

The background features abstract geometric shapes in various shades of green and a dark blue-grey. On the left, there's a yellow circle partially cut off. On the right, there are several overlapping green shapes, including a large dark blue-grey semi-circle. At the bottom, there's a solid green horizontal bar with a dark blue-grey circle on the left and a rounded green shape on the right.

Diagnostics of allergic diseases.

As a result of the immune system being affected by various pathogenic factors, the changes related to the pathologies of the immunocompetent tissue, that is, immunopathological processes - Immune deficiency syndromes (defects or deviations from the norm in one or more indicators of the immune response mechanisms), allergic reactions and autoimmune processes (pathologies that occur as a result of excessive activation of immune mechanisms).

Allergy - is a pathological form of immunological reactivity, which is a response accompanied by tissue damage as a result of increased and distorted sensitivity of the body to substances of antigenic nature.

Clinically significant appearances for the diagnosis of allergy:

- *Skin: itching, rash, swelling, erythema;*
- *Eye: itching, watering, swelling, hyperemia, crusts;*
- *Nose: rhinorrhea, itching, congestion (congestion), sneezing;*
- *Lungs: wheezing, coughing, chest tightness, shortness of breath (dyspnea);*
- *Gastrointestinal tract: nausea, vomiting, bloating, diarrhea;*
- *Cardiovascular system: anaphylaxis, fainting, weakness.*
- *Correlation of anamnesis with the etiological role of known allergens (household, dust, epidermal, insect, food, etc.). As a result of repeated exposure to the allergen, the allergy symptoms are aggravated and when the allergen is removed, the allergy symptoms disappear or their intensity is reduced; Pathogenetically based treatment should alleviate the symptoms of the disease.*



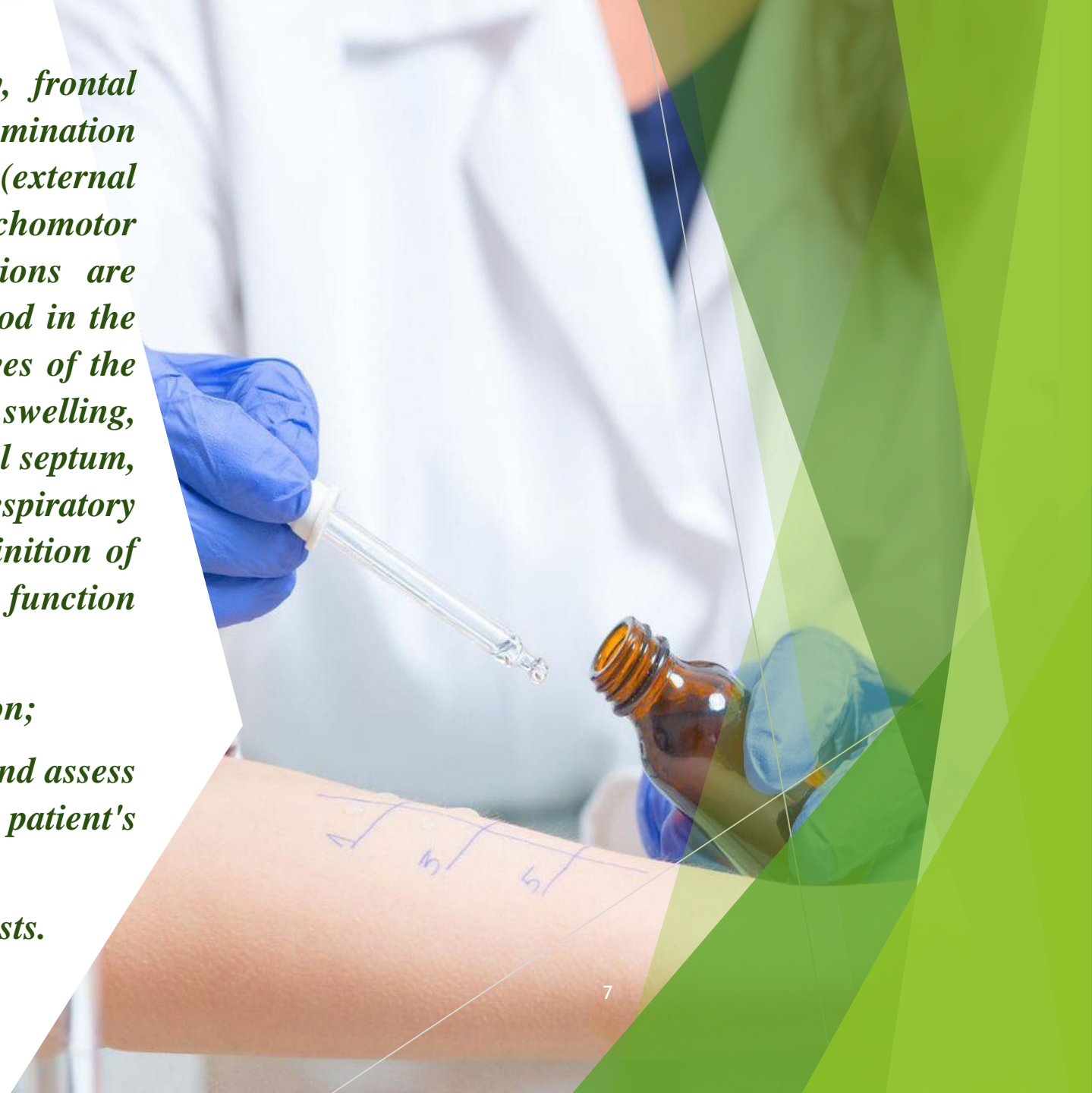
Clinical-laboratory methods:

