International Journal of Colorectal Disease (2024) 39:191
https://doi.org/10.1007/s00384-024-04760-2

RESEARCH

The risk factors of lymph node metastasis in early colorectal cancer: a predictive nomogram and risk assessment

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Accepted: 11 November 2024 / Published online: 28 November 2024

Abstract

Purpose Endoscopic procedures and surgery are common treatments for early colorectal cancer (CRC). However, only approximately 10% of patients who undergo surgery have lymph node metastases (LNM) detected on postoperative pathology, which often leads to overtreatment. This study aims to comprehensively analyze the risk factors for LNM in early CRC patients, establishing a predictive model to aid in treatment decisions.

Methods This study reviewed the clinicopathologic data of patients with early CRC who underwent surgery from January 2015 to June 2023. Univariate and multivariate logistic regression analyses were employed to identify LNM risk factors. The receiver operating characteristic (ROC) analysis and calibration curves were also constructed to verify the model's discrimination and calibration. A simplified scale was calculated to promote the risk stratification for LNM.

Results The study analyzed medical records of 375 patients. Of these, 37 (9.9%) cases had LNM. Univariate analysis identified age, nerve invasion, depth of submucosal invasion, histologic grade, LVI, and tumor budding as risk factors. The multivariate analysis confirmed histologic grade (OR, 13.403; 95% CI, 1.415–126.979; $P = 0.024$), LVI (OR, 6.703; 95% CI, 2.600–17.284; $P < 0.001$), and tumor budding (OR, 3.090; 95% CI, 1.082–8.820; $P = 0.035$) as independent predictors. The optimal nomogram, incorporating six risk factors, demonstrated strong predictability with an area under the ROC curve (AUC) of 0.837 (95% CI, 0.762–0.912). A simplified risk assessment scale with a total score of 19 points was developed.

Conclusion The study developed a nomogram and a simplified risk assessment scale to predict LNM risk, potentially optimizing the management of early CRC patients.

Keywords Early colorectal cancer · Lymph node metastasis · Predictive model · Risk stratification · Background

Colorectal cancer (CRC) ranks as the third most prevalent cancer and the second leading cause of cancer-related mortality globally [25]. Early CRC is characterized by tumors confined to the mucosa or submucosa, irrespective of lymph node metastasis (LNM) [35]. Advances in endoscopic technology have enabled the resection of increasingly many early CRC cases endoscopically, avoiding the trauma and intestinal dysfunction associated with surgical interventions [5]. The current guidelines recommend endoscopic treatment for intramucosal cancers or mildly invasive submucosal cancers (depth of invasion < 1000 μm) [14, 27]. However, the management of cases with submucosal invasion depths > 1000 μm, lymphovascular invasion (LVI), poor histopathological types, and high-grade tumor budding remains under debate. Coloproctectomy plus lymph node dissection is generally advised for these higher-risk cases due to the increased likelihood of LNM [12, 23, 28]. Recent studies indicated that approximately 10% of patients who underwent surgery had LNM and the majority of them did not develop LNM postoperatively [10, 17, 26]. Preoperative prediction of LNM risk could significantly benefit these patients by potentially allowing for the selection of less invasive treatments, thereby avoiding reduced quality of life and complications associated with more extensive surgical procedures.

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Despite advancements, the preoperative detection of LNM in patients scheduled for coloproctectomy remains challenging, even with the use of computed tomography (CT), magnetic resonance imaging (MRI), and endoscopic ultrasound (EUS) [15]. Although some researchers have developed predictive models incorporating depth of invasion, LVI, histopathological type, and tumor budding for evaluating the risk of LNM [14], these models, based on non-Chinese populations, may not be directly applicable to Chinese patients without further validation. Besides, patients with T1 carcinoma may also undergo endoscopic resection based on comprehensive clinical evaluations of their overall state and radiographic and pathological findings [3] without adverse effects on prognosis [30]. A prediction model that integrates multiple clinicopathological features could enhance the accuracy of LNM risk estimation in these patients, aiding the decision-making process regarding the necessity of additional surgical interventions. An effective and precise prediction model could broaden the criteria for early CRC treatment by identifying individuals with a low risk of LNM from a surgically resected cohort. This study aims to develop a nomogram based on the clinicopathological features of early CRC patients to enhance the effectiveness of predicting LNM.

