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PATHOGENETIC PRINCIPLES OF ACUTE INFECTIOUS INTESTINAL INFECTIONS AND FEATURES OF CLINICAL COURSE AMONG CHILDREN OF DIFFERENT AGES

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Abstract. This article presents the pathogenetic aspects of the diarrhea syndrome that occurs in acute intestinal infections and the characteristics of the clinical course in children of different ages.

Key words: acute intestinal infections, salmonellosis, dysentery, children, pathogenesis, clinic.

Relevance of the problem. 90-95% of the diseases associated with diarrhea syndrome correspond to acute intestinal infections and acute intestinal diseases. As a result of increasing temperature during summer time in the territory of our republic, favorable conditions are created for the survival and reproduction of pathogens that cause acute intestinal infections. During this period, there is an increase in the variety of various food items like fruits and vegetables, unfortunately it sometimes becomes the main reasons for the spread of these pathogens.

Absorption of water in the intestines is related to the absorption of electrolytes. It is carried out with the help of villi epithelium, that is, enterocytes and colonocytes. In the small intestine, water and electrolytes are transported passively, and in the large intestine, they are absorbed by the active sodium pump, against the chemical concentration balance. Glucose and amino acids enhance the absorption of water and electrolytes. The amount of water absorbed in the large intestine is 5 liters per day. If more water enters the large intestine, it causes diarrhea. The clinical course of diarrhea indicates a violation of the absorption of water and electrolytes in the intestines. There are 4 types of diarrheas, which are based on different mechanisms of pathogenicity. Diarrhea of one or another type is characteristic of every intestinal disease.

Secretory diarrhea. In this type of diarrhea there is an increased secretion of sodium and water in the intestinal cavity. For example, diarrhea in cholera. Exotoxin enters enterocytes through the receptor zone and activates adenylate cyclase, which increases the synthesis of cyclic 3-5- adenosine monophosphate (tsAMF). This, in turn, increases the secretion of electrolytes and water by enterocytes in a constant ratio (5 g of sodium chloride, 4 g of sodium bicarbonate

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and 1 g of potassium chloride in 1 liter of excretion. The main place is occupied by prostaglandins that stimulate the synthesis of tsAMF. Also, secretory diarrhea occurs again in salmonellosis, terminal ileitis, post cholecystectomy syndrome Secretory diarrhea occurs under the influence of highly active peptides, serotonin, calcitonin, secretin, free and chain fatty acids. Character of feces: watery, sometimes green.

Hyperosmolar diarrhea. Diarrhea of this type occurs in malabsorption syndrome, disaccharide deficiency and by taking saline laxatives. The osmotic pressure in the fecal mass is higher than of osmotic pressure in the blood plasma. Unwanted wastes are abundant in it with liquid and undigested nutrients.

Hyper and hypokinetic diarrhea. It causes a transient disturbance of the mass in the intestines. This type of diarrhea is a result of decreased or increased intestinal motor function. Neurosis is observed on taking laxatives and antacids. The osmotic pressure in the waste mass corresponds to the osmotic pressure in the blood plasma. In this type of diarrhea, the stool is liquid, porridge-like, and its quantity is small.

Hyper exudative diarrhea. The cause of this type of diarrhea is the release of blood, mucus, plasma protein into the intestinal cavity. This type of diarrhea occurs in intestinal inflammatory diseases (salmonellosis, dysentery), nonspecific ulcerative colitis, Crohn's disease, intestinal tuberculosis, lymphoma, carcinoma and intestinal ischemic diseases. In this case, the osmotic pressure of the waste mass is higher than the osmotic pressure of the blood plasma. The waste is liquid, a mixture of deposits and pus.

It is known that the pathogenesis of infectious acute intestinal infections is a complex and still unsolved. It is related to a number of factors, the interaction of which determines one or another level of expression of the infectious process. These factors are implemented by the characteristic of the pathogen (pathogenicity of pathogens and their toxins for the macro-organism and foreign antigen), and on the other hand, through the protective factors of the macro-organism. According to most researchers , the following mechanisms are distinguished in the pathogenesis of infectious UTIs: increased intestinal secretion, inhibition of active transport of ions from the intestine, impaired permeability of the intestinal wall, and impaired intestinal motility. enterotoxigenic and entero-invasive types are distinguished.In the initial mechanism, bacteria do not digest enterocytes, in this case, strong enterotoxins are the cause of the disease, which disrupt the transport of ions and water through the membrane of intestinal epithelial cells by activating adenylate cyclase. Invasive microorganisms also affect the absorption of electrolytes and water from the small intestine, so this mechanism of diarrhea does not result in

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fluid loss in the feces, but most invasive bacteria are capable of synthesizing enterotoxins.Therefore, in such cases, the mechanism of diarrhea has a mixed character.

