Research Square

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Constrction and validation of a nomogram model for pulmonary infection after aneurysm embolization

Xiaoyong Lin
Guangdong Medical University
Yifan Deng
dengyifanhzyy@outlook.com
Guangdong Medical University
Honghai Luo
Huizhou Central People's Hospital
Ke Xu
Huizhou First People's Hospital
Yingcong Wei
Guangdong Medical University
Wencai Li
Huizhou Central People's Hospital
Haiyong Zeng
Huizhou Central People's Hospital
Gang Zhu
Huizhou Central People's Hospital

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Abstract

Blackground and objective:

In order to analyze the risk factors associated with pulmonary infection following aneurysm embolization, we developed a nomogram model and validated its predictive accuracy.

Methods:

Clinical data pertaining to patients who underwent aneurysm embolization procedures at Huizhou Central People's Hospital during the period from 2019 to 2023 were retrieved utilizing the hospital's electronic medical record system.Variables including age, Platelet-to-Lymphocyte Ratio (PLR), Systemic Immune-Inflammation Index (SIRI), Red Blood Cell Distribution Width-to-Albumin/Globulin Ratio (RDWAGR), and creatinine (CREA) levels were calculated. The variables identified were subsequently incorporated into a logistic regression model to evaluate their associations with the incidence of pulmonary infections subsequent to aneurysm embolization surgery.A nomogram model was subsequently developed and validated, with its predictive performance assessed via the Receiver Operating Characteristic (ROC) curve.

Results:

Logistic regression analysis identified that age, PLR, SIRI, RDWAGR, d-NLR, and FIB were significantly associated with postoperative pulmonary infection ($P < 0.05$). Drawing upon these significant variables, the constructed nomogram model demonstrated robust discriminatory power in forecasting the risk of pulmonary infection, with an area under the receiver operating characteristic curve (AUC) of 0.769, signifying a high level of predictive precision.The calibration plot exhibited satisfactory alignment between the model's predicted probabilities and the observed outcomes. Additionally, internal validation assessments further underscored the nomogram model's stability and reproducibility.

Conclusions:

The nomogram model established in this research accurately forecasts the risk of pulmonary infection in patients post-aneurysm embolization surgery, showcasing robust predictive capabilities and significant clinical utility. This tool can be a pivotal asset for clinicians in postoperative care, facilitating early identification of patients at high risk and guiding the customization of intervention strategies.

Introduction

The prevalence of intracranial aneurysms in the general populace ranges from 3–5%, with an annual risk of rupture of approximately 0.95%[1–4]. However, upon rupture, an aneurysm can result in subarachnoid hemorrhage (SAH), the mortality rate can reach up to 50%[5–6]. This significantly affects the patient's well-being and poses a serious threat to their health and safety. Therefore, prompt treatment for intracranial aneurysms is absolutely essential.Surgical treatment is the preferred option for patients with intracranial aneurysms. The main surgical approaches include endovascular embolization and craniotomy clipping[7]. Among these, endovascular treatment has gradually supplanted craniotomy clipping as the preferred method due to its minimally invasive nature and favorable prognosis[8].Surgical complications may include delayed cerebral ischemia, hydrocephalus, and pulmonary infections. Large-scale pathological analyses and literature reviews report that the incidence of pulmonary infection following surgery has been reported to vary between 13.2% and 29.15%[9], making it a serious complication that increases patient mortality and prolongs hospital stays, thereby imposing a significant burden on patients and their families. Despite the high prevalence and substantial clinical impact of postoperative pulmonary infections following aneurysm embolization, limited research has focused on identifying biomarkers to predict these infections, leaving a gap in the literature.

Previous studies indicate that immune dysregulation and excessive inflammatory responses triggered by subarachnoid hemorrhage may increase the risk of pulmonary infections. Due to the complexity of the underlying pathological mechanisms, univariate analysis alone is likely insufficient to fully elucidate the occurrence and progression of these infections[8].Therefore, combining multiple factors to establish a more accurate predictive model for pulmonary infection is necessary.

This study focuses on examining and pinpointing the risk factors contributing to pulmonary infections in patients undergoing aneurysm embolization through retrospective analysis and statistical evaluation. By integrating clinical practice, we will extract high-value predictive risk indicators, clarify associated risk factors, and construct a risk prediction nomogram model. For high-risk patients, timely interventions and personalized treatment plans can be implemented to prevent postoperative pulmonary infections, ultimately improving patient outcomes.

Materials and methods

This study was a single- center retrospective study.

Patients and participants

This retrospective study reviewed clinical data from 290 patients who underwent aneurysm embolization at Huizhou Central People's Hospital between 2019 and 2023. All diagnoses were independently confirmed by three experienced neurosurgeons."

The inclusion criteria were: (1) Patients diagnosed with aneurysmal subarachnoid hemorrhage confirmed by computed tomography (CT), computed tomography angiography (CTA), or digital subtraction angiography (DSA); (2) Patients who met the surgical indications and were able to tolerate the

procedure; (3) Patients who signed informed surgical consent; (4) Patients who underwent endovascular embolization after admission.

The exclusion criteria were: (1) Death within 24 hours of admission; (2) Diagnosed with community-acquired pneumonia or pulmonary infection prior to admission; (3) Presence of pseudoaneurysms; (4) Comorbid infectious diseases, systemic autoimmune diseases, or malignancies; (5) Interruption of treatment or loss to follow-up; (6) Age under 18 years.