Material and methods

Patient selection

Patients with early CRC who underwent coloproctectomy involving lymph node dissection, with or without preceding endoscopic resection, at the Affiliated Hospital of Qingdao University from January 2015 to June 2023 were enrolled in the study. The exclusion criteria were metastatic colorectal cancer or multiple malignant lesions, recurrence of cancer after surgical operation, preoperative treatment with neoadjuvant chemotherapy or radiotherapy, and concurrent life-threatening conditions such as cardiac failure, renal failure, or respiratory failure.

Data collection

The clinicopathologic data were retrieved from the hospital's medical database, encompassing age, sex, body mass index (BMI), histories of hypertension and diabetes, smoking and alcohol consumption habits, levels of carcinoembryonic antigen (CEA) and cancer antigen 199 (CA199), endoscopic features, and pathological findings. Endoscopic features included tumor location, size, and gross type. The preoperative pathologic findings included depth of submucosal invasion, histologic grade, LVI, tumor budding, and nerve invasion (NI). The postoperative pathological outcomes documented LNM. All samples were examined independently by two experienced pathologists. In cases of diagnostic discrepancies, a third senior pathologist reviewed the samples. A definitive diagnosis was established when at least two pathologists reached an agreement.

Written informed consent was obtained from all participants prior to coloproctectomy. The study received approval from the ethics committee of the Affiliated Hospital of Qingdao University.

Histologic evaluation

In this study, the pathological diagnosis was based on the third English edition of the *Japanese Classification of Colorectal, Appendiceal, and Anal Carcinoma* [35]. Endoscopic classification followed the Paris classification criteria for colorectal cancers [34]. The measurements of submucosal invasion depth were defined using the following criteria: (1) For non-pedunculated lesions, the depth of invasion was measured from the recognized lowest border of the muscularis mucosae (MM) to the deepest invasive site of the tumor. (2) If the MM was unidentifiable, the depth was measured from the surface of the lesion. (3) For pedunculated lesions with MM involvement, the depth of submucosal invasion was determined by measuring the distance from the deepest invasion point to the reference line. The reference line was defined as the border between the head and the stalk of the tumor. In cases where tumor invasion was confined to the head, the submucosal (SM) invasion depth was considered 0 μm. Tumors were graded based on their predominant histologic type: well-differentiated adenocarcinoma and papillary adenocarcinoma as G1, moderately differentiated adenocarcinoma as G2, and poorly differentiated adenocarcinoma, mucinous adenocarcinoma, or signet ring cell carcinoma as G3. Tumor budding was defined as a single cell or a cluster of < 5 neoplastic cells at the advancing edge of the tumor. Budding was assessed on hematoxylin and eosin-stained slides within a selected "hot-spot" area under a $\times 20$ objective lens and categorized into three levels: Bd1 (0–4), Bd2 (5–9), and Bd3 (≥ 10).

Statistical analyses

Initially, continuous variables were converted to categorical ones for statistical evaluation. The association between variables and LNM was determined using either the chi-square test or Fisher's exact test in the univariate analysis. Variables significant at $P < 0.05$ were subsequently included in the multivariate logistic regression analysis to identify independent risk factors for LNM. All analyses were conducted using SPSS software, version 26.0 (SPSS, Chicago, IL, USA).

Nomograms were constructed based on the findings from both the univariate and multivariate logistic regression analyses. The effectiveness of these nomograms was evaluated by comparing the area under the receiver operating characteristic (ROC) curve (AUC) for each model. The optimal nomogram was further validated through assessments of its discrimination and calibration abilities. The discriminative performance was measured by the concordance index, which ranges from 0.5 to 1.0; a value closer to 1.0 indicates higher accuracy in predicting actual outcomes. Calibration curves were generated using the bootstrap method, involving 1000 bootstrap samples, to ensure internal validity. A well-calibrated model closely aligns the calibration curve with the ideal reference line, represented by a slope of 1. These validation processes were carried out using R Software version 4.3.2 (www.r-project.org).

