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ORIGINAL RESEARCH

Analysis of Predictive Value of Cellular Inflammatory Factors and T Cell Subsets for Disease Recurrence and Prognosis in Patients with Acute Exacerbations of COPD

Haoran Deng, Shiping Zhu, Fei Yu, Xue Song, Xinlai Jin, Xuchun Ding

Department of Respiratory and Critical Care Medicine, Hangzhou TCM Hospital Affiliated to Zhejiang Chinese Medical University, Hangzhou City, Zhejiang Province, People's Republic of China

Correspondence: Xuchun Ding, Email archon_01@163.com

Objective: To explore the predictive value of cellular inflammatory factors and T cell subsets for disease recurrence and prognosis in patients with acute exacerbations of chronic obstructive pulmonary disease (COPD).

Methods: Serum samples were collected from the two groups to detect and compare the levels of inflammatory cytokines [interleukin-1β (IL-1β), interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α)], T cell subsets (CD4+, CD8+), and clinical related indicators. Pearson correlation analysis was used to analyze the correlation between inflammatory cytokines, T cell subsets, and clinical indicators. Receiver operating characteristic (ROC) curves were plotted to analyze the predictive value of serum inflammatory factors and T cell subsets for acute exacerbations of COPD.

Results: The observation group had higher levels of IL-1β, IL-6, TNF-α, and CD8+, and lower CD4+ levels ($P<0.05$). The ratio of forced expiratory volume in 1 second to forced vital capacity (FEV1/FVC) was lower, while procalcitonin (PCT) and white blood cell count (WBC) were higher ($P<0.05$). Correlation analysis showed positive correlations between IL-1β, IL-6, TNF-α, and CD8+, and negative correlations with CD4+ and FEV1/FVC ($P<0.05$). After 6 months, 15 out of 73 patients had acute recurrences, with higher IL-1β, IL-6, TNF-α, and CD8+ levels ($P<0.05$). Binary logistic regression identified IL-1β, IL-6, TNF-α, and CD8+ as significant predictors of exacerbations, while CD4+ was protective. ROC analysis showed that combined biomarkers had the highest predictive efficiency (AUC = 0.907).

Conclusion: This study is the first to integrate multiple serum inflammatory factors and T cell subsets into a comprehensive predictive model for acute recurrence of COPD within six months (AUC = 0.907), offering a more accurate prediction than traditional methods. The findings underscore the value of these biomarkers in clinical follow-up and highlight their independent predictive power, providing new insights into the interaction between immune markers and clinical indicators in COPD exacerbations.

Keywords: Cellular inflammatory factors, T cell subsets, COPD, acute phase, disease recurrence, predictive value, prognosis

Introduction

Chronic obstructive pulmonary disease (COPD) is a common respiratory condition characterized by persistent airflow limitation and an underlying state of chronic inflammation. Acute exacerbations of COPD (AECOPD) are episodes of symptom worsening, including increased dyspnea, cough, and sputum production, leading to frequent hospitalizations and significantly impacting patient prognosis. These acute events contribute not only to a rapid decline in lung function but also to an increased risk of mortality and long-term disability.[1] Clinical studies[2] indicate that acute exacerbations are closely related to the long-term prognosis of COPD, and the frequency and severity of exacerbations have a significant impact on the overall prognosis of patients. Moreover, The burden of AECOPD on both individual health and healthcare systems is substantial, making the prediction and prevention of exacerbations a critical priority in COPD management.[3] Recent research has emphasized the heterogeneous nature of COPD, proposing an updated definition and taxonomy that

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accounts for various pathological processes beyond traditional causes like smoking. This new framework aims to improve early identification and targeted interventions by considering the diverse mechanisms contributing to COPD progression.[4] Currently, the prediction of acute exacerbations of COPD primarily relies on traditional methods like medical history and pulmonary function tests, but these often lack accuracy and timeliness, highlighting the need for new biomarkers to improve prediction and early intervention.[5]

