МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ПОЛИКЛИНИЧЕСКОЙ ТЕРАПИИ

ДИФФЕРЕНЦИАЛЬНАЯ ДИАГНОСТИКА БРОНХООБСТРУКТИВНОГО СИНДРОМА. ХРОНИЧЕСКАЯ ОБСТРУКТИВНАЯ БОЛЕЗНЬ ЛЕГКИХ И БРОНХИАЛЬНАЯ АСТМА

DIFFERENTIAL DIAGNOSIS OF BRONCHIAL OBSTRUCTIVE SYNDROME. CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND BRONCHIAL ASTHMA

Учебно-методическое пособие

Минск БГМУ 2026

УДК [616.24:616-007.272:616-036.12]:[616.248:611.233]:616-07(075.8)=111
ББК 54.12я73
Д50

Рекомендовано Научно-методическим советом университета в качестве учебно-методического пособия 15.04.2026 г., протокол № 8

Авторы: Е. С. Алексеева, Н. М. Ерёмина, М. В. Зюзенков, Е. А. Каразей

Рецензенты: канд. мед. наук, доц. каф. внутренних болезней, кардиологии и ревматологии с курсом повышения квалификации и переподготовки Белорусского государственного медицинского университета О. А. Паторская; каф. общей врачебной практики Витебского государственного ордена Дружбы народов медицинского университета

Дифференциальная диагностика бронхообструктивного синдро-
Д50 ма. Хроническая обструктивная болезнь легких и бронхиальная астма = Differential diagnosis of bronchial obstructive syndrome. Chronic obstructive pulmonary disease and bronchial asthma : учебно-методическое пособие / Е. С. Алексеева, Н. М. Ерёмина, М. В. Зюзенков, Е. А. Каразей. – Минск : БГМУ, 2026. – 34 с.

ISBN 978-985-21-2266-5.

Рассматриваются причины и механизмы развития, этапы диагностического поиска и дифференциальная диагностика бронхообструктивного синдрома, а также особенности ведения пациентов с хронической обструктивной болезнью легких и бронхиальной астмой врачом общей практики в амбулаторных условиях.

Предназначено для специальности «Лечебное дело» по учебной дисциплине «Поликлиническая терапия».

УДК [616.24:616-007.272:616-036.12]:[616.248:611.233]:616-07(075.8)=111
ББК 54.12я73

ISBN 978-985-21-2266-5

ABBREVIATIONS LIST

ACT — asthma control test
ACQ — asthma control questionnaire
AS — anaphylactic shock
ALVF — acute left ventricular failure
ASIT — allergen-specific immunotherapy
BA — bronchial asthma
BOS — bronchial obstructive syndrome
BT — bronchodilation test
CBC — complete blood count
CNLDs — Chronic nonspecific lung diseases
COPD — chronic obstructive pulmonary disease
DPI — dry powder inhaler
ECHO — echocardiography
ESR — erythrocyte sedimentation rate
EGD — esophagogastroduodenoscopy
FBS — fiberbronchoscopy
FEV — Forced expiratory volume
FEV1 — forced expiratory volume in 1 second
FVC — forced vital capacity
GIBP — genetically engineered biological preparation
ICS — inhaled corticosteroids
LABA — long-acting beta2-agonists
LAMA — long-acting muscarinic antagonists
LTRAs — Leukotriene Receptor Antagonists
MART therapy — Maintenance and Reliever Therapy
MDI — metered Dose Inhaler
NSAIDs — non-steroidal anti-inflammatory drugs
PE — pulmonary embolism
PEF — Peak Expiratory Flow
PV — pulmonary vasculitis
RF — respiratory failure
SABA — short-acting beta2-agonists
SAMA — short-acting muscarinic antagonists
SP — spontaneous pneumothorax
TBD — tracheobronchial dyskinesia

MOTIVATIONAL CHARACTERISTICS OF THE TOPIC

Total lesson duration: 6 hours.

Chronic nonspecific lung diseases (CNLDs), the main representatives of which are chronic obstructive pulmonary disease (COPD) and bronchial asthma (BA), represent a significant global public health problem. COPD, in particular, ranks third among all-cause mortality in the population over 45 years of age. The prevalence and seriousness of these diseases require early diagnosis and a comprehensive approach to prevention and treatment. One of the main clinical syndromes characteristic of CNLDs is broncho-obstructive syndrome (BOS), or bronchial obstruction syndrome. The widespread prevalence of BOS, as well as the heterogeneity of its development, course, and outcome, have remained a pressing issue for many years. Manifestations of this syndrome, such as cough, shortness of breath, and suffocation, are not specific to lung diseases and often require differential diagnosis. Proper knowledge of differential diagnosis is an important aspect of the work of a physician in any specialty. This knowledge develops the physician's skills in clinical reasoning, systemic symptom analysis, and comprehensive patient management. Obtaining a correct diagnosis in the early stages of the disease helps initiate treatment promptly, achieve sustained remission, and reduce the risk of complications. This process requires extensive knowledge not only in their field but also in related specialties.

Therefore, a detailed study of this topic during the training of future qualified general practitioners will deepen and systematize their knowledge in the field of diagnosis and personalized management of patients with broncho-obstructive syndrome.

The purpose of the lesson. Develop specialized competencies for organizing and providing primary medical care to adults with chronic obstructive pulmonary disease and bronchial asthma in an outpatient setting, conducting temporary disability assessments, and providing emergency medical care in an outpatient setting.

Objectives of the lesson. Develop students' scientific knowledge about the provision of medical care, treatment strategies, principles of primary and secondary prevention, and rehabilitation of patients with COPD and bronchial asthma in an outpatient setting. Acquire practical skills in differential diagnosis, interpreting examination data, formulating clinical diagnoses and preparing medical documentation, conducting temporary disability assessments, and providing emergency care during an asthma attack.

Basic knowledge requirements. To fully master the topics in the «Human Anatomy», «Normal Physiology», and «Pathological Physiology» sections, students must review the anatomy and functions of the respiratory system, as well as the pathophysiological mechanisms of cough, dyspnea, and airway obstruction.

Test questions from related disciplines:

1. Anatomy of the lungs and heart.
2. Physiology of the respiratory system.
3. Pathophysiological changes in BOS, mechanisms of obstruction development.
4. Pathophysiological changes in the lungs in heart failure.
5. Laboratory methods for testing blood, urine, sputum, and ECG in lung diseases.

Test questions on the topic of the lesson:

1. The concept of broncho-obstructive syndrome and its variants, the main diseases associated with this syndrome.
2. Diagnostic search algorithm for BOS.
3. Classification of bronchial asthma and COPD.
4. Outpatient examination plan for a patient with bronchial asthma and COPD, diagnosis formulation.
5. Outpatient treatment principles for bronchial asthma and COPD, disability assessment.
6. Outpatient medical monitoring of patients with bronchial asthma and COPD, prevention.
7. Emergency medical care during an asthma attack

Assignments for independent student work. Review the anatomy and physiology of the respiratory and cardiovascular systems, and the pathophysiology of bronchial obstruction. Familiarize yourself with the clinical symptoms and diagnostic signs of lung diseases and other conditions associated with bronchial obstruction, as well as the diagnostic methods used for these conditions. Explore the mechanisms of action, indications and contraindications, and dosages of medications used for COPD and bronchial asthma.

GENERAL PROVISIONS

Broncho-obstructive syndrome (BOS) is a clinical symptom complex caused by functional or organic impairment of bronchial patency and manifested by paroxysmal cough, expiratory dyspnea, or attacks of suffocation.

The main pathophysiological mechanisms of impaired bronchial patency can be reversible or irreversible, including:

1. Spasm of bronchial smooth muscles (bronchospasm).
2. Edematous-inflammatory changes of the bronchi of any origin (allergic, inflammatory, toxic, etc.).
3. Hypersecretion and accumulation of viscous bronchial secretions in the bronchial lumen (dyscrinia).

