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Relationship of NLR, Th17/Treg with the severity and prognosis of acute pancreatitis

SU Hongxia, LI Zhuan, HAN Haijing, JIANG Yongqiang, WANG Chunying

Department of Gastroenterology, China North Industries Corporation (NORINCO) General Hospital, Xi'an, Shaanxi 710065, China

Corresponding author: LI Zhuan, E-mail: 18329906464@163.com

Abstract: Objective To explore the correlations of neutrophil-to-lymphocyte ratio (NLR), T helper 17 (Th17) cell to regulatory T cell (Treg) ratio with disease severity of acute pancreatitis (AP), and analyze their predictive value for prognosis. **Methods** A total of 152 AP patients admitted to NORINCO General Hospital between March 2021 and March 2025 were enrolled. On the one hand, patients were classified into 3 groups, including mild acute pancreatitis (MAP, *n*=51), moderately severe acute pancreatitis (MSAP, *n*=56), and severe acute pancreatitis (SAP, *n*=45) according to the severity of the disease. On the other hand, patients were divided into poor prognosis and good prognosis groups based on clinical outcomes during hospitalization (followed up for 1 month or death) . The whole blood NLR and peripheral blood Th17/Treg levels were measured and compared among different severity groups or between different prognosis groups, respectively. Receiver operating characteristic (ROC) curves were plotted to analyze the prognosis predictive value of each index for AP. Kaplan-Meier method was used to draw survival curves for prognostic evaluation. **Results** Whole blood NLR and peripheral Th17/Treg levels progressively increased in MAP, MSAP, and SAP groups, with statistically significant differences (P<0.05) . Among 152 patients, 42 had poor prognosis and 110 had good prognosis. The poor prognosis group showed significantly higher NLR (11.07±3.98 *vs* 8.71±2.71, t=3.542, P<0.05) and Th17/Treg (3.34±0.52 *vs* 2.95±0.54, t=2.528, P<0.05) levels than those in the good prognosis group. ROC analysis demonstrated that the area under the curve (AUC) of combined NLR and Th17/Treg for predicting the prognosis of patients with AP was 0.835 (95%*CI*: 0.766-0.890), which was higher than that of NLR alone (AUC=0.638, 95%*CI*: 0.556-0.714) and Th17/Treg alone (AUC=0.662, 95%*CI*: 0.581-0.736) .Kaplan-Meier survival analysis revealed that patients with elevated NLR or Th17/Treg levels exhibited markedly poor prognoses (P<0.05) . **Conclusion** The levels of NLR and Th17/Treg in AP patients increased with the aggravation of disease severity. The combined application of NLR and Th17/Treg demonstrates substantial clinical value in predicting prognosis, providing guidance for clinical management of AP.

Keywords: Acute pancreatitis; Neutrophil-to-lymphocyte ratio; Helper T cell 17 to regulatory T cell ratio; Diagnosis; Prognosis; Severity

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Acute pancreatitis (AP) is a self-digestive pancreatic disease caused by the abnormal activation of pancreatic enzymes. Its clinical manifestations and disease progression exhibit significant heterogeneity, and it can be classified into mild acute pancreatitis (MAP), moderately severe acute pancreatitis (MSAP) and severe acute pancreatitis (SAP)[1]. Among them, 85% of patients have MAP with a favorable prognosis; however, 5%–10%may progress to SAP, with a mortality rate as high as 36%–50%, making clinical management extremely challenging [2-3]. Studies have shown that the progression of AP is closely associated with excessive inflammatory responses. Neutrophils directly damage tissues by releasing reactive oxygen species and proteolytic enzymes, while immune dysregulation of lymphocyte subsets further exacerbates the inflammatory cascade [4-5]. The neutrophil-to-lymphocyte ratio (NLR) reflects the degree of innate immune activation [6], whereas the T helper 17 cell (Th17)/regulatory T cell (Treg) ratio reflects adaptive immune imbalance [7]. Multiple clinical studies have confirmed that elevated NLR and Th17/Treg imbalance are both significantly associated with the clinical progression of AP [8-10]. Combined detection of NLR and Th17/Treg can simultaneously cover innate immune and adaptive immune imbalance, thereby providing a more comprehensive evaluation of the inflammatory status and immune dysregulation characteristics in patients with AP. This study aimed to analyze the predictive value of combined NLR and Th17/Treg detection for disease severity and prognosis in patients with AP.

1 Subjects and Methods

1.1 Study subjects

A total of 152 patients with acute pancreatitis (AP) treated at the General Hospital of Ordnance Industry from March 2021 to March 2025 were enrolled.

Inclusion criteria: (1) Meeting at least two of the three criteria from the *Guidelines for diagnosis and treatment of acute pancreatitis in China (2021)* [11]. ① Typical clinical manifestations including sudden, persistent severe upper abdominal pain, often radiating to the back, frequently accompanied by nausea, vomiting, and other gastrointestinal symptoms; ② Serum amylase and/or lipase ≥ 3 times the upper limit of normal; ③

Imaging features: contrast-enhanced CT/MRI showing inflammatory changes such as pancreatic enlargement, blurred margins, or peripancreatic fluid collections. (2) Age 18–75 years. (3) Admitted within 48 hours of disease onset. (4) Complete clinical data. (5) Patients and their families were informed of the study and signed informed consent.

Exclusion criteria: (1) Presence of serious comorbidities, including severe liver or kidney disease, malignant tumours, immune system diseases, haematological diseases, infectious diseases, etc. (2) Pregnant or breastfeeding women. (3) Patients who had received anticoagulant therapy or used immunomodulators within the past 3 months that might interfere with study indicators. (4) Incomplete clinical data or inability to cooperate with the study. This study was approved by the Ethics Committee of the General Hospital of Ordnance Industry (approval No.: 202102261102000383896).

According to the revised Atlanta classification in the *Guidelines for diagnosis and treatment of acute pancreatitis in China (2021)*, the 152 AP patients were divided into three groups based on disease severity: MAP group (n=51), MSAP group (n=56), and SAP group (n=45). There was no statistically significant difference in general data among the three groups (P>0.05). See **Table 1**.

