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IMPORTANT BIOLOGICAL FACTORS IN THE ORIGIN OF YOUR ANGIOEDEMA DISEASE

Annotation: Angioedema is a pathological condition accompanied by the accumulation of fluid in the skin tissues and subcutaneous adipose tissue due to an increase in the permeability of the vascular walls of the microcirculatory bed. It is manifested by edema of the skin of various localization (face, neck, limbs), often combined with urticaria and itching of the skin. Diagnosis is made by physical examination, laboratory tests of blood, studying the hereditary and allergic history of the patient. Therapeutic tactics depend on the causes that caused the development of the syndrome, and may include the use of antihistamines, androgens, fibrinolysis inhibitors, and diuretics.

Key words: angioedema , blood vassels, neck, face.

Angioedema was first described in 1882 by the German scientist Heinrich Quincke. The researcher considered it an independent disease-angioedema. Currently, it is established that approximately half of the cases of pathology occur as a result of allergic processes of the reagin type, while the rest are independent acquired or hereditary conditions. The term "isolated angioedema" refers to a number of diseases that are based on impaired fluid circulation between the circulatory system and tissues. Angioedema can be diagnosed at any age, women are detected approximately 1.5-2 times more often than men. Genetically determined variants of the pathological condition are transmitted according to the autosomal dominant type.

There are a huge number of external and internal factors that contribute to the development of angioedema. The immediate cause of the pathology is changes in the complement system and a violation of some other physiological processes (blood coagulation, fibrinolytic and kinin reactions). In the vast majority of cases, the anomaly is caused by a deficiency or insufficient activity of the C1 inhibitor, a blood protease that slows down and stops a number of biochemical reactions in the blood and tissues. This phenomenon occurs under the influence of the following factors::

- Genetic features. A significant proportion of episodes of congenital angioedema are caused by a mutation of the SEPRING1 gene located on chromosome 11. It encodes the protein sequence of the C1 inhibitor, so when this gene is changed, there is a protease deficiency or loss of its function.
- Lymphoproliferative pathologies. Some conditions with accelerated proliferation of lymphocytes or their progenitor cells (for example, lymphomas) may be accompanied by angioedema. The reason for this is an increase in the rate of destruction of the C1 inhibitor.
- Autoimmune reactions. Under certain conditions, antibodies to C1 esterase are formed, binding this enzyme and contributing to its destruction. The process can occur both in isolation and with systemic allergic reactions or other disorders.

A rare variant of pathology is angioedema, which develops at the optimal level of C1-inhibitor – mainly it includes the so-called estrogen-dependent edema. It is assumed that they are caused by genetic factors and are transmitted by an X-linked mechanism, the course of the disease is aggravated by taking estrogen preparations. There is also a type of disease provoked by the use of angiotensin-converting enzyme inhibitors, which are part of numerous antihypertensive drugs.

Pathogenesis

The C1-inhibitor deficiency that occurs for one reason or another leads to activation of the complement system, increases the concentration of kallikrein and bradykinin. The latter stimulate the

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formation of vasoactive peptide compounds that affect vascular walls and smooth muscles. As a result, the precapillary arterioles first expand, then the cell plasma elements exit from the bloodstream into the intercellular space. Thus, a local angioedema is formed, which is manifested by a characteristic clinical picture. In addition, reactions of the bradykinin link can cause spasm of smooth muscle cells of the digestive and respiratory systems, disrupting their motor skills.

Classification

Taking into account the peculiarities of the clinical course in clinical allergology, all cases of angioedema are divided into two large groups – isolated and combined. The former are manifested only by edema of subcutaneous fat and skin, the latter can be accompanied by urticaria, respiratory spasm and other symptoms. The division is rather conditional and poorly reflects the causes of the disease. Hereditary and acquired forms of angioedema are distinguished by etiological characteristics. Congenital varieties account for approximately 2-5% of the total number of cases of the disease, and include the following types::

- Type 1. Is caused by the almost complete absence of a C1 inhibitor, which occurs when the SEPRING1 gene is mutated. It is characterized by a rather severe course-generalization and severity of edema, their appearance not only on the skin, but also on the mucous membranes of the respiratory tract or digestive tract. It is registered in 85% of cases of hereditary angioedema.
- Type 2. Develops with a relative lack of an inhibitor due to its slow formation or reduced activity due to an incorrect structure of the enzyme. The clinic is less severe, edema mainly affects the tissues of the extremities, sometimes the face. This variant of the disease is diagnosed in 12-14% of patients with hereditary forms of angioedema.
- Type 3. It is extremely rare, usually due to the absence of C1-esterase deficiency. As a rule, it is represented by estrogen-dependent edema-exacerbations of pathology during pregnancy, taking combined oral contraceptives, and replacement therapy during menopause.

All variants of hereditary angioedema are isolated, not accompanied by urticaria or other disorders. Acquired varieties have a different classification, which includes only two main types of the disease:

- Type 1. It is detected against the background of lymphoproliferative conditions – lymphomas, with some infectious lesions. The reason is an increase in the consumption of C1-inhibitor and its subsequent lack.
- Type 2. It occurs due to the synthesis of autoantibodies to the inhibitor, which sharply reduces its concentration in the blood. This phenomenon occurs in some autoimmune and allergic conditions, immunodeficiency and other pathologies.

Symptoms of angioedema

The main symptom of the pathology is the appearance of painless edema of the skin of various sizes. Patients note a feeling of fullness and tension, there are no other subjective complaints. Unlike inflammatory edema, the affected area is characterized by a paler color than the surrounding skin areas and the absence of a local increase in tissue temperature. Most often, edematous manifestations are found on the upper and lower extremities, face (lips, cheeks, eyelids, ears), neck, in the genital area. Pruritus of the skin is uncharacteristic, but can be detected when angioedema is combined with urticaria.