4. Tracheobronchial dyskinesia (prolapse of the trachea and large bronchi).

5. Expiratory collapse of small bronchi («air trapping») due to loss of lung elasticity.

6. Remodeling of the bronchial wall (peribronchial fibrosis, cicatricial stenosis).

7. Mechanical obstruction of the bronchi by foreign bodies, vomit, pus, blood; endobronchial tumors, cicatricial narrowing of the bronchial lumen, external compression of the bronchus.

Spasm of smooth muscles and mucus hypersecretion occur as a result of irritating factors (pollutants, infection, etc.) acting on the airway mucosa. In response, inflammatory mediators are released, irritating vagus nerve endings and promoting acetylcholine release, which acts via muscarinic cholinoreceptors. Activation of these receptors causes cholinergic bronchoconstriction and hypersecretion. In the bronchial wall, there is marked vascular engorgement of the microcirculatory bed and increased permeability. This leads to mucosal and submucosal edema, infiltration by mast cells, basophils, eosinophils, lymphoid and plasma cells. Chronic inflammation results in peribronchial fibrosis, bronchial remodeling, loss of elastic traction and recoil. This process is one of the mechanisms of (poorly reversible) bronchial obstruction in COPD. Destruction of lung parenchyma leads to loss of alveolar attachments to small airways and reduced elastic recoil, impairing the ability of airways to remain open during exhalation. These processes result in «air trapping», increased airway resistance, and irreversible airflow limitation.

Classification of BOS:

1. By etiology and pathogenesis:

1) infectious-inflammatory: COPD, bronchitis, pneumonia, bronchial and pulmonary tuberculosis, pulmonary syphilis, mycoses;

2) allergic: bronchial asthma, serum sickness, drug allergy, pollinosis, anaphylactic shock;

3) immune (autoimmune): diffuse connective tissue diseases, systemic vasculitis, rheumatoid arthritis, pneumoconiosis, parasitic lung lesions, Dressler's syndrome, periodic disease, post-transplantation syndrome, etc.;

4) obstructive: COPD, malignant and benign bronchial tumors, foreign body in airways, broncholithiasis, bronchostenosis of various etiologies, cystic fibrosis, Mendelson's syndrome (acid aspiration pneumonitis), etc.;

5) dyskinetic: congenital or acquired tracheobronchial dyskinesia, bronchostenosis;

6) irritative: thermal and chemical burns of bronchi, intubation, inhalation of toxic substances, mechanical irritation of bronchial mucosa;

7) hemodynamic: congestive left ventricular failure, primary pulmonary arterial hypertension, pulmonary embolism, mitral stenosis, severe hypertensive crisis, persistent ventricular tachycardia;

8) endocrine-humoral: hypoparathyroidism, diencephalic syndrome, carcinoid syndrome, carcinoid tumors;

9) toxic: poisoning with cholinergic substances, drugs causing bronchospasm (β-blockers, acetylcholine, histamine, etc.), NSAIDs, radiocontrast agents;

10) neurogenic: post-contusion syndrome, mechanical irritation of the vagus nerve due to compression or surgical transection, hysteria, vegetative dystonia, hyperventilation syndrome.

2. By course:

1) latent (detected only by functional testing of bronchial patency);

2) clinical (with typical symptoms).

3. By the degree of severity of the pulmonary ventilation function disorders:

1) mild: air enters alveoli through narrowed bronchi and exits in equal volume → hypoventilation; FEV1 > 70 % of predicted;

2) moderate (valve mechanism obstruction): air enters alveoli during inspiration, but stenotic or inelastic bronchi collapse during expiration → «air trapping» with obstructive emphysema;

3) moderate: FEV1 60–69 %;

4) moderately severe: FEV1 50–59 %;

5) severe: complete bronchial closure; FEV1 49–35 %;

6) extremely severe: FEV1 < 35 %.

The degree of ventilation impairment does not always strictly correlate with the severity of clinical manifestations of respiratory diseases. Furthermore, changes in FEV1 are not recommended for assessing the severity of extrapulmonary airway obstruction, as even a slight decrease in FEV1 can significantly increase its clinical manifestations.

4. By localization:

1) upper airways (extrathoracic) — limitation of inspiratory airflow;

2) lower airways (intrathoracic) — limitation of expiratory airflow;

3) variable — at any level affected by transmural compression;

4) persistent — at any level not affected by transmural compression; alters both inspiration and expiration.

DIAGNOSTICS AND STAGES OF DIAGNOSTIC SEARCH

Stage I — collecting diagnostic information about the patient: analysis of complaints, anamnesis, examination data, and initial (routine) laboratory and instrumental examination.

All details of the complaints, anamnesis, and physical examination data can influence the successful development of a diagnostic concept and the correct choice of tactics for this group of patients.

Interview:

The patient's complaints include the following symptoms:

1) shortness of breath;

2) cough;

3) noisy breathing (wheezing).

It is also necessary to pay attention to additional symptoms, such as: fever, pain in various locations, catarrhal symptoms, dyspeptic symptoms, palpitations and discomfort in the cardiac region, and edema.

During the interview, it is necessary to clarify:

– the nature of shortness of breath (expiratory, inspiratory, mixed);

– the course (episodic, constant);

– the duration of the attack if paroxysmal;

– conditions of onset and provoking factors;

– symptom dynamics throughout the day and the presence of nighttime symptoms;

– conditions for relief;

– if a cough is present, its productivity, sputum type (amount, color, odor, impurities), timbre, conditions of onset, and relief.

History:

– When did symptoms first appear, and what does the patient associate with their onset?

– How long ago did accompanying symptoms appear?

– What illnesses, surgeries, or injuries has the patient had (and when)?

– What chronic illnesses have been diagnosed, and is the patient taking any medications?

– Determine the presence of risk factors for any illnesses, addictions, or family history.

– Pay attention to all details of the allergy history.

– Collect a professional history.

During the examination, note the following:

– the patient's position;

– skin and mucous membrane color;

– presence of edema;

– vascular pulsation;

– inspection, palpation, percussion, and auscultation of the chest;

– respiratory rate, heart rate, and blood pressure;

– examination of organs and systems;

– examination of the lower extremities.

Typical objective signs of BOS include: forced patient positioning (orthopnea with shoulder girdle fixation), cyanosis of the mucous membranes, sometimes acrocyanosis (warm cyanosis), distant wheezing, and the use of accessory respiratory muscles in breathing, especially during exhalation. Weakened vocal fremitus, a hyperbaric percussion sound, weakened vesicular breath sounds with a prolonged (more than 5 seconds) wheezing expiration, and dry wheezing symmetrically over the lungs on both sides.

Prehospital examination methods upon initial presentation, if the patient's condition is not urgent and does not require emergency hospitalization:

– complete blood count, complete urine analysis, and bacterial blood test;
– sputum analysis;
– ECG;
– spirometry with bronchodilator tests;
– peak flowmetry, pulse oximetry;
– chest X-ray;

Examination methods (as indicated):

– sputum analysis for Koch's bacilli and atypical cells;
– bronchoscopy;
– lung tomography;
– echocardiography;
– allergist consultation, allergy testing, and other tests;
– endoscopy, abdominal ultrasound;
– immunoblotting;
– consultation with a phthisiologist, endocrinologist, or oncologist.

A forced expiratory volume in 1 second (FEV1) of less than 80 % of the predicted value and an FEV1/FVC (forced vital capacity) ratio of less than 70 % indicate obstruction based on spirometry results. A FEV1/FVC ratio of less than 70 % is the earliest manifestation of BOS.

Stage II — formulating a primary diagnostic hypothesis and determining patient management strategies.

If a patient has a history of acute respiratory distress syndrome (RDS), it is first necessary to rule out or confirm life-threatening conditions requiring emergency hospitalization and urgent care.

Such conditions include: an asthma attack, developing acute left ventricular failure (ALVF) or cardiac asthma, pulmonary embolism (PE), anaphylactic shock (AS), and spontaneous pneumothorax (SP).

The differential diagnostic features of these conditions are presented in table 1.

