Preoperative predictors of concurrent endometrial carcinoma in patients with endometrial intraepithelial neoplasia: the role of HALP score and other inflammatory markers

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Abstract

Objective: The aim of this study was to identify preoperative factors that predict concurrent endometrial carcinoma in patients with endometrial intraepithelial neoplasia (EIN), focusing on inflammatory markers, such as hemoglobin, albumin, lymphocyte, and platelet (HALP) score, prognostic nutritional index (PNI), the modified systemic inflammatory score (mSIS), clinical characteristics, and imaging findings.

Material and Methods: A retrospective review was conducted of patients diagnosed with EIN who underwent hysterectomy and bilateral salpingo-oophorectomy between 2019 and 2024. Data collected included demographic details, cancer antigen-125 levels, hematological parameters, HALP score, PNI, mSIS, and preoperative endometrial thickness. Statistical analyses were performed to evaluate the associations between these factors and concurrent endometrial carcinoma.

Results: Concurrent endometrial carcinoma was identified in 39 (19.9%) of the total of 196 patients included. Significant predictors included older age ($p<0.001$), lower platelet count ($p<0.001$), and endometrial thickness greater than 13 mm ($p=0.044$). Inflammatory markers such as the HALP score, PNI, and mSIS did not show significant associations. The majority of cases with carcinoma were International Federation of Gynecology and Obstetrics stage IA (76.9%) and grade 1 endometrioid tumors (94.9%).

Conclusion: Advanced age, reduced platelet count, and increased endometrial thickness are key predictors of concurrent endometrial carcinoma in patients with EIN. These findings may be useful for improved preoperative risk stratification and inform surgical planning. Further research is needed to explore the role of inflammatory biomarkers in this context. (J Turk Ger Gynecol Assoc. 2025; 26: 34-40)

Keywords: Endometrial intraepithelial neoplasia, endometrial carcinoma, HALP score, Inflammatory markers, preoperative predictors

Received: 24 December, 2024 **Accepted:** 11 February, 2025 **Publication Date:** 12 March, 2025

Introduction

Endometrial intraepithelial neoplasia (EIN) is recognized as a precursor lesion that significantly increases the risk for the development of endometrioid endometrial carcinoma. Histopathological overlap between EIN and endometrial cancer is not uncommon, with studies reporting that up to 40% of patients diagnosed with EIN may harbor concurrent endometrial carcinoma at the time of hysterectomy (1-3). Several risk factors for the development of EIN and its

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DOI: *10.4274/jtgga.galenos.2025.2024-12-5*

Cite this article as: *Aytekin O, Karagöz Ç, Göktaş E, Tokalıoğlu AA, Tiryaki Güner G, Özkaya Uçar Y, et al. Preoperative predictors of concurrent endometrial carcinoma in patients with endometrial intraepithelial neoplasia: the role of HALP score and other inflammatory markers. J Turk Ger Gynecol Assoc. 2025; 26: 34-40*

progression to cancer have been identified, including metabolic conditions linked to prolonged unopposed estrogen exposure, leading to precancerous endometrial alterations (4).

The potential relationship between inflammation and cancer was first highlighted by Balkwill and Mantovani (5) in the 19th century, who suggested that chronic inflammation might contribute to tumorigenesis. Recent studies have demonstrated that systemic inflammation plays a critical role in the development and progression of some cancers, influencing both tumor growth and patient outcomes (6). Several serum-based inflammatory biomarkers, such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and more complex indices like the modified systemic inflammatory score (mSIS), prognostic nutritional index (PNI), and hemoglobin, albumin, lymphocyte, and platelet (HALP) score, have been investigated for their prognostic value across various malignancies (7-12). There are studies showing that high NLR is associated with poorer prognosis in patients with endometrial cancer (13), and studies reporting that both NLR and PLR are important predictors of prognosis in ovarian cancer (14). Similarly, emerging indices, such as HALP score and PNI have been evaluated in cervical cancer (15,16).

In the present study, the aim was to evaluate whether preoperative factors, including patient demographics, imaging findings, and laboratory parameters, and in particular a range of inflammatory markers, could serve as predictors of concurrent endometrial carcinoma in patients diagnosed with EIN who underwent hysterectomy. It is hoped that this may help refine preoperative risk stratification and guide surgical decision-making.

