

THE IMPACT OF METABOLIC SYNDROME ON CLINICAL AND INFLAMMATORY BIOMARKERS IN BRONCHIAL ASTHMA

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ARTICLE INFORMATION

ABSTRACT:

ARTICLE HISTORY:

Received: 01.05.2026

Revised: 02.05.2026

Accepted: 03.05.2026

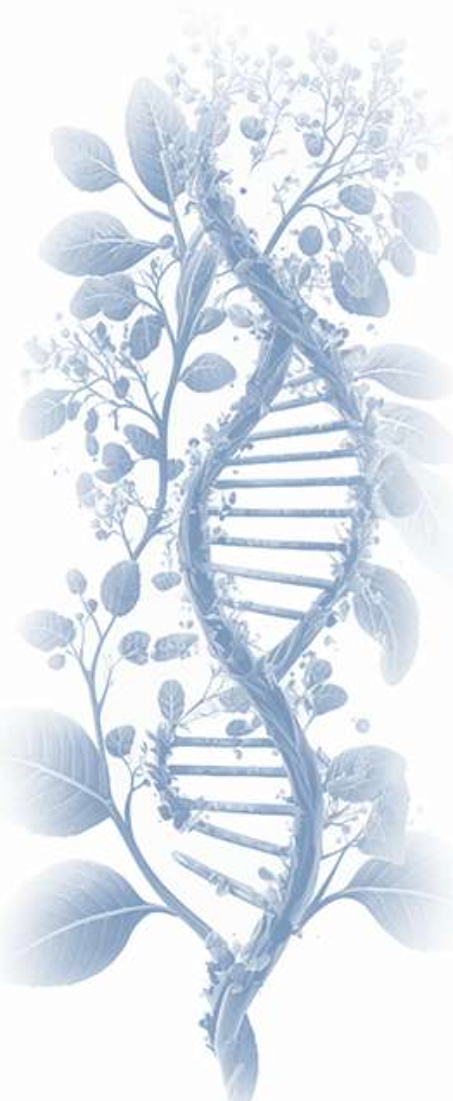
KEYWORDS:

Obstructive airway diseases, bronchial asthma, metabolic syndrome, systemic inflammation, airway hyperresponsiveness, insulin resistance, oxidative stress, asthma phenotypes, asthma endotypes, cytokines, IL-4, IL-5, IL-6, IL-13, TNF- α

In this study, we conducted a

Bronchial asthma is a chronic inflammatory disease of the respiratory tract characterized by airway hyperresponsiveness, reversible airflow obstruction, and persistent inflammation. In recent years, it has become evident that asthma is not merely a localized pulmonary disorder but rather a systemic disease, especially when it is associated with metabolic syndrome. Metabolic syndrome (MS), which includes abdominal obesity, insulin resistance, dyslipidemia, and arterial hypertension, significantly modifies the clinical course, severity, and therapeutic response of bronchial asthma.

comprehensive evaluation of clinical manifestations, pulmonary functional parameters, metabolic indices, oxidative stress markers, and inflammatory cytokines in patients suffering from bronchial asthma with and without metabolic syndrome. Particular attention was paid to the role of interleukins (IL-4, IL-5, IL-6, IL-13), tumor necrosis factor-alpha (TNF- α), C-reactive protein (CRP), and oxidative stress biomarkers (MDA, SOD, GPx, CAT, TAC). The results demonstrate that metabolic syndrome significantly worsens asthma control, increases systemic inflammation, enhances oxidative stress, and contributes to corticosteroid resistance, thereby forming a distinct



*metabolic asthma
phenotype.*

Introduction and Relevance

Bronchial asthma is one of the most widespread chronic respiratory diseases worldwide, affecting hundreds of millions of individuals. Traditionally, it has been considered an allergic and inflammatory disease of the airways; however, modern scientific evidence has redefined asthma as a heterogeneous systemic disorder with multiple phenotypes and endotypes.

One of the most clinically significant comorbid conditions associated with asthma is metabolic syndrome. Metabolic syndrome is a complex metabolic disorder characterized by central obesity, insulin resistance, dyslipidemia, and elevated blood pressure. The coexistence of metabolic syndrome with bronchial asthma has become increasingly common due to global increases in obesity, sedentary lifestyle, and dietary changes.

The interaction between asthma and metabolic syndrome is multifactorial and includes several pathogenetic mechanisms:

- chronic low-grade systemic inflammation
- increased production of pro-inflammatory cytokines such as IL-6 and TNF- α
- adipokine imbalance (increased leptin and decreased adiponectin)
- enhanced oxidative stress leading to cellular damage
- insulin resistance contributing to immune dysregulation

These mechanisms collectively lead to worsening of airway inflammation, increased bronchial hyperresponsiveness, and reduced response to standard anti-asthmatic therapy. Therefore, the study of metabolic syndrome in asthma patients is of high scientific and clinical importance in modern pulmonology.

Aim of the Study

The aim of this research is to comprehensively investigate the influence of metabolic syndrome on the clinical course of bronchial asthma, to evaluate changes in pulmonary function, metabolic status, inflammatory mediators, and oxidative stress markers, and to identify phenotype-specific therapeutic approaches for personalized management of patients.

Materials and Study Design

Study Setting

The study was conducted at the Department of Internal Medicine No. 1, Samarkand State Medical University, which serves as a tertiary referral center for respiratory and internal diseases.

