

Effect of dynamic platelet-to-lymphocyte ratio on the prognosis of patients with esophageal squamous cell carcinoma receiving chemoradiotherapy

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Abstract

Systemic inflammatory load affects the long-term developmental outcomes in patients with malignancy. The purpose of this study was to investigate the effect of the dynamic levels of platelet-to-lymphocyte ratio (PLR) at different treatment stages on the prognosis of patients with esophageal squamous cell carcinoma (ESCC) undergoing chemoradiotherapy. This study included 168 patients who received chemoradiotherapy between 2012 and 2018. PLR levels at different treatment stages were calculated based on blood test results. The association between PLR and overall survival (OS) was determined using the Kaplan–Meier method and Cox proportional regression models. The cutoff values of PLR before and after treatment of 168 patients with ESCC were 195.7 and 403.6, respectively. The 5-year OS rates of patients in the low and high pre-PLR groups were 42.1% and 21.7%, respectively. The overall 5-year OS rate of all patients was 27.1%. Multivariate analysis results showed that patient age (hazard ratio [HR] = 1.736; 95% confidence interval (CI) = 1.129–2.669; P = .012), alcohol consumption (HR = 1.622; 95%Cl = 1.050–2.508; P = .029), T stage (HR = 12.483; 95%Cl = 3.719–41.896; P < .001), pre-PLR (HR = 1.716; 95%Cl = 1.069–2.756; P = .025), post-PLR (HR = 1.664; 95%Cl = 1.106–2.503; P = .015) were independent factors of the prognosis of patients with ESCC undergoing chemoradiotherapy.

Abbreviations: CI = confidence interval, CRT = chemoradiotherapy, ESCC = esophageal squamous cell carcinoma, HR = hazard ratio, OS = overall survival, PLR = platelet-to-lymphocyte ratio, RT = radiotherapy.

Keywords: esophageal squamous cell carcinoma, lymphocytes, platelets, prognosis

1. Introduction

Esophageal cancer is a malignant tumor with high incidence and mortality rates, with over 600,000 new cases globally each year.^[1] In China, it ranks sixth and fifth in terms of incidence and mortality, respectively, among all malignant tumors.^[2] Furthermore, the pathological types of esophageal cancer differ significantly among different regions, with adenocarcinoma being more prevalent in European and American countries, while squamous cell carcinoma in China.^[3]

Surgical treatment is the primary therapeutic approach for early-stage esophageal cancer, which can significantly improve the short-term survival rate of patients.^[4] For those who are unwilling or unable to undergo surgery, radical chemoradiotherapy (CRT) is the standard alternative.^[5–7] Increasing evidence suggests that systemic inflammatory responses play a vital role in the occurrence and development of tumors and are closely associated with tumor proliferation, invasion, and metastasis.^[8] Changes in tumor-related inflammatory cells reflect the degree of the body's inflammatory response to the tumor: the higher the levels of inflammation, the poorer the prognosis.^[9,10]

In recent years, clinical trials have reported that systemic inflammatory markers, such as the systemic

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The datasets generated during and/or analyzed during the current study are not publicly available, but are available from the corresponding author on reasonable request.

This study was approved by the Ethics Committee and Institutional Review Board of Guangyuan Central Hospital (No. GYZXLL202310) and was conducted in accordance with the principles of the Helsinki Declaration and its amendments. In addition, they have written informed consent from the patients for the inclusion of their medical and treatment history within this work. We confirm that the data has been anonymized and conducted a confidential analysis.

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immune-inflammation index,^[11] advanced lung cancer inflammation index (ATI),^[12] neutrophil-to-lymphocyte ratio (NLR)^[13] and platelet-to-lymphocyte ratio (PLR),^[14] are important prognostic indicators for patients with malignant tumors such as colorectal cancer, lung cancer, and cervical cancer.

