

CLINICAL, LABORATORY, AND IMMUNOLOGICAL FEATURES OF PATIENTS WITH BRONCHOPULMONARY DISEASES

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Introduction

Respiratory system diseases continue to represent a major challenge in contemporary medicine due to their high prevalence and significant contribution to morbidity and mortality worldwide, including in Uzbekistan. Numerous reports indicate that respiratory diseases rank among the leading causes of illness, with incidence rates increasing approximately 2.5 times in recent years [1]. Pneumonia, in particular, remains one of the most life-threatening infectious diseases; in 2019 alone, it was responsible for nearly 2.5 million deaths globally, including about 672,000 pediatric cases [2]. Bronchopulmonary disorders—such as chronic bronchitis, chronic obstructive pulmonary disease (COPD), pneumonia, and related conditions—not only impair patients' quality of life and work capacity but also substantially increase disability and mortality rates [3]. Bronchopulmonary diseases are characterized by both local and systemic inflammatory responses. Alterations in immune system function play a pivotal role in disease development and progression. Previous investigations have demonstrated that patients with chronic bronchopulmonary pathology often exhibit reduced immunological reactivity, manifested by decreased T-lymphocyte counts, disrupted lymphocyte subpopulation balance, diminished natural killer (NK) cell activity, and abnormalities in immunoglobulin levels [4]. These findings suggest the presence of secondary immunodeficiency, particularly in patients with severe disease courses [5], which in turn predisposes them to recurrent infections and dysregulated inflammatory responses.

Study objective: To evaluate clinical manifestations, laboratory parameters, and immunological alterations in patients with bronchopulmonary diseases according to disease severity, and to perform statistical analysis in order to formulate evidence-based conclusions and clinical recommendations.

Clinical Characteristics

The study included 100 patients diagnosed with bronchopulmonary pathology, predominantly pneumonia and chronic bronchitis. Based on clinical severity, patients were stratified into three groups: mild (n=40), moderate (n=40), and severe (n=20). The overall mean age was 52.3 ± 8.1 years, with younger patients predominating in the mild group (≈ 45 years) and older patients in the severe group (≈ 60 years). Clinical assessment revealed a clear association between disease severity and symptom intensity. Mild cases were characterized by relatively subtle manifestations with minimal risk to life, whereas moderate cases showed more pronounced symptoms. In

contrast, severe cases were associated with marked clinical deterioration and a substantially higher risk of complications.

Mild disease

1. Cough: intermittent dry or productive cough, usually more noticeable in the morning and of low intensity.
2. Dyspnea: absent at rest; mild shortness of breath may occur during physical exertion.
3. Body temperature: normal or slightly elevated ($37\text{--}37.5\text{ }^{\circ}\text{C}$), with preserved general condition.

Moderate disease

1. Cough: persistent productive cough throughout the day, accompanied by increased sputum production (approximately 100–200 ml/day).
2. Dyspnea: present even at rest and clearly aggravated by mild physical activity such as climbing stairs.
3. Body temperature: typically febrile (around $38\text{ }^{\circ}\text{C}$), often associated with fatigue, headache, and occasional chest discomfort that may worsen during respiration.

Severe disease

1. Cough: continuous, intense cough with copious purulent sputum ($>300\text{ ml/day}$); hemoptysis was noted in some patients.
2. Dyspnea: severe respiratory insufficiency characterized by tachypnea and shallow breathing at rest, reduced arterial oxygen saturation ($<90\%$), cyanosis, and use of accessory respiratory muscles.
3. Body temperature: persistently high fever ($38\text{--}39\text{ }^{\circ}\text{C}$) with chills and pronounced signs of systemic intoxication, including pallor, diaphoresis, and tachycardia; severe pleuritic chest pain and diffuse wheezing were frequently observed. Patients in the severe group demonstrated a high incidence of complications such as respiratory failure, pulmonary edema, pleural involvement, and sepsis. Conversely, individuals with mild and moderate disease generally achieved rapid clinical recovery with timely and adequate therapy, and complications were uncommon.

Laboratory Parameters

All participants underwent comprehensive laboratory evaluation, including complete blood count, urinalysis, biochemical profiling, and sputum examination with microbiological culture. The extent of inflammatory changes closely correlated with disease severity. In mild cases, leukocyte counts were within normal limits or mildly elevated ($7\text{--}9 \times 10^9/\text{L}$), whereas severe cases exhibited pronounced leukocytosis (up to $12\text{--}15 \times 10^9/\text{L}$). A marked increase in neutrophil proportion (up to 80%) and a

