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## ORIGINAL ARTICLE

# The Predictive Value of Lactate Dehydrogenase (LDH) for Lymphovascular Invasion in Endometrial Cancer: a Retrospective Cohort Study

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**SUMMARY**

***Background:*** **This study aims to explore the predictive value of serum lactate dehydrogenase (LDH) for lymphovascular invasion in endometrial cancer.**
***Methods:*** **A retrospective analysis was performed on a cohort of 147 patients diagnosed with endometrial cancer at Fujian Provincial Cancer Hospital between January 2018 and January 2020. The study focused on preoperative relevant test indicators, including lactate dehydrogenase (LDH) levels and the extent of postoperative lymphovascular invasion (LVI). Patients were stratified based on the degree of LVI, and intergroup differences were assessed using the chi-squared test ($\chi^2$). Stratification was conducted according to the median values of LDH, CA125, glucose (Glu), and Ki67. Survival analyses and comparisons of differences were executed using Kaplan-Meier survival curves and the log-rank test. Spearman's rank correlation coefficient was used to perform a correlation analysis between LDH and other biological indicators. Independent prognostic factors influencing patient outcomes were identified via multivariate Cox regression analysis. A prognostic nomogram was subsequently developed from the Cox regression results to estimate survival probabilities at 1, 2, and 4 years. Furthermore, the predictive efficacy of LDH was assessed through the construction of time-dependent receiver operating characteristic (ROC) curves. A p-value of less than 0.05 was deemed statistically significant.**
***Results:*** **The levels of serum lactate dehydrogenase (LDH), cancer antigen 125 (CA125), glucose (Glu), and Ki67 were significantly elevated in the group with lymphovascular invasion (LVI+) compared to the group without lymphovascular invasion (LVI-) ($p < 0.05$). Elevated LDH levels were inversely associated with patient survival log-rank $p < 0.05$), whereas no significant associations were observed between CA125, Glu, Ki67 levels, and patient survival. Spearman's correlation analysis revealed a significant correlation between LDH and Glu levels ($p < 0.05$). Cox regression analysis identified LDH as an independent prognostic factor, with elevated LDH levels increasing the risk of mortality by 2.120 times (95% confidence interval: 1.351 - 3.327, $p = 0.001$). The prognostic nomogram demonstrated that patients with lower total scores had higher probabilities of survival at 1, 2, and 4 years. Time-dependent receiver operating characteristic (ROC) analysis indicated that the predictive efficacy of LDH was limited, with area under the curve (AUC) values of 0.654, 0.653, and 0.719 for 1, 2, and 4 years, respectively.**
***Conclusions:*** **Lactate dehydrogenase (LDH) can serve as a potential biomarker for assessing lymphovascular metastasis and prognostic outcomes in endometrial cancer.**
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**KEYWORDS**

endometrial cancer, lactate dehydrogenase (LDH), lymphovascular invasion (LVI), predictive factor

## INTRODUCTION

Endometrial carcinoma (EC) ranks among the most prevalent malignant neoplasms affecting the female reproductive system globally, with an elevated incidence observed particularly in individuals with obesity and metabolic syndrome [1-4]. While patients diagnosed at an early stage often achieve favorable prognoses through surgical intervention combined with adjuvant therapy, approximately 20 - 30% of cases nonetheless experience tumor recurrence, attributed to lymphovascular invasion (LVI) and lymph node metastasis, resulting in an average 5-year survival rate of less than 50% for these patients [5-7]. Consequently, the identification and exploration of appropriate biomarkers to predict the occurrence and progression of LVI are of critical importance for the prevention and management of recurrence in endometrial carcinoma.

Lactate dehydrogenase (LDH), a pivotal enzyme in glycolysis, is frequently observed at elevated levels in association with increased tumor aggressiveness and decreased survival rates in various solid malignancies, including colorectal and lung cancers [8-9]. Numerous studies on gynecological tumors have demonstrated a positive correlation between serum LDH levels and the risk of peritoneal metastasis in ovarian cancer, as well as lymph node metastasis in cervical cancer [10-12]. However, its prognostic significance in endometrial cancer remains underexplored. Furthermore, empirical evidence suggests that PIK3CA mutations in endometrial cancer cells may enhance LDH release through activation of the PI3K/AKT/mTOR signaling pathway [13-15], indicating that LDH could potentially serve as a biomarker for the invasive phenotype associated with metabolic dysregulation in endometrial cancer.