Results

Baseline characteristics and follow-up data

A total of 375 patients who underwent radical colorectal resection with lymph node dissection were enrolled in the study. Among these, 37 cases (9.9%) presented with LNM. The cohort predominantly consisted of males (59.20%) and individuals over 60 years of age (65.33%). A minority of patients reported histories of diabetes, hypertension, smoking, and alcohol consumption. Most patients had normal levels of CEA and CA199. The rectum was the most common tumor location, accounting for 70.40% of cases. Overall, the gross tumor type of 291 patients (77.60%) was pedunculated, and the majority of lesions were smaller than 3 cm. NI and LVI were present in 17 (4.53%) and 29 (7.73%) patients, respectively. Regarding the depth of invasion, 68 patients (18.13%) had tumors less than 1000 μm deep, while 307 patients (81.87%) presented with tumors deeper than 1000 μm, including 117 patients (31.20%) with tumors exceeding 3000 μm in depth. Histologically, the majority were classified as G2, indicating moderately differentiated carcinomas. For tumor budding, 268 cases (71.47%) were identified as Bd1, 9 cases (24.3%) as Bd2, and 11 cases (29.7%) as Bd3. The clinical characteristics of the cohort are detailed in Table 1. Within 5 years post-surgery, recurrence or metastasis occurred in seven patients. The remaining 368 patients showed no signs of recurrence or metastasis until October 2023.

Risk factors for LNM

According to the univariate analysis presented in Table 1, significant risk factors for LNM included young age, deep submucosal invasion, poor histologic grade, NI, LVI, and high-grade tumor budding. Furthermore, multivariate logistic regression analysis demonstrated that poor histologic grade (OR, 13.403; 95% CI, 1.415–126.979; $P=0.024$), LVI (OR, 6.703; 95% CI, 2.600–17.284; $P<0.001$), and tumor budding (OR, 3.090; 95% CI, 1.082–8.820; $P=0.035$) were independent risk factors of LNM. These findings are presented in Table 2. As shown in Table 3, 11.4% of patients with invasion depth exceeding 1000 μm experienced LNM. When the depth of invasion was deeper than 1000 μm but there were no other risk factors, the rate of LNM was 2.48%. In cases where there were at least one risk factor and the invasion depth was greater than 1000 μm, the rate of LNM was 17.20%.

Receiver operating characteristic analysis

Based on the results from the univariate and multivariate analyses, two nomograms were constructed and evaluated through ROC analysis to compare their predictive value on LNM. Nomogram 1 shown in Fig. 1 included six variables from the univariate analysis: age, NI, depth of submucosal invasion, histologic grade, LVI, and tumor budding. In contrast, nomogram 2 shown in Fig. S1 incorporated only three variables from the multivariate analysis: LVI, histologic grade, and tumor budding. The ROC curves demonstrating the predictability of LNM for both nomograms are displayed in Fig. 2. The AUC of the two nomograms were 0.837 and 0.792, respectively, indicating that nomogram 1 offered a more precise prediction of LNM in early CRC patients. Consequently, the more comprehensive nomogram 1 was selected for further analysis in our study.

The validation of the nomogram

The nomogram with six risk factors assigned scores ranging from 0 to 100 to each variable, reflecting their contributions to the predicted outcome. The total score can be obtained by summing the scores for all variables. The total score was then used to determine the probability of LNM occurrence by aligning a vertical line with the "Risk of LNM" scale, as depicted in Fig. 1. Calibration of the nomogram was conducted using the bootstrap method, which involved over 1000 repetitive samples for internal validation. The calibration curve, displayed in Fig. 3, closely matches the ideal curve, indicating a satisfactory degree of concordance. The consistency index (CI) of the model was 0.837, signifying a moderate discriminative ability.