The diagnostic criteria for postoperative pulmonary infection were: (1) New-onset pulmonary infection indicated by chest imaging; (2) Body temperature > 38.3°C, white blood cell count < 4.0 × 10^9/L or > 12 × 10^9/L; (3) Reduced blood oxygen levels; (4) At least two accompanying symptoms, such as purulent sputum, dyspnea, cough, increased oxygen demand, or need for respiratory support.

A total of 290 patients participated in this research, which was granted approval from the Ethics Committee of Huizhou Central People's Hospital. All research procedures adhered strictly to the principles outlined in the Declaration of Helsinki and local ethical standards. Prior to enrollment, informed consent was obtained from each patient or their designated next of kin, in accordance with the hospital's established policies.

Data collection

Clinical data for all eligible patients were collected, including admission and follow-up information. Data collection encompassed the following aspects: (1) General information, such as the patient’s name, age, gender, height, weight, and other relevant details; (2) Current medical history obtained through patient inquiry; (3) Past medical history and personal history, including any comorbidities such as hypertension, diabetes, and others; (4) Assessment of disease severity upon admission, including Glasgow Coma Scale (GCS) score and Hunt-Hess grade; (5) Laboratory data obtained within 24 hours of admission through peripheral venous blood collection. The laboratory results included a comprehensive analysis of peripheral blood parameters, such as white blood cell count, neutrophil count, lymphocyte count, monocyte count, platelet count, prothrombin time, activated partial thromboplastin time, fibrinogen, D-dimer, and albumin levels.

Statistical analysis

Patients were randomly divided into a modeling group and a validation group in a 7:3 ratio using SPSS 27.0 software. Clinical data were collected for all patients, and based on the occurrence of postoperative pneumonia, the modeling cohort was further subdivided into a pneumonia group and a non-pneumonia group. In the univariate analysis, continuous variables that did not conform to a normal distribution were compared using the Mann-Whitney U test and presented as median ± interquartile range (IQR), while continuous variables that followed a normal distribution were compared using the Student’s t-test and presented as mean ± standard deviation (SD). Categorical variables were compared using Pearson’s chi-square test. Variables with a P-value < 0.05 were included in the multivariate analysis, which was conducted using binary logistic regression to identify independent risk factors for postoperative pneumonia following aneurysm embolization.A risk prediction nomogram model for pneumonia occurrence was constructed using R software, with internal validation performed via bootstrap resampling 1000 times. For each variable, odds ratios (ORs) and 95% confidence intervals (CIs) were computed. To evaluate the sensitivity and specificity of the predictive factors, a receiver operating characteristic (ROC) curve was plotted. Additionally, the area under the ROC curve (AUC) was calculated to assess the model's discriminatory power.An AUC greater than 0.7 was considered indicative of good discrimination. The Hosmer-Lemeshow goodness-of-fit test was employed to evaluate the model’s fit, and calibration curves were plotted to assess the nomogram's performance in predicting the occurrence of pneumonia post-embolization.

Results

Patients' characteristics

Our flow chart is shown in Fig. 1, which is filtered by strict nanosteric criteriaThis retrospective study included 290 patients, comprising 128 males (44.1%) and 162 females (55.9%), with a median age of 55 years (range: 21 to 88 years).Among these patients, 99 cases (34%) experienced pulmonary infection, while 191 cases (66%) did not. Table 1presents the clinical characteristics of all patients.

Using SPSS 27.0 software, patients were randomly assigned to two groups in a 7:3 ratio, creating a training set of 203 patients and a validation set of 87 patients. According to Table 2, there were no statistically significant differences in the variables between the training and validation sets, suggesting that they had similar baseline characteristics.

Predictive factors selection and construction of nomogram

Table 2 presents the results of the univariate analysis conducted on the training set, identifying relevant variables associated with pulmonary infection following aneurysm embolization. These variables include Age (P < 0.01), RDW (P = 0.041), WBC (P < 0.01), Neut (P < 0.01), Eo (P = 0.032), UREA (P = 0.014), CREA (P = 0.016), AG (P = 0.011), AST (P = 0.006), LDH (P = 0.001), ADA (P = 0.016), FIB (P = 0.006), D-D (P = 0.004), HR (P = 0.03), NLR (P = 0.005), d-NLR (P = 0.041), SII (P = 0.025), SIINI (P = 0.009), SIRI (P < 0.001), Hunt-Hess (P < 0.001), GCS (P < 0.001), RDWAGR (P = 0.001), and PLR (P < 0.01). Among these 22 variables with statistically significant differences, potential predictive factors were selected through logistic regression.

Principal component analysis (PCA) was employed to screen variables with high variance inflation factors, reducing multicollinearity. These variables were further assessed using binary logistic regression. As shown in Table 3, Age (OR = 1.053, 95% CI: 1.019–1.089), PLR (OR = 1.006, 95% CI: 1.002–1.011), SIRI (OR = 1.047, 95% CI: 1.003–1.092), RDWAGR (OR = 1.14, 95% CI: 1.007–1.289), and CREA (OR = 1.015, 95% CI: 1.001–1.03) were identified as independent predictors of pulmonary infection after aneurysm embolization. A nomogram was subsequently constructed based on these predictors Fig. 2.

Validation and clinical use of nomogram

Internal validation of the nomogram model in the training set was performed using the Bootstrap method with 1,000 resamples, resulting in an area under the ROC curve (AUC) of 0.769 (as shown in Fig. 3). In ROC curve analysis, a predictor variable is generally deemed to possess good discriminative ability if its AUC surpasses 0.7. Furthermore, Decision Curve Analysis (DCA) revealed that our nomogram provided a positive net benefit compared to the all-or-none strategy across a threshold probability range of 10–95% in both the training set (Fig. 4A) and the validation set (Fig. 4B).