Tab.1 Comparison of general information among three groups of patients [case(%)]

Indicator	MAP group (n=51)	MSAP group (n=56)	SAP group (n=45)	F/χ^2 value	P value
Gender					
Male	26 (50.98)	30 (53.57)	21 (46.67)	0.479	0.489
Female	25 (49.02)	26 (44.94)	24 (53.55)		
Age (year, $\bar{x}\pm s$)	45.08±9.60	46.30±10.93	44.33±9.94	0.484	0.617
BMI ($\bar{x}\pm s$)	22.45±2.41	22.07±2.33	22.03±2.17	0.506	0.604
Etiology					
Biliary	26 (50.98)	27 (48.21)	22 (48.89)		
Hyperlipidemia	16 (31.37)	17 (30.36)	13 (28.89)	0.385	0.825
Alcoholic and other causes	9 (17.65)	12 (21.43)	10 (22.22)		
Combined underlying diseases	35 (68.63)	40 (71.43)	32 (71.11)	2.392	0.122

1.2 Observation Indicators and Detection Methods

1.2.1 Blood Sample Collection

Immediately after admission, 3 mL of venous blood was collected from each patient and placed into two EDTA-K2 anticoagulant tubes. Among them, 2 mL was used for routine blood testing by an automated hematology analyzer (Sysmex Corporation, Japan, XN-900), and 1 mL was used for the detection of Th17 cells and Treg cells by a flow cytometer (Beckman Coulter, USA, MoFlo Astrios™ EQ).

1.2.2 NLR Calculation

A 2 mL whole blood sample was analyzed using an automated hematology analyzer under the complete blood count plus five-part white blood cell differential mode (DIFF channel). Neutrophil count (NEUT) and absolute lymphocyte count (LYMPH) were obtained. NLR = NEUT / LYMPH.

1.2.3 Th17/Treg Calculation

Four whole blood samples of 100 μL each were prepared and added with reagents respectively: (1) 20 μL CD3 PerCP, 20 μL CD4 FITC, 5 μL CD25 APC, and 20 μL CD127 PE; (2) 20 μL CD3 PerCP and 20 μL CD4 FITC; (3) 20 μL CD3 FITC, 20μL CD4 PE, and 20 μL IL-17 PerCP-Cy5.5; (4) 20 μL CD3FITC and 20 μL CD4 PE. After incubation at room temperature in the dark for 15 minutes,2 mL hemolysin was added. Ten minutes later, the samples were centrifuged at 1,500 r/min (centrifugal radius 16 cm) for 5 minutes, and the supernatant was discarded. The samples were then washed with 2 mL phosphate-buffered saline (PBS), centrifuged again under the same conditions for 5 minutes, the supernatant discarded, and finally resuspended in 500 μL PBS. The values of Treg cells (Treg/$CD4^+$ T%) and Th17 cells (Th17/$CD4^+$ T%) were measured using the respective instruments. Th17/Treg=(Th17/$CD4^+$T%)/(Treg/$CD4^+$T%) [12].

1.2.4 Assessment of AP severity

Within 24 hours of admission, patients underwent evaluation using the Acute Physiology and Chronic Health Evaluation II (APACHE II) score, Ranson score, Modified CT Severity Index (MCTSI) score, and Marshall Organ Failure (Marshall) score to determine the severity of AP. Higher APACHE II, Ranson, and MCTSI scores indicate more severe disease. A Marshall score >2 indicates organ failure [13].

1.2.5 Prognostic Evaluation Indicators

The follow-up period was 1 month (or until death). Based on the clinical outcomes during hospitalization, patients were divided into a good prognosis group and a poor prognosis group. (1) Good prognosis group: Patients showed significant improvement in their condition after systematic treatment, with stable vital signs, marked relief of clinical symptoms such as abdominal pain, laboratory parameters essentially returning to normal, imaging showing significant resolution of pancreatic inflammation, and no complications requiring hospitalization. (2) Poor prognosis group: Patients developed complications such as multiple organ dysfunction syndrome, infected pancreatic necrotic tissue, sepsis, acute respiratory distress syndrome, disseminated intravascular coagulation, or died during hospitalization.

1.3 Statistical Methods

SPSS 26.0 and MedCalc software were used for data analysis. The Shapiro-Wilk test was used to assess the normality of continuous variables. All variables conformed to normal distribution and were expressed as $\bar{x}\pm s$. Independent-sample t-tests were used for comparisons between two groups, while analysis of variance was used for comparisons among multiple groups. Post hoc pairwise comparisons were performed using the LSD-t test. Categorical data were expressed as number of cases (%) and analyzed using the chi-square test. Pearson correlation analysis was used to evaluate correlations between indicators and disease severity. Receiver

operating characteristic (ROC) curves were plotted to analyze the predictive value of each indicator for poor prognosis. Kaplan-Meier survival curves were used to assess the relationship between different NLR and Th17/Treg levels and the prognosis of AP patients. A *P* value less than 0.05 was considered significant.

2 Results

2.1 Comparison of NLR and Th17/Treg among the three groups

The whole-blood NLR and peripheral blood Th17/Treg levels in the MAP group, MSAP group, and SAP group increased progressively, and the differences were statistically significant ($P<0.05$). See **Table 2**.

2.2 Comparison of clinical scores among the three groups

The APACHE II score, Ranson score, MCTSI score, and Marshall score in the MAP group, MSAP group, and SAP group increased sequentially, and the differences were statistically significant ($P<0.05$). See **Table 3**.

