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Original Article

Construction and validation of a Cox regression-based nomogram model for predicting recurrence risk in early endometrial cancer

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Abstract: Objectives: To develop and validate a Cox regression-based nomogram model for predicting recurrence risk in early-stage endometrial cancer. Methods: We retrospectively analyzed 1,540 patients with FIGO stage I-II disease treated between January 2013 and December 2021, of whom 247 (16.04%) experienced recurrence and 1,293 did not. Key predictive factors were identified using Lasso-Cox regression, and a nomogram was constructed and evaluated in training (n=924), validation (n=308), and testing (n=308) cohorts. Results: The model demonstrated strong discriminative ability, with C-index values of 0.748, 0.684, and 0.677, and AUCs of 0.767, 0.701, and 0.694 across the three cohorts. Compared with the traditional Naples Prognostic Score, the nomogram showed significantly better performance in both the training cohort (AUC 0.767 vs. 0.687, P=0.009) and the validation cohort (AUC 0.701 vs. 0.580, P=0.041). Calibration curves showed good agreement between predicted and observed outcomes, and decision curve analysis confirmed substantial net clinical benefit, with net reclassification improvement supporting superior accuracy. Conclusions: The developed nomogram provides a reliable and effective tool for individualized recurrence risk assessment in early-stage endometrial cancer, demonstrating significant clinical potential for improved risk prediction and treatment planning.

Keywords: Endometrial cancer, recurrence risk, nomogram, Cox regression, prediction model, naples prognostic score

Introduction

Endometrial cancer (EC) is one of the most common malignancies of the female reproductive system, with incidence rates steadily increasing globally. In developed countries, it is now the fourth most prevalent cancer among women [1]. Early-stage EC (FIGO stages IA and IB), characterized by localized disease and low invasiveness, typically presents with favorable outcomes, with 5-year overall survival rates of 80%-90% [2]. However, approximately 10%-20% of patients still experience recurrence. Research by Hong et al. [3] indicates that high-risk subgroups, including those with non-endometrioid histology and G3 grade, face poorer prognoses [4].

The recurrence patterns further complicate prognosis. Gaudet et al. [5] reported that patients with peritoneal carcinomatosis recurrence have significantly worse outcomes, with a median survival of only 12 months. Recurrence not only reduces quality of life but also increases treatment complexity and healthcare resource use. Therefore, accurately identifying high-risk subgroups for recurrence in early-stage EC is crucial for developing individualized treatment protocols, optimizing follow-up strategies, and improving long-term outcomes.

Traditionally, recurrence risk assessment has relied on clinical and pathological factors [6]. Studies have identified factors such as age ≥55, BMI ≥28 kg/m^2, G3 grading, FIGO stage

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IB, myometrial invasion ≥1/2, and presence of lymphovascular space invasion (LVSI) as significant risk indicators for recurrence [7]. However, these individual factors do not provide a comprehensive risk assessment. Traditional prognostic systems, such as the Neoadjuvant Prognostic Score (NPS), integrate some clinical features but lack precision, particularly in early-stage patients [8]. Thus, developing a comprehensive multivariable prediction model for recurrence risk assessment has become an urgent need.

In recent years, multivariable analysis-based prediction models, especially nomograms, have shown substantial potential in oncological prognostics. Nomograms integrate multiple variables and can quantify individualized disease risks. They have been widely used in predicting outcomes in cancers such as breast and prostate cancer [9, 10]. Unlike traditional scoring systems, nomograms offer more precise risk assessments, providing specific recurrence-free survival probabilities at 24 and 36 months, thus aiding clinicians in developing personalized treatment and follow-up plans. Lasso-Cox regression analysis further enhances predictive accuracy by screening key factors while minimizing overfitting risk [11]. However, research on nomogram models for early-stage EC recurrence is limited, with most studies focusing on advanced-stage patients or single-variable analyses.

This study aims to develop and validate a multivariable nomogram model for predicting recurrence risk in early-stage EC. The objectives are: (1) To retrospectively analyze clinical and pathological data from early-stage EC patients, identifying risk factors associated with recurrence; (2) To construct a nomogram model predicting 24-month and 36-month recurrence-free survival probabilities using Lasso-Cox regression; (3) To evaluate the model's performance using ROC curves, calibration curves, decision curve analysis (DCA), and net reclassification improvement (NRI), and compare its predictive performance with the traditional NPS. This research will provide a precise, practical tool for recurrence risk prediction, optimizing clinical decision-making and follow-up strategies for early-stage EC patients.

Methods and materials

Sample size calculation

This study referenced Huang et al. [12], where the recurrence rate for stage I EC patients was 17.6%. Using the formula $N = Z^2 \times [P \times (1-P)]/E^2$, where Z=1.96, E=0.05, and P=0.176, the required sample size was calculated as 223. Additionally, based on Dou et al. [13], which reported a 36-month recurrence rate of 6.4% in EC patients with LVSI (HR=3.36), the Schoenfeld formula indicated that a minimum of 334 cases would be necessary.

General data collection

We retrospectively analyzed 1,540 patients with FIGO stage I-II endometrial cancer treated at our institution between January 2013 and December 2021. Patients were divided into a recurrence group (247 cases, 16.04% recurrence rate) and a non-recurrence group (1,293 cases, 83.96%). Using R software, we randomly split the samples into training, validation, and testing groups for independent model assessment (6:2:2). The study was approved by the medical ethics committee of Northwest Women's and Children's Hospital.

Inclusion and exclusion criteria

Inclusion criteria: (1) Patients confirmed with FIGO stage I EC [14]. (2) Age ≥18 years. (3) Definitive diagnosis of EC based on pathological standards. (4) Complete clinical data and follow-up records.

Exclusion criteria: (1) Patients who received other treatments before initial surgery (e.g., preoperative radiotherapy, chemotherapy, hormone therapy). (2) Patients who did not undergo total hysterectomy with bilateral salpingo-oophorectomy. (3) Concurrent malignancies (e.g., ovarian cancer, breast cancer, colorectal cancer) or severe comorbidities (e.g., heart failure, respiratory failure) that significantly affect prognosis.

Clinical data collection

We systematically collected clinical and pathological data from electronic medical records and outpatient follow-up. Variables collected included:

Demographics: Age, BMI, menopausal status, comorbidities.

Tumor characteristics: Tumor diameter, histology, grade, FIGO stage, myometrial invasion depth, etc.

Pathological features: LVSI, cervical stromal invasion, lymph node metastasis, estrogen receptor (ER) and progesterone receptor (PR) status.

Treatment information: Surgical approach, lymph node dissection, adjuvant therapy.

Prognostic data: NPS, newly established risk score, and follow-up data (e.g., recurrence status, survival).

Immunohistochemistry and molecular testing

Immunohistochemistry (IHC) for p53 and Ki-67 was performed on formalin-fixed, paraffin-embedded tumor sections using automated staining (Ventana BenchMark ULTRA, Roche Diagnostics). Primary antibodies included p53 (clone DO-7, Roche Ventana CONFIRM) and Ki-67 (clone MIB-1, Agilent/Dako M7240). Staining was interpreted as mutant-type (p53-abn) when ≥80% of tumor nuclei showed diffuse strong nuclear positivity or absent nuclear staining with intact internal controls. The Ki-67 labeling index was calculated by counting at least 500 tumor cells, defining ≥50% positivity as high proliferation. Discrepancies in p53 interpretation were resolved by consensus or molecular testing (NGS, droplet digital PCR, or Sanger sequencing).

Calculation of the NPS

The NPS was calculated based on four factors: serum albumin, total cholesterol, neutrophil-to-lymphocyte ratio (NLR), and lymphocyte-to-monocyte ratio (LMR). Adverse factors (albumin <40 g/L, total cholesterol <180 mg/dL, NLR >2.96, LMR ≤4.44) received 1 point each. The total score ranged from 0 to 4, with higher scores indicating worse prognosis.

Follow-up

Follow-up occurred over three years with outpatient visits and telephone contact: every 3 months during the first year and every 6 months in subsequent years. Recurrence was confirmed by clinical symptoms and imaging, with biopsy confirmation when feasible. Recurrence-free survival (RFS) was calculated from primary surgery to recurrence or last follow-up without recurrence.

Outcome measurements

The primary outcome was the construction of a new risk prediction model using clinical and pathological data, compared with the traditional NPS system. Model performance was evaluated using C-index and NRI. Secondary outcomes included comparing recurrence-free survival between risk groups, analyzing recurrence pattern distribution, and validating the model in independent cohorts.

Statistical analysis

All statistical analyses were performed using R software (version 4.3.3). Continuous variables were presented as mean ± standard deviation (SD) if normally distributed or as median [interquartile range (IQR)] if non-normally distributed, following assessment of normality using the Kolmogorov-Smirnov test. Categorical variables were presented as frequency and percentage [n (%)]. Group comparisons for continuous variables used independent samples t-tests for normally distributed data or Mann-Whitney U tests for non-normally distributed data. Categorical variables were compared using chi-square tests or Fisher's exact tests. Cox proportional hazards regression models were used for survival analysis. Variables with $P<0.05$ in univariate analysis were included in multivariable Cox regression. LASSO-Cox regression was applied to select predictors. Model performance was assessed using Harrell's C-index, calibration curves, and DCA. Differences in ROC curves were compared using DeLong tests, and NRI indices were calculated for model comparison. Kaplan-Meier survival curves and log-rank tests were used to evaluate survival differences. Statistical significance was set at $P<0.05$. Main R packages used included: dplyr, survival, survminer, ggplot2, patchwork, forestplot, and nricens.

