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Differential expression of c-kit, E-cadherin, and beta-catenin in endometriosis and normal endometrial tissue

Endometriozis ve normal endometriyal dokuda c-kit, E-kadherin ve beta-kateninin farklı ekspresyonları

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Abstract

Objective: C-kit, E-cadherin and beta-catenin adhesion molecules and proto-oncogenes are thought to be associated with molecular mechanisms related to the invasion, implantation and persistence of ectopic endometrial cells. Comparing the expression levels of these molecules in endometriomas, other types of endometriosis, and normal endometrial tissue may provide further insight into the mechanisms driving endometriosis development. The present study sought to examine the molecular pathophysiological roles of these molecules by determining their expression profiles in different types of endometriosis and in the healthy endometrium.

Materials and Methods: Retrospective data from 180 cases were analyzed, comprising 60 endometriomas, 60 cases of other types of endometriosis (superficial and deep), and 60 normal proliferative endometrial tissue samples. Immunohistochemical staining for c-kit, E-cadherin, and beta-catenin was performed. The expression levels of E-cadherin and beta-catenin were quantified using the H-score method.

Results: C-kit positivity was found in 9% of endometriomas and 10% of other endometriosis tissues, but was absent in normal endometrium. Beta-catenin H-scores were significantly lower in endometriosis tissues compared with normal endometrial tissues ($p<0.001$). E-cadherin levels showed no significant difference between the groups. A post-hoc power analysis confirmed that the study was adequately powered to detect group differences in E-cadherin, indicating that the non-significant finding likely reflects a true absence of a difference.

Conclusion: Increased c-kit expression, along with reduced beta-catenin expression in endometriosis samples, suggests that these molecules contribute to endometriosis pathogenesis. However, because no significant difference was found in E-cadherin expression, a definitive conclusion cannot be made regarding the involvement of E-cadherin in endometriosis development.

Keywords: Endometriosis, endometrioma, c-kit, E-cadherin, beta-catenin

Öz

Amaç: C-kit, E-kaderin ve beta-katenin gibi adezyon molekülleri ve proto-onkogenlerin, ektopik endometriyal hücrelerin invazyonu, implantasyonu ve persistansı ile ilişkili moleküler mekanizmalarla bağlantılı olduğu düşünülmektedir. Bu çalışma, farklı endometriozis tiplerinde ve sağlıklı endometriyumda bu moleküllerin ekspresyon profillerini belirleyerek moleküler patofizyolojik rollerini incelemeyi amaçlamıştır.

Gereç ve Yöntemler: Yüz seksen olgunun retrospektif verileri analiz edildi; bunlar arasında 60 endometrioma, 60 diğer endometriozis türü (yüzeyel ve derin) ve 60 normal proliferatif endometriyal doku örneği bulunmaktaydı. C-kit, E-kadherin ve beta-katenin için immünohistokimyasal boyama yapıldı. E-kadherin ve beta-katenin ekspresyon düzeyleri H-skor yöntemi kullanılarak ölçüldü.

PRECIS: This retrospective case-control study evaluates immunohistochemical expression of c-kit, E-cadherin, and beta-catenin in endometriotic and normal endometrial tissues, revealing molecular differences linked to endometriosis pathogenesis.

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Bulgular: C-kit pozitifliği endometriomaların %9'unda ve diğer endometriozis dokularının %10'unda saptandı, ancak normal endometriyumda gözlenmedi. Beta-katenin H-skorları, normal endometrial dokulara kıyasla endometriozis dokularında anlamlı olarak daha düşüktü (p<0,001). E-kaderin düzeyleri gruplar arasında anlamlı farklılık göstermedi. Post-hoc güç analizi, çalışmanın E-kadherin'deki grup farklılıklarını tespit etmek için yeterli güce sahip olduğunu doğruladı ve bu da anlamlı olmayan bulgunun muhtemelen gerçek bir farkın olmadığını yansıttığını gösterdi.

Sonuç: Endometriozis dokularında artmış c-kit ekspresyonu ile azalmış beta-katenin ekspresyonu, bu moleküllerin endometriozis patogenezine katkıda bulunabileceğini düşündürmektedir. Ancak, E-kaderin ekspresyonunda önemli bir fark bulunmadığından, endometriozis gelişiminde E-kaderinin rolünü kesin olarak belirlemek mümkün değildir.