The Hosmer-Lemeshow goodness-of-fit test was employed to assess the model's fit, and a calibration curve was plotted to evaluate the model's accuracy. The p-value for the Hosmer-Lemeshow test was 0.659 ($P > 0.05$), indicating no statistically significant difference between the predicted and observed outcomes, thus suggesting that the model demonstrates good accuracy.Through internal validation, it was determined that the model exhibits both strong discrimination and calibration.

Discussion

Intracranial aneurysms result from damage and lesions in the walls of intracranial arteries, leading to abnormal bulging, and they are a leading cause of subarachnoid hemorrhage[10–12].These aneurysms are associated with high mortality and morbidity. Patients often present with a sudden onset of symptoms, including increased intracranial pressure and altered consciousness, which render them vulnerable to aspiration and vomiting[13].Endovascular embolization, one of the primary surgical treatments for intracranial aneurysms, has a high cure rate but can result in various complications, including pulmonary infections, which significantly impact patient prognosis, hospital stay duration, and associated costs[14–15]. In severe cases, such infections may lead to death. Currently, there is a lack of reliable predictive models for identifying risk factors for post-embolization pulmonary infections.

Elderly patients often experience a weakened vomiting reflex, diminished mucociliary function, reduced immune response, and compromised cardiopulmonary function, all of which elevate their risk of developing pneumonia[16–17].According to data from the Centers for Disease Control and Prevention (CDC), approximately 150,000 adults aged 65 and older are hospitalized for pneumonia annually in the United States[18]. Many patients present with varying degrees of altered consciousness at the onset of illness, which further heightens the risk of aspiration and substantially increases the likelihood of pulmonary infections[19].

SIRI (Systemic Inflammatory Response Index) and PLR (Platelet-to-Lymphocyte Ratio) are emerging inflammatory markers that have gained significant attention in recent years. These markers are increasingly employed to predict the likelihood and prognosis of various clinical outcomes, including rheumatic diseases, acute pulmonary embolism, and acute pancreatitis[20–22].However, relatively few studies have confirmed the relationship between PLR, SIRI, and postoperative pulmonary infections following aneurysm embolization. In this study, the median Systemic Inflammatory Response Index (SIRI) in patients with pulmonary infections was 7.22, whereas in non-pulmonary infection patients, it was 4.68 ($P < 0.01$). Similarly, the median Platelet-to-Lymphocyte Ratio (PLR) in patients with pulmonary infections was 247.7, compared to 230 in non-pulmonary infection patients ($P < 0.01$), both of which were statistically significant.

Following aneurysmal rupture resulting in subarachnoid hemorrhage, the body’s inflammatory response escalates rapidly and is strongly linked to the development of pulmonary infections[23].It is well established that the immune system’s primary role is to eliminate infections; however, excessive activation of the immune response can result in host tissue damage[24]. The most extreme form of this is known as a “cytokine storm.” For instance, during the late stages of Ebola virus infection, the cytokine storm is a primary cause of mortality. Several viruses, including avian flu and SARS, can trigger the immune system to initiate an aggressive response, attacking the body. Cytokines, which are small molecules released into the bloodstream, are critical in immune cell communication. They mobilize immune cells to infection sites, enabling them to engulf damaged cells and even penetrate blood vessel walls[25]. Similarly, aneurysmal rupture leading to subarachnoid hemorrhage elicits a pronounced inflammatory response, which is strongly correlated with the development of pulmonary infections.

Neutrophils produce neutrophil extracellular traps (NETs), which play a role in the formation of blood clots in the lungs. These NETs can trigger an inflammatory response and contribute to damage to the lung tissue.Neutrophils also release enzymes such as myeloperoxidase and neutrophil elastase, which exacerbate lung injury through oxidative stress and DNA damage[26–28]. Fluctuations in lymphocyte count may reflect the patient’s immune status, with moderate lymphocyte elevations potentially supporting infection prevention and reducing the risk of pulmonary infections[29]. The Systemic Inflammatory Response Index (SIRI) and Platelet-to-Lymphocyte Ratio (PLR) incorporate peripheral white blood cell, neutrophil, and lymphocyte counts, providing a more comprehensive evaluation of the immune-inflammation balance than white blood cell count alone.Furthermore, studies suggest that PLR has superior predictive ability for post-stroke pulmonary infections compared to SIRI. Given that inflammation is a dynamic process, clinicians routinely monitor inflammatory markers to track disease progression in clinical practice.

Red blood cell distribution width (RDW) is strongly associated with the body’s stress response, with elevated levels generally indicating stress-induced inflammation. In this state, abnormal secretion of inflammatory cytokines, such as interleukin-6 (IL-6), impairs red blood cell maturation, resulting in greater variability in red blood cell size and volume[30–32]. Albumin and globulin are essential components of serum proteins, with albumin synthesized primarily in the liver[33]. In critically ill neurological patients, low albumin levels are recognized as an independent risk factor for various complications, indicating impaired immune function and poor nutritional status. The albumin-to-globulin (A/G) ratio is a key marker for assessing liver function[34].

Multiple studies have established red cell distribution width (RDW) as an independent prognostic factor for patient outcomes in lung infections[35]. However, limited research has investigated the association between RDW and the albumin/globulin (A/G) ratio in this clinical setting. Previous investigations into lung

infections have predominantly centered on inflammatory markers, with relatively little emphasis placed on liver function-related markers. Our findings demonstrate that the combined assessment of RDW and the albumin/globulin (A/G) ratio offers substantial diagnostic value for lung infections, comparable to conventional inflammatory markers such as white blood cell (WBC) and neutrophil (Neut) counts.