Results

Comparison of clinical and pathological characteristics between recurrence and non-recurrence groups

Our comparative analysis of early-stage (IA, IB) EC patients revealed significant differences in

Table 1. Comparison of clinical and pathological characteristics between recurrence and non-recurrence groups in early endometrial cancer patients

Variable	Total	Recurrence Group (n=247)	Non-recurrence Group (n=1293)	χ^2 Value	*P* Value
Age				55.531	<0.001
≥55 years	885 (57.47%)	195 (78.95%)	690 (53.36%)		
<55 years	655 (42.53%)	52 (21.05%)	603 (46.64%)		
BMI				51.007	<0.001
≥28	482 (31.30%)	125 (50.61%)	357 (27.61%)		
<28	1058 (68.70%)	122 (49.39%)	936 (72.39%)		
Menopausal Status				0.709	0.400
Yes	854 (55.45%)	143 (57.89%)	711 (54.99%)		
No	686 (44.55%)	104 (42.11%)	582 (45.01%)		
Parity				2.799	0.094
≥2	1319 (85.65%)	220 (89.07%)	1099 (85.00%)		
<2	221 (14.35%)	27 (10.93%)	194 (15.00%)		
Hypertension History				0.474	0.491
Yes	273 (17.73%)	40 (16.19%)	233 (18.02%)		
No	1267 (82.27%)	207 (83.81%)	1060 (81.98%)		
Diabetes History				2.435	0.119
Yes	146 (9.48%)	30 (12.15%)	116 (8.97%)		
No	1394 (90.52%)	217 (87.85%)	1177 (91.03%)		
Surgical Approach				0.645	0.422
Laparotomy	778 (50.52%)	119 (48.18%)	659 (50.97%)		
Laparoscopy	762 (49.48%)	128 (51.82%)	634 (49.03%)		
Hysterectomy Extent				0.602	0.438
Extrafascial	1329 (86.30%)	217 (87.85%)	1112 (86.00%)		
Other	211 (13.70%)	30 (12.15%)	181 (14.00%)		
Pelvic LN Removal				0.587	0.444
Yes	1273 (82.66%)	200 (80.97%)	1073 (82.99%)		
No	267 (17.34%)	47 (19.03%)	220 (17.01%)		
Pathological Grading				63.382	<0.001
G1	484 (31.43%)	40 (16.19%)	444 (34.34%)		
G2	886 (57.53%)	149 (60.32%)	737 (57.00%)		
G3	170 (11.04%)	58 (23.48%)	112 (8.66%)		
Pathological Type				0.913	0.339
EAC	1350 (87.66%)	212 (85.83%)	1138 (88.01%)		
NEAC	190 (12.34%)	35 (14.17%)	155 (11.99%)		
FIGO Staging				75.224	<0.001
IB	430 (27.92%)	125 (50.61%)	305 (23.59%)		
IA	1110 (72.08%)	122 (49.39%)	988 (76.41%)		
Tumor Lesion Diameter				0.822	0.365
≥2 cm	1407 (91.36%)	222 (89.88%)	1185 (91.65%)		
<2 cm	133 (8.64%)	25 (10.12%)	108 (8.35%)		
Myometrial Invasion Depth				75.224	<0.001
≥1/2	430 (27.92%)	125 (50.61%)	305 (23.59%)		
<1/2	1110 (72.08%)	122 (49.39%)	988 (76.41%)		
LVSI				33.255	<0.001
Yes	182 (11.82%)	56 (22.67%)	126 (9.74%)		
No	1358 (88.18%)	191 (77.33%)	1167 (90.26%)		

ER				5.716	0.017
Positive	1188 (77.14%)	205 (83.00%)	983 (76.02%)		
Negative	352 (22.86%)	42 (17.00%)	310 (23.98%)		
PR				0.814	0.367
Positive	1319 (85.65%)	207 (83.81%)	1112 (86.00%)		
Negative	221 (14.35%)	40 (16.19%)	181 (14.00%)		
P53				29.254	<0.001
Mutant type	869 (56.43%)	178 (72.06%)	691 (53.44%)		
Wild type	671 (43.57%)	69 (27.94%)	602 (46.56%)		
Ki-67 Positivity Rate (%) ≥38%				19.836	<0.001
Yes	533 (34.61%)	116 (46.96%)	417 (32.25%)		
No	1007 (65.39%)	131 (53.04%)	876 (67.75%)		
P16				0.513	0.474
Positive	1176 (76.36%)	193 (78.14%)	983 (76.02%)		
Negative	364 (23.64%)	54 (21.86%)	310 (23.98%)		
NPS	2.00 [1.00, 3.00]	2.00 [2.00, 3.00]	2.00 [1.00, 3.00]	5.571	<0.001

Note: BMI, Body Mass Index; FIGO, International Federation of Gynecology and Obstetrics; LVSI, Lymphovascular Space Invasion; ER, Estrogen Receptor; PR, Progesterone Receptor; NPS, Neoadjuvant Prognostic Score.

clinical and pathological characteristics between recurrence and non-recurrence groups. Statistical significance was observed for age (P<0.001), BMI (P<0.001), pathological grading (P<0.001), FIGO staging (P<0.001), myometrial invasion depth (P<0.001), LVSI (P<0.001), ER status (P=0.017), P53 status (P<0.001), Ki-67 positivity rate (P<0.001), and NPS score (P<0.001). The recurrence group had higher proportions of patients with age ≥55 years, BMI ≥28, higher pathological grade (G3), FIGO stage IB, myometrial invasion depth ≥1/2, presence of LVSI, ER positivity, mutant-type P53, and Ki-67 positivity rate ≥38%, as well as higher NPS scores, suggesting these factors are correlated with increased recurrence risk. Other variables showed no significant differences, including menopausal status (P=0.400), parity (P=0.094), hypertension history (P=0.491), diabetes history (P=0.119), surgical approach (P=0.422), hysterectomy extent (P=0.438), pelvic lymphadenectomy (P=0.444), pathological type (P=0.339), tumor lesion diameter (P=0.365), PR (P=0.367), and P16 status (P=0.474) (**Table 1**).

Comparison of clinical and pathological characteristics among training, validation, and testing groups

We compared the distribution of clinical and pathological characteristics among the training, validation, and testing groups in early-stage (IA, IB) EC patients. No statistically significant differences were observed across the three groups for variables such as age (P=0.583), BMI (P=0.346), menopausal status (P=0.671), parity (P=0.615), hypertension history (P=0.495), diabetes history (P=0.096), surgical approach (P=0.853), hysterectomy extent (P=0.556), pelvic lymphadenectomy (P=0.515), pathological grading (P=0.909), pathological type (P=0.251), FIGO staging (P=0.356), tumor lesion diameter (P=0.446), myometrial invasion depth (P=0.356), LVSI (P=0.992), ER (P=0.664), PR (P=0.117), P53 status (P=0.416), Ki-67 positivity rate (P=0.759), P16 status (P=0.399), and NPS (P=0.419). This consistency in baseline characteristics ensures the suitability of these groups for subsequent model training and validation (**Table 2**).

Comparison of clinical and pathological characteristics between recurrence and non-recurrence groups in training cohort

In the training cohort, significant differences were found between recurrence and non-recurrence groups for age (P<0.001), BMI (P<0.001), pathological grading (P<0.001), FIGO staging (P<0.001), myometrial invasion depth (P<0.001), LVSI (P<0.001), P53 status (P=0.002), Ki-67 positivity rate (P=0.030), tumor lesion diameter (P=0.028), and NPS (P<0.001). The recurrence group showed higher proportions of patients with age ≥55 years, BMI ≥28, higher pathological grade (G3), FIGO stage IB,

Table 2. Comparison of clinical and pathological characteristics among training, validation, and testing groups in early endometrial cancer patients

Variable	Total	Training Group (n=924)	Validation Group (n=308)	Testing Group (n=308)	χ^2 Value	*P* Value
Age					1.080	0.583
≥55 years	885 (57.47%)	526 (56.93%)	174 (56.49%)	185 (60.06%)		
<55 years	655 (42.53%)	398 (43.07%)	134 (43.51%)	123 (39.94%)		
BMI					2.122	0.346
≥28	482 (31.30%)	299 (32.36%)	97 (31.49%)	86 (27.92%)		
<28	1058 (68.70%)	625 (67.64%)	211 (68.51%)	222 (72.08%)		
Menopausal Status					0.799	0.671
Yes	854 (55.45%)	504 (54.55%)	176 (57.14%)	174 (56.49%)		
No	686 (44.55%)	420 (45.45%)	132 (42.86%)	134 (43.51%)		
Parity					0.972	0.615
≥2	1319 (85.65%)	786 (85.06%)	269 (87.34%)	264 (85.71%)		
<2	221 (14.35%)	138 (14.94%)	39 (12.66%)	44 (14.29%)		
Hypertension History					1.407	0.495
Yes	273 (17.73%)	162 (17.53%)	50 (16.23%)	61 (19.81%)		
No	1267 (82.27%)	762 (82.47%)	258 (83.77%)	247 (80.19%)		
Diabetes History					4.696	0.096
Yes	146 (9.48%)	82 (8.87%)	39 (12.66%)	25 (8.12%)		
No	1394 (90.52%)	842 (91.13%)	269 (87.34%)	283 (91.88%)		
Surgical Approach					0.319	0.853
Laparotomy	778 (50.52%)	467 (50.54%)	152 (49.35%)	159 (51.62%)		
Laparoscopy	762 (49.48%)	457 (49.46%)	156 (50.65%)	149 (48.38%)		
Hysterectomy Extent					1.175	0.556
Extrafascial	1329 (86.30%)	801 (86.69%)	260 (84.42%)	268 (87.01%)		
Other	211 (13.70%)	123 (13.31%)	48 (15.58%)	40 (12.99%)		
Pelvic LN Removal					1.326	0.515
Yes	1273 (82.66%)	767 (83.01%)	258 (83.77%)	248 (80.52%)		
No	267 (17.34%)	157 (16.99%)	50 (16.23%)	60 (19.48%)		
Pathological Grading					1.005	0.909
G1	484 (31.43%)	286 (30.95%)	104 (33.77%)	94 (30.52%)		
G2	886 (57.53%)	535 (57.90%)	171 (55.52%)	180 (58.44%)		
G3	170 (11.04%)	103 (11.15%)	33 (10.71%)	34 (11.04%)		
Pathological Type					2.762	0.251
EAC	1350 (87.66%)	807 (87.34%)	278 (90.26%)	265 (86.04%)		
NEAC	190 (12.34%)	117 (12.66%)	30 (9.74%)	43 (13.96%)		
FIGO Staging					2.065	0.356
IB	430 (27.92%)	249 (26.95%)	96 (31.17%)	85 (27.60%)		
IA	1110 (72.08%)	675 (73.05%)	212 (68.83%)	223 (72.40%)		
Tumor Lesion Diameter					1.613	0.446
≥2 cm	1407 (91.36%)	840 (90.91%)	287 (93.18%)	280 (90.91%)		
<2 cm	133 (8.64%)	84 (9.09%)	21 (6.82%)	28 (9.09%)		
Myometrial Invasion Depth					2.065	0.356
≥1/2	430 (27.92%)	249 (26.95%)	96 (31.17%)	85 (27.60%)		
<1/2	1110 (72.08%)	675 (73.05%)	212 (68.83%)	223 (72.40%)		
LVSI					0.017	0.992
Yes	182 (11.82%)	110 (11.90%)	36 (11.69%)	36 (11.69%)		
No	1358 (88.18%)	814 (88.10%)	272 (88.31%)	272 (88.31%)		
ER					0.820	0.664
Positive	1188 (77.14%)	715 (77.38%)	232 (75.32%)	241 (78.25%)		
Negative	352 (22.86%)	209 (22.62%)	76 (24.68%)	67 (21.75%)		