Anahtar Kelimeler: Endometriozis, endometriyoma, c-kit, E-kadherin, beta-katenin

Introduction

Endometriosis is a chronic inflammatory disease that affects 5-10% of women during their reproductive years, characterized by endometrial glands and stroma located outside the uterine cavity[1]. Although asymptomatic cases are observed, common symptoms of endometriosis include pelvic pain and infertility. Endometriosis has negative effects on patients' daily activities, overall well-being, and sexual function. It is often associated with fatigue and depression, resulting in work loss and a significant economic burden[2]. There are three phenotypes of endometriosis: Superficial endometriosis, ovarian endometrioma and deep infiltrating endometriosis[3]. Understanding the pathogenesis of endometriosis is crucial for achieving effective treatment. Although the disease is common and reduces patients' overall well-being, the underlying molecular and cellular processes remain incompletely elucidated. Key questions, such as the origins of different types of endometriosis and why deep infiltrating endometriosis exhibits cancer-like behavior, remain unanswered. Even though endometriosis is a non-malignant condition, it mimics tumor cells through its formation of new blood vessels, tissue infiltration, and ectopic implantation into distant organs[4,5]. Studies have shown that adhesion molecules and proto-oncogenes involved in tumor pathogenesis may also play a role in the development of endometriosis. In this study, we analyzed the roles of stem cells and adhesion molecules, such as c-kit, E-cadherin, and beta-catenin, potentially involved in endometriosis pathogenesis.

The c-kit receptor (CD117), the gene product of c-kit, constitutes a transmembrane glycoprotein. Several studies provide evidence that the stem cell factor/c-kit signaling axis is involved in the molecular mechanisms driving endometriosis. The E-cadherin/beta-catenin complex is involved in epithelial cell-cell adhesion. Disruption of this integrity and the reduction or absence of adhesion molecules has been associated with the proliferation, migration, and invasion of tumor cells. Endometrial cells located outside the uterus in patients with endometriosis invade other tissues and organs in a manner resembling tumor cells. Various studies have been conducted based on the hypothesis that stem cell expression increases and adhesion molecule levels decrease to enable endometrial cells to migrate and invade other areas during the development of endometriosis, similar to tumor progression. While some studies report increased c-kit proto-oncogene expression and decreased E-cadherin and beta-catenin levels in endometriosis, findings remain inconsistent[6-8]. In order to better understand the mechanisms underlying endometriosis, we analyzed pathological preparations from 180 cases, focusing on the proto-oncogene c-kit and the cell-cell adhesion molecules E-cadherin and beta-catenin.

Materials and Methods

Patient and Tissue Sample Selection

This retrospective case-control study was approved by the Ege University Research Ethics Committee, İzmir, Türkiye (decision number: 21-7T/50, dated 08.07.2021), and was designed following the approval. Between 2015 and 2021, patients aged 18-55 years who underwent surgery for endometriosis or other benign conditions and had pathological examinations were retrospectively selected from the archives of the Department of Obstetrics and Gynecology, Ege University Faculty of Medicine. Pathology slides from the selected cases were retrieved from the medical pathology archive. The slides were examined under a microscope in the presence of a pathologist, and samples containing an adequate number of assessable epithelial and stromal cells were selected for analysis. Based on the review of surgical notes and definitive pathology reports, patients were divided into three groups: those with ovarian endometriotic cysts were assigned to the endometrioma group; those with endometriosis foci in tissues other than the ovary were assigned to the endometriosis group; and those with proliferative endometrium obtained for benign reasons were assigned to the control group. Sixty patients were selected from each group. Patients with genital system malignancies and those with insufficient endometriosis tissue in paraffin blocks were excluded from the study.

Immunohistochemical Examination

Histological evaluation was performed on 3-5 μm-thick sections obtained from formalin-fixed paraffin blocks using immunohistochemical staining for E-cadherin (monoclonal, clone 36, JO1182, Ventana, ready to use), beta-catenin (monoclonal, clone 14, V0003124, Cellmarque, ready-to-use),

and c-kit (CD117, monoclonal, clone YR145, Cellmarque, concentrate; 1:100). Paraffin sections were deparaffinized in xylene and rehydrated sequentially with graded alcohol using a fully automated immunohistochemical staining device (Benchmark XT; Ventana Medical Systems), following the manufacturers' instructions.