Few studies have focused on early markers for detecting such infections. In this retrospective analysis, we evaluated the predictive value of age, Platelet-to-Lymphocyte Ratio (PLR), Systemic Inflammatory Response Index (SIRI), Red Blood Cell Distribution Width-to-Albumin/Globulin Ratio (RDWAGR), and creatinine (CREA) in assessing the risk of pulmonary infections following aneurysm embolization.The results revealed a 34% incidence rate of pulmonary infection. Through multi-factor logistic regression, we established a significant relationship between these variables and the occurrence of infections. The ROC curve analysis yielded an area under the curve (AUC) of 0.769 (95% CI: 0.7–0.839). Validation in 87 post-embolization patients demonstrated a sensitivity of 0.481 and a specificity of 0.883, indicating satisfactory predictive performance. Our findings indicate that this model may facilitate early recognition and prediction of pulmonary infections following aneurysm embolization.

In conclusion, the risk factors for pulmonary infections in patients undergoing aneurysm embolization are complex and multifactorial. The risk prediction model developed in this study demonstrates promising predictive capability and may serve as a valuable reference for assessing the likelihood of pulmonary infection. However, the findings are constrained by the single-center design, a relatively small sample size, and the possibility of inaccuracies or missing data within patient records.Additionally, the limited sample size hindered a more nuanced analysis of pulmonary infections categorized by severity (mild, moderate, or severe). Future research should prioritize the inclusion of multi-center studies with larger sample sizes and a broader scope of observational parameters to further refine the model's accuracy and generalizability.

Declarations

Author Contribution

a proposed the generation of the research concept and completed the draft writing, data analysis, and mapping, b completed the data analysis and contributed to the data collection,all the authors reviewed the manuscript

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Data Availability

All data used or analyzed during this study are included in this article and supplementary documents

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Tables

Table 1Baseline clinical characteristics associated with patients after aneurysm

Characterists		Pneumonia=1，n=99，	No pneumonia，n=191，	P
Gender，n，%，	Female	58，59，	104，54，	0.501
	Male	41，41，	87，46，	
Age， Median (Q1,Q3)		59，50，68，	54(47,61)	<0.01
Hb， Median (Q1,Q3)		126(114,141)	132(120,142)	0.032
RBC， Median (Q1,Q3)		4.3(3.85,4.73)	4.47(4.06,4.76)	0.033
HCT， Median (Q1,Q3)		0.365(0.344,0.411)	0.386(0.355,0.418)	0.024
MCV， Median (Q1,Q3)		89.3(83.6,92.6，	88.3，83.6，91.9，	0.443
MCH， Median (Q1,Q3)		30.4(28.4,31.5)	30.3(28.5,31.2)	0.654
MCHC， Median (Q1,Q3)		339(329,347)	340(329,349)	0.548
RDW， Median (Q1,Q3)		13.5(12.6,14.2)	13(12.4,13.7)	0.037
WBC， Median (Q1,Q3)		14(11.5,17.5)	12.3(9.7,15)	<0.01
Neut， Median (Q1,Q3)		12.47，9.23，15.49，	10.58(7.51,13.43)	<0.01
Lymph， Median (Q1,Q3)		0.96，0.69，1.4，	1.1(0.77,1.55)	0.081
Mono， Median (Q1,Q3)		0.66，0.38，1.04，	0.56(0.34,0.78)	0.024
Eo， Median (Q1,Q3)		0(0,0.14)	0(0,0.02)	0.046
Baso， Median (Q1,Q3)		0.2(0.1,0.4)	0.02(0.01,0.03)	0.723
PLT， Median (Q1,Q3)		228(180,270)	238(200,281)	0.111
PCT， Median (Q1,Q3)		0.24(0.2,0.28)	0.24(0.21,0.28)	0.446
MPV， Median (Q1,Q3)		10.2(9.8,10.8)	10.2(9.6,10.61)	0.206
PDW， Median (Q1,Q3)		11.6(10.7,12.5)	11.3(10.3,12.6)	0.267
PAB， Median (Q1,Q3)		0.246(0.201,0.296)	0.25(0.208,0.292)	0.685
NA， Median (Q1,Q3)		139(137.1,142)	139(137,141)	0.922
CL， Median (Q1,Q3)		102.5(99.6,105.7)	103.6(100.4,105.7)	0.698
K， Median (Q1,Q3)		3.49(3.29,3.81)	3.61(3.38,3.82)	0.058
CA， Median (Q1,Q3)		2.22(2.12,2.31)	2.22(2.14,2.3)	0.649
UREA， Median (Q1,Q3)		4.9(3.7,6.3)	4.3(3.5,5.3)	0.009
CREA， Median (Q1,Q3)		69(58,88)	65(51,79)	0.014
TP， Median (Q1,Q3)		73(68.4,79)	71.5(68.1,76.8)	0.166
ALB， Median (Q1,Q3)		41.6(37.7,45.1)	41.8(39,44.8)	0.243
A/G， Median (Q1,Q3)		1.305(1.16,1.44)	1.4(1.24,1.57)	0.002
TBIL， Median (Q1,Q3)		10.75(7.4,13.63)	11.9(8.6,14.2)	0.238
DBIL， Median (Q1,Q3)		4.2(2.9,4.9)	4.3(3.2,5)	0.524
ALT， Median (Q1,Q3)		18(12,24)	16(11.4,22)	0.173
AST， Median (Q1,Q3)		24(17,37)	18.4(15.3,24.76)	<0.01
ALP， Median (Q1,Q3)		67(57,81)	68(55,83)	0.858
GCT， Median (Q1,Q3)		23(16,39)	24(15,36)	0.579
LDH， Median (Q1,Q3)		205(187,243)	194(165,215)	<0.01
ADA， Median (Q1,Q3)		10.12(8,13.25)	9(7,12)	0.008
FIB， Median (Q1,Q3)		3.39(3,4.14)	3.28(2.81,3.72)	0.009
D-D， Median (Q1,Q3)		1810(1160,3870)	1380(610,2863)	0.002
GCS， n，%，	<8	13(13)	10(5)	<0.01
	8-12	26(26)	10(5)	

