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PREVALENCE, SYMPTOMS AND SPECIFIC ASPECTS OF THE DISEASE IN CHILDREN WITH DRUG ALLERGY

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Yomgurova Ozoda Rajabturdievna
Bukhara State Medical Institute

Summary.
The systematization of issues related to clinical symptoms, treatment and prevention of drug allergy is currently of great importance for practical medicine. Knowledge of the clinical manifestations of an allergic reaction to drugs, therapeutic tactics in the relief of its acute phase, organizational and preventive measures can largely reduce the likelihood of adverse outcomes of the disease. According to the Boston Collaborative Drug Surveillance Program, the prevalence of cutaneous adverse drug reactions in hospitalized patients was 2.2%, with antibiotics causing such reactions in 7% of cases.

Keywords.
Lyell's syndrome, Stevens-Johnson syndrome, Bronchial asthma, Toxicoderma.

Clinical forms
I. By prevalence:
1. Generalized:
anaphylactic shock;
serum sickness and serum-like syndrome (skin-visceral form of drug allergy);
fever;
generalized vasculitis in combination with other lesions.
2. Localized (organ and system):
skin lesions;
toxicoderma with damage to internal organs (Lyell's syndrome, Stevens-Johnson syndrome);
hematological lesions;
vasculitis;
visceral (internal organs);
mucous membranes and respiratory system;
nervous system.
II. By severity:
light;

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moderate;
heavy.
III. With the flow:
acute;
subacute;
chronic.
IV. According to the presence of complications:
uncomplicated;
complicated.

Although the clinical manifestations of drug allergy are diverse, there are about 40 variants of their course, in adults they most often occur in the form of skin, hematological, respiratory and visceral manifestations. Drug-induced anaphylactic shock (MAS) is the most severe generalized manifestation of drug allergy, caused by a type 1 immunological reaction and the release of a large number of cytokines, which is accompanied by a pronounced impairment of the activity of various organs and systems. It is characterized by initial excitation followed by depression of the central nervous system, bronchospasm, and a sharp decrease in blood pressure.

Depending on the course, the following variants of anaphylactic shock are distinguished: acute, malignant, benign, protracted, recurrent, abortive.

The main clinical manifestations of anaphylactic shock:
hemodynamic disorders;
respiratory failure (shortness of breath, bronchospasm, suffocation);
disruption of the gastrointestinal tract (nausea, vomiting, diarrhea);
skin rashes (urticaria and Quincke's edema).

Anaphylactoid reactions are caused by the release of allergy mediators without a preliminary immune response. They can develop by the following main mechanisms: direct activation of the complement system by a substance (most often by an alternative, properdin mechanism, i.e. bypassing the initial components of the system due to the activation of the C3 component); histamine-liberator effect

Serum sickness is a systemic generalized allergic reaction involving many organs and systems in the pathological process, which occurs in response to the introduction of foreign serum or its protein fractions, as well as some drugs (for example, penicillin). Approximately 5–6 days after the introduction of the allergen, antibodies to these antigens appear in the patient's blood, interacting with them and forming immune complexes, which then fall out in small vessels and cause

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inflammatory reactions in the skin, kidneys, joints, heart muscle. At the same time, the complement system is activated, which is also included in the process and enhances inflammation. An early sign of serum sickness is redness, swelling, and itching at the injection site, sometimes 1–2 days before the general manifestations. At 7-12 (6-21) days after administration, the general reaction develops - swollen lymph nodes, skin rashes, joint pain, fever. In parallel, there are moderate changes in the function of the heart and kidneys, on the part of white blood - an increase in the number of lymphocytes. The reaction is usually mild and goes away after a few days even without treatment. Deaths are rare. of the drug; direct activation of humoral enhancement systems.