Focal limited staining was observed in endometriosis foci with the c-kit antibody and was considered positive. Samples showing no staining were considered negative. Evaluation of E-cadherin and beta-catenin expression was performed separately based on the percentage of stained cells and staining intensity. The percentage of staining was determined as the ratio of stained cells to the total number of cells (0-100%). Staining intensity was classified into four groups: 0=no staining, 1=weak, 2=moderate, and 3=strong staining intensity. The histochemical scoring method (H-score), obtained by multiplying the percentage of cells at each staining intensity by the corresponding intensity score, was used to evaluate staining extent and intensity together, as previously described by McCarty et al.[9].

Since E-cadherin staining was observed only in epithelial cells, it was evaluated exclusively in these cells. However, because beta-catenin staining was observed in both epithelial and stromal cells, these two components were evaluated separately. Representative images of c-kit, E-cadherin, and beta-catenin staining are shown in Figure 1.

Statistical Data Analysis Methods

IBM SPSS Statistics 25.0 (IBM Corp., Released 2017. IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.) was used for statistical analyses.

The normality of numerical variables was assessed using the Kolmogorov-Smirnov test within subgroups. Numerical variables were compared among the three groups using the Kruskal-Wallis test, and Dunn's test with Bonferroni correction was applied for post-hoc pairwise comparisons when significant differences were detected. Ordinal variables were analyzed using the chi-square test (with exact probabilities calculated), and cells contributing to significant differences were identified by adjusted residual Z-scores ($z>1.96$ or $z<-1.96$). A p-value <0.05 was considered statistically significant.

Results

Although no significant difference was found between the endometrioma and endometriosis groups in terms of c-kit positivity rates, both groups exhibited a marked elevation relative to the control group (p=0.005). C-kit positivity rates for each group are presented in Table 1.

No notable difference was detected among the groups in terms of E-cadherin H-scores. The E-cadherin H-scores across the groups are shown in Table 1. E-cadherin staining intensities were also similar among the groups, as shown in Table 2 (p=0.14).

When beta-catenin H-scores were evaluated, a significant difference was observed among the groups (p<0.001). Comparisons between individual groups showed no significant difference between the endometrioma and endometriosis groups (p=0.53), whereas both groups had significantly lower beta-catenin H-scores than the control group (p<0.001). Beta-catenin H-scores for each group are detailed in Tables 1, 3, and 4.

Beta-catenin epithelial and stromal staining intensities were evaluated separately, and a significant difference was found among the groups (p<0.001). Beta-catenin epithelial and stromal staining intensities are summarized in Table 5. Tables 1 and 4 show significant differences in age distribution among the three groups (p<0.001). The mean age in both study groups was lower than that of the control group (p<0.001). The correlation between age and immunohistochemical variables was then analyzed. Table 3 shows a statistically significant but weak positive correlation between beta-catenin H-scores (epithelial and stromal intensity) and age across all cases. Because no significant differences in age distribution were observed among the endometriosis, endometrioma, and control groups with respect to beta-catenin expression, this weak positive correlation was considered not to be clinically meaningful, and no additional statistical analyses were performed.

Discussion

In this study, we evaluated the expression of c-kit, E-cadherin, and beta-catenin—molecules recognized for their role in stem cell signaling and cell-cell adhesion—in different types of endometriosis and compared them with normal proliferative endometrium. Our results demonstrated a significant increase in c-kit expression and a significant decrease in beta-catenin expression in endometriosis tissues. These findings contribute to the growing evidence that stem cell-related pathways and adhesion molecules could play a role in the molecular mechanisms underlying endometriosis, particularly in the initiation and persistence of ectopic lesions.

