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ETIOLOGY OF ALIMENTARY TOXIC ALEIKIA , TREATMENT AND PREVENTION METHODS

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Annotation: Alimentary toxic aleikia is a severe food intoxication caused by mycotoxins of fungi of the genus Fusarium. The specific effect of the toxin is damage to the lymphoid tissue and bone marrow. The main manifestations of the disease are necrotic angina and hemorrhagic syndrome. Diagnosis consists in detecting the pathogen during bacteriological examination of the patient's blood and tissues, no less important is the careful collection of anamnesis, the presence of characteristic changes in the tonsils and leukopenia in a general clinical blood test. Treatment consists of detoxification, systemic and local antibacterial therapy, and may include transfusion of blood components.

Key words: alimentary toxic aleikia, alimentary hemorrhagic tonsillitis.

Alimentary toxic aleikia, also known as septic, alimentary hemorrhagic tonsillitis, and acute alimentary mycotoxicosis, is a severe poisoning caused by the fusarium fungal toxin. The path of infection is food, most often associated with eating products from overwintered grain containing fungal spores. The pathology was first described in 1932 during an epidemic in Kazakhstan; it occurs all over the globe. It was found that residents of agricultural areas are most susceptible to infection, usually an increase in the number of cases is observed in the period from April to June. A sick person is not contagious to others.

The causative agents of the disease are fungi of the genus Fusarium, which produce the toxin poin, which causes local manifestations in the form of necrosis and has a tropicity to hematopoietic and lymphoid tissues. The source of infection is grain crops that served as a breeding ground for reproduction and accumulation of poin during wintering. These agricultural plants include millet, buckwheat, wheat, rye, oats, and barley. Favorable conditions for the growth of the fungus are high humidity, the presence of oxygen and heat. In the cold season, fungi turn into spores and survive the winter, and after the onset of spring, they begin to produce poin again.

Warm winter and early spring, as well as high humidity and other violations of grain storage rules are considered risk factors for an outbreak of the disease. The toxin is not destroyed by heat treatment, fermentation, and is able to maintain its properties for up to five years, especially in the storage temperature range from -1°C to +5°C. Patients with immunosuppression (HIV infection, long-term therapy with corticosteroids, immunosuppressants), people after splenectomy, children, agricultural workers, food industry workers, and public catering are considered risk groups for morbidity.

Pathogenesis

After getting poin on the mucous membranes, a local reaction occurs, associated with necrotic tissue changes due to extensive inflammation. Through the wound surface, the toxin is

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intensively absorbed into the blood, spreads throughout the body, showing tropicity to myeloid (red oblique brain) and later to lymphoid tissue. Under the influence of poin, there is a suppression of hematopoiesis, inflammatory changes in the thymus, spleen, lymph nodes, Peyer's plaques and appendix. There is a gradual decrease in the number of red blood cells, monocytes, granulocytes, platelets, T-lymphocyte precursors, B-lymphocytes and NK-cells, as well as macrophages and dendritic cells. Hemorrhagic changes occur in parenchymal organs, skin, and septic conditions develop. Immunity after a previous illness is stable, but its duration has not been studied.

Classification

In the clinic of alimentary mycotoxicosis, two variants of the course are distinguished, which are directly dependent on the amount of grain product eaten, the toxigenicity of the strain, the concentration of toxin in the food product, and the immune competence of the patient's body:

1. Lightning fast. It is characterized by a rapid increase in clinical manifestations and a fatal outcome during the first day of the disease due to advanced DIC syndrome or sepsis.
2. Typical. It lasts about 3-4 weeks, is characterized by a gradual increase in the severity of the condition and pronounced staging. It includes toxic, leukopenic, and anginal-hemorrhagic stages.

Symptoms

The incubation period is from 2 to 6 weeks, and if you consume a large number of products from infected grains (more than 500 g), symptoms appear after a few days. The first signs of poisoning are nausea, vomiting, loose watery stools, weakness, decreased performance and fatigue. The described manifestations last about 3 days, followed by a period of leukopenia. Clinically, this condition manifests itself as an increase in weakness, malaise, drowsiness (this stage usually lasts 2-3, less often 6-8 weeks).

In the future, with the aggravation of toxic effects, the anginal-hemorrhagic stage occurs. Patients complain of high rises in body temperature (more than 39°C), stunning chills, the appearance of spot hemorrhages (petechiae) on the body, less often – ecchymosis; gingival and nasal bleeding. At the same time, there are severe pain when swallowing, an unpleasant putrid smell from the mouth, dirty gray deposits on the tonsils, in the oral cavity, larynx and pharynx. With the progression of the disease, phlegmons and abscesses of the skin, fiber and internal organs are formed.