Recent studies have identified cellular inflammatory factors as key mediators in the pathogenesis of AECOPD.[6] Specifically, inflammatory cytokines such as interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α) are critically involved in the amplification of airway inflammation.[7] IL-1β plays a pivotal role in the recruitment and activation of neutrophils, a hallmark of the acute inflammatory response in COPD.[8] Meanwhile, IL-6 acts as a multifunctional cytokine, not only driving inflammation but also playing a role in immune regulation, which further exacerbates chronic inflammation and immune system imbalances.[9] TNF-α, another potent pro-inflammatory cytokine, is heavily involved in promoting airway remodeling and worsening airflow limitation, contributing to the progressive decline in lung function.[10] Elevated levels of these inflammatory markers during AECOPD are associated with worse clinical outcomes, making them potential biomarkers for predicting exacerbations.[11] In addition to inflammatory cytokines, the role of T cell subsets, particularly CD4+ and CD8+ T cells, has gained increasing attention in the study of COPD. CD4+ T cells, which help regulate the immune response by aiding B cells in antibody production and activating other immune cells, are often diminished in patients with AECOPD, suggesting a compromised immune defense.[12] Conversely, CD8+ T cells, which are cytotoxic and involved in the direct destruction of infected or damaged cells, are typically elevated during exacerbations, reflecting heightened immune activation and tissue damage.[13] The imbalance between CD4+ and CD8+ T cells is indicative of immune dysregulation, which has been linked to both the severity of AECOPD and the risk of future exacerbations.[12] In COPD patients, the proportion and functional status of CD4+ and CD8+ T cells change, especially during acute exacerbations. The increase in CD8+ T cells and the decrease in CD4+ T cells are believed to be closely related to the progression of airway inflammation and damage.[14] The significance of these biomarkers in the context of AECOPD is multifaceted. First, they offer insights into the underlying mechanisms of exacerbations, shedding light on the inflammatory and immune pathways that drive acute events. Second, they provide potential predictive value for identifying patients at higher risk of recurrence, enabling earlier and more targeted interventions. Despite the established roles of inflammatory cytokines and T cell subsets in the progression of COPD,[15,16] there remains a gap in understanding how these markers can be used effectively to predict disease recurrence and long-term outcomes. Furthermore, recent studies have highlighted the role of failed regeneration in disease progression, where impaired lung progenitor cells contribute to the pathological remodeling seen in COPD.[17] The inflammatory markers and T cell subsets explored in this study may not only reflect the inflammatory state of COPD but also provide insight into the underlying regenerative failures that exacerbate disease progression. By investigating these biomarkers, we aim to better understand how immune dysregulation and failed regeneration contribute to acute exacerbations and long-term prognosis.

Therefore, this study aims to analyze the predictive value of cellular inflammatory factors and T cell subsets for disease recurrence and prognosis in patients with acute exacerbations of COPD. By comparing the levels of inflammatory factors and T cell subsets between patients in the acute phase and the stable phase, and combining clinical indicators and follow-up results, we aim to explore the potential application of these biomarkers in predicting acute recurrence of COPD.

Data and Methods

Basic Data

A retrospective analysis was conducted on the clinical data of 73 patients with acute exacerbations of COPD admitted to our hospital from January 2022 to January 2024, classified as the observation group. Additionally, 35 patients with stable COPD during the same period were selected as the control group. Inclusion criteria: all COPD cases met the relevant diagnostic criteria of the Global Initiative for Chronic Obstructive Lung Disease (GOLD);[18] patients with acute exacerbations of COPD exhibited symptoms such as dyspnea, chronic cough, sputum production, persistent airflow limitation, and a short-term

worsening of symptoms such as cough, wheezing, and sputum production, requiring a change in treatment regimen; stable patients had a stable disease condition for more than one month without needing to change the treatment regimen. Exclusion criteria: patients with severe organ dysfunction; malignant tumors; immune system or hematological diseases; severe infections; cognitive, communication, or mental disorders; use of antibiotics or hormonal drugs within 30 days before participating in the study; incomplete participation in the study or incomplete clinical data. This study was approved by the medical ethics committee of Zhejiang University of Chinese Medicine. Informed consent was obtained from all study participants. All the methods were carried out in accordance with the Declaration of Helsinki.