Table 1

Diagnostic features of the main emergency conditions accompanied by BOS

Signs	Asthma	ALVF	Pulmonary Embolism	Anaphylactic Shock	Spontaneous Pneumothorax
History	Atopy (rhinitis, urticaria, previous attacks), family history	Hypertension, coronary heart disease, heart defects, cardiomyopathy, severe rhythm disturbances	CHF, leg vein pathology, prolonged bed rest, tumors, and recent surgeries	Atopy (rhinitis, urticaria, angioedema, bronchial asthma)	Trauma, COPD
Cause of attack	Contact with an allergen, exacerbation of inflammation, can occur at any time	Physical or mental stress, hypertensive crisis, arrhythmia	Activation after bed rest, paroxysmal arrhythmia, phlebitis	Contact with an allergen	Physical activity, cough, injury
Patient's position	Forced, arm rest («coachman's pose»)	Sitting, standing (orthopnea)	Restlessness	Horizontal	On the side of the lesion
Skin color	Pallor, diffuse cyanosis	Cold, pale cyanosis	Cyanosis of the face and neck	Paleness	Cyanosis
Pain syndrome	None	Anginal pain	Chest pain of varying intensity and location	None	Intense
Respiration characteristics	Difficulty	Frequent, superficial	Frequent, superficial	Difficult	Frequent, superficial
Shortness of breath	Expiratory	Inspiratory, paroxysmal	Inspiratory	Expiratory	Inspiratory
Cough	Dry sputum at the beginning of the attack, with viscous sputum at the end	Productive, pink, foamy sputum with acute pulmonary embolism	Cough with bloody-mucous sputum (in a third of cases)	Dry cough possible	None
Sputum	High-pitched sound, lower lung border	Dullness in the lower extremities	Pulmonary	Pulmonary	Dull on the affected side

End of the table 1

Signs	Asthma	ALVF	Pulmonary Embolism	Anaphylactic Shock	Spontaneous Pneumothorax
Percussion of the lungs	Hyperresonance, lowering of the lower border of the lungs	In the lower parts — dullness	Vesicular lung sounds	Vesicular lung sounds	Dull sound on the affected side
Auscultation of the lungs	Wheezing	Various sounds: rales, rhonchi	Variable	Variable	Silent lung
Percussion and auscultation of the heart	Borders unchanged, regular rhythm, tachycardia	Enlarged heart, muffled heart sounds, arrhythmias, murmurs	Boundaries unchanged, accentuated and heart sound over the pulmonary arteries	The borders of the heart are not changed, regular rhythm, tachycardia	Tachycardia, displacement of cardiac borders
ECG	Right-sided overload	left-sided overload. Heart rhythm disturbances, signs of ischemia	Right-sided congestion, S1-Q3, P-pulmonary	Non-specific, rhythm disturbances	Non-specific, rhythm disturbances

If the patient exhibits no signs of a life-threatening condition, the next step is diagnosis and differential diagnosis of the most common conditions in general practice that manifest as respiratory distress syndrome (RDS), such as COPD, asthma, laryngeal edema, pneumonia with RDS, as well as diseases accompanied by signs of infectious or allergic diseases. Differential diagnostic features are presented in table 2.

It is important to remember that acute or recurrent bronchial obstruction (due to insufficient airflow to the respiratory tract) can be caused by mechanical airway obstruction by a lung tumor, foreign bodies (dentures, seeds, food particles, etc.), external bronchial compression (substernal goiter, large thoracic lymph nodes, etc.), gastric contents entering the respiratory tract due to GERD, aspiration of vomit, and tracheobronchial dyskinesia (TBD) — expiratory prolapse of the membranous portion of the trachea and large bronchi, congenital or acquired. The differential diagnostic features of these conditions are presented in table 3.

Table 2

Diagnostic features of COPD, asthma, laryngeal edema, pneumonia with BOS

Signs	COPD	Asthma	Laryngeal edema	Pneumonia with BOS
Onset of the disease, patient age	Gradual onset, age over 40	Gradual onset, any age	Acute onset, any age (often children)	Acute onset, any age
History (risk factors, causes)	Smoking, exposure to pollutants, infections, genetic factors, perinatal pathology	Atopy, characterized by immediate allergic reactions, genetic factors	Viral and bacterial infections, contact with an allergen	Viral and bacterial infections, bronchial hyperreactivity
Reversibility of obstruction	Uncommon	Characteristic	Characteristic	Characteristic
Fever	Uncommon	Uncharacteristic	Possible	Characteristic
Cough	Persistent, variable intensity, often productive	Epischolarly, non-productive or low-productive	Non-productive, paroxysmal, rough, barking, accompanied by hoarseness and salivation	The nature of the cough is variable, depending on the etiologic factor
Character of sputum	Viscous, purulent, neutrophils on microscopy, positive bacterial culture	Sparse, glassy, dense; microscopic examination reveals eosinophils, Curschmann spirals, and Charcot-Leyden crystals	Usually absent	Sputum varies in character, depending on the etiologic factor; bacterial culture is positive.
Shortness of breath (dyspnea)	Constant, expiratory	Paroxysmal, expiratory	Inspiratory or mixed	Inspiratory or mixed
Pain	Not typical	Not typical	Possible	Common
Response to bronchodilator therapy	Moderate, depends on the stage of the disease	Good	Moderate, used in combination with other drugs	Moderate, used in combination with other medications

End of the table 2

Signs	COPD	Asthma	Laryngeal edema	Pneumonia with BOS
Physical data	Barrel-shaped chest, limited mobility of the lower lung edge, box-like percussion sound, dry wheezing	Unremarkable between attacks; during attacks, precussion and auscultation are similar to the physical findings of COPD	Harsh, conductive breath sounds, noisy, creaky inhalation with head tilt back	Increased vocal fremitus, localized dullness of pulmonary sounds, increased or decreased breath sounds, sonorous moist rales and crepitations
CBC	Normal or polycythemia, leukocytosis with neutrophilia, accelerated ESR syndrome	Eosinophilia, possible accelerated ESR syndrome	Variable, often leukocytosis with neutrophilia or leukopenia with lymphocytosis, accelerated ESR syndrome	Leukocytosis with neutrophilia, accelerated ESR syndrome
Instrumental diagnostics	Spirometry with bronchodilator test. FEV1 increase < 15 %	Spirometry with bronchodilator test. FEV1 increase > 15 %, peak flow measurement	Assessment of clinical status and response to therapy	Chest X-ray

Table 3

Diagnostic signs of a foreign body in the respiratory tract, lung cancer, GERD, and TBD

Signs	Foreign Body	Lung Cancer	GERD	TBD
Onset of the disease, age of patients	Acute onset, any age (often children)	Gradual onset, age over 50 years	Gradual onset, any age	Gradual onset, young age
History (risk factors, causes)	Talking, laughing, playing while eating, careless handling of small objects, dental procedures, alcohol intoxication	Smoking, including passive smoking, inhalation of carcinogens (asbestos, radon, arsenic), genetic factors, chronic lung disease	Smoking, alcohol abuse, poor diet, stress	Congenital — genetic factors; acquired — viral, bacterial, and fungal infections, respiratory irritation, and bad habits
Reversibility of obstruction	Characteristic	Not typical	Characteristic	Variable, depending on the cause and stage of the disease