Complications

Complications usually manifest with a lightning-fast course of food mycotoxicosis or in the anginal-hemorrhagic period of a typical variant of the disease. The most common of these are pneumonia, bronchitis, lung abscesses, soft tissue phlegmons, and osteomyelitis. In conditions of increasing immunosuppression, these conditions can lead to sepsis. In some cases, the appearance of necrotic changes in the distal parts of the limbs is observed. Deficiency of the blood coagulation system, damage to parenchymal organs (kidneys, liver, spleen) lead to the development of DIC-syndrome with a characteristic clinic of massive uncontrolled bleeding.

Diagnostics

If alimentary-toxic aleikia is suspected, consultation with an infectious disease specialist, otorhinolaryngologist, pulmonologist is mandatory; after the appearance of ulcers of the skin and internal organs, a surgeon is required. Laboratory and instrumental examinations are performed to identify the pathogen, assess the nature and severity of changes:

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- Clinical and biochemical blood tests. In the general blood test, pronounced leukopenia, thrombocytopenia, anemia, and accelerated ESR are observed. Biochemical parameters reflect an increase in the activity of AST, ALT, creatinine, and urea.
- Identification of infectious agents. Isolating fungi from human tissues and blood is a time-consuming and expensive process. Bacanalysis is performed by seeding the material on nutrient media. Detection of the toxin in grain is carried out using a biological sample (feeding laboratory pigeons with suspicious grain, skin test on rabbits), grain chromatography.
- Endoscopy of ENT organs. When examining the pharynx-pharyngoscopy-signs of catarrhal, necrotic or gangrenous sore throat are found. Typical deposits of dirty gray or brown color, passing from the tonsils to the mucous membrane of the larynx, pharynx, oral cavity; bleeding of the nasal and throat mucosa, formation of necrosis.

If signs of pneumonia appear, a chest X-ray is performed; for the purpose of differential diagnosis, ultrasound of the abdominal cavity, pelvis, retroperitoneal space, and lymph nodes is used. Differential diagnosis is carried out with diphtheria, sepsis, systemic mycoses, agranulocytosis, Simanovsky-Plaut-Vincent angina, poisoning with heavy metals, radioactive substances.

Treatment of alimentary-toxic aleikia

Therapeutic measures begin with the removal of poisoned foods from food, gastric lavage and a siphon enema (or taking a laxative). It is recommended to exclude alcohol, seasonings, fatty, fried, and cereal products from the diet, adhere to frequent fractional meals, and serve food at room temperature (to avoid additional injury to the mucous membranes). It is necessary to monitor the drinking regime, while it is preferable to use water or non-acidic juices. After each meal, it is recommended to rinse the mouth with antiseptic solutions (chlorhexidine, furacilin, calendula, sage, chamomile).

Treatment of alimentary toxic alaikia is usually inpatient, including detoxification therapy (glucose-salt solutions) and broad-spectrum antibiotics (penicillins, cephalosporins). With a prolonged course of pathology, it may be necessary to replace the loss of blood components: transfusion of red blood cells, platelet mass, and leukocyte concentrate is performed. There is evidence of a positive effect of the colony-stimulating factor on the course, severity and outcome of the disease. For the duration of treatment, it is recommended to reduce the dose of immunosuppressive drugs or cancel them if possible. When localized or spilled purulent processes appear, they are opened and drained, and local antibacterial agents (ointments, suspensions) are used.

Prognosis and prevention

With an uncomplicated course, the disease lasts approximately 3-4 weeks. With timely access to medical care, withdrawal from the use of contaminated products, the manifestations of mycotoxicosis tend to disappear on their own. With the appearance of hemorrhagic syndrome and necrotic changes in the tonsils, the proportion of deaths is 50-80%. However, for the occurrence of life-threatening conditions, it takes about two to three months of constant consumption of an infected cereal product, even against the background of obvious malaise. Timely hospitalization and a thorough epidemiological investigation reduce the risk of death and complications.

No specific prevention (vaccine) has been developed at this stage of medical development. To prevent contamination, the sanitary surveillance authorities conduct periodic grain inspections

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and explanatory work with the population. The sale and purchase of overwintered grain for the food needs of humans and animals is prohibited (delivery to distilleries is allowed). In the territory where sporadic cases of toxic aleikia have been recorded, grain products and raw materials from them are subject to disposal; the population should be examined for leukopenia.

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