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Short Communication

Relationship between serum CA125, prolactin and cortisol levels with disease stage and pain level in endometriosis patients

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Abstract

Endometriosis affects approximately 10% of women of reproductive age and is characterized by the presence of endometrial tissue outside the uterine cavity. Diagnostic delays are common due to nonspecific symptoms and the absence of reliable biomarkers. Serum CA125, prolactin, and cortisol have been implicated in the pathophysiology of endometriosis through inflammatory, neuroendocrine, and stress-response mechanisms. However, their role as biomarkers in endometriosis remains poorly studied. This study aimed to investigate the relationships between serum CA125, prolactin, and cortisol levels with endometriosis staging and pain severity in endometriosis patients. A cross-sectional study was conducted at Dr. Zainoel Abidin General Hospital, Banda Aceh, Indonesia, involving women with laparoscopically confirmed endometriosis. Serum CA125, prolactin, and cortisol levels were measured using electrochemiluminescence immunoassay (ECLIA). Disease staging followed the American Society for Reproductive Medicine (ASRM) classification, and pain severity was assessed using the Numeric Rating Scale (NRS). Statistical analyses were performed using the Spearman correlation test. A total of 30 women with confirmed endometriosis were included in this study, with a mean age of 37.2 years. Endometriosis stages were distributed as stage II (20.0%), stage III (16.7%), and stage IV (63.3%), and the mean pain score was 5.60±1.48. Elevated serum biomarker levels were observed with CA125 of 72.65±55.39 U/mL, prolactin of 1456.77±1799.79 µIU/mL, and cortisol of 341.92±189.02 nmol/L. The serum CA125 level was positively correlated with endometriosis staging (r=0.580, p=0.001) but not with pain severity. Prolactin and cortisol had no significant correlations with disease stage or pain severity (all p>0.05). This study shows that serum CA125 levels are significantly correlated with endometriosis staging, supporting its potential as a biomarker of disease progression. Although prolactin and cortisol levels were elevated, their lack of association with clinical parameters suggests broader neuroendocrine dysregulation rather than direct markers of disease severity.

Keywords: Endometriosis, biomarker, cancer antigen 125, prolactin, cortisol

Introduction

Endometriosis is a chronic, estrogen-dependent gynecological disorder characterized by the presence of endometrial glands and stroma outside the uterine cavity [1]. Affecting approximately 10–15% of women of reproductive age and up to 70% of those with chronic pelvic pain, it represents one of the most prevalent causes of dysmenorrhea, dyspareunia, and infertility

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worldwide [2,3]. Despite its high prevalence, the diagnosis of endometriosis is often delayed for six to seven years after symptom onset due to nonspecific clinical manifestations and the lack of reliable non-invasive biomarkers [4,5]. The current diagnostic gold standard—laparoscopic visualization with histopathological confirmation—remains invasive, costly, and limited to specialized healthcare centers [6].

The pathogenesis of endometriosis is multifactorial, involving retrograde menstruation, immune dysfunction, and hormonal and inflammatory alterations that promote ectopic implantation, neuroangiogenesis, and fibrosis [7-9]. Estrogen plays a central role by stimulating local proliferation of endometriotic tissue, whereas progesterone resistance contributes to impaired apoptosis and aberrant inflammatory signaling [10]. The chronic peritoneal inflammation characteristic of endometriosis leads to macrophage activation, cytokine release, and oxidative stress, which perpetuate pain and lesion progression [11,12]. These complex biological interactions highlight the need for integrative biomarkers that reflect both the inflammatory and neuroendocrine dimensions of the disease.

Among circulating biomarkers, cancer antigen 125 (CA125) has been the most extensively studied. CA125 is a high-molecular-weight glycoprotein secreted by coelomic and Müllerian epithelium, and its serum concentration increases in response to peritoneal irritation and inflammatory stimulation [13,14]. Elevated CA125 levels have been observed in moderate to severe endometriosis and correlate positively with disease stage [15]. However, its diagnostic accuracy remains limited, particularly for early-stage disease [16].

Beyond inflammatory biomarkers, neuroendocrine mediators such as prolactin and cortisol may also play important roles in endometriosis pathophysiology. Prolactin exerts both immunomodulatory and pro-inflammatory effects through macrophage activation and cytokine secretion [17]. Elevated serum prolactin levels have been documented in patients with endometriosis and are associated with infertility and enhanced pain perception [18]. Similarly, cortisol, the major glucocorticoid produced via activation of the hypothalamic-pituitary-adrenal (HPA) axis, reflects physiological stress responses that interact with inflammatory and immune mechanisms [19]. Dysregulation of the HPA axis, manifesting as initial hypercortisolism followed by adaptive hypocortisolism, has been proposed as a feature of chronic inflammatory conditions, including endometriosis [20,21].

