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THE LEVEL OF EOSINOPHILIC CATIONIC PROTEIN IN PATIENTS WITH ATOPIC BRONCHIAL ASTHMA IN COMBINATION WITH GASTROESOPHAGEAL REFLUX DISEASE

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ABSTRACT

The aim of the study was to investigate the level of eosinophil cationic protein (ECP) in the blood serum of patients with atopic bronchial asthma (ABA) and its combination with gastroesophageal reflux disease (GERD). 40 people were examined, including 10 healthy individuals (control), 12 patients with isolated A B A, and 18 patients with A B A + GERD. The ECP level was determined by ELISA. The values mean were: control - 15.6 ± 4.3 μg/L; isolated A B A - 46.2 ± 11.1 μg/L ($p < 0.01$); ABA + GERD - 68.9 ± 14.6 μg/L ($p < 0.001$). ECP correlated positively with the eosinophil count ($r = 0.67$) and IgE level ($r = 0.54$) and negatively with FEV_1 ($r = –0.61$). The most pronounced systemic eosinophil activation was revealed in patients with combined pathology. The obtained data confirm the diagnostic and prognostic value of determining ECP as a marker of systemic inflammation and therapeutic effectiveness.

KEYWORDS

Eosinophil cationic protein, atopic bronchial asthma, gastroesophageal reflux disease, systemic inflammation, IgE, eosinophil activation, biomarkers.

Introduction

Bronchial asthma (BA) is one of the most common chronic respiratory diseases and is characterized by heterogeneous clinical manifestations, variable bronchial obstruction, and persistent inflammation. Comorbidities significantly influence the course of BA, potentially worsening disease control, increasing the frequency of exacerbations, and reducing the effectiveness of standard therapy. Current research on BA management emphasizes the need for active identification and correction of comorbidities, among which gastroesophageal reflux disease (GERD) occupies an important place [2]. GERD is widespread in the general population: according to a systematic review and meta-analysis, the global prevalence of gastroesophageal reflux disease is estimated at approximately 13.98%, with significant variability [4]. Moreover, the frequency of GERD is significantly higher in patients with asthma, which makes the problem particularly clinically significant. According to a systematic review by Havemann et al. (Gut, 2007), the average prevalence of GERD symptoms in patients with asthma is 59.2% (in the control group - 38.1%); patients with asthma also often have objective signs of reflux: pathological acid reflux according to 24-hour pH-metry - about 50.9%, esophagitis during endoscopy - about 37.3% [1]. These data reflect the global trend of high comorbidity between GERD and asthma and explain the need to consider reflux-associated mechanisms when assessing asthma symptoms and control.

The pathophysiological relationship between asthma and GERD is multifactorial and includes potential microaspiration of gastric contents, esophago-bronchial vagus reflexes, maintenance of chronic cough and nocturnal symptoms, as well as impact on sleep quality and treatment adherence. Clinical guidelines for GERD specifically discuss extraesophageal manifestations and the complexities of causal interpretation of respiratory symptoms, including the possible influence of GERD on the course of asthma [3]. Additionally, a meta-analysis by Mallah et al. (2022) confirms the clinical significance of the combination of GERD and asthma and examines their relationship in the context of exacerbations and disease severity [5].

In the context of the high prevalence of GERD in patients with asthma, the search for objective laboratory markers reflecting the activity of allergic inflammation and allowing the assessment of systemic eosinophil activation in comorbid pathology is relevant. Eosinophil cationic protein (ECP), a degranulation product of activated eosinophils, is considered as an indicator of the functional activity of the eosinophil link and a potentially more sensitive marker of inflammatory activity compared to a simple count of blood eosinophils. The review by Koh et al. emphasizes that ECP is associated with inflammation in asthma and can be used to monitor disease activity and response to therapy [6]. Considering that GERD can maintain or exacerbate respiratory symptoms and the inflammatory background, the study of ECP in patients with atopic asthma in combination with GERD seems pathogenetically justified and clinically significant [2,3].

The aim of the study was to evaluate the characteristics of serum eosinophil cationic protein levels in patients with atopic bronchial asthma and gastroesophageal reflux disease, and to determine the relationships between eosinophil cationic protein and markers of atopy (total IgE), eosinophilia, and respiratory function parameters.