Study Period

Patients were observed over a period from April 2025 to February 2026. Clinical and laboratory evaluations were performed at multiple time points, including baseline, 3 months, 6 months, and final follow-up at 12 months, allowing for dynamic assessment of disease progression and treatment response.

Sample Size

A total of 92 patients diagnosed with bronchial asthma were included in the study.

Age Range

Participants were aged between 18 and 70 years, representing both young adults and elderly populations.

Patient Groups

Group I (Main Group)

This group included 46 patients diagnosed with bronchial asthma in combination with metabolic syndrome. These patients received standard anti-asthmatic therapy along with individualized metabolic and anti-inflammatory correction strategies, including lifestyle modification and supportive pharmacotherapy aimed at improving insulin sensitivity and reducing systemic inflammation.

Group II (Control Group)

This group consisted of 46 patients with bronchial asthma without metabolic syndrome who received only standard guideline-based therapy. This group served as a comparative baseline to evaluate the influence of metabolic syndrome on disease progression.

Clinical and Laboratory Assessment

A comprehensive diagnostic approach was used, including:

Clinical assessment

Patients were evaluated using the Asthma Control Test (ACT), assessment of dyspnea severity using the mMRC scale, frequency of exacerbations, and physical exercise tolerance. Clinical observation also included monitoring of cough severity, sputum production, and overall quality of life.

Pulmonary function testing

Spirometric parameters such as FEV1, FVC, FEV1/FVC ratio, peak expiratory flow (PEF), and oxygen saturation (SpO₂) were measured to assess the degree of airway obstruction and respiratory impairment.

Metabolic evaluation

Body mass index (BMI), waist circumference, fasting glucose levels, lipid profile (LDL, HDL, triglycerides), and insulin resistance index (HOMA-IR) were analyzed to evaluate metabolic disturbances.

Inflammatory biomarkers

Serum levels of IL-4, IL-5, IL-6, IL-13, TNF- α , CRP, and total IgE were measured as indicators of systemic and airway inflammation.

Oxidative stress markers

Malondialdehyde (MDA), superoxide dismutase (SOD), glutathione peroxidase (GPx), catalase (CAT), and total antioxidant capacity (TAC) were used to assess oxidative balance in the organism.

Results

Clinical findings

Patients with asthma and metabolic syndrome demonstrated significantly more severe clinical manifestations compared to the control group. These patients experienced more frequent exacerbations, persistent dyspnea, reduced exercise tolerance, and poor disease control despite standard therapy. In contrast, patients without metabolic syndrome showed relatively stable disease progression with fewer exacerbations.

Pulmonary function

Significant reduction in FEV1, FEV1/FVC ratio, and PEF was observed in the metabolic syndrome group, indicating more severe airway obstruction. Oxygen saturation levels were also lower, suggesting impaired respiratory efficiency. The control group exhibited only mild to moderate functional impairment.

Metabolic alterations

Patients in the main group showed increased body mass index, central obesity, elevated fasting glucose, and significant insulin resistance (HOMA-IR). Dyslipidemia was also prominent, characterized by elevated LDL cholesterol and triglycerides with reduced HDL levels.

Inflammatory response

A marked increase in pro-inflammatory cytokines was observed in the metabolic asthma group. IL-6 and TNF- α levels were significantly elevated, indicating systemic inflammation.

IL-5 and IL-13 were also increased, reflecting mixed eosinophilic and neutrophilic inflammation. CRP levels were consistently higher in this group, confirming active inflammatory status.

Oxidative stress

A pronounced imbalance between oxidative and antioxidant systems was observed in patients with metabolic syndrome. Increased MDA levels indicated enhanced lipid peroxidation, while decreased SOD, GPx, CAT, and TAC reflected weakened antioxidant defense mechanisms.

Discussion

The findings of this study confirm that metabolic syndrome plays a crucial role in modifying the clinical course of bronchial asthma. The coexistence of these two conditions creates a complex pathophysiological interaction characterized by systemic inflammation, metabolic dysregulation, and oxidative stress.

This leads to the formation of a distinct **metabolic asthma phenotype**, which is characterized by:

- late disease onset
- obesity-associated airway inflammation
- predominance of neutrophilic inflammation
- reduced responsiveness to inhaled corticosteroids
- increased frequency of exacerbations

These findings support the concept of asthma as a heterogeneous disease requiring phenotype-based personalized treatment strategies rather than uniform therapeutic approaches.

Conclusion

1. Metabolic syndrome significantly aggravates the clinical course of bronchial asthma.
2. Pulmonary function is more severely impaired in patients with metabolic syndrome.
3. Pro-inflammatory cytokines (IL-6, TNF- α) are markedly elevated.
4. Oxidative stress is significantly increased, while antioxidant defense is reduced.
5. A distinct metabolic asthma phenotype is identified, requiring personalized management.
6. Integrated metabolic and anti-inflammatory therapy should be considered in such patients.

Practical Recommendations

- Early screening for metabolic syndrome in all asthma patients is essential.
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- Routine assessment of IL-6, CRP, and oxidative stress markers is recommended.
- Incorporation of insulin resistance evaluation (HOMA-IR) into clinical practice is advised.
- Phenotype-based individualized therapy should be implemented.
- Antioxidant and metabolic correction strategies should be included in complex treatment protocols.

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