PR					4.283	0.117
Positive	1319 (85.65%)	803 (86.90%)	253 (82.14%)	263 (85.39%)		
Negative	221 (14.35%)	121 (13.10%)	55 (17.86%)	45 (14.61%)		
P53					1.754	0.416
Mutant type	869 (56.43%)	510 (55.19%)	176 (57.14%)	183 (59.42%)		
Wild type	671 (43.57%)	414 (44.81%)	132 (42.86%)	125 (40.58%)		
Ki-67 Positivity Rate (%) ≥38%					0.553	0.759
Yes	533 (34.61%)	317 (34.31%)	104 (33.77%)	112 (36.36%)		
No	1007 (65.39%)	607 (65.69%)	204 (66.23%)	196 (63.64%)		
P16					1.837	0.399
Positive	1176 (76.36%)	697 (75.43%)	235 (76.30%)	244 (79.22%)		
Negative	364 (23.64%)	227 (24.57%)	73 (23.70%)	64 (20.78%)		
NPS	2.00 [1.00, 3.00]	2.00 [1.00, 3.00]	2.00 [1.75, 3.00]	2.00 [2.00, 3.00]	1.741	0.419

Note: BMI, Body Mass Index; FIGO, International Federation of Gynecology and Obstetrics; LVSI, Lymphovascular Space Invasion; ER, Estrogen Receptor; PR, Progesterone Receptor; NPS, Neoadjuvant Prognostic Score.

myometrial invasion depth ≥1/2, presence of LVSI, mutant-type P53, Ki-67 positivity rate ≥38%, tumor lesion diameter ≥2 cm, and higher NPS, suggesting these factors may contribute to increased recurrence risk.

No statistical significance was found for menopausal status (P=0.117), parity (P=0.183), hypertension history (P=0.098), diabetes history (P=0.442), surgical approach (P=0.613), hysterectomy extent (P=0.113), pelvic lymphadenectomy (P=0.154), pathological type (P=0.414), ER (P=0.182), PR (P=0.704), and P16 status (P=0.690) (**Table 3**).

Lasso-Cox regression analysis results for high-risk factors in training cohort

Lasso-Cox regression analysis was performed on 9 variables that showed baseline differences between recurrence and non-recurrence groups in the early EC training cohort. All 9 variables were selected, indicating their predictive value for recurrence risk. Tumor lesion diameter, age, FIGO staging, pathological grading, and BMI had higher regression coefficients, suggesting they significantly contribute to recurrence risk prediction. Myometrial invasion depth, LVSI, Ki-67 positivity rate, and P53 status had relatively lower coefficients. These findings provide essential evidence for constructing a recurrence risk prediction model in early EC (**Figure 1**).

Correlation analysis results for 9 variables in training group

Correlation analysis of the 9 variables (age, BMI, pathological grading, FIGO staging, tumor lesion diameter, myometrial invasion depth, LVSI, P53 status, Ki-67 positivity rate) revealed that most variables had weak correlations (R values ≤0.3), except for FIGO staging and myometrial invasion depth (R=1, P=0). Statistically significant correlations were found between BMI and FIGO staging (R=0.096, P=0.003), BMI and myometrial invasion depth (R=0.096, P=0.003), BMI and LVSI (R=0.067, P=0.041), FIGO staging and P53 status (R=0.067, P=0.043), myometrial invasion depth and P53 status (R=0.067, P=0.043), and LVSI and age (R=0.063, P=0.054), though all R values remained below 0.3. Due to the perfect correlation between FIGO staging and myometrial invasion depth (R=1, P=0), FIGO staging was excluded from subsequent analysis to avoid multicollinearity. Other variable pairs showed weak or non-significant correlations, confirming their suitability for inclusion in further analysis (**Figure 2**).

Cox proportional hazards model analysis and PH assumption testing results for training cohort

In the training cohort (n=924, events =142), we constructed a Cox proportional hazards model using 8 variables: age, BMI, pathological grading, tumor lesion diameter, myometrial invasion depth, LVSI, P53 status, and Ki-67 positivity rate. Significant regression coefficients were found for all variables (P<0.05), with pathological grade G3 (P<0.001), age (P<0.001), myometrial invasion depth (P<0.001), tumor lesion diameter (P=0.008), and Ki-67 positivity rate (P=0.006) contributing substantially to recurrence risk prediction. Hazard ratios (HRs) ranged from 1.475 (P53) to 3.900 (pathologi-

Table 3. Comparison of clinical and pathological characteristics between recurrence and non-recurrence groups in early endometrial cancer training cohort

Variable	Total	Recurrence Group (n=142)	Non-recurrence Group (n=782)	χ^2 Value	*P* Value
Age				26.919	<0.001
≥55 years	526 (56.93%)	109 (76.76%)	417 (53.32%)		
<55 years	398 (43.07%)	33 (23.24%)	365 (46.68%)		
BMI				21.988	<0.001
≥28	299 (32.36%)	70 (49.30%)	229 (29.28%)		
<28	625 (67.64%)	72 (50.70%)	553 (70.72%)		
Menopausal Status				2.451	0.117
Yes	504 (54.55%)	86 (60.56%)	418 (53.45%)		
No	420 (45.45%)	56 (39.44%)	364 (46.55%)		
Parity				1.776	0.183
≥2	786 (85.06%)	126 (88.73%)	660 (84.40%)		
<2	138 (14.94%)	16 (11.27%)	122 (15.60%)		
Hypertension History				2.737	0.098
Yes	162 (17.53%)	18 (12.68%)	144 (18.41%)		
No	762 (82.47%)	124 (87.32%)	638 (81.59%)		
Diabetes History				0.592	0.442
Yes	82 (8.87%)	15 (10.56%)	67 (8.57%)		
No	842 (91.13%)	127 (89.44%)	715 (91.43%)		
Surgical Approach				0.255	0.613
Laparotomy	467 (50.54%)	69 (48.59%)	398 (50.90%)		
Laparoscopy	457 (49.46%)	73 (51.41%)	384 (49.10%)		
Hysterectomy Extent				2.512	0.113
Extrafascial	801 (86.69%)	129 (90.85%)	672 (85.93%)		
Other	123 (13.31%)	13 (9.15%)	110 (14.07%)		
Pelvic LN Removal				2.034	0.154
Yes	767 (83.01%)	112 (78.87%)	655 (83.76%)		
No	157 (16.99%)	30 (21.13%)	127 (16.24%)		
Pathological Grading				31.711	<0.001
G1	286 (30.95%)	25 (17.61%)	261 (33.38%)		
G2	535 (57.90%)	84 (59.15%)	451 (57.67%)		
G3	103 (11.15%)	33 (23.24%)	70 (8.95%)		
Pathological Type				0.668	0.414
EAC	807 (87.34%)	127 (89.44%)	680 (86.96%)		
NEAC	117 (12.66%)	15 (10.56%)	102 (13.04%)		
FIGO Staging				25.858	<0.001
IB	249 (26.95%)	63 (44.37%)	186 (23.79%)		
IA	675 (73.05%)	79 (55.63%)	596 (76.21%)		
Tumor Lesion Diameter				4.806	0.028
≥2 cm	840 (90.91%)	136 (95.77%)	704 (90.03%)		
<2 cm	84 (9.09%)	6 (4.23%)	78 (9.97%)		
Myometrial Invasion Depth				25.858	<0.001
≥1/2	249 (26.95%)	63 (44.37%)	186 (23.79%)		
<1/2	675 (73.05%)	79 (55.63%)	596 (76.21%)		
LVSI				13.606	<0.001
Yes	110 (11.90%)	30 (21.13%)	80 (10.23%)		
No	814 (88.10%)	112 (78.87%)	702 (89.77%)		

ER				1.780	0.182
Positive	715 (77.38%)	116 (81.69%)	599 (76.60%)		
Negative	209 (22.62%)	26 (18.31%)	183 (23.40%)		
PR				0.144	0.704
Positive	803 (86.90%)	122 (85.92%)	681 (87.08%)		
Negative	121 (13.10%)	20 (14.08%)	101 (12.92%)		
P53				9.298	0.002
Mutant type	510 (55.19%)	95 (66.90%)	415 (53.07%)		
Wild type	414 (44.81%)	47 (33.10%)	367 (46.93%)		
Ki-67 Positivity Rate (%) ≥38%				4.701	0.030
Yes	317 (34.31%)	60 (42.25%)	257 (32.86%)		
No	607 (65.69%)	82 (57.75%)	525 (67.14%)		
P16				0.160	0.690
Positive	697 (75.43%)	109 (76.76%)	588 (75.19%)		
Negative	227 (24.57%)	33 (23.24%)	194 (24.81%)		
NPS	2.00 [1.00, 3.00]	2.00 [2.00, 3.00]	2.00 [1.00, 3.00]	7.281	<0.001

Note: BMI, Body Mass Index; FIGO, International Federation of Gynecology and Obstetrics; LVSI, Lymphovascular Space Invasion; ER, Estrogen Receptor; PR, Progesterone Receptor; NPS, Neoadjuvant Prognostic Score.

