THE COURSE OF CHOLERA IN CHILDREN, WAYS OF TRANSMISSION AND TREATMENT

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Annotation: *In this article, the problem of acute infectious intestinal diseases (AIID) remains one of the main and urgent problems of modern medicine. Difficulties in early diagnosis in children, recurrence of the disease, chronic state of the disease and low life expectancy of patients are important for pediatric medicine. Timely detection and early diagnosis of OYUIK, research of the clinical course and laboratory tests of the disease, are necessary for treatment and prevention of the disease.*

Key words: *infectious, cholera disease, symptoms, cause, group, adhesion, invasion, toxin production, intracellular replication*

Cholera is an acute infectious disease characterized by general symptoms of intoxication and inflammation of the gastrointestinal tract, especially the colon.

Etiology. Shigella, belonging to the Enterobacteriaceae group, is the causative agent of shingles. Shigella are rod-shaped, gram negative, non-motile, do not form spores and capsules. S. dysenteriae, S. flexneri, S. boydii, S. sonnei types are distinguished. There are different biochemical and serological variants of the causative agents (Grigoryev-Shig, Lordge-Sax, Schmitz-Stuser, etc.). Each type of Shigella has serovars (1-15) and subserovars (1a,b; 2a 2b; 3a 3b and others). Pathogenicity of pathogens consists of 4 main factors: adhesion, invasion, toxin production and intracellular replication abilities. S. dysenteriae bacteria produce exotoxin, and other microbes produce endotoxin. Toxins are of great importance in the pathogenesis of the disease and in the manifestation of clinical symptoms. The resistance of intestinal bacteria in the external environment is different. If S. sonnei has high resistance (up to 3 months in water, up to 10 days in milk), S. dysenteriae has medium resistance (up to 3 days in water and milk). Shigella die quickly under the influence of sunlight, high temperature and disinfectants.

Epidemiology. The source of the disease is the sick person, carriers. A sick person begins to excrete bacteria through feces from the first hours of the disease. The transmission mechanism is fecal-oral. Contaminated food products (milk, dairy products, vegetables), household contact (contaminated dishes, clothes, toys), water is transmitted. Transmission through food products is mainly due to S.sonne bacteria, transmission through contaminated water is mainly due to S. flexneri pathogen, household-contact transmission methods are specific for S. flexneri and S.sonne.

Immunity is typospecific in patients with acute type of hives. In patients, the disease may have a prolonged and chronic course. Being healthy people, carriers do not seek medical help, do not receive treatment, often do not go to the hospital and cause a wide spread of the disease. Summer-autumn seasonality is typical for Ichburug. Children of kindergarten and school age are prone to the disease.

Pathogenesis and pathomorphology. Shigella enters the human body through the mouth. The acid in the gastric juice removes the irritant and does not allow it to enter the intestine. But if the acidity level of the gastric juice is low, or if a large amount of the causative agent enters the body at once, some bacteria quickly break through the physiological barrier in the stomach and enter the intestine. There, they are affected by digestive juices and intestinal flora. In this case, some of the drivers will die. Enteric bacteria do not shoot outside the intestinal wall. When bacteria die, toxins are released from them. Toxins damage the epithelium of the mucous membrane of OIT, have a sensitizing effect on it. The amount of endotoxin increases in the intestine and slowly enters the blood and lymph vessels. Toxins circulating in the blood affect various tissues, organs and nervous system. Increased hypoxia in tissues and development of metabolic acidosis. As a result, primary clinical symptoms of the disease (increased body temperature, headache, shivering, loss of appetite, weakness) appear. Bacteria affect the nerve endings located in the sigmoid part of the large intestine. These parts of the colon are shortened. Clinically, false alarms appear. In ulcer disease, morphological changes develop mostly in the distal part of the colon. These changes are expressed by the appearance of catarrhal, fibrinous-necrotic and ulcers. In recent years, mainly symptoms of catarrhal inflammation appear. Then the mucous membrane of the wall of the large intestine becomes red, light hemorrhages, sometimes erosions appear. Mucous or blood-tinged fluid is found in the intestine. Often, solitary follicles in the mucous and submucosal layers of the intestinal wall swell during the process. Sometimes, fibrin accumulates in the inner parts of the intestinal wall. Focal necrotic changes are observed in mucous membranes and follicles. Ulcers appear. The disease begins acutely. The patient's body temperature rises to 380-390C, headache, weakness, abdominal pain appear. Intestinal disorders become more and more pronounced day by day. Attacking pains in the abdomen, often in the lower part or on the left side. The sigmoid colon is palpated in the form of a thickened rope and pain is detected. Tenesmus and false calls are observed. Feces can be small, mucous, bloody, similar to "rectal sputum".

In the subclinical form, there are no clinical symptoms, the disease is diagnosed by bacteriological or serological methods. According to epidemiological instructions, patients are found during the examination of the source of infection. In its hypertoxic form, the symptoms of intoxication are evident. The patient faints, the temperature rises to 39-400C, signs of neurotoxicosis appear, infectious-toxic shock is observed, the patient becomes confused, coma occurs. In the relapsing course of the disease, there are periods of exacerbation and reduction of the pathological process. The period of

exacerbation of the disease is characterized by restlessness, sleep disturbances, abdominal pain, increased frequency of constipation, and weight loss. The Song boom period alternates with the countdown period. The duration of remission can last from 1 month to 3-6 months. Patients with a chronic form and bacterial isolates are considered very dangerous. It is caused by the presence of concomitant diseases, poor quality nutrition, hypovitaminosis, worm infestation, stomach and intestinal secretory activity disorders. In the chronic form of goiter, S. flexner bacteria are often detected in feces. Complications. Specific complications associated with ileus infection include rectal mucosal prolapse, intestinal intussusception, anal fissure, DVS-syndrome, infectious-toxic and hypovolemic shock. Intestinal rupture and bleeding are rare. Non-specific complications are mainly associated with the addition of ORVI and changes in SHPM activity. Intestinal dysbacteriosis, otitis, zotiljam, pyelonephritis are often observed.

Prevention and anti-epidemic measures. In the fight against infectious diseases, the following measures are implemented: early diagnosis, isolation of the patient, sending an urgent notification to the SES, measures related to sanitation and hygiene, and final disinfection in the hearth. Patients with acute diarrhea should undergo a bacteriological examination of feces once after clinical recovery. Children who are chronic carriers of gonorrhea are not allowed to attend kindergarten. Medical observation of those in contact will last for 7 days (every day they will be asked about their health, a smear test, thermometry, diarrhea). Prevention with phages is recommended. Bacterial carriage is formed when patients who have had a bowel movement excrete their bacteria. They will be under the supervision of a polyclinic doctor for 3 months, they will undergo a bacteriological examination once a month. Carriers of bacteria do not go to kindergarten or school.

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