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GIARDIASIS IN CHILDREN AND ITS SIGNIFICANCE IN THE ETIOPATHOGENESIS OF DISEASES IN CHILDREN

Ibatova Sh.M.,
Mamatkulova F.Kh.,
Normakhmatov B.B.
Samarkand State Medical University, Samarkand, Uzbekistan

ABSTRACT	***KEYWORDS***
Giardiasis is one of the most common helminthic infestations in the world. The problem of giardiasis is also very relevant for Uzbekistan. In Uzbekistan, among parasitic diseases, giardiasis accounts for a large proportion (60-70%) of children in organized children's groups [1-5]. The discussion about the pathogenicity of Giardia, which lasted for a long time, can be said to have ended with the recognition of their damaging effect on the villi of the intestinal epithelium, where they vegetate [10]. According to the conclusion of the WHO expert group, Lamblia intestinalis should be considered a pathogenic protozoan [6-9].	

According to the World Health Organization (WHO), giardiasis affects approximately 20-25% of children in the world. Giardia ranks third in prevalence after enterobiasis and ascariasis (WHO, 2006). Previously, it was believed that giardiasis occurs in endemic areas of Asia, Africa, Latin America with poorly developed infrastructure. In connection with the development of tourism in developing countries, giardiasis is found everywhere, often together with pathogens of intestinal infections and helminthiases, such as Hymenolepis nana, Strongyloides stercoralis, Taenia spp. etc.

Infection with giardiasis occurs through the alimentary route. Transmission factors are hands, water, food, and household items contaminated with faeces containing Giardia cysts [11,18]. The clinical manifestations of giardiasis are varied, but lesions of the gastrointestinal tract prevail, which is associated with the localization of parasites in the duodenum and jejunum [12,15,17,20].

At the same time, infested patients often experience nausea, heartburn, belching, vomiting, cramping pains in the abdomen, a decrease in acidity up to complete achylia [13,16,19].

Giardiasis with severe clinical manifestations of intestinal damage, according to Pashinsky E.S. [2018] is about 40%. This group includes patients with acute and chronic duodenitis, acute and chronic enterocolitis. With duodenitis of giardia etiology, the persistence of the pain syndrome, flatulence, rumbling in the abdomen and unstable stools are noted [6-9].

Asylgareeva, G. M., Kobiakov, A. I., Mavziutov, A. R [2015] in a study of 214 patients aged 15 to 68 years old, affected by Lamblia intestinalis, duodenitis was established in 93 patients (43.4%).

In the conditions of our region, the clinical picture of intestinal giardiasis was studied by Inoyatova F.I., Nurmatova N.F. The authors studied 265 patients, duodenitis was detected in 40.5%, enteritis in 36.5%, enterocolitis in 23.0%.

Giardia lesion of the small intestine is characterized by constant moderate pain in the umbilical region, a feeling of stomach fullness even after a small portion of food, bloating, liquid, sometimes foamy stools from 3 to 10 times a day, pale skin [14]

In giardiasis enterocolitis, Kornienko, E.A., Minina [2009] note severe pain in the navel and somewhat less pain along the colon. The frequency of stool is on average 5 times a day. Tenesmus is rare. Chronically flowing giardiasis enterocolitis give frequent relapses.

Kaidanek, T.V., Mukhametzianov, A.M., [2014] observed atrophic processes and eosinophilic infiltration of Lamblia propria in the intestines of mice infested with Giardia against the background of catarrhal inflammation.

Romero, D., Rosales, M.J. [2014], examining the biopsy material of patients with giardiasis, found that changes in the intestine ranged from infiltration of the intestinal wall with mononuclear cells to severe changes resembling celiac disease (villous atrophy, increased mitosis, crypt expansion).

Seshadri V., Martens C. [2010], subjected to electron microscopic examination of biopsy specimens of the small intestine of patients with giardiasis, revealed violations of the brush border of the villi. Supaluk Popruk, Kanthinich Thima [2011] found a decrease in the height of enterocytes, cellular infiltration, areas of villous atrophy in biopsies of the small intestine of patients with giardiasis. Similar results were obtained by Saghaug C.S., Sørnes, S. [2015].

Minina S.N. [2009] in the study of biopsy specimens of the small intestine and stomach of patients with giardiasis using electron microscopic, histological, histochemical and quantitative morphometric methods revealed damage to the glycocalyx of the microvilli of prismatic absorptive cells. The range of pathological changes in the small intestine varied from superficial diffuse enteritis to moderate and severe atrophy of the mucous membrane.