Although several studies have explored individual associations between these biomarkers and endometriosis, few have simultaneously examined CA125, prolactin, and cortisol within a unified analytical framework [22]. Understanding the integrated profiles of inflammatory and neuroendocrine biomarkers may improve the accuracy of non-invasive disease assessment and deepen insight into endometriosis-related pain mechanisms. This study aimed to investigate the relationships between serum CA125, prolactin, and cortisol levels with endometriosis stage and pain severity among patients with endometriosis.

Methods

Study design and setting

A cross-sectional study was conducted to investigate the relationships between serum CA125, prolactin, and cortisol levels and endometriosis stage and pain severity in the Department of Obstetrics and Gynecology at Dr. Zainoel Abidin General Hospital, the major tertiary referral center in Aceh Province, Indonesia. The study was carried out between January and June 2025, following approval from the Health Research Ethics Committee of Dr. Zainoel Abidin General Hospital. Data collection was carried out between January and June 2025. During this period, all eligible women undergoing diagnostic or operative laparoscopy for suspected endometriosis were screened. The diagnosis of endometriosis was confirmed intraoperatively and supported by postoperative histopathology, following the routine clinical workflow of the hospital's Minimally Invasive Gynecology Unit. Standardized perioperative protocols were used for preoperative assessment, anesthesia, surgical procedures, specimen handling, and postoperative evaluation, ensuring methodological uniformity across cases.

All blood samples were collected preoperatively in the hospital's surgical preparation unit and processed in accordance with institutional laboratory protocols. Serum aliquots were

transported under controlled conditions to the ISO 15189–accredited Prodia Central Laboratory for biomarker quantification. Clinical symptoms, laparoscopic findings, and staging based on the revised American Society for Reproductive Medicine (rASRM) classification were documented by attending gynecologist consultants trained in minimally invasive gynecological surgery.

Patients and criteria

Eligible participants were women of reproductive age who underwent diagnostic or therapeutic laparoscopy with intra-operative confirmation of endometriosis. The inclusion criteria were: (1) clinical and ultrasonographic suspicion of endometriosis; (2) no prior history of laparotomy or hormonal therapy within three months before enrolment; and (3) willing to provide blood samples and participate in the study. Exclusion criteria included: (1) histopathological results inconsistent with endometriosis; (2) evidence of malignant ovarian or pelvic pathology; and (3) systemic disorders or medication use that could alter cortisol or prolactin levels (e.g., corticosteroids, dopamine agonists/antagonists).

Sample size and sampling method

Sample size was determined using a correlation formula with an expected moderate effect size (r=0.5), a confidence level of 95%, and 80% statistical power, yielding a minimum of 29 participants. Thirty patients meeting the inclusion criteria were consecutively recruited using a purposive sampling method to ensure all subjects had confirmed endometriosis through laparoscopic and histopathological findings.

Study variables and measurements

The independent variables in this study were serum concentrations of CA125, prolactin, and cortisol, whereas the dependent variables were endometriosis stage and pain severity. Serum CA125 (U/mL) was quantified using an electrochemiluminescence immunoassay (ECLIA) on a Cobas e601 analyzer (Roche Diagnostics, Mannheim, Germany). Serum prolactin (µIU/mL) was measured using the same ECLIA platform, following the manufacturer's specified procedures. The serum cortisol (nmol/L) was also analyzed by ECLIA from morning samples collected between 08:00 and 10:00 a.m. to minimize diurnal variation.

Endometriosis stage was determined intraoperatively and classified according to the rASRM scoring system, which categorizes the disease into stages I–IV based on lesion size, depth, and extent of adhesions. Pain severity was evaluated preoperatively using the Numeric Rating Scale (NRS), ranging from 0 (no pain) to 10 (worst imaginable pain), and the highest score reported during menstruation within the previous three months was recorded.

Data collection

Venous blood samples (5 mL) were collected during the preoperative phase. Samples were centrifuged at 2000×g for 10 minutes, and sera were stored at –80°C until analysis. Biomarker assays were performed at the Prodia Central Laboratory (Jakarta, Indonesia), which follows ISO 15189–accredited procedures. Clinical data, including age, parity, menstrual history, and clinical symptoms, were recorded using standardized case report forms. The endometriosis stage was documented by the attending laparoscopic surgeon based on intraoperative findings and photographic documentation.