Risk stratification for LNM

A simplified scale, depicted in Table 4, was developed from the nomogram for risk stratification of LNM. Weighting points were assigned to each variable within the nomogram,

Table 1 Comparisons of clinicopathological characteristics between LNM positive and LNM negative groups

Variable			LNM		*P* value
		No. of patients, *n* (%) 375	Negative *n* = 338	Positive *n* = 37	
Age (yr)	< 60	130 (34.67)	110 (32.5%)	20 (54.1%)	0.009
	≥ 60	245 (65.33)	228 (67.5%)	17 (45.9%)	
Sex	Male	222 (59.20)	201 (59.5%)	21 (56.8%)	0.750
	Female	153 (40.80)	137 (40.5%)	16 (43.2%)	
BMI (kg/m^2)	< 18.5	8 (2.13)	8 (2.4%)	0 (0.0%)	0.590
	18.5–24.9	187 (49.87)	167 (49.4%)	20 (54.1%)	
	≥ 25	180 (48.00)	163 (48.2%)	17 (45.9%)	
Hypertension	No	245 (65.33)	224 (66.3%)	21 (56.8%)	0.248
	Yes	130 (34.67)	114 (33.7%)	16 (43.2%)	
Diabetes	No	317 (84.53)	285 (84.3%)	32 (86.5%)	0.729
	Yes	58 (15.47)	53 (15.7%)	5 (13.5%)	
Smoke	No	259 (69.07)	232 (68.6%)	27 (73.0%)	0.588
	Yes	116 (30.93)	106 (31.4%)	10 (27.0%)	
Alcohol	No	257 (68.53)	232 (68.6%)	25 (67.6%)	0.894
	Yes	118 (31.47)	106 (31.4%)	12 (32.4%)	
CEA (ng/mL)	0–5	338 (90.13)	305 (90.2%)	33 (89.2%)	0.839
	> 5	37 (9.87)	33 (9.8%)	4 (10.8%)	
CA199 (U/mL)	0–37	367 (97.87)	330 (97.6%)	37 (100.0%)	0.432
	> 37	8 (2.13)	8 (2.4%)	0 (0.0%)	
Location	Rectum	264 (70.40)	235 (69.5%)	29 (78.4%)	0.477
	Left hemicolon	75 (20.00)	68 (20.1%)	7 (18.9%)	
	Transverse colon	6 (1.60)	6 (1.8%)	0 (0.0%)	
	Right hemicolon	30 (8.00)	29 (8.6%)	1 (2.7%)	
Gross type	Pedunculated	291 (77.60)	261 (77.2%)	30 (81.1%)	0.593
	Others	84 (22.40)	77 (22.8%)	7 (18.9%)	
Size (cm)	< 1.5	126 (33.60)	112 (33.1%)	14 (37.8%)	0.809
	1.5–2.9	177 (47.20)	160 (47.3%)	17 (45.9%)	
	≥ 3.0	72 (19.20)	66 (19.5%)	6 (16.2%)	
Depth of invasion (μm)	< 1000	68 (18.13)	66 (19.5%)	2 (5.4%)	0.020
	1000–1999	86 (22.93)	80 (23.7%)	6 (16.2%)	
	2000–2999	104 (27.74)	94 (27.8%)	10 (27.0%)	
	≥ 3000	117 (31.20)	98 (29.0%)	19 (51.4%)	
NI	No	358 (95.47)	327 (96.7%)	31 (83.8%)	0.001
	Yes	17 (4.53)	11 (3.3%)	6 (16.2%)	
LVI	No	346 (92.27)	323 (95.6%)	23 (62.2%)	< 0.001
	Yes	29 (7.73)	15 (4.4%)	14 (37.8%)	
Histologic grade	G1	78 (20.80)	77 (22.8%)	1 (2.7%)	< 0.001
	G2	261 (69.60)	234 (69.2%)	27 (73.0%)	
	G3	36 (9.60)	27 (8.0%)	9 (24.3%)	
Tumor budding	Bd1	268 (71.47)	251 (74.3%)	17 (45.9%)	< 0.001
	Bd2	68 (18.13)	59 (17.5%)	9 (24.3%)	
	Bd3	39 (10.40)	28 (8.3%)	11 (29.7%)	