In particular, PLR can be referred to in predicting the prognosis of patients with esophageal squamous cell carcinoma (ESCC), most studies on the predictive role of PLR tend to focus on the impact of PLR levels before treatment on prognosis.^[14-16] However, there are few studies on the impact of changes in PLR levels in different treatment stages on the prognosis of cancer patients. At the same time, its use is controversial because its accuracy and optimal cutoff value remain unclear.

Therefore, this study retrospectively analyzed the clinicopathological data of 168 ESCC patients undergoing CRT, in order to explore the influence of dynamic changes in PLR at different treatment stages (before and after treatment) on the prognosis of ESCC patients undergoing radiotherapy (RT) and chemotherapy. The accuracy of PLR in predicting prognosis was also evaluated through subgroup analysis.

2. Materials and methods

2.1. Study object

This retrospective study was approved by the Ethics Committee and Institutional Review Board of Guangyuan Central Hospital (No. GYZXLL202310). A total of 168 patients with ESCC who received CRT from January 2012 to December 2018 were included. The 7th edition of the American Joint Committee on Cancer TNM staging was adopted. The inclusion criteria were as follows: age > 18 years; KPS score \geq 70 points; pathologically confirmed squamous cell carcinoma of the esophagus; patients receiving curative RT; patients with laboratory and clinical data; and no distant tumor metastasis. The exclusion criteria included previous surgery history, incomplete follow-up information, and any other primary tumors aside from esophageal cancer.

2.2. Treatment

Three-dimensional conformal radiotherapy or intensitymodulated RT was performed. Bilateral lung V20 \leq 20%, bilateral lung V5 < 50%, heart V30 \leq 30%, and the maximum spinal cord dose \leq 45.0 Gy. Simultaneous chemotherapy was given at the same time, using platinum-based combined chemotherapy for 1 to 5 cycles: (1) paclitaxel + cisplatin, (2) paclitaxel + carboplatin, and (3) docetaxel + cisplatin. For patients who cannot tolerate intravenous chemotherapy, 1–3 cycles of oral chemotherapy with S-1/capecitabine are given.

2.3. Observation indicators and follow-up

A follow-up evaluation was conducted every 3 months in the first year, then every 6 months in the next 2 years, and then at the end of each year or until death. All patients were followed up through outpatient examinations and telephone calls until December 2022.

2.4. PLR calculation method

PLR = platelet/lymphocyte, pretreatment PLR is the blood test index 1 week before treatment; mid-treatment PLR (mid-PLR)



Figure 1. Pre-PLR using the Log-rank test to calculate optimal stratification cutoffs for continuous covariates.

is the lowest level of blood test index during the treatment process; posttreatment PLR (Post-PLR) is the blood test index 1 week after treatment.

2.5. Statistical analysis

R 4.2.1 software was used for statistical analysis. The continuous variables conforming to the normal distribution were compared by t test or analysis of variance, and the χ 2 test was used to compare count data. Log-rank test and optimal stratification were performed to determine the optimal cutoff value of continuous covariate PLR.^[17,18] The best cutoff value of PLR was used as reference in dividing the patients into the low PLR and high PLR groups (Figs. 1 and 2). For survival analysis, the Kaplan– Meier method and Log-rank test were performed. For univariate and multivariate analyses, Cox risk model was used. Test level α was 0.05, and P < .05 was set for statistical significance.

3. Results

3.1. Patient and tumor characteristics

The median age of the 168 patients was 63 years (34–86 years), including 131 males (78.0%) and 37 females (22.0%). Of the 168 patients, patients (57.7%) had smoking history, while 71 (42.3%) did not have smoking history; 91 (54.2%) had drinking history, while 77 patients (45.8%) did not have drinking history. A total of 68 patients (40.5%) had a tumor length < 5 cm, while 100 (59.5%) had a tumor length \ge 5 cm. 6 patients (4.2%) were stage II, while 137 (95.8%) were stage III. In terms of RT dose, 26 patients (15.4%) received < 60 Gy, 142 (84.6%) received \ge 60 Gy, 142 patients (84.6%) received

