled 20 patients with endometriosis and 20 healthy women aged 25-45 years. Participants were selected via consecutive sampling from patients presenting to our gynecology clinic for laparoscopic investigation of chronic pelvic pain or infertility. Healthy controls were recruited from volunteers with no symptoms related to endometriosis. A definitive diagnosis of endometriosis was confirmed by laparoscopy by a gynecologist. In this study, the diagnosis of Long COVID relied on RT-PCR confirmation of SARS-CoV-2 infection (based on individuals' self- reports), along with a 25-question participant survey. Inclusion Criteria: for all participants: Regular menstrual cycles (26-32 days). For Endometriosis Group: Women aged 25-45 years with laparoscopically and histologically confirmed endometriosis. For Control Group: Age- matched healthy women with no history or clinical symptoms suggestive of endometriosis. Long COVID Status: Confirmation of prior SARS-CoV-2 infection via a positive RT-PCR test (self- report) and the presence of at least 5 symptoms beyond 2 months, as defined by a positive response to our 25-item Long COVID questionnaire. Exclusion Criteria (for all participants): History of hepatitis B/C or HIV. Cardiovascular disease, systemic inflammatory diseases (e.g., rheumatoid arthritis, lupus), cancer, premature ovarian failure, or other hormonal disorders. Hospitalization for acute COVID-19 infection. Any infectious or systemic inflammatory illness in the 6 months prior to blood collection. Use of hormonal contraceptives or other hormonal medications within 3 months prior to the study. Pregnancy or lactation. The key potential confounders considered in this study included age, body mass index (BMI), time since COVID-19 infection, and vaccination status. Participants in the endometriosis and control groups were matched for age and BMI. All study participants had received at least one dose of the vaccine. Data on the time interval between COVID-19 infection and blood sampling were collected for all participants. Due to the limited sample size, it was not possible to adjust for this variable. All individuals provided written informed consent. The study protocol and procedures were approved by the Ethics Committee of Shahid Beheshti University of Medical Sciences (Approval No. IR.SBMU.MSP.REC.1403.265). After completing the questionnaires, blood samples were collected from the participants, and a complete blood count was performed.

Evaluation of inflammatory parameters

We evaluated routine clinical hematology using a Mindray BC-6200 automated hematology analyzer (Full diff, Flowcytometery, Laser Light Scattering). The Systemic Immune-Inflammation Index (SII) was determined using the formula: SII = (platelet count × neutrophil count) / lymphocyte count, where all values are expressed in units of

$\times 10^9$/L. The pan-immune inflammation value (PIV) was computed using the following formula: PIV = (neutrophil count × monocyte count × platelet count) / lymphocyte count ($\times 10^9$/L). The following formula was used to calculate the systemic inflammatory response index (SIRI): neutrophil count × monocyte count/lymphocyte count (10^9/L).

Statistical analysis

The Shapiro- Wilk test was used to analyze the distribution of participants' data, which indicated a non- parametric distribution. Owing to the non-normal distribution of the variables, the Kruskal- Wallis ANOVA was used to compare the four groups. Differences in variables between women with and without endometriosis were assessed using the non-parametric Mann- Whitney U test. Statistical significance was set at $p < 0.05$. Statistical analyses were performed using GraphPad Prism 10.

Ethical consideration

The protocol has been confirmed by institutional ethical committee (Ethical code: IR.SBMU.MSP.REC.1403.663).

RESULTS

A total of 40 participants were enrolled in this study, 20 of whom were confirmed to have endometriosis. Of these 20, 10 had Long COVID based on the questionnaire. In the control group, 10 out of 20 people without endometriosis had Long COVID. Based on the presence or absence of endometriosis, the participants were divided into four groups. Healthy subjects: no endometriosis and Long COVID, LC^+ EM^-: no endometriosis and with Long COVID, EM^+ LC^-: endometriosis and Long COVID, EM^+ LC^+: endometriosis and with Long COVID.

The median age (interquartile range) of the healthy participants was 39 (6.75) years, 38 (13.75) years for LC^+ EM- participants, 37.5 (10.5) years for EM^+ LC^- participants, and 43.5 (6.75) years for EM^+ LC^+ participants. There were no significant differences in age among the four groups.