Material and Methods

This retrospective study analyzed patients diagnosed with EIN who underwent hysterectomy and bilateral salpingo-oophorectomy between 2019 and 2024. Data were obtained from electronic medical records, patient files, and pathology reports. Patients who had undergone fertility-sparing management following an EIN diagnosis were excluded from the study. Furthermore, individuals with concurrent endometrial malignancy identified during endometrial sampling were also excluded. This study was approved by the Ethics Committee of Ankara Bilkent City Hospital (approval number: TABED 1-24-157, date: 24.04.2024).

The collected data included demographic information, cancer antigen-125 (CA-125) levels, hematological parameters, calculated HALP score, PNI, and mSIS, and ultrasound findings in particular endometrial thickness, together with final definitive pathology results. Hematological parameters such as serum albumin, hemoglobin, platelet, lymphocyte, monocyte, and neutrophil counts were recorded preoperatively. The HALP score was calculated using the formula; hemoglobin (g/L) × albumin (g/L) × lymphocyte (10^9/L)/platelet (10^9/L) (17). PNI was determined using the formula: [10 × albumin (g/L) + 0.005 × total lymphocyte count] (12). The mSIS was defined as follows: patients with an albumin level <40 g/L and lymphocyte-to-monocyte ratio (LMR) <4.44 were assigned a score of 2; those with either an albumin level ≥40 g/L or LMR ≥4.44 were assigned a score of 1; and in patients with both an albumin level ≥40 g/L and LMR ≥4.44 the assigned score was 0 (18).

Frozen/section analysis was routinely performed on all EIN patients. Patients who met one of the following criteria underwent routine pelvic and para-aortic lymphadenectomy: grade 1 or 2 endometrioid adenocarcinoma with a tumor size ≥2 cm, >50% myometrial invasion, all grade 3 endometrioid adenocarcinomas, extrauterine metastasis, cervical involvement, and any non-endometrioid adenocarcinomas. Staging was carried out based on the revised 2009 International Federation of Gynecology and Obstetrics (FIGO) criteria (19).

Statistical analysis

Statistical analysis was performed using IBM SPSS, version 22.0 for Windows (SPSS Inc., Chicago, IL, USA). Descriptive statistics were expressed as mean ± standard deviation or median (range) for continuous variables and as number (percentage) for categorical variables. The chi-square test was used to assess categorical variables. A p-value of less than 0.05 was considered statistically significant.

Results

A total of 196 patients diagnosed with EIN based on endometrial sampling were included in this study. The mean age of the patients was 51.4±9.5 years, ranging 31-83 years. The median preoperative values for CA-125, albumin, hemoglobin, platelet count, lymphocytes, monocytes, and neutrophils were 10.9 IU/mL, 45.5 g/L, 12.8 g/dL, 291x10^9/L, 2x10^9/L, 0.4x10^9/L, and 4.5x10^9/L, respectively. The mean NLR was 2.5±1.2, MLR was 0.2±0.2, PLR was 153.8±56.2, HALP score was 43.3±21.9, and PNI was 46.2±3.4. The mean preoperative endometrial thickness was 11±5.8 mm. The mSIS was 0 in 170 patients (86.7%) and 1 in 26 patients (13.3%). Final pathology results showed that 39 patients (19.9%) had concurrent endometrial cancer. The clinical characteristics and pathological outcomes of the patients are detailed in Table 1.

The pathological characteristics of the 39 patients with concurrent endometrial cancer are presented in Table 2. The most common FIGO stage was IA, which was found in 30 (76.9%). All patients had endometrioid-type tumors, with 37 (94.9%) being classified as FIGO grade 1. Twelve (30.8%) had

Table 1. Clinical characteristics of the patients (n=196 patients)