This study uniquely employed a selection of multidimensional biomarkers, including those related to inflammation (e.g., neutrophil-to-lymphocyte ratio [NLR], fibrinogen-to-albumin ratio [FAR]), tumor burden (e.g., cancer antigen 125 [CA125], Ki67), and glycolysis (e.g., lactate dehydrogenase [LDH]), to comprehensively assess their predictive efficacy for lymphovascular invasion in endometrial cancer. Utilizing the Cox regression model, a prognostic nomogram was developed to overcome the limitations associated with single biomarkers. The results of this study provide a scientific foundation for identifying high-risk patients with endometrial cancer and for the development of dynamic monitoring strategies.

## MATERIALS AND METHODS

### Subjects

A retrospective inclusion of 147 patients with endometrial cancer who underwent surgical treatment at Fujian Provincial Cancer Hospital from January 2018 to January 2020. Inclusion criteria: 1) Diagnosed with endometrial cancer by postoperative histopathological examination (according to WHO 2020 classification standards); 2) Complete data on serum LDH and all covariates (age, BMI, CA125, etc.) within 7 days before surgery; 3) Underwent standard surgical staging for endometrial cancer (at least including total hysterectomy + bilateral salpingo-oophorectomy + pelvic lymphadenectomy/ sampling) and postoperative paraffin specimens that could clearly assess the status of lymphovascular invasion (LVI). Exclusion criteria: 1) Presence of other malignant tumors; 2) Preoperative intervention: received radiotherapy, chemotherapy, or hormone therapy before surgery; 3) Missing data or insufficient specimen quality.

This study was approved by the Ethics Committee of Fujian Provincial Cancer Hospital (Ethics approval number: K025-159-01), and informed consent was waived.

### Observation indicators

Age, Body Mass Index (BMI), serum lactate dehydrogenase (LDH), carbohydrate antigen 125 (CA125), human epididymis protein 4 (HE4), neutrophil ratio (NEU), lymphocyte ratio (LYM), plasma fibrinogen (FIB), serum albumin (ALB), blood glucose (Glu), triglyceride (TG) values within 7 days before surgery; postoperative serum Ki67 value, lymph node metastasis, and lymphovascular invasion (LVI) degree (see Table 1).

### Follow-up

The survival period is defined from the date of diagnosis to the date of death or the last follow-up cutoff date. Measured in months, the first follow-up occurs one month post-surgery and continues until the patient's death or the end of the study (January 2025). The follow-up method investigates the patient's survival status through reviewing pathological data and methods such as phone calls and ID checks.

### Statistical analysis

All statistical analyses were performed using SPSS software version 29.0 (IBM SPSS Statistics for Windows; IBM Corp., Armonk, NY, USA) and R language version 4.2.1 (R Foundation, Vienna, Austria). Non-normally distributed variables such as LDH levels and Ki67 index were analyzed using the Mann-Whitney U test, while normally distributed variables (e.g., age, BMI) were analyzed using independent samples *t*-test. For survival data analysis, Kaplan-Meier curves were constructed, and statistical analysis was performed using the log-rank test. Univariate and multivariate Cox

**Table 1. Variable definitions.**

| Variable | Definition and Measurement |
|---|---|
| LDH | Preoperative serum lactate dehydrogenase (U/L), measured by enzymatic UV assay [16] |
| Lymphovascular Invasion (LVI) | Postoperative pathology-confirmed tumor cells in endothelial-lined spaces (CD34+/D2-40+), independently reviewed [17,18] |
| Age | Age (years) at surgery. |
| BMI | Body mass index (kg/m²), calculated from preoperative height and weight. |
| FIGO Stage (2018) | 2018 FIGO staging: IA (myometrial invasion < 50%), IB (≥ 50%), II (cervical stromal involvement), III (regional spread), IV (distant metastasis) [19,20] |
| Grade | WHO 2020 criteria: G1 (well-differentiated), G2 (moderately differentiated), G3 (poorly differentiated) [19-20] |
| Ki67 Index (%) | Percentage of MIB-1 antibody-positive cells via immunohistochemistry; ≥ 30% indicates high proliferation [21,22] |
| NEU | Preoperative neutrophil count (× 10⁹/L), measured via complete blood count |
| LYM | Preoperative lymphocyte count (× 10⁹/L), measured via complete blood count |
| FIB | Preoperative fibrinogen (g/L), measured via coagulation panel |
| ALB | Preoperative albumin (g/L), measured via biochemical assay |
| Glu | Preoperative fasting blood glucose (mmol/L), measured via biochemical assay |
| TG | Preoperative fasting triglycerides (mmol/L), measured via biochemical assay |
| CA125 | Preoperative serum CA125 (U/mL), measured by electrochemiluminescence [20] |
| HE4 | Preoperative serum HE4 (pmol/L), measured by electrochemiluminescence [20] |
| NLR | Neutrophil-to-lymphocyte ratio (NEU ÷ LYM) [23] |
| FAR | Fibrinogen-to-albumin ratio (FIB ÷ ALB) [23] |
| TyG Index | Calculated as ln [TG (mmol/L) × Glu (mmol/L)/2] [20] |