The NLR ratio varied across the study groups (Healthy, LC^+EM^-, EM^+LC^-, EM^+LC^+, Control, and EM). No statistically significant difference was observed between the Healthy and LC^+EM^- groups (1.63 vs. 1.96; $p > 0.9999$). Similarly, the comparison between EM^+LC^- and EM^+LC^+ showed no significant difference (2.06 vs. 2.89; $p = 0.3347$). However, a significant increase in NLR was detected in the EM group compared to the control group (1.73 vs. 2.5; $p = 0.0227$) (Figure 1).

The PLR varied across the study groups. No statistically significant difference was observed between the Healthy and LC^+EM^- groups (137.55 vs. 194.56; $p = 0.2225$). However, a significant increase in PLR was detected in the EM^+LC^+ group compared to the EM^+LC^- group (168.65 vs. 99.24; $p = 0.0471$). The comparison between the Control and EM groups showed no significant difference (170.04 vs. 141.43; $p = 0.0911$) (Figure 2).

In the comparison between all groups, the SIRI varied across the study groups, but no statistically significant difference was observed between the groups. A difference between the Healthy group and the LC^+EM^- group (0.65 vs 0.83; $p > 0.9999$) and the comparison between EM^+LC^- and EM^+LC^+ (0.75 vs 1.2; $p > 0.9999$) also the EM group compared with the Control group (1.14 vs 0.75; $p = 0.3013$) are shown in Figure 3.

The SII was elevated in LC^+ patients compared to the controls (Figure 4). No statistically significant difference was observed between the Healthy and LC^+EM^- groups (525.85 vs. 781.76; $p = 0.3064$). The SII was significantly higher in the EM^+LC^+ group than in the EM^+LC^- group (995.25 vs. 612.48; $p = 0.0193$). However, no significant difference was observed between the Control and EM groups (674.68 vs. 808; $p = 0.0596$).

In the comparison between all groups, the PIV varied across the study groups, but no statistically significant difference was observed between the groups. A difference between the Healthy group and the LC^+EM^- group (194.54 vs 330.29; $p = 0.4516$) and the comparison between EM^+LC^- and EM^+LC^+ (207.75 vs 428.7; $p = 0.2930$), also the EM group compared with the Control group (269.64 vs 317.832; $p = 0.4450$), is shown in Figure 5.

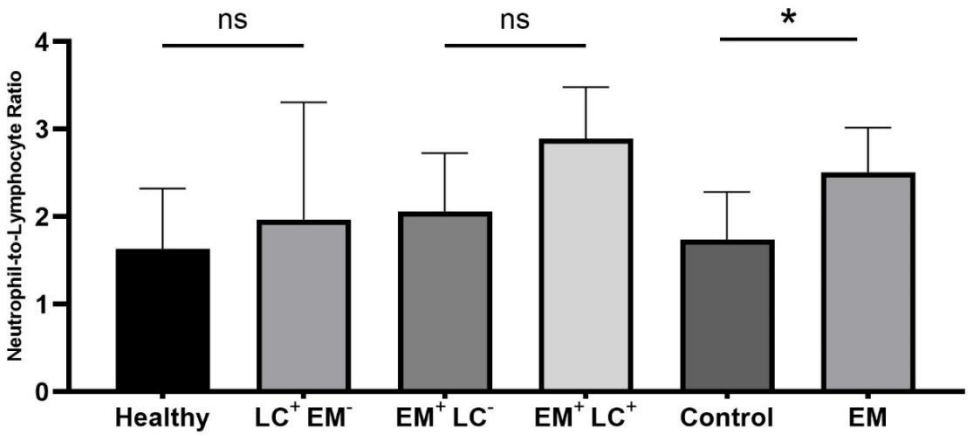

FIGURE 1. Comparison of neutrophil- to- lymphocyte ratios (NLR) between the different groups. The bar charts demonstrate the differences in the median NLR between the different groups. The chart shows that the Healthy group had the lowest NLR compared to the other groups. $EM^+ LC^+$ has the highest NLR compared to the other groups. The median of NLR is significantly (0.0227) higher in EM compared to controls. LC: Long COVID, EM: Endometriosis, ns denotes no significant, * denotes $p < 0.05$.