Materials and Methods

The study included 40 participants, divided into three groups:

1. Control: 10 healthy individuals without clinical signs of allergic diseases and gastrointestinal pathology.
2. Isolated atopic bronchial asthma - 12 patients with an established diagnosis of atopic bronchial asthma of mild to moderate severity in the exacerbation phase or unstable remission.
3. Atopic bronchial asthma + GERD - patients with an established diagnosis of atopic bronchial asthma of mild to moderate severity in the acute phase or unstable remission in combination with gastroesophageal reflux disease

The average age of the subjects was 44.4 ± 11.6 years (18 men, 22 women). The diagnosis of atopic bronchial asthma was confirmed by clinical data, spirometry results (FEV_1, Tiffeneau index), and immunological parameters (increased total and specific IgE, eosinophilia). GERD was diagnosed based on complaints, the GerdQ questionnaire (GERD Questionnaire), and fibrogastroduodenoscopy (FGDS) data.

Serum eosinophil cationic protein (ECP) levels were determined by enzyme-linked immunosorbent assay (ELISA) using the Phadia 200 System (Thermo Fisher Scientific, Sweden). Total IgE levels were also determined, and peripheral blood eosinophil counts were also performed.

Results and Discussion

In the control group, the average ECP level was 15.6 ± 4.3 μg/L. In patients with isolated BA, ECP was significantly elevated — 46.2 ± 11.1 μg/L ($p < 0.01$ relative to the control). The highest values were recorded in patients with BA + GERD — 68.9 ± 14.6 μg/L, which was significantly higher than both the control ($p < 0.001$) and the isolated BA group ($p < 0.05$).

Table 1. Serum ECP levels (μg/L) in the study groups

Group	n	ECP, M ± SD (μg/L)	p vs control	p vs isol. BA
Control	10	15.6 ± 4.3	—	—
Isolated BA	12	46.2 ± 11.1	<0.01	—
BA + GERD	18	68.9 ± 14.6	<0.001	<0.05

Patients with asthma (both isolated asthma and asthma + GERD) showed elevated total IgE levels and higher eosinophil counts compared to controls. In the asthma + GERD group, the trend toward higher values persisted.

Table 2. Additional markers

Indicator	Control (n=10)	Isolated asthma (n=12)	BA + GERD (n=18)
Eosinophils, %	2.1 ± 0.9	5.8 ± 1.6	6.6 ± 1.9
Total IgE, IU/ml	58 ± 22	238 ± 84	286 ± 96
FEV_1, % of expected	96 ± 7	78 ± 9	72 ± 10

Correlation analysis revealed a positive association between the ECP level and the peripheral blood eosinophil count ($r = 0.67$; $p < 0.01$), as well as with the total IgE level ($r = 0.54$; $p < 0.05$). A negative correlation was also found between the ECP concentration and the FEV_1 value ($r = –0.61$; $p < 0.01$), indicating that increased eosinophilic activation is associated with a more pronounced decrease in bronchial patency and confirming the pathogenetic role of eosinophilic inflammation in the development of functional disorders in asthma.

Conclusion

The study showed that the serum level of eosinophil cationic protein (ECP) is a sensitive and informative biomarker of systemic eosinophil activation in patients with atopic bronchial asthma. In patients with bronchial asthma, the ECP concentration was significantly higher than in apparently healthy individuals: 15.6 ± 4.3 μg/L in the control group versus 46.2 ± 11.1 μg/L in isolated bronchial asthma ($p < 0.01$). The highest ECP values were found in patients with comorbid pathology of bronchial asthma + GERD - 68.9 ± 14.6 μg/L, which was significantly higher than the control values ($p < 0.001$) and was higher than in isolated bronchial asthma ($p < 0.05$). The findings suggest that GERD is associated with additional increases in systemic inflammatory activity in atopic asthma.

Correlation analysis confirmed the pathogenetic significance of ECP: a positive association was found between the ECP level and the eosinophil count ($r = 0.67$; $p < 0.01$) and total IgE levels ($r = 0.54$; $p < 0.05$), as well as a negative correlation with the FEV_1 indicator ($r = –0.61$; $p < 0.01$), indicating a relationship between eosinophil activation and impaired bronchial patency. Thus, ECP can be considered as an additional diagnostic and prognostic marker in patients with atopic bronchial asthma, especially when combined with GERD, allowing for an objective assessment of the severity of the inflammatory process and the effectiveness of anti-inflammatory therapy.

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