LNM, lymph node metastasis; *BMI*, body mass index; *CEA*, carcinoembryonic antigen; *CA199*, cancer antigen 199; *NI*, nerve invasion; *LVI*, lymphovascular invasion; *G1*, well-differentiated adenocarcinoma and papillary adenocarcinoma; *G2*, moderately differentiated adenocarcinoma; *G3*, poorly differentiated adenocarcinoma, mucinous adenocarcinoma, and signet ring cell carcinoma

Table 2 Comparative multivariate analysis of LNM positive and LNM negative groups

Parameters		Multivariate analysis			
		OR	95% CI		*P* value
Age (yr)	< 60	1			
	≥ 60	0.525	0.237	1.164	0.113
Depth of invasion (μm)	< 1000	1			
	1000–1999	1.036	0.178	6.028	0.969
	2000–2999	1.469	0.273	7.911	0.654
	≥ 3000	3.333	0.683	16.274	0.137
NI	No	1			
	Yes	2.901	0.755	11.149	0.121
LVI	No	1			
	Yes	6.703	2.6	17.284	< 0.001
Histologic grade	G1	1			
	G2	6.578	0.805	53.741	0.079
	G3	13.403	1.415	126.979	0.024
Tumor budding	Bd1	1			
	Bd2	1.758	0.692	4.463	0.235
	Bd3	3.090	1.082	8.820	0.035

LNM, lymph node metastasis; *NI*, nerve invasion; *LVI*, lymphovascular invasion; *G1*, well-differentiated adenocarcinoma and papillary adenocarcinoma; *G2*, moderately differentiated adenocarcinoma; *G3*, poorly differentiated adenocarcinoma, mucinous adenocarcinoma, and signet ring cell carcinoma

Table 3 Comparison of LNM at invasion depth > 1000 μm

	LNM, *n*	No. of patients, *n*	Rate of LNM (%)
> 1000 μm	35	307	11.40
> 1000 μm, without other risk factors	3	121	2.48
> 1000 μm, with at least one risk factor	32	186	17.20

Risk factors defined as age < 60 years, lymphovascular invasion, nerve invasion, Bd3 tumor budding, and G3 histological grading

LNM, lymph node metastasis; *G3*, poorly differentiated adenocarcinoma, mucinous adenocarcinoma, and signet ring cell carcinoma

rounded to the nearest whole number and proportional to their beta regression coefficient values. The total score of the scale was calculated to be 19, and the cohort was divided into three risk categories: low, medium, and high risk. Patients with a total score of 0–5 were classified as "low-risk," within which there were three cases of LNM, corresponding to an LNM rate of 1.89% and a 5-year cancer-specific survival (CSS) rate of 98.82%. For patients scoring 6–9 points, termed "medium-risk," there were 19 LNM cases, resulting in an LNM rate of 10.16% and a 5-year CSS of 96.72%. Patients with scores above 10 were categorized as "high-risk," among whom 15 experienced LNM, representing an LNM rate of 51.72% and a CSS rate of 91.30%. The risk stratification based on LNM is detailed in Table 5.

Discussion

Endoscopic resection, specifically endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD), has become a preferred treatment method for early CRC [5, 7]. Guidelines and studies have shown that lesions confined to the muscularis mucosa or submucosal lesions with invasion depths of less than 1000 μm are suitable for these less invasive endoscopic techniques [7, 12, 14, 19, 23, 27, 31]. A retrospective study highlighted impressive 5-year survival rates following endoscopic resection, with 100% for intramucosal cancer (Tis) and 96% for submucosal invasive cancer (T1) [27]. Previous studies indicated that only 2–10% of patients who undergo coloproctectomy exhibit LNM [2, 4, 13, 21, 24, 32], suggesting that the remaining 90% might undergo unnecessary surgical resections, leading to prolonged hospital stays, diminished quality of life, and potential postoperative mortality rates of 1.5–2% [6, 16, 29]. Additionally, while some patients meet the endoscopic indications, a small proportion still develop LNM during the follow-up of ESD. Moreover, deciding on endoscopic treatment based solely on endoscopic findings remains challenging for some patients. It is crucial to comprehensively analyze and identify risk factors for LNM and develop a practical method to assist in clinical decision-making. Although existing studies contribute to this field, they often lack comprehensive long-term validation. Consequently, our study proposes an innovative algorithm designed to predict LNM risk by integrating contemporary insights with data from our center.