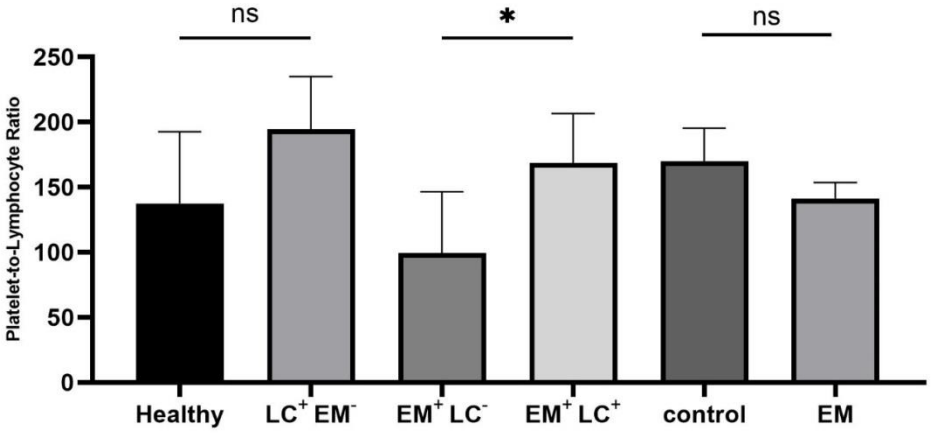

FIGURE 2. Comparison of platelet- to- lymphocyte ratio (PLR) between the different groups. The bar charts demonstrate the differences in the median PLR between the different groups. The chart shows that the $EM^+ LC^-$ group had the lowest PLR value compared to the other groups. $LC^+ EM^-$ has the highest PLR compared to the other groups. The median PLR was significantly (0.0227) higher in $EM^+ LC^+$ than in $EM^+ LC^-$ ($p = 0.0471$). LC: Long COVID, EM: Endometriosis, ns denotes no significant, * denotes a $p < 0.05$.

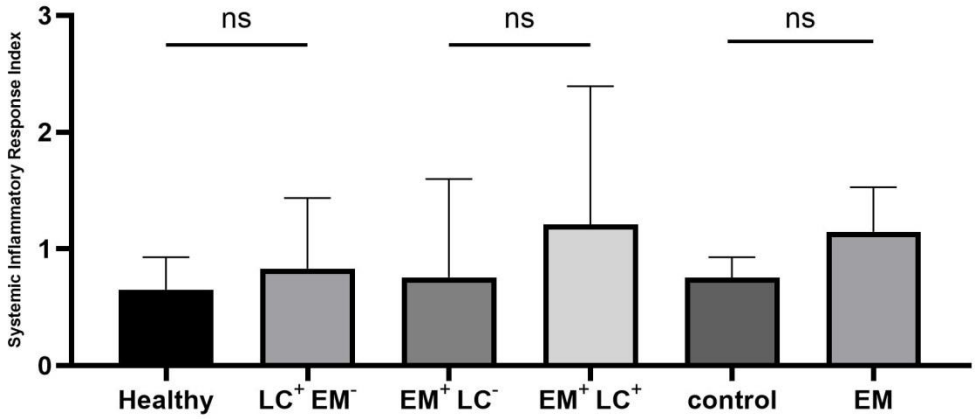

FIGURE 3. Comparison of the systemic inflammatory response index (SIRI) between the different groups. The chart shows that the Healthy group had the lowest SIRI value compared to the other groups. $EM^+ LC^+$ has the highest SIRI compared to the other groups. No statistically significant difference was observed between the groups. LC: Long COVID, EM: Endometriosis, ns denotes no significant.