Bronchial asthma is a chronic relapsing disease characterized by asthma attacks due to bronchial hyperreactivity due to various reasons. The pathogenesis of bronchial asthma is based on an immunological mechanism, the starting link of which is the combination of reagins with the corresponding antigen on the surface of the cell membranes of basophils and mast cells located in the respiratory mucosa. The result is damage to the cell membrane, the release of allergy mediators - primarily histamine, as well as the slowly reacting substance of anaphylaxis (MRS-A), acetylcholine, bradykinin, serotonin, prostaglandins, leukotrienes and, as a result, a sharp spasm of the smooth muscles of the bronchi, accompanied by the release of bronchial glands viscous mucus and obstruction of the bronchi. This leads to the development of an asthma attack, during which the patient takes a forced position, straining the pectoral muscles in order to overcome the difficulty in exiting. In these moments, the patient is seized with fear, it seems to him that the attack will certainly end in death, although, as a rule, the tragic ending does not occur: difficulty in breathing gradually weakens, sputum leaves, and normal breathing is restored. In some patients, seizures are prolonged and often recur.

Allergic rhinitis is a symptom of drug allergy, often combined with bronchial asthma, and sometimes acting as an independent disease. An allergic reaction unfolds in this case in the nasal mucosa.

Allergic rhinitis is characterized by profuse liquid mucous discharge from the nose. At the same time, the mucous membrane swells, swells, acquires a pale gray color. Nasal itching, sneezing, and headache are common. Sometimes the swelling of the mucosa is so pronounced that it protrudes and the so-called allergic polyps are formed.

Allergic conjunctivitis can occur as an independent disease or be combined with other allergies - allergic rhinitis and bronchial asthma. The leading symptom

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of the disease is the development of a sharp itchy inflammation of the mucous membrane of the eyes, followed by the occurrence of organic tissue changes. Sometimes the cause of the development of allergic conjunctivitis is the use of eyewash solutions used in ophthalmic practice. These drugs sometimes contain chemical compounds that have an allergenic effect.

Four types of allergic reactions (Gell-Coombs classification) are based on the involvement of vessels and connective tissue in the process, which leads to damage to the nervous system, in which the connective tissue is located in the vessels and membranes of the brain. The basis of the defeat of the nervous system is allergic vasculitis.

Drug-induced vasculitis is a systemic disease, which is based on a generalized lesion of arteries and veins of various sizes with secondary involvement of internal organs and tissues in the pathological process. Neurological symptoms are based on acute or chronic cerebrovascular and peripheral insufficiency as a result of immune vascular inflammation due to the prolonged action of allergens.

Skin itching can occur when taking any drug, but more often it is caused by hypnotics, sulfonamides, penicillin, novocaine, insulin, etc. An objective examination of patients shows traces of scratching. Itching can be widespread and limited, moderate and unbearable, constant or paroxysmal. It must be differentiated from pruritus in diabetes mellitus, renal failure, lymphoproliferative diseases, oncopathologies, etc.

Drug-induced systemic lupus erythematosus is a generalized drug-induced syndromic disease clinically and immunologically similar to systemic lupus erythematosus (SLE).

It can be induced by hydralazine derivatives, apressin, adelfan, phenothiazine derivatives, methyldopa, novocainamide, inderal, sulfonamides, penicillin, isoniazid, oral contraceptives, β-blockers, lithium preparations, antibiotics, antipsychotics, etc. The clinic is characterized by fever, joint pain, muscles, pleurisy, allergic rashes associated with taking drugs.

Differential diagnosis is based on the following criteria: prescribing drugs before the appearance of clinical and paraclinical signs; reverse development of complications after discontinuation of the drug (clinical manifestations of the syndrome disappear, as a rule, earlier than immunological ones); the development of similar symptoms after repeated administration of the drug.

Skin rashes with drug allergies are diverse. Small-spotted rashes appear when the superficial blood vessels of the skin expand. Roseolous rash usually begins with

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itching, sometimes small red spots and is localized more often on the face and trunk. Soon it begins to peel off (3-4 days). Sometimes there are new rashes that merge and combine with other elements (urticarial, cystic, etc.). This roseolous rash must be distinguished from syphilitic roseola, pink lichen, measles, scarlet fever.

A maculopapular rash is most often manifested in the treatment of antibiotics, sulfonamides, barbiturates, vitamins, mercury diuretics, nitrofuran and other drugs and has a scarlet or measles-like appearance. Accompanied by severe itching. Gradually, its color changes to bluish-pink, sometimes with a marble tint. The duration of the rash is 1-2 weeks. One of the hallmarks of an allergic skin lesion is blood eosinophilia in the affected area.

