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Establishment and validation of a prognostic nomogram for overall survival in type II endometrial carcinoma patients

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This study aimed to develop and validate a nomogram model to predict overall survival (OS) in patients with type II endometrial carcinoma (EC). Data from patients with confirmed type II EC enrolled between 2010 and 2018 were extracted from the Surveillance, Epidemiology, and End Results (SEER) database. Patients were randomly allocated to training and validation groups in a 7:3 ratio. Univariable and multivariable analyses were performed to identify independent prognostic risk factors, which were included in constructing the nomogram model. The concordance index (C-index), receiver operating characteristic (ROC) curve, calibration curve, and decision curve analysis (DCA) were used to assess the prediction accuracy and clinical utility of the nomogram model. The effects of different variables on survival probability were analyzed using the Kaplan-Meier method. A total of 3,933 eligible patients with type II EC were identified and included in this study. Independent risk factors for type II EC were found to be race, tumor size, histology, grade, T stage, N stage, M stage, examination of para-aortic lymph nodes, examination of pelvic lymph nodes, surgery, lung metastasis, radiation therapy, and chemotherapy. A prognostic nomogram was constructed based on these variables. The C-index for the training cohort was 0.791 (95% CI 0.780–0.802) and for the validation cohort was 0.798 (95% CI 0.778–0.818). The ROC curve demonstrated good prediction accuracy. The calibration curve indicated strong agreement between predicted and actual values. The DCA showed that the nomogram model has significant clinical utility and potential. This study developed a survival prediction model for patients with type II EC to assist clinicians in evaluating prognostic factors, predicting OS, and determining appropriate treatment protocols to improve patient outcomes.

Keywords Type II endometrial carcinoma, Nomogram, SEER database, Prognostic factor, Overall survival

Endometrial carcinoma (EC) is the most common gynecological cancer in high-income countries, and its incidence is increasing globally[1]. Among all cancer cases in women in the United States, EC is the fourth most common in terms of incidence and the sixth most common in terms of mortality[2]. Previous studies have found that EC is associated with several risk factors, including advanced age, obesity, hormone replacement therapy, and genetic predisposition[3]. In 1983, Bockman proposed that EC be divided into two pathological types[4]:Type I, endometrioid adenocarcinoma, which is associated with long-term estrogen stimulation; and Type II, non-endometrioid carcinoma. The pathological types of Type II primarily include serous carcinoma (SC), clear cell carcinoma (CCC), mixed carcinoma (MC), and carcinosarcoma (CS). Its occurrence is generally unrelated to estrogen stimulation. Despite accounting for only 1–10% of all EC, Type II EC exhibits aggressive characteristics—including deep myometrial invasion, metastasis, and recurrence—that lead to a disproportionately high mortality rate[5]. Even at early stages, patients with Type II EC typically experience worse prognoses than those with the more common EC. However, because Type II EC is rare, previous studies have been limited by small sample sizes[6,7], hindering comprehensive analysis and leaving its prognostic factors largely undefined. Therefore, a comprehensive understanding of type II EC is essential for its prevention, treatment, and prognosis.

The Surveillance, Epidemiology, and End Results (SEER) database is a large public resource in the field of cancer. Since 1973, it has regularly collected extensive information and data on millions of malignant tumor

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patients across the United States. The database includes social information, clinically relevant indicators, tumor-related conditions, and treatment methods, providing researchers with comprehensive and free access to valuable data[8]. In this study, we assessed type II EC patients registered in the SEER database between 2010 and 2018, aiming to develop and validate prognostic nomograms for overall survival (OS) to help clinicians make better treatment decisions.

Methods

The SEER∗Stat software (version 8.4.3; National Cancer Institute, USA) was used to extract clinical information of patients diagnosed with EC between 2010 and 2018 from the SEER database. The inclusion criteria for this study were: (a) the primary site of the tumor was the uterus (Primary Site-Labeled: C54.0-C54.9, C55.9) and the first primary cancer was EC; (b) for type II EC, pathological tissue types were classified according to the ICD-O-3 code: SC (8441, 8460, 8461), CCC (8310), MC (8323), and CS (8950, 8951, 8980, 8981); and (c) known cause of death and survival duration after diagnosis. The exclusion criteria were: (a) incomplete or unclear clinical data (e.g., marital status, ethnic information, tumor grade, tumor size, T stage, N stage, and survival time); and (b) survival time recorded as zero.

The variables in the SEER database included age, race, marital status, tumor size, grade, T stage, N stage, M stage, metastatic site, surgery, radiotherapy, chemotherapy, cause of death, survival time, and survival status. In this study, OS was defined as the time from surgery to death from any cause. Ultimately, our study included 3,933 eligible patients with type II EC, as shown in Fig. 1. We referred to prior literature[9,10] and employed X-tile software (Yale University, New Haven, Connecticut, USA) to determine the optimal cutoff values for tumor size at 46 mm and 66 mm.

