

**COMPREHENSIVE OVERVIEW OF BRONCHIAL ASTHMA: ETIOLOGY,  
PATHOGENESIS, CLINICAL MANIFESTATIONS, AND TREATMENT STRATEGIES**

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**Abstract:** This article provides a comprehensive overview of bronchial asthma, focusing on its etiology, pathogenesis, clinical manifestations, and treatment strategies. It discusses the role of allergens, central nervous system disorders, and chronic lung diseases in triggering asthma attacks. The article emphasizes the importance of early diagnosis and tailored treatment plans to manage symptoms and prevent exacerbations. Written for healthcare professionals and researchers, this article aims to enhance understanding and improve management of bronchial asthma, ultimately improving patient outcomes and quality of life.

**Keywords:** bronchial asthma, inflammation, hyperreactivity, allergens, airway obstruction, shortness of breath, Exacerbations, pulmonary function tests, bronchodilators, corticosteroids, allergen avoidance, respiratory gymnastics, quality of life.

**Introduction:** Bronchial asthma is a chronic disease on the basis of which lies chronic inflammation of the respiratory tract and hyperreactivity, which develops in the bronchi. It is accompanied by bronchial obstruction and various degrees of shortness of breath attacks, depending on the severity of the disease. Bronchial asthma has spread in different countries depending on factors such as climate, vegetation, weather pollution, urbanization rate of the same feed. According to the European Respiratory Society and the Russian pulmonologiary Congress in 2002, more than 150,000 people in the world are currently suffering from bronchial asthma. Severe types of bronchial asthma are increasing. For this reason, cases of dying from bronchial asthma are also increasing. This condition is due to the fact that the diagnosis of bronchial asthma is not made in time.

**Etiology:** There are many factors that can cause the appearance of bronchial asthma. Many scientists believe that a change in the state of reactivity of the human organism is one of the main causes of this disease. In bronchial asthma, as in all allergic diseases, an increase in the body's state of sensitivity to certain substances called "allergens" (especially when allergens have been acting on the body for a long time) is the cause of the disease. This means that the appearance of certain changes in the body under the influence of allergens sets the stage for the disease. But in some cases, the disease can also begin at once. In this case, the effect of the allergen on the body will not occur and will be passed on. The Allergen, consisting mainly of particles in protein, is completely safe for people with normal body sensitivity, healthy. Allergens can enter the body through breath, digestive tract, skin, and mucous membranes. In addition to bronchial asthma, allergens can also cause diseases such as donkeys, inflammation of the mucous membranes of the eyes (conjunctivitis), inflammation of the mucous membranes of the nasal cavity (rhinitis), rapid-onset tumors (Quincke's tumors), occasional numb headaches (migraines). Allergens can come in extremely different forms. Take, for example, the usual household dust. Its composition depends on the type of equipment in each household, pets, plants grown at home, and finally the insects

(cockroaches, woodworms, spiders, etc. In other words, each household will have its own allergen. Also, in manufacturing enterprises, libraries, the composition of dust is specific and can act as an allergen under certain conditions. Again, dust from cats, dogs, domestic birds, horses and other animals, droppings from poultry skins, wool, feathers can also cause bronchial asthma in young children. For example, it has been found that the digger and hair that fall out of a person's skin have an allergen property. In most houses, wonderful small fish are fed in the aquarium. But it should be remembered that in some cases, fish mints and, in particular, Daphnia, which is fed to fish as feed, processed by talcans, can cause an allergic condition, even bronchial asthma. Chemicals that are increasingly used in our daily routine (detergents, toxic chemicals used against insects, drugs that are sprayed on wet and dry fruits for long storage, etc.) also have allergenic properties. Another type of allergen is the substances contained in food. Products such as eggs, fish, chocolate, honey, strawberries, oranges, nuts can have an allergic effect on some people. When in infancy, some children who are separated from the breast early and fed artificial foods are given the above products, symptoms of an allergic disease can appear that can lead to their body from a rash to an attack of bronchial asthma. Allergens are classified into infectious and non-infectious types. Accordingly, the onset of the disease, its symptoms, development and complications differ from each other. The non-infectious allergic type of bronchial asthma occurs in most children. Older people hardly get sick with this type of disease. Bronchial asthma is more common in people with a cold airway, who often have a tummy tuck.

