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CHARACTERISTICS OF ALLERGENS IN THE ASTHMATIC TRIAD

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Abstract

Aspirin bronchial asthma, aspirin or asthmatic triad (AT) is characterized by three main clinical manifestations - bronchial asthma (BA), polypous rhinosinusitis (PRS) and intolerance to aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs). Despite the fact that bronchial asthma in patients with AT makes up, according to various sources, from 10 to 20% of its clinical and pathogenetic variants, torpid course of the disease, risk of sudden death, sharp decrease in the quality of life of patients, classifies it as the most severe forms of BA. A distinctive feature of the disease is also the high cost of treatment and diagnostic measures. The relevance of the AT problem is evidenced by the fact that the European Coordination Committee was created to study it, and in 2022 - the International Society of Scientists, which coordinates research on the problem of PRS. According to medical statistics, out of 7 million patients with BA in Russia, 1 million have severe forms of the disease, of which 40% of patients are represented by AT or aspirin bronchial asthma. The asthmatic triad has been known to medical science for about 80 years, however, to this day it remains unclear what causes the severity, polymorphism and uniqueness of clinical manifestations of AT. Many issues of pathogenesis, clinical and diagnostic approaches have not yet been resolved and are discussed by allergists, immunologists, otolaryngologists, pulmonologists.

Introduction

However, at present, it is believed that: NSAID intolerance in AT is not a true allergic reaction and is not based on IgE-mediated mechanisms [97, 215]; the main manifestations of AT - polypous rhinosinusitis and bronchial asthma itself in the asthmatic triad are not a random coincidence of symptoms, but a pathogenetically related pathology.

The key role in modern studies of the pathogenesis of AT is still given to the peculiarities of arachidonic acid metabolism, stimulation of increased production

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of leukotrienes by aspirin , and the study of increased sensitivity of the respiratory tract to them. In this regard, in recent years, the term " aspirin-exacerbated respirator disease » (AERD) is a respiratory disease that worsens after taking aspirin. The platelet theory of AT development is also of interest. Thus, Evsyukova E.K. [25] found that AT patients have reduced synthesis of the hormone melonin , while the sensitivity and perverted reaction of the platelet receptor apparatus to melonin are increased . It has been shown that platelets of AT patients, under the influence of NSAIDs, reduce the production of leukotrienes , which have a bronchodilation effect , and stimulate the release of cytotoxic anti-inflammatory mediators. A number of studies pay attention to changes in the function of lymphocytes, mast cells, and eosinophils as a result of chronic intracellular infection, studying in detail the immunological aspects of AT. However, the presented values of immune status indicators in different authors have significant differences. In this regard, it is not clear which link of immunity is the leading one in this disease. All currently existing hypotheses explain only individual links in the pathogenesis of AT and, in particular, the development of aspirin-induced bronchospasm . In this regard, the treatment and diagnostic approaches to AT are considered from the point of view of the pathogenetic mechanisms of the asthmatic triad development based on the features of arachidonic acid metabolism. Theories of AT pathogenesis exist independently and do not explain the features of the course and development of the complete asthmatic triad in individual clinical and immunological groups. Thus, to date, the causes of excessive formation of leukotrienes , rapid progression of the disease and the formation of glucocorticosteroid dependence in patients who excluded NSAIDs and aspirin from taking have not been clarified. The modern concept of the asthmatic triad does not distinguish between the forms, severity, stages of development and combinations of the main clinical manifestations of the disease - bronchial asthma and polypous rhinosinusitis . Risk groups are not considered and the conditions under which initial clinical BA and PRS in the final stage of the disease are able to form a picture of the complete asthmatic triad are not taken into account. The influence of individual symptoms - bronchial asthma, allergic rhinitis, NSAIDs on the final formation of the complete asthma syndrome has not been studied, and the stages of disease development for individual groups of patients with asthma have not been investigated. It is interesting that to date there are no data on a full allergological

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examination of patients with the complete asthmatic triad and various combinations of its main manifestations - allergic rhinitis, polypous rhinosinusitis , bronchial asthma. There is no classification in the literature and the stages of development of the complete clinical picture of the asthmatic triad for individual forms of bronchial asthma have not been studied, there are no practical recommendations for prevention and a systemic therapeutic and diagnostic approach to this severe category of patients. Currently, there is no generally accepted concept for the diagnosis and treatment of AT. There are individual recommendations from allergists, immunologists, pulmonologists and otolaryngologists on symptomatic treatment of AT without taking into account the characteristics of the disease. Great hopes were pinned on compounds of a new biological class - antagonists leukotriene receptors. However, the insufficient clinical efficacy of monotherapy and the high cost of treatment with these drugs limit their use. In addition, it has been shown that the effectiveness of antileukotriene therapy is comparable in patients with and without aspirin intolerance. Various domestic and foreign centers have been developing methods of aspirin desensitization for many years . Until now , some authors consider this method to be the most effective in the treatment of AT. However, the need for long-term inpatient monitoring of the patient, the possibility of developing adverse pharmacological reactions to aspirin, as well as the unstable clinical effect of treatment when the drug is discontinued, limited the use of this method. Identification of risk groups for the development of a complete clinical picture of AT and a systemic therapeutic and diagnostic approach to the disease is possible only with a holistic view of the pathogenesis, clinical and immunological features of AT and a study of the mutual influence of the main manifestations of AT.

Objective of the Study

To present the clinical and immunological characteristics of AT, the ways of its formation and therapeutic approaches to control the main manifestations of AT.