Characterists		Pneumonia=1（n=99）	No pneumonia（n=191）	P
	>12	60(61)	171(90)	
Hunt-Hess（ n（%）	I	2(2)	60(31)	<0.01
	II	34(34)	81942)	
	III	35(35)	37(19)	
	IV	24(24)	13(7)	
	V	4(4)	0(0)	
BMI（ Median (Q1,Q3)		22.34(20.49,24.03)	23.27(21.28,24.85)	0.024
Systolic pressure（ Median (Q1,Q3)		145(123,167)	144(128,163)	0.914
Diastolic pressure（ Median (Q1,Q3)		82(70,94)	87(76,95)	0.119
Pulse pressure（ Median (Q1,Q3)		61(47,76)	58(49,68)	0.296
HR（ Median (Q1,Q3)		82(70,96)	77(71,87)	0.06
R（ Median (Q1,Q3)		20(17,23)	19(16,21)	0.021
NLR（ Median (Q1,Q3)		12.61(7.9,20.33)	9.81(5.48,15.17)	0.002
d-NLR（ Median (Q1,Q3)		8.55(5.39,11.61)	7.24(4.66,10.65)	0.043
PLR（ Median (Q1,Q3)		231.68(140.71,325.40)	224.62(150,295.23)	P<0.01
SII（ Median (Q1,Q3)		2903.34(1518.47,4949.72)	2532.78(1294.29,3706.94)	0.028
SIINI（ Median (Q1,Q3)		379.74(220.04,681.60)	316.56(182.97,463.97)	0.009
PNI（ Median (Q1,Q3)		47.45(42,50.2)	48.05(44.95,51.2)	0.034
SIRI（ Median (Q1,Q3)		7.24(4.65,16.71)	4.65(2.29,8.19)	<0.01
Emesis（n%）	1	79(80)	132(70)	0.053
	0	20(20)	59930）	
Hypertension（n%）	1	61（61）	124（65）	0.672
	0	38（39）	67（35）	
Diabetes（n%）	1	14（14）	7（5）	0.001
	0	85（86）	184（95）	
Drink（n%）	1	7（7）	6（3）	0.125
	0	92（93）	185（97）	
	0			
RDWAGR（ Median (Q1,Q3)		10.51(9.07,12.62)	9.56(8.06,11.06)	<0.001

Table 2.Baseline clinical characteristics of patients in training set and validation set and univariate analysis in training set.

Characterists		Training set	Validation set	P	Training set		P
		(n=203)	(n=87)		Pneumonia（n=72）	No pneumonian=131）	
Gender（n（%）	Female	113(55.67%)	49(56.32%)	0.918	41（（57%）	72（60%）	
	Male	90(44.33%)	38(43.68%)		31（43%）	59（40%）	
Age, Mean ± SD		55(48,64)	56(47,65)	0.946	59.57±12.16	52.73±10.70	<0.01
Hb ,Median (Q1,Q3)		130(118,142)	128(117,141)	0.692	123.5(113,75,1442)	133(121,142)	0.058
RBC（ Median (Q1,Q3)		4.41((3.99,4.79)	4.32(4,4.69)	0.345	4.32(3.87,4.76)	4.5(4.06,4.81)	0.072
HCT（ Median (Q1,Q3)		0.383(0.351,0.416)	0.384(0.348,0.412)	0.573	0.36(0.34,0.41)	0.39(0.36,0.42)	0.055
MCV（ Median (Q1,Q3)		88.4(83.5,92)	88.5(84.8,92.3)	0.336	88.35(83.45,92.5)	88.3(83.3,92)	0.97
MCH（ Median (Q1,Q3)		30.3(28.5,31.3)	30.5（28.8（31.5（	0.511	30.15(28.13,31.4)	30.3(28.6,31.2)	0.717
MCHC（ Median (Q1,Q3)		339(329,348)	340（329（348（	0.9	339(329,346)	340(329,350)	0.474
RDW（ Median (Q1,Q3)		13.1(12.5,14.1)	13（12.5（13.8（	0.197	13.5(12.7,14.325)	13(12.4,13.8)	0.041
WBC（ Median (Q1,Q3)		13.3(10.1,16.5)	12.2(10.1,15.5)	0.615	14.65(12.73,18.13)	12.5(9.7,15.6)	<0.01
Neut（ Median (Q1,Q3)		11.65(8.53,14.55)	10.72(7.83,13.78)	0.275	12.96(10.07,15.64)	10.59(7.13,13.54)	<0.01
Lymph（ Median (Q1,Q3)		1.05(0.7,1.55)	1.05(0.75,1.46)	0.603	0.98(0.66,1.50)	1.08(0.77,1.55)	0.215
Mono（ Median (Q1,Q3)		0.57(0.35,0.86)	0.6(0.43,0.84)	0.559	0.63(0.37,1.05)	0.55(0.34,0.78)	0.074
Eo（ Median (Q1,Q3)		0(0,0.018)	0(0,0.02)	0.382	0(0,0.11)	0(0,0.03)	0.032
Baso（ Median (Q1,Q3)		0.02(0.01,0.03)	0.02(0.01,0.03)	0.392	0.02(0.01,0.42)	0.02(0.01,0.03)	0.611
PLT（ Median (Q1,Q3)		236(197,281)	227(198,273)	0.449	232(192.75,274.25)	241(200,290)	0.258
PCT（ Median (Q1,Q3)		0.244(0.21,0.28)	0.24(0.2,0.27)	0.294	0.24(0.21,0.28)	0.24(0.21,0.28)	0.468
MPV（ Median (Q1,Q3)		10.3（9.6（10.8（	10.1(9.7,10.61)	0.356	10.3(9.8,10.8)	10.3(9.6,10.8)	0.739
PDW（ Median (Q1,Q3)		11.6（10.6（12.7（	11.1(10.4,12.1)	0.079	11.64(10.7,12.53)	11.5(10.4,12.9)	0.798
PAB（ Median (Q1,Q3)		0.25（0.21（0.3（	0.245(0.204,0.283)	0.522	0.24(0.20,0.3)	0.25(0.21,0.30)	0.656
NA（ Median (Q1,Q3)		139（137（141（	140(138,142)	0.174	139(137,142)	139(137,141)	0.489
CL（ Median (Q1,Q3)		102.9（99.6（105.5（	104(100.3,105.9)	0.217	102.4(99.38,105.63)	103.2(99.8,104.9)	0.946
K（ Median (Q1,Q3)		3.6（3.35（3.82（	3.51(3.31,3.82)	0.372	3.53(3.30,3.84)	3,64(3.42,3.82)	0.061

