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ETIOLOGY AND PATHOGENESIS OF AFLATOXICOSIS

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Annotation: Aflatoxicosis is an acute poisoning or chronic intoxication that develops as a result of eating foods containing mycotoxins of Aspergillus fungi. High doses of aflatoxins cause acute liver failure, coagulopathy, seizures; long-term use of mycotoxins in smaller doses can cause developmental delay in children, congenital anomalies in offspring, cirrhosis and liver cancer. The etiological diagnosis is established by backseeding food samples and biomaterial of the patient. Additionally, liver tests, hemostasiogram, and ultrasound of the liver are performed. Treatment – detoxification, hepatoprotectors, antimycotics, B vitamins.

Key words: detoxification, hepatoprotectors, antimycotics.

Aflatoxicosis is a mycotoxicosis caused by secondary metabolites of aflatoxigenic fungi. Aflatoxins (AFTS) were first isolated in 1961 from peanut flour contaminated with the mold fungus Aspergillus flavus. Subsequently, the symptom complex associated with the use of contaminated products was called aspergilloflavotoxicosis, or aflatoxicosis. The last major outbreak of aflatoxicosis with fatal outcomes was registered in Tanzania in 2016.Children show the greatest sensitivity to the pathogenic effects of aflatoxins.

Causes of aflatoxicosis
Sources of infection
Sources of aflatoxins are Aspergillus molds: A. Flavus, A. Parasiticus and some others. These fungi usually affect products of plant origin:

- cereals (corn, rice, wheat, sorghum);
- legumes (cocoa, soy, chickpeas);
- nuts (peanuts, almonds, hazelnuts, walnuts),
- spices (nutmeg, pepper);
- dried fruits (dried apricots, raisins, figs);
- products of processing of these crops (flour, butter, paste).

Aspergillus Flavus produces aflatoxins B1 and B2, A. parasiticus-B1, B2, G1, G2. In addition, both types of fungus can synthesize aflatoxins M1 and M2. The latter are also formed as a result of hydroxylation of mycotoxins B1 and B2 in the tissues of farm animals that have consumed fungal-contaminated feed, so they are often found in milk, dairy products, eggs, and meat.
The permissible concentration of aflatoxin B1 in plant products is 5 mcg/kg, aflatoxin M1 in animal products is 0.5 mcg/kg. Acute aflatoxicosis develops with daily intake of mycotoxin B1 in the amount of 20-120 mcg/kg of body weight per day for 1-3 weeks. AFTS are thermally stable, i.e. they are practically not destroyed during heat and culinary processing.
Infection routes

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Aflatoxicosis is considered mainly as an alimentary disease, since the main route of aflatoxins entering the body is food (with food, mother's milk). Transplacental transfer of mycotoxins from mother to fetus is possible. Employees of grain storage facilities, feed mills, flour mills, and farms may be exposed to aflatoxin inhalation or transdermal penetration. Cases of contamination with heroin aflatoxins have been reported, so in some cases, infection occurs with intravenous drug use.

Risk factors

Conditions that contribute to a higher incidence of aflatoxigenic fungi infestation in food crops are:

- subtropical and tropical climate (high temperature and humidity);
- adverse weather conditions for plant growth;
- plant damage caused by pests;
- violation of harvesting technology, grain transportation and storage conditions (lack of light and ventilation);
- lack of sanitary and epidemiological control over food storage.

Pathogenesis

Aflatoxins are absorbed in the small intestine, enter the liver with the bloodstream, where they undergo hydroxylation with the formation of metabolites (AFQ1, AFM1, aflatoxicol, 8,9-epoxy-AFB1, etc.). Aflatoxins form compounds with liver proteins (albumin, thrombin), block the synthesis of many enzymes, polypeptides and nucleic acids. Their excretion occurs with bile, feces, and urine. Aflatoxin B1 is the strongest hepatotropic poison and one of the most dangerous carcinogens for humans. Its secondary metabolites bind to the nitrogenous bases of DNA (in particular, guanine), activating mutations of the tumor suppressor gene TP53 in codon 249.

Under the action of AFT in the liver, hepatocyte dystrophy, the formation of foci of fat and coagulation necrosis, cholangiolar proliferation, and intrahepatic cholestasis occur. Necrotic changes are also found in the heart muscle, spleen, and kidneys. With aflatoxicosis, vitamin D metabolism is disrupted, prothrombin formation occurs, cellular and humoral immunity is suppressed, and oncogenes are activated. Aflatoxins also have mutagenic, embryotoxic and teratogenic effects.