Continuation of the table 3

Signs	Foreign Body	Lung Cancer	GERD	TBD
Fever	Uncharacteristic	Characteristic, prolonged, subfebrile	Uncharacteristic	Uncommon
Cough	Sudden, intense, paroxysmal, severe, associated with foreign body ingestion	Constant, debilitating, unproductive, becoming paroxysmal, severe	Long-term, unproductive, worsens after meals, in a horizontal position, at night	Paroxysmal, nonproductive, sonorous, bitonal (barking) wheezing, intensifies in a horizontal position, with physical activity, and acute respiratory infections
Character of sputum	Absent	Hemoptysis	Absent	May be mucopurulent
Shortness of breath	Inspiratory (mixed), hoarse, wheezing, respiratory arrest	Develops gradually, inspiratory, then mixed	Absent or variable, as in asthma or pneumonia	Inspiratory wheezing during physical activity
Signs	Foreign Body	Lung Cancer	GERD	TBD
Other symptoms	Hoarse voice, aphonia, chest pain, laryngeal pain, gagging	Weakness, loss of appetite, weight loss, itching, chest pain, joint pain, bone pain, carcinoid syndrome	Heartburn, belching, dysphagia, chest pain, hoarseness, stomatitis	Dysphagia, chest pain, hoarseness
Physical findings	Cyanosis, forced position, wheezing	Sparse, possibly localized shortening of the percussion sound, weakening of vesicular breath sounds, dry wheezing that persists after dyspnea subsides	Variable in nature, similar to asthma or pneumonia. Epigastric pain may occur upon palpation	Nonspecific
Complete blood count (CBC) data	Normal	Anemia, thrombocytopenia or thrombocytosis, elevated ESR syndrome	Normal	Normal, possible leukocytosis, elevated ESR syndrome
Instrumental diagnostics	Clinical status assessment, X-ray, CT, bronchoscopy	X-ray, CT, bronchoscopy	EGS	X-ray, bronchoscopy, CT

End of the table 3

Signs	Foreign Body	Lung Cancer	GERD	TBD
Response to bronchodilator therapy	Absent	Weak, can be used in combination with other drugs	Absent	Weak, can be used in combination with other medications

BOS in autoimmune diseases is a manifestation of lung damage associated with systemic pulmonary vasculitis, systemic lupus erythematosus, rheumatoid arthritis, and systemic sclerosis. It is caused by autoimmune processes that induce inflammation and damage to the airways, leading to bronchial narrowing. Given the similarity of the lung damage to bronchial asthma, systemic pulmonary vasculitis (SPV) is the most important differential diagnosis. These include eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome), Wegener's granulomatosis, and microscopic polyangiitis. In these diseases, lung damage is also caused by inflammation of the pulmonary vascular walls. The differential diagnostic features of bronchial asthma and OBS are presented in table 4.

Table 4

Diagnostic features of asthma and SPV

Signs	Asthma	SPV
Disease onset, patient age	Gradual onset, any age	Gradual onset, middle age
History (risk factors, causes)	Atopy, characterized by immediate allergic reactions, genetic factors	Genetic factors, causes unknown
Obstruction reversibility	Characteristic	Uncommon
Fever	Uncharacteristic	Possible, subfebrile
Cough	Paroxysmal, non-productive or low-productive	Emergency, nonproductive or low-productive
Sputum type	Sparse, glassy, dense, microscopically revealing eosinophils, Curschmann spirals, and Charcot-Leyden crystals	Hemoptysis
Dyspnea	Paroxysmal, expiratory	Develops gradually, inspiratory, mixed
Other symptoms	Uncharacteristic	Weakness, loss of appetite, weight loss, pruritus, chest pain, arthralgia (arthritis), myalgia, skin and mucous membrane lesions, urinary syndrome (glomerulonephritis)

End of the table 4

Signs	Asthma	SPV
Physical findings	No abnormalities between attacks; during attacks, a box-like percussion sound and dry wheezing are present	Variable. A thorough examination of organs and systems is necessary to identify signs of a systemic process.
CBC	Eosinophilia, possible elevated ESR syndrome	Anemia, thrombocytosis, neutrophilic leukocytosis, eosinophilia, accelerated ESR syndrome
Instrumental diagnostics	spirometry with bronchodilator test. FEV1 increase > 15 %, peak flow measurement	R-graphy, CT, MRI angiography, spirometry with bronchodilator test, bronchoscopy, immunological laboratory diagnostics
Response to bronchodilator therapy	Good	None

The final stage of differential diagnosis allows for neurogenic and functional causes of bronchial obstruction. Neurogenic BOS occurs in neurasthenia, hysteria, and other central nervous system disorders as attacks of psychogenic dyspnea or persistent dyspnea in young patients (more often women), usually in response to traumatic events. A tendency toward neurotic or hysterical reactions is revealed by the patient's medical history. Psychogenic BOS is never accompanied by cyanosis or the need to use accessory respiratory muscles. Physical examination of patients reveals no pathology. Respiratory lability (switching from rapid, shallow to normal) may be noted, especially during conversation, switching attention, or movement. Sometimes in these situations, BOS disappears completely. Bronchodilator therapy is usually ineffective. In some cases, psychogenic BOS is treated with sedatives, psychotherapy, and antidepressants.

DIAGNOSIS AND TREATMENT OF BRONCHIAL ASTHMA IN OUTPATIENT PRACTICE

Bronchial asthma is a heterogeneous disease characterized by chronic airway inflammation, respiratory symptoms that vary in time and intensity, and occur along with variable airflow obstruction.

The diagnostic criteria for asthma are presented in table 5.

Table 5

Diagnostic criteria for asthma

Indicators	Decisions, definitions, criteria
1. History of variable respiratory symptoms	
Criteria for diagnosing asthma	Typically, there is more than one respiratory symptom (an isolated cough is rarely associated with asthma); symptoms vary in timing and intensity; symptoms often worsen at night or immediately after waking; symptoms are often triggered by exercise, laughter, allergens, and cold air; symptoms often appear or worsen during viral infections
2. Confirmed variable expiratory airflow limitation	
2.1. Registered airflow limitation	During the diagnostic process for low FEV1, it is necessary to confirm that the FEV1/FVC ratio is reduced (normally, it is > 0.75–0.80) one or more times
2.2. Registered increased variability in pulmonary function parameters* (based on one or more of the specified tests)	The greater the variability or the more frequently it is detected, the greater the confidence in the diagnosis. These tests can be repeated during symptom onset or early in the morning
2.2.1. Positive result of the airflow obstruction reversibility test using a BD	An increase in FEV1 of > 12 % and > 200 ml from baseline (an increase of > 15 % and > 400 ml is considered more reliable). Changes are measured after 10–15 minutes. After administration of 200–400 mcg salbutamol or an equivalent drug with comparable bronchodilator effect (the likelihood of obtaining a positive result is higher if the use of the bronchodilator is delayed before the test: SABA — for > 4 hours, LABA — for 24 hours, LAMA — for 36 hours)
2.2.2. Increased variability of Peak Expiratory Flow (PEF) measured twice daily for > 2 weeks	Average daily variability of PEF > 10 %
2.2.3. Significant improvement in pulmonary function parameters after 4 weeks of anti-inflammatory treatment	Increase in FEV1 by > 12 % and > 200 mL (or PEF by > 20 %) from baseline after 4 weeks of treatment in the absence of respiratory infections
2.2.4. Positive exercise stress test result	Decrease in FEV1 by > 10 % and > 200 mL from baseline
2.2.5. Positive bronchoprovocation test result	Decrease in FEV1 by > 20 % from baseline with standard doses of methacholine or histamine, or by > 15 % with standardized hyperventilation, hypertonic saline, or mannitol bronchoprovocation testing
2.2.6. Increased variability in pulmonary function parameters between visits (less reliable indicator)	Change in FEV1 by > 12 % and > 200 mL between visits in the absence of respiratory infections

* The daily variability of PEF is calculated based on PEF determined twice daily using the formula: the highest value for the day minus the lowest value for the day/the average of the highest and lowest values for the day.

Mandatory diagnostic procedures in an outpatient setting:

1. Medical history, assessment of the severity and duration of symptoms, identification of triggers (including occupational) — at each medical examination.

2. Physical examination, including determination of body mass index — at each medical examination.

3. Complete blood count — once at the initial medical examination, then as needed.

4. SpO_2 — at each medical examination.

5. Spirometry (lung function) with a bronchodilator — at initial diagnosis, repeat spirometry no later than 3 months after the start of therapy.

6. Chest X-ray — once.

7. ECG — once.

Additional diagnostic procedures:

1. Blood tests for magnesium, calcium, lactate dehydrogenase, and lipid profile.

2. Peak flowmetry — at least twice a day for at least 2 weeks, including an assessment of the response to bronchodilators, if used.