2.3 Correlation of NLR and Th17/Treg with clinical scores

NLR and Th17/Treg were each positively correlated with the APACHE II score, Ranson score, MCTSI score, and Marshall score ($P<0.05$). See **Table 4.**

Tab.2 Comparison of NLR and Th17/Treg among three groups

Indicator	MAP group (*n*=51)	MSAP group (*n*=56)	SAP group (*n*=45)	*F* value	*P* value
NLR	7.81 ± 2.23	11.02 ± 2.85	13.12±3.98	37.050	< 0.01
Th17/Treg	2.88±0.52	3.13±0.57	3.65±0.41	29.093	< 0.01

Tab.3 Comparison of AP severity scores among three groups

Indicator	MAP group (*n*=51)	MSAP group (*n*=56)	SAP group (*n*=45)	*F* value	*P* value
APACHE II	6.41±1.23	9.62±2.65	16.42±3.97	159.471	< 0.01
Ranson Score	2.81±1.07	3.75±1.34	5.78±1.43	66.058	< 0.01
MCTSI	3.14±1.39	4.55±2.13	5.24±2.01	15.903	< 0.01
Marshall Score	1.03±0.23	2.31±0.84	3.46±1.25	95.905	< 0.01

Tab.4 Correlation analysis between NLR, Th17/Treg and clinical scores

Indicator	NLR		Th17/Treg	
	r value	*P* value	*r* value	*P* value
APACHE Ⅱ	0.355	< 0.01	0.288	< 0.01
Ranson Score	0.401	< 0.01	0.227	< 0.01
MCTSI	0.387	< 0.01	0.198	0.014
Marshall Score	0.422	< 0.01	0.297	< 0.01

2.4 Comparison of whole-blood NLR and peripheral blood Th17/Treg between different prognosis groups

After 1 month of follow-up, among the 152 AP patients, 110 had a good prognosis and 42 had a poor prognosis. The whole-blood NLR (11.07±3.98 *vs* 8.71±2.71, $t=3.542$, $P<0.001$) and peripheral blood Th17/Treg (3.34±0.52 *vs* 2.95±0.54, $t=2.528$, $P<0.001$) levels in the poor prognosis group were significantly higher than those in the good prognosis group.

2.5 Predictive value of NLR and Th17/Treg for poor prognosis in AP patients

ROC curve analysis showed that the area under the curve (AUC) of the combination of NLR and Th17/Treg for predicting poor prognosis in AP patients was 0.835 (95%CI: 0.766–0.890), which was higher than that of NLR alone (AUC=0.638, 95%CI: 0.556–0.714) and Th17/Treg alone (AUC=0.662, 95%CI: 0.581–0.736). See **Table 5** and **Figure 1**.

2.6 Impact of NLR and Th17/Treg on survival of AP patients

Based on the ROC curve results, 152 AP patients were divided into two groups: NLR > 10.031 ($n=56$) and NLR ⩽ 10.031 ($n=96$). Survival curve analysis showed that the 30-day survival rate of patients with high NLR was lower than that of patients with low NLR (log-rank $\chi^2=9.147$, P=0.003). Similarly, 152 AP patients were divided into two groups: Th17/Treg > 3.230 ($n=61$) and Th17/Treg ⩽ 3.230 ($n=91$). The 30-day survival rate of patients with high Th17/Treg was lower than that of patients with low Th17/Treg (log-rank $\chi^2=14.608$, $P<0.001$). See **Figure 2.**

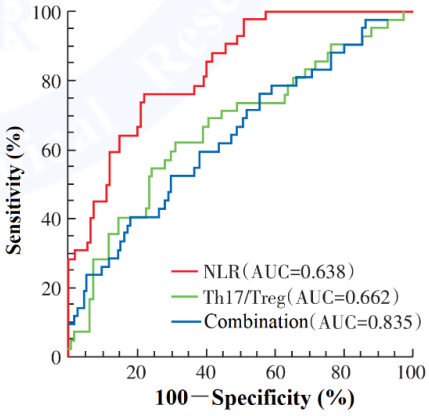
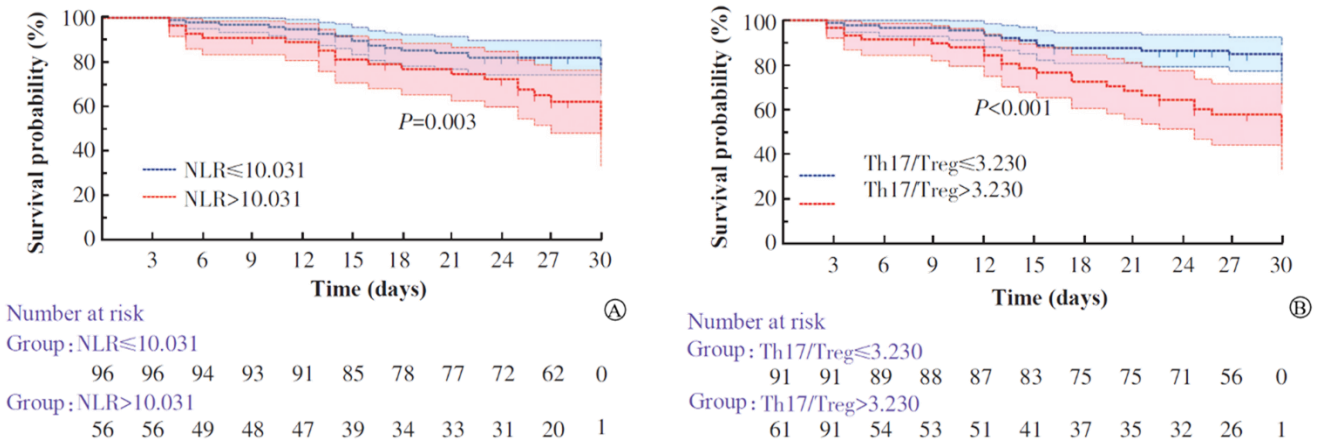


Fig.1 ROC curve of NLR and Th17/Treg in predicting the prognosis of AP patients

Tab.5 Prognostic value of combined detection of NLR and Th17/Treg in patients with acute pancreatitis

Indicator	Sensitivity (%)	Specificity (%)	AUC (95% *CI*)	Cut-off value	Youden's index	*Z* value	*P* value
NLR	52.38	70.00	0.638(0.556-0.714)	10.031	0.224	2.667	0.008
Th17/Treg	61.90	69.09	0.662(0.581-0.736)	3.230	0.310	3.149	0.002
Combination	76.19	78.18	0.835(0.766-0.890)			9.853	< 0.001



Note: A, NLR; B, Th 17/Treg.