The significantly higher c-kit expression detected in endometriosis samples relative to normal endometrium supports the hypothesis that the stem cell factor (SCF)/c-kit axis could be involved in endometriosis development. Previous studies have demonstrated elevated SCF levels in the peritoneal fluid of women with endometriosis[10] and increased c-kit expression in ectopic endometrial tissue[10,11]. Our findings are consistent with these reports and suggest that c-kit may contribute to lesion formation and persistence. Although Osuga et al.[10] detected c-kit mRNA expression in both eutopic and ectopic endometrium, this finding may be attributed to the high sensitivity of everse transcription polymerase chain reaction, which allows detection of even very low levels of gene expression. In addition, although Pacchiarotti et al.[11] reported low-level c-kit expression

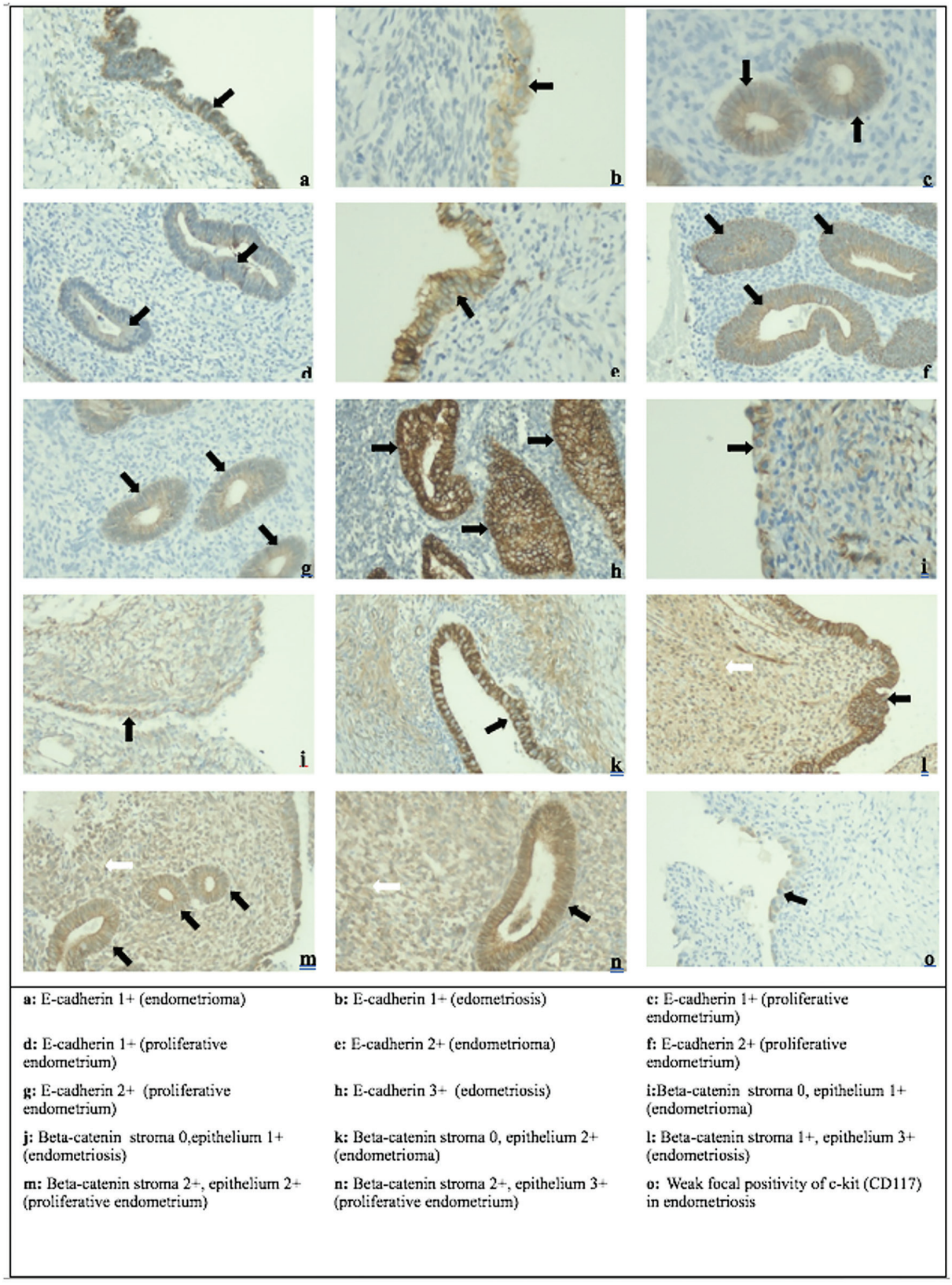

Figure 1. Immunohistochemical staining of c-kit, E-cadherin, and beta-catenin

**Immunopositive cells are indicated by arrows. Black arrows indicate epithelial positivity, and white arrows indicate stromal positivity. Staining intensity was graded as 0 (negative), 1+ (weak), 2+ (moderate), and 3+ (strong), and was semi-quantitatively scored separately in epithelial and stromal components, with higher scores indicating stronger immunoreactivity. For beta-catenin, both epithelial and stromal staining patterns are shown*