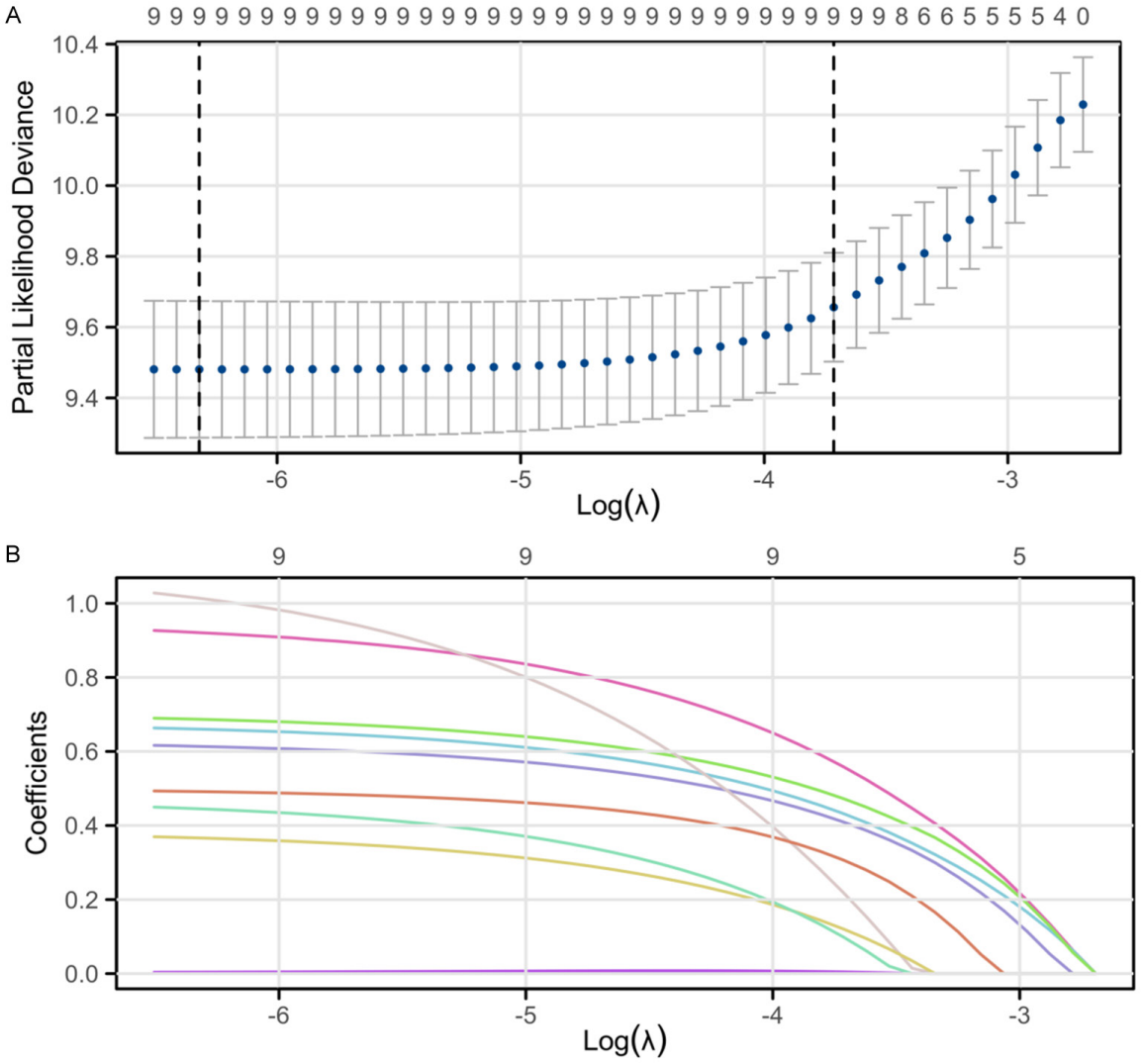

Figure 1. Lasso-Cox regression coefficients for recurrence-related variables in early endometrial cancer training group. A. Bar chart of Lasso-Cox regression coefficients for each variable, displaying the relative contribution of 9 variables to recurrence risk. B. Variable selection path plot, showing the trajectory of each variable coefficient as the regularization parameter changes in Lasso regression. Note: BMI, Body Mass Index; FIGO, International Federation of Gynecology and Obstetrics; LVSI, Lymphovascular Space Invasion.

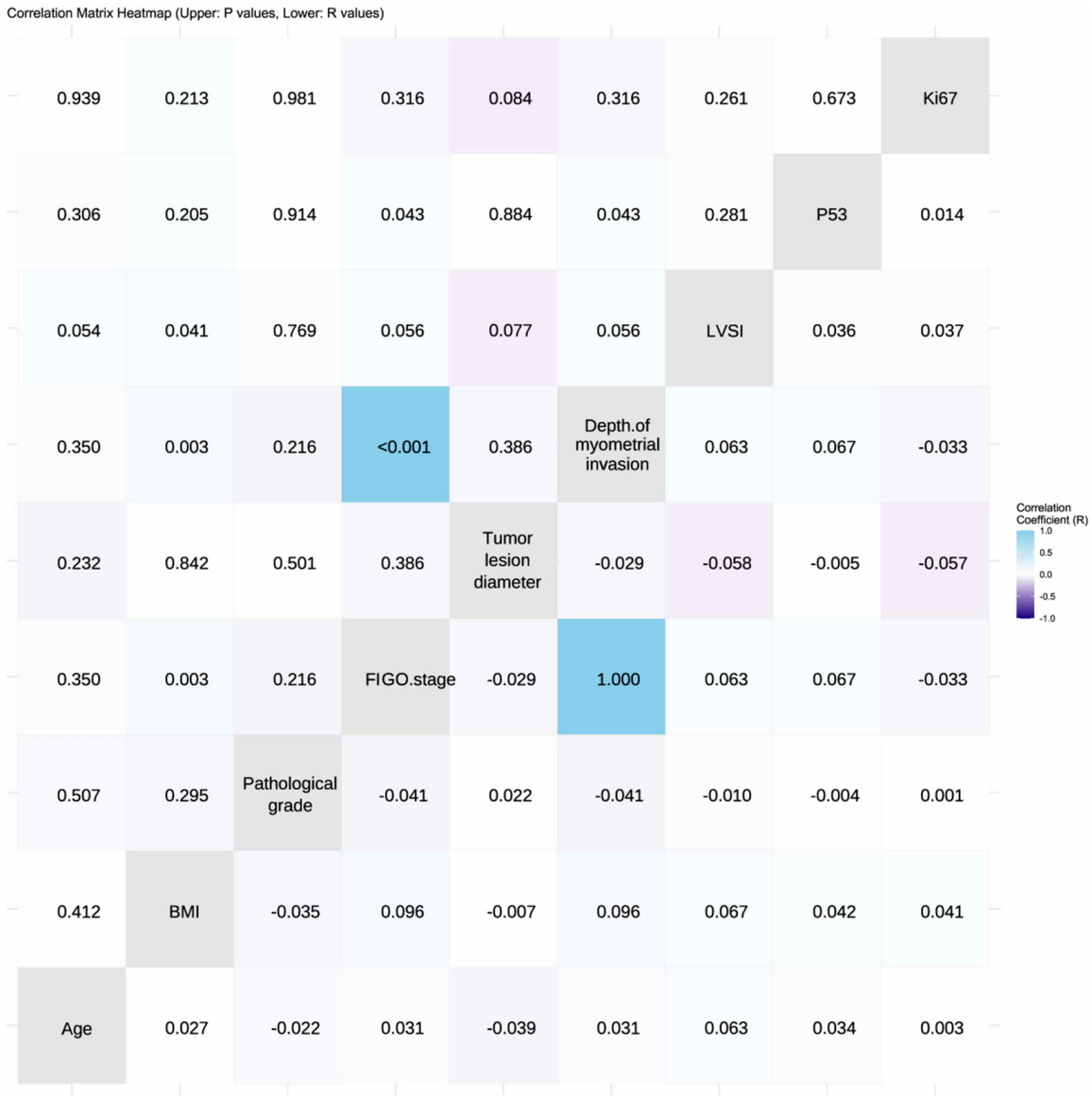

Figure 2. Correlation heatmap of 9 variables in early endometrial cancer training group. Note: BMI, Body Mass Index; FIGO, International Federation of Gynecology and Obstetrics; LVSI, Lymphovascular Space Invasion.

cal grade G3), with the model showing a concordance index of 0.748 (P<2e-16), indicating good discriminative ability.

Schoenfeld residual testing showed all *P* values exceeded 0.05 (age P=0.87, BMI P=0.29, pathological grading P=0.18, tumor lesion diameter P=0.48, myometrial invasion depth P=0.87, LVSI P=0.85, P53 P=0.47, Ki-67 P=0.75), with a global test P=0.75, supporting that the model satisfies proportional hazards assumptions (**Figure 3**).