chemotherapy, while 26 (15.4%) did not receive chemotherapy (Table 1). With regard to the changes in PLR levels before and after treatment, results of the paired analysis showed that the levels of Post-PLR in patients significantly decreased to varying degrees compared with those before treatment (P < .05, Fig. 3). The distribution of background variables for survival stratification is shown in Table 2. Compared with the patients who died, the surviving patients smoked and drank less, had shorter tumor length, were in earlier T stage, and had lower PLR, with significant differences in T stage, pre-PLR, and post-PLR Lower (P < .05, Table 1).

3.2. Univariate and multivariate analyses

Results of the univariate analysis showed that tumor length, T stage, N stage, pre-PLR, and post-PLR could predict the prognosis of patients with ESCC (all P < .05, Table 2). On multivariate analysis, age (hazard ratio [HR] = 1.736; 95% confidence interval (CI) = 1.129–2.669; P = .012), drinking (HR = 1.622; 95% CI = 1.050–2.508; P = .029), T stage (HR = 12.483; 95% CI = 3.719–41.896; P < .001), pre-PLR (HR = 1.716; 95% CI = 1.069–2.756; P = .025), post-PLR (HR = 1.664; 95% CI = 1.106–2.503; P = .015) were independent factors of the prognosis of patients with ESCC (Table 2).

3.3. Demographic and clinical characteristics of cancer patients stratified by PLR at different treatment stages

Using the Log-rank test, the optimal stratification was used to resolve the optimal cutoff value of the continuous covariate,





 Table 1

 Clinicopathological features of 168 patients with ESCC.

Characteristics	Ν	Alive (n = 70)	Death (n = 98)	P value
Gender, n (%)				.176
Female	37 (22.0%)	19 (11.3%)	18 (10.7%)	
Male	131 (78.0)	51 (30.4%)	80 (47.6%)	
Age (years), n (%)		- (.249
<65	92 (54.8%)	42 (25%)	50 (29.8%)	
≥65	76 (45.2%)	28 (16.7%)	48 (28.6%)	
KPS score, n (%)	((,.)	()	.169
70	5 (3%)	0 (0%)	5 (3%)	
80	68 (40.5%)	27 (16.1%)	41 (24.4%)	
90	94 (56.0%)	43 (25.6%)	51 (30.4%)	
100	1 (0.6%)	0 (0%)	1 (0.6%)	
Smoking history, n (%)	1 (0.070)	0 (070)	1 (0.070)	.444
No	71 (42.3%)	32 (19%)	39 (23.2%)	
Yes	97 (57.7%)	38 (22.6%)	59 (35.1%)	
Alcohol history, n (%)	01 (01.170)	00 (22.070)	00 (00.170)	.063
No	77 (45.8%)	38 (22.6%)	39 (23.2%)	.000
Yes	91 (54.2%)	32 (19%)	59 (35.1%)	
Location, n (%)	01 (04.270)	02 (1070)	00 (00.170)	.459
Cervical	6 (3.6)	3 (1.8%)	3 (1.8%)	.100
Upper thoracic	34 (20.2%)	16 (9.5%)	18 (10.7%)	
Middle thoracic	56 (33.3%)	22 (13.1%)	34 (20.2%)	
Lower thoracic	64 (38.1%)	28 (16.7%)	36 (21.4%)	
Abdomina	8 (4.8%)	1 (0.6%)	7 (4.2%)	
Tumour length (cm), n (%)	0 (4.070)	1 (0.070)	7 (7.270)	.034
<5	68 (40.5%)	35 (20.8%)	33 (19.6%)	.004
≥5	100 (59.5%)	35 (20.8%)	65 (38.7%)	
T-stage, n (%)	100 (00.070)	55 (20.070)	00 (00.770)	<.001
T2	22 (13.1%)	19 (11.3%)	3 (1.8%)	<.001
T3	77 (45.8%)	34 (20.2%)	43 (25.6%)	
T4	69 (41.1%)	17 (10.1%)	52 (31%)	
N-stage, n (%)	09 (41.170)	17 (10.170)	52 (5170)	.130
NO	6 (3.6%)	5 (3%)	1 (0.6%)	.150
N1	54 (32.1%)	23 (13.7%)	31 (18.5%)	
N2	79 (47.0%)	23 (13.7 %) 33 (19.6%)	46 (27.4%)	
N3	29 (17.3%)	9 (5.4%)	20 (11.9%)	
RT dose (Gy), n (%)	29 (17.370)	9 (3.4 %)	20 (11.970)	.349
<60	26 (15.4%)	13 (7.7%)	13 (7.7%)	.349
<00 ≥60	142 (84.6%)	57 (33.9%)	85 (50.6%)	
	142 (04.0%)	07 (33.9%)	00 (00.0%)	710
Chemotherapy, n (%) No	0C (1E 40/)	10 (69/)	16 (0 50()	.718
	26 (15.4%)	10 (6%)	16 (9.5%)	
Yes Dro DI P n (9()	142 (84.6%)	60 (35.7%)	82 (48.8%)	.022
Pre-PLR, n (%)	100 (00 EN/)	61 (26 20/)	71 (40 20/)	.022
<195.7	132 (88.5%)	61 (36.3%)	71 (42.3%)	
≥195.7	36 (21.5%)	9 (5.4%)	27 (16.1%)	000
Post-PLR, n (%)		40 (00 00()		.032
<403.6	99 (59.0%)	48 (28.6%)	51 (30.4%)	
≥403.6	69 (41.0%)	22 (13.0%)	47 (28%)	