**Table 2. Variable assignment of the Cox proportional risk regression model.**

| Variables | Assignment |
|---|---|
| LDH (U/L) | 0 = < 175, 1 = ≥ 175 |
| CA125 (U/mL) | 0 = < 26.5, 1 = ≥ 26.5 |
| Glu (mmol/L) | 0 = < 5.4, 1 = ≥ 5.4 |
| Ki67 (%) | 0 = < 60, 1 = ≥ 60 |
| FIGO Stage | 0 = stage I and stage II, 1 = stage III and stage IV |
| Grade | 0 = G1, 1 = G2 and G3 |

regression analyses were employed to identify independent risk factors associated with endometrial cancer and to create prognostic nomograms; the distribution of related variables is shown in (Table 2). The association between LDH and other variables was assessed using Spearman's rank correlation analysis. Continuous variables are presented as mean ± standard deviation or median (IQR), and categorical variables are presented as frequency (percentage). All analyses were conducted using two-sided tests, with $p < 0.05$ considered statistically significant.

## RESULTS

### Comparison of clinical traits and biomarkers by lymphovascular invasion status in endometrial cancer patients

This study encompassed a cohort of 147 patients diagnosed with endometrial cancer, categorized based on the extent of lymphovascular invasion (LVI). The cohort was divided into two groups: 66 patients in the LVI- group and 81 patients in the LVI+ group. No statistically significant differences were observed between the groups concerning age, body mass index (BMI), neutro-

**Table 3. Analysis of the relationship between LVI, and the clinical characteristics of EC patients.**

| Characteristics | LVI- | LVI+ | p-value |
|---|---|---|---|
| n | 66 | 81 | |
| Age (years), mean ± sd | 57.848 ± 7.9696 | 56.272 ± 6.8228 | 0.198 |
| BMI (kg/m²), mean ± sd | 24.274 ± 4.3424 | 24.43 ± 3.654 | 0.814 |
| LDH (U/L), mean ± sd | 181.27 ± 59.524 | 234.12 ± 182.43 | 0.016 |
| CA125 (U/mL), mean ± sd | 39.814 ± 6.482 | 108.5 ± 83.38 | 0.035 |
| HE4 (pmol/L), mean ± sd | 77.665 ± 81.387 | 80.346 ± 131.2 | 0.906 |
| NEU (× 10⁹/L), mean ± sd | 4.0394 ± 2.1966 | 4.243 ± 2.6087 | 0.615 |
| LYM (× 10⁹/L), mean ± sd | 1.8482 ± 0.5241 | 1.9221 ± 0.58314 | 0.425 |
| NLR, mean ± sd | 2.389 ± 1.7372 | 2.4107 ± 1.771 | 0.941 |
| FIB (g/L), median (IQR) | 2.92 (2.6875, 3.4075) | 3.12 (2.65, 3.8) | 0.248 |
| ALB (g/L), median (IQR) | 42.5 (39.825, 44.95) | 41.6 (40.2, 43.1) | 0.070 |
| FAR, median (IQR) | 0.070082 (0.061872, 0.081198) | 0.077596 (0.06401, 0.092766) | 0.076 |
| Glu (mmol/L), median (IQR) | 5.6 (5.115, 6.255) | 5.32 (4.82, 5.61) | 0.021 |
| TG (mmol/L), median (IQR) | 1.29 (1.0325, 1.9) | 1.31 (1, 1.65) | 0.602 |
| TyG, median (IQR) | 0.047232 (0.036516, 0.078531) | 0.050879 (0.037833, 0.067747) | 0.969 |
| Ki67 (%), median (IQR) | 50 (30, 63.75) | 70 (45, 80) | 0.001 |
| Grade, n (%) | | | |
| G1 | 8 (5.4%) | 39 (26.5%) | < 0.001 |
| G2 | 30 (20.4%) | 39 (26.5%) | |
| G3 | 28 (19%) | 3 (2%) | |
| FIGO Stage, n (%) | | | |
| I | 17 (11.6%) | 14 (9.5%) | < 0.001 |
| II | 7 (4.8%) | 33 (22.4%) | |
| III | 42 (28.6%) | 23 (15.6%) | |
| IV | 0 (0%) | 11 (7.5%) | |