Statistical analyses were performed using SPSS (v22.0, IBM) and R (v3.6.1). To ensure that participants were randomly assigned to the training and validation groups, we used the "comparegroups" package in R to allocate

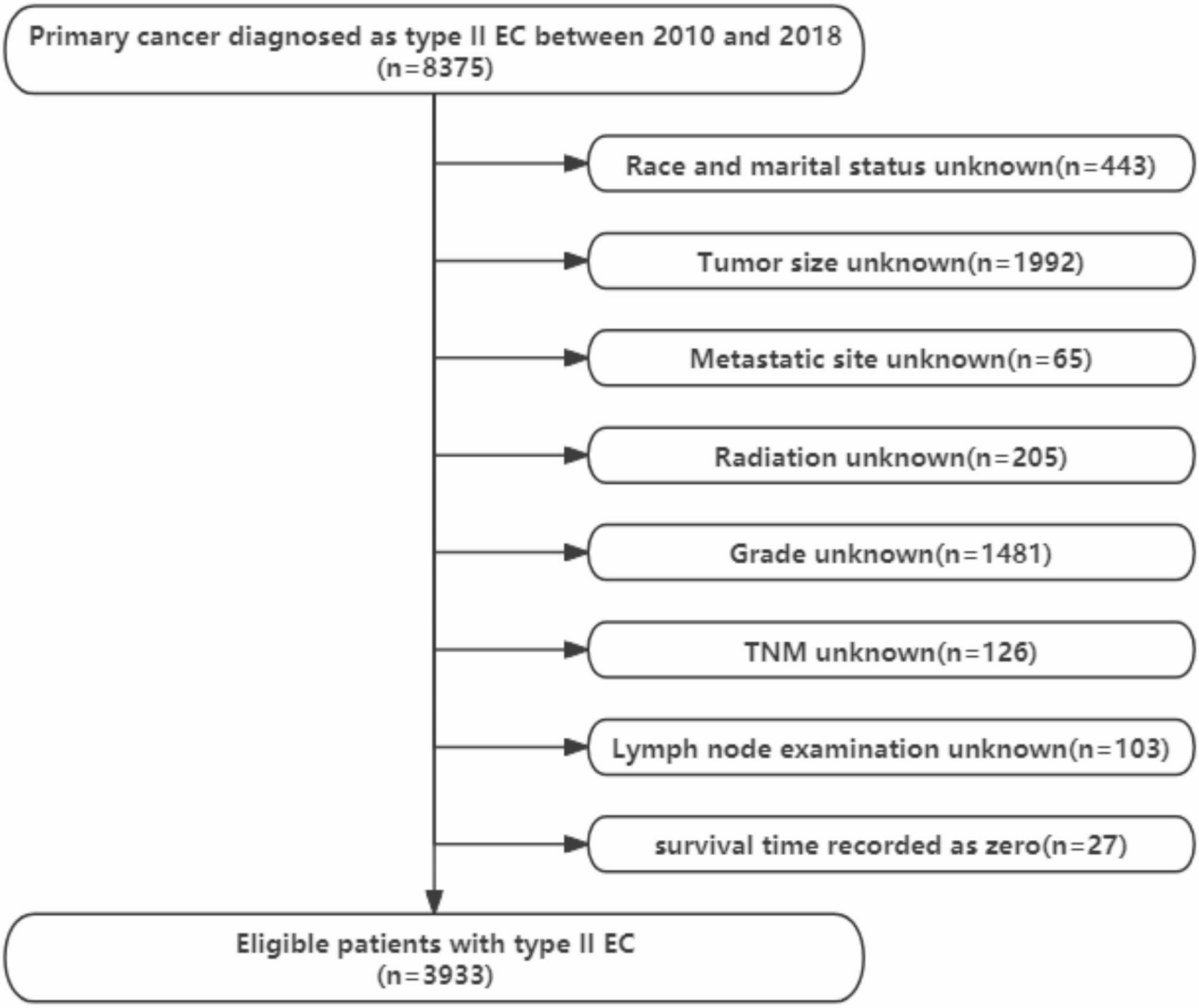

Fig. 1. The data filtering flowchart.

patients in a 7:3 ratio. Patients were randomly allocated into a training cohort (70%, $n = 2,753$) and validation cohort (30%, $n = 1,180$) for model development and testing. A statistical description of the baseline clinical characteristics of the patients was performed, and the differences between the training and validation cohorts were compared using the chi-square test. To identify independent prognostic factors for patients with type II EC, univariable Cox regression analysis was performed using the "survival" package in R on data extracted from the SEER database. Variables with a P -value of less than 0.05 in the univariable analysis were subsequently included in the multivariable Cox regression analysis. Based on the results of the multivariable analysis, significant variables were identified as independent prognostic factors. The independent prognostic factors identified through multivariable Cox regression analysis were used to construct a prognostic prediction model for type II EC patients using the "rms" package in R. A nomogram was developed to predict 1-year, 3-year, and 5-year OS. The performance of the prognostic model was evaluated in both the training and validation cohorts in terms of discrimination, calibration, and clinical benefit. The model's discrimination was assessed by calculating the area under the curve (AUC) from the ROC curves generated using the "survivalROC" package in R. Calibration was evaluated using calibration curves plotted with the calibrate function in the "rms" package. Clinical benefit was assessed through decision curve analysis (DCA), with the corresponding DCA curves created using the "dcurves" package in R. Finally, Kaplan-Meier (KM) survival curves were plotted for the identified independent prognostic factors, and differences between groups were compared using the log-rank test. We utilized log-log transformed KM survival plots to visually assess the proportional hazards assumptions. In this study, the significance level was set at 0.05.

Results

Characteristics of patients

The baseline characteristics of patients are summarized in Table 1. A total of 3,933 patients with type II EC were included in our study: 2,753 in the training cohort and 1,180 in the validation cohort. There was no significant difference in the evaluation indexes between the two groups ($P > 0.05$), indicating a balanced distribution.

Prognostic factors for type II EC

The results of the univariable and multivariable analyses of the training cohort are shown in Table 2. Univariable analysis revealed that race, marital status, tumor size, histology, grade, T stage, N stage, M stage, examination of para-aortic lymph nodes, examination of pelvic lymph nodes, surgery, lung metastasis, liver metastasis, bone metastasis, radiation therapy, and chemotherapy were all significant risk factors for OS ($P < 0.05$). These risk factors were included in the multivariable analysis. Race, tumor size, histology, grade, T stage, N stage, M stage, examination of para-aortic lymph nodes, examination of pelvic lymph nodes, surgery, lung metastasis, radiation therapy, and chemotherapy were identified as independent risk factors for adverse OS ($P < 0.05$).