ESCC = esophageal squamous cell carcinoma, PLR = platelet-to-lymphocyte ratio, RT = radiotherapy.

and the data on both sides of the PLR value were distinguished to obtain the best difference. Using this method, the pre-PLR cutoff value was 195.7, and the patients were divided into low pre-PLR group (PLR < 195.7; n = 132) and high pre-PLR group (PLR \geq 195.7; n = 36). Pre-PLR was associated with sex, T stage, and N stage (all *P* < .05, Fig. 1, Table 3). Post-PLR cutoff value was 403.6: low post-PLR group (PLR < 403.6; n = 132) and high post-PLR group (PLR \geq 403.6; n = 36; Fig. 2, Table 4).

3.4. PLR at different treatment stages and survival prognosis

The pre-PLR value was 153.5 ± 88.9 (low pre-PLR group: PLR < 195.7, n = 132, 78.5%; high pre-PLR group: PLR ≥ 195.7 , n = 36, 21.5%). The mid-PLR value was 474.9 \pm 402.3 (low mid-PLR group: PLR < 248.6, n = 44,

26.2%; high mid-PLR group: PLR \ge 248.6, n = 124, 73.8%). The post-PLR value was 332.8 ± 266.7 (low post-PLR group: PLR < 403.6, n = 99, 58.9%; high post-PLR group: PLR \ge 403.6, n = 69, 41.1%). The level of pre-PLR showed statistically significant differences compared to mid-PLR and post-PLR. (*P* < .0001, Fig. 3).

Among the 168 patients, the 1-, 2-, 3-, and 5-year overall survival (OS) rates were 73.7%, 56.8%, 48.0%, and 37.5%, respectively. In the high pre-PLR group, the 1-, 2-, 3-, and 5-year OS rates were 62.9%, 34.1%, 21.7%, and 21.7%, respectively; in the low pre-PLR group were 76.5%, 62.8%, 54.9%, and 42.1%, respectively. Results of the Kaplan–Meier analysis showed that the OS rate of the low pre-PLR group was higher than that of the high pre-PLR group (P < .001, Fig. 4A). The 1-, 2-, 3-, and 5-year OS rates of patients in the high Post-PLR group were 57.4%, 47.0%, 34.4%, and 27.1%, respectively; In the low post-PLR group were 83.8%, 63.6%, 57.3%, and 44.7%, respectively. Based on the Kaplan–Meier analysis, the OS rate of the low post-PLR group was higher than that of the high post-PLR group (P = .0026, Fig. 4B).