Methods

Basic Data Collection

Including gender (male, female), age, body mass index (BMI), family history of COPD (yes, no), comorbidities (hypertension, diabetes, hyperlipidemia, etc)., smoking history (smoking index ≥400, smoking index = number of cigarettes smoked per day × years of smoking), history of alcohol abuse (drinking ≥5 bottles of beer per session or blood alcohol content ≥0.08 g/dl).

Laboratory Indicators Collection

Including procalcitonin (PCT), white blood cell count (WBC), pulmonary function indicators [ratio of forced expiratory volume in 1 second to forced vital capacity (FEV1/FVC), percentage of predicted FEV1 (FEV1%pred)], inflammatory cytokines (IL-1β, IL-6, TNF-α), and T cell subsets (CD4+, CD8+) levels. Fasting venous blood (10 mL) was collected from patients at admission, placed in ethylenediaminetetraacetic acid tubes, and left standing at room temperature in the dark for 10 minutes. It was centrifuged at 1500 r/min for 15 minutes with a centrifugal radius of 10 cm, and the supernatant was separated. PCT was detected using luminescence immunoassay, WBC was detected using an automatic blood cell analyzer, inflammatory cytokines (IL-1β, IL-6, TNF-α) were detected using enzyme-linked immunosorbent assay (ELISA) kits, T cell subsets (CD4+, CD8+) were detected using a DxFLEX flow cytometer, and FEV1/FVC and FEV1%pred were detected using the Vmax6200 pulmonary function system.

Statistical Analysis

Statistical analysis was conducted using SPSS 20.0. Continuous variables were reported as mean ± standard deviation (SD), and group comparisons were made using independent-sample *t*-tests. Categorical variables were expressed as frequencies and percentages (n, %), and analyzed using the chi-square test. Pearson correlation analysis was used to analyze the correlation between inflammatory cytokines, T cell subsets, and clinical indicators. Binary logistic regression was used to calculate the predictive value of combined detection for acute exacerbations of COPD, and receiver operating characteristic (ROC) curves were plotted to evaluate the performance of single or multiple indicators in predicting acute exacerbations of COPD. A P value of less than 0.05 was considered statistically significant.

Results

Comparison of Levels of Cellular Inflammatory Factors and T Cell Subsets

The levels of IL-1β (53.96 ± 10.15 pg/mL), IL-6 (30.37 ± 6.91 pg/mL), TNF-α (55.82 ± 7.43 ng/mL), and CD8+ (29.83 ± 2.51%) were significantly higher in the observation group compared to the control group, while CD4+ levels (28.59 ± 5.36%) were significantly lower in the observation group ($P<0.05$), as shown in Table 1. These specific differences highlight the pronounced inflammatory response and immune dysregulation during acute exacerbations of COPD.

Comparison of Clinical Indicators

The level of FEV1/FVC (54.09 ± 12.28%) was significantly lower in the observation group compared to the control group, while the levels of PCT (0.26 ± 0.05 ng/mL) and WBC (12.09 ± 2.23 × 10^9/L) were significantly higher in the observation group ($P<0.05$). No significant differences were observed in the other indicators ($P>0.05$), as shown in Table 2.