phil count (NEU), lymphocyte count (LYM), fibrinogen (FIB), triglycerides (TG), and other measured indicators ($p > 0.05$). However, significant differences were identified in lactate dehydrogenase (LDH), cancer antigen 125 (CA125), glucose (Glu), Ki67 expression, tumor grade, and the International Federation of Gynecology and Obstetrics (FIGO) stage ($p < 0.05$). Specifically, the LDH level in the LVI+ group was 234.12 ± 182.43 U/L, significantly exceeding that of the LVI-group, which was 181.27 ± 59.524 U/L ($p = 0.016$). The CA125 level in the LVI+ group was 108.5 ± 83.38 U/mL, higher than the LVI-group's level of 39.814 ± 6.482 U/mL ($p = 0.035$). Conversely, the Glu level in the LVI-group was 5.6 mg/dL (IQR: 5.115 - 6.255), which was higher than that in the LVI+ group at 5.32 mg/dL (IQR: 4.82 - 5.61) ($p = 0.021$). The Ki67 expression level in the LVI+ group was 70% (IQR: 45% - 80%), surpassing that of the LVI- group, which was 50% (IQR: 30% -63.75%) ($p = 0.001$) (see Table 3).

**Analysis of biomarkers grouping's impact on patient's survival**

Based on the median values of LDH, CA125, Glu, and Ki67, patients were categorized into high and low expression groups. The analysis revealed that the overall survival rate for patients in the low LDH expression group was significantly greater than that of the high LDH expression group (Log-rank $p < 0.001$) (refer to Figure 1A). Throughout the follow-up period, the survival probability for patients in the low expression biomarker group consistently exceeded that of the high expression group across all time points. This trend was particularly evident during the early follow-up period (0 - 20 months), where the disparity in survival rates between the two groups was more pronounced and statistically significant ($p < 0.05$). These findings suggest that LDH levels may serve as an independent prognostic factor, with elevated LDH expression being inversely associated with survival outcomes. Conversely, the expression levels of CA125 (Log-rank $p = 0.273$) (Figure 1B), Glu (Log-rank $p = 0.915$) (Figure 1C), and Ki67

**Table 4. Univariate and multivariate analysis of factors affecting vascular invasion in endometrial cancer.**

| Characteristics | Total (n) | Univariate analysis | | Multivariate analysis | |
|---|---|---|---|---|---|
| | | Hazard ratio (95% CI) | p-value | Hazard ratio (95% CI) | p-value |
| LDH (U/L) | 147 | | | | |
| 0 | 73 | Reference | | Reference | |
| 1 | 74 | 2.239 (1.429 - 3.508) | < 0.001 | 2.120 (1.351 - 3.327) | 0.001 |
| CA125 (U/mL) | 147 | | | | |
| 1 | 75 | Reference | | | |
| 0 | 72 | 1.204 (0.778 - 1.862) | 0.405 | | |
| Glu (mmol/L) | 147 | | | | |
| 0 | 70 | Reference | | | |
| 1 | 77 | 0.894 (0.577 - 1.385) | 0.616 | | |
| Ki67 (%) | 147 | | | | |
| 1 | 75 | Reference | | | |
| 0 | 72 | 1.105 (0.715 - 1.708) | 0.654 | | |
| FIGO Stage | 147 | | | | |
| 1 | 53 | Reference | | Reference | |
| 0 | 94 | 0.435 (0.279 - 0.679) | < 0.001 | 0.599 (0.333 - 1.076) | 0.086 |
| Grade | 147 | | | | |
| 1 | 46 | Reference | | Reference | |
| 0 | 101 | 0.460 (0.296 - 0.715) | < 0.001 | 0.659 (0.369 - 1.174) | 0.157 |

(Log-rank p = 0.136) (Figure 1D) did not exhibit a statistically significant impact on survival rates.

**Correlation analysis of LDH with Glu, CA125, and Ki67 markers**

The Spearman's correlation analysis reveals a weak negative correlation between LDH levels and glucose (Glu), with a correlation coefficient of -0.175 and a p-value of 0.034, indicating statistical significance ($p < 0.05$) as illustrated in Figure 2A. Conversely, a weak positive correlation is observed between LDH levels and CA125 levels, with a correlation coefficient of 0.118 and a p-value of 0.157, which does not reach statistical significance (Figure 2B). Additionally, no correlation is detected between LDH levels and Ki67 expression levels, as evidenced by a correlation coefficient of -0.016 and a p-value of 0.846 (Figure 2C).