Nomogram development and validation

Based on the results of Cox multivariable analysis, a nomogram for OS in the training cohort was constructed using R, as shown in Fig. 2. According to the nomogram, T stage contributed the most to the prognosis, followed by grade, surgery, M stage, tumor size, chemotherapy, race, N stage, lung metastasis, histology, radiation therapy, examination of pelvic lymph nodes, and examination of para-aortic lymph nodes. Each variable was assigned a different score, and the total score for patients with type II EC was calculated by summing the scores for each variable. A vertical line drawn at the total score on the horizontal axis indicates the 1-, 3-, and 5-year OS. In the training cohort, the C-index of the model was 0.791 (95% CI 0.780–0.802). In the validation cohort, the model's C-index was 0.798 (95% CI 0.778–0.818). The AUC and calibration curves were used to determine the prediction accuracy of the nomogram. The AUC values for predicting the 1-, 3-, and 5-year OS rates were 0.856, 0.827, and 0.816, respectively, in the training group (Fig. 3), and 0.863, 0.811, and 0.803 in the validation group, indicating good predictive ability. The calibration curve showed that the predicted survival probability of type II EC by the model was close to the actual probability, indicating good calibration (Fig. 4). The results of DCA suggested that the model has good clinical efficacy and application value (Fig. 5).

Survival analysis

The Kaplan-Meier method was used to evaluate the OS of patients with type II EC (Fig. 6). Survival analysis demonstrated a significantly reduced OS in patients with adverse prognostic factors, including tumor size ≥ 67 mm, Black race, grade III/IV tumors, carcinosarcoma histology, advanced TNM stages, and the presence of lung metastases. Additionally, lack of surgical intervention and receipt of radiation therapy or lymph node examinations were also associated with worse OS (all $P < 0.001$).

Discussion

This study identified key prognostic factors for type II EC using a large cohort of 3,933 patients from the SEER database. Independent predictors included race, tumor size, histology, grade, T stage, N stage, M stage, examination of para-aortic and pelvic lymph nodes, surgery, lung metastasis, and the use of radiation therapy or chemotherapy. Based on these factors, we developed and validated a nomogram with strong discriminative and predictive ability for estimating OS in patients with type II EC. Unlike most studies on type II EC, which are based on small samples, our research provides robust evidence from a large dataset. Our findings underscore the necessity of establishing a personalized staging system to guide prognosis assessment and management for these patients.

According to this study's analysis, race is an independent factor affecting the prognosis of type II EC, with Black individuals having a worse prognosis. In a population-based study on the diagnosis of type II EC in

Variables	Total (*N* = 3933)	Training cohort (*N* = 2753)	Validation cohort (*N* = 1180)	*p*-value
Age				0.531
< 45	98 (2.5%)	63 (2.3%)	35 (3.0%)	
45–59	888 (22.6%)	627 (22.8%)	261 (22.1%)	
60–74	2194 (55.8%)	1528 (55.5%)	666 (56.4%)	
≥75	753 (19.1%)	535 (19.4%)	218 (18.5%)	
Race				0.920
Black	659 (16.8%)	460 (16.7%)	199 (16.9%)	
White	2734 (69.5%)	1911 (69.4%)	823 (69.7%)	
Other	540 (13.7%)	382 (13.9%)	158 (13.4%)	
Marital				0.966
Divorce	442 (11.2%)	311 (11.3%)	131 (11.1%)	
Married	2030 (51.6%)	1427 (51.8%)	603 (51.1%)	
Single	1447 (36.8%)	1005 (36.5%)	442 (37.5%)	
Widowed	14 (0.4%)	10 (0.4%)	4 (0.3%)	
Tumor size, mm				0.633
≤46	2178 (55.4%)	1511 (54.9%)	667 (56.5%)	
47–66	808 (20.5%)	573 (20.8%)	235 (19.9%)	
≥67	947 (24.1%)	669 (24.3%)	278 (23.6%)	
Histology				0.479
Serous carcinoma (SC)	1425 (36.2%)	1000 (36.4%)	425 (36.0%)	
Carcinosarcoma (CS)	990 (25.2%)	700 (25.4%)	290 (24.6%)	
Clear cell carcinoma (CCC)	260 (6.6%)	171 (6.2%)	89 (7.5%)	
Mixed carcinoma (MC)	1258 (32.0%)	882 (32.0%)	376 (31.9%)	
Grade				0.902
I	388 (9.9%)	276 (10.0%)	112 (9.5%)	
II	335 (8.5%)	230 (8.4%)	105 (8.90%)	
III	2064 (52.5%)	1442 (52.4%)	622 (52.7%)	
IV	1146 (29.1%)	805 (29.2%)	341 (28.9%)	
T stage				0.132
T1	2374 (60.4%)	1689 (61.4%)	685 (58.1%)	
T2	463 (11.8%)	311 (11.3%)	152 (12.9%)	
T3	968 (24.6%)	671 (24.3%)	297 (25.1%)	
T4	128 (3.2%)	82 (3.0%)	46 (3.9%)	
N stage				0.261
N0	2938 (74.7%)	2037 (74.0%)	901 (76.4%)	
N1	537 (13.7%)	390 (14.2%)	147 (12.5%)	
N2	458 (11.6%)	326 (11.8%)	132 (11.1%)	
M stage				0.134
M0	3378 (85.9%)	2380 (86.5%)	998 (84.6%)	
M1	555 (14.1%)	373 (13.5%)	182 (15.4%)	
Examination of para-aortic lymph nodes				0.374
No	1859 (47.3%)	1288 (46.8%)	571 (48.4%)	
Yes	2074 (52.7%)	1465 (53.2%)	609 (51.6%)	
Examination of pelvic lymph node				0.058
No	821 (20.9%)	552 (20.1%)	269 (22.8%)	
Yes	3112 (79.1%)	2201 (79.9%)	911 (77.2%)	
Surgery				1.000
No	65 (1.7%)	45 (1.6%)	20 (1.7%)	
Yes	3868 (98.3%)	2708 (98.4%)	1160 (98.3%)	
Lung metastasis				0.571
No	3846 (97.8%)	2695 (97.9%)	1151 (97.5%)	
Yes	87 (2.2%)	58 (2.1%)	29 (2.5%)	
Liver metastasis				0.159
No	3886 (98.8%)	2725 (99.0%)	1161 (98.4%)	
Continued				