In some patients, edema is detected in the area of the mucous membranes, sometimes the pathological process also affects the submucosal plate. The most frequently affected organs are the oral cavity (tongue, soft palate), respiratory tract, and gastrointestinal tract. With the development of angioedema of the respiratory system, there is a feeling of lack of air, hoarseness or complete loss of voice, barking cough. Involvement of the gastrointestinal tract is manifested by a pronounced abdominal syndrome –

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pain, nausea, vomiting. There is tension in the abdominal wall muscles, which creates a false picture of peritonitis or acute intestinal obstruction.

Extremely rare symptoms of the disease are signs of pleural effusion (cough, chest pain, difficulty breathing). Other rare variants of pathology include local cerebral edema (registered depression of consciousness, hemiparesis), angioedema of the bladder (accompanied by acute urinary retention), muscle and joint damage. Isolated forms of the disease develop slowly over 12-48 hours. After that, in the absence of complications, the swelling slowly resolves within 5-8 days. Some combined variants of angioedema (especially allergic ones) can progress much faster – within a few minutes or hours.

Complications

The probability of complications in angioedema depends on the localization of the pathological process. Most common (approximately half of all complicated cases) patients have difficulty breathing due to narrowing of the laryngeal lumen or bronchi. In the absence of medical attention, the violation can lead to death. Abdominal forms of pathology are relatively dangerous, which can cause disorders of peristalsis with the development of obstruction and peritonitis. Often, edema of the digestive tract organs causes unnecessary surgical intervention due to erroneous diagnosis. Brain damage can lead to coma and a number of neurological consequences (impaired coordination, speech, perception). Acute urinary retention with edematous phenomena in the bladder is the cause of fluid reflux, hydronephrosis and renal failure.

Diagnostics

In most cases, the diagnosis of angioedema is made by an immunologist. Less often, this pathology is encountered by specialists in other areas – dermatologists, pediatricians, gastroenterologists, internists. The definition of the disease is often difficult due to its diverse etiology and very wide range of clinical manifestations. The main focus is on anamnestic information and the results of specific laboratory tests. Diagnosis of angioedema includes the following methods:

- Survey and inspection. During external examination, the prevalence and localization of the edematous area are clarified, and the absence of soreness is confirmed. The method of questioning is used to find out what preceded the development of pathological manifestations (stress, eating any products, taking medications), whether similar reactions occurred in relatives.
- Laboratory tests. A specific method for diagnosing angioedema is to determine the level of C1-inhibitor in blood plasma – its absence or decrease in the amount indicates the presence of the disease. It is possible to determine the titer of antibodies to the C1 inhibitor – this technique allows you to find out whether the disease has an acquired autoimmune character.
- Additional research. If the respiratory system is affected, bronchoscopy and chest radiography are performed. Usually, edema of the laryngeal tissues, bronchospasm is detected, and occasionally pleural effusion is detected. Ultrasound of the abdominal cavity allows differentiating abdominal forms of angioedema from peritonitis and other pathologies of the gastrointestinal tract.

In addition to the above methods, a huge number of different factors are taken into account when diagnosing this condition. For example, the age of the patient: hereditary varieties are more often found in people under 20 years of age, acquired forms – in people over 40 years of age with a burdened anamnesis. Take into account the presence or absence of concomitant symptoms – urticaria, respiratory disorders. Differential diagnosis is carried out with edema of a different genesis-as a result of renal pathology, venomous insect bites, local allergic and inflammatory reactions.

Treatment of angioedema

Therapeutic measures for angioedema are divided into two groups-methods for stopping an acute attack and techniques for preventing its subsequent development. In both cases, similar medicinal substances are used – depending on the purpose of their appointment, only the scheme of

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administration and dosage change. Most often, the following drugs are used to treat angioedema in modern immunology::

- Androgens. Some analogues of male sex hormones (danazol, methyltestosterone) they can enhance the synthesis of C1-esterase in liver cells. They reduce the severity of symptoms of pathology and reduce the likelihood of an attack of the disease in the future.
- Inhibitors of fibrinolysis. Drugs that prevent fibrinolytic processes also slow down the reactions of the kallikrein pathway. This reduces the rate of plasma diffusion into the tissues, reduces the likelihood of angioedema. The use of drugs of this group (e-aminocaproic or tranexamic acids) is carried out under the control of the state of the blood coagulation system.
- Fresh frozen plasma. Transfusion of donor plasma containing a C1-inhibitor is an effective method for stopping acute edema, especially of a hereditary nature.

In the presence of autoantibodies against complement components, their removal from the bloodstream by plasmapheresis is indicated. This is a temporary measure that can significantly reduce the severity of edematous manifestations. If the patient's life is threatened (for example, due to airway obstruction), epinephrine is recommended, and if it is ineffective, conico-or tracheotomy is recommended. If the cause of angioedema is the presence of another disease (allergic, autoimmune, or other), a treatment regimen is developed according to the indications. There are also promising inhibitor drugs that are used in some countries to treat this condition.

Prognosis and prevention

The prognosis of angioedema is considered uncertain until its etiology is clarified in a particular patient. With the hereditary nature of the pathology, there is always a risk of developing fatal laryngeal edema, so it is advisable for patients to have a card indicating the diagnosis. With proper preventive treatment, seizures are rare and do not pose a threat to the patient's life. The prognosis of acquired forms depends on the nature of the underlying disease. Preventive measures include timely treatment of allergic and autoimmune conditions.

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