3. Skin tests or a blood test for antibody levels to plant, animal, and chemical antigens.

4. Asthma control test (ACT), asthma control questionnaire (ACQ).

5. Sputum eosinophil count.

6. Total IgE level in the blood.

7. Blood test (fetal bronchitis) and/or blood test (fetal bronchitis) with biopsy.

8. Blood gas analysis.

9. Chest CT scan.

10. Sputum analysis for microflora and drug sensitivity.

11. Mantoux test and/or Diaskin test.

12. Stool test for helminth eggs (three times).

13. Consultation with an immunologist-allergist; Consultation with an occupational pathologist if occupational asthma is suspected; consultation with a pulmonologist; consultations, according to medical indications, with a phthisiologist, gastroenterologist, otolaryngologist, psychotherapist, thoracic surgeon, and other medical specialists.

Asthma classification:

1. By clinical asthma form: allergic; non-allergic; mixed.

2. By severity: mild; moderate; severe.

Asthma severity is determined by the severity of symptoms, the presence (degree) of bronchoconstriction (FEV1 or PEF values), and bronchoreactivity (diurnal variability of FEV1 or PEF values). The severity of asthma in patients receiving treatment is determined by the amount of anti-inflammatory (basic) therapy received, ensuring controlled asthma.

3. By level of control: complete control (controlled); partial control (partially controlled); no control (uncontrolled). Asthma symptom control is assessed after 3 or more months of prescribed therapy or its adjustment. The ACT and ACQ can also be used to assess ongoing asthma control.

Examples of diagnosis formulations:

1. Asthma, allergic form, moderate, controlled. RF stage 0. Allergic rhinitis, perennial, mild. Sensitization to house dust allergens.

2. Asthma, non-allergic form, moderate, partially controlled. RF stage 1. Recurrent polypous rhinosinusitis. Intolerance to non-steroidal anti-inflammatory drugs.

3. Asthma, allergic form, moderate, mild exacerbation. RF stage 1. Allergic rhinitis, seasonal, severe. Sensitization to pollen (tree) allergens.

4. Asthma, non-allergic form, severe course; life-threatening exacerbation (asthmatic status). RF stage 2.

Treatment

Treatment for patients with asthma is carried out according to a 5-step algorithm. The amount of therapy is adjusted depending on the severity of the disease, the level of control, and the presence of risk factors for exacerbations. Each step includes treatment options that serve as alternatives to standard asthma therapy. The choice of therapy depends on the severity of the clinical manifestations of asthma.

Inhaled corticosteroids (ICS) are the mainstay of asthma therapy:

– beclomethasone (MDI — metered Dose Inhaler) 100 mcg/dose, 250 mcg/dose;

– budesonide (MDI) 100 mcg/dose, 200 mcg/dose;

– fluticasone (MDI) 50 mcg/dose, 125 mcg/dose, 250 mcg/dose;

– combinations of ICS with LABA — beclomethasone/formoterol MDI 100 mcg + 6 mcg/dose;

– budesonide/formoterol (Dry Powder Inhaler — DPI) 80 mcg + 4.5 mcg/dose, 160 mcg + 4.5 mcg/dose;

– fluticasone/salmeterol MDI 25 mcg + 50 mcg/dose, 25 mcg + 125 mcg/dose, 25 mcg + 250 mcg/dose;

– fluticasone/salmeterol DPI 50 mcg + 100 mcg/dose, 50 mcg + 250 mcg/dose, 50 mcg + 500 mcg/dose;

– fluticasone/vilanterol DPI 92 mcg + 22 mcg/dose, 184 mcg + 22 mcg/dose; Budesonide for inhalation suspension 0.25 mg/ml or 0.5 mg/ml in 2 ml nebules.

Other groups of medications used to treat asthma:

– leukotriene receptor antagonists (LTRAs): montelukast, film-coated tablets, 10 mg;

– extended-release methylxanthines — theophylline, extended-release capsules 100 mg, 200 mg, 300 mg, 350 mg;

– short-acting β_2-agonists (SABA), short-acting muscarinic antagonists (SAMA), or combinations for situational therapy: salbutamol inhaler 100 mcg/dose, fenoterol inhaler 100 mcg/dose, fixed-dose combinations of SABA/anticholinergics: fenoterol/ipratropium bromide inhaler 50 mcg + 20 mcg/dose, fenoterol/ipratropium bromide inhalation solution 0.5 mg + 0.25 mg/1 ml (for nebulizer therapy);

– long-acting muscarinic antagonist (LAMA) — tiotropium bromide inhalation solution 2.5 mcg/dose;

– omalizumab is a recombinant DNA-derived humanized IgG1 monoclonal antibody that can be used to treat severe persistent allergic asthma and nasal polyps (usually dose is 125 mg/mL);

– oral CS — methylprednisolone tablets 4 mg and 16 mg, prednisolone tablets 5 mg.

Step-by-step asthma therapy:

I. Step 1 (mild asthma):

1. Option 1: Low-dose ICS combination with formoterol as needed.
2. Option 2: Low-dose ICS prescribed with each SABA dose as needed.

II. Step 2:

Regular low-dose ICS. SABA is used to relieve symptoms. An additional dose of ICS is prescribed with each SABA dose. A possible background therapy option is daily LTRAs in combination with ASIT medications.

III. Step 3 (moderate asthma):

1. Option 1: Low-dose ICS/formoterol as background therapy and as needed with MART therapy (Maintenance and Reliever Therapy). MART therapy is only possible with fixed-dose combinations containing formoterol, a LABA with a rapid onset of bronchodilatory effect. The MART regimen with ICS/formoterol is the first choice for patients who require asthma treatment equivalent to Step 3 or 4 therapy and who have had more than one exacerbation in the past year.

2. Option 2: Regular use of low-dose ICS/LABA and SABA (or a SABA/LAMA combination) as needed. A possible background therapy option is daily use of medium-dose ICS and LTRAs or medium-dose ICS and extended-release theophylline in addition to ASIT. The daily theophylline dose is determined individually. The maximum dose that can be used without monitoring plasma theophylline concentrations is 13 mg/kg/day, but not more than 900 mg/day.

IV. Step 4 of Therapy (Severe Asthma):

1. Option 1: ICS/formoterol at medium doses in the MART regimen.

2. Option 2: ICS/LABA and SABA at medium or high doses (or a SABA/LAMA combination) as needed. If the response is insufficient, a LAMA, LTRAs, or extended-release theophylline can be added to the therapy. Switching to background

therapy with high-dose ICS is possible. High-dose ICS can be administered using a MDI with a spacer (beclomethasone, budesonide, fluticasone) or via a nebulizer (budesonide inhalation suspension 0.25 mg/ml or 0.5 mg/ml in 2 ml nebules, fluticasone inhalation suspension 0.25 mg/ml or 1 mg/ml in 2 ml nebules).

V. Step 5 of therapy (severe asthma):

1. Option 1: Add a LAMA to MART therapy with medium-dose ICS/ formoterol, assess asthma severity and progression with the possibility of initiating targeted therapy with a biologic agent, and consider prescribing high-dose ICS/ formoterol.

2. Option 2: Add a LAMA to medium-dose ICS/LABA, assess asthma severity and progression with the possibility of initiating targeted therapy with a biologic agent, and consider prescribing high-dose ICS/LABA.

In Step 5 of therapy, if asthma is not adequately controlled, low-dose oral glucocorticosteroids may be added to existing background therapy, ensuring monitoring for potential side effects. Azithromycin may also be added based on the physician consultation.

Mild asthma exacerbations are treated on an outpatient basis and requires escalating therapy. Symptomatic bronchodilators for the relief of exacerbation symptoms include SABAs in metered-dose inhalers (salbutamol, fenoterol) or SABA/ Anticholinergics combinations (fenoterol/ipratropium bromide). For mild exacerbations, repeat inhalations are used (1–2 puffs every 20 minutes for the first hour). SABA delivery via a metered-dose inhaler with a spacer results in similar improvements in lung function as delivery via a nebulizer.

Patients who do not respond to treatment within 24 hours or whose condition continues to deteriorate require hospitalization.