Variables	Total (*N* = 3933)	Training cohort (*N* = 2753)	Validation cohort (*N* = 1180)	*p*-value
Yes	47 (1.2%)	28 (1.0%)	19 (1.6%)	
Bone metastasis				0.160
No	3914 (99.5%)	2743 (99.6%)	1171 (99.2%)	
Yes	19 (0.5%)	10 (0.4%)	9 (0.8%)	
Radiation therapy				0.412
No	2247 (57.1%)	1585 (57.6%)	662 (56.1%)	
Yes	1686 (42.9%)	1168 (42.4%)	518 (43.9%)	
Chemotherapy				0.757
No/unknown	1546 (39.3%)	1087 (39.5%)	459 (38.9%)	
Yes	2387 (60.7%)	1666 (60.5%)	721 (61.1%)	

Table 1. Demographic and clinical characteristics of type II EC patients.

women, both EC-specific and overall mortality were found to be higher in non-Hispanic Black women compared to non-Hispanic White women. Further analysis revealed that clinicopathological factors were the most significant contributors to this difference, followed by sociodemographic and therapeutic factors[11]. In addition, a retrospective study found that racial differences in endometrial cancer prognosis may be related to genetic mutations. Mutations in TP53 and PIK3R1 were associated with poor prognosis, and the frequency of TP53 and FAT1 mutations was higher in Black/African American individuals, correlating with a worse prognosis[12].

Previous studies have shown that the relationship between tumor size and prognosis in EC patients is non-linear, with mortality risk increasing as tumor size increases[13]. Similarly, the analysis results of this study also showed that tumor size is a significant factor affecting the prognosis of patients with type II EC. The TNM stage is an important predictor for EC patients; the higher the stage, the greater the invasion of the primary tumor, resulting in a worse prognosis and lower survival rate[2,14]. Furthermore, lymph node metastasis significantly impacts EC, with studies showing it to be an important prognostic factor for EC[14,15]. In this study, the TNM staging from the AJCC 7th edition was used due to limitations of the SEER database. The analysis results indicated that higher TNM stages were associated with worse survival in patients with type II EC. The lung is the most common site of extraperitoneal organ metastasis in EC, with an incidence of 1.5%[16,17]. An analysis of prognostic factors for metastatic EC based on the SEER database showed that liver and lung metastases are independent prognostic factors for OS[18]. Interestingly, this study found that lung metastasis was an independent prognostic factor for type II EC, while liver metastasis was not.

Tumor pathologic grade is an important prognostic parameter in EC. Previous studies have described the prognostic significance of tumor pathologic grade in EC[19,20]. Studies have shown that highly malignant EC types are associated with significantly poorer prognoses. For instance, SC and CCC are linked to unfavorable outcomes due to their histological characteristics and aggressive behavior[21,22]. In addition to its impact on patient prognosis, tumor pathologic grade can also serve as an important reference for guiding adjuvant therapy[23]. In this study, we concluded that tumor pathologic grade is an independent prognostic factor for OS in patients with type II EC, consistent with previous findings.

Since the SEER database only includes information on surgical treatment and chemoradiotherapy, other treatment methods were not studied, analyzed, or discussed. Surgery is the most effective treatment for patients with early-stage EC[1]. Surgery can not only remove the primary lesion but also help determine factors that influence prognosis. Even in cases of advanced disease or distant tumor metastasis, the study recommends cytoreductive surgery[24,25]. In 2009, FIGO classified patients with pelvic lymph node metastasis as stage IIIC1 and those with positive para-aortic lymph nodes as stage IIIC2. The correlation between lymph node metastasis status and the prognosis of endometrial cancer has been well established[26]. This study suggests that the examination of para-aortic lymph nodes and pelvic lymph node sampling has a prognostic survival benefit in patients with type II EC. This benefit may be due to the implementation of further treatment upon finding positive lymph nodes. However, the effectiveness of radiation and chemotherapy remains controversial. An international, open-label, multicenter, randomized phase 3 trial showed that adjuvant chemotherapy given during and after radiotherapy for high-risk endometrial cancer did not improve 5-year OS[27]. In contrast, a retrospective study showed improved OS in women with stage IB and II uterine serous carcinoma and clear cell carcinoma who received radiation, chemotherapy, or both[28]. A study based on SEER data showed that brachytherapy was beneficial for stage I-II serous carcinoma and clear cell carcinoma, chemotherapy was beneficial for stage III serous carcinoma and clear cell carcinoma, and both chemotherapy and brachytherapy were beneficial for stage I-II serous carcinoma[29]. The results of this study indicate that radiation therapy and chemotherapy are independent risk factors affecting the prognosis of type II EC.