Symptoms of aflatoxicosis

Acute poisoning

Signs of acute aflatoxicosis manifest within half an hour after ingestion of contaminated food. There is general weakness, lethargy, dizziness, short-term fever. There is abdominal pain, vomiting, upset stools. Worries about heaviness and pain in the liver, the skin and sclera become jaundiced. Symptoms of neurointoxication include ataxia, seizures, and paresis.

Subcutaneous hemorrhages and spontaneous bleeding occur, with acute aflatoxicosis, hepatosplenomegaly, edema, ascites quickly progress. If emergency care is not provided, patients fall into a coma, death occurs within a few days from acute liver or kidney failure, brain edema.

Chronic aflatoxicosis

The long-term cumulative effect of subtoxic doses of aflatoxins leads to chronic liver damage with the development of cirrhosis, portal hypertension, and hepatocellular carcinoma. Against the background of dyspepsia, children have protein and energy insufficiency, delayed physical development and growth, and adults have progressive weight loss.

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Complications

Aflatoxicosis is associated with the development of several types of cancer, in particular, cholangiocarcinoma, primary cancer of the liver, lungs, and intestines. The risk of developing hepatocellular carcinoma is 25-30 times higher when aflatoxicosis is combined with viral hepatitis B or C. It is believed that aflatoxins are involved in the pathogenesis of kwashiorkor and Reye's syndrome in children.

The course of aflatoxicosis can be aggravated by the presence of other mycotoxins (for example, fumonisins) in one product, with the simultaneous development of combined mycotoxicoses. Mortality from aflatoxicosis is high, especially in the pediatric population.

Diagnostics

Diagnosis of aflatoxicosis is based on the establishment of a link between poisoning and the use of products contaminated with mycotoxin. This requires a detailed nutritional history and a thorough study of clinical and laboratory data. It is necessary to remove food products that are suspected of causing poisoning, their special study (organoleptic, mycological, luminescent, chromatographic analysis, bioassays)in a sanitary laboratory. Examination of the victim includes:

- **Detection of aflatoxins in the body.** The concentration of AFT metabolites is determined in urine, blood plasma, and breast milk. For microbiological isolation of the producing fungus, food residues, feces, and vomit are used.
- **Blood tests.** UAC, liver enzymes, coagulogram, vitamin D levels, and electrolytes are examined. Aflatoxicosis is characterized by an increase in the level of transaminases, bilirubin, and an increase in prothrombin time.
- **Other studies.** To assess the degree of damage to internal organs, ultrasound of the liver, spleen, kidneys, and ECG registration are performed. If necessary, perform a liver biopsy with the morphology of the biopsy.

Differential diagnosis

As part of differential diagnosis, other mycotoxicoses (fusariotoxicoses), as well as food toxicoses of other etiologies, are excluded.:

- salmonellosis;
- staphylococcal food poisoning;
- botulism;
- viral gastroenteritis;
- poisoning with organochlorine, organophosphate compounds, pesticides, etc.

Treatment of aflatoxicosis

Like any acute food poisoning, aspergilloflavotoxicosis requires urgent care. There are no specific antidotes. At the first stage, to remove the maximum amount of mycotoxins, gastric lavage is performed, a siphon enema is set up, saline laxatives are introduced through a probe, and activated carbon suspensions are added. To detoxify the body, enterosorbents are prescribed, forced diuresis is performed, according to indications – hemo- / plasma sorption, albumin dialysis.

Routine therapy for aflatoxicosis includes taking antimycotic drugs that are active against Aspergillus, immunomodulators, hepatoprotectors, vitamin and mineral complexes, and probiotic bacteria.

Prognosis and prevention

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Aflatoxicosis is dangerous with severe irreversible damage to the liver and nervous system, a high risk of death, and serious unforeseen consequences. Outbreaks of acute aflatoxicosis are regularly reported in Asia and Africa, and both adults and children are victims. In addition, these regions have a high percentage of deaths from cirrhosis of the liver and liver cell cancer.

Measures to combat aflatoxicosis include prevention of contamination of agricultural products with mold fungi at all stages of their cultivation, processing, and storage. For this purpose, breeding is carried out, genetic engineering methods are introduced, temperature and humidity control is provided in warehouses, chemical decontamination is performed. Before preparing and consuming products, it is necessary to carefully inspect them for the presence of mold fungi, do not use products of questionable quality, expired shelf life, or violation of the integrity of the package.

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