Giardiasis with complete atrophy of the villi of the small intestine, clinically manifested by diarrhea, loss of appetite, nausea, spasmodic pain in the epigastric region and weakness, was described by Zakharova I.N., Abdyukina T.I. [2015]. Giardia invasion aggravates the course of a number of infectious diseases [13]. Viral hepatitis occurring against the background of giardiasis are characterized by a longer duration of dyspeptic syndrome and icteric period, more profound changes in the enzymatic systems of the liver [10] .

Giardiasis associated with typhoid fever, paratyphoid fever and salmonellosis worsened the clinical manifestations of the infection and contributed to an increase in the duration of bacterial excretion [14]. Giardiasis has a similar effect on the course of bacterial dysentery and lesions of the hepatobiliary system [13] in the form of gallbladder dyskinesia, acute and chronic cholecystitis, and chronic hepatitis. With giardiasis in combination with helminthic invasion, the clinical manifestations are more pronounced and persistent [10].

Giardiasis often causes malabsorption syndrome. There are different opinions regarding the mechanism of malabsorption in giardiasis. Akhmedov V.A. [2011], Agafonova N.A. [2018] believed that massive giardia invasion creates a mechanical barrier that prevents the absorption of nutrients. Babak O.Ya. [2012] believed that steatorrhea in giardiasis is of pancreatic origin due to the penetration of parasites into the pancreatic ducts.

In the small intestine (and according to some reports in the mouth and stomach), fats undergo hydrolytic cleavage under the influence of lipase, which acts only on emulsified fats. In the mouth and stomach, lipase partially breaks down only milk fats, since they are in the form of an emulsion. Their rapid transport and acid reaction in the stomach destroys the fat emulsion of milk and inactivates salivary lipase. Most of the digestion of fats takes place in the intestines. Under the influence of bile acids, fat is emulsified, due to which its surface increases, which facilitates its hydrolysis by pancreatic lipase. Lipase is secreted by the pancreas in an inactive state and is activated by bile acids.

Under the action of pancreatic lipase, which cleaves fatty acids from triglycerides, di- and monoglycerides, as well as fatty acids and glycerol, are formed in the intestinal cavity. Intestinal lipase, the enterocyte monoglyceride lipase, also takes part in the final hydrolysis of fats [9]. The resulting di- and monoglycerides, glycerides, glycerol, and fatty acids are converted by intestinal juice and bile into micelles and water-soluble complexes (soaps), which are absorbed through the microvilli membranes of the brush border of the small intestine epithelium [12].

It is known that not all fat is completely hydrolyzed in the small intestine. Part of it in the form of di- and monoglycerides, consisting of glycerol and stearic acids, in the form of droplets can be absorbed by the intestinal wall without prior splitting - by pinocytosis. Fat particles during pinocytosis from the intestinal epithelium enter the lymphatic system, and then into the superior vena cava, which explains the milky appearance of blood serum after ingestion of fat. They are converted into chylomicrons, of which 80% are triglycerides, and 8% are phospholides and 4% are cholesterol [11].

Fats in the form of chylomicrons are carried with the blood stream, then their catabolism or, conversely, resynthesis occurs. Fatty acids with a short carbon chain from enterocytes enter the blood of the v.portae system and enter the liver, where fat resynthesis can occur. Fatty acids with a long carbon chain are transported by the lymph, which largely explains the easier digestibility of short-chain fats. These include vegetable fats. Excess fat is deposited in adipose tissue as a reserve energy material. However, the mechanism of fat absorption in the intestine cannot be considered as a diffusion process, since different fats are absorbed at different rates depending on the quantity, quality, and chemical structure of the molecules [15]. At the absorption stage, some fats can affect the resorption of other fats, and the main mechanism of their absorption is active transport through cell membranes [5].

The absorption and digestion of fat are interrelated processes that are carried out with the participation of the same structural elements of the intestine - the "brush border". Zalipaeva T.L. [2002] suggested the existence of a digestive transport conveyor that provides direct transfer of hydrolysis products from a digestive enzyme molecule to a system of transport carriers that are localized on membranes. There is an inverse relationship between transport mechanisms and the activity of digestive enzymes.

Due to the close physiological relationship between digestion and absorption of fat, both of these processes under pathological conditions are often disturbed simultaneously. However, the absorption function of the intestine is often predominantly impaired [10].