Based on the identified independent risk factors, a nomogram model was constructed to predict 1-, 3-, and 5-year OS in patients with type II EC. The C-index of the model was 0.791 (95% CI 0.780–0.802) in the training group and 0.798 (95% CI 0.778–0.818) in the validation group, indicating good predictive accuracy. The ROC curve, calibration curve, and DCA all demonstrated that the prediction model had good consistency and clinical utility. Additionally, the effects of different variables on patients' OS were further analyzed using the Kaplan-Meier method. Although the nomogram prediction model demonstrated good accuracy in this study, there are some limitations. First, this study is a retrospective analysis based on the SEER database, which may introduce

Variables	Univariable analysis		Multivariable analysis	
	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value
Age				
< 45	Reference			
45–59	0.76 (0.52–1.09)	0.135		
60–74	0.77 (0.54–1.11)	0.16		
≥75	0.83 (0.57–1.21)	0.329		
Race				
Black	Reference		Reference	
White	0.57 (0.49–0.66)	< 0.001	0.84 (0.72–0.99)	0.035
Other	0.56 (0.45–0.69)	< 0.001	0.65 (0.52–0.81)	< 0.001
Marital				
Divorce	Reference		Reference	
Married	0.86 (0.70–1.05)	0.136	0.84 (0.68–1.04)	0.106
Single	1.36 (1.11–1.67)	0.003	0.98 (0.80–1.21)	0.866
Widowed	0.29 (0.04–2.10)	0.222	0.55 (0.08–3.98)	0.555
Tumor size				
< 46	Reference		Reference	
47–66	1.73 (1.48–2.02)	< 0.001	1.02 (0.86–1.20)	0.825
≥67	3.05 (2.66–3.50)	< 0.001	1.18 (1.00-1.38)	0.048
Histology				
Serous carcinoma	Reference		Reference	
Carcinosarcoma	1.47 (1.28–1.69)	< 0.001	1.56 (1.35–1.82)	< 0.001
Clear-cell carcinoma	1.06 (0.83–1.35)	0.645	1.50 (1.17–1.93)	< 0.001
Mixed cell adenocarcinoma	0.43 (0.37–0.52)	< 0.001	1.01 (0.84–1.21)	0.937
Grade				
I	Reference		Reference	
II	2.28 (1.37–3.80)	0.002	1.87 (1.11–3.14)	0.019
III	6.41 (4.19–9.81)	< 0.001	2.58 (1.65–4.04)	< 0.001
IV	7.40 (4.82–11.37)	< 0.001	2.60 (1.65–4.08)	< 0.001
T stage				
T1	Reference		Reference	
T2	2.18 (1.80–2.64)	< 0.001	1.25 (1.03–1.52)	0.027
T3	4.50 (3.92–5.16)	< 0.001	1.43 (1.22–1.69)	< 0.001
T4	7.42 (5.78–9.53)	< 0.001	1.62 (1.22–2.16)	< 0.001
N stage				
N0	Reference		Reference	
N1	2.19 (1.87–2.56)	< 0.001	1.10 (0.93–1.31)	0.273
N2	2.47 (2.11–2.90)	< 0.001	1.41 (1.17–1.70)	< 0.001
M stage				
M0	Reference		Reference	
M1	4.71 (4.12–5.38)	< 0.001	1.37 (1.15–1.63)	< 0.001
Examination of para-aortic lymph nodes				
No	Reference		Reference	
Yes	0.70 (0.62–0.79)	< 0.001	0.80 (0.68–0.93)	0.005
Examination of pelvic lymph node				
No	Reference		Reference	
Yes	0.55 (0.48–0.63)	< 0.001	0.82 (0.68–0.98)	0.033
Surgery				
No	Reference		Reference	
Yes	0.12 (0.09–0.16)	< 0.001	0.44 (0.31–0.63)	< 0.001
Lung metastasis				
No	Reference		Reference	
Yes	5.50 (4.14–7.30)	< 0.001	1.52 (1.09–2.13)	0.014
Liver metastasis				
No	Reference		Reference	
Continued				

Variables	Univariable analysis		Multivariable analysis	
	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value
Yes	6.77 (4.64–9.88)	< 0.001	1.07 (0.68–1.67)	0.776
Bone metastasis				
No	Reference		Reference	
Yes	8.54 (4.82–15.12)	< 0.001	1.83 (0.98–3.41)	0.056
Radiation therapy				
No	Reference		Reference	
Yes	0.57 (0.51–0.65)	< 0.001	0.86 (0.75–0.99)	0.039
Chemotherapy				
No/unknown	Reference		Reference	
Yes	1.19 (1.05–1.34)	0.008	0.56 (0.49–0.65)	< 0.001

Table 2. Univariable and multivariable Cox analyses on variables for the prediction of OS of patients.