Fig.2 Survival curves of AP patients with different levels ofNLR or Th17/Treg

3 Discussion

At present, the pathogenesis of APP has not yet been fully elucidated, and the various inflammatory cells, such as neutrophils, lymphocytes, macrophages, and dendritic cells, are abnormally activated and subsequently release large amounts of inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin (IL)-6, IL-1β, and others, thereby triggering a cascade amplification effect that ultimately leads to systemic inflammatory responses and organ damage [14–15]. As the first line of defense of the innate immune system, neutrophils are extensively activated and infiltrate pancreatic tissue during the early stage of AP onset. Through the release of proteolytic enzymes, such as elastase and myeloperoxidase, as well as reactive oxygen species, neutrophils directly induce injury to pancreatic acinar cells. At the same time, activated neutrophils secrete large amounts of pro-inflammatory cytokines, further amplifying the inflammatory response. As the disease progresses, this inflammatory response exhibits a cascade amplification effect, resulting in a significant increase in NEUT [14,16]. In addition, during SAP, inflammatory stress induces lymphocyte apoptosis, while the pro-inflammatory microenvironment suppresses lymphocyte proliferation [17–18]. The marked increase in NEUT together with the relative decrease in lymphocytes jointly leads to a sustained elevation of the NLR, which is positively correlated with disease severity. The results of the present study showed that whole blood NLR levels increased sequentially among the MAP group, MSAP group, and SAP group. These findings suggest that NLR levels are positively correlated with the severity of AP, with higher NLR values indicating more severe disease. NLR may serve as a simple and inexpensive inflammatory biomarker for assisting in the evaluation of disease severity and risk stratification in patients with AP. Furthermore, the APACHE II score and Ranson score directly reflect the degree of systemic inflammatory response, while the extent of pancreatic necrosis evaluated by the MCTSI is closely associated with the degree of neutrophil infiltration. Organ failure assessed by the Marshall score represents the terminal manifestation of systemic inflammatory responses. Correlation analysis also demonstrated that elevated NLR was positively correlated with these scoring systems evaluating systemic inflammation and organ injury. These findings further confirm the cascade amplification effect of inflammation in AP and additionally demonstrate the advantages of using NLR in the clinical assessment of AP severity.

During the occurrence and development of AP, $CD4^+$T lymphocytes play a major role. These cells secrete various lymphokines during the effector phase of cellular immunity and further differentiate into different subtypes, among which Th17 cells mediate inflammatory responses and Treg cells mediate immune tolerance. [19–20]. In the absence of inflammatory stimulation, transforming growth factor-beta 1 (TGF-β1) induces the differentiation of Th0 cells into Treg cells, thereby suppressing autoimmunity and maintaining immune tolerance within the immune system. When the body experiences injury, antigen-presenting cells, such as dendritic cells, promote the differentiation of $CD4^+$T cells into Th17 cells under stimulation by inflammatory cytokines. During this process, Treg cells may transiently increase in an attempt to suppress excessive inflammation. However, when injury persists, the pro-inflammatory effects of Th17 cells may become uncontrolled. Excessive activation of Th17 cells is accompanied by inhibition of Treg differentiation, resulting in Th17/Treg imbalance and disruption of the pro-inflammatory and anti-inflammatory equilibrium [19–21]. In the present study, peripheral blood Th17/Treg ratios increased sequentially among the MAP group, MSAP group, and SAP group. Correlation analysis further demonstrated that elevated Th17/Treg ratios were positively correlated with scoring systems evaluating systemic inflammation and organ injury.

In this study, the 30-day survival outcomes of 152 patients with AP were analyzed. The results showed that patients in the high-NLR group and those in the high-Th17/Treg group had poorer prognoses, suggesting that both biomarkers are associated with the mechanisms

underlying adverse prognostic outcomes in patients. ROC curve analysis demonstrated that the combined use of NLR and Th17/Treg had the highest predictive value for adverse prognosis in patients with AP, with an AUC of 0.835, sensitivity of 76.19%, and specificity of78.18%, indicating good diagnostic accuracy. Combined detection of these two biomarkers may serve as a reliable biomarker combination for evaluating the prognosis of AP patients and provide a new laboratory basis for clinical prognostic assessment. By reflecting the acute inflammatory state of innate immunity dominated by neutrophils, NLR can provide early warning of the intensity of systemic inflammatory responses, whereas Th17/Treg reflects dysregulation of adaptive immunity and can dynamically monitor the risk of chronic disease progression. The combination of these two indicators not only covers the entire process of AP from acute injury to persistent deterioration, but also integrates interactions across different immune system levels, thereby improving the sensitivity and specificity of the predictive model.

In summary, NLR and Th17/Treg levels in patients with AP are significantly positively correlated with disease severity, and the combination of NLR and Th17/Treg may be used to evaluate the prognosis of AP. Future multicenter clinical studies are needed to validate their synergistic value and to explore precision intervention strategies based on these biomarkers.