Statistical analysis

Data analysis was performed using SPSS software version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Shapiro–Wilk test and presented as mean ± standard deviation (SD) or median (range) as appropriate. Categorical variables were summarized as frequencies and percentages. The Kruskal–Wallis test was used to compare the serum biomarker levels (CA125, prolactin, and cortisol) across endometriosis stages, as the data were non-normally distributed.

The Spearman rank correlation test was applied to evaluate associations between serum biomarker levels (CA125, prolactin, cortisol) and (1) endometriosis stage and (2) pain severity (NRS score). A two-tailed p<0.05 was considered statistically significant.

Results

Participant characteristics

Thirty women with laparoscopically and histologically confirmed endometriosis were included in the analysis. The detailed demographic and clinical characteristics of patients are presented in **Table 1**. The mean age of patients was 37.23±7.44 years (range 21–52 years). All patients were married and of reproductive age. The majority presented with dysmenorrhea (90.0%), followed by dyspareunia (43.3%) and infertility (63.3%). The mean NRS pain score was 5.60±1.48, indicating moderate-to-severe pain intensity. Regarding parity, 12 participants (40.0%) were nulliparous, while 16 (53.3%) were multiparous (**Table 1**).

In terms of disease severity, 6 participants (20.0%) were classified as stage II, 5 (16.7%) as stage III, and 19 (63.3%) as stage IV according to rASRM classification (**Table 1**). The predominance of advanced disease likely reflects delayed presentation to the tertiary referral center.

Table 1. Characteristics of women with confirmed endometriosis included in the study (n=30)

Characteristic	Frequency (percentage)
Age (years), mean±SD	37.23±7.44
Clinical symptoms	
Dysmenorrhea	27 (90.0)
Dyspareunia	13 (43.3)
Infertility	19 (63.3)
Pain level (NRS), mean±SD	5.60±1.48
Parity	
Nullipara	12 (40.0)
Multipara	16 (53.3)
Endometriosis stage based on ASRM classification	
Stage II	6 (20.0)
Stage III	5 (16.7)
Stage IV	19 (63.3)

ASRM: American Society for Reproductive Medicine; NRS: Numeric Rating Scale

Serum biomarker profiles

All three biomarkers demonstrated mean serum concentrations above their respective reference ranges (**Table 2**). The mean serum CA125 level was 72.65±55.39 U/mL, the mean prolactin level was 1456.77±1799.79 µIU/mL, and the mean cortisol level was 341.92±189.02 µg/dL.

Table 2. Comparisons of biomarker levels (CA125, prolactin, and cortisol) based on disease staging in endometriosis patients (n=30)

Variable	Endometriosis stage	Median	Min	Max	*p*-value
CA125 level (U/mL)	Stage II	24.1	13.4	40.6	0.007
	Stage III	13.9	8.4	101.4	
	Stage IV	87.4	12.9	887.0	
Prolactin level (µIU/mL)	Stage II	621	245	2098	0.094
	Stage III	2395	482	9241	
	Stage IV	577	123	3718	
Cortisol level (nmol/L)	Stage II	400.9	221.0	532.7	0.471
	Stage III	253.9	56.7	605.8	
	Stage IV	300.8	71.6	822.4	

When stratified by disease stage, the median CA125 values were 24.05 U/mL in stage II, 13.9 U/mL in stage III, and 87.4 U/mL in stage IV (**Table 2**). Statistical testing revealed a significant difference across stages (p=0.007), indicating a progressive increase in CA125 with increasing disease severity. Conversely, neither serum prolactin (stage II: 621 µIU/mL; stage III: 2395 µIU/mL; stage IV: 577 µIU/mL; p=0.094) nor cortisol (stage II: 400.9 µg/dL; stage III: 253.9 µg/dL; stage IV: 300.8 µg/dL; p=0.471) demonstrated significant inter-stage variation (**Table 2**). These findings suggest that only CA125 displays a consistent stage-dependent pattern among the biomarkers measured.

Correlation between biomarkers and endometriosis stage

Spearman's correlation analysis showed a significant positive correlation between serum CA125 level and endometriosis stage (r=0.580, p=0.001) (**Table 3**). This finding indicated that higher CA125 concentration was associated with more advanced disease. However, no significant correlations were observed between serum prolactin (r=-0.069, p=0.718) or cortisol (r=-0.039, p=0.837) and disease stage (**Table 3**).

The observed magnitude of correlation supports CA125 as a potential indicator of disease progression, while the lack of consistent associations for prolactin and cortisol implies that their variability may reflect neuroendocrine fluctuations rather than lesion burden.