In salmonellosis, the causative agent enters the lymph and blood through the lymphoid ring of the larynx, and then spreads throughout the internal organs. A small amount of Salmonella can also enter the lungs via aspiration and blood, where it remains inside the cytoplasm of alveolar macrophages for a certain period of time . Their main mass enters the intestines and immediately enters the tissues of the 12-finger and small intestines, as well as the blood and lymph vessels of these organs. Most of the salmonella left in the intestinal cavity either die or excreted with feces. In the generalization of the process, the accumulation and multiplication of the pathogen occurs in the intestine, mesenteric nodes, spleen and liver. This process is significantly facilitated by the ability of microbes to parasitize in the cytoplasm of macrophages .

It has been proved that the basis of the pathogenesis of dysentery is the parasitism of its causative agents in the epithelium of the mucous membrane of the large intestine. Some enteric pathogens, such as Escherichia, can interact with the human body in the same way as Salmonella and Shigella . The important toxic effects of bacterial lipopolysaccharides include their entry into leukocytes, resulting in their degranulation. Epidemic processes and clinical course of intestinal infections have changed in recent years, and as a result, their diagnosis has become more difficult. For example, salmonellosis, dysentery, typhoid-paratyphoid diseases, and many other intestinal infections are now characterized by a rather mild course, a decrease in the number of severe forms and the degree of intoxication, and a significant reduction in relapses and lethal cases .

The course of dysentery in newborns and children in the first weeks of life is of particular concern because they are very sensitive to this disease due to the reactive properties of the organism. Dysentery at this age often begins slowly: children become lethargic, do not latch on to the breast and suck less. Firstly, the character of stool changes, then after 2-3 days defecation accelerates up to 8-10 times a day with the increase of mucous, bluish-colored mixture, sometimes with the appearance of blood streaks. The child is especially restless during feeding. There is redness around the anus and buttocks, the anus becomes loose. A subfebrile condition is noted in some children. In some rare cases, dysentery in babies begins very acutely: from the first day of the disease, the stool is usually subfebrile, rarely develops with an acceleration of 10-25 times against the background of a high temperature of up to 390. At first, stools are mixed with mucus, bluish color, later with pus and streaks of blood. Deep redness around the

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anus and buttocks, causing rectal prolapse in some children. Feces may normalize within 10-15 days. Despite the severity of the disease, most children's weight not only decreases but can also increase. Sometimes, dysentery in babies can occur in a subclinical form .

Toxicosis begins gradually like primary dysenteric intoxication in older children, and is accompanied by symptoms of dehydration. It seems that the absence of toxic forms of dysentery in infants should be explained by this, and the severity of the disease is related to the destruction of the metabolism. The development of intestinal toxicosis is also explained by the anatomic and physiological characteristics of young children . Along with symptoms of toxicosis and exsiccosis, colitic syndrome is also expressed, but not as the same extent as in older children, which is probably due to the lack of formation of the autonomic nervous system. Feces are usually scanty and their character is maintained, mixed with mucus and sometimes with streaks of blood. The number of stools reaches 8-15 times. Tenesmus equivalents in the form of flushing of the face, relaxation or opening of the anus are observed during agitation, crying, defecation .

Propensity for protracted course, general and secondary, frequent complications due to infection, higher percentage of lethality, as well as the occurrence of secondary toxicosis are characteristic feature of dysentery in early childhood .

Secondary toxicoses can be observed in any form of dysentery. Various factors are of infectious and non-infectious nature depends on the allergic background of the body and the history of significant disruption of metabolism could be the reasons of secondary toxicoses. Secondary toxicoses, which usually appear in the 2-4th week after the onset of the disease, significantly worsens the child's condition and can lead to a fatal outcome. Often they are acute, sometimes develop slowly against the background of a satisfactory condition during the recovery period after dysentery. In the acute development of secondary toxicosis, repeated vomiting occurs, the child refuses to eat and drink and the stool often becomes liquid, abundant and scattered. Exycosis increases rapidly, weight decreases drastically and the baby can go into serious condition. The edges of the face become sharper, the skin becomes gray and dry, the mucous membranes dry up, the subcutaneous fat layer swells, and the limbs become cold. Cardiovascular activity decreases (tachycardia, marked hypotension, muffled heart sounds), unconsciousness, convulsions may occur . The severity of the disease depends not only on the age of the child, but also on the nature of feeding, premorbid background, the presence of concomitant diseases and the type of the pathogen. Climatic-geographical conditions, lifestyle of a person, nature of eating,

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availability of vitamins in food and other factors contribute to dysentery. In areas with hot, dry summers, dysentery is more pronounced and more severe .

The course of dysentery also depends on the type of causative agent, for example, the disease caused by Sh. dysenteriae is characterized by a severe course with expressed hemocolith syndrome, 10 - 50% mortality during epidemics. When it provokes Sh. The disease caused by Sh. sonnei has a rapid onset, similar to food intoxication, and then is short-term and mild, rarely turning into a chronic form .