Conflict of Interest None

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·急性胰腺炎专题·论著·

NLR、Th17/Treg与急性胰腺炎严重程度及预后的关系

苏红霞，李转，韩海静，江永强，王春莹
兵器工业总医院消化内科，陕西 西安 710065

摘要：目的　探讨中性粒细胞与淋巴细胞比值（NLR）、辅助性T细胞（Th）17与调节性T细胞（Treg）比值（Th17/Treg）与急性胰腺炎（AP）病情严重程度的关系，及对预后的预测价值。**方法**　选取2021年3月至2025年3月兵器工业总医院收治的152例AP患者，依据病情分为轻症（MAP）51例、中度重症（MSAP）56例和重症（SAP）45例3组，依据患者住院期间的临床转归情况（随访1个月或死亡）分为预后良好组与预后不良组。对比全血NLR与外周血Th17/Treg在不同严重程度组、不同预后组之间的差异，绘制受试者工作特征（ROC）曲线分析各指标对AP患者预后的预测价值，采用Kaplan-Meier法绘制生存曲线评估预后。**结果**　全血NLR与外周血Th17/Treg在MAP、MSAP和SAP 3组中依次升高，差异有统计学意义（$P<0.05$）。152例AP患者中110例预后良好，42例预后不良，预后不良组患者NLR（11.07±3.98 *vs* 8.71±2.71，$t=3.542$，$P<0.05$）与Th17/Treg（3.34±0.52 *vs* 2.95±0.54，$t=2.528$，$P<0.05$）水平高于预后良好组。ROC曲线结果显示，NLR与Th17/Treg联合预测AP患者预后的曲线下面积（AUC）为0.835（95%*CI*：0.766~0.890），高于NLR（AUC=0.638，95%*CI*：0.556~0.714）与Th17/Treg（AUC=0.662，95%*CI*：0.581~0.736）单独预测。Kaplan-Meier生存曲线显示，高水平NLR或Th17/Treg患者的预后明显较差（$P<0.05$）。**结论**　AP患者NLR与Th17/Treg水平随病情严重程度升高而增加，NLR与Th17/Treg的联合应用在评估AP患者预后方面具有较高应用价值，有助于指导AP的临床诊治。

关键词：急性胰腺炎；中性粒细胞与淋巴细胞比值；辅助性T细胞17与调节性T细胞比值；诊断；预后；严重程度
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SU Hongxia, LI Zhuan, HAN Haijing, JIANG Yongqiang, WANG Chunying
Department of Gastroenterology, China North Industries Corporation (NORINCO) General Hospital, Xi'an, Shaanxi 710065, China
Corresponding author: LI Zhuan, E-mail: 18329906464@163.com

Abstract: Objective　To explore the correlations of neutrophil-to-lymphocyte ratio (NLR), T helper 17 (Th17) cell to regulatory T cell (Treg) ratio with disease severity of acute pancreatitis (AP), and analyze their predictive value for prognosis. **Methods**　A total of 152 AP patients admitted to NORINCO General Hospital between March 2021 and March 2025 were enrolled. On the one hand, patients were classified into 3 groups, including mild acute pancreatitis (MAP, $n=51$), moderately severe acute pancreatitis (MSAP, $n=56$), and severe acute pancreatitis (SAP, $n=45$) according to the severity of the disease. On the other hand, patients were divided into poor prognosis and good prognosis groups based on clinical outcomes during hospitalization (followed up for 1 month or death). The whole blood NLR and peripheral blood Th17/Treg levels were measured and compared among different severity groups or between different prognosis groups, respectively. Receiver operating characteristic (ROC) curves were plotted to analyze the prognosis predictive value of each index for AP. Kaplan-Meier method was used to draw survival curves for prognostic evaluation. **Results**　Whole blood NLR and peripheral Th17/Treg levels progressively increased in MAP, MSAP, and SAP groups, with statistically significant differences ($P<0.05$). Among 152 patients, 42 had poor prognosis and 110 had good prognosis. The poor prognosis group showed significantly higher NLR (11.07±3.98 *vs* 8.71±2.71, $t=3.542$, $P<0.05$) and

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通信作者：李转，E-mail：18329906464@163.com
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Th17/Treg (3.34±0.52 *vs* 2.95±0.54, t=2.528, P<0.05) levels than those in the good prognosis group. ROC analysis demonstrated that the area under the curve (AUC) of combined NLR and Th17/Treg for predicting the prognosis of patients with AP was 0.835 (95%*CI*: 0.766-0.890), which was higher than that of NLR alone (AUC=0.638, 95%*CI*: 0.556-0.714) and Th17/Treg alone (AUC=0.662, 95%*CI*: 0.581-0.736). Kaplan-Meier survival analysis revealed that patients with elevated NLR or Th17/Treg levels exhibited markedly poor prognoses (P<0.05). **Conclusion** The levels of NLR and Th17/Treg in AP patients increased with the aggravation of disease severity. The combined application of NLR and Th17/Treg demonstrates substantial clinical value in predicting prognosis, providing guidance for clinical management of AP.

Keywords: Acute pancreatitis; Neutrophil-to-lymphocyte ratio; Helper T cell 17 to regulatory T cell ratio; Diagnosis; Prognosis; Severity

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急性胰腺炎(acute pancreatitis, AP)是一种由胰酶异常激活引发的胰腺自身消化性疾病,其临床症状和疾病进程存在显著异质性,可分为轻症AP(mild acute pancreatitis, MAP)、中度重症AP(moderately severe acute pancreatitis, MSAP)和重症AP(severe acute pancreatitis, SAP)[1]。其中,80%~85%的患者为MAP,预后良好;但5%~10%可进展为SAP,病死率为36%~50%,临床救治极具挑战性[2-3]。研究表明,AP的进展与过度炎症反应密切相关,中性粒细胞通过释放活性氧和蛋白水解酶直接损伤组织,而淋巴细胞亚群的免疫调节失衡进一步加剧炎症级联反应[4-5]。中性粒细胞与淋巴细胞比值(neutrophil-to-lymphocyte ratio, NLR)可反映先天免疫激活程度[6],而辅助性T细胞17(T helper 17 cell, Th17)/调节性T细胞(regulatory T cell, Treg)比值则体现适应性免疫失衡[7]。多项临床数据证实,NLR升高和Th17/Treg失衡均与AP临床进展显著相关[8-10]。联合NLR与Th17/Treg检测可以同时覆盖先天免疫和适应性免疫失衡,能更全面地评估AP患者的炎症状态和免疫紊乱特征。本研究旨在分析NLR与Th17/Treg联合检测对AP患者病情严重程度及预后的预测价值。