Common skin manifestations of drug allergy usually belong either to the group of toxicoderma or to the group of allergic dermatitis.

Toxicoderma is an inflammatory skin lesion (rash), which is a reaction to the circulation of substances in the blood that have sensitizing, toxic, dysmetabolic and other mechanisms of action and are introduced into the body by oral, parenteral, inhalation, transepidermal routes.

Drug toxicoderma is toxicoderma caused by drugs administered for therapeutic (drugs) or diagnostic (eg, contrast agents) purpose.

Acute toxic-allergic reactions (OTAR) to drugs is an acute systemic immunological reaction of the body that occurs in response to a therapeutic dose of a drug, more often against the background of a viral or bacterial infection, characterized by an acute onset, rapid progression, severe symptoms of toxemia and high mortality.

The main mechanisms for the development of OTAR for medicines:

1) the interaction of the drug with viral and bacterial agents or with proteins of the body ensures the development of immune mechanisms of allergic tissue damage, mainly of types 2, 3, and 4;

2) the release of humoral mediators of an allergic reaction;

3) damaging effect of lymphocytes and macrophages;

4) toxic effects of products of destroyed tissues and microbial bodies on the body.

Erythema multiforme exudative (MEE) is an acute, rarely recurrent disease of the skin and mucous membranes that occurs from various causes and is characterized by a characteristic combination of many primary elements of the rash (spots, papules, blisters, vesicles, blisters).

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It can be caused by both infection and drugs. The most common MEE inducers are sulfonamides, pyrazolone derivatives, tetracyclines, barbiturates, acetylsalicylic acid, diuretics, progesterone, streptomycin, etc.

The primary element is an erythema spot, which within two days increases to 1-2 cm in diameter, turns into a papule. The formed element has sharp borders, darker cyanotic edges and a center with a lighter pink stripe between them, which gives the appearance of a bull's eye, iris or target. A papule or vesicle may be located in the center of the spot.

The most common localization is the dorsal and palmar surfaces of the hands and the plantar surface of the feet, the extensor surfaces of the forearms and lower legs, and the external genitalia. The rash is located symmetrically. With widespread MEE, almost all areas of the skin are affected, with the exception of the scalp.

Severe bullous forms of MEE are characterized by a tendency to merge elements and general phenomena (fever, malaise).

Stevens-Johnson syndrome (SSD) is a severe malignant exudative erythema (acute mucocutaneous-ocular syndrome). Skin lesions (from 10% to 30% of the body surface) occur during drug therapy, provoked by hypothermia and focal infection. Sulfonamides, antipyretics, penicillin, tetracycline and other drugs can cause this disease. The etiopathogenesis involves the herpes simplex virus and early therapy with acyclovir and prednisolone. In the vessels of the dermis, deposits of IgM and C3-complement and fibrin were found, infiltration with basophils, monophages and lymphocytes, which indicates the participation of allergic reactions of types II–IV.

The onset is stormy with obligatory damage to the mucous membranes. Temperature 39–40 °C. Pain in the throat, joints, pronounced salivation, herpetic eruptions on the lips, oral mucosa, genitals, in the perianal region. Abundant rashes appear on the skin, as with erythema multiforme exudative (erythematous, papular and vesiculo-bullous elements, there may be single rashes). The blisters are grouped, purple-bluish, with small vesiculations, fixed on the hands and feet, in the interdigital spaces, often with hemorrhagic contents. Erosions quickly form on the mucous membranes, which ulcerate and become covered with deposits of a dirty gray color. Severe conjunctivitis, nosebleeds, lesions of internal organs develop. The general condition of patients is severe, leukocytosis, high ESR, thrombocytopenia, eosinophilia appear in the blood. With the progression of the disease, the likelihood of death is high. The temperature may be elevated for 1-2

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weeks, then decrease. At 4–6 weeks after the rash, moderate pigmentation remains. The duration of the disease is 4-6 weeks.