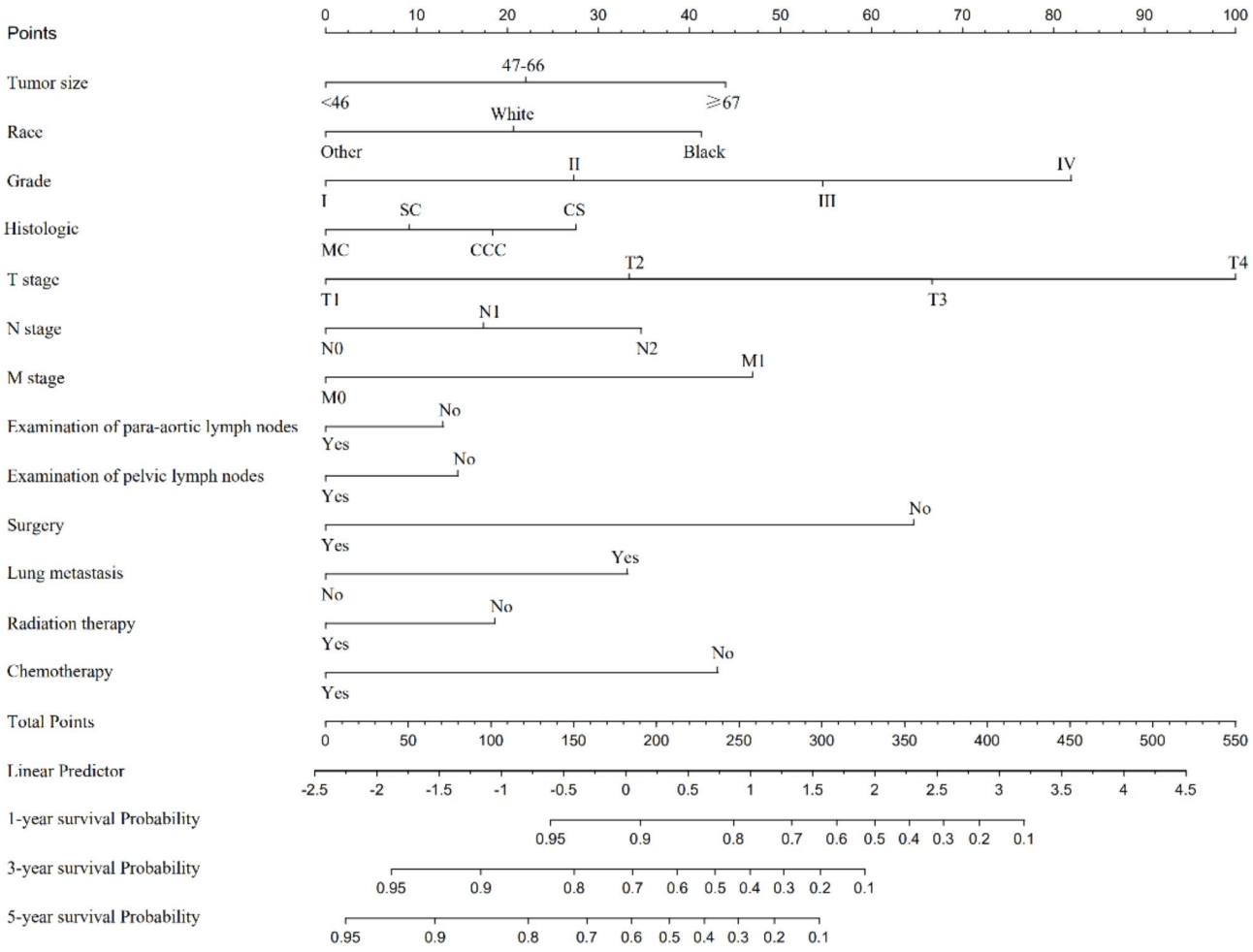

Fig. 2. Nomogram for predicting OS of patients with type II EC.

selection bias. Second, the SEER data pertains to patients in the United States, potentially limiting the model's applicability to other regions. Therefore, data from various national institutions and centers are needed to refine the model. Finally, to enhance the accuracy and clinical utility of the prediction model, it is essential to validate and evaluate the model using external datasets[30]. However, this study lacks sufficient external data, preventing external validation of the model. Only the original data was divided into training and validation sets, making the general applicability of the nomogram prediction model unclear. Therefore, further verification with large sample sizes and multi-center data is necessary.

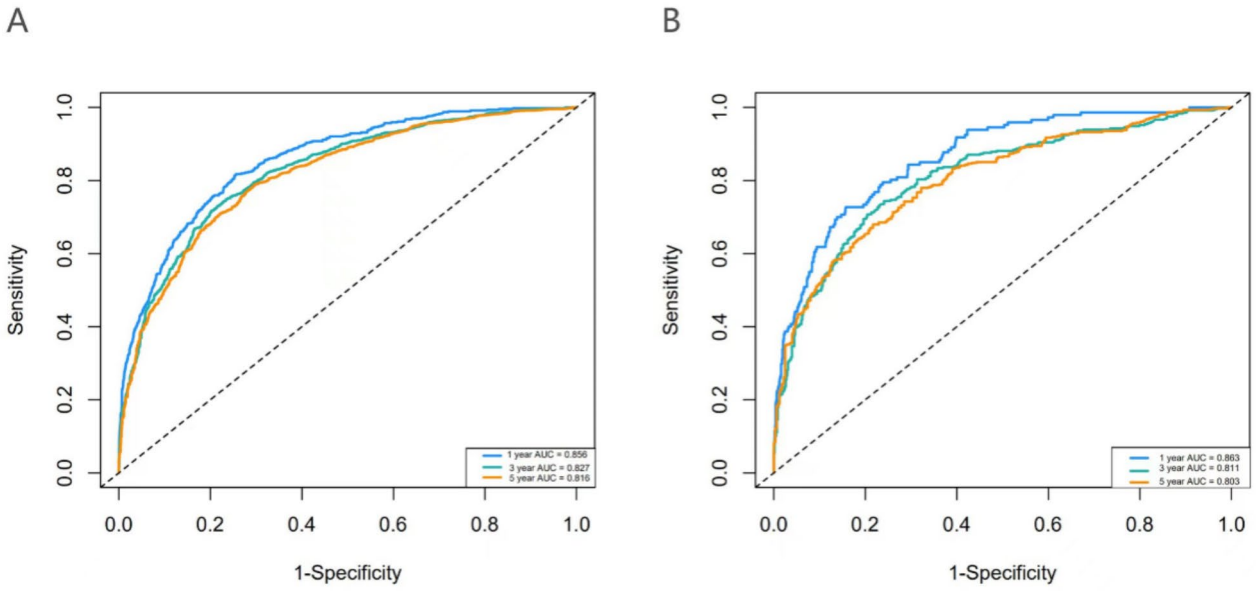

Fig. 3. Receiver operating characteristics curves in the training (**A**) and validation (**B**) groups for 1-, 3-, and 5-year OS.