Features		Mean ± SD	Median (range)
Age (years)		51.4±9.5	50 (31-83)
CA-125 (IU/mL)		16.3±26.1	10.9 (2-213)
Albumin (g/L)		45.2±3.4	45.5 (28-52)
Hemoglobin (g/dL)		12.8±1.6	12.9 (6.3-16.6)
Platelet (10^9/L)		300±80	291 (127-538)
Lymphocyte (10^9/L)		2.2±1.1	2 (0.6-14.1)
Monocyte (10^9/L)		0.4±0.4	0.4 (0.2-5.8)
Neutrophil (10^9/L)		4.9±1.9	4.5 (0.3-12.6)
Neutrophil-to-lymphocyte ratio		2.5±1.2	2.2 (0.1-8.7)
Monocyte-to-lymphocyte ratio		0.2±0.2	0.2 (0.03-2.1)
Platelet-to-lymphocyte ratio		153.8±56.2	142.7 (21-422)
HALP score[1]		43.3±21.9	40.4 (11-243)
PNI[2]		46.2±3.4	46.5 (28.3-53.3)
Endometrial thickness (mm)[3]		11±5.8	10.5 (1-35)
		n	**%**
mSIS[4]	0	170	86.7
	1	26	13.3
	2	0	0
Final pathology	EIN	157	80.1
	Cancer	39	19.9
Ovarian pathology	Benign	195	99.5
	Cancer[5]	1	0.5

[1]: HALP: Hemoglobin (g/L) x albumin (g/L) x lymphocyte (n/L)/platelet (n/L), [2]: PNI: Prognostic nutritional index, [3]: (n=170) patients (endometrial thickness value was not reported in 26 patients), [4]: mSIS: Modified systemic inflammatory score, [5]: Adult granulosa cell tumor, SD: Standard deviation, CA-125: Cancer antigen-125

Table 2. Pathologic features of patients with endometrial cancer (n=39)

Pathologic characteristics		n	%
FIGO 2009 stage	IA	30	76.9
	IB	6	15.4
	II	2	5.1
	IIIC2	1	2.6
FIGO grade	Endometrioid grade 1	37	94.9
	Endometrioid grade 2	1	2.6
	Endometrioid grade 3	1	2.6
Depth of myometrial invasion	No invasion	12	30.8
	<1/2	18	46.2
	≥1/21	8	4.1
	Serosal invasion	1	0.5
Lymphovascular space invasion	Negative	34	87.2
	Positive	5	12.8
Cervical invasion	Negative	35	89.7
	Glandular invasion	1	2.6
	Stromal ± glandular invasion	3	7.7
Peritoneal cytology	Negative	39	100
	Positive	0	0
Lymphadenectomy	Not performed	30	76.9
	Performed	9	23.1

FIGO: International Federation of Gynecology and Obstetrics, [1]: Except serosal invasion

no myometrial invasion, while 1 (0.5%) had serosal invasion. Lymphovascular space invasion was present in 5 (12.8%), and cervical stromal and/or glandular invasion was noted in 3 (7.7%).

Table 3 presents the relationship between preoperative clinical factors and inflammatory markers and the presence of concurrent endometrial cancer. A significant relationship was found between higher age, lower platelet counts and the presence of concurrent endometrial carcinoma. A significant correlation (p=0.044) was identified between an endometrial thickness exceeding 13 mm and the presence of concurrent endometrial cancer, with a sensitivity of 42.4%, specificity of 75.2%, positive predictive value of 29.2%, and negative predictive value of 24.4% (Table 4).

Discussion

The aim of the present study was to evaluate predictive factors for concurrent endometrial carcinoma in patients diagnosed with EIN undergoing hysterectomy. Among 196 patients included in the study, 19.9% were found to have concurrent endometrial

Table 3. Association of preoperative clinical variables and inflammatory markers with concurrent endometrial cancer