- *clinical analysis of peripheral blood;*
- *biochemical blood test (the list of studies is determined by the doctor depending on the presence of accompanying diseases); clinical analysis of urine;*
- *from the nasal cavity, conjunctival mucus, etc. cytological examination of secretions, smears;*
- *general and bacteriological analysis of sputum and other secretions from mucous membranes and skin according to the instructions;*
- *blood gas analysis, rheological tests, hormone testing, virological studies, etc. inspections according to instructions;*
- *Coprogram;*
- *Parasitological examination.*



- *In addition, instrumental (frontal rhinoscopy, frontal rhinomanometry, endoscopic examination (bronchoscopy), etc.), functional examinations (external respiratory function study), examination, bronchomotor tests, specific methods of allergic examinations are checked. Anterior rhinoscopy is the main method in the diagnosis of diseases and non-allergic derivatives of the nasal mucosa. The color of the nasal mucus, swelling, the presence of polyps, the curvature of the nasal septum, etc. allows to work. Study of external respiratory function in bronchial asthma diseases and definition of bronchial asthma. External respiratory function examination:*

- *detection and assessment of bronchial obstruction;*
- *check the effectiveness of anti-asthma therapy and assess the dynamics of the bronchial asthma patient's condition;*
- *allows evaluating the results of bronchomotor tests.*





Bronchomotor tests:

- ▶ ***Tests can be performed only by a doctor who owns the test methodology. When conducting tests, it is required to strictly follow the technique. When evaluating the results of bronchomotor tests, not only indicators of forced exhalation volume in the first second, but also other indicators of external respiratory function should be taken into account.***



Skin tests:

- ▶ *Skin tests are a standard method of allergy diagnosis and are performed during remission of allergic diseases. Depending on the method of application of the allergen, different methods of skin tests are used: Prick tests, scarification, intradermal, drop, application, etc. Standard allergens are used for skin testing. Positive skin tests indicate the presence of IgE antibodies to a specific allergen, but are not considered indicative of clinical sensitization. The relationship between the clinical picture and the results of skin tests is imperative.*



- ▶ *In general, allergy skin tests are reliable for diagnosing allergies to airborne substances, such as pollen, pet dander and dust mites. Skin testing may help diagnose food allergies. But because food allergies can be complex, you mairing allergy skin tests, your skin is exposed to suspected allergy-causing substances (allergens) and is then observed for signs of an allergic reaction.*
- ▶ *Along with your medical history, allergy tests may be able to confirm whether a particular substance you touch, breathe or eat is causing symptoms.*
- ▶ *Information from allergy tests may help your doctor develop an allergy treatment plan that includes allergen avoidance, medications or allergy shots (immunotherapy).*

- ▶ *Allergy skin tests are widely used to help diagnose allergic conditions, including:*
 - ▶ *Hay fever (allergic rhinitis)*
 - ▶ *Allergic asthma*
 - ▶ *Dermatitis (eczema)*
 - ▶ *Food allergies*
 - ▶ *Penicillin allergy*