CA Median (Q1,Q3)	2.23 2.14 2.3	2.2(2.12,2.28)	0.303	2.23(2.14,2.3)	2.23(2.14,2.3)	0.969
UREA Median (Q1,Q3)	4.4 3.4 5.5	4.6(3.8,5.8)	0.496	4.9(3.38,6.33)	4.3(3.3,5.2)	0.014
CREA Median (Q1,Q3)	66 54 82	66(52,81)	0.783	69(58,88.25)	65(52,79)	0.026
TP Median (Q1,Q3)	72.5 68.1 77.3	71.2(68.4,77)	0.38	73.05(68.48,78.98)	72.37(67.8,77)	0.383
ALB Median (Q1,Q3)	42.1 38.4 45.1	41.3(38.1,44.5)	0.3	41.85(37.68,45.23)	42.4(39.6,44.8)	0.244
AG Median (Q1,Q3)	1.36 1.2 1.55	1.34(1.17,1.51)	0.602	1.3(1.17,1.46)	1.39(1.26,1.58)	0.011
TBIL Median (Q1,Q3)	11.9 8.15 14	11(9.1,13.7)	0.844	10.85(7.38,13.45)	12.1(8.3,14.4)	0.326
DBIL Median (Q1,Q3)	4.2 3 5	4.4(3.2,5.1)	0.654	3.85(2.9,5.05)	4.3(3.1,5)	0.619
ALT Median (Q1,Q3)	17 12 22.1	16.9(11,23)	0.862	18(12,23.85)	16,1(11.3,22)	0.313
AST Median (Q1,Q3)	20 16 27	20(15,30)	0.72	23(17,33)	19(16,24.76)	0.006
ALP Median (Q1,Q3)	68 57 84	67(53,82)	0.296	68.5(60.75,83.25)	67(56,84)	0.616
GCT Median (Q1,Q3)	24 16 37	20(16,30)	0.082	23(16.75,39.25)	24(16,37)	0.994
LDH Median (Q1,Q3)	200.5 170 226	197(168,224)	0.579	205(187.75,237)	195(163,219)	0.001
ADA Median (Q1,Q3)	10.12 7 12	9(7,13.25)	0.555	10.12(9,12.25)	9(7,112)	0.016
FIB Median (Q1,Q3)	3.37 2.89 3.85	3.3(2.81,3.36)	0.998	3.51(3.02,4.15)	3.28(2.83,3.62)	0.006
D-D Median (Q1,Q3)	1650 780 2863	1400(780,2460)	0.286	2380(1232.5,3942,5)	1380(570,2863)	0.004
BMI Median (Q1,Q3)	22.94(20.96,24,79)	22.86(20.92,24.42)	0.466	22.18(20.49,24.38)	23.53(21.22,24.92)	0.069
Median (Q1,Q3)	59(48,70)	59(49,69)	0.649	61.5(48.5,77)	58(48,70)	0.185
Median (Q1,Q3)	80(72,89)	75(67,89)	0.166	83.5(72.75,96.5)	79(72,87)	0.03
Median (Q1,Q3)	20(16,22)	19(16,20)	0.053	20(18,23)	19(16,21)	0.053
NLR Median (Q1,Q3)	11.24(6.23,18.18)	9.6(6.47,16.40)	0.589	12.92(8.13,21.34)	10.18(5.46,15.65)	0.005
d-NLR Median (Q1,Q3)	7.74(4.91,11.21)	6.74(5.08,10.33)	0.555	8.78(5.83,12.01)	7.41(4.44.10.88)	0.041
PLR Median (Q1,Q3)	231.68(144.02,302)	214.49(156.41,317.46)	0.847	247.70(140.71,347.56)	230(145.52,290)	<0.01
SII Median (Q1,Q3)	2618(1351.40,4291.76)	2274.22(1382.68,3661.81)	0.446	3969.8(2323.8,6874.1)	2534.6(1180.7,3747.8)	<0.01
SIINI Median (Q1,Q3)	350.35(201.72,566.75)	321.67(206.38,482.09)	0.424	422.54(242.14,764.18)	316.85(158.54,473.20)	0.009
PNI Median (Q1,Q3)	48.1(44.6,51.2)	47.15(44.15,50.35)	0.19	47.65(41.95,50.53)	48.3(45.3,51.4)	0.076
SIRI Median (Q1,Q3)	5.59(2.85,11.96)	5.19(3.25,9.84)	0.966	7.22(4.56,16.88)	4.68(2.02,8.26)	<0.01