A prognostic model was constructed according to the changes in PLR levels during different treatments, group A (high pre-PLR + high post-PLR), group B (high pre-PLR + high post-PLR), and group D (low pre-PLR group + low post-PLR group) were formed according to the change level of PLR. Group D had a significantly different OS rate than the other groups (P < .001, Table 5 and Fig. 5).

4. Discussion

Inflammation is involved in several processes such as tumor occurrence and development, which makes hematology-related indicators a prerequisite for predicting tumor prognosis.^[19,20] Although the specific mechanism between hematological indicators and tumors is unclear, their correlation can be explained by inflammatory factors and lymphocytes. Pre-PLR has been widely studied to assess the inflammatory response and predict patient prognosis. In recent years, more and more studies have shown that Pre-PLR is of great significance owing to its predictive value on the prognosis of patients with esophageal cancer.^[15,21,22] However, in the present study, our results demonstrated the predictive value of PLR on ESCC at different treatment stages, including the significant predictive value of pre-PLR, post-PLR, pre-PLR + post-PLR in patients with ESCC receiving CRT. The OS of the low-PLR group was significantly better than that of the high PLR group (P < .001), and pre-PLR, post-PLR, drinking history, and age were independent prognostic factors based on the multivariate analysis, indicating that pre-PLR and post-PLR were the most important factors for patients receiving CRT. The effective predictor of prognosis in patients with ESCC.

Since Balkwill first put forward the hypothesis of the correlation between inflammatory response and tumor, he believed that 'tumor originates from chronic inflammation."^[23] Since then, a large number of studies have confirmed that it is closely related to the prognosis of malignant tumors such as lung cancer, colorectal cancer, and Breast cancer.[24-26] Inflammation plays an important role in many processes such as tumor development, invasion, and metastasis, and it is listed as one of the top 10 biological characteristics of tumor cells.^[27] As an indicator of body inflammation, PLR can reflect the relative changes in platelet and lymphocyte counts. In recent years, Pre-PLR has been commonly used in the evaluation of various tumors to predict the prognosis of cancer patients. Malignant tumors are often accompanied by elevated platelets. Platelets stimulate the proliferation of tumor cells by releasing some growth factors, including vascular endothelial growth factor, plateletderived growth factor, transforming growth factor-B and other



Figure 3. Changes of PLR in different treatment stages. (A) The levels of pre-PLR and post-PLR. (B) Paired analysis of pre-PLR and post-PLR (PLR before treatment: pre-PLR; PLR after treatment: post-PLR).

cytokines, thereby promoting tumor growth and metastasis.^[28,29] In addition, platelets can also protect tumor cells from antitumor immune responses.^[30] Lymphocytes are an important component of the body's immune response. Some lymphocytes leave the blood and migrate to tumor tissues to play a tumor-killing immune response. In addition, lymphocytes play an important role in the immune surveillance that inhibits the development of tumors.^[31,32]

This study shows that the survival benefit of patients in the low -PLR group may come from multiple aspects, including tumor size. On the contrary, a high PLR may be related to an increased tumor burden and increased metabolic rate mediated by inflammation. At present, there is no consensus on the cutoff value of PLR before treatment. The present study showed that the average PLR value of 168 patients with ESCC was higher than that reported in related studies. The results of He et al showed that the low PLR group (≤ 150) had a longer survival time, and an independent prognostic factor for OS, elevated PLR indicates a poor prognosis; their patients underwent radical surgical resection.[33] Compared with this study, the present study had patients with earlier tumor stage and smaller tumor burden and tumor inflammatory response. However, our patients were undergoing CRT for ESCC, and the tumor stage was later and the inflammatory load was higher, which may lead to an overall higher level of PLR in this study.