Salmonellosis disease in children is clinically manifested with complete polymorphism. It depends on intoxication and series of pathological processes that occur in various organs and systems. A single serotype of Salmonella can cause symptoms ranging from mild to severe form. Undoubtedly, the clinical manifestation and intensity of the symptoms of the disease depend on the interaction between the infectious microbe and the child's body, that is, the amount of the microbe, the route and landscape of the infection, the biological characteristics of the pathogen, the child's premorbid condition, the child's immune strength, age, and diet.

Gastrointestinal manifestations of salmonellosis are more common among children: according to some researchers, this clinical manifestation can occur in 43-93 % cases. This type of disease begins with acute dysfunction of the gastrointestinal tract, to which cases of general intoxication of the body are added. At the beginning of the disease these signs and symptoms can be acknowledged: Swelling of Patient’s face, rise in temperature, headache, paleness, stomach aches, nausea and vomiting. In the special form of gastritis, the stool of the patient is normal, the temperature rises, the head hurts, the body relaxes, and the body tingles. Intestinal manifestations of salmonellosis are common among diseases such as gastroenteritis and enteritis. In this type of disease, at the same time as vomiting or a few hours after vomiting, the abdomen swells and is accompanied with occasional diffuse pain. When palpating the abdomen, 83% of patients have pain in the stomach area, around the navel, and in the iliac region, sometimes along the colon. At the same time, the patient has frequent bowel movements, and stools are large, frothy liquid, slimy mixture. It is noted that vomiting starts before diarrhea in 60% of patients. It is recognized that 19.2% of patients vomited once, 30% 2-3 times, and 50% many times.

In the case of gastroenteritis, enteritis, stool is watery, bluish, without pathological impurities, and has an odor when onset is acute. Frequency of defecation reaches 5 to 20 times a day. Sometimes the feces are liquid and resemble the color of mud. In very severe types of the disease, stools resemble "boiled rice" due to persistent diarrhea. There are also clinical variants (colitis,

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enterocolitis, gastroenterocolitis) that occur when the large intestine is damaged. 40% of one-year-old children have signs of hemocolitis. The dyspeptic variant of the disease was observed in children under one year old and often suffering from concomitant diseases: in such patients, the temperature rises up to 39.0 degrees, vomiting, belching, flatulence, 5-7 stools per day is normal but contains more amount of liquid consisting of undigested nutrients and mucus.

In the gastrointestinal form of salmonellosis, the intestinal symptoms of the disease last from several days to 2-3 weeks, and in children under the age of one year, they can last up to a month or even more. In the mild form of the disease, short-term dyspeptic disorders are observed, there are almost no signs of general intoxication, and in the severe form death can occur. The temperature curve has an irregular wavy character, sometimes rising and sometimes falling, corresponding to the subfebrile period.

It should be noted that symptoms of intoxication are one of the characteristic features of salmonella disease. In this case, the color of the patient becomes pale, the liver becomes enlarged and skin may turn yellow due to the development of toxic hepatitis. The spleen is enlarged and kidneys can also be affected.

The typhoid variant of salmonellosis is less frequent (up to 5.0-20%) than the gastrointestinal variant in children, and is often severe and moderately severe. In this condition, general intoxication plays an important role that ensures a clearer manifestation of the disease. The disease often begins slowly, its symptoms increase over 2-4 days. The temperature rises significantly and intoxication increases. The child becomes apathetic, has a headache, sleep is disturbed, there is a loss of appetite and child does not feel energetic.

The septic variant of the disease occurs mainly in infants, more often in new and premature babies and in children with a premorbid background. Usually, the disease is severe and leaves serious complications, often leading to the death of the child.

coli infection in children is clinically severe with symptoms of gastrointestinal tract dysfunction and general intoxication. Toxic syndrome is characterized by a long, severe course and in some cases death can occur. Coli infection in newborns is mainly characterized by a mild course, but there may also be severe forms with a toxic-septic component and lethal consequences, depending on the premorbid background and the type of the pathogen.

In children of the first six months of life, especially with an unfavorable premorbid background and early artificial feeding, the course of the disease is characterized by toxicosis and exicosis syndrome, rapid weight loss, severe vomiting, pale-yellow watery stools, flatulence, and sometimes intestinal paresis.

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Disease pneumonia , urinary tract complications such as infections, toxic myocarditis. Despite intensive treatment, the percentage of lethal consequences is high.

Conclusion As it is known from the above, significant progress has been made in the interpretation of the pathogenesis of acute intestinal infections, but this problem requires further study. Also, the clinical course of acute intestinal infections is manifested differently in different age groups, in addition, the course of the disease acquires its own characteristics under the influence of various external and internal factors, it is necessary to thoroughly study these diseases in our region and draw logical conclusions, which in turn, plays an important role in the development of methods of treatment and prevention of diarrheal diseases.

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