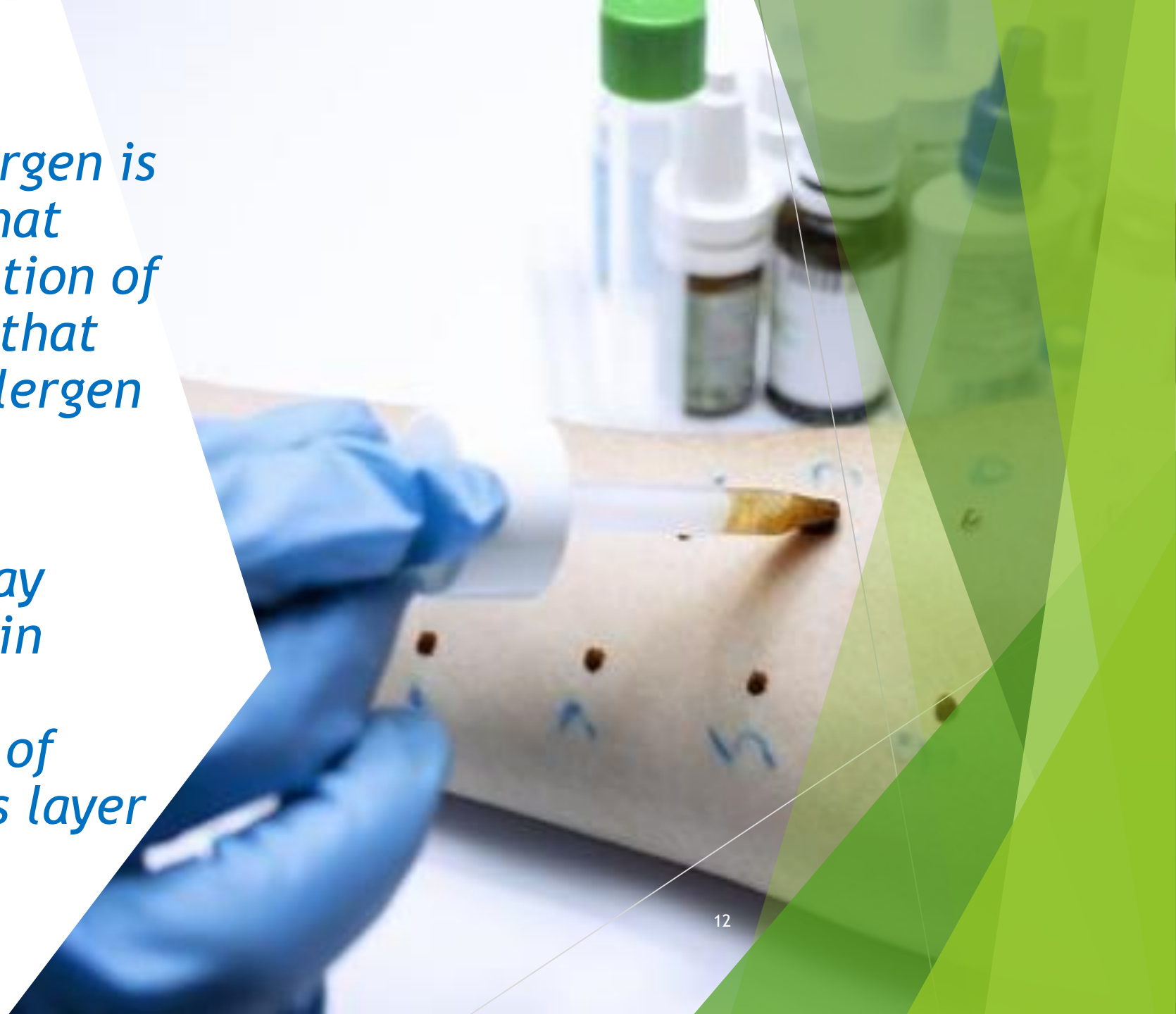


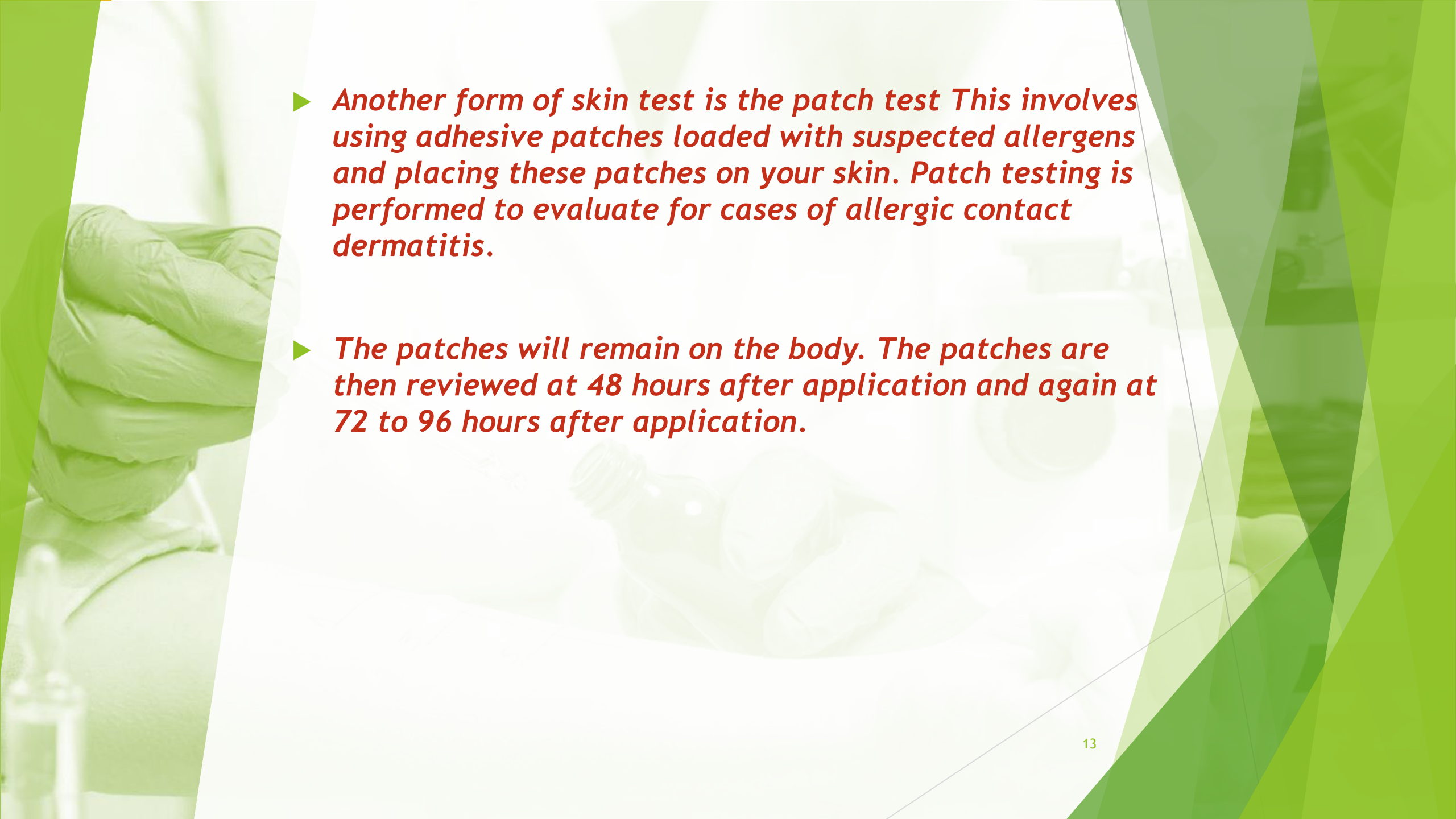
- ▶ Skin prick testing will be done at doctor's office. For this test, a doctor or nurse will lightly prick the skin on patients back or arm with a comb-like tool. Then, they will add a small amount of a suspected allergen over the pricked area.
- ▶ You'll know, and feel, the results more quickly than with a blood test. If the doctor sees swelling or if the area begins to itch, that's a positive reaction. This means you're more likely to be allergic to that particular allergen. A positive reaction could happen right away, or it could take 15 to 20 minutes. If there's no reaction, it's unlikely you're allergic to the substance.
- ▶ Skin prick testing is more sensitive than blood testing. It's also less expensive. However, there's more risk. Though rare, it's possible to have a serious reaction. For this reason, a doctor may avoid skin testing if risk for anaphylaxis or a severe reaction is high. Doctors and staff should be trained to deal with any reaction that might occur.
- ▶ *If you're getting a skin prick test, you'll be asked to stop taking antihistamine medication a few days before the test.*