Table 3. Correlation between biomarker levels (CA125, prolactin, and cortisol) with disease stage and pain severity in endometriosis patients (n=30)

Variable	Correlation	p-value	Correlation coefficient (r)
CA125 level	Endometriosis stage	0.001	0.580
Prolactin level	Endometriosis stage	0.718	-0.069
Cortisol level	Endometriosis stage	0.837	-0.039
CA125 level	Pain level	0.312	0.191
Prolactin level	Pain level	0.720	-0.068
Cortisol level	Pain level	0.054	-0.356

Correlation between biomarkers and pain severity

No biomarker demonstrated a statistically significant correlation with pain severity as measured by NRS (**Table 3**). The correlation coefficients were r=0.191 (p=0.312) for CA125, r=-0.068 (p=0.720) for prolactin, and r=-0.356 (p=0.054) for cortisol. Although the inverse correlation between cortisol and pain intensity almost approached significance, the overall pattern suggests that circulating biomarker levels alone do not adequately capture inter-individual variability in pain perception among patients with endometriosis. These findings reinforce the multifactorial etiology of endometriosis-associated pain, in which lesion location, neuroinflammatory sensitization, and psychosocial factors likely play a more dominant role than systemic biomarker levels.

Discussion

This study investigated the relationship between serum CA125, prolactin, and cortisol levels with disease stage and pain severity among women with confirmed endometriosis. Among these biomarkers, only CA125 showed a significant positive correlation with disease stage, whereas prolactin and cortisol exhibited no significant association with either disease stage or pain severity. These findings highlight the potential of CA125 as a biochemical indicator of disease burden, while prolactin and cortisol may reflect systemic neuroendocrine responses rather than direct pathological processes within endometriotic tissue.

The significant correlation between CA125 concentration and disease stage observed in this study is consistent with prior evidence showing that CA125 reflects peritoneal inflammation and lesion extent in endometriosis [1-4]. CA125 is a high–molecular-weight glycoprotein derived from coelomic and Müllerian epithelia, released into the circulation during epithelial disruption and inflammatory activation [5]. As the disease advances, deeper endometrial infiltration, adhesion formation, and greater involvement of the peritoneal surface lead to increased systemic CA125 levels [6,7].

Previous studies have reported comparable results, showing that serum CA125 levels increased significantly with the severity of endometriosis [8-10]. A study found CA125 levels were significantly higher in stages III–IV than in I–II [11], while another study observed a direct correlation between CA125 and rASRM stage scores [12]. A previous study further demonstrated that serum CA125 concentrations were strongly associated with the extent of adhesion formation [13]. Although CA125 lacks specificity—since elevations may also occur in adenomyosis, pelvic inflammatory disease, or ovarian cysts [14,15]—its reproducible association with disease progression confirms its role as a surrogate indicator of lesion burden rather than a standalone diagnostic test. A meta-analysis encompassing 22 studies found that CA125 has moderate sensitivity (52%) but high specificity (92%) for endometriosis diagnosis, with notable

improvement in moderate-to-severe disease [16]. These findings are congruent with our results, in which CA125 increased proportionally with higher disease stage, highlighting its relevance as an adjunct biomarker for staging rather than early detection.

Beyond its correlation with disease severity, the findings suggest that CA125 may hold translational value as a practical triage marker, particularly in low-resource settings where advanced imaging and laparoscopy are limited. Although not diagnostic on its own, elevated CA125 levels could help clinicians identify women at a higher likelihood of moderate-to-severe endometriosis, prompting earlier referral, prioritization for surgical evaluation, or more targeted use of scarce diagnostic resources. Integrating CA125 into preliminary assessment pathways may therefore improve timely recognition of endometriosis and reduce delays in management, especially in peripheral healthcare facilities.

In contrast, this study found no significant association between prolactin levels and endometriosis stage or pain severity. Prolactin, traditionally known for its role in lactation, also exerts important immunomodulatory and angiogenic functions [17,18]. Elevated prolactin has been detected in women with endometriosis, potentially linked to dopaminergic dysregulation and inflammatory stimulation of the pituitary axis [19,20]. Studies have shown that prolactin receptors are expressed in both eutopic and ectopic endometrial tissues, mediating cell proliferation and survival through Janus kinase–signal transducer and activator of transcription (JAK–STAT), mitogen-activated protein kinase (MAPK), and phosphoinositide 3-kinase (PI3K) pathways [21-23]. A study observed significantly higher prolactin levels among women with endometriosis compared to healthy controls [24], suggesting a pathogenic role in lesion growth. However, other studies failed to find consistent associations [25,26], possibly due to methodological variability in sampling times and hormonal influences.