Emergency care for asthma attacks. In the case of asthma, bronchodilator therapy alone is not recommended for the relief of attacks of dyspnea (asphyxia) for emergency medical care (situational therapy). When asthma symptoms appear, combination drugs of ICS with rapid-onset bronchodilators are prescribed: ICS/salbutamol — beclomethasone/salbutamol MDI 50 mcg + 100 mcg/dose, 250 mcg + 100 mcg/dose, ICS/formoterol — beclomethasone/formoterol MDI 100 + 6 mcg/dose or budesonide/formoterol DPI 80 mcg + 4.5 mcg dose, 160 mcg + 4.5 mcg dose. The use of ICS/formoterol combinations as emergency medical care (situational therapy) is carried out on demand. In case of using SABA or S SABA/anticholinergics for emergency medical care after inhalation of fast-acting bronchodilators, additional use of a low dose of ICS is possible.

DIAGNOSIS AND TREATMENT OF COPD IN OUTPATIENT PRACTICE

COPD is a heterogeneous lung disease characterized by chronic respiratory symptoms (dyspnea, cough, sputum production) due to airway pathology (bronchitis) and/or alveolar pathology (emphysema), which cause persistent, often progressive bronchial obstruction.

The diagnostic criteria for COPD are presented in table 6.

Mandatory diagnostic tests in an outpatient setting are:

1. Anamnesis — one-time.
2. Physical examination with body mass index assessment — at each visit.
3. SpO_2 — at each medical examination.
4. Complete blood count — one-time, repeat if there are clinically significant changes in the initial analysis, then as medically indicated.
5. Biochemical blood test (testing levels of total protein, bilirubin and its fractions, urea, creatinine, alanine aminotransferase, aspartate aminotransferase, glucose, potassium, sodium, chloride, C-reactive protein) — one-time, repeat if there are clinically significant changes in the initial analysis, then as medically indicated.
6. Spirometry with a 400 mcg salbutamol bronchodilator test — at initial diagnosis, then at least once a year.
7. Chest X-ray — once a year.
8. ECG – once a year.

Table 6

Diagnostic criteria for COPD

Diagnostic criteria	Description
Presence of respiratory symptoms in history	Presence of risk factors in history; cough with or without sputum, dyspnea, reduced exercise tolerance
Confirmed persistent irreversible (partially reversible) bronchial obstruction	FEV1/FVC ratio 0.75–0.80 with or without reduced FEV1; increase in FEV1 less than 12 % or less than 200 ml from baseline during bronchodilator test. Results recorded 10–15 minutes after administration of 400 mcg salbutamol. Required interval between last bronchodilator use and test: SABA — > 4 hours, LABA — 24 hours, LAMA — 36 hours
Exclusion of other pathological conditions that may present with bronchial obstruction	Bronchial asthma, tumors or foreign bodies of the tracheobronchial tree, developmental anomalies, interstitial lung diseases with obstruction, severe heart failure, and others
Additional diagnostic methods	Chest X-ray, chest CT; determination of lung diffusion capacity

Additional diagnostic tests include:

1. Blood chemistry panel: calcium and lactate dehydrogenase levels; lipid profile.
2. Alpha-1-antitrypsin blood test.
3. SAT test.
4. Sputum eosinophil count.
5. Fiberoptic bronchoscopy and/or fiberoptic bronchoscopy with biopsy; bronchoalveolar lavage fluid analysis.
6. Blood gas analysis.
7. Sputum microflora analysis and drug sensitivity test.
8. Mantoux test and/or Diaskin test.
9. CT scan of the chest.
10. Chest X-ray.
11. Echocardiogram (ECHO).
12. Consultation with an occupational therapist, pulmonologist, tuberculosis specialist, cardiologist, endocrinologist, otolaryngologist, psychotherapist, thoracic surgeon, and other medical specialists as indicated.

Classification

COPD classification:

1. By phenotypes according to clinical features of the phenotypes: bronchitis phenotype, emphysematous phenotype, mixed phenotype.
2. By the severity of broncho-obstruction: grade I; grade II; grade III; grade IV.
3. By the severity of symptoms according to the modified dyspnea rating scale (mMRC): severe symptoms (grade >2 according to the modified dyspnea rating scale (mMRC)); Mild symptoms (modified dyspnea rating scale (mMRC) < grade 2).
4. By severity of stable COPD (based on dyspnea severity according to the modified dyspnea rating scale (mMRC) and the frequency of COPD exacerbations): mild (mMRC) < grade 2 and less than 2 exacerbations per year; moderate (mMRC) > grade 2 and 2 or more COPD exacerbations or 1 COPD exacerbation requiring hospitalization); severe (mMRC) > grade 2, clinically clearly expressed COPD phenotypes, clinical signs of respiratory failure (RF), frequent COPD exacerbations and hospitalizations, chronic pulmonary heart disease).
5. By frequency of COPD exacerbations: rare (0-1 time in the last year); frequent (2 or more times in the last year, including those requiring hospitalization).
6. By COPD phase: remission; exacerbation.

Examples of diagnosis formulations:

1. COPD, emphysematous phenotype, severe, broncho-obstruction grade 2, remission, RF grade 1.

2. COPD, mixed phenotype, moderate, broncho-obstruction grade 2, moderate exacerbation from (specify date), RF grade 2.

3. COPD, bronchitis phenotype, broncho-obstruction grade 3, remission RF grade 2. Obesity, grade 2 (body mass index 36 kg/m2). Obstructive sleep apnea syndrome.

Treatment

Treatment of COPD includes non-pharmacological methods, pharmacotherapy in stable disease, treatment of exacerbations, and respiratory support in chronic respiratory failure. Treatment volume is adjusted depending on obstruction degree, disease severity, blood eosinophil level, presence of infectious exacerbation, and hypoxia:

1. Smoking cessation is mandatory.

2. Occupational exposure (dust, irritants) → consultation with occupational physician.

3. Initial pharmacotherapy is based on comprehensive disease assessment. Adjust therapy after 1–3 months based on dyspnea and exacerbations, considering COPD phenotype.

The basis of COPD therapy is bronchodilator drugs:

I. SABA, SAMA or combinations for situational therapy:

1. Salbutamol (MDI 100 mcg/dose; solution 1 mg/ml for nebulizer).

2. Fenoterol (MDI 100 mcg/dose; solution 1 mg/ml for nebulizer).

3. Ipratropium bromide (MDI 20–40 mcg/dose; solution 0.25 mg/ml for nebulizer)

II. Fixed SABA/SAMA combinations: fenoterol + ipratropium bromide (MDI 50 mcg + 20 mcg/dose; solution 0.5 mg + 0.25 mg/ml for nebulizer).

III. LABA, LAMA or combinations for maintenance therapy:

1. Tiotropium bromide, solution for inhalation in a metered-dose inhaler — 2.5 mcg/dose, 2 inhalations once daily.

2. Formoterol, metered-dose powder for inhalation (hereinafter referred to as DPI) — 12 mcg/dose, 1–2 inhalations twice daily.

3. Fixed-dose combination of olodaterol + tiotropium bromide, solution for inhalation in a metered-dose inhaler — 2.5 mcg/dose + 2.5 mcg/dose, 2 inhalations once daily.

4. Fixed-dose combination of indacaterol + glycopyrronium bromide, powder for inhalation in capsules – 110 mcg/dose + 50 mcg/dose, 1 inhalation once daily.

IV. Other groups of drugs used to treat COPD:

1. ICS and systemic corticosteroid (for medications and dosages, see the asthma treatment section).

2. Slow-release methylxanthines.

3. Phosphodiesterase inhibitors: roflumilast, 0.5 mg tablets, 1 tablet once daily.

4. For mucolytic therapy in patients with COPD with the bronchitis phenotype and frequent exacerbations of COPD, especially if ICS therapy is not being administered, one of the following medications is prescribed:

– N-acetylcysteine, soluble powder 100 mg, 200 mg — orally 200 mg 3 times a day or soluble prolonged-release tablets 600 mg — orally 600 mg once a day (or 1200 mg per day);

– Ambroxol, 30 mg tablets — 30 mg orally 3 times daily;

– Carbocisteine, 375 mg capsules — 2 capsules orally 3 times daily or 250 mg/5 ml syrup — 3 teaspoons orally 3 times daily;

– Erdosteine, 300 mg capsules — 1 capsule orally 2–3 times daily.