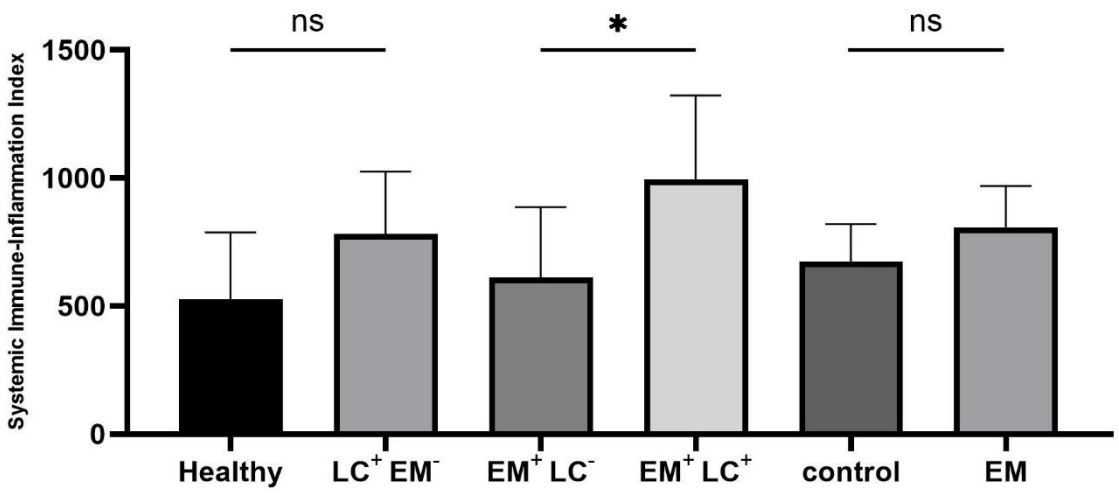

FIGURE 4. Comparison of the systemic immune- inflammation index (SII) between the different groups. The bar charts demonstrate the differences in median SII between the groups. The chart shows that the Healthy group had the lowest SII compared to the other groups. $EM^+ LC^+$ has the highest SII compared to the other groups. The median SII was significantly (0. 0.0193) higher in $EM^+ LC^+$ compared to $EM^+ LC^-$ ($p = 0.0471$). LC: Long COVID, EM: Endometriosis, ns denotes no significant, * denotes $p < 0.05$.

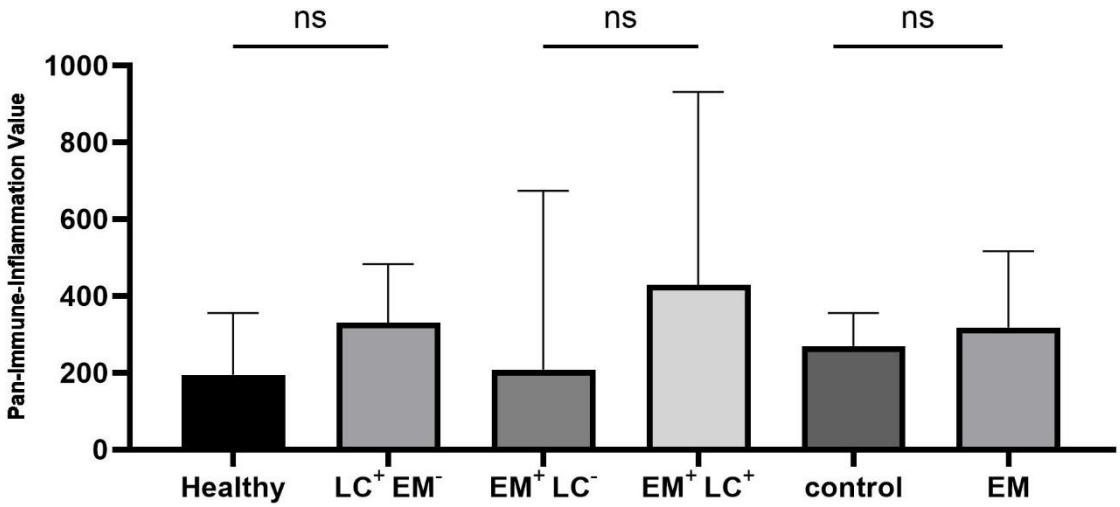

FIGURE 5. Comparison of pan- immune- inflammation value (PIV) between the different groups. The chart shows that the Healthy group had the lowest PIV value compared to the other groups. $EM^+ LC^+$ has the highest PIV compared to the other groups. No statistically significant differences were observed between the groups. LC: Long COVID, EM: Endometriosis, ns denotes no significant.