Table 1 Levels of Cellular Inflammatory Factors and T Cell Subsets ($\bar{x} \pm s$)

Index	Control (n=35)	Observation (n=73)	*T*	*P*
IL-1β (pg/mL)	36.41±9.76	53.96±10.15	8.513	<0.001
IL-6 (pg/mL)	14.82±4.75	30.37±6.91	12.008	<0.001
TNF-α (ng/mL)	27.69±5.32	55.82±7.43	20.069	<0.001
CD4+ (%)	32.07±3.11	28.59±5.36	3.559	<0.001
CD8+ (%)	26.64±2.07	29.83±2.51	6.525	<0.001

Table 2 Comparison of Clinical Indicators ($\bar{x} \pm s$, n[%])

Index	Control (n=35)	Observation (n=73)	T/x^2	*P*
Gender	–	–	0.168	0.681
Male	23 (65.71)	45 (61.64)	–	–
Female	12 (34.29)	28 (38.36)	–	–
Age (years)	73.02±10.39	71.86±10.14	0.552	0.582
BMI (kg/m²)	28.65±2.04	28.27±1.96	0.930	0.354
Family History of COPD	–	–	0.184	0.667
Yes	11 (31.43)	26 (35.62)	–	–
No	24 (68.57)	47 (64.38)	–	–
Comorbidities	–	–	0.091	0.762
Yes	21 (60.00)	46 (63.01)	–	–
No	14 (40.00)	27 (36.99)	–	–
Smoking History	–	–	0.297	0.585
Yes	12 (34.29)	29 (39.73)	–	–
No	23 (65.71)	44 (60.27)	–	–
Alcohol History	–	–	0.034	0.853
Yes	9 (25.71)	20 (27.40)	–	–
No	26 (74.29)	53 (72.60)	–	–
FEV1/FVC (%)	68.74±13.52	54.09±12.28	5.614	<0.001
FEV1%pred	58.67±12.09	56.59±13.12	0.790	0.431
PCT (ng/mL)	0.12±0.04	0.26±0.05	14.480	<0.001
WBC ($\times 10^9$/L)	10.17±2.75	12.09±2.23	3.876	<0.001

Correlation Between Inflammatory Cytokines, T Cell Subsets, and Clinical Indicators

Correlation analysis indicated that IL-1β, IL-6, and TNF-α were positively correlated with CD8+, smoking history, and PCT levels, and negatively correlated with CD4+ and FEV1/FVC levels in the observation group (P<0.05), as shown in Table 3.

Comparison of Cellular Inflammatory Factors and T Cell Subsets in Patients with Different Prognoses

After a 6-month follow-up, 15 out of 73 patients in the observation group experienced acute recurrence. At the time of first admission, the levels of IL-1β (59.78 ± 11.04 pg/mL), IL-6 (37.43 ± 5.59 pg/mL), TNF-α (57.96 ± 7.44 ng/mL), and CD8+ (33.51 ± 3.82%) were significantly higher in the recurrence group compared to the non-recurrence group, while CD4+ levels (25.64 ± 4.79%) were significantly lower (P<0.05), as shown in Table 4. These findings suggest a strong association between elevated inflammatory markers and T cell imbalance with the risk of recurrence in COPD patients.

Table 3 Correlation Between Inflammatory Cytokines, T Cell Subsets, and Clinical Indicators

Index	IL-1β		IL-6		TNF-α	
	r	*P*	*r*	*P*	*r*	*P*
CD4+	−0.472	0.001	−0.419	0.002	−0.443	0.015
CD8+	0.415	0.001	0.376	0.005	0.391	0.011
Gender	0.051	0.653	0.069	0.681	0.048	0.670
Age	0.146	0.211	0.108	0.343	0.127	0.272
BMI	0.157	0.181	0.124	0.283	0.140	0.233
Family History	0.204	0.081	0.151	0.191	0.167	0.147
Comorbidities	0.125	0.276	0.094	0.423	0.113	0.320
Smoking History	0.408	0.006	0.369	0.006	0.384	0.004
Alcohol History	0.047	0.684	0.039	0.732	0.042	0.710
FEV1/FVC	0.465	0.018	0.397	0.008	0.426	0.002
FEV1%pred	0.114	0.312	0.103	0.366	0.109	0.338
PCT	0.397	0.004	0.364	0.011	0.372	0.010
WBC	0.163	0.136	0.139	0.239	0.141	0.232

Table 4 Comparison of Cellular Inflammatory Factors and T Cell Subsets in Patients with Different Prognoses ($\bar{x} \pm s$)