Lyell's syndrome, or toxic epidermal necrolysis (TEN), bullous necroepidermolysis, scorched skin syndrome, bullous erythroderma, was described in 1956 [10]. Lethality is from 30–50%. They get sick at any age. The process is polyetiological. It is sometimes difficult to differentiate from other severe toxicodermas and begins as MEE or SJS, which have many similarities. Differences between them are determined by the form and prevalence of the lesion: with lesions of less than 30% of the body surface and mucous membranes and the predominance of blisters - this is SJS, more than 30% with necrolysis and detachment of the epidermis - Lyell's syndrome. Already in 1970-1980. some Soviet and foreign researchers noted the relationship between the development of MEE, SJS and Lyell's syndrome. Sometimes it is difficult to draw a line between MEE and SDS, SDS and PETN.

Lyell's syndrome, or toxic epidermal necrolysis (TEN), bullous necroepidermolysis, scorched skin syndrome, bullous erythroderma, was described in 1956 [10]. Lethality is from 30–50% [15]. They get sick at any age. The process is polyetiological. It is sometimes difficult to differentiate from other severe toxicodermas and begins as MEE or SJS, which have many similarities. Differences between them are determined by the form and prevalence of the lesion: with lesions of less than 30% of the body surface and mucous membranes and the predominance of blisters - this is SJS, more than 30% with necrolysis and detachment of the epidermis - Lyell's syndrome. Already in 1970-1980. some Soviet and foreign researchers noted the relationship between the development of MEE, SJS and Lyell's syndrome. Sometimes it is difficult to draw a line between MEE and SDS, SDS and PETN.

The disease develops after 10 hours - 21 days from the moment of taking the medicine. The process often begins as a common urticaria, not amenable to therapy with antihistamines and calcium preparations. The onset is acute, sudden. There are chills, vomiting, diarrhea, headache, sore throat, joints, muscles. Within a few hours, the temperature rises to 39–40 °C. There is soreness and burning of the skin, then a rash appears in the form of erythematous painful and slightly swollen spots of various sizes, often merging with each other and localized on the skin of the face, trunk, limbs and mucous membranes. Often the spots become bluish-ashy in color. Against the background of spreading erythema, flabby thin-walled irregularly shaped blisters are formed, ranging in size from a nut to a palm and more, in places

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merging with each other. The contents of the blisters are serous or serosanguineous. In place of the opened blisters of the skin and mucous membranes, extensive erosions are formed. They separate abundant serous or serous-bloody exudate, which leads to rapid dehydration of the body. There is a sharply positive Nikolsky syndrome - when you press a finger on the skin, the epidermis exfoliates, exposing the erosive surface. Areas devoid of epidermis resemble burns of II-III degree. The unshed epidermis looks like corrugated paper. Hyperesthesia is very pronounced - even the touch of a sheet causes pain. The skin of the hands comes off like a glove. The tongue is covered with a dirty yellow coating, swollen, the mouth opens with difficulty, there are many erosions in the pharynx. Dried crusts on lips. The oral cavity and tongue represent a continuous wound surface. The conjunctiva is hyperemic, headaches increase, damage to internal organs, loss of consciousness is noted.

There can be three types of Lyell's syndrome: hyperacute with a fatal outcome; acute with the addition of a toxic-infectious process and, as a result, with a possible fatal outcome; favorable when the process is allowed for 6-10 days. In a severe form, during the first 2–6 days, the area of damage to the skin and mucous membranes progressively increases, symptoms of severe pathology of the kidneys, lungs, and heart appear. Death can occur due to developing toxic lesions, dehydration, anuria, coma.

Drug photodependent toxicoderma and dermatitis. Photodependent drug toxicoderma is a group of rashes that is an inflammatory response to systemically administered drugs, developed under the influence of ultraviolet radiation (UV).

Photodependent allergic toxicoderma develops regardless of the dose of the drug after a latent period of sensitization to drug metabolites formed during exposure from several days to months. The reaction develops in 1–2 days under the action of UV rays with a length of 290–320 nm (B) and is a T-cell, delayed reaction. The clinical feature of photo-dependent toxicoderma is the localization of the rash on areas of the skin (face, hands) that are open to insolation, which is also typical for other photodermatosis, as well as for aerogenic contact dermatitis.

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