DISCUSSION

Endometriosis is a chronic inflammatory disease that is characterized by dysregulation of the immune system. The pathogenesis of endometriosis involves complex interactions between hormonal, immune, and environmental factors (15). Immune dysregulation plays a crucial role in the development and progression of endometriotic lesions, with various immune cells contributing to the inflammatory environment (15). Understanding the immunopathophysiology of endometriosis is crucial for developing novel diagnostic tools and targeted therapies (16).

In recent years, Systemic Inflammatory Indices have emerged as accessible and cost-effective biomarkers for quantifying immune system activation and inflammatory burden. These indices are recognized as indirect markers of systemic inflammation, and their increase can indicate chronic immune system activation. These indices, derived from routine complete blood counts, provide valuable insights into the interaction between innate and acquired immunity and are increasingly recognized for their prognostic and diagnostic potential in various chronic diseases.

The COVID-19 pandemic, particularly the Long- term sequelae post- acute known as Long COVID, has introduced

an additional layer of immune dysregulation. Persistent low- grade inflammation, immune fatigue, and coagulopathy abnormalities recorded in Long COVID may exacerbate the pathophysiological mechanisms already active in endometriosis. Understanding this interaction is not only clinically relevant but also crucial for guiding targeted monitoring and potential interventions in patient populations already affected by chronic symptoms. Evaluating NLR, PLR, SII, PIV, and SIRI in women with endometriosis related to Long COVID may illuminate common inflammatory pathways, highlight biomarkers of disease persistence or exacerbation, and ultimately provide strategies for prognosis, management, and risk stratification. This study is one of the first to explore this association through Systemic Inflammatory Indices.

Most studies that have examined Systemic Inflammatory Indices in endometriosis have found a significant increase in these parameters in the endometriosis group compared to the control group. In a study conducted by Liang Peng and colleagues on 3,390 subjects, a significant positive correlation between SII and endometriosis was demonstrated (17).

Ying Zhou also conducted a study that included 434 women with confirmed endometriosis and 517 individuals as a control group, comparing the SII, SIRI, NLR, and PIV levels in women with endometriosis to the control group. This is one of the most recent studies conducted, in which the systemic inflammatory response indices SII, SIRI, NLR, and PIV were significantly higher ($p < 0.0001$) in women with endometriosis than in the control group (18).

A significant increase in SII ($p = 0.0193$) was observed in the group of patients with endometriosis who have Long COVID, compared to individuals without Long COVID, highlighting the presence of chronic systemic inflammation in this population. This increase in SII supports the hypothesis that chronic inflammation related to Long COVID may enhance the inflammatory baseline characteristic of endometriosis and potentially contribute to the persistence or worsening of clinical symptoms. Such an interaction between two chronic inflammatory diseases could have significant implications for disease progression, symptom burden, and treatment strategies. This enhanced inflammatory response provides a plausible biological mechanism for the exacerbation and persistence of reported debilitating symptoms such as anxiety, depression, fatigue, and dysmenorrhea in patients with endometriosis following SARS-CoV-2 infection (19).

Furthermore, while a similar trend of increased SII in individuals without endometriosis who had Long COVID was observed compared to those without it, this difference did not reach statistical significance. This finding highlights an important point: the underlying inflammatory environment of endometriosis may create a unique vulnerability, making these patients more susceptible to the Long- term pre- inflammatory effects of SARS-CoV-2, resulting in a more pronounced inflammatory response than what is seen in the general population.

The observed increase in PIV among patients with Long COVID strongly indicates a complex and concurrent dysregulation in these immune axes. This likely reflects a state of persistent activation of the innate immune system alongside relative lymphopenia, which are prominent features of both the acute and post-acute phases of SARS-CoV-2 infection and contribute to a profile of immune system fatigue and dysregulation. In our group, a consistent trend was observed where the PIV values in groups with Long COVID were higher compared to groups without it, a pattern that was true for both individuals with endometriosis and healthy individuals. Nevertheless, despite this clear trend, the differences between groups did not reach statistical significance. The persistence of this trend, even in the absence of statistical significance, is clinically and biologically noteworthy. This suggests a common inflammatory pathway following SARS-CoV-2 infection that may affect all individuals regardless of endometriosis diagnosis. Lack of statistical significance may be attributed to various factors, including a high degree of inter-individual variability in immune response to Long COVID or the possibility that PIV, while comprehensive, may be influenced by confounding variables not accounted for in this analysis.