Intestinal absorption disorders can be primary - due to congenital, genetically determined enzyme defects and secondary (acquired). More common are secondary disorders of intestinal absorption, which are observed in parasitic diseases [12].

The absorption of most substances occurs mainly in the proximal jejunum. For example, in humans, the fastest absorption of lipids occurs in the lower horizontal branch of the duodenum and in the proximal jejunum [14]. These data are confirmed by histochemical studies of Belousov Yu.V. [2007]

and Gerasimova N.A. [2011], who showed the greatest conversion of fatty acids into triglycerides in the mucous membrane of the jejunum. Cai, J., Wang, R., provide a diagram of the site of absorption of fats and bile salts in the intestine [2015].

Since fats are the most difficult to digest group of nutrients [15], therefore, in various diseases, their absorption first of all suffers and a large amount of fat is excreted in the feces - steatorrhea. In children aged 1 to 18 months, up to 99.26% of the administered amount of lipids is resorbed [13]. According to Azamova Z.Sh. [2011], fat malabsorption can be identified when more than 25% of undigested fat is excreted in the feces.

Along with a violation of the absorption of fats, with giardiasis, there is a violation of the resorption of carbohydrates. So Baldursson, S., Karanis. P. [2011] indicates that with intestinal giardiasis, the absorption of lactose is 46%, lactose and sucrose - 16% and glucose only 6%.

Carbohydrates are the main source that satisfies the energy needs of the body. About half of your calorie needs come from carbohydrates. the main sources of which are starch and glycogen (60%), disaccharides and sucrose (30%) and lactose (10%).

The clinical expression of a violation of the utilization of carbohydrates is loose stools of an acidic reaction, which indicates a pronounced decompensation. However, subcompensated forms can also be observed, which are manifested by flatulence, due to a shift in the topography of carbohydrate utilization in the small intestine. Carbohydrate malabsorption is based on congenital or acquired insufficiency of functional elements that ensure the absorption of di- and monosaccharides in the small intestine (enzymes).

Polysaccharides that enter the body with food are cleaved in the oral cavity under the influence of salivary & - amylase, which cleaves 1,4 - glycosidic bonds in starch and glycogen molecules. But since food does not stay long in the oral cavity, the breakdown of carbohydrates during this period is short and incomplete.

The main breakdown of carbohydrates occurs in the small intestine. & - pancreatic juice amylase completes the breakdown of the starch residue after salivary amylase to di- and monosaccharides. Amylase of saliva and pancreatic juice breaks down starch into maltose, maltotriose and branched & - dextrins, but does not give free glucose. It is considered proven that the main hydrolysis of polysaccharides - starch is carried out by enzymes that are freely located in the intestinal lumen [13-15]. The so-called cavity digestion of carbohydrates can be thought of as the conversion of water-insoluble large molecules of polysaccharides into soluble disaccharides. Here, small molecules are released from the surface of the food bolus under the action of cavity enzymes as the chyme moves through the small intestine [15].

There are data characterizing changes in the activity of intestinal enzymes depending on age. It is shown that lactase has a high degree of activity by the time of birth and during 1 year of life with lactotrophic nutrition, especially breast milk. Then its activity decreases. Along with this, maltase and sucrose activity increase during the first weeks of life from low in newborns to high in adults [12-15]).

Small intestine enzymes play an important role in the digestion and absorption of carbohydrates. Hydrolysis of carbohydrates is carried out both in the intestinal lumen (abdominal digestion) and on the membranes of microvilli of epithelial cells of the intestinal mucosa.

The absorption of carbohydrates takes place almost throughout the entire gastrointestinal tract. However, the place of their most intensive resorption is the duodenum and proximal jejunum.

It has been established that in the absence of reinvasion, the life expectancy of Giardia in humans ranges from 3 to 40 days and averages 4 weeks. However, in recent years, the problem of resistance of the intestinal mucosa during invasion by these protozoa has been widely discussed. It has been shown that with a decrease in local immunity of the intestine, mainly its cellular link, which is typical for children, giardiasis can acquire a protracted course, including chronic. The latter can continue for many months and years without specific therapy.

Thus, the histochemical changes in the microvilli of the intestinal epithelium, established in recent years, in giardiasis determine the expediency of studying the process of absorption of fat and carbohydrates, depending on the level of cystic secretion of protozoa and their possible effect on the severity of the clinical manifestations of the disease.

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