Factor		EIN	Cancer	Sensitivity, (%)	Specificity, (%)	PPV, (%)	NPV, (%)	p
		n (%)	n (%)					
Age (years)[1]	≤50	97 (89.8)	11 (10.2)	71.8	61.8	31.8	89.8	**<0.001**
	>50	60 (68.2)	28 (31.8)					
CA-125 (IU/mL)[1]	≤10.9	83 (82.2)	18 (17.8)	53.8	52.9	22.1	82.2	0.453
	>10.9	74 (77.9)	21 (22.1)					
Albumin (g/L)[1]	>45.5	80 (81.6)	18 (18.4)	53.8	51.0	21.4	81.6	0.591
	≤45.5	77 (78.6)	21 (21.4)					
Hemoglobin (g/dL)[1]	>12.9	77 (80.2)	19 (19.8)	51.3	49.0	20.0	80.2	0.971
	≤12.9	80 (80.0)	20 (20.0)					
Platelet (10^9/L)[1]	>291	89 (90.8)	9 (9.2)	76.9	56.7	30.6	90.8	**<0.001**
	≤291	68 (69.4)	30 (30.6)					
Lymphocyte (10^9/L)[1]	≤2	77 (76.2)	24 (23.8)	38.5	49.0	15.8	76.2	0.162
	>2	80 (84.2)	15 (15.8)					
Monocyte (10^9/L)[1]	≤0.4	100 (83.3)	20 (16.7)	48.7	63.7	25.0	83.3	0.154
	>0.4	57 (75.0)	19 (25.0)					
Neutrophil (10^9/L)[1]	≤4.5	79 (79.0)	21 (21.0)	46.2	50.3	18.8	79.0	0.693
	>4.5	78 (81.3)	18 (18.8)					
Neutrophil-to-lymphocyte ratio[1]	≤2.2	78 (83.0)	16 (17.0)	59.0	49.7	22.5	83.0	0.333
	>2.2	79 (77.5)	23 (22.5)					
Monocyte-to-lymphocyte ratio[1]	≤0.2	100 (82.6)	21 (17.4)	46.2	63.7	24.0	82.6	0.257
	>0.2	57 (76.0)	18 (24.0)					
Platelet-to-lymphocyte ratio[1]	≤142.7	78 (78.8)	21 (21.2)	46.2	49.7	18.6	78.8	0.642
	>142.7	79 (81.4)	18 (18.6)					
HALP score[1,2]	>40.4	76 (77.6)	22 (22.4)	43.6	48.4	17.3	77.6	0.371
	≤40.4	81 (82.7)	17 (17.3)					
PNI[1,3]	>46.5	82 (82.8)	17 (17.2)	56.4	52.2	22.7	82.8	0.334
	≤46.5	75 (77.3)	22 (22.7)					
Endometrial thickness (mm)[1,4]	≤10.5	68 (80.0)	17 (20.0)	48.5	49.6	18.8	80.0	0.846
	>10.5	69 (81.2)	16 (18.8)					
mSIS[1,5]	0	138 (81.2)	32 (18.8)	17.9	87.9	26.9	81.2	0.335
	1	19 (73.1)	7 (26.9)					

[1]: Median value, [2]: HALP: Hemoglobin (g/L) x albumin (g/L) x lymphocyte (n/L)/platelet (n/L), [3]: PNI: Prognostic nutritional index, [4]: (n=170) patients (endometrial thickness value wasn't reported in 26 patients), [5]: mSIS: Modified systemic inflammatory score, EIN: Endometrial intraepithelial neoplasia, PPV: Positive predictive value, NPV: Negative predictive value

Table 4. Association of endometrial thickness with concurrent endometrial cancer

Endometrial thickness[1]	EIN	Cancer	Sensitivity, (%)	Specificity, (%)	PPV, (%)	NPV, (%)	p
	n (%)	n (%)					
≤3 mm	9 (75.0)	3 (25.0)	90.9	6.6	19.0	75.0	0.612
>3 mm	128 (81.0)	30 (19.0)					
≤4 mm	13 (68.4)	6 (31.6)	81.8	9.5	17.9	68.4	0.155
>4 mm	124 (82.1)	27 (17.9)					
≤5 mm	21 (75.0)	7 (25.0)	78.8	15.3	18.3	75.0	0.413
>5 mm	116 (81.7)	26 (18.3)					
≤6 mm	25 (73.5)	9 (26.5)	72.7	18.2	17.6	73.5	0.245
>6 mm	112 (82.4)	24 (17.6)					
≤7 mm	36 (78.3)	10 (21.7)	69.7	26.3	18.5	78.3	0.640
>7 mm	101 (81.5)	23 (18.5)					
≤8 mm	50 (78.1)	14 (21.9)	57.6	36.5	17.9	78.1	0.528
>8 mm	87 (82.1)	19 (17.9)					
≤9 mm	58 (79.5)	15 (20.5)	54.5	42.3	18.6	79.5	0.745
>9 mm	79 (81.4)	18 (18.6)					
≤10 mm	68 (80.0)	17 (20.0)	48.5	49.6	18.8	80.0	0.846
>10 mm	69 (81.2)	16 (18.8)					
≤11 mm	78 (81.3)	18 (18.8)	45.5	56.9	20.3	81.3	0.804
>11 mm	59 (79.7)	15 (20.3)					
≤12 mm	90 (83.3)	18 (16.7)	45.5	65.7	24.2	83.3	0.232
>12 mm	47 (75.8)	15 (24.2)					
≤13 mm	103 (84.4)	19 (15.6)	42.4	75.2	29.2	84.4	**0.044**
>13 mm	34 (70.8)	14 (29.2)					