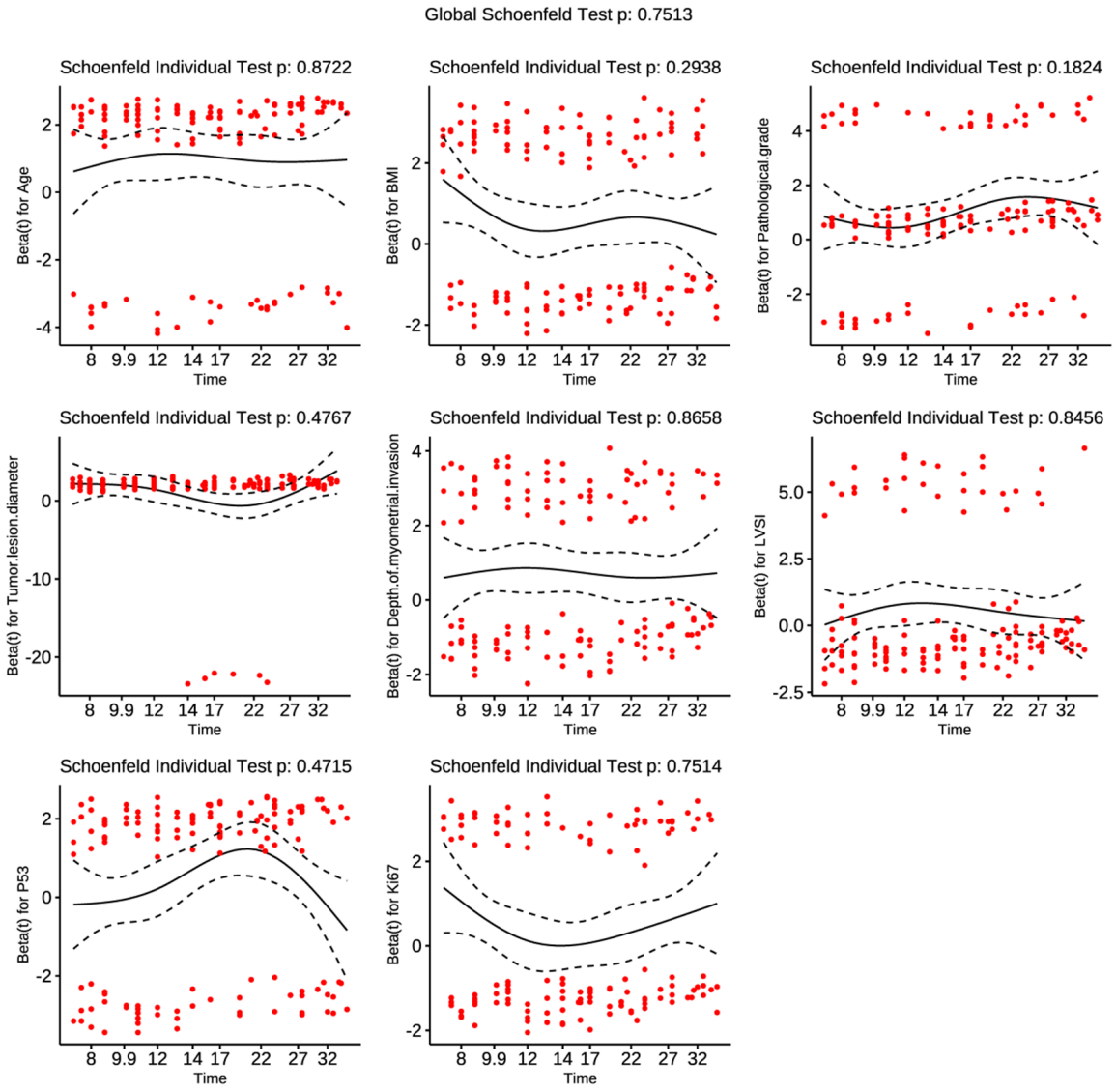

Figure 3. Schoenfeld residual plot for proportional hazards assumption testing of Cox model in early endometrial cancer training group. Note: BMI, Body Mass Index; LVSI, Lymphovascular Space Invasion.

Recurrence survival curve analysis for 8 variables in training cohort

Kaplan-Meier survival curves were constructed for 8 variables (age, BMI, pathological grading, tumor lesion diameter, myometrial invasion depth, LVSI, P53 status, Ki-67 positivity rate) in the training cohort. Statistically significant differences in recurrence survival curves were observed for age (P<0.001), BMI (P<0.001), pathological grading (P<0.001), tumor lesion diameter (P=0.030), myometrial invasion depth (P<0.001), LVSI (P<0.001), P53 status (P=0.002), and Ki-67 positivity rate (P=0.0027) (**Figure 4**).

Univariate and multivariable Cox regression analysis results for 8 variables in training cohort

Univariate Cox regression revealed significant associations with recurrence risk for age ≥55 years (P<0.001, HR=2.683), BMI ≥28 (P<0.001, HR=2.205), pathological grade G2 (P=0.007, HR=1.851), G3 (P<0.001, HR=4.134), tumor lesion diameter ≥2 cm (P=0.036, HR=2.4), myometrial invasion depth ≥1/2 (P<0.001, HR=2.356), LVSI presence (P<0.001, HR=2.175), mutant-type P53 (P=0.003, HR=1.705), and Ki-67 positivity rate ≥38% (P=0.028, HR=1.451).

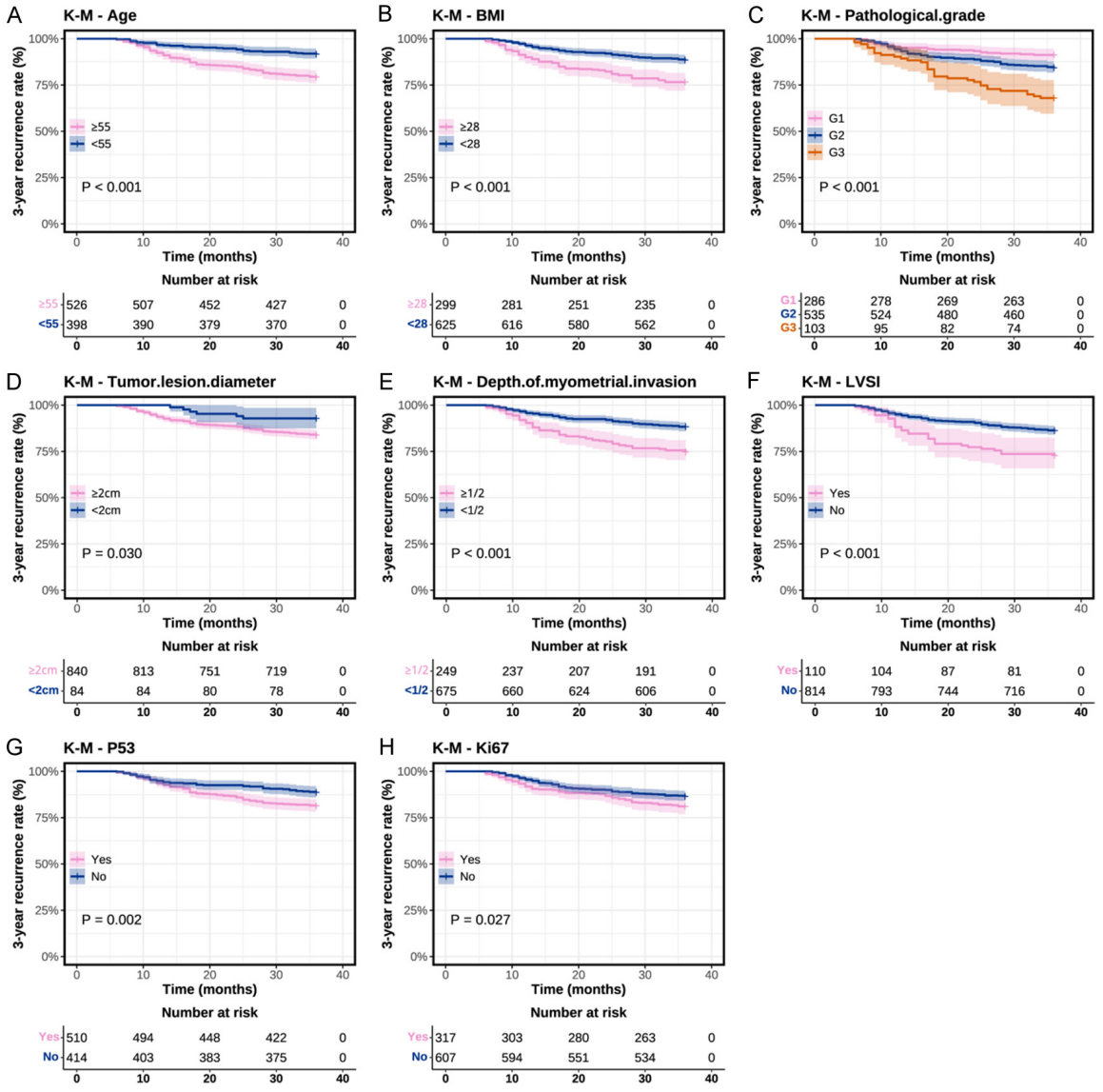

Figure 4. Kaplan-Meier recurrence survival curves for 8 variables in early endometrial cancer training group. A. Recurrence curves for patients of different ages. B. Recurrence curves for patients with different BMI. C. Recurrence curves for patients with different pathological grades. D. Recurrence curves for patients with different tumor lesion diameters. E. Recurrence curves for patients with different myometrial invasion depths. F. Recurrence curves for patients with different LVSI status. G. Recurrence curves for patients with different P53 status. H. Recurrence curves for patients with different Ki-67 positivity rates. Note: BMI, Body Mass Index; LVSI, Lymphovascular Space Invasion; P53, Tumor Protein 53; Ki-67, Ki-67 Antigen.

Multivariable analysis confirmed the independent predictive roles of these variables: age ≥55 years (P<0.001, HR=2.604), BMI ≥28 (P<0.001, HR=1.882), pathological grade G2 (P=0.004, HR=1.928), G3 (P<0.001, HR=3.9), tumor lesion diameter ≥2 cm (P=0.008, HR=3.036), myometrial invasion depth ≥1/2 (P<0.001, HR=2.038), LVSI (P=0.017, HR=1.661), mutant-type P53 (P=0.032, HR=1.475), and Ki-67 positivity rate ≥38% (P=0.006, HR=1.611) (**Figure 5**).