**Identifying key prognostic factors: Independent risk assessment of LDH levels**

The findings from both univariate and multivariate Cox regression analyses indicate that lactate dehydrogenase (LDH) level serves as an independent prognostic factor influencing patient outcomes. Specifically, the risk of mortality in patients with elevated LDH levels is 2.120 times greater than in those with lower LDH levels (95% confidence interval: 1.351 - 3.327, p = 0.001). Conversely, variables such as CA125, glucose (Glu), FIGO stage, Ki67, and tumor grade did not emerge as independent prognostic indicators in the multivariate analysis, as detailed in (Table 4).

**Development of a prognostic nomogram and survival prediction using multifactor Cox regression**

Utilizing the results from the Cox regression analysis, a prognostic nomogram was developed to estimate the survival probabilities of patients at 1, 2, and 4 years. The construction of this nomogram incorporated the following variables: lactate dehydrogenase (LDH), cancer antigen 125 (CA125), glucose (Glu), International Federation of Gynecology and Obstetrics (FIGO) Stage, Ki67, and Grade. The findings indicated that patients with a lower total score (e.g., 0 to 100 points) exhibited a higher 1-year survival probability, approximately 0.9, whereas those with a higher total score (e.g., 200 to 300 points) demonstrated a reduced 1-year survival probability, approximately 0.6. This trend was consistent for the 2-year survival probability, where patients with lower total scores had a probability close to 0.9, and those with higher scores had a probability near 0.6. At 4 years, patients with lower total scores maintained a higher survival probability, around 0.8, while those with higher scores experienced a markedly decreased survival probability, approximately 0.3 (refer to Figure 3).