Hunt-Hessn n%)	<3	119(58.6%)	58((66.7%)	0.488	25(35)	97(72)	<0.001
	≥3	84(41.4%)	29(33.3%)		47(65)	34(28)	
GCS	<8	20	6	0.728	14 29	6(4)	<0.01
	≥8	83	81		58(71)	125(96)	
RDWAGR		9.82(8.54,11.52)	9.77(8.23,11.86)	0.799	10.75(9.1,12.61)	9.56(8.17,10.75)	0.001

Table 3.Multivariate Logistic Regression Analysis of patients with pulmonary infection after aneurysm embolization. OR: odd ratio; CI: confdence interval

Variables	OR	95%CI	P-value
Age	1.053	1.019-1.089	0.002
PLR	1.006	1.002-1.011	0.002
SIRI	1.047	1.003-1.092	0.035
RDWAGR	1.14	1.007-1.289	0.038
CREA	1.015	1.001-1.03	0.042

Figures

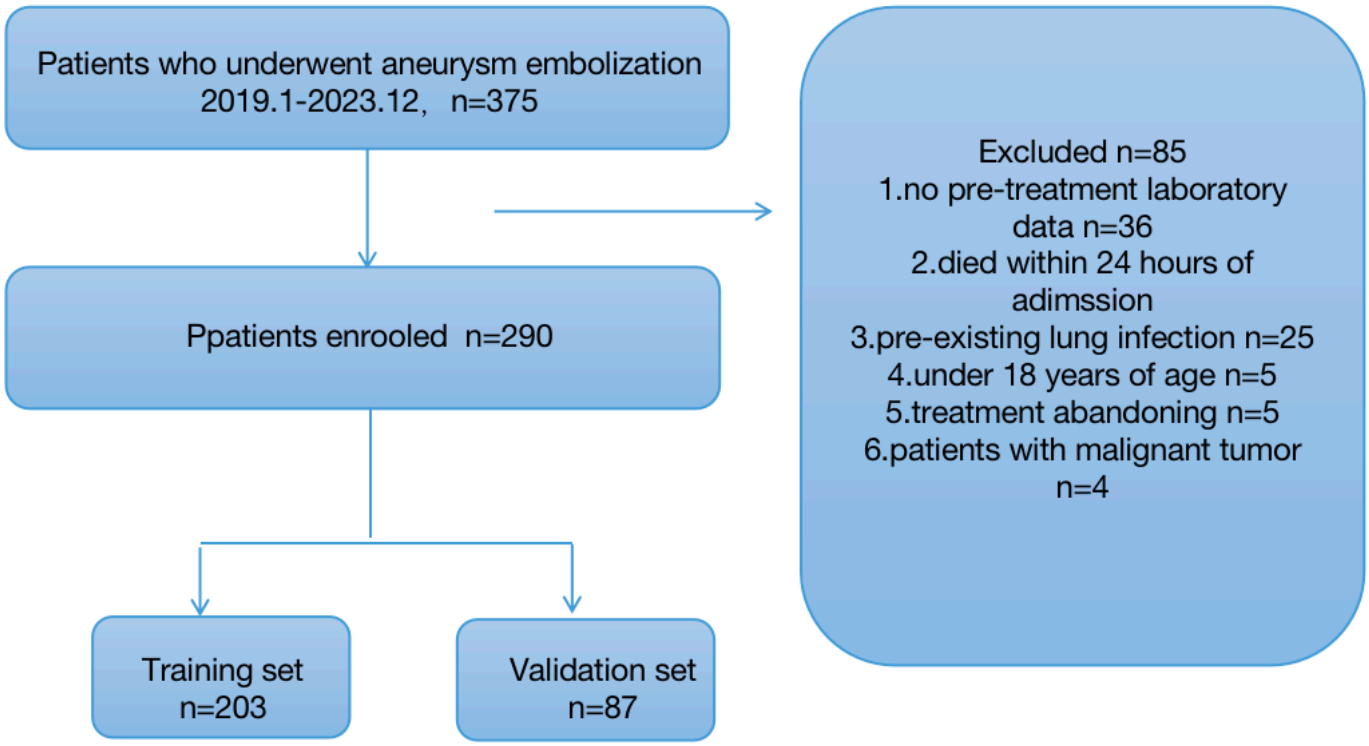


Figure 1

Diagram of Patient Selection Procedure for the Research Study

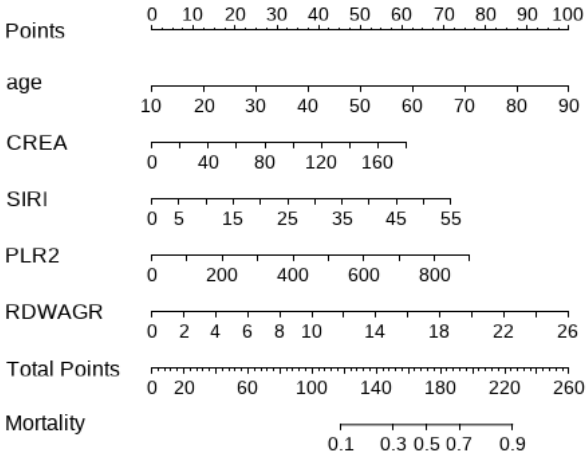
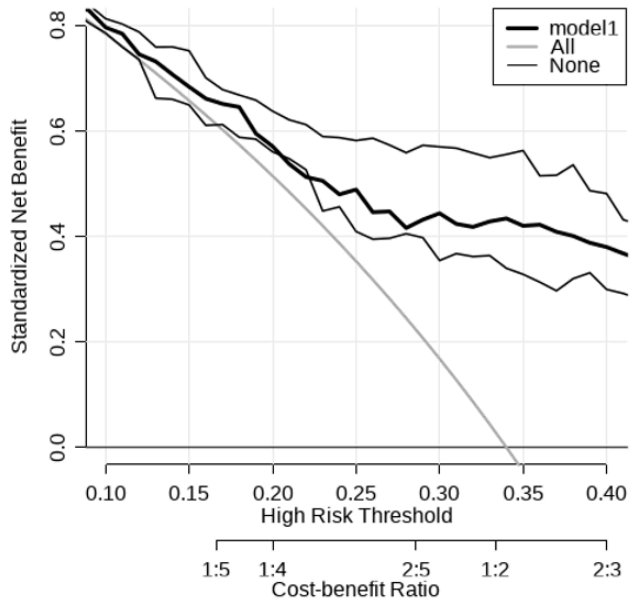