In our analysis, patients with LNM were typically younger and exhibited deeper tissue invasion, poorer differentiation, higher grades of tumor budding, and increased risks of NI and LVI. A previous study also indicated that younger CRC patients were more likely to develop LNM than older patients [11]. This phenomenon could be attributed to the higher metabolic rates and greater tissue regeneration capacities of younger individuals, which may create more favorable conditions for tumor growth and metastasis.

Our findings align with previous studies that have shown invasion depth was not an independent risk factor in multivariate analyses despite its significant correlations in univariate analyses [21, 22, 24]. Furthermore, evidence suggested that invasion depth was a weaker predictor of LNM than other pathological factors such as LVI,

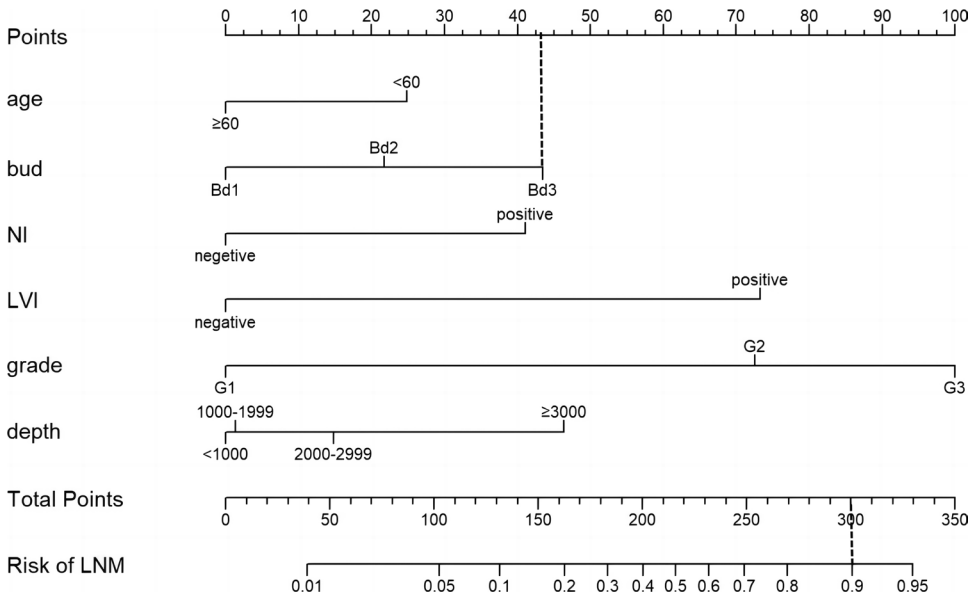

Fig. 1 Nomogram for predicting LNM in early CRC patients. LNM, lymph node metastasis; CRC, colorectal cancer; NI, nerve invasion; LVI, lymphovascular invasion; G1, well-differentiated adenocarcinoma and papillary adenocarcinoma; G2, moderately differentiated adenocarcinoma; G3, poorly differentiated adenocarcinoma, mucinous adenocarcinoma, and signet ring cell carcinoma