Toru et al's^[34] study on pre-PLR in patients with esophageal cancer who received radical treatment showed that the 5-year OS rate of the low pre-PLR group was 53.8% and that of the high pre-PLR group was 38.1% (P < .05) and that PLR was a significant prognostic factor. However, the results of the present study showed that the 5-year OS rate of patients in the high Pre-PLR group was 21.7%, and the 5-year OS rate of patients in the high other factors led to a worse prognosis. However, the prognosis of patients with ESCC patients was related not only to T stage and pre-PLR but also to patient age, alcohol consumption, T stage, post-PLR, and other factors. Treatments such as CRT will have different degrees of toxicity and side effects, among

which bone marrow suppression is the most common, leading to an increase in PLR levels, which could predict the prognosis of patients.

There are certain limitations in this study. First of all, this was a retrospective study with a small sample, thus the possibility of bias. In addition, as blood samples can be easily affected by factors such as treatment and infection, there might have been unknown factors affecting PLR that might have affected our results.

5. Conclusion

Pre-PLR and post-PLR can be used to predict the clinical outcome of patients with ESCC receiving radical CRT. However, since this study is a small-scale retrospective study, a large prospective cohort study is needed to verify the accuracy of PLR in different treatment phases and to assess other tumor nutrition assessment indicators.

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Writing - review & editing: Dan He, Chuan Yang.

Table 2

Univariate and multivariate analysis of prognostic factors.

		Univariate	analysis	Multivariate analysis	
Characteristics	Total (N)	Hazard ratio (95% CI)	P value	Hazard ratio (95% CI)	P value
Gender, n (%)			.219		
Male	37	Reference			
Female	131	1.365 (0.818-2.277)	.234		
Age (years), n (%)			.068		
<65	92	Reference		Reference	
≥65	76	1.450 (0.975-2.158)	.067	1.736 (1.129-2.669)	.012
KPS score, n (%)			.368	· · · · · · · · · · · · · · · · · · ·	
70	5	Reference			
80	68	0.437 (0.172-1.108)	.081		
90	94	0.404 (0.161–1.014)	.054		
100	1	0.714 (0.083–6.130)	.758		
Smoking history, n (%)			.403		
No	71	Reference			
Yes	97	1.188 (0.792-1.781)	.405		
Alcohol history, n (%)			.089		
No	77	Reference		Reference	
Yes	91	1.416 (0.944–2.122)	.092	1.622 (1.050–2.508)	.029
Location, n (%)	0.		.809		
Cervical	6	Reference	1000		
Upper thoracic	34	1.211 (0.356–4.113)	.759		
Middle thoracic	56	1.264 (0.388–4.122)	.698		
Lower thoracic	64	1.245 (0.383–4.053)	.715		
Abdomina	8	2.045 (0.527–7.931)	.301		
Tumour length (cm), n (%)	0	2.040 (0.021 1.001)	.010		
<5	68	Reference	.010	Reference	
≥5	100	1.722 (1.130–2.623)	.011	1.239 (0.797–1.925)	.340
T-stage, n (%)	100	1.722 (1.100 2.020)	<.001	1.200 (0.101 1.020)	.010
T2	22	Reference	1.001	Reference	
T3	77	5.719 (1.773–18.453)	.004	5.039 (1.541–16.477)	.007
T4	69	14.447 (4.458–	<.001	12.483 (3.719–	<.001
I T	00	46.814)	1.001	41.896)	1.001
N-stage, n (%)		40.014)	.007	41.090)	
NO NO	6	Reference	.007	Reference	
N0 N1	54	4.359 (0.594–31.954)	.148	1.867 (0.247–14.115)	.545
N2	79	5.118 (0.705–37.142)	.106	1.720 (0.228–12.960)	.599
N3	29	9.599 (1.284–71.770)	.100	1.480 (0.182–12.028)	.714
RT dose (Gy), n (%)	29	9.599 (1.204-71.770)	.638	1.400 (0.102-12.020)	./ 14
<60	26	Reference	.030		
≥60	142	1.148 (0.640–2.058)	.644		
<i>Chemotherapy, n (%)</i>	142	1.148 (0.040-2.058)	.536		
No	26	Reference	.000		
Yes	142	0.841 (0.492–1.439)	.528		
Pre-PLR	142	0.041 (0.492-1.409)	.020 .001		
<196.15	132	Reference	.001	Reference	
≥196.15	36	2.165 (1.383–3.387)	<.001	1.716 (1.069–2.756)	.025
Post-PLR	30	2.103 (1.303–3.307)	.003	1.110 (1.009-2.150)	.020
<403.6	99	Reference	.003	Reference	
<403.6 ≥403.6	99 69	1.827 (1.228–2.718)	.003	1.664 (1.106–2.503)	.015
≥403.0	09	1.021 (1.220-2.110)	.003	1.004 (1.100–2.303)	.015