1 资料与方法

1.1 研究对象 纳入2021年3月至2025年3月于兵器工业总医院就诊的152例AP患者。纳入标准:(1)满足《中国急性胰腺炎诊治指南(2021)》[11]三项标准中至少两项,①典型临床表现为突发持续性上腹剧痛,常放射至背部,多伴恶心、呕吐等消化道症状;②血清淀粉酶和/或脂肪酶≥正常值上限3倍;③影像学特征,增强CT/核磁共振成像(MRI)显示胰腺肿大、边缘模糊或胰周积液等炎症改变。(2)年龄18~75岁。(3)发病48 h内入院。(4)临床资料完整。(5)患者及家属知晓本研究并签署同意书。排除标准:(1)有严重合并症,包括严重肝肾疾病、恶性肿瘤、免疫系统疾病、血液系统疾病、传染性疾病等;(2)妊娠期、哺乳期女性;(3)近3个月内接受抗凝治疗或使用免疫调节剂可能干扰研究指标的患者;(4)临床资料缺失或不能配合完成研究者。本研究已获得兵器工业总医院伦理委员会批准(批号:20210226110200038396)。

根据《中国急性胰腺炎诊治指南(2021)》中的修订版Atlanta分级,将152例AP患者依据病情分为MAP组(51例)、MSAP组(56例)和SAP组(45例)。3组患者一般资料比较差异无统计学意义(P>0.05)。见表1。

1.2 观察指标及检测方法

1.2.1 血液样本采集 患者入院后立即采集静脉血3 mL,分别置于两个EDTA-K2抗凝管中,2 mL用于全自动血细胞分析仪(日本Sysmex公司,XN-900)检测血常规,1 mL用于流式细胞仪(美国Beckman Coulter公司,MoFlo AstriosTM EQ)检测Th17细胞与Treg细

表1 3组患者一般资料比较 [例(%)]

Tab.1 Comparison of general information among three groups of patients [case(%)]

项目	MAP组(n=51)	MSAP组(n=56)	SAP组(n=45)	F/χ^2值	P值
男/女(例)	26/25	30/26	21/24	0.479	0.489
年龄(岁, $\bar{x}\pm s$)	45.08±9.60	46.30±10.93	44.33±9.94	0.484	0.617
BMI(kg/m^2, $\bar{x}\pm s$)	22.45±2.41	22.07±2.33	22.03±2.17	0.506	0.604
病因					
胆源性	26(50.98)	27(48.21)	22(48.89)		
高脂血症	16(31.37)	17(30.36)	13(28.89)	0.385	0.825
酒精性及其他	9(17.65)	12(21.43)	10(22.22)		
合并基础病	35(68.63)	40(71.43)	32(71.11)	2.392	0.122

注:BMI为身体质量指数。

胞数量。

1.2.2　NLR 计算　取 2 mL 全血样本于全自动血细胞分析仪进样，选择全血细胞计数+白细胞五分类检测模式（DIFF 通道），获取中性粒细胞计数（neutrophil count，NEUT）与淋巴细胞绝对值（lymphocyte，LYMPH），NLR=NEUT / LYMPH。

1.2.3　Th17/Treg 计算　取 4 支全血样本，各 100 μL，分别加入：（1）20 μL CD3 PerCP、20 μL CD4 FITC、5 μL CD25 APC 和 20 μL CD127 PE；（2）20 μL CD3 PerCP 和 20 μL CD4 FITC；（3）20 μL CD3 FITC、20 μL CD4 PE 和 20 μL IL-17 PerCP-Cy5.5；（4）20 μL CD3 FITC 和 20 μL CD4 PE。样本避光室温放置 15 min 后加入 2 mL 溶血素，10 min 后以 1 500 r/min 转速（离心半径 16 cm）离心 5 min 后，弃去上清，加入 2 mL 磷酸盐缓冲液洗涤，相同离心条件下，离心 5 min 后弃去上清，并加入 5 00 μL PBS 缓冲液重悬。分别上机检测 Treg 细胞（Treg/CD4$^+$T%）和 Th17 细胞（Th17/CD4$^+$T%）的数值。Th17/Treg=（Th17/CD4$^+$T%）/（Treg/CD4$^+$T%）[12]。

1.2.4　AP 严重程度判断　患者入院 24 h 内进行急性生理学与慢性健康状况评分Ⅱ（Acute Physiology and Chronic Health Evaluation Ⅱ，APACHE Ⅱ）、Ranson 评分、改良 CT 严重指数（Modified CT Severity Index，MCTSI）评分以及马歇尔器官功能衰竭（Marshall Organ Failure，Marshall）评分评估，以判断 AP 严重程度。APACHE Ⅱ、Ranson、MCTSI 评分分值越高，患者病情越严重，Marshall 评分>2 分提示器官衰竭[13]。

1.2.5　预后判断指标　随访 1 个月（或死亡）为期限，根据患者住院期间的临床转归情况，将患者分为预后良好组与预后不良组。（1）预后良好组：患者经过系统治疗后病情显著改善，生命体征平稳，腹痛等临床症状明显缓解，实验室指标基本恢复正常，影像学显示胰腺炎症明显吸收，无需要住院治疗的并发症；（2）预后不良组：患者住院期间发生多器官功能障碍综合征、胰腺坏死组织感染、脓毒症、急性呼吸窘迫综合征、弥散性血管内凝血等并发症或死亡。

1.3　统计学方法　采用 SPSS 26.0、MedCalc 软件分析数据。用 Shapiro-Wilk 检验评估连续变量的正态性，均符合正态分布，用 $\bar{x}\pm s$ 表示，两组比较采用独立样本 t 检验，多组比较采用方差分析，事后两两比较采用 LSD-t 检验；计数资料用例（%）表示，比较采用 χ^2 检验。采用 Pearson 相关分析各指标与病情严重程度的相关性，绘制受试者工作特征（receiver operating characteristic，ROC）曲线分析各指标对预后不良的预测价值，采用 Kaplan-Meier 法绘制生存曲线评估不同 NLR、Th17/Treg 水平与 AP 患者预后的关系。P<0.05 为差异有统计学意义。