**Pathogenesis:** Three phases are differentiated in the pathogenesis of bronchial asthma:

- 1) Immunological phase in which antigens and antibodies interact with each other;
- 2) The pathochemical phase in which biologically active substances (histamine, acetylcholine, serotonin, bradykinin) are formed;
- 3) Pathophysiological phase;

In this, biologically active substances affect the bronchi muscle. The muscles tighten and contract, the small bronchi become narrower, the mucous membrane of the bronchi swells. A Talai sticky mucus comes out and makes it difficult to breathe. In addition to allergens, disorders of the functioning of the central nervous system also play a large role in the development of bronchial asthma. Excessive strain on the nerves, intense excitement, severe fear often cause the seizure of a bronchial asthma attack. While the smell of a single flower causes the disease to snore in this patient, the image of that flower can also cause him to squeeze his breath and choke. Bronchial asthma has been found to be dependent on several chronic lung diseases: bronchitis, stretched pneumonia. Bronchial asthma often leads to an outbreak of chronic bronchitis. Among other factors that affect the course of the disease, a large place is given to disorders of the endocrine system, including the functioning of the adrenal glands.

**Pathological anatomy:** In this disease, first of all, symptoms of emphysema, blockage of the bronchi from mucous or mucous-purulent clots, expressed bronchospasm are observed. At the same time, symptoms of bronchitis and chronic lung diseases are seen, which are satellite to bronchial asthma. When examined under a microscope, it is observed that the ciliated epithelium decreases and is replaced by goblet cells. There is a decrease in capillary cohesion by a small circulatory circle, extensive anastomosing cohesion, functionally active bridging formation, muscle floor hypertrophy, and infiltration of the thickened inner floor.

**Clinic:** The main clinical sign of bronchial asthma is a shortness of breath attack. Factors that provoke an outbreak of sensitivity are: the influence of non-infectious allergens, recurrence

of inflammatory diseases, nervous-mental tensions. Bronchial asthma attack occurs in three periods:

The first period is the period of combative symptoms of an attack. Symptoms of an attack are different in different patients, with a cough in the patient a few minutes or hours before the attack, itching of the throat, eyes, skin or ear. Supra, a lot of urination, etc. are observed. If the patient is able to detect the symptoms in a timely manner, he can achieve regression of the attack with the help of drugs.

The second period - expiratory is accompanied by signs of suffocation. During this period, the patient feels a "crunch" in the chest, unable to fully exhale. Exhaling is accompanied by remote whistling wheezes. The patient's face swells, bruises. The chest is emphysematous enlarged, standing in a deep breathing position. The neck vein is swollen. From time to time, a tormented cough is observed. Sputum is dark, sticky, sometimes foamy. When percussive, the sound of a box is heard, while pulmonary auscultation reveals that there is a talay of dry wheezing. The breathing period lasts 3-4 times longer than the breathing period. At the expense of a strong tumor of the lungs, the boundaries of the heart are not determined. The heart tone is sluggish, with a tone II accent in the pulmonary artery. The pulse is accelerated, the completeness is reduced. In long-term shortness of breath attacks, there are signs of heart right ventricular failure-enlarged liver, swelling in the legs. An X-ray examination shows increased airiness of the lungs, low aperture position, and alertness. The ribs settle horizontally, the range expands, the image of the lungs increases on X-ray. In long-term severe shortness of breath attacks, changes in the ECG appear. The electrical axis of the heart is shifted to the right. In the I Standard direction, the S tooth is deep, in the III direction, the R tooth is high. The large axial P is often negative in the standard directions II and III and in the direction V. In the positive, sometimes bi-peaked P tooth, V2, V2, an inversion of the ST segment is found in the right thoracic orientations. Giss tutami may have a blockage on his right leg. The severity of the course of bronchial asthma is assessed on the basis of clinical signs of the disease, as well as signs of pulmonary dysfunction detected in the spirometer and picfloumetr.

The third period is the relapse of the attack. During this period, a large amount of phlegm cough appears, which gives the patient relief. In Hoi, which depends on the pathogenesis of the disease, disease regression occurs every xiI. This period is fast without change in other members, sometimes it can be slow by disabling the patient as the opposite. Bronchial asthma can lead to complications with the addition of chronic bronchitis, which in turn causes pulmonary emphysema, chronic respiratory failure, and pulmonary-heart disease.

International consensus according to the course of bronchial asthma (USA, 1995-Y.) in 4 stages based on the classification:

1. Intermittent asthma (I-stage). Short-term relapse (cough attacks, whistling breathing, dyspnoe) does not exceed once a week. Relapse lasts from several hours to several days. A night attack is returned up to 2 times a month. In the period between attacks, there are no symptoms of the disease, the activity of the external breath has not changed, that is, the required indicators of the volume of rapid breathing in the spirometer and the speed of the exhalation peak in the pikiofluometer are more than 80%. During the day, the change limit of this kursatkich is up to 20%.

2. Symptoms of relapse in mild persisting asthma (Stage 2) are one or more times a week, but no more than once a day. The patient's sleep and physical activity are disturbed. Night attacks

increase more than twice a month. This group consists of 80% or more of the necessary indicators of the volume of accelerated breathing and the speed of the exhalation peak in patients, the change limit of indicators will be 20-30%. Symptoms of bronchial asthma are controlled with the intake of permanent broncholytic drugs.