concomitant decrease in lymphocyte percentage ($<15\%$) were characteristic of severe disease. Lymphopenia, defined as a significant reduction in circulating lymphocytes, is recognized as a marker of severe inflammation and an unfavorable prognostic factor in community-acquired pneumonia and sepsis [6]. Elevated risks of intensive care admission and mortality have been reported in patients presenting with lymphopenia [6]. Biochemical analysis further confirmed disease severity, as inflammatory markers were substantially higher in severe cases. Mean C-reactive protein (CRP) levels reached 80 ± 25 mg/L in severe pneumonia compared with 10 ± 8 mg/L in mild cases ($p < 0.001$). Erythrocyte sedimentation rate (ESR) values were likewise significantly increased, reflecting heightened inflammatory activity. Urinalysis findings were largely unremarkable. However, transient proteinuria or microhematuria was occasionally detected during periods of severe intoxication and high fever, suggesting functional renal involvement secondary to systemic inflammation. Sputum examination was essential for etiological diagnosis. Microscopy revealed abundant polymorphonuclear leukocytes in all samples, with purulent characteristics predominating in severe cases. Pathogenic organisms were isolated in approximately 60% of mild-to-moderate cases and in up to 80% of severe cases. The most commonly identified pathogens included *Streptococcus pneumoniae* ($\sim 40\%$), *Haemophilus influenzae*, *Staphylococcus aureus*, and *Klebsiella* species [7]. In elderly and immunocompromised patients with severe pneumonia, Gram-negative bacteria and nosocomial pathogens, such as *Pseudomonas aeruginosa*, were more frequently detected.

Immunological Alterations

Immunological assessment demonstrated that immune dysregulation plays a critical role in determining disease severity. Severe bronchopulmonary disease was consistently associated with lymphopenia, indicating compromised cellular immunity and reduced host defense capacity [5,6]. Humoral immunity parameters varied according to clinical severity. While immunoglobulin levels (IgG, IgM, IgA) generally remained within reference ranges in mild and moderate cases, severe disease was frequently accompanied by reduced IgG concentrations (<7 g/L). Such reductions were associated with recurrent exacerbations and repeated pneumonia episodes within one year. International data indicate that approximately 10% of COPD patients exhibit decreased IgG levels, particularly involving IgG1 and IgG3 subclasses [8,9]. In the present study, markedly reduced total IgG (<5 g/L) was observed in 10% of severe cases, correlating with a more aggressive and recurrent disease course. Mild reductions in IgM were also noted in some severe cases, suggesting attenuation of the primary humoral immune response [8]. Conversely, elevated IgA levels were detected in a subset of patients with severe chronic bronchitis, possibly reflecting enhanced mucosal immune activation. Pro-inflammatory cytokine profiling revealed significantly increased levels of interleukin-6 (IL-6), interleukin-8 (IL-8), and tumor necrosis

factor- α (TNF- α) in severe disease. IL-6 and IL-8 concentrations in sputum were 2–3 times higher in severe cases compared with mild disease ($p < 0.05$) [10]. Elevated IL-6 levels were also detected systemically and demonstrated an inverse correlation with respiratory function indices, including FEV₁ and arterial oxygenation ($r \approx -0.43$, $p < 0.05$) [8]. These findings underscore the contribution of excessive cytokine production to disease severity and respiratory compromise. Increased IL-8 levels likely reflect intensified neutrophil recruitment to the inflammatory focus [9].

Statistical Analysis

Statistical processing was performed using standard analytical methods. Quantitative variables were expressed as mean \pm standard deviation. Comparisons between groups were conducted using Student's t-test and one-way analysis of variance (ANOVA), while categorical variables were analyzed using the χ^2 test. Statistical significance was defined as $p < 0.05$. Analysis revealed significant intergroup differences across multiple clinical, laboratory, and immunological parameters. Severe disease was associated with higher leukocyte counts, lower lymphocyte percentages, elevated CRP levels, increased pathogen detection rates, prolonged hospitalization, and higher complication and mortality rates compared with mild disease ($p < 0.05$).

Conclusions and Practical Implications

1. Disease severity and clinical presentation: Increasing severity of bronchopulmonary disease is directly associated with intensified clinical manifestations and a higher risk of complications. Early diagnosis and prompt treatment are crucial to prevent progression to severe forms.

2. Laboratory indicators: Severe disease is characterized by leukocytosis, neutrophilia, lymphopenia, and markedly elevated inflammatory markers. Continuous monitoring of laboratory parameters, particularly lymphocyte counts, is recommended.

3. Immunological monitoring: Severe cases exhibit pronounced immune imbalance, including cellular immunodeficiency and disturbances in humoral immunity. Assessment of immune parameters and, where appropriate, cautious consideration of immunomodulatory or immunoglobulin replacement therapy may be justified.

4. Prevention and management strategies: Smoking cessation, vaccination against respiratory infections, early control of exacerbations, and adequate inpatient management of severe pneumonia are essential components of effective disease prevention and treatment.

5. Future perspectives: Further research should focus on expanding the range of immunological biomarkers and evaluating targeted immunomodulatory approaches to improve outcomes in patients with bronchopulmonary diseases.

Conclusion

The above recommendations are aimed at improving the effectiveness of treatment and reducing complications in patients with bronchopulmonary pathology. Adherence to these recommendations in clinical practice contributes to better disease outcomes and overall improvement in patient prognosis.

References

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