2　结　果

2.1　3 组 NLR 与 Th17/Treg 比较　MAP、MSAP 和 SAP 3 组全血 NLR 与外周血 Th17/Treg 水平依次增高，差异有统计学意义（P<0.05）。见表 2。

2.2　3 组患者临床评分比较　MAP、MSAP 和 SAP 3 组 APACHE Ⅱ评分、Ranson 评分、MCTSI 评分、Marshall 评分依次升高，差异有统计学意义（P<0.05）。见表 3。

2.3　NLR、Th17/Treg 与临床评分的相关性　NLR、Th17/Treg 分别与 APACHE Ⅱ评分、Ranson 评分、MCTSI 评分、Marshall 评分呈正相关（P<0.05）。见表 4。

2.4　不同预后组全血 NLR 与外周血 Th17/Treg 比较　经 1 个月随访，152 例 AP 患者中 110 例预后良好，42 例预后不良。预后不良组全血 NLR（11.07±3.98 *vs* 8.71±2.71，t=3.542，P<0.001）以及外周血 Th17/Treg（3.34±0.52 *vs* 2.95±0.54，t=2.528，P<0.001）显著高于预后良好组。

表 2　3 组患者 NLR 与 Th17/Treg 比较　（$\bar{x}\pm s$）

Tab.2　Comparison of NLR and Th17/Treg among three groups of patients　（$\bar{x}\pm s$）

项目	MAP 组（n=51）	MSAP 组（n=56）	SAP 组（n=45）	F 值	P 值
NLR	7.81±2.23	11.02±2.85^a	13.12±3.98^{ab}	37.050	<0.001
Th17/Treg	2.88±0.52	3.13±0.57^a	3.65±0.41^{ab}	29.093	<0.001

注：与 MAP 组比较，^{a}P<0.05；与 MSAP 组比较，^{b}P<0.05。

表 3　3 组患者 AP 严重程度评分比较　（$\bar{x}\pm s$）

Tab.3　Comparison of AP severity scores among three groups of patients　（$\bar{x}\pm s$）

项目	MAP 组（n=51）	MSAP 组（n=56）	SAP 组（n=45）	F 值	P 值
APACHE Ⅱ评分	6.41±1.23	9.62±2.65^a	16.42±3.97^{ab}	159.471	<0.001
Ranson 评分	2.81±1.07	3.75±1.34^a	5.78±1.43^{ab}	66.058	<0.001
MCTSI 评分	3.14±1.39	4.55±2.13^a	5.24±2.01^{ab}	15.903	<0.001
Marshall 评分	1.03±0.23	2.31±0.84^a	3.46±1.25^{ab}	95.905	<0.001

注：与 MAP 组比较，^{a}P<0.05；与 MSAP 组比较，^{b}P<0.05。

表 4　NLR、Th17/Treg 与临床评分的相关性分析

Tab.4　Correlation analysis between NLR, Th17/Treg and clinical scores

参数	NLR		Th17/Treg	
	r 值	P 值	r 值	P 值
APACHE Ⅱ评分	0.355	<0.01	0.288	<0.01
Ranson 评分	0.401	<0.01	0.227	<0.01
MCTSI 评分	0.387	<0.01	0.198	<0.05
Marshall 评分	0.422	<0.01	0.297	<0.01

2.5　NLR 与 Th17/Treg 对 AP 患者预后不良的预测价值　ROC 曲线分析显示，NLR 与 Th17/Treg 联合预测 AP 患者预后不良的曲线下面积（area under the curve，AUC）为 0.835（95%*CI*：0.766~0.890），高于 NLR（AUC=0.638，95%*CI*：0.556~0.714）与 Th17/Treg（AUC=0.662，95%*CI*：0.581~0.736）单独预测。见表 5、图 1。

2.6　NLR 与 Th17/Treg 对 AP 患者生存的影响　根据 ROC 曲线结果，将 152 例 AP 患者分为 NLR>10.031（56 例）与 NLR≤10.031（96 例）两组，生存曲线分析结果显示，高 NLR 的患者 30 d 生存率低于低 NLR 的患者（log-rank χ^2=9.147，*P*=0.003）。同样，将 152 例 AP 患者分为 Th17/Treg>3.230（61 例）与 Th17/Treg≤3.230（91 例）两组，高 Th17/Treg 患者的 30 d 生存率低于低 Th17/Treg 组（log-rank χ^2=14.608，*P*<0.001）。见图 2。

表 5　NLR 与 Th17/Treg 联合检测对 AP 患者预后的预测价值

Tab.5　Prognostic value of combined detection of NLR and Th17/Treg in patients with AP

项目	敏感度（%）	特异度（%）	AUC（95%*CI*）	截断值	约登指数	*Z* 值	*P* 值
NLR	52.38	70.00	0.638（0.556~0.714）	10.031	0.224	2.667	0.008
Th17/Treg	61.90	69.09	0.662（0.581~0.736）	3.230	0.310	3.149	0.002
二者联合	76.19	78.18	0.835（0.766~0.890）	–	–	9.853	<0.001