Table 1. Comparison of demographic and clinical characteristics among the endometriosis, endometrioma, and control groups

		Endometriosis	Endometrioma	Control group
E-cadherin H-score	Mean	241.33	236.67	251.83
	Std. deviation	34.369	39.172	31.000
	Median	250	240	255
	Minimum	150	150	160
	Maximum	290	300	300
	IQR	48	70	30
	p-value	0.12		
Beta-catenin H-score	Mean	237.83	225	259
	Std. deviation	30.426	42.486	24.886
	Median	240	240	260
	Minimum	160	80	180
	Maximum	300	290	300
	IQR	40	50	30
	p-value	<0.001		
Age	Mean	33.38	35.12	40.85
	Std. deviation	6.268	8.201	7.813
	Median	33	34	42.5
	Minimum	23	21	20
	Maximum	48	48	53
	IQR	10	16	8
	p-value	<0.001		
C-kit	C-kit (-) n (%)	50 (83.3)	51 (85.0)	60 (100.0)
	C-kit (+) n (%)	10 (16.7)	9 (15.0)	0 (0)
	p-value	0.005		

c-kit (+/-): Indicates the presence or absence of c-kit expression. Statistical analyses were performed using the Kruskal-Wallis test for continuous variables and Fisher's exact test for categorical variables. IQR: Interquartile range, Std.: Standard

in eutopic endometrium, no c-kit immunoreactivity was detected in eutopic endometrial samples in the present study. This discrepancy may be explained by the inherently minimal expression of c-kit in eutopic endometrium and by the possibility that immunohistochemistry may fall below the detection threshold, particularly in retrospective studies.

Similar to our study, Pacchiarotti et al.[11] and Osuga et al.[10] did not perform a separate analysis comparing deep and superficial endometriosis. Future studies could be designed to evaluate c-kit expression specifically in deep versus superficial lesions.

Table 2. Distribution of E-cadherin staining intensity among the endometriosis, endometrioma, and control groups

	E-cadherin staining intensity			
	1	2	3	p-value
	n (%)	n (%)	n (%)	
Endometriosis	2 (3.3)	17 (28.3)	41 (68.3)	0.14
Endometrioma	5 (8.3)	19 (31.7)	36 (60.0)	
Control group	1 (1.7)	11 (18.3)	48 (80.0)	

In contrast to c-kit, beta-catenin expression was significantly decreased in endometriosis tissues, particularly within the stromal compartment. Beta-catenin is a key component of the E-cadherin-mediated adhesion complex and plays an important role in maintaining epithelial integrity and cell-cell adhesion[7]. Reduced beta-catenin expression may weaken intercellular adhesion, thereby facilitating cellular detachment, migration, and implantation of endometrial cells at ectopic sites. This finding is consistent with previous studies reporting diminished beta-catenin levels in endometriotic lesions[7,12]. The similar reduction in beta-catenin observed in both endometrioma and other types of endometriosis suggests that impaired adhesion mechanisms may represent a shared molecular feature underlying different endometriosis phenotypes rather than being specific to invasiveness.

E-cadherin, another important adhesion molecule, did not show significant differences in expression levels among the groups in our study. This finding is consistent with several previous reports[7,12,13], although conflicting data exist in the literature. For example, Jedryka et al.[14] reported reduced E-cadherin levels in the serum or peritoneal fluid of patients with endometriosis, whereas other studies have linked decreased expression primarily to recurrent or deep infiltrative forms of the disease[15,16]. These discrepancies may be attributed to methodological heterogeneity, differences in sample type (circulating versus tissue-based measurements), and variability in disease stage or phenotype. Importantly, our findings suggest that alterations in E-cadherin expression may not represent a universal feature of endometriosis but may instead be restricted to specific clinical subtypes or more advanced disease, which could explain the inconsistent results across studies.