The PLR, an indicator of a prothrombotic state and relative immune system suppression, showed a continuous increase among individuals with Long COVID. This trend was observed in both the healthy control group and the endometriosis group. Importantly, this difference was statistically significant specifically in the endometriosis group ($p = 0.0471$). This finding suggests that the intersection of chronic inflammatory conditions and pre-existing endometriosis with the thrombo- inflammatory complications of Long COVID may create a unique inflammatory environment in which disturbances in platelet activation and a reduction in lymphocytes are most pronounced. The significant increase in PLR in this group adds another dimension to the observed increase in SII, further emphasizing the exacerbated thrombo- inflammation state.

The NLR index did not show a significant difference in individuals without endometriosis between the group with

Long COVID and those without it. Also, in individuals with endometriosis, this index was higher in the group with Long COVID compared to those without Long COVID, but again this difference was not statistically significant.

SIRI has not only demonstrated its value in the prognosis of cancer (20, 21) but has also been shown to accurately reflect the body's inflammatory status. Many previous studies have shown that the composite inflammatory index has very good outcomes in assessing prognosis, status, and the progression of inflammatory diseases (22, 23). The potential of composite inflammatory indices based on complete blood counts and their ratios in predicting and preventing inflammatory diseases is very high and deserves to be further investigated in clinical applications (24).

However, in our study, no significant difference was observed between the groups under study. In comparing individuals with endometriosis to healthy individuals, despite the increase in these inflammatory indices, contrary to previous studies, this difference was not significant, except for the NLR index, which was significantly higher ($p = 0.0227$). The lack of significance in this difference could be attributed to the sample size, as other studies that examined these indices used larger sample sizes.

A significant limitation is the variability in the time interval between the acute SARS-CoV-2 infection and the measurement of inflammatory indices in our Long COVID groups. This interval was recorded for each participant but due to sample size constraints, we could not stratify our analysis based on it. The inflammatory characteristics of Long COVID are dynamic and are likely to change over time. The absence of a standardized timeline following infection may lead to variability and obscure the relationship. Future longitudinal studies with serial measurements at fixed time points will be essential to clarify the course of inflammation in this patient population. Our findings should be interpreted with appropriate caution in light of potential residual and unmeasured confounding. While factors such as the specific SARS-CoV-2 variant and underlying undiagnosed comorbidities were not fully accounted for in our analysis, the study design and exclusion criteria minimized their potential impact.

CONCLUSION

As a result, this study provides preliminary evidence that Long COVID may exacerbate systemic inflammatory status in patients with endometriosis. Our key finding is a statistically significant increase in the SII and PLR indices in endometriosis patients with Long COVID compared to patients without Long COVID. This suggests a potential synergistic interaction between these two conditions, where the ongoing immune regulation disorder characteristic of Long COVID may enhance the chronic inflammatory baseline inherent to endometriosis. The observed increase in NLR, PIV, and SIRI in the group of endometriosis patients with Long COVID, although not statistically significant, consistently followed an upward trend and further supported the hypothesis of increased inflammation. Notably, these inflammatory indices did not significantly increase in non- endometriosis patients with Long COVID, indicating a unique vulnerability in the endometriosis population. This indicates that the pre- existing inflammatory microenvironment in endometriosis may make these individuals prone to a stronger and more measurable inflammatory response following SARS-CoV-2 infection.

The main limitations of this study are the relatively small sample size and its cross-sectional design, which prevents establishing causality. Nevertheless, these findings highlight the potential applicability of inflammatory indicators derived from CBC, which is readily available, particularly SII and PLR, as biomarkers for monitoring the inflammatory burden in endometriosis patients suffering from Long COVID. This relationship necessitates further research through larger and longitudinal studies to confirm these results, elucidate the underlying mechanisms, and explore the clinical implications for managing this specific patient population, which may experience exacerbated symptoms due to complex inflammatory pathways.

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Not declared.

CONFLICT OF INTEREST

All authors declare no conflicts of interest.

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