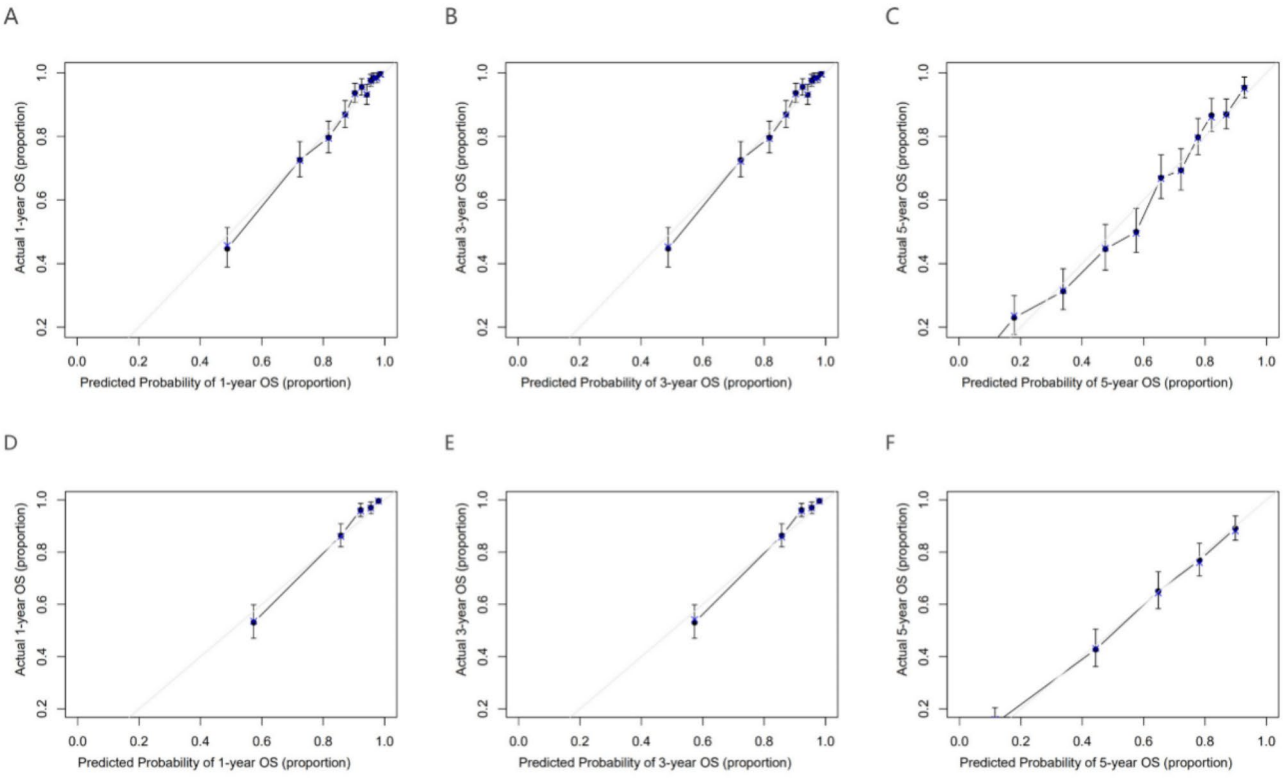

Fig. 4. Calibration curves of the nomogram for 1-, 3-, and 5-year OS of type II EC patients in the training cohort (**A**–**C**); and in the validation cohort (**D**–**F**).