Scratch test:

During this test, an allergen is placed in liquid, then that liquid is placed on a section of skin with a special tool that lightly punctures the allergen into skin's surface.

If the scratch test is inconclusive, doctors may order an intradermal skin test. This test requires injecting a tiny amount of allergen into the dermis layer of skin.



- 
- A hand in a white glove is shown holding a small vial in a laboratory setting. The background is a blurred image of laboratory equipment, including a pipette and a beaker. The overall color scheme is light green and white.
- ▶ ***Another form of skin test is the patch test. This involves using adhesive patches loaded with suspected allergens and placing these patches on your skin. Patch testing is performed to evaluate for cases of allergic contact dermatitis.***
 - ▶ ***The patches will remain on the body. The patches are then reviewed at 48 hours after application and again at 72 to 96 hours after application.***



One of the specific methods of allergy tests is the method of in vitro laboratory tests. Among the in vitro tests, various methods are currently used for the diagnosis of allergic diseases. Based on the results of the total IgE study, the diagnosis of allergy is not made, but this information is useful for the final clinical outcome. In addition to allergic diseases, the level of IgE is high in many other diseases - hepatitis associated with viral, drug, radiation and other factors; parasitic diseases; immune deficiency; genetic factors (high and low IgE levels), etc. may increase.

Immunoglobulins - Test

HE
(IGE) B KPOBI

2/22/2023

Allergic diseases.



Atopic dermatitis.

- ▶ *Atopic dermatitis - is a chronic relapsing allergic skin disease that usually occurs in early childhood in people with a hereditary predisposition to atopic diseases and is characterized by itching and hypersensitivity reactions on the skin to both allergens and non-specific irritants.*



Etiology and pathogenesis.



- ▶ The main factors that cause atopic dermatitis are household, epidermal, food allergens, fungi and pollen. In most people, the disease develops before the age of 5 years, in many by the age of 1 year; but atopic dermatitis can start even in adulthood. The development of atopic dermatitis is caused by genetic factors, dysfunction of the epidermal barrier, immunological mechanisms, and environmental factors (irritants). Genes encoding epidermal and immunological proteins are involved in the development of atopic dermatitis. In recent years, a mutation of the filaggrin gene has been found in many patients with atopic dermatitis, which has been shown to cause disruption of the epidermal barrier function. **Filaggrin** is a component of the cell membrane, which is formed as a result of the differentiation of keratinocytes. These recent molecular discoveries provide new insights into the relationship between atopic dermatitis and skin inflammation, T-cell sensitization, and immediate hypersensitivity allergic conditions such as asthma and allergic rhinitis. Dysfunction of the epidermal skin as a result of mutations in the filaggrin gene explains the skin irritations and xerosis (the skin peels off violently, small cracks appear, the skin surface becomes rough) during atopic dermatitis. However, skin inflammation is a T-cell-mediated hypersensitivity reaction that is typical of slow-onset hypersensitivity reactions. This type of hypersensitivity interferes with the activity of antibacterial peptides (for example - beta-defensins), which in turn increases the sensitivity of patients with atopic dermatitis to bacterial and viral skin infections.

Classification:

- ▶ *Age periods of the disease:*
 - *I – baby (up to 2 years);*
 - *II – child (from 2 to 13 years old);*
 - *III – teenagers and adults (over 13 years old).*
- ▶ *Stages of the disease:*
 - *Aggravation stages: stage of severe clinical appearances, stage of moderate clinical appearances;*
 - *Stages of remission: incomplete, complete remission.*
- ▶ *Dissemination of the process: limited localized, general, diffuse.*
- ▶ *Degree of severity of the process: light, medium, heavy.*
- ▶ *When determining the severity of the disease, the duration and frequency of Aggravations, the duration of remissions, the distribution and morphological characteristics of the skin process, the intensity of skin itching, sleep disorders, and the result of the treatment are taken into account.*

Diagnostics:

- ▶ *Absolute clinical signs:*
 - *Itching of the skin;*
 - *Characteristic morphological characteristics of skin lesions depending on age*
 - *Inflammation (acute, chronic);*
- ▶ *The most common symptoms are:*
 - *The onset of the disease is in early childhood;*
 - *Atopy, along with allergic diseases, atopic anamnesis in other family members;*
 - *Dry skin.*
- ▶ *Other signs (important to confirm the diagnosis, but not mandatory and specific to make the diagnosis):*
 - *Atypical vascular reactions (white dermographism, facial pallor, etc.)*
 - *Follicular keratosis;*
 - *Damage to the periorbital region and eyelids, hyperpigmentation of the skin of the periorbital region, Deny-Morgan symptom (additional folding of the lower eyelid);*
 - *Damage to the perioral region, parotid region, external auditory canals.*



▶ *Laboratory diagnostics.*

▶ Clinical analysis of blood - eosinophilia (not considered a specific sign).