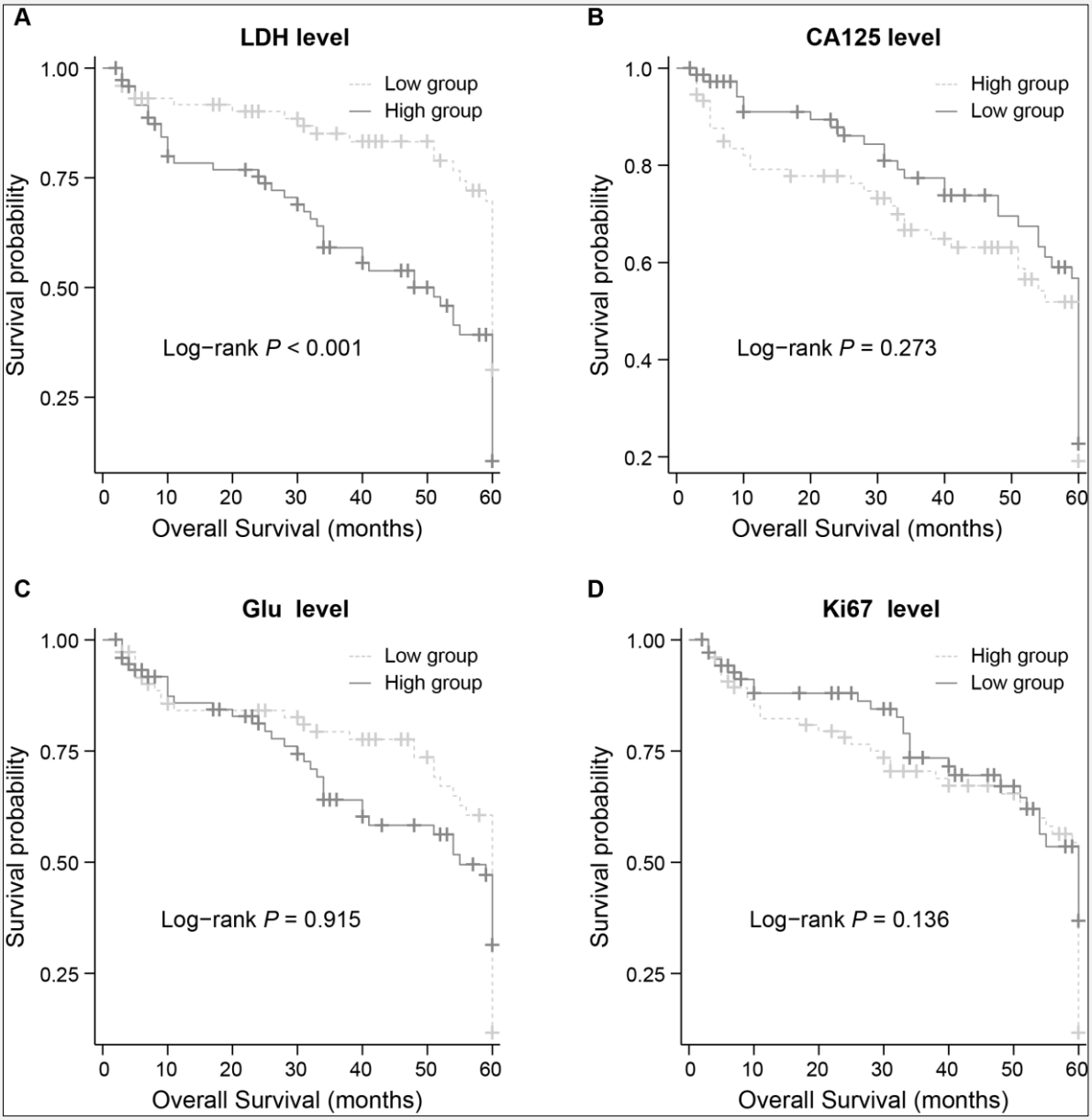


**Figure 1. The link between biomarker levels and survival rate.**

**A The relationship between LDH levels and overall survival rate. B The relationship between CA125 levels and overall survival rate. C The relationship between Glu levels and overall survival rate. D The relationship between Ki67 levels and overall survival rate.**

### Time-dependent analysis of LDH levels in survival prognostication

The utility of lactate dehydrogenase (LDH) levels in forecasting patient survival at 1, 2, and 4 years was assessed using time-dependent receiver operating characteristic (ROC) analysis. The analysis revealed that the area under the curve (AUC) for predicting 1-year survival was 0.654, for 2-year survival was 0.653, and for 4-year survival was 0.719 (refer to Figure 4). These findings suggest that while LDH levels possess some prognostic value in predicting patient survival over a 1 to 4-year period, their predictive efficacy is limited. Consequently, LDH may be considered as an auxiliary indicator in survival prediction models.

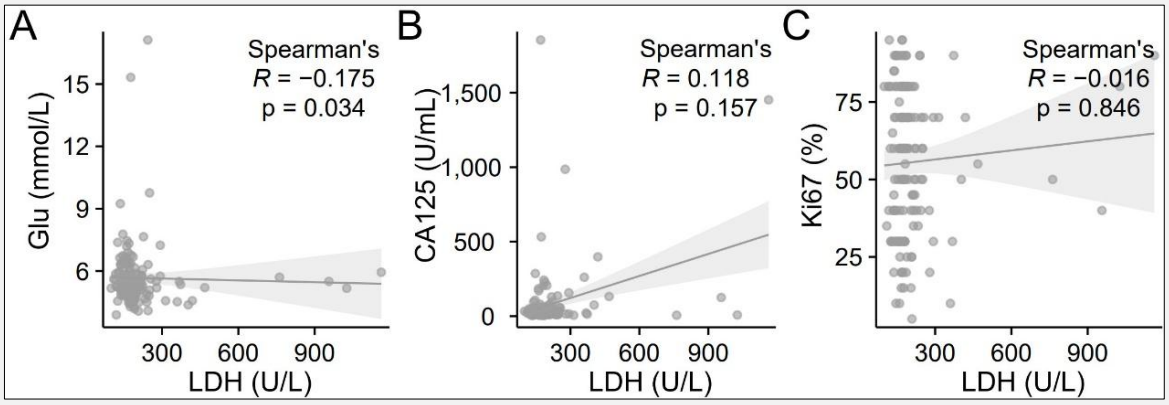


**Figure 2. The association of LDH levels with Glu, CA125, and Ki67 biomarkers.**

**A The relationship between LDH levels and Glu levels. B The relationship between LDH levels and CA125 levels. C The relationship between LDH levels and Ki67 levels.**