Index	Non-recurrence (n=58)	Recurrence (n=15)	*T*	*P*
IL-1β (pg/mL)	39.34±8.82	59.78±11.04	7.587	<0.001
IL-6 (pg/mL)	15.71±5.02	37.43±5.59	14.595	<0.001
TNF-α (ng/mL)	29.56±4.97	57.96±7.44	17.681	<0.001
CD4+ (%)	33.15±3.26	25.64±4.79	7.175	<0.001
CD8+ (%)	25.43±2.92	33.51±3.82	8.945	<0.001

The Value of Inflammatory Cytokines and T Cell Subsets in Predicting Acute Recurrence

ROC curve analysis revealed that IL-1β, IL-6, TNF-α, CD4+, and CD8+ all had good application efficiency in predicting acute recurrence in patients with acute exacerbations of COPD (AUC>0.7). Among these, IL-1β had the highest efficiency when used alone (AUC=0.802, 95% CI: 0.674~0.923), while the combined application of indicators had the highest predictive efficiency (AUC=0.907,95% CI:0.726~0.971), as shown in Table 5 and Figure 1.

Binary Logistic Regression Analysis

To further assess the independent predictive value of cellular inflammatory factors and T cell subsets for acute exacerbations of COPD, binary logistic regression analysis was performed. The model included IL-1β, IL-6, TNF-α,

Table 5 The Value of Inflammatory Cytokines and T Cell Subsets in Predicting Acute Recurrence

Index	Threshold	AUC	95% CI	P	Sensitivity	Specificity
IL-1β	49.86	0.802	0.674~0.923	<0.05	71.8	81.9
IL-6	26.97	0.763	0.625~0.894	<0.05	72.5	80.6
TNF-α	43.62	0.781	0.651~0.914	<0.05	70.8	76.7
CD4+	29.71	0.749	0.612~0.876	<0.05	73.1	79.4
CD8+	29.58	0.772	0.638~0.905	<0.05	74.7	69.5
Union	-	0.907	0.726~0.971	<0.05	77.9	92.6

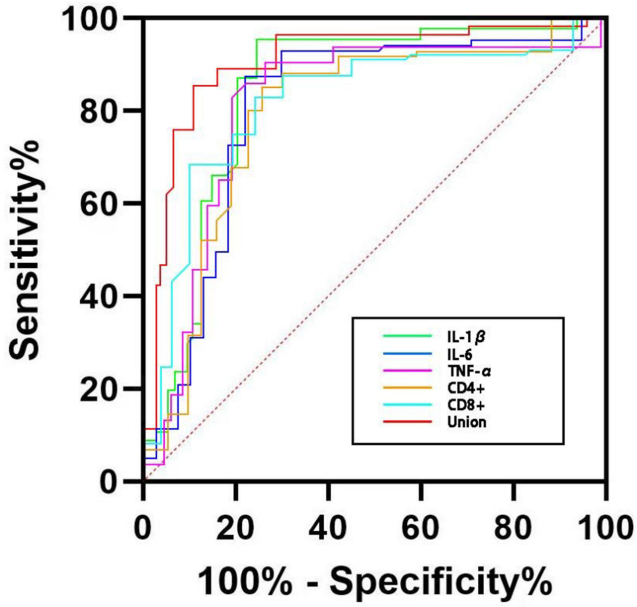

Figure 1 ROC Curve Analysis of Inflammatory Cytokines and T Cell Subsets in Predicting Acute Recurrence.

CD4+, and CD8+ as independent variables. The analysis revealed that IL-1β (OR = 1.42, 95% CI: 1.20–1.68, P < 0.001), IL-6 (OR = 1.36, 95% CI: 1.14–1.60, P < 0.001), and TNF-α (OR = 1.28, 95% CI: 1.08–1.51, P = 0.004) were significant predictors of acute exacerbations. Additionally, CD4+ (OR = 0.72, 95% CI: 0.55–0.92, P = 0.008) was found to be a protective factor, while CD8+ (OR = 1.54, 95% CI: 1.25–1.90, P < 0.001) was positively associated with an increased risk of exacerbations. These results demonstrate that elevated levels of inflammatory cytokines and CD8+ T cells, along with reduced CD4+ T cells, are significant independent predictors of acute exacerbations in COPD patients (Table 6).