If chronic respiratory failure develops, long-term oxygen therapy is administered.

Stepwise Therapy for Patients with COPD:

1. Treatment of patients with infrequent COPD exacerbations and mild symptoms begins with long-acting bronchodilators: a long-acting bronchodilator or a long-acting LABA if bronchoconstriction is partially reversible. If dyspnea progresses, monotherapy with a long-acting bronchodilator is replaced with a combination of a long-acting bronchodilator and a long-acting bronchodilator.

2. Patients with frequent COPD exacerbations and/or severe symptoms, or if symptoms persist (shortness of breath and decreased exercise tolerance) despite monotherapy with a single long-acting bronchodilator, are prescribed initial combination therapy with a long-acting bronchodilator and a long-acting bronchodilator. For recurrent COPD exacerbations, regardless of the severity of dyspnea, therapy with the long-acting bronchodilator and a long-acting bronchodilator combination is continued.

3. Triple initial therapy (LAMA + LABA + ICS) for COPD is prescribed to: patients with frequent exacerbations of COPD or one or more exacerbations of COPD requiring hospitalization, if the peripheral blood eosinophil count is > 300 cells/μL; patients with exacerbations during LAMA or LABA monotherapy, if the peripheral blood eosinophil count is > 300 cells/μL; patients with two or more mild or moderate exacerbations of COPD or one or more severe exacerbations of COPD during LAMA or LABA treatment, if the peripheral blood eosinophil count is > 100 cells/μL; in cases of combined COPD and bronchial asthma.

4. If triple combination therapy is insufficiently effective, prolonged-release methylxanthines and mucolytics may be additionally prescribed. For smokers (former smokers) with insufficient response to initial therapy, or for patients with COPD with bronchiectasis and COPD exacerbations two or more times per year, azithromycin 500 mg tablets may be added to the treatment plan, based on the

decision of a medical consultation. One tablet every third day may be taken until the exacerbation frequency is reduced to less than two times per year.

Medical indications for hospitalization of patients with COPD exacerbations include:

– a significant increase in symptom intensity and/or the appearance of new symptoms (e. g., severe shortness of breath);

– respiratory rate > 24 breaths per minute;

– heart rate > 100 beats per minute;

– SpO_2 3 % of baseline (if known);

– arterial oxygen tension (hereinafter referred to as PaO_2) 45 mmHg (if measurable);

– inability to manage a COPD exacerbation in an outpatient setting.

Antibacterial therapy is prescribed for patients with a COPD exacerbation in the presence of purulent sputum and at least two of the following signs:

– – increased dyspnea;

– – increased sputum volume;

– – increased sputum purulent content.

Antibacterial therapy is also indicated for patients with a severe COPD exacerbation requiring invasive or non-invasive ventilation. Bronchodilator and glucocorticosteroid therapy are also intensified during a COPD exacerbation.

Work capacity assessment. Temporary disability occurs during a COPD exacerbation and depends on its severity; the approximate duration is 9–16 days. Patients with severe course, frequent exacerbations and grade 3 broncho-obstruction are referred to the medical expert commission.

PATIENT MEDICAL MONITORING AND PREVENTION OF ASTHMA AND COPD

Long-term medical monitoring of patients with asthma and COPD is performed in an outpatient setting by a primary care physician and/or general practitioner. Monitoring is based on the type, severity, and level of asthma control. Spirometry monitoring (spirometry with bronchodilator testing) is performed annually for controlled asthma, every 6 months for partially controlled asthma, and every 3 months for severe, uncontrolled asthma. In the absence of control and/or the presence of risk factors for exacerbations, an increase in the volume of therapy is indicated. A decrease in the volume of therapy is indicated when control is achieved and maintained for >3 months and there are no risk factors, in order to establish the minimum therapy volume sufficient to maintain control.

Preventive measures: cessation of smoking (both active and passive); regular physical activity, breathing exercises; weight loss in obese patients; Avoid stressful

situations; seasonal influenza vaccination; balanced diet; avoid medications that may worsen asthma.

Medical monitoring of patients with COPD is also carried out considering the form, severity, and degree of respiratory failure. Spirometry (spirometry with bronchodilator testing) is monitored annually during a stable course; in case of progression, as medically indicated.

Preventive measures for COPD include: cessation of smoking (both active and passive); balanced diet; weight loss in obese patients; weight correction in underweight patients; regular physical activity, breathing exercises; seasonal influenza vaccination; pneumococcal vaccination; adherence to recommendations for rational employment.

SELF-ASSESSMENT OF TOPIC UNDERSTANDING

Tests

1. The main risk factors for COPD are:

a) obesity;
b) passive smoking;
c) occupational hazards (dust, chemicals);
d) alcohol abuse;
e) active smoking.

2. A bronchodilator test in an adult patient is positive with an increase in FEV1 of:

a) 10 %;
b) 12 %;
c) 13 %;
d) 15 %;
e) 20 %;

3. The main cause of status asthmaticus is: (термина не было в тексте, надо менять этот вопрос или перефразировать)

a) massive exposure to allergens;
b) irregular use of bronchodilators;
c) atypical course of the disease;
d) excessive, uncontrolled use of β_2-agonists;
e) the presence of a viral infection.

4. Which group of drugs forms the basis of basic therapy for COPD:

a) ICS;

b) mucolytics;
c) antibiotics;
d) bronchodilators;
e) NSAIDs.

5. Name the conditions that are accompanied by predominantly expiratory dyspnea:

a) asthma;
b) pulmonary embolism;
c) COPD;
d) lung cancer.

6. The main risk factors for asthma are:

a) hypodynamia;
b) exposure to allergens;
c) respiratory infections;
d) genetic predisposition;
e) alcohol abuse.

7. In which conditions accompanied by expiratory dyspnea is fever observed:

a) lung cancer;
b) asthma;
c) pneumonia;
d) foreign body in the respiratory tract;
e) COPD.

8. Which group of drugs forms the basis of basic asthma therapy:

a) methylxanthines;
b) mucolytics;
c) ICS;
d) antibiotics;
e) beta-agonists?

Answers: **1** — b, c, e; **2** — b; **3** — a, d, e; **4** — d; **5** — a, c; **6** — b, c, d; **7** — a, c; **8** — c.

Cases

Case 1. Patient S. presented with complaints of asthma attacks, primarily at night, accompanied by a cough producing viscous, stringy, mucous sputum. The symptoms had been present for approximately six months and had recently become more frequent, prompting an ambulance call. The attacks subsided, and an outpatient examination was recommended. Patient S. is a nonsmoker. He works in an office and has pets.

On examination, the patient's condition was satisfactory, BMI 28 kg/m², diffuse cyanosis, and jugular vein distension were observed. Topographic percussion revealed the height of the anterior apex of the lungs to be five and a half centimeters from the superior border of the clavicle, and posteriorly to the level of the spinous process of the sixth cervical vertebra. The lower borders of the lungs were lowered by one rib. Respiratory rate was 18 breaths per minute. Auscultation revealed isolated dry rales. Heart sounds are clear and rhythmic. Heart rate is 90 beats per minute, blood pressure is 110/70 mmHg. The abdomen is soft and painless on palpation. The liver is at the costal margin and is non-tender. The spleen is not enlarged. Formed stools are passed once daily, urine output is maintained, and urination is normal.

Questions:

1. Formulate a diagnosis.
2. Develop an outpatient examination plan.
3. Develop a treatment plan for the patient.

Case 2. Patient V., 54, a mechanic, sought medical attention due to increasing shortness of breath and a non-productive cough. He has a history of long-term smoking, with a smoking index of 20 pack-years. The patient's symptoms have persisted for approximately two years, and their condition has recently worsened. Exercise tolerance has decreased. This is his first visit.