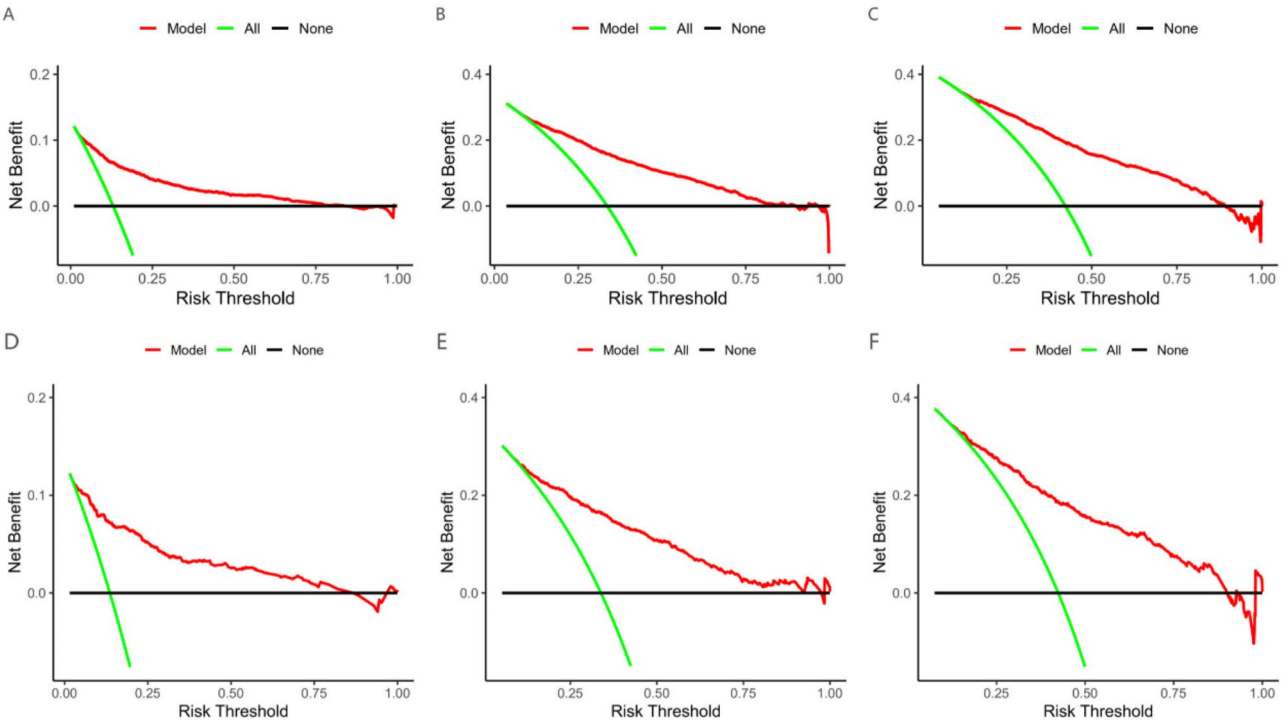

Fig. 5. Decision curve analysis curves of the nomogram for 1-, 3-, and 5-year OS of type II EC patients in the training cohort (**A**–**C**); and in the validation cohort (**D**–**F**).

Conclusions

In summary, using the SEER database, we identified independent predictors of OS in patients with type II EC and established a nomogram model that can intuitively and accurately predict individual survival prognosis. This model can guide clinicians in adopting appropriate measures for personalized treatment by predicting survival time.

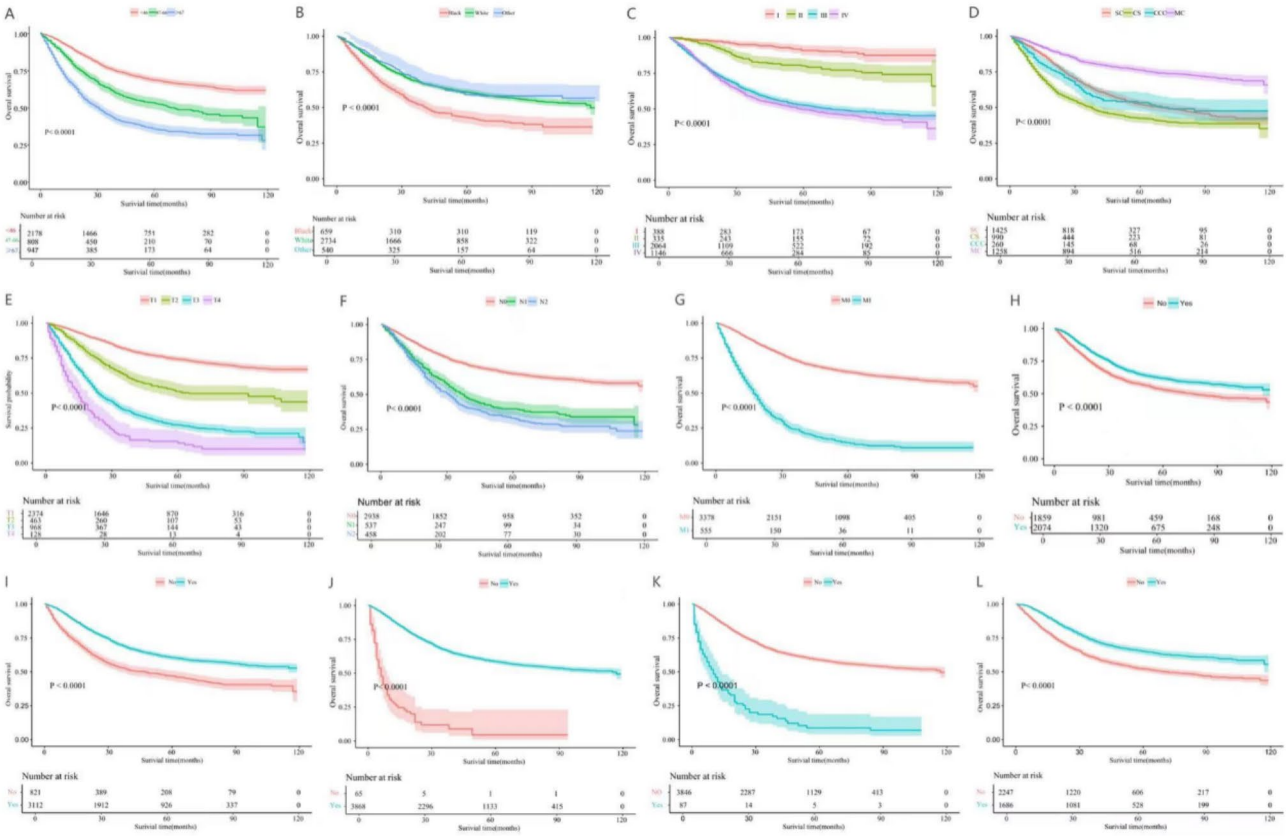

Fig. 6. Kaplan–Meier curves of type II EC patients stratified by (**A**) Tumor size; (**B**) Race; (**C**) Grade; (**D**) Histologic; (**E**) T stage; (**F**) N stage; (**G**) M stage; (**H**) Examination of para-aortic lymph nodes; (**I**) Examination of pelvic lymph nodes; (**J**) Surgery; (**K**) Lung metastasis; and (**L**) Radiation therapy.

Data availability

The dataset analyzed in this study is available through the SEER database.

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Author contributions

YL and JHS designed and wrote the first draft of the study. JH analyzed the data. QL assisted the data analysis. XJJ and SMT revised the manuscript. All authors contributed to the article and approved the submitted version.

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Declarations

Competing interests

The authors declare no competing interests.

Additional information

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