▶ *Allergic and immunological examinations.*

▶ Skin test. It is recommended if there are no contraindications (prick tests with a standard set of non-infectious allergens);

▶ Determination of total IgE in blood serum:

▶ • An increase in the amount of IgE is not considered a specific sign;

▶ • A low level of total IgE does not indicate the absence of atopy and is not a criterion to exclude the diagnosis of atopic dermatitis.

Anaphylactic shock.

How does anaphylaxis present?

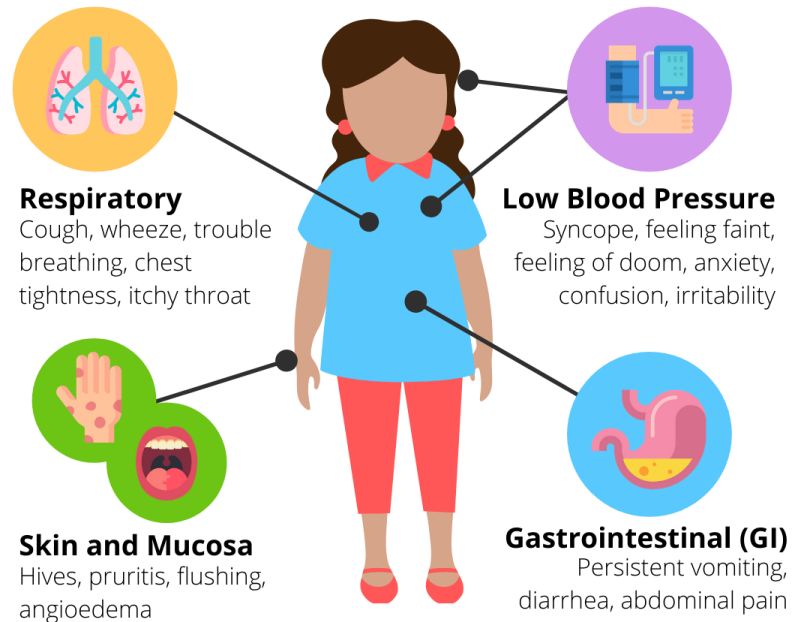


Image Credits: Freepik, Flat Icons, Smashicons, Monkk

Infographic by @AnnYoungMD for CRB SIDERS

- ▶ Anaphylactic shock - an acute life-threatening condition accompanied by severe hemodynamic disorders (according to the international recommendations of the World Allergy Organization: a drop in systolic blood pressure below 90 mm Hg or up to 30% of the initial level) causing circulatory failure and hypoxia in all vital organs is a hypersensitivity reaction. Anaphylactic shock is the most severe clinical manifestation of anaphylaxis associated with high mortality.

► Classification.

I. Depending on the dominant clinical symptoms of anaphylactic shock – typical, hemodynamic, asphyxial, abdominal, cerebral options;

II. Determined by the degree of hemodynamic disturbances, four degrees are distinguished depending on the severity of the course of anaphylactic shock - first, second, third, fourth degrees;

III. Depending on the nature of anaphylactic shock, acute malignant, acute benign, prolonged, recurrent (relapsing) and abortive.

► Diagnostics.

- As a rule, the diagnosis of anaphylactic shock is based on the clinical picture of the disease and the conditions under which the reaction occurs.
- 1. Complaints and history. The patient's complaints depend on the severity of anaphylactic shock, dominant clinical symptoms and course. Collecting anamnesis is possible after the patient's condition is stabilized and plays an important role in diagnosing shock, determining the cause of its development and preventing repeated reactions.
- Reasons that can cause anaphylactic shock - injection of drugs, food, insect stings, specific-allergen immunotherapy, physical stress, etc.
- Time of onset of anaphylactic shock - the sudden onset of characteristic symptoms (minutes or hours) after exposure to the allergen. Symptoms usually progress rapidly after that.
- Complaints of the patient (in cases of preserved consciousness) - anxiety, fear, excitement, tremors, weakness, dizziness, numbness of the tongue, fingers, ringing in the ears, blurred vision, nausea, cramp-like pains in the abdomen.



Laboratory diagnostics:

Performing laboratory tests during anaphylactic shock is important for differential diagnosis with other types of shock.

- ▶ *Determination of serum tryptase level - examination is carried out twice: 15 minutes-3 hours after the onset of the first symptoms and after recovery;*
- ▶ *Determination of serum histamine level -is performed within 15-60 minutes after the first symptoms of anaphylactic reaction appear.*

Allergic rhinitis.

- ▶ Allergic rhinitis (AR) is an inflammatory disease of the mucous membrane of the nasal cavity (which develops under the influence of allergens).
- ▶ **Classification.**
- ▶ Allergic rhinitis is classified according to its form, character, degree of severity, stages of the disease.
- ▶ • *seasonal allergic rhinitis* occurs against dust (pollinosis) and fungal allergens: wood dust, grains and weeds, Cladosporium, Penicillium, Alternaria, etc. fungal spores;
- ▶ • household powders, library powders, anti-mold fungus, etc. year-round allergic rhinitis.
- ▶ *In addition, allergic rhinitis is classified according to severity:*
- ▶ • mild form – only mild clinical symptoms of the disease are observed, and these symptoms do not affect the patient's ability to work and sleep;
- ▶ • moderately severe form – symptoms disrupt the patient's sleep pattern, significantly reduce the quality of life;
- ▶ • severe form – the symptoms become so deep that the patient cannot continue with normal life activities (work, education, sports, sleep patterns, etc.) without treatment.
- ▶ According to the period of the disease, ***acute and remission*** periods are distinguished.