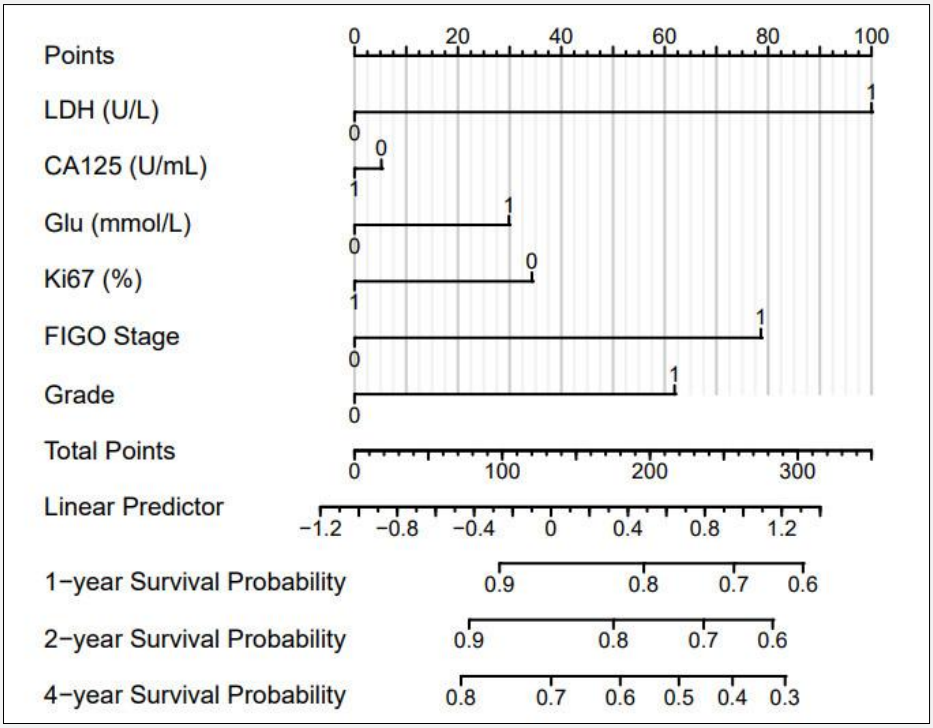


**Figure 3. This nomogram predicts 1-year, 2-year, and 4-year survival probabilities using various biomarkers and clinical traits.**

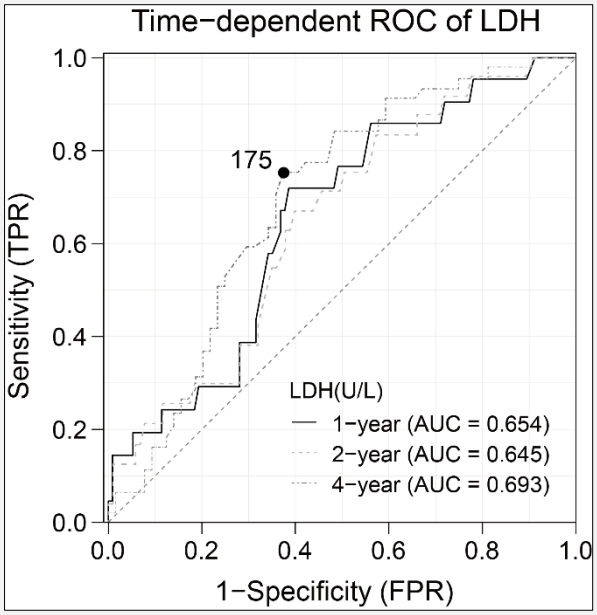


**Figure 4. Time-dependent ROC curves for LDH levels in survival prediction at different time points (1 year, 2 years, 4 years).**

## DISCUSSION

This study examines endometrial cancer (EC), a prevalent malignant tumor among women [4]. Through a retrospective analysis of clinical and biomarker data from 147 patients diagnosed with EC, this research systematically evaluates preoperative lactate dehydrogenase (LDH) levels for their predictive value regarding lymphovascular invasion (LVI) and disease prognosis, marking the first investigation of its kind. The findings reveal that the levels of LDH, CA125, and Ki67 are significantly elevated in the LVI-positive group compared to the LVI-negative group, with LDH identified as an independent risk factor influencing overall survival (hazard ratio = 2.120, p = 0.001), thereby addressing a critical gap in the existing literature. From a clinical practice standpoint, measuring LDH levels offers physicians a straightforward and effective tool for assessing patient prognosis. Elevated LDH levels are associated not only with the occurrence of vascular metastasis but also with a marked reduction in patient survival rates. This suggests that LDH can serve as a vital prognostic indicator, assisting clinicians in more accurately stratifying patient risk and optimizing treatment strategies [24-26]. Consequently, it enables the identification of high-risk patients who may benefit from more aggressive therapeutic interventions.