The marked elevation but high variability of prolactin observed in this cohort suggests systemic neuroendocrine dysregulation rather than lesion-specific pathology. Stress-related prolactin secretion through activation of the hypothalamic–pituitary axis may contribute to this heterogeneity [27,28]. In addition, prolactin's role as a proinflammatory mediator that augments macrophage activation and cytokine production [29] could reflect an adaptive rather than causative process in chronic disease. Therefore, while elevated prolactin indicates altered endocrine homeostasis, it appears insufficiently specific to serve as a clinical marker for endometriosis activity.

Similarly, serum cortisol showed no significant relationship with either disease stage or pain severity. Cortisol, the end product of the HPA axis, regulates immune and metabolic responses to stress [30]. Chronic inflammatory states, such as endometriosis, can alter HPA function, resulting in an initial hypercortisolism phase followed by a secondary hypocortisolism phase as an adaptive response [31-33]. A study reported significantly higher plasma and salivary cortisol in women with endometriosis, consistent with HPA axis activation by persistent inflammatory stress [34]. In contrast, another study observed blunted cortisol responses and altered circadian rhythm in patients with chronic pelvic pain, indicative of long-term stress adaptation [35]. The borderline negative correlation between cortisol and pain in our study may support this hypothesis, suggesting that chronic nociceptive stimulation can attenuate cortisol release over time. Furthermore, glucocorticoid resistance at the receptor or post-receptor level has been described in chronic inflammatory conditions [36], potentially explaining the coexistence of high cortisol levels with persistent inflammation. These mechanisms may contribute to the heterogeneity observed in cortisol responses across different disease stages and clinical phenotypes of endometriosis.

The lack of significant correlation between the measured biomarkers and pain intensity highlights the complex and multifactorial nature of pain in endometriosis. Pain severity does not necessarily correspond to lesion size or stage, as nerve fiber density, lesion location, and neuroimmune crosstalk are pivotal determinants [37,38]. Lesions involving the uterosacral ligaments, rectovaginal septum, and pelvic plexus are particularly associated with higher pain scores regardless of overall disease extent [39]. Additionally, central sensitization, characterized by heightened spinal and supraspinal pain processing, plays a major role in endometriosis-related pain and is influenced by psychological and endocrine factors [40]. This complexity explains why biochemical markers of inflammation or stress fail to predict pain severity, underscoring the need

for integrative models that combine molecular, anatomical, and psychosocial parameters to better capture the patient's pain experience.

These findings reinforce the potential utility of serum CA125 as an accessible and reproducible marker to estimate endometriosis stage in clinical practice, especially where advanced imaging or laparoscopy is unavailable. However, the absence of significant associations for prolactin and cortisol suggests that neuroendocrine biomarkers alone have limited diagnostic value. A multimodal approach integrating biochemical assays, imaging, and psychometric assessment may enhance diagnostic accuracy and individualized management of endometriosis-related symptoms.

Several limitations should be acknowledged. The cross-sectional design precludes causal inference and temporal assessment of biomarker fluctuations. The modest sample size limits statistical power, particularly for prolactin and cortisol. Single-time-point measurements cannot capture hormonal variations across the menstrual cycle or diurnal cortisol rhythm. Future longitudinal studies incorporating serial biomarker sampling and stratification by clinical phenotype and stress indices may clarify the dynamic interplay between endocrine regulation and disease progression.

Conclusion

Serum CA125 exhibited a significant positive correlation with endometriosis stage, supporting its role as a practical indicator of disease burden. In contrast, prolactin and cortisol levels, although frequently elevated, showed no direct relationship with disease severity or pain intensity, likely reflecting broader neuroendocrine dysregulation. These findings highlight the need for integrative biomarker frameworks encompassing inflammatory, endocrine, and neuroimmune pathways to improve diagnostic and prognostic precision in endometriosis management.

Ethics approval

This study received ethical clearance from the Health Research Ethics Committee of the Dr Zainoel Abidin General Hospital, Banda Aceh, Indonesia (090/ETIK-RSUDZA/2025). All procedures followed the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment.

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None.

Competing interests

All the authors declare that there are no conflicts of interest.

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Underlying data

Derived data supporting the findings of this study are available from the corresponding author on request.

Declaration of artificial intelligence use

We hereby confirm that no artificial intelligence (AI) tools or methodologies were utilized at any stage of this study, including during data collection, analysis, visualization, or manuscript preparation. All work presented in this study was conducted manually by the authors without the assistance of AI-based tools or systems.

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