CI = confidence interval, ESCC = esophageal squamous cell carcinoma, HR = hazard ratio, PLR = platelet-to-lymphocyte ratio, RT = radiotherapy.

Table 3

Demographic and clinical characteristics of cancer patients stratified by pre-PLR.

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Table 4
Demographic and clinical characteristics of cancer patients
stratified by post-PLR.

Characteristics	Total (N)	Pre-PLR < 195.7 (n = 132)	Pre-PLR ≥ 195.7 (n = 36)	P value	Characteristics	Total (N)	Post-PLR < 403.6	$\textbf{Post-PLR} \geq 403.6$	P value
	(,	(11 = 102)	(11 – 00)		Gender, n (%)				.049
Gender, n (%)				.674	Male	131	72 (42.9%)	59 (35.1%)	
Male	131	102 (60.7%)	29 (17.3%)		Female	37	27 (16.1%)	10 (6%)	
Female	37	30 (17.9%)	7 (4.2%)		Age (years), n				.233
Age (years),				.627	(%)				
n (%)					<65	92	58 (34.5%)	34 (20.2%)	
<65	92	71 (42.3%)	21 (12.5%)		≥65	76	41 (24.4%)	35 (20.8%)	
≥65	76	61 (36.3%)	15 (8.9%)		KPS score, n (%)				.339
KPS score, n (%)				.5	70	5	2 (1.2%)	3 (1.8%)	
70	68	55 (32.7%)	13 (7.7%)		80	68	44 (26.2%)	24 (14.3%)	
80	94	5 (3%)	0 (0%)		90	94	53 (31.5%)	41 (24.4%)	
90	5	71 (42.3%)	23 (13.7%)		100	1	0 (0%)	1 (0.6%)	
100	1	1 (0.6%)	0 (0%)		Smoking history,				.101
Smoking history,				.497	n (%)				
n (%)					No	71	47 (28%)	24 (14.3%)	
No	97	78 (46.4%)	19 (11.3%)		Yes	97	52 (31%)	45 (26.8%)	
Yes	71	54 (32.1%)	17 (10.1%)		Alcohol history,			()	.254
Alcohol history,		01 (021170)		.571	n (%)				
n (%)				.071	No	77	49 (29.2%)	28 (16.7%)	
No	91	70 (41.7%)	21 (12.5%)		Yes	91	50 (29.8%)	41 (24.4%)	
Yes	77	62 (36.9%)	15 (8.9%)		Location, n (%)	01	00 (20.070)	+1 (Z+1+70)	.866
Location, n (%)	11	02 (30.970)	13 (0.970)	.702	Cervical	6	3 (1.8%)	3 (1.8%)	.000
Cervical	6	51 (30.4%)	13 (7.7%)	.102	Upper	34	21 (12.5%)	13 (7.7%)	
Upper	34	27 (16.1%)	7 (4.2%)		thoracic	54	21 (12.070)	10 (1.170)	
	54	27 (10.170)	7 (4.270)		Middle	56	20 (100/)	04 (14 20/)	
thoracic	50		14(0,00())			50	32 (19%)	24 (14.3%)	
Middle	56	42 (25%)	14 (8.3%)		thoracic	0.4	07 (000)	07 (10 10/)	
thoracic			- ////		Lower	64	37 (22%)	27 (16.1%)	
Lower	64	6 (3.6%)	2 (1.2%)		thoracic	_			
thoracic					Abdomina	8	6 (3.6%)	2 (1.2%)	