[1]: (n=170) patients (endometrial thickness value was not reported in 26 patients), EIN: Endometrial intraepithelial neoplasia, PPV: Positive predictive value, NPV: Negative predictive value

carcinoma based on final pathology results. This is consistent with previous studies that report concurrent carcinoma rates ranging from 20% to 40% in patients with EIN, underscoring the significant overlap between EIN and endometrial cancer (1,20).

A key finding of our study was the association between higher age and the presence of concurrent endometrial carcinoma. This result is consistent with prior studies, which highlights increasing age as a significant risk factor for endometrial hyperplasia progression and cancer development (21). Aging is associated with prolonged exposure to unopposed estrogen, as well as age-related alterations in immune and inflammatory responses, which may further predispose patients to carcinogenesis. In the study conducted by Giannella et al. (22), and similar to our findings, patients with concurrent cancer were older.

Endometrial thickness, as measured preoperatively on ultrasound, was also found to be significantly associated with concurrent endometrial carcinoma, particularly for values exceeding 13 mm in our cohort. This finding confirms previous studies that have identified increased endometrial thickness as a predictor of malignancy in patients with EIN (23,24). However, the sensitivity and specificity of this cut-off remain suboptimal (42.4% and 75.2%, respectively), highlighting the need for multimodal risk assessment strategies that include clinical, radiological, and laboratory parameters.

Inflammatory biomarkers, including the HALP score, PNI, and mSIS were also evaluated. Although these indices have been shown to predict prognosis and recurrence in various malignancies (9,25-29), they did not reach statistical significance in predicting concurrent endometrial carcinoma in the present study. This may be attributed to the early-stage nature of the disease in most patients or the relatively small sample size, or both. However, given their availability in other malignancies, future studies with larger cohorts are needed to validate their predictive value in patients with EIN.

The current study also identified an association between lower platelet counts and concurrent endometrial carcinoma in patients with EIN. While the underlying mechanism remains unclear, this finding may reflect alterations in the inflammatory or hematopoietic environment associated with malignancy. Further studies are needed to explore this relationship.

Study Limitations

The strengths of our study include the relatively large sample size and comprehensive evaluation of preoperative factors, including inflammatory biomarkers, hematological parameters, and imaging findings. However, certain limitations should be acknowledged. First, the retrospective nature of the study introduces potential selection and information biases. Second, the sample size, while sufficient for preliminary analysis, may limit the power to detect associations between inflammatory markers and concurrent carcinoma.

Conclusion

The present study found and confirmed earlier results that older age, lower platelet counts, and endometrial thickness greater than 13 mm were significant predictors of concurrent endometrial carcinoma in patients with EIN undergoing hysterectomy. It is hoped that these findings can aid in refining preoperative risk stratification and surgical decision-making. Further, larger prospective studies are needed to validate the role of systemic inflammatory biomarkers and other preoperative factors in predicting concurrent malignancy in this patient population.

Ethics

Ethics Committee Approval: *This study was approved by the Ethics Committee of Ankara Bilkent City Hospital (approval number: TABED 1-24-157, date: 24.04.2024).*

Informed Consent: *Retrospective study.*

Footnotes

Author Contributions: *Surgical and Medical Practices: O.A., Ç.K., Y.Ö.U., T.T., Concept: Y.Ö.U., F.K., T.T., Design: F.K., T.T., Data Collection or Processing: O.A., Ç.K., E.G., G.T.G., Analysis or Interpretation: O.A., Ç.K., A.A.T., Literature Search: O.A., E.G., A.A.T., G.T.G., Writing: O.A., T.T.*

Conflict of Interest: *No conflict of interest is declared by the authors.*

Financial Disclosure: *The authors declared that this study received no financial support.*

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