Discussion

Studies[19] have shown that the levels of IL-1β, IL-6, and TNF-α are significantly elevated during acute exacerbations of COPD and are associated with the severity of the condition. The results of this study also show that the levels of IL-1β, IL-6, and TNF-α in the serum of the observation group were significantly higher than those of the control group (P<0.05), which is consistent with previous studies[20], further confirming that the inflammatory response in patients with acute COPD is more intense than in the stable phase. Comparison of clinical indicators showed that the FEV1/FVC level of the observation group was lower than that of the control group, and the PCT and WBC levels were higher than those of the control group (P<0.05), suggesting that acute COPD affects lung function, immunity, and nutrition. In terms of correlation, this study found that IL-1β, IL-6, and TNF-α were positively correlated with smoking history and PCT levels, and negatively correlated with FEV1/FVC levels (P<0.05) in COPD patients. This result confirms that the massive release of inflammatory cells can lead to a further decline in lung function in COPD patients.

Table 6 Binary Logistic Regression Analysis for Predicting Acute Exacerbations of COPD

Variable	Odds Ratio (OR)	95% CI	P-value
IL-1β	1.42	1.20–1.68	<0.001
IL-6	1.36	1.14–1.60	<0.001
TNF-α	1.28	1.08–1.51	0.004
CD4+	0.72	0.55–0.92	0.008
CD8+	1.54	1.25–1.90	<0.001

The results of this study show that the levels of CD8+ in the observation group were higher and the levels of CD4+ were lower than those of the control group (P<0.05), which is consistent with previous related studies.[21] Correlation analysis showed that IL-1β, IL-6, and TNF-α were positively correlated with CD8+ levels and negatively correlated with CD4+ levels (P<0.05) in COPD patients, further confirming the close association between abnormal expression of T lymphocyte subsets and the inflammatory response and disease severity in COPD patients. A brief analysis of the possible reasons shows that CD4+ lymphocytes play an important role in coordinating and maintaining the body's immune function. If COPD patients have abnormally low expression of CD4+, it indicates a decline in immune capability, making them susceptible to bacterial or viral infections, leading to disease progression and repeated exacerbations, continuously damaging lung tissue and further worsening COPD.[22] CD8+ T lymphocytes, also known as cytotoxic T lymphocytes, once activated, can induce cell activation, proliferation, differentiation, and produce effector cells, triggering targeted immune responses.[23] COPD patients with elevated CD8+ T lymphocyte levels may enhance the cytotoxic effects mediated by granzyme and perforin, damaging the body's lymphocytes and exacerbating airway damage.[24] At the same time, elevated CD8+ T lymphocyte levels can stimulate the body to produce various inflammatory mediators and cytokines, promoting the aggregation of macrophages and neutrophils in the lungs, and producing large amounts of metalloproteinases and oxidants, aggravating lung tissue damage and further promoting the progression of COPD.[25]

To date, although the clinical understanding of the changes in inflammatory cytokines and T cell subsets in COPD is relatively comprehensive, systematic studies on their predictive value and prognostic relationship in patients with acute exacerbations are still limited. Understanding how these biomarkers affect the risk of acute exacerbations of COPD can provide important information for clinicians to develop individualized treatment and management strategies, thereby improving patient prognosis. This study, after six months of follow-up observation, found that out of 73 patients in the observation group, 15 were readmitted due to another acute exacerbation. The levels of IL-1β, IL-6, TNF-α, and CD8+ in the readmission group were higher, and the levels of CD4+ were lower than those in the non-readmission group at the first admission (P<0.05). The ROC curve plotted showed that IL-1β, IL-6, TNF-α, CD4+, and CD8+ all had good application efficacy (AUC>0.7) in predicting another acute exacerbation in patients with acute COPD, with the combined application of these indicators having the highest predictive efficacy (AUC reaching 0.907).

