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Current State of the Problem Giardiasis in Children

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Abstract: Giardiasis is a serious problem in modern pediatrics. Its relevance is due to both the high prevalence of this type of protozoa and the polymorphism of clinical manifestations, the complexity of diagnosis and ambiguous approaches to treatment.

The review examines modern aspects of the diagnostic search and therapeutic options for giardiasis in children. It is currently known that about 500 parasites can exist in the human body, including more than 15 species of protozoa. The undoubted leader in prevalence among the entire group of protozoa in childhood is Giardia. The polymorphism of the clinical picture of giardiasis, certain difficulties in diagnosis, ambiguity of approaches to treatment and often dissatisfaction with its results determine the interest in this topic among doctors of various specialties.

Keywords: giardiasis; children; intestinal microbiota; lactase deficiency; diarrhea syndrome; antigiardiasis drugs.

Members of the genus Lamblia are currently classified into 3 species based on morphological criteria: L. muris, L. agilis and L. intestinalis. In turn, among L. intestinalis there are about a dozen subspecies. L. muris infects mainly rodents, birds and reptiles, and L. agilis is found only in amphibians. The reservoir of L. intestinalis infection can be humans, dogs, cats, and beavers.

Various strains of L. intestinalis have now been typed using isoenzyme analysis, polymerase chain reaction (PCR), in vitro excystation test and culture method. Eight different genotypes have been identified, only two of which, strains A and B, are capable of infecting humans.

The data obtained cast doubt on the possibility of human infection with G. intestinalis from animals. In other words, Giardia is species-specific and the source of invasion is a person who secretes mature cysts.

The routes of infection are fecal-oral and contact-household. Our children's bad habits of eating unwashed fruits and vegetables, biting their nails, holding a pencil in their mouth, and not washing their hands after a walk or before eating are the reason for the high prevalence of giardiasis in the child population. Infection often occurs by drinking water and food containing cysts.

Cysts remain viable in water for up to 86 days, in soil for up to 75 days. They can be stored on glass, metal, cardboard, polymers for up to 20 days, on a common towel - up to two days, in products - about a day. Cysts are resistant to UV irradiation, but die when boiled after 5 minutes. and when frozen. Disinfectants act only in concentrations that are 5–10 times higher than those usually used.

People's susceptibility to infection varies. The leading role in the formation of resistance to infection is given to the barrier function of the mucous membrane of the small intestine, the state of local and activity of cellular immunity. It is the state of the intestinal microflora, which is largely determined by immune mechanisms, that explains why some people are resistant to the effects of parasites.

In the development of the disease, and most importantly, in the severity of its clinical manifestations, both the role of the host (its immune status, nutritional status and age) and the parasite itself (virulence and pathogenicity of strains) are important.

The incubation period for primary invasion is 1–4 weeks. The period of cyst release begins on the 9th–12th day after infection and can last for months. It alternates with periods of subsidence of the process (from 1 to 17 days), when cysts are not released. The latter provision explains frequent diagnostic failures during a single examination using coproscopy.

Cysts are a non-motile form. They are capable of division and are acid-resistant. When they enter the stomach, they are not destroyed and move into the duodenum, where from each cyst 2 vegetative forms are formed, which are attached to the villi of the epithelium of the duodenum and proximal jejunum. In a microscope, under the cyst shell, 2 or 4 nuclei and a coiled flagellar apparatus of Giardia are distinguished.

In the upper parts of the small intestine, cysts transform into vegetative forms (trophozoites). These forms are active, mobile, and pear-shaped. The length of the trophozoite is 9–21 μm, and there is a suction disc on the anterior surface. The formation of vegetative forms from cysts occurs in 10–12 minutes, their doubling occurs every 9–12 hours. With the help of a suction disc they are held on the mucous membrane of the duodenum, where they receive the necessary food and reproduce by division. In the colon, the reverse transformation of vegetative forms into cysts occurs, which are excreted in the feces. The formation of cysts from vegetative forms occurs within 12–14 hours. In most cases, cysts are released into the external environment, however, in some patients, especially with diarrhea syndrome, vegetative forms may also be released. Trophozoites persist in the external environment for 30–60 minutes, cysts - from 9 to 123 days (depending on temperature and humidity).