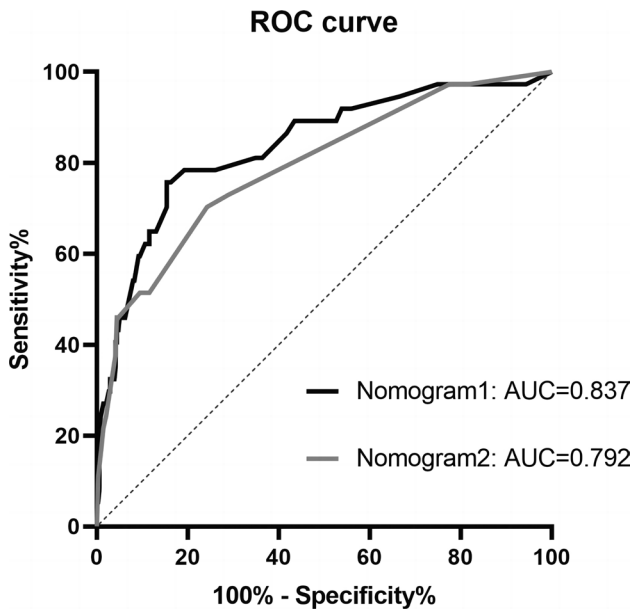

Fig. 2 ROC curves for nomogram 1 and nomogram 2. AUC, area under the receiver operating characteristic (ROC) curve

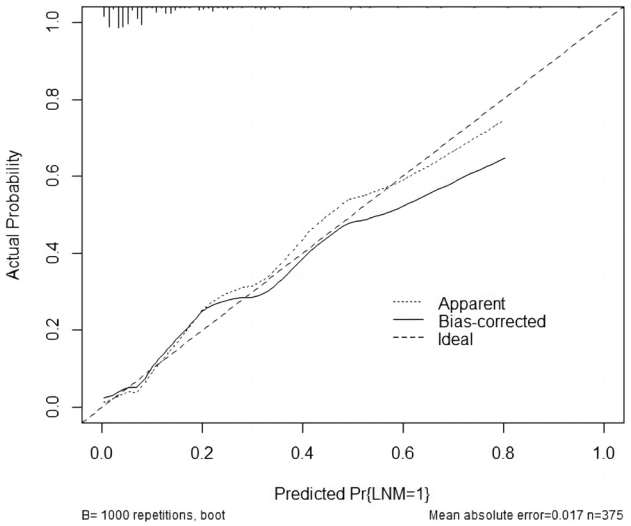

Fig. 3 Calibration curve of the nomogram. LNM, lymph node metastasis

histological differentiation, and tumor budding [32]. A meta-analysis revealed that invasion depth greater than 1500 μm was substantially associated with LNM [8]. Kim et al. reported a low LNM rate of 1.5% (4 out of 271 patients) when the depth of submucosal infiltration reached 2000 μm [18]. Despite the unclear predictive value of invasion depth, we have included it in our nomogram based on the area under the AUC analysis. The effectiveness of invasion depth as a predictor of LNM needs further exploration.

Our findings showed that positive LVI, high-grade tumor budding, and poorly differentiated histology were associated with LNM, aligning with findings from previous studies [2, 9, 33]. In this study, LVI and histology grading emerged as the most decisive risk factors for LNM. This finding is supported by a retrospective analysis of 428 individuals, which identified only LVI-positive status and poorly differentiated histology as statistically significant predictors of LNM [18]. NI has been recognized as a predictive factor in multiple studies and should not be overlooked in LNM risk assessments [8]. Some studies demonstrated that NI was associated with LNM in early CRC, which may be particularly relevant to large caliber axons often in

Table 4 The scoring scale based on the nomogram

Variable	Points			
Age	≥60	<60		
	0	2		
Bud	Bd1	Bd2	Bd3	
	0	2	3	
NI	Negative	Positive		
	0	3		
LVI	Negative	Positive		
	0	4		
Grade	G1	G2	G3	
	0	4	5	
Depth	<1000	1000–1999	2000–2999	≥3000
	0	1	1	2

NI, nerve invasion; *LVI*, lymphovascular invasion; *G1*, well-differentiated adenocarcinoma and papillary adenocarcinoma; *G2*, moderately differentiated adenocarcinoma; *G3*, poorly differentiated adenocarcinoma, mucinous adenocarcinoma, and signet ring cell carcinoma

Table 5 The risk stratification for LNM

Risk category	Total points	LNM ($n=37$)	Rate of LNM (%)	Five-year CSS (%)
Low	0–5	3	1.89	98.82
Intermediate	6–9	19	10.16	96.72
High	>10	15	51.72	91.30

LNM, lymph node metastasis; *CCS*, cancer-specific survival rate

contact with lymph nodes [1, 20]. Consequently, a detailed assessment of tumor differentiation, budding, NI, and LVI is critical for predicting LNM risk in post-ESD patients.