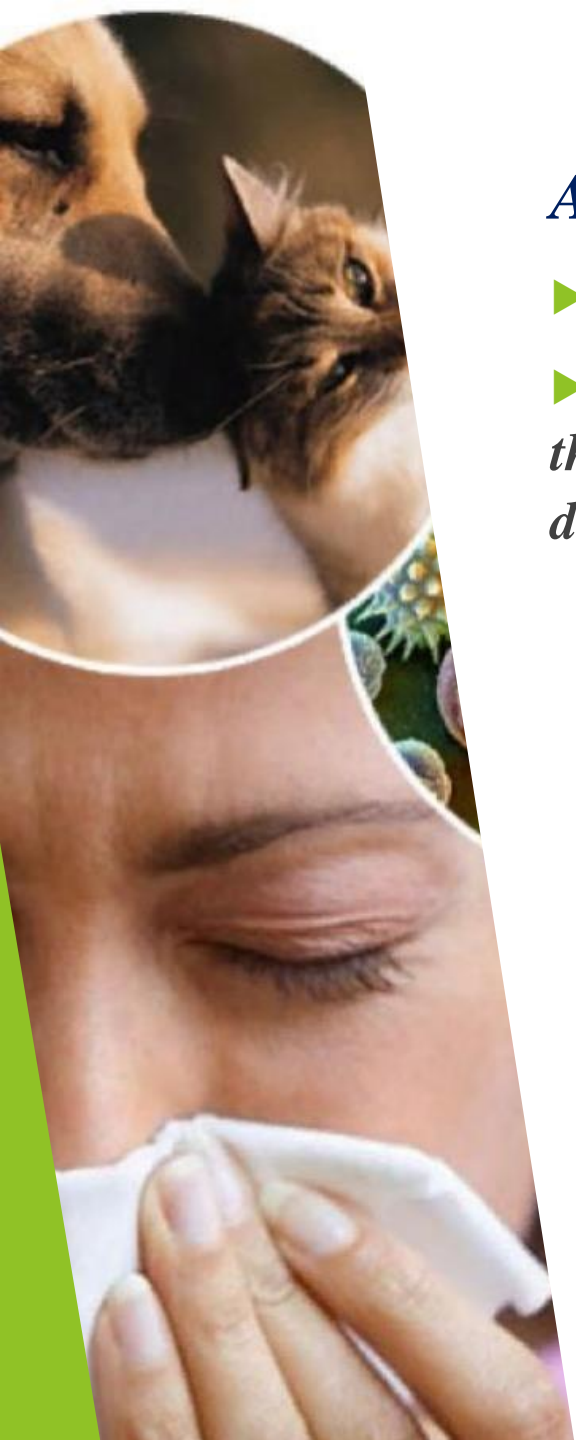
*Diagnos*tics.

▶ *Clinical and physical indicators.*

- ▶ • Runny nose (rhinorrhea), frequent sneezing, itching in the nasal cavity are the most common symptoms of seasonal allergic rhinitis.
- ▶ • Nasal congestion. It is more common during year-round allergic rhinitis.
- ▶ • Other clinical signs. In addition to classic symptoms, symptoms characteristic of bronchial asthma is also observed; general weakness, headache, earache, hearing loss, smell disturbance, nosebleed, sore throat, cough, eye symptoms are often noted.
- ▶ *Physical examinations.* The characteristic symptoms of rhinitis include lack of nasal breathing, swelling of the face, dermatitis on the upper lip and on the wings of the nose, etc. belongs to. In cases of secondary infection, nasal discharge can be purulent-mucous in nature.

Allergic anamnesis.

- ▶ • *Allergen contact.* In year-round allergic rhinitis, the symptoms are observed constantly or regularly with worsening of the condition. In the case of seasonal allergic rhinitis, the relationship between the symptoms and the seasons is necessarily observed.
- ▶ • *Association with bronchial asthma.* Allergic rhinitis is considered a risk factor for bronchial asthma. Therefore, at present, such patients must be examined to check for bronchial asthma (hidden bronchospasm check).



Allergic and immunological examination.

- ▶ • *Skin tests with non-infectious allergens;*
- ▶ • *Determination of specific IgE - is carried out to confirm the diagnosis, especially when the results of skin tests are doubtful.*

Laboratory diagnostics.

- **General blood analysis - eosinophilia is possible during the acute period of the disease;**
- **Cytology of nasal secretion (rhinocytogram) - Increase in the number of eosinophils by 10% or more.**



Hereditary angioneurotic edema.



- ▶ *Hereditary angioedema is a rare, life-threatening, genetic disease that manifests as bradykinin-induced edema of the skin and mucous membranes.*
- ▶ *In most cases, hereditary angioedema is caused by a deficiency or reduced function of the C1 inhibitor, but there are rare forms of hereditary angioedema without changes in the complement system. The main manifestations of hereditary angioedema are various localized recurrent edema of the deep layers of the dermis lasting from several hours to several days. The characteristic features of edema in hereditary angioedemas are the absence of itching, skin hyperemia, as well as non-responsiveness to treatment with corticosteroids and antihistamines. Hereditary angioedema refers to primary immune deficiencies, in the pathogenesis of this disease disorders in the complement system play a key role.*

Etiology and pathogenesis.