Abdomina	8	6 (3.6%)	0 (0%)		Tumor length				.115
Tumour length				.08	(cm), n (%)				
(cm), n (%)					<5	68	45 (26.8%)	23 (13.7%)	
<5	68	58 (34.5%)	10 (6%)		≥5	100	54 (32.1%)	46 (27.4%)	
≥5	100	74 (44%)	26 (15.5%)		T-stage, n (%)				.102
T-stage, n (%)				.023	T2	22	14 (8.3%)	8 (4.8%)	
T2	22	21 (12.5%)	1 (0.6%)		T3	77	51 (30.4%)	26 (15.5%)	
T3	77	63 (37.5%)	14 (8.3%)		T4	69	34 (20.2%)	35 (20.8%)	
T4	69	48 (28.6%)	21 (12.5%)		N-stage, n (%)				.187
N-stage, n (%)			(<i>'</i>	.004	NO	6	6 (3.6%)	0 (0%)	
NO	6	47 (28%)	7 (4.2%)		N1	54	33 (19.6%)	21 (12.5%)	
N1	79	63 (37.5%)	16 (9.5%)		N2	79	44 (26.2%)	35 (20.8%)	
N2	54	6 (3.6%)	0 (0%)		N3	29	16 (9.5%)	13 (7.7%)	
N3	29	16 (9.5%)	13 (7.7%)		RT dose (Gy),		· · · ·		.061
RT dose (Gy),				.414	n (%)				
n (%)					<60	26	11 (6.5%)	15 (8.9%)	
<60	26	22 (13.1%)	4 (2.4%)		≥60	142	88 (52.4%)	54 (32.1%)	
≥60	142	110 (65.5%)	32 (19%)		Chemotherapy,			(0/0)	.061
Chemotherapy,	144	110 (00.070)	02 (10/0)	.414	n (%)				.001
1.2.				.414	No	26	11 (6.5%)	15 (8.9%)	
n (%) No	06	00 (10 10/)	1 (0 10/)		Yes	142	88 (52.4%)	54 (32.1%)	
No Yes	26 142	22 (13.1%) 110 (65.5%)	4 (2.4%) 32 (19%)		100	142	00 (JZ.470)	J4 (J2.170)	

radiotherapy.





Table 5

Relationship between pre-PLR and post-PLR.

Group		Post-PLR	Patients, n (%)	Overall survival		
	Pre-PLR			Median (95%Cl)	P value	
A	High PLR(≥195.7)	High PLR(≥403.6)	18 (10.7%)	16.733 (6.904–26.563)	<.001	
В	High PLR(≥195.7)	Low PLR(<403.6)	18 (10.7%)	16.300 (8.816-23.784)		
С	Low PLR(<195.7)	High PLR(≥403.6)	51 (30.4%)	24.800 (9.655–39.945)		
D	Low PLR(<195.7)	Low PLR(<403.6)	81 (48.2%)	52.800 (24.912-80.688)		

CI = confidence interval, PLR = platelet-to-lymphocyte ratio.



Figure 5. Relationship between PLR changes and prognosis in different treatment stages. Group A (high pre-PLR + high post-PLR), group B (high pre-PLR + high post-PLR), C (low pre-PLR + high post-PLR), and group D (low pre-PLR group + low post-PLR group) were formed according to the change level of PLR. Group D had a significantly different OS rate than the other groups.

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