3. In moderate-to-severe persisting asthma (stage 3), the symptoms of everyday illness are long-lasting. Night shortness of breath attacks are observed more than once a week. The patient drinks broncholytic drugs daily. In this group of patients, JNCHH and NCHCHT make up between 60% and 80% of the required indexing, with the daily variation limit of these rates increasing by more than 30%.

4. In severe persisting asthma (stage 4), the patient has long symptoms of the disease, attacks are repeated more often, physical activity is limited, severe shortness of breath attacks are repeated despite taking medications. In this group of patients, JNCHH and NCHCHT are less than 60% of the required indexing in the period up to the reception of the drug, with a daily change limit of this indexing of more than 30%.

**Treatment:** While the treatment of bronchial asthma is aimed at treating an asthma attack or asthmatic condition on the one hand, on the other hand, the pathogenetic process of the disease is taken into account. When stopping a bronchial asthma attack, simpatomimetics, xanthine compounds, cholinolytic drugs are used. A positive change is observed as sympathomimetics increase the functional activity of b-adrenergic receptors in the bronchial smooth muscle. Sympathomimetics are divided into 4 groups according to what type of adrenergic receptor they affect:

- 1) A-and B-receptor stimulants (adrenaline, ephedrine);
- 2) a-adrenergic receptor stimulants (norepinephrine, rnezaton, sympathol);
- 3) Non-selective J3-stimulants (novodrin, isadrin, astmopent, alupent, etc.);
- 4) Selective 132 stimulant (berotek, salbutamol, terbutalin, phenoterol, formoterol);

The broncholytic effect of ephedrine is slower than that of adrenaline and is relatively long-lasting. It is recommended to administer 0.4 ml of 0.1% II adrenaline solution and 1 ml of 5% II ephedrine solution under the skin at the same time. Ephedrine can be prescribed to drink from 25 mg. Non-selective 13-stimulants are commonly used as pocket thinners or drugs used under the tongue. From selective 132 - stimulants, dosed ingaliers are used: terbutalin (bricanyl), salbutamol (ventolin), berotek, formoterol. Resistance to these drugs is poorly developed. In some cases, when treated with sympathomimetics, patients have a worsening condition, a shortness of breath attack is aggravated, seizures are more recurrent. When treated with sympathomimetics, the obstruction of the bronchi, the exacerbation of cirrhosis indicates that treatment with them should be discontinued. When treated with sympathomimetics, as a result of their negative effects, an increase in blood pressure, hand tremors, dizziness are observed. Cardiac arrhythmias or sudden death may occur in patients with circulatory disorders in large crown vessels of the heart as a result of the toxic effects of sympathomimetics on the myocardium. When returning an asthma attack, 2.4% II is administered 5-10 ml of euphylline to the vein. This treatment has a hypotensive effect on a small circle of blood circulation, in addition to broncholytic action. In the elimination of tachycardia caused by Eufillin treatment, especially when the disease is accompanied by heart failure, verapamil (isoptin) is given to drink from 80-120 mg. In the return of mild attacks of asthma, euphylline can be used as a suppository (0.3-0.5 g). Cholinolytics (atropine, platifillin) have a weaker effect on sympathomimetics and euphylline, but they enhance the effect of