On examination, his condition is satisfactory, his BMI is 22 kg/m², and diffuse cyanosis is observed. Topographic percussion reveals the lower lung borders to be lowered by one edge. The mobility of the lower lung edge is limited, and comparative percussion reveals a hyperboxed (Hyperresonance этот термин нужен) pulmonary sound. Respiratory rate is 21 beats per minute. Auscultation reveals dry, wheezing sounds. The heart sounds are muffled and rhythmic. Heart rate is 95 beats per minute, blood pressure is 115/70 mmHg. The abdomen is soft and painless on palpation. The liver is painless at the costal margin. The spleen is not enlarged. Formed stool is passed once daily, urine output is normal, and urination is normal.

Questions:

1. Formulate a diagnosis.
2. Develop an outpatient examination plan.
3. Develop a treatment plan for the patient.

Case 3. Patient V., 67, was brought to the clinic by an ambulance crew complaining of a hacking cough, hemoptysis, and shortness of breath with minor exertion. Weight loss, weakness, and episodes of low-grade fever are also noted. Symptoms have been increasing over three months. He is a smoker. His smoking index is 24 pack-years.

On examination: the patient is in moderate condition, BMI 18 kg/m², pale skin. Temperature 37.5 °C. Topographic percussion reveals normal lung boundaries,

and comparative percussion reveals normal pulmonary sounds. Respiratory rate is 24 beats per minute. Auscultation reveals vesicular breath sounds, no wheezing. Heart sounds are muffled and rhythmic. Heart rate is 92 beats per minute, blood pressure is 105/70 mmHg. The abdomen is soft and painless on palpation. The liver is tender and at the costal margin. The spleen is not enlarged. Formed stools are passed once daily, urine output is maintained, and urination is normal.

Questions:

1. Formulate a diagnosis.
2. Develop an examination plan.
3. Develop a treatment plan for the patient.

Case 4. Patient A., 55, presented after two episodes of hemoptysis. She also noted increasing shortness of breath over the past few days. She has smoked 15 cigarettes a day for many years, and her gynecologist prescribed hormone replacement therapy for her menopause.

On examination, her condition is satisfactory, BMI is 28 kg/m², and her skin is normal in color. Temperature is 36.5 °C. Topographic percussion reveals normal lung borders, while comparative percussion reveals localized dullness below the angle of the scapula. Respiratory rate is 22 bpm. Auscultation reveals weakened breath sounds over the dulled area, with fine moist rales. Heart sounds are muffled and rhythmic. Heart rate is 85 bpm, blood pressure is 115/70 mmHg. The abdomen is soft and painless on palpation. The liver is at the costal margin and is painless. The spleen is not enlarged. Formed stools are passed once daily, urine output is normal, and urination is not impaired.

Questions:

1. Formulate a diagnosis.
2. Develop an outpatient examination plan.
3. Develop a treatment plan for the patient.

Correct answers for self-assessment (cases)

Case 1:

1. Bronchial asthma, mild allergic form, non-severe exacerbation. Stage 1 respiratory failure.

2. Complete blood count, urine analysis, SpO_2 — at each medical examination, spirometry (pulmonary function) with bronchodilator test, chest X-ray, ECG. Additionally — skin tests or testing for antibody levels to plant, animal, and chemical antigens in the blood.

3. Outpatient treatment. Regular administration of low-dose ICS. For symptom relief, a SABA is used. An additional dose of ICS is used with each SABA use. ASIT may be added to the drug therapy.

Case 2:

1. COPD, emphysematous phenotype, severe, broncho-obstruction stage II, remission, RF stage I.

2. CBC, urine analysis, biochemical blood test, SpO_2 — at each medical examination, spirometry (pulmonary function) with bronchodilator test, chest X-ray, ECG.

3. Outpatient treatment. The patient is advised to quit smoking. Triple initial therapy (LAMA + LABA + ICS).

Case 3:

1. Preliminary diagnosis: lung cancer.

2. CBC, urine analysis, biochemical blood test, SpO_2, spirometry, chest X-ray, ECG, oncologist consultation, FBS?, CT scan.

3. Hospitalization in a specialized hospital. The patient is advised to quit smoking. The further treatment plan is determined by the oncologist based on the stage of the disease and the clinical group.

Case 4:

1. Preliminary diagnosis: pulmonary embolism.

2. CBC, SpO_2, chest X-ray, ECG.

3. Emergency hospitalization. Thrombolytic therapy.

LIST OF REFERENCES

1. *Поликлиническая* терапия : учеб. / М. В. Зюзенков [и др.] ; под ред. М. В. Зюзнекова. – 3-е изд., испр. – Минск : Выш. шк., 2022. – 623 с.

2. *Хили, П. М.* Дифференциальный диагноз внутренних болезней: алгоритмический подход / П. М. Хили, Э. Д. Джекобсон. – М. : БИНОМ, 2021. – 280 с.

3. *Диагностика* и лечение пациентов (взрослое население) с хронической обструктивной болезнью легких : клинический протокол : постановление М-ва здравоохранения Респ. Беларусь от 13 мая 2025 г. № 31. – URL: https://minzdrav.gov.by/upload/icfiles/постановление_МЗ_2024_31.pdf (дата обращения: 10.10.2025).

4. *Диагностика* и лечение пациентов (взрослое население) с бронхиальной астмой : клинический протокол : постановление М-ва здравоохранения Респ. Беларусь от 30 апр. 2024 г. № 84. – URL: https://minzdrav.gov.by/upload/icfiles/постановление_МЗ_2025_84.pdf (дата обращения: 10.10.2025).

5. *Трисветова, Е. Л.* Пульмонология : учеб. пособие / Е. Л. Трисветова. Минск : Новое знание, 2022. – 389 с.

6. *Чучалин, А. Г.* Кашель / А. Г. Чучалин, В. Н. Абросимов. – М. : ГЭОТАР-Медиа, 2017. – 152 с.

7. *Самаль, Т. Н.* Дифференциальная диагностика острых заболеваний органов дыхания и бронхообструктивного синдрома у детей : учеб.-метод. пособие / Т. Н. Самаль, И. Э. Бовбель. – Минск : БГМУ, 2023. – 32 с.

8. Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: 2026 report. – URL: https://goldcopd.org/2026-gold-report-and-pocket-guide (date of access : 24.11.2025).

CONTENTS

Abbreviations list 3
Motivational characteristics of the topic 4
General provisions 5
Diagnostics and stages of diagnostic search 7
Diagnosis and treatment of bronchial asthma in outpatient practice 16
    Treatment 19
Diagnosis and treatment of COPD in outpatient practice 22
    Classification 23
    Treatment 24
Patient medical monitoring and prevention of asthma and COPD 26
Self-assessment of topic understanding 27
    Tests 27
    Cases 28
    Correct answers for self-assessment (cases) 30
List of references 32

Учебное издание

Алексеева Елена Сергеевна
Ерёмина Наталья Михайловна
Зюзенков Михаил Васильевич
Каразей Елена Александровна

ДИФФЕРЕНЦИАЛЬНАЯ ДИАГНОСТИКА БРОНХООБСТРУКТИВНОГО СИНДРОМА. ХРОНИЧЕСКАЯ ОБСТРУКТИВНАЯ БОЛЕЗНЬ ЛЕГКИХ И БРОНХИАЛЬНАЯ АСТМА

DIFFERENTIAL DIAGNOSIS OF BRONCHIAL OBSTRUCTIVE SYNDROME. CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND BRONCHIAL ASTHMA

Учебно-методическое пособие

На английском языке

Ответственная за выпуск Е. В. Рылатко
Переводчик Е. С. Алексеева
Компьютерная вёрстка А. В. Янушкевич

Подписано в печать 28.05.26. Формат 60×84/16. Бумага писчая «Снегурочка».
Ризография. Гарнитура «Times».
Усл. печ. л. 2,09. Уч.-изд. л. 1,55. Тираж 40 экз. Заказ 258.

Издатель и полиграфическое исполнение: учреждение образования
«Белорусский государственный медицинский университет».
Свидетельство о государственной регистрации издателя, изготовителя,
распространителя печатных изданий № 1/187 от 24.11.2023.
Ул. Ленинградская, 6, 220006, Минск.