Nomogram prediction model for early endometrial cancer recurrence risk

Based on the training cohort data, we constructed a nomogram for predicting early endometrial cancer recurrence risk, incorporating 8 variables: age, body mass index (BMI), pathological grading, tumor lesion diameter, myometrial invasion depth, LVSI, P53 status, and Ki-67 positivity rate. The nomogram calculates a total score by assigning points to different variable

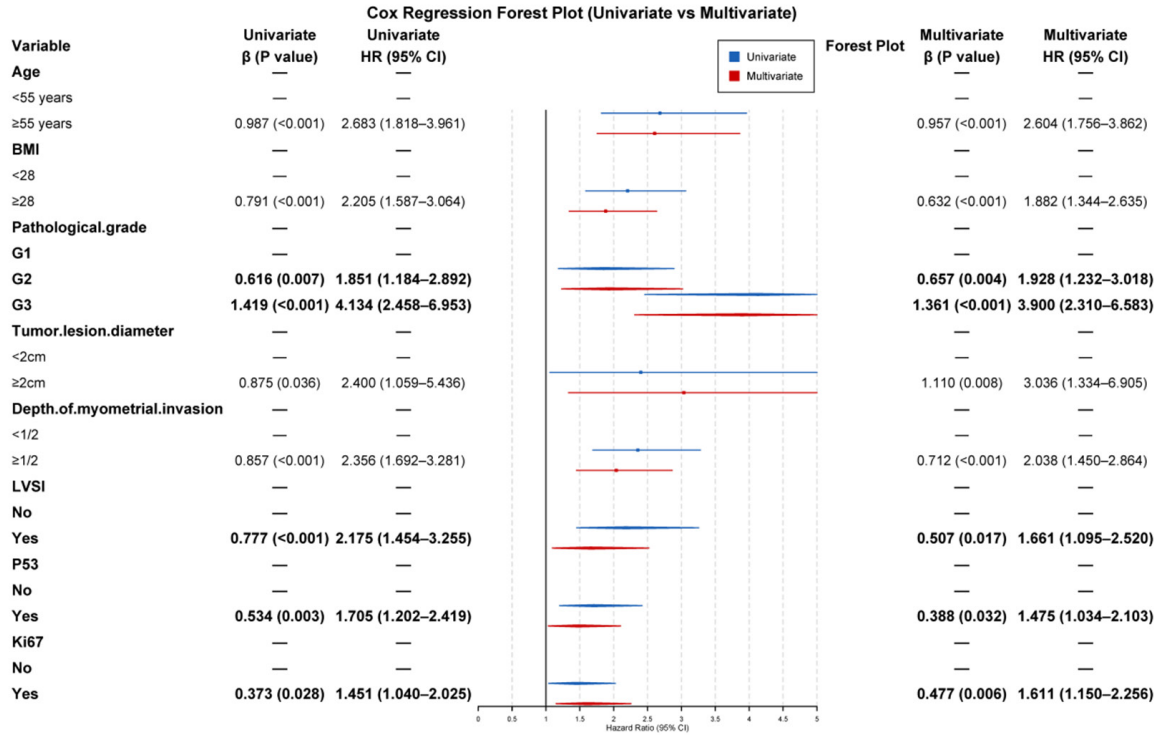

Figure 5. Forest plot of univariate and multivariable Cox regression analysis for 8 variables in early endometrial cancer training group. Note: BMI, Body Mass Index; LVSI, Lymphovascular Space Invasion; P53, Tumor Protein 53; Ki-67, Ki-67 Antigen.

levels, predicting patients' 24-month and 36-month recurrence-free survival probabilities.

The results indicate that certain variables achieve higher scores at specific thresholds (e.g., age ≥55 years, BMI ≥28 kg/m^2, pathological grade G3, tumor diameter ≥2 cm, myometrial invasion depth ≥1/2, presence of LVSI, mutant-type P53, Ki-67 positivity rate ≥38%). Higher total scores correlate with increased recurrence risk, accompanied by progressively decreasing 24-month and 36-month survival probabilities. This nomogram provides an individualized recurrence risk assessment through an intuitive scoring system, highlighting its significant potential for clinical application (**Figure 6**).

Validation and evaluation of nomogram model in early endometrial cancer training cohort

We validated the nomogram model using the training cohort. **Figure 7A** shows the 3-year recurrence risk ROC curve with an AUC of 0.767, indicating strong discriminative ability. **Figure 7B** presents the calibration curve, with a C-index of 0.748 (95% CI: 0.728-0.768). The Le Cessie-van Houwelingen goodness-of-fit test yielded P=0.445, indicating no evidence of poor fit. Bootstrap-corrected calibration revealed a mean absolute error of 0.006, confirming excellent agreement between predicted and observed probabilities. **Figure 7C** displays the DCA, with the model showing net benefit across threshold probability ranges of 0-79%, achieving a maximum net benefit rate of 14.82%, demonstrating the model's practical value in clinical decision-making.

Validation and evaluation of nomogram model in early endometrial cancer validation cohort

The nomogram model was further validated in the validation cohort. **Figure 8A** shows the 3-year recurrence risk ROC curve with an AUC of 0.701, indicating reasonable discriminative ability. **Figure 8B** presents the calibration curve with a C-index of 0.684 (95% CI: 0.645-0.723). The Le Cessie-van Houwelingen goodness-of-fit test yielded P=0.015, suggesting mild miscalibration. However, the bootstrap-corrected calibration slope was close to 1.04 and the mean absolute error was 0.021, indi-

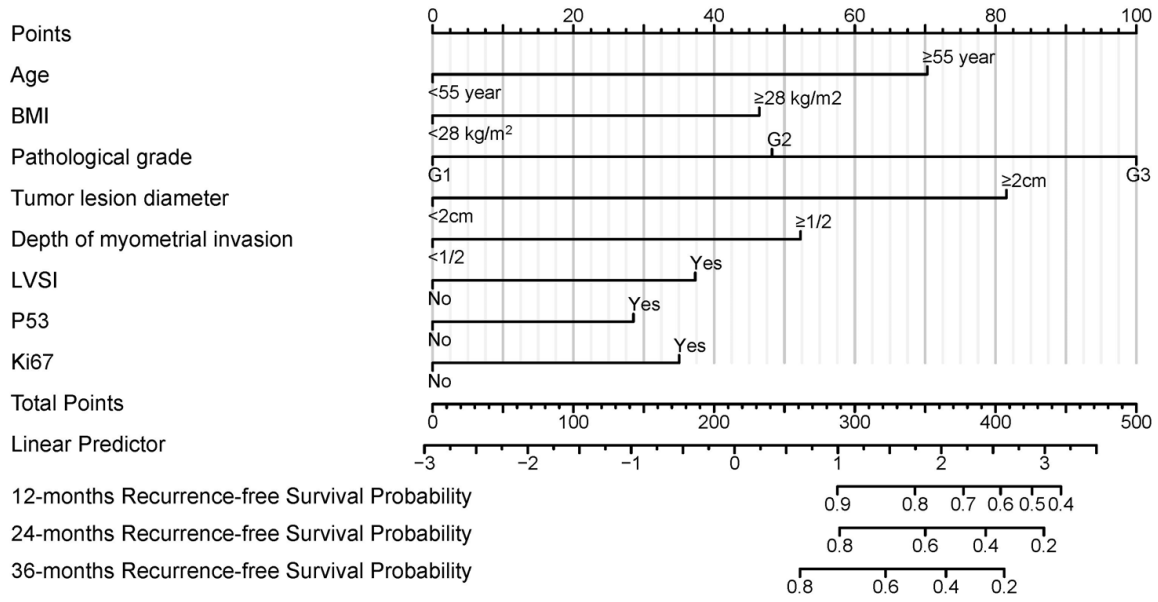

Figure 6. Nomogram prediction model for recurrence risk in early endometrial cancer training group. Note: BMI, Body Mass Index; LVSI, Lymphovascular Space Invasion; P53, Tumor Protein 53; Ki-67, Ki-67 Antigen. In the nomogram, each variable is assigned a score (top scale). The total score corresponds to the predicted probability of recurrence-free survival (RFS) at 12, 24, and 36 months, shown in the bottom three scales.

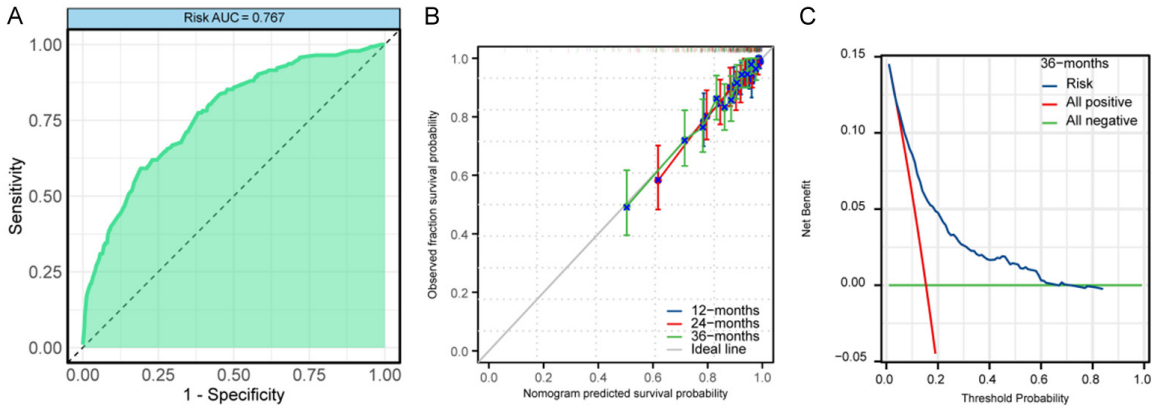

Figure 7. Validation and evaluation charts of nomogram model in early endometrial cancer training group. A. ROC curve for 3-year recurrence risk, showing the AUC value of model discriminative ability. B. Calibration curve, displaying the model's C-index and global test results. C. DCA showing the net benefit of the model at different thresholds. Note: ROC, Receiver Operating Characteristic; AUC, Area Under the Curve; C-index, Concordance Index; DCA, Decision Curve Analysis.

cating acceptable agreement between predicted and observed values. **Figure 8C** shows the DCA, with the model demonstrating net benefit across threshold probability ranges of 0-50%, achieving a maximum net benefit rate of 16.45%, highlighting its practical clinical utility.