sympathomimetics. Atropine is given subcutaneously (0.5 ml of 0.1% II solution) or in a thinning case (0.1% II solution). Ipratropine bromide (atrovent) from atropine derivatives is prescribed in the Prevention of asthma attacks (from 20-40 mg to 3 times). Sympathomimetics and cholinolithics - berodual, ephatin, troventol-are used in medical practice. In the case of a slight shortness of Breath Attack of bronchial asthma, drugs are given to drink, as a suppository or dosed ingestion. In the treatment of moderate to severe attacks, the drug is injected into the vein, under the skin and between the muscles. In the Prevention of an asthma attack, long-acting (12-18 hours) compounds of theophylline can be used: theopek, theobiolong and others. In the treatment of stage I of asthmatic condition, 200-250 ml of blood is excreted, followed by infusion treatment. In order to combat developing dehydration, 2 I and more liquids (isotonic solution of table salt, 5% II glucose solution, reopoliglyukin) are injected per day. In addition, 10 ml of 0.9% II sodium iodide solution (in the absence of allergies to iodine treatment) is administered to the vein for the purpose of diluting eufillin (2.4% Ii solution 10 ml), Heparin (5000 TB), phlegm. In order to eliminate acidosis in the patient's body, a solution of 200-400 ml of 4% II sodium bicarbonate is dripped into a vein. In an effort to re-establish sensitivity of J3-adrenergic receptors to cypmatomimetics, venase is administered slowly but or drip 30-60 mg prednisolone (250-300 mg metipred or 125-300 mg hydrocortisone) every 3 hours at an indicated dose until the asthma attack stops. When the " silent lung " stage is formed, the dose of prednisolone administered to the vein is increased to 90-120 mg, using previous stage treatment methods, and its administration returns every 1.5-2 hours. If signs of congestion appear within the framework of a small circulation and there is a risk of pulmonary edema, it is necessary to carry out a dehydrating treatment by adding urinary drugs with potassium salts. If the treatment does not work, then in a short time - 2-3 hours the patient is transferred to controlled breathing, aspiration of the bronchi is carried out. Treatment of Stage III of asthmatic condition is carried out in the conditions of the intensive care unit. Treatment carried out in the middle of seizures, measures to prevent seizures depend on the pathogenetic type of bronchial asthma. In patients with atopic bronchial asthma, hygienic measures should be carried out to stop exposure to allergens in the apartment, exclude foods that cause allergies from the composition of food, when there is hypersensitivity to house dust. If there is no possibility to stop the causes of allergies, desensitizing treatment is carried out. Specific desensitization is performed by injecting a specific allergen into an overdose. In infectious-allergic asthma, desensitization can be performed with autovaccine made from patient sputum flora. Antibacterial treatment is carried out only in cases where infectious colds are confirmed. Unreasonable use of antibiotics can aggravate the course of the disease. In order to prevent recurrence of infectious-allergic asthma, it is recommended to use immunostimulants (levamisol, polysaccharides, T-activin, sodium nucleinate). In physical exertion asthma, calcium antagonists are prescribed: corinfar (3 times a day from 0.01 g), isoptin (2 times a day from 120 mg), cenizite (3 times a day from 50 mg), diltiazem. In physical activities, it is recommended to gradually increase physical exertion. In addition to hormones, immunodepressants (azathioprine, 6-mercaptopurine) are also used when the disease is very severe. Extracorporeal blood purification methods (hemisorption, immunosorption, plasmaphoresis) are used. The most used in the treatment of bronchial asthma is hemisorption. In medicine, it is also observed that bronchial asthma can be treated surgically, physiotherapeutic, Without Medication (respiratory Gymnastics, psychotherapy, barocamera).

**Conclusion:** In conclusion, bronchial asthma is a complex and chronic condition that affects millions of people worldwide. It is characterized by chronic inflammation of the respiratory

tract, bronchial obstruction, and varying degrees of shortness of breath attacks. The prevalence of severe types of bronchial asthma is increasing, leading to a rise in asthma-related deaths. The etiology of bronchial asthma is multifactorial, with allergens playing a significant role in triggering the disease. Allergens can enter the body through various routes, such as inhalation, ingestion, or contact with the skin, leading to allergic reactions that contribute to the development of bronchial asthma. Treatment of bronchial asthma aims to manage acute asthma attacks and prevent future attacks. Bronchial asthma is a complex and challenging disease that requires a comprehensive approach to management. Early diagnosis, proper treatment, and lifestyle modifications can help improve the quality of life for individuals living with bronchial asthma.

**REFERENCES:**

1. Ärztliches Zentrum für Qualität in der Medizin. Nationale Versorgungs-Leitlinie Asthma bronchiale *Dtsch Arztebl.* 2005;102(40):A 2734–A 2734.
2. Bateman ED, Hurd SS, Barnes PJ, et al. Global strategy for asthma management and prevention: GINA executive summary. *Eur Respir J.* 2008;31:143–178.
3. Buhl R, Berdel D, Criege C-P, Gillissen A, Kardos P, Kroegel C, et al. Leitlinie zur Diagnostik und Therapie von Patienten mit Asthma. *Pneumologie.* 2006;60:139–183.
4. Suissa S, Ernst P. Inhaled corticosteroids: impact on asthma morbidity and mortality. *J Allergy Clin Immunol.* 2001;107:937–944.
5. Reddel HK, Salome CM, Peat JK, Woolcock AJ. Which index of peak expiratory flow is most useful in the management of stable asthma? *Am J Respir Crit Care Med.* 1995;15:1320–1325.
6. Cockcroft DW, Murdock KY, Berscheid BA, Gore BP. Sensitivity and specificity of histamine PC20 determination in a random selection of young college students. *J Allergy Clin Immunol.* 1992;89:23–30.
7. Green RH, Brightling CE, McKenna S, Hargadon B, Parker D, Bradding P, et al. Asthma exacerbations and sputum eosinophil counts: a randomised controlled trial. *Lancet.* 2002;360:1715–1721.