Lactate dehydrogenase (LDH) demonstrates a significant biological association with lymphovascular invasion (LVI). As a pivotal enzyme in glycolysis, elevated LDH levels generally indicate heightened metabolic activity within tumor cells and a hypoxic microenvironment [27]. This study identified a notable elevation in LDH levels within the LVI-positive group, suggesting that tumor cells may acquire an invasive phenotype by augmenting their glycolytic capacity, exemplified by the Warburg effect, thereby facilitating vascular invasion. This finding aligns with previous research indicating that increased LDH levels correlate with enhanced metastatic potential across various solid tumors, including melanoma and lung cancer [28]. Additionally, LDH may contribute to angiogenesis and immune evasion by modulating lactate accumulation in the tumor microenvironment, thus expediting vascular metastasis. Importantly, this study is the first to establish an independent correlation between LDH and LVI in endometrial cancer, proposing that LDH could serve as an auxiliary marker for the preoperative evaluation of vascular metastasis risk.

The interaction between lactate dehydrogenase (LDH) and other biomarkers has garnered significant attention in tumor research in recent years [29]. Notably, elevated LDH levels exhibit a certain correlation with alterations in glucose metabolism (R = -0.175, p = 0.034). LDH is integral to glycolysis and gluconeogenesis, facilitating the reversible conversion between lactate and pyruvate. Consequently, variations in LDH levels may indicate changes in cellular metabolic status, particularly con-

cerning energy metabolism. Nonetheless, these correlations are relatively weak, implying that the prognostic value of LDH may derive more from its direct reflection of tumor biological behavior rather than merely serving as an indicator of inflammation or metabolic disorders. Therefore, the interpretation of LDH in clinical applications for tumors should be approached with caution, and further research is warranted.

The results of the survival analysis demonstrate that patients in the high lactate dehydrogenase (LDH) group exhibit a significantly lower survival rate compared to those in the low LDH group (Log-rank $p < 0.05$). Furthermore, in the Cox multivariate model analysis, LDH emerged as an independent prognostic factor. This finding aligns with existing research on ovarian and cervical cancers, reinforcing the association between elevated LDH levels and increased tumor burden and aggressiveness [30]. It is important to note that, although traditional tumor markers such as CA125 and Ki67 are frequently employed in survival prediction [31,32], this study indicates their predictive value is limited ($p > 0.05$), potentially due to the small sample size or insufficient follow-up duration. Additionally, the predictive efficacy of LDH improves over time (4-year AUC = 0.719), suggesting that LDH is more appropriate as an auxiliary indicator for medium- to long-term prognostic assessment.

In clinical applications, prognostic assessment encounters numerous challenges yet exhibits significant potential [33]. The prognostic nomogram developed in this study incorporates various key indicators, including lactate dehydrogenase (LDH) and the International Federation of Gynecology and Obstetrics (FIGO) staging, and demonstrates predictive capability for patients' 1- to 4-year survival probabilities. Nevertheless, the model's performance, with AUC values ranging from 0.653 to 0.719, requires further enhancement. Consequently, future research should integrate additional molecular markers, such as POLE mutations and microsatellite instability [34], to improve the model's accuracy. Moreover, given that LDH is a pan-tumor marker with limited specificity, a comprehensive assessment should be conducted in conjunction with imaging examinations or molecular typing. For instance, in patients with elevated LDH levels, it is advisable to prioritize advanced imaging studies (such as PET-CT) to facilitate early detection of potential occult metastases. These strategies will enhance the clinical applicability of prognostic models, thereby better supporting the individualized treatment of patients.

## CONCLUSION

This study corroborates the significant association between serum lactate dehydrogenase (LDH) levels and both lymphovascular invasion and poor prognosis in endometrial cancer, demonstrating that its predictive value is independent of conventional clinicopathological factors. While the predictive efficacy of LDH in isolation is limited, its integration with other markers as a cost-effective and readily accessible blood biomarker holds promise for enhancing risk stratification and postoperative monitoring strategies in patients. Future research should aim to elucidate the biological mechanisms underlying LDH's role in the metastasis of endometrial cancer and investigate its potential as a therapeutic target.

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**Availability of Data and Materials:**
The datasets used and/or analyzed during the present study are available from the corresponding author on reasonable request.

**Ethics Approval and Consent to Participate:**
The present study was approved by the Ethics Committee for Research and New Technologies of Fujian Provincial Cancer Hospital, and written informed consent was provided by all patients prior to the study start. All procedures were performed in accordance with the ethical standards of the Institutional Review Board and the Declaration of Helsinki, and its later amendments or comparable ethical standards.

**Declaration of Interest:**
The authors have no conflicts of interest to declare.

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