The pathogenesis of the disease is caused by the increase of vascular permeability due to the influence of bradykinin, the release of intravascular fluid, and the occurrence of local edema in the skin, subcutaneous tissue, and mucous membranes. Accumulation of bradykinin in hereditary angioedema is caused by a deficiency in the amount of C1 inhibitor or a weakening of its functional activity. C1-inhibitor is a multifunctional enzyme that participates in the activity of the complement system, kallikrein-kinin system, activates the internal mechanism of blood coagulation and the fibrinolysis system. In patients with type I and II hereditary angioedema, the lack of C1-inhibitor leads to uncontrolled activation of the kallikrein-kinin system, resulting in the breakdown of high molecular weight kininogen and the formation of bradykinin. Bradykinin is considered a key mediator in the formation of edema. In addition to the above properties, bradykinin affects the nerve endings, causing pain, as well as a decrease in blood pressure, narrowing of the bronchi and dilation of large vessels (in the skin, mucous membranes, brain, kidneys and other organs). At the same time, the absence of C1 inhibitor leads to the activation of the fibrinolysis system, resulting in the accumulation of plasmin, which enhances the formation of bradykinin from high molecular weight kininogen. In the case of C1 inhibitor deficiency, uncontrolled activation of coagulation factor XII develops, which accelerates blood coagulation by an internal mechanism and enhances the activation of the kallikrein-kinin system.

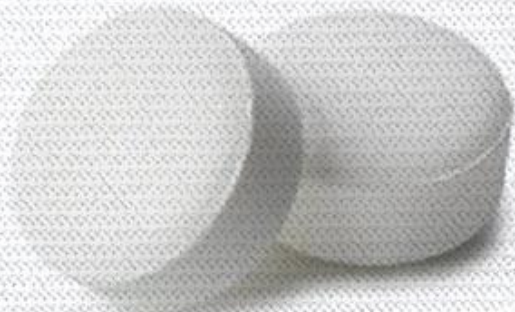
Classification

- ▶ 1. Hereditary angioneurotic edema observed with C1 inhibitor deficiency:
 - ▶ • Type I hereditary angioedema - deficiency of C1 inhibitor in plasma is noted;
 - ▶ • Type II hereditary angioedema – weakening of the functional activity of the C1 inhibitor is noted.
- ▶ 2. Hereditary angioneurotic edema with a normal level of C1 inhibitor:
 - ▶ • Hereditary angioneurotic edema followed by factor XII mutation;
 - ▶ • Hereditary angioneurotic edema followed by mutation of the angiotensin-converting enzyme-1 gene;
 - ▶ • Hereditary angioneurotic edema caused by mutation of the plasminogen gene.

Diagnostics

- ▶ Characteristics of edemas. Edema associated with the presence of bradykinin is pale, non-itchy (burning, pain), dense (pit symptom is not observed). Hyperemia is not observed in the skin covering the edema. In some cases, erythema is observed. Regression (withdrawal) of edema is possible within 72 hours.
- ▶ **Laboratory diagnostics.**
 - ▶ 1. Study of the level of C1-inhibitor and its functional activity. It is considered the gold standard in the diagnosis of type I and II hereditary angioneurotic edema. Changed indicators require reconfirmation at least 1 month apart.
 - ▶ 2. Determination of the C4 component of the complement system. It is used for screening purposes. The examination is carried out at least 2 weeks after stopping medication and no less than three days after receiving C1 inhibitor concentrate or blood plasma.
 - ▶ 3. Examination of C1q, C1-INH95 Kd levels and determination of the presence of antibodies against C1q. A decrease in the level is observed in patients with acquired angioedema.

Allergic to
ASPIRIN



«*Asthma on
Aspirin*» -

- ▶ *-is a pseudo-allergic chronic inflammatory process, which usually occurs in the respiratory tract due to increased sensitivity to non-steroidal anti-inflammatory drugs and is observed with nasal congestion, rhinorrhea, difficulty breathing, coughing and suffocation attacks.*

Triad Asthma

"Samter's Triad"



Aspirin/NSAID



Nasal polyp



Asthma

- ▶ It occurs in women aged 30 - 50 and is usually twice as common as in men. It accounts for 9 - 22 % of all bronchial asthma cases. At the same time, aspirin intolerance occurs in 30 - 40 % of patients suffering from bronchial asthma, sinusitis, and nasal polyps.
- ▶ The classic aspirin triad includes aspirin intolerance, nasal polyps, and bronchial asthma.
- ▶ The mechanism of development of bronchospasm and suffocation attacks during asthma on aspirin is related to disruption of arachidonic acid metabolism due to the effect of non-steroidal drugs. At this time, inflammatory mediators - leukotrienes, which increase inflammation in the respiratory tract, cause bronchospasm and the development of excessive mucus in the bronchi, and increase vascular permeability - are produced more. All this makes it possible to consider the pathology as a respiratory pseudoallergy.

Clinical course of «asthma on aspirin» :

At an early age, chronic rhinitis aggravated by taking aspirin, and later nasal polyps, hypertrophic sinusitis, purulent sinusitis, eosinophilia, bronchial asthma are observed.