Figure 2

Nomogram for predicting pulmonary infection after aneurysm embolization.

A



B

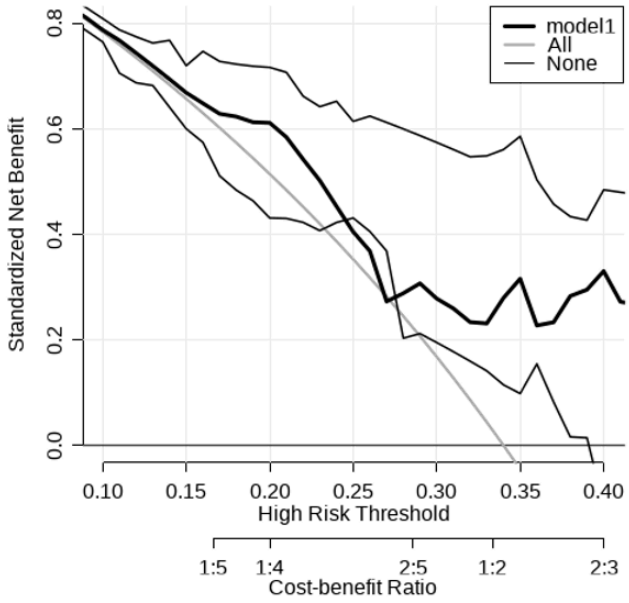


Figure 3

ROC curves and calibration curves of nomogram for the composite inflammatory and nutritional markers for predicting pulmonary infection after aneurysm embolization.(A) ROC curve of the nomogram in the training sett; (B) ROC curve of the nomogram in the validation set; (C) the calibration curve of nomogram in the training set; (D) the calibration curve of the nomogram in the validation set

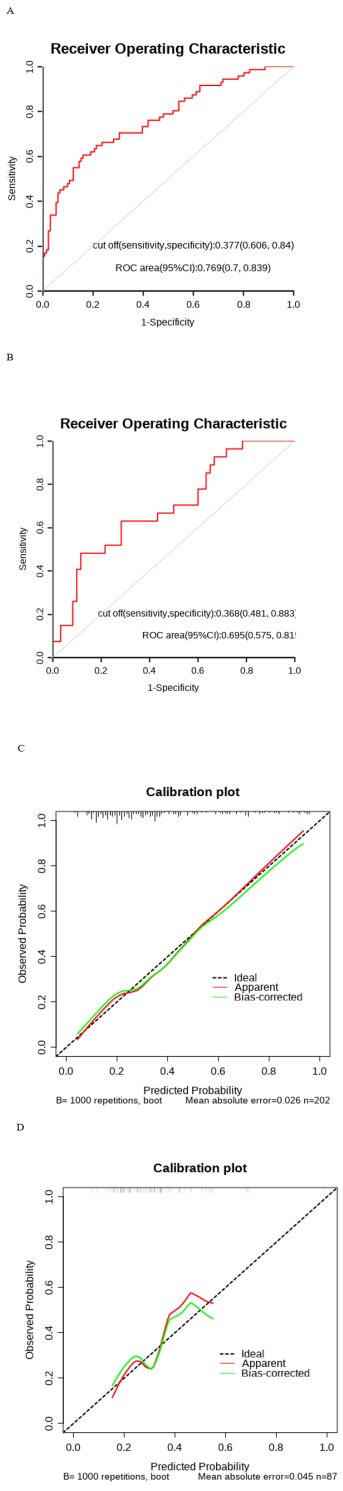


Figure 4

Decision curve analysis of the nomogram for prediction of pulmonary infection. (A) Training set; (B) validation set

Supplementary Files

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- ORIGINALDATA1.xlsx