The body of the trophozoite is covered with a cytoplasmic membrane, under which vacuole-like formations—pinocytic (digestive) vacuoles—are found on the dorsal surface. Due to the presence of devices for communication with the villi of the small intestine (suction disk, groove of the caudal part of the body with central cords), vegetative forms exist in close contact with the villi of the brush border of the small intestine. The central harnesses function as a “pump”, pumping out liquid from under the dome of the suction disk. Having attached to the brush border, trophozoites pump out the contents of the spaces between the villi.

The vital activity of Giardia in the host's intestine depends on the intensity of membrane digestion. This is confirmed by the fact of the predominance of Giardia in children and young animals, as well as the peculiarity of the distribution of Giardia in the intestine, due to the gradient of digestive activity.

The question of the presence of Giardia in the biliary tract remains controversial. The results of many studies show that Giardia lives in the gallbladder and bile ducts only when bile loses its bactericidal properties, as a result of which persistence of parasites in a pathologically altered gallbladder is possible.

There is an opinion that Giardia does not live in an intact gallbladder and ducts, and therefore cannot serve as a primary etiological factor in the development of diseases of the hepatobiliary zone. Damage to the biliary tract with giardiasis is usually secondary. This is facilitated, on the one hand, by duodenal dyskinesia, and on the other hand by severe dysfunction of the sphincter of Oddi. In both cases, the key mechanism is the entry of Giardia into the biliary system due to duodenobiliary reflux.

When vegetative forms attach to the villi of the duodenum, the latter are traumatized, which is accompanied by frequent changes of epithelium with the replacement of mature ones by functionally immature enterocytes. As a result, the absorption of digestive substrates and the processes of parietal digestion are disrupted (the activity of disaccharidases, in particular lactase, is reduced), the microbial landscape of the intestine changes, the production of secretory Ig A decreases. These disorders are often accompanied by the development of diarrheal syndrome (secretory or osmolar diarrhea).

Long-term persistence of Giardia leads to disruption of the metabolism of proteins, fats, carbohydrates, vitamins and microelements. The following factors are involved in the implementation of these disorders: a decrease in the content of enterokinase and alkaline phosphatase in the duodenal contents → worsening enzymatic deficiency → diarrhea, flatulence; development of motor-evacuation disorders in the intestines, biliary system and pancreas → worsening enzyme deficiency → diarrhea, flatulence; change in the microbial landscape in the intestine → diarrhea; toxic effects on the glycocalyx of the small intestine → diarrhea; sensitization of the body → allergic manifestations.

As a result of the impact on the nerve endings of the intestinal wall, viscero-visceral reflexes arise, which leads to the appearance of abdominal pain syndrome.

According to E. A. Kornienko and S. N. Minina, morphologically, giardiasis is always accompanied by chronic duodenitis, half of the children show signs of villous atrophy, and a third have deepening of the crypts; in almost all cases there is an increase in the number of interepithelial lymphocytes, dense lymphoplasmacytic infiltration with an admixture of neutrophils and eosinophils. A similar nature of changes is described in the works of other authors.

According to foreign authors, inflammatory changes in the duodenum occur in all examined patients. The inflammation is predominantly eosinophilic in nature, and the presence of clear atrophic changes requires excluding other pathologies, including celiac disease.

An important role is played by the immunological characteristics of the host organism and the nature of the immune response to parasitic invasion. The inflammatory reaction in the mucous membrane of the small intestine during giardiasis is characterized by a predominant increase in anti-inflammatory cytokines.

Perhaps the prevalence of the Th2 response characteristic of giardiasis explains the tendency of patients to allergic reactions, as well as eosinophilic infiltration of the mucous membrane of the small intestine.

An insufficient pro-inflammatory Th1 response in combination with a decrease in secretory IgA levels likely contributes to the long-term chronic course of giardiasis.

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