Validation and evaluation of nomogram model in early endometrial cancer testing cohort

Validation in the testing cohort showed similar results. **Figure 9A** illustrates the 3-year recurrence risk ROC curve with an AUC of 0.694, indicating reasonable discriminative ability. **Figure 9B** presents the calibration curve with a C-index of 0.677 (95% CI: 0.638-0.717). The Le Cessie-van Houwelingen goodness-of-fit test resulted in P=0.021, indicating some degree of miscalibration. Nevertheless, the bootstrap-corrected calibration slope remained near 1.05, with a mean absolute error of 0.036, supporting adequate agreement between predicted and observed values. **Figure 9C** displays

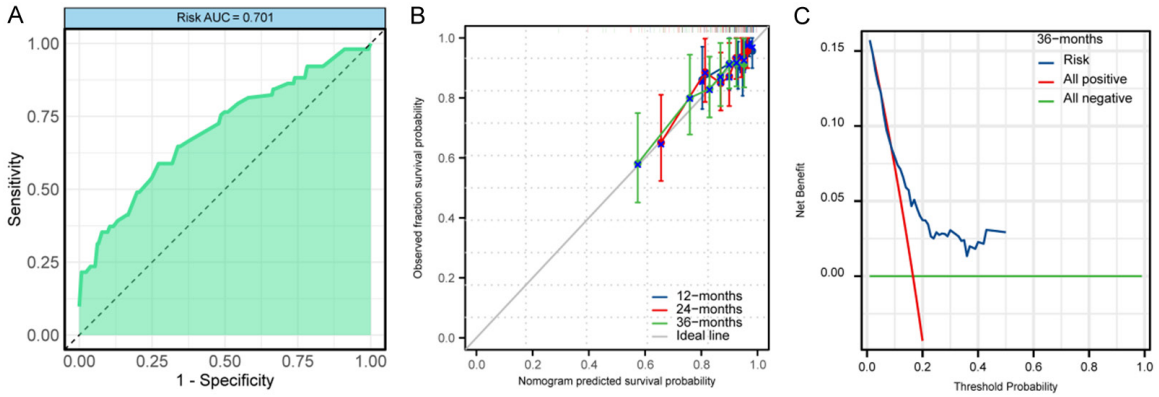

Figure 8. Validation and evaluation charts of nomogram model in early endometrial cancer validation group. A. ROC curve for 3-year recurrence risk, showing the AUC value of model discriminative ability. B. Calibration curve, displaying the model's C-index and global test results. C. DCA, showing the net benefit of the model at different thresholds. Note: ROC, Receiver Operating Characteristic; AUC, Area Under the Curve; C-index, Concordance Index; DCA, Decision Curve Analysis.

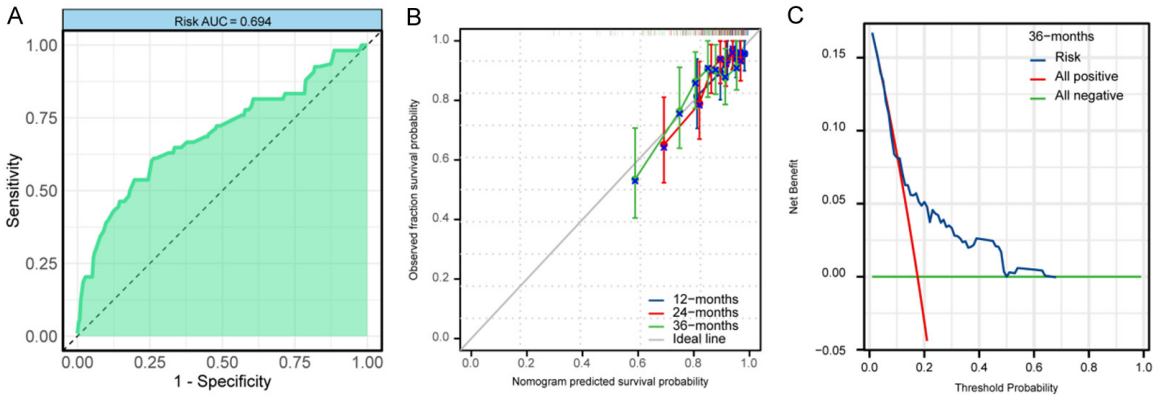

Figure 9. Validation and evaluation charts of nomogram model in early endometrial cancer testing group. A. ROC curve for 3-year recurrence risk, showing the AUC value of model discriminative ability. B. Calibration curve, displaying the model's C-index and global test results. C. DCA, showing the net benefit of the model at different thresholds. Note: ROC, Receiver Operating Characteristic; AUC, Area Under the Curve; C-index, Concordance Index; DCA, Decision Curve Analysis.

DCA, showing the model's net benefit across threshold probability ranges of 0-48%, achieving a maximum net benefit rate of 17.18%, suggesting strong practical value in clinical decision-making.

ROC curve comparison between nomogram model and NPS in training, validation, and testing cohorts

We compared the performance of our nomogram model to the NPS for predicting 3-year recurrence risk across the three cohorts. In **Figure 10A**, the training cohort showed an AUC of 0.767 for our model versus 0.687 for NPS (DeLong test P=0.009), demonstrating significant superiority of the nomogram. In **Figure 10B**, the validation cohort showed an AUC of 0.701 for our model versus 0.580 for NPS (DeLong test P=0.041), indicating superior performance of the nomogram. In **Figure 10C**, the testing cohort revealed an AUC of 0.694 for the nomogram compared to 0.600 for NPS (DeLong test P=0.109), suggesting no significant difference between the two models in predictive capability.

NRI analysis of the new risk prediction model compared to traditional NPS

We evaluated the predictive performance improvement of our new risk prediction model

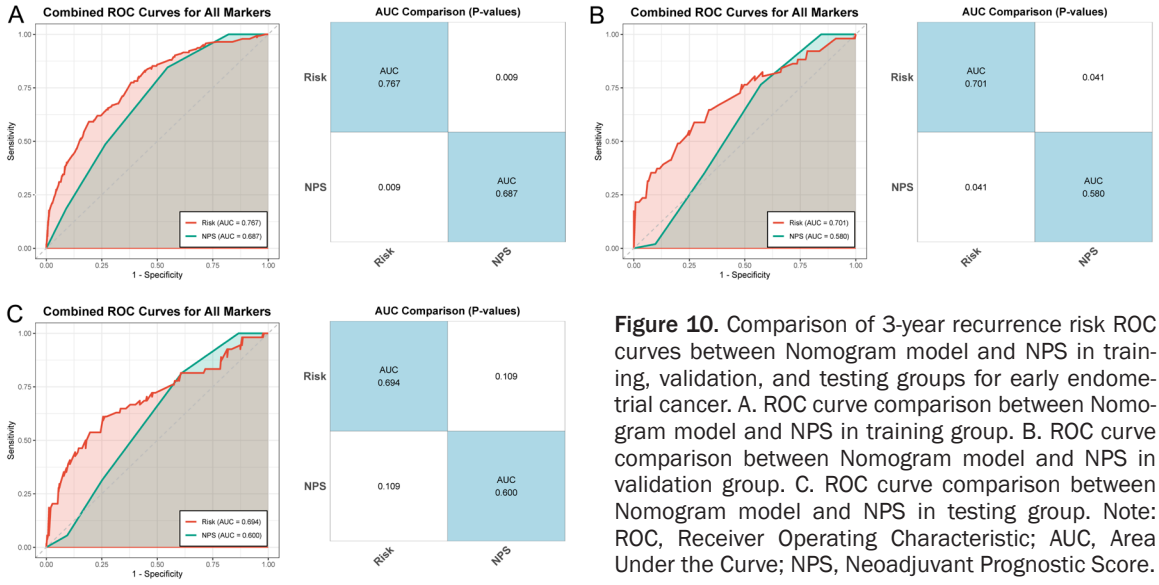

Figure 10. Comparison of 3-year recurrence risk ROC curves between Nomogram model and NPS in training, validation, and testing groups for early endometrial cancer. A. ROC curve comparison between Nomogram model and NPS in training group. B. ROC curve comparison between Nomogram model and NPS in validation group. C. ROC curve comparison between Nomogram model and NPS in testing group. Note: ROC, Receiver Operating Characteristic; AUC, Area Under the Curve; NPS, Neoadjuvant Prognostic Score.