Study Limitations

This study presents certain limitations. Due to its retrospective nature, a potential for selection bias exists, which could restrict causal inferences. Although the sample size was adequate to detect significant differences in c-kit and beta-catenin expression, detailed subgroup analyses according to disease phenotype were not performed.

Table 3. Correlation between age and immunohistochemical variables

	Immunohistochemical variables			Endometriosis group			Endometrioma group			Control group		
	Coef	n	p-value	Coef	n	p-value	Coef	n	p-value	Coef	n	p-value
E-cadherin H-score	0.008	180	0.92	0.087	60	0.51	-0.123	60	0.35	-0.084	60	0.52
E-cadherin severity	-0.044	180	0.56	0.010	60	0.94	-0.128	60	0.33	-0.226	60	0.08
Beta-catenin H-score	0.215	180	0.004	0.114	60	0.39	0.288	60	0.03	-0.144	60	0.27
Beta-catenin severity	0.184	180	0.01	0.129	60	0.32	0.181	60	0.17	-0.129	60	0.33
Beta-catenin stromal severity	0.273	180	<0.001	0.250	60	0.05	-0.222	60	0.09	0.053	60	0.69

Coef: Correlation coefficient, n: Sample size

Table 4. Statistical comparison of beta-catenin H-scores and age among the endometrioma, endometriosis, and control groups

Groups	p-value for beta-catenin H-scores	p-value for age
Endometrioma-endometriosis	0.53	0.55
Endometrioma-control group	**<0.001**	**<0.001**
Endometriosis-control group	**<0.001**	**<0.001**

Table 5. Distribution of beta-catenin epithelial and stromal staining intensity among the endometriosis, endometrioma, and control groups

	Beta-catenin epithelial staining intensity				Beta-catenin stromal staining intensity				
	1	2	3	p-value	0	1	2	3	p-value
	n (%)	n (%)	n (%)		n (%)	n (%)	n (%)	n (%)	
Endometriosis	2 (3.3)	18 (30.0)	40 (66.7)	**p<0.001**	23 (38.3)	26 (43.3)	10 (16.7)	1 (1.7)	**p<0.001**
Z-score	-0.7	0.7	-0.3		**4.7**	**2.4**	**-2.4**	**-4.6**	
Endometrioma	6 (10.0)	24 (40.0)	30 (50.0)		11 (18.3)	26 (43.3)	16 (26.7)	7 (11.7)	
Z-score	**2.2**	**2.9**	**-3.7**		-0.1	**2.4**	-0.2	**-2.3**	
Control group	1 (1.7)	6 (10.0)	53 (88.3)		0 (0.0)	5 (8.3)	24 (40.0)	31 (51.7)	
Z-score	**-1.5**	**-3.6**	**4.1**		**-4.6**	**-4.8**	**2.6**	**6.9**	

Z-score: Adjusted residual z-score from chi-square analysis

Immunohistochemistry provided semi-quantitative protein expression data but did not allow functional or molecular-level evaluation. In addition, clinical variables such as prior treatment and disease duration could not be fully controlled.

Conclusion

This study demonstrates that altered expression of c-kit and beta-catenin is associated with the pathogenesis of endometriosis, highlighting their potential roles in lesion formation and maintenance. In contrast, E-cadherin expression did not show a consistent association. These findings provide insight into molecular mechanisms underlying endometriosis and may guide future diagnostic and therapeutic strategies.

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Ethics

Ethics Committee Approval: This retrospective case-control study was approved by the Ege University Research Ethics Committee, İzmir, Türkiye (decision number: 21-7T/50, dated 08.07.2021), and was designed following the approval.
Informed Consent: Informed consent was obtained from all patients who participated in the study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: E.A.A., F.Ş., A.A., G.S., Concept: E.A.A., F.Ş., G.S., Design: E.A.A., G.S. F.Ş., Data Collection or Processing: E.A.A., Analysis or Interpretation: E.A.A., G.S., Literature Search: E.A.A., Writing: E.A.A.
Conflict of Interest: No conflict of interest was declared by the authors.

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