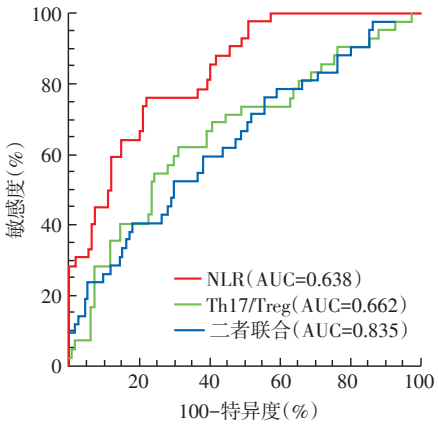

图 1　NLR 与 Th17/Treg 预测 AP 患者预后的 ROC 曲线

Fig.1　ROC curve of NLR and Th17/Treg in predicting the prognosis of AP patients

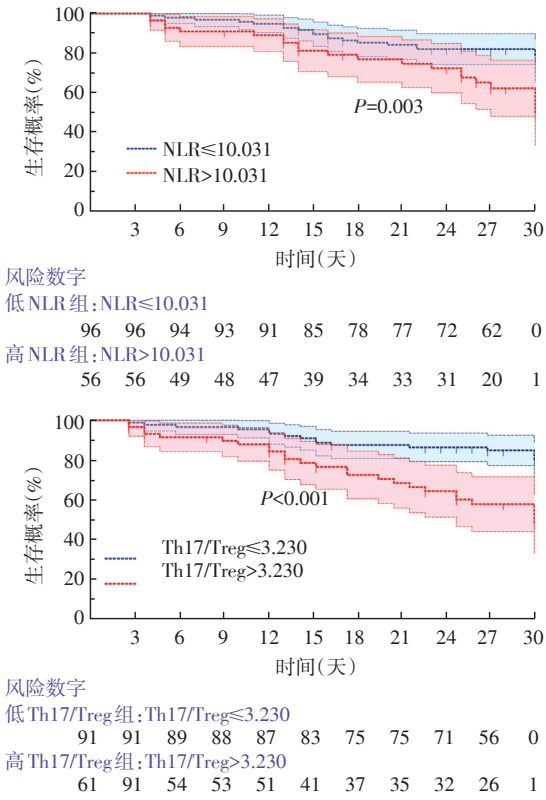

图 2　不同 NLR 或 Th17/Treg 水平 AP 患者的生存曲线

Fig.2　Survival curves of AP patients with different levels of NLR or Th17/Treg

3　讨　论

目前，AP 的发病机制尚未完全阐明，炎症反应过度激活学说已成为研究热点。该学说认为多种炎症细胞（如中性粒细胞、淋巴细胞、巨噬细胞及树突状细胞）被异常激活后，通过释放大量炎症因子，如肿瘤坏死因子α（TNF-α）、白细胞介素（IL）-6、IL-1β等，触发级联放大效应，最终导致全身炎症反应及器官损伤[14-15]。中性粒细胞作为先天免疫系统的第一道防线，在 AP 发病早期即被大量激活并浸润胰腺组织。其通过释放弹性蛋白酶、髓过氧化物酶等蛋白水解酶和活性氧自由基，直接导致胰腺腺泡细胞损伤。同时，活化的中性粒细胞分泌大量促炎因子，进一步放大炎症反应。随着病情加重，这种炎症反应呈现级联放大效应，导致 NEUT 显著增加[14, 16]。除此之外，在 SAP 时，炎症应激诱导淋巴细胞凋亡，促炎微环境抑制淋巴细胞增殖[17-18]。NEUT 的显著增加与淋巴细胞相对减少，共同导致 NLR 持续升高，与疾病严重程度呈正相关。本研究结果显示，全血 NLR 在 MAP、MSAP 和 SAP 3 组中依次升高，该结果提示 NLR 水平与 AP 的严重程度呈正相关，NLR 越高，疾病越严重。NLR 可能作为一种简便、价廉的炎症标志物，用于辅助评估 AP 患者的病情严重程度和风险分层。此外，APACHE Ⅱ 和 Ranson 评分直接反映全身炎症反应程度，MCTSI 评估的胰腺坏死范围与中性粒

细胞浸润程度密切相关，Marshall 评分中的器官功能衰竭更是全身炎症反应的终末表现。相关性研究结果也显示，NLR 升高与这些评估全身炎症和器官损伤的评分呈正相关，这些结果印证了 AP 中的炎症级联放大效应，也进一步显示采用 NLR 作为临床评估 AP 严重程度的优势。

在 AP 发生发展过程中主要发挥作用的是 $CD4^+$ T 淋巴细胞，该细胞在细胞免疫的效应阶段中能分泌多种淋巴因子并进一步分化为不同的亚型细胞，其中 Th17 细胞介导炎症反应，Treg 细胞介导免疫耐受[19-20]。在机体没有受到炎症刺激时，TGF-β1 诱导 Th0 分化为 Treg 细胞，抑制自身免疫来维持机体免疫系统的自身免疫耐受。在机体受到损伤时，抗原呈递细胞（如树突状细胞）在炎症因子刺激下，促进 $CD4^+$ T 细胞分化为 Th17 细胞，Treg 细胞可能短暂增多，试图抑制过度炎症。当损伤持续时，Th17 细胞的促炎作用可能失控，Th17 过度活化伴随着 Treg 分化被抑制，即 Th17/Treg 细胞失衡，促炎-抗炎平衡破坏[19-21]。在本研究中，外周血 Th17/Treg 在 MAP、MSAP 和 SAP 3 组中依次升高，相关性分析也显示，Th17/Treg 升高与这些评估全身炎症和器官损伤的评分呈正相关。

本研究对 152 例 AP 患者的 30 d 生存情况进行分析，结果显示，高水平 NLR 组患者与高水平 Th17/Treg 组患者的预后较差，提示这两种标志物均与患者不良预后的发生机制有关。ROC 曲线分析显示，NLR 与 Th17/Treg 联合对 AP 患者不良预后的预测价值最高，其 AUC 达到 0.835，敏感度为 76.19%，特异度为 78.18%，具有较好的诊断准确性，两者联合检测可作为评估 AP 患者预后的可靠标志物组合，为临床预后评估提供了新的实验室依据。NLR 通过反映中性粒细胞主导的先天免疫急性炎症状态，能够早期预警全身炎症反应强度，而 Th17/Treg 则揭示适应性免疫的调控失衡，可动态监测疾病慢性化风险。二者联合既覆盖了 AP 从急性损伤到持续恶化的全过程，又整合了不同免疫层级的交互作用，使预测模型具有更高的敏感性和特异性。

综上所述，AP 患者 NLR 与 Th17/Treg 水平与其病情严重程度呈显著正相关，NLR 与 Th17/Treg 的联合可用于评估 AP 预后。未来需通过多中心临床研究验证其协同价值，并探索基于此的精准干预策略。

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