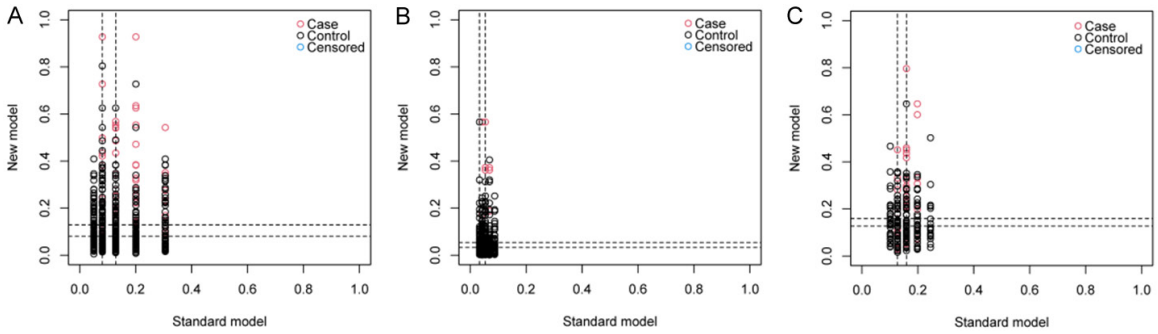

Figure 11. Net reclassification improvement comparison between new risk prediction model and traditional NPS scoring system. A. Training Group NRI Analysis Results: Shows the reclassification performance of the new risk prediction model relative to the NPS scoring system in the training cohort. The forest plot displays point estimates and 95% confidence intervals for overall NRI and its components (NRI+ and NRI-). B. Validation Group NRI Analysis Results: Demonstrates the reclassification performance of the new model in the validation cohort. Despite negative event reclassification improvement, the control reclassification improvement was significantly positive, resulting in good overall NRI performance. C. Testing Group NRI Analysis Results: Presents reclassification results in the independent testing cohort, confirming the stable performance of the new model across different populations. Note: NRI, Net Reclassification ImprovementNRI; NPS, Neuropathological Score; C-index, Concordance Index; CI, Confidence Interval.

relative to the traditional NPS scoring system using NRI analysis. In **Figure 11A**, the training cohort (n=924, event rate 15.4%) showed that the new model achieved a C-index of 0.748, significantly higher than the NPS C-index of 0.665 (improvement: 0.083, improvement rate: 12.5%). The overall NRI was 0.315 (95% CI: -0.001 to 0.432), with NRI+ of 0.172 (95% CI: -0.166 to 0.273) and NRI- of 0.143 (95% CI: -0.094 to 0.360).

In **Figure 11B**, the validation cohort (n=308, event rate 16.6%) demonstrated the new model's strong discriminative ability, with a C-index of 0.684, compared to 0.564 for NPS (improvement: 0.120, improvement rate: 21.3%). The overall NRI was 0.365 (95% CI: -0.055 to 0.671), with NRI+ of 0.102 (95% CI: -0.354 to 0.462) and NRI- of 0.263 (95% CI: -0.022 to 0.617).

In **Figure 11C**, the testing cohort (n=308, event rate 17.5%) showed a C-index of 0.677 for the new model, superior to the NPS C-index of 0.580 (improvement: 0.098, improvement rate: 16.9%). The overall NRI was 0.170 (95% CI:

-0.016 to 0.710), with NRI+ of -0.204 (95% CI: -0.283 to 0.556) and NRI- of 0.375 (95% CI: -0.091 to 0.543).

Discussion

Early-stage EC (FIGO stage I) generally has a favorable prognosis, but 10%-20% of patients still experience recurrence, which diminishes quality of life, increases treatment complexity, and raises medical costs [15]. A nationwide cohort study by Jeppesen et al. [16] reported a 3-year recurrence rate of 7% in early-stage EC patients, which is similar to our observed recurrence rate. However, their study included stage II patients, which contributed to a higher overall recurrence risk. Traditional recurrence risk assessments rely on factors such as age, BMI, pathological grading, FIGO staging, myometrial invasion depth, and LVSI. However, these individual factors often fail to comprehensively reflect risk [17].

Using Lasso-Cox regression, our study identified 8 key predictive factors: age, BMI, pathological grading, tumor lesion diameter, myometrial invasion depth, LVSI, P53 status, and Ki-67 positivity rate. These factors are consistent with findings from previous studies, though predictive efficacy may vary. Çakır et al. [18] specifically studied stage 1A grade 1-2 patients and found that a P53 index cutoff of 17.5% yielded a 5-year recurrence-free survival rate of 94.6% for low P53 groups versus 65.4% for high P53 groups, demonstrating strong predictive ability. Comparatively, our study, which included both IA and IB stage patients, showed significant but relatively moderate predictive efficacy for P53. This difference may reflect the heterogeneity of P53 expression across different stages. Biologically, mutated P53, known as the "guardian of the genome", leads to checkpoint loss and DNA damage repair defects, which promote tumor progression and metastasis, particularly in low-risk early-stage patients.

Myometrial invasion depth was consistently identified as a key predictive factor Nwachukwu et al. [7] found that any degree of myometrial invasion significantly increased recurrence risk in stage IA grade 1 EC. Han et al. [19] highlighted the importance of stage differences, noting that myometrial invasion is an independent prognostic factor in stage IA, while histological grading becomes more significant in stage IB. This aligns with our results and may explain why our predictive efficacy is slightly lower than that of Kong et al. [20], who focused on stage IA patients and achieved a C-index of 0.862, compared to our model's C-index of 0.677-0.748. The higher myometrial invasion in stage IB patients with tumors ≥38% contributes to increased biological invasiveness, making prediction more complex. From a biological standpoint, myometrial invasion reflects tumor aggressiveness and angiogenesis, with deeper invasion providing more metastatic pathways for tumor cells.

Hormone receptor status was validated as a predictive factor in our study. Li et al. [21] established optimal positive thresholds for ER and PR at 12% and 8%, respectively, showing that low expression groups had significantly decreased recurrence-free survival. While we did not define specific thresholds, our study found that hormone receptor-negative patients had a markedly higher recurrence risk, consistent with the hormone-dependent nature of EC. Notably, ER status was not included in our final model, suggesting that molecular markers like P53 and Ki-67 may better capture overlapping biological pathways than hormonal status in early-stage disease. ER and PR negativity often indicate high tumor dedifferentiation, reduced hormone sensitivity, and enhanced proliferative and invasive potential.

LVSI showed strong predictive value in our study. Altın et al. [22] found that even LVSI-negative early endometrioid cancer patients had a 4.5% recurrence rate, indicating that grading remains an independent predictor of recurrence in traditionally low-risk populations. Interestingly, our model placed more weight on tumor diameter ≥2 cm than LVSI, challenging traditional paradigms that prioritize vascular invasion. The greater predictive value of tumor size likely reflects sustained proliferative capacity and the increased likelihood of aggressive subclones, with the 2 cm threshold indicating a critical biological transition in early-stage tumors. LVSI presence provides pathways for lymphatic and hematogenous metastasis, crucial in tumor dissemination.

Molecular marker integration further enhanced prediction accuracy. Buchynska et al. [23] found that elevated KRAS, ATR, and CHEK1

expression were closely associated with recurrence, involved in cell proliferation and DNA damage repair. While our study did not include these markers, Ki-67, a proliferation marker, served a similar role. Ki-67 demonstrated robust predictive value, while P53 showed the least effect among selected variables, suggesting that proliferative markers may reflect the cumulative effect of multiple molecular alterations, including P53 pathway disruption. High Ki-67 expression correlates with tumor invasiveness and metastatic potential [24, 25].

Our nomogram model, incorporating 8 variables, performed well across training, validation, and testing cohorts, with C-index values of 0.748, 0.684, and 0.677, respectively, and AUC values of 0.767, 0.701, and 0.694, demonstrating good discriminative ability. In contrast, Zheng et al. [26] reported a higher C-index in their multi-institutional study on early-onset EC, though their cohort had a narrower age range (mostly 45-49 years), resulting in higher patient homogeneity. Age, as a predictive factor, may reflect immune senescence, reduced DNA repair capacity, and changes in the tumor microenvironment.

Serum markers also showed predictive value. Erturk et al. [27] identified preoperative CA-125 elevation as an independent predictor of early recurrence (OR: 3.43), and Liu et al. [28] confirmed that combining ctDNA and CA-125 enhances short-term recurrence prediction. Elevated CA-125 may reflect tumor burden, increased vascular permeability, or tumor-associated inflammatory responses.

Calibration curves indicated high consistency between predicted and actual risks, with DCA confirming significant net clinical benefit in clinical decision-making, achieving maximum benefit rates of 14.82%, 16.45%, and 17.18%. Compared to traditional NPS scoring systems, our model outperformed, with NRI analysis showing overall improvement rates of 12.5%-21.3%. Dou et al. [13] emphasized that early recurrence patients have significantly worse prognoses, with 75% of recurrences occurring within 36 months, highlighting the importance of early identification of high-risk patients. Our model predicts 24-month and 36-month recurrence-free survival probabilities through a scoring system, accurately quantifying individual risk and guiding personalized treatment and follow-up.

Different recurrence patterns may require tailored prediction strategies. Shin et al. [29] found local recurrence mainly associated with high-grade disease, while distant metastasis was linked to non-endometrioid histology and parametrial invasion. Huang et al. [12] confirmed that FIGO staging, LVSI, ER negativity, and P53 abnormal expression were independent risk factors for early recurrence, consistent with our findings.

Our model, while showing slightly lower prediction precision compared to studies targeting specific stages, offers broader clinical applicability. Kong et al. [20] achieved a higher C-index in stage IA patients (C-index =0.862), whereas our model, which applies to both IA and IB stage patients, better meets clinical needs. This trade-off between precision and applicability highlights the practical considerations in model development. Including more heterogeneous patient populations increases prediction complexity but provides more realistic clinical scenarios.

This study has limitations. The retrospective design and single-center data may introduce selection bias. Differences in immunohistochemistry techniques could affect the generalizability of P53 and Ki-67 interpretations. Although we identified 8 robust predictors, we did not perform subgroup validation, which may limit statistical power in some small cohorts. The lack of emerging markers such as MMR status or POLE mutations and the 3-year follow-up duration may further limit predictive capability. Future research should focus on multi-center validation, integrating genomic and imaging biomarkers, developing dynamic nomogram models, and exploring deep learning methods to optimize prediction performance and clinical applicability.

Conclusion

This study successfully constructed and validated a Cox regression-based nomogram model for early EC recurrence risk, integrating 8 key variables: age, BMI, pathological grading, tumor lesion diameter, myometrial invasion depth, LVSI, P53 status, and Ki-67 positivity

rate. The model demonstrated excellent discriminative ability, calibration, and clinical net benefit across training, validation, and testing cohorts, outperforming traditional NPS scoring systems. This model provides a precise, individualized recurrence risk assessment tool for early-stage EC patients, optimizing treatment decisions and follow-up strategies with significant clinical application potential.

Disclosure of conflict of interest

None.

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