Our study analyzed the risk factors for LNM in patients by evaluating detailed pathological characteristics and developed a nomogram incorporating six variables: age, invasion depth, histologic grade, tumor budding, NI, and LVI. Compared with models from Kajiwara et al. and Fujino et al., our nomogram showed superior discriminative ability, with an AUC of 0.837 (95% CI, 0.762–0.912). This study presents a novel model specifically developed for the Chinese population, aiming to assist endoscopists in making therapeutic decisions for early CRC patients. We transformed the nomogram into a straightforward, stratified scoring table to enhance its practicality and simplify its use. This risk assessment scale, the first of its kind in our study, is expected to significantly enhance the clarity of treatment indications.

In our simplified scoring table, patients with a total score not exceeding 5 have a nearly 1.89% probability of LNM, making endoscopic treatment a preferred option. These patients exhibit a 5-year survival rate of up to 90%. Additionally, our study found that the absolute risk of LNM in patients with deep submucosal invasive carcinoma (greater than 1000 μm) but without other risk factors was relatively low at 2.48%. In such scenarios, ESD may be a safer alternative than surgical treatment. For patients classified as medium risk for LNM, initial endoscopic treatment could be considered appropriate, with the possibility of additional surgery based on subsequent follow-up assessments. These findings indicate that the current guidelines for selecting initial treatment in CRC may need refinement, and an expansion of the indications for endoscopic treatment should be considered. For high-risk patients, coloproctectomy could be a more suitable treatment option, given its considerable LNM rate of 51.72%.

The current study presents several limitations. Firstly, as a retrospective, single-center study, it is subject to inherent selection bias, which may influence the results. Consequently, a large-scale, prospective study involving a multi-center cohort is necessary to validate the scoring table and refine the indications for endoscopic therapy in patients with early CRC. Secondly, our methodology included only patients who underwent surgical procedures, excluding those who received only ESD. This approach might introduce selection bias, but it is unlikely to substantially affect the clinical utility of our nomogram. Thirdly, while the bootstrap method was used for repeated sampling verification, external validation with a larger cohort from multiple medical centers is still needed. Moreover, the risk level assessment table requires comprehensive internal and external validation data.

Conclusions

In conclusion, we have established a nomogram incorporating age, depth of invasion, histologic grade, LVI, NI, and tumor budding to predict the risk of LNM in patients with early CRC. We have also introduced a comprehensive risk assessment scale. Together, this nomogram and risk assessment scale can serve as reliable and effective prediction tools, potentially enhancing treatment strategies for early CRC.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00384-024-04760-2.

Author contribution Jiahui Xu wrote the main manuscript text. Fan Yin and Yushuang Xu prepared Tables and Figures. Linlin Ren and Congcong Min contributed significantly to the translation. Peng Zhang and Mengyu Cao collected and analyzed the data. Xiaoyu Li reviewed the manuscript. Zibin Tian and Tao Mao helped perform the analysis with constructive discussion. All authors reviewed the manuscript.

Funding The work was completed with the support of the National Natural Science Foundation of China (No. 82273393) Association to Ren LL.

Data availability No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate This research study was conducted retrospectively from data obtained for clinical purposes and was reviewed and approved by the Ethics Committee of the Affiliate Hospital of Qingdao University (Reference Number QYFYWZLL28762). The procedures used in this study adhere to the tenets of the Declaration of Helsinki. The need for written informed consent was waived by the Ethics Committee of the Affiliate Hospital of Qingdao University due to the retrospective nature of the study.

Consent for publication Not applicable.

Competing interests The authors declare no competing interests.

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