Research objectives

1. To study the stages and patterns of development of individual manifestations of the asthmatic triad (BA, PRS, NSAIDs) and their influence on the formation of the complete AT syndrome.

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2. Present the features of the allergological characteristics of the complete AT syndrome at various stages of its development.
3. To identify the features of the immunogram and indicators of local immunity in patients with AT at various stages of its development.
4. To establish the main risk factors for the development of asthma, to identify and present clinical, laboratory, allergological and immunological markers of risk groups of patients with initial manifestations of the asthmatic triad.
5. Develop and substantiate clinical diagnostic criteria for establishing the diagnosis of the asthmatic triad, depending on the variants of the clinical course.
To present the features of the histological characteristics of polyps in patients with different variants of the clinical course of AT. To identify the features of the landscape of pathogens of the upper respiratory tract infection in patients with AT depending on the severity and variants of the clinical course. To develop methodological approaches to allergen-specific immunotherapy and pharmacotherapy in patients with AT. Based on the conducted studies, to develop a treatment algorithm depending on the variants of the clinical course of AT and ways to prevent the development of the full clinical syndrome of the asthmatic triad. Based on the analysis of the results of in-depth clinical, laboratory, allergological and immunological examination, heterogeneity of the contingent of patients with AT was established and differential diagnostic criteria for various forms of the asthmatic triad were developed for the first time. It was established for the first time that about half of the patients with AT have atopic mechanisms of bronchial asthma formation and sensitization to non-bacterial allergens. For the first time, the stages of formation of the full AT syndrome were established for its various forms and variants of the course. It has been proven that the development of aspirin intolerance in patients with AT always develops against the background of chronic inflammation of the upper and lower respiratory tract, in the mechanism of which allergic inflammation is of paramount importance. It has been established that all patients with AT have a variety of pathogens of intracellular infection: bacteria, viruses, fungi, which are not only the cause of exacerbation of infection of the upper and lower respiratory tract, but also its main manifestations - bronchial asthma and PRS. The role of eosinophilic inflammation in the development of AT has been scientifically substantiated and it has been shown that in patients with AT, eosinophilic inflammation in shock organs is accompanied by a decrease in the

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barrier function of the mucous membranes of the respiratory tract, increased inflammatory reactions and the formation of clinical signs of secondary immune deficiency. Clinical manifestations of SID in patients with AT are accompanied by changes in the functioning of the immune system - a decrease in the content and functional activity of phagocytes, a deficiency of secretory IgA , the presence of high levels of specific immunoglobulins to pathogens of chronic intracellular infection. It has been proven that long-term allergic inflammation of the respiratory tract can form nasal and bronchial hyperreactivity even in the absence of constant allergen exposure. For the first time, a pathogenetically substantiated clinical classification of AT has been proposed, the forms and severity of the main manifestations of AT - bronchial asthma and polypous rhinosinusitis have been identified. For the first time, the effectiveness of ASIT in controlling allergic inflammation in patients with the asthmatic triad has been applied and proven . Based on the identified differential diagnostic criteria and classification of AT, an algorithm for treating patients depending on the variants of the clinical course of AT and ways to prevent the development of the full clinical syndrome of the asthmatic triad has been proposed. The results presented in the work are of great value for practical health care. Contrary to popular belief, the role of allergy in the mechanism of AT development has been established for the first time. It is shown that among patients with asthmatic triad the proportion of patients with atopy has increased by 2.6 times over the past 20 years, and half of them have sensitization to the non-bacterial spectrum of allergens. For the first time the risk factors and stages of development of the complete clinical syndrome of asthmatic triad have been identified. The relationship between the severity and duration of individual clinical manifestations, as well as the activity of chronic infectious inflammation of the upper and lower respiratory tract of patients with AT has been established. The obtained results made it possible to substantiate the need for timely adequate therapy of bacterial, viral, fungal intracellular infection in patients with asthmatic triad. Based on the analysis of the sequence of development of clinical manifestations of AT, combinations and combinations of individual (intermediate) stages of the asthmatic triad have been considered and the need for complex treatment of patients depending on the stage of the disease has been substantiated. The identified clinical and laboratory signs of secondary immune deficiency in

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patients with AT made it possible to substantiate the need for and criteria for the appointment of immunomodulatory therapy.

Conclusions

Complete asthmatic triad syndrome (bronchial asthma combined with polypous rhinosinusitis and intolerance to nonsteroidal anti-inflammatory drugs) was found in 273 (3.1%) of 4452 examined patients with bronchial asthma. Clinical and immunological examination revealed heterogeneity of the group of patients with asthmatic triad. Depending on the mechanism of development, three forms of asthmatic triad were distinguished: asthmatic triad with atopic asthma (ATA), asthmatic triad with mixed asthma (ATS), asthmatic triad with infection-dependent asthma (IDBA). The leading link in the pathogenesis of the asthmatic triad is eosinophilic allergic inflammation, which is accompanied by chronic inflammation of the respiratory tract in patients with polypous rhinosinusitis and bronchial asthma and serves as a background for the formation of increased sensitivity to aspirin and other non-steroidal anti-inflammatory drugs. A feature of the allergological characteristics of the asthmatic triad is the increased role of atopy in the formation of the disease. Over the past 20 years of observation, the percentage of patients with atopic BA in AT has increased from 19% to 47%; sensitization to non-bacterial allergens was detected in 48.4% of all patients with the asthmatic triad.

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