The binary logistic regression analysis further solidified the independent predictive value of these biomarkers. Specifically, IL-1β, IL-6, and TNF-α were all found to be significant independent predictors of acute exacerbations, with IL-1β showing the strongest association (OR = 1.42, 95% CI: 1.20–1.68, P < 0.001). These findings suggest that elevated inflammatory cytokine levels may directly contribute to the worsening of airway inflammation, exacerbating lung function decline and leading to more frequent exacerbations. This is consistent with prior research, which has highlighted the role of these cytokines in driving the inflammatory processes underlying COPD exacerbations.[26] In terms of T cell subsets, the regression analysis revealed that elevated CD8+ T cells significantly increased the risk of exacerbations (OR = 1.54, 95% CI: 1.25–1.90, P < 0.001), while higher CD4+ T cell levels were protective (OR = 0.72, 95% CI: 0.55–0.92, P = 0.008). The imbalance between these T cell subsets is indicative of immune dysregulation in COPD patients, where an increase in cytotoxic CD8+ T cells exacerbates airway damage by promoting the release of pro-inflammatory mediators and cytotoxic factors.[27] Conversely, the decline in CD4+ T cells likely reflects impaired immune surveillance, making patients more susceptible to infections and subsequent exacerbations.[28–30] These results highlight the importance of monitoring both cellular inflammatory factors and T cell subsets in clinical practice. The ability of these biomarkers to predict acute exacerbations independent of traditional clinical measures underscores their potential utility in guiding more personalized treatment strategies.

Because the detection methods for inflammatory cytokines and T cell subsets are simple and have good efficacy in reflecting the condition of acute COPD, this study suggests that the above indicators can be used as reliable clinical indicators for assessing the risk of acute exacerbation in COPD.

Limitation

It should be noted that there are some limitations in this study, such as: (1) Small sample size: The sample size included in this study is relatively small, which may affect the reliability and generalizability of the research, and a small sample

size may limit the statistical significance of some results, leading to bias. (2) Research design limitations: This study is a retrospective analysis, which may have potential information bias and treatment selection preferences, making its persuasiveness slightly weaker compared to randomized controlled trials or prospective research designs. (3) Single-center study: This study was conducted in a single hospital, which may limit the external validity of the research results. Differences between medical institutions may affect the generalizability of the results. (4) Lack of dynamic observation and mechanism research: This study only detected the expression levels of various indicators at specific time points, lacking dynamic observation of the changes in indicators at different stages of the disease. Additionally, this study is mainly a clinical observation study and does not deeply explore the specific mechanisms of inflammatory cytokines, T cell subsets, and the recurrence of acute COPD. Therefore, in future studies, we will address the above deficiencies by increasing the sample size, improving the research design, and conducting multi-center, large-sample joint studies to make the research results more reliable and comprehensive. (5) One potential limitation of this study is that it does not account for the possible effects of COPD medications, such as inhaled corticosteroids and bronchodilators, on the levels of inflammatory markers and T cell subsets before hospital admission. Inhaled corticosteroids, for instance, are known to reduce airway inflammation by suppressing the production of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α.[31] Similarly, bronchodilators can alleviate symptoms of airway obstruction, potentially affecting immune cell activity and the expression of T cell subsets.[32] As these medications are commonly used by COPD patients, their effects on the inflammatory and immune profiles of patients prior to admission may have confounded the observed results.

Conclusion

This study is the first to integrate multiple serum inflammatory factors and T cell subsets into a comprehensive predictive model for acute recurrence of COPD within six months (AUC = 0.907), offering a more accurate prediction than traditional methods. The findings underscore the value of these biomarkers in clinical follow-up and highlight their independent predictive power, providing new insights into the interaction between immune markers and clinical indicators in COPD exacerbations.

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Disclosure

The authors report no conflicts of interest in this work.

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