Symptoms:

- ▶ *Abdominal pain with flushing;*
- ▶ *suffocation cough;*
- ▶ *Rhinitis;*
- ▶ *conjunctivitis;*
- ▶ *Quincke's edema;*
- ▶ *fever;*
- ▶ *diarrhea;*
- ▶ *nausea and vomiting.*



Diagnosis:

Diagnosis is based on history and clinical picture.

- ▶ *The patient receives non-steroid drugs in gradually increasing doses. If no response is observed in patients after taking 650 mg of the drug, it is considered as a lack of sensitivity.*
- ▶ *Currently, in the laboratory diagnosis of aspirin asthma, examination methods such as determination of LTE4 in urine and LTC4 in nasal mucus are being worked on. It should be noted that the amount of LTE4 in urine and LTC4 in mucus increases rapidly during non-steroidal anti-inflammatory drug sensitivity tests in patients with aspirin asthma.*

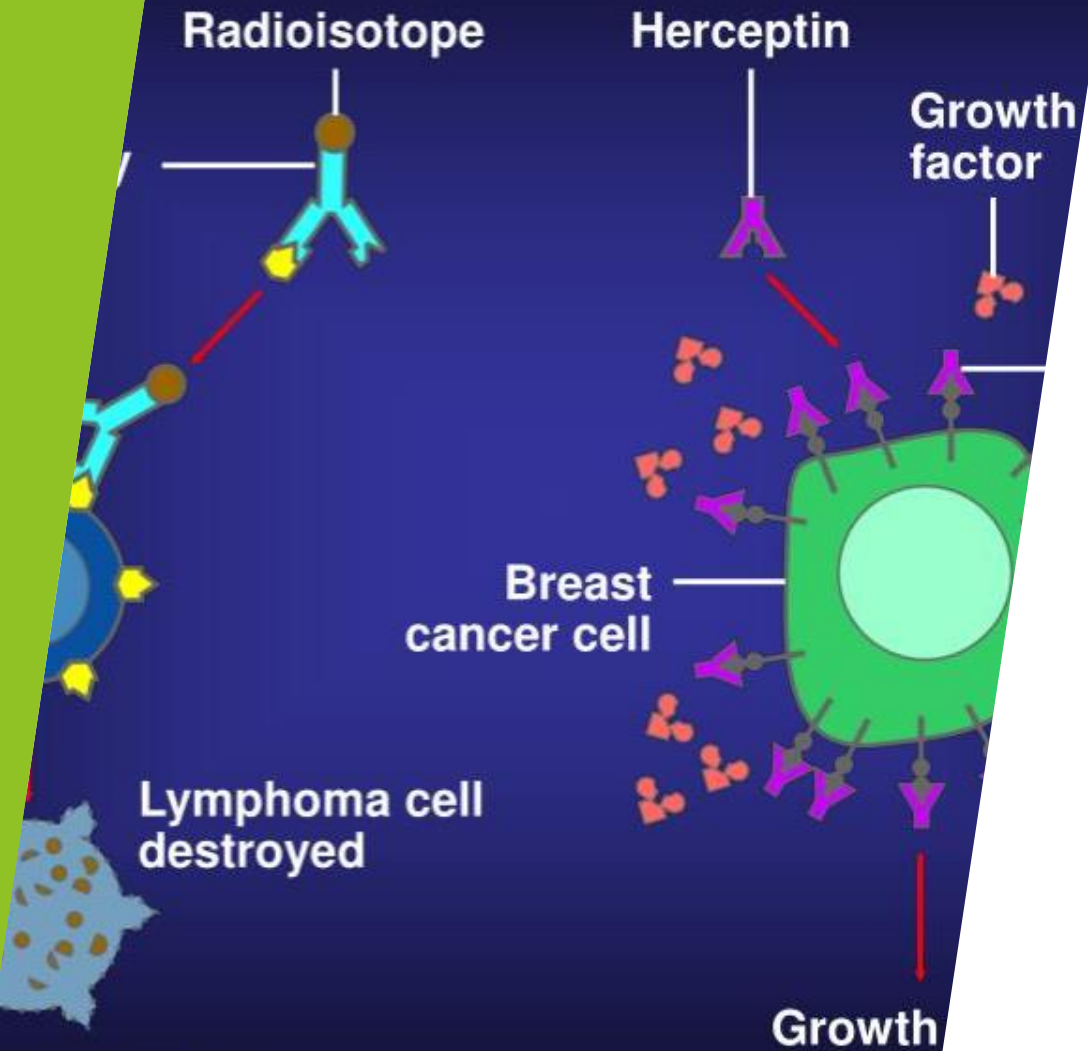


Immunoprophylaxis and immunotherapy:

It is a branch of immunology and studies specific methods of prevention and treatment of infectious and non-infectious diseases.

- ▶ *Immunoprophylaxis is a complex of measures aimed at creating active or passive immunity against the causative agents of infectious diseases or their antigens in order to form insensitivity to diseases that may occur in the body.*
- ▶ *Immunotherapy is a complex of measures aimed at disorders of the body's immune system functions and treatment of advanced diseases.*

Immunotherapy



► Immunoprophylaxis and immunotherapy:

- • Creating specific immunity or activating the activity of the immune system;
- • To activate or weaken individual parts (circles) of the immune system;
- • To normalize the work of the immune system in case of deviations in one or another case, etc. is applied for the purpose.

The background features abstract geometric shapes in various shades of green and blue. In the top left, there is a yellow circle partially cut off. In the top right, there are several overlapping leaf-like shapes in dark blue and light green. The bottom of the slide is a solid green bar with a dark blue circle on the left and a light green rounded shape on the right.

Thank you for attention!