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MODERN ASPECTS OF ETIOLOGY AND EPIDEMIOLOGY OF GIARDIAS

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Abstract
Currently, six species of Giardia are recognized, among which the ones responsible for human disease are L. intestinalis (syn. Giardia duodenalis, G. lamblia, Giardia intestinalis). The introduction of molecular-genetic diagnostic methods has identified eight main genetic subtypes of L. intestinalis (A–H). Human giardiasis is associated with subtypes A and B, which also exhibit intragroup variations (AI–AIII, BIII–B1V) [7]. Giardia that infect humans can also infect other mammalian species, both in the wild and in domestic animals.

Keywords: humans, illness, transmission, tropical and subtropical countries, life cycle.

Introduction

Human infection occurs solely via the oral route through the ingestion of mature infectious cysts (infective dose: 10–100 cysts). The transmission mechanism is fecal-oral. The primary route of transmission for the pathogen is through water. This route is responsible not only for sporadic cases of illness but also for outbreaks. From 2004 to 2010, 70 outbreaks of giardiasis related to waterborne transmission were recorded worldwide [2]. In addition to the waterborne route, foodborne and contact transmission routes are also significant, with the latter being especially important in childcare settings, where giardiasis can spread through toys, dishes, and shared towels, on which cysts can remain viable for 6 hours to 2 days. Giardia is a common cause of "traveler's diarrhea" in tropical and subtropical countries [8].

PATHOGENESIS

The life cycle of Giardia, like that of many other protozoa, includes both a vegetative (trophozoite) and a dormant (cyst) stage. The active reproducing vegetative stage is particularly important, as its structural and physiological characteristics enable these parasites to inhabit the surface of the

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brush border of the intestinal epithelium in humans, which is a critical part of the digestive system. The highest number of trophozoites is found in the upper loops (2.5 meters in length) of the small intestine, where the intensity of contact digestion is greatest. By colonizing the brush border and absorbing hydrolysis products, Giardia affect the process of membrane digestion and can alter the functional state of the epithelial villi. This is manifested by an increase in the number of mitoses in the intestinal epithelial crypts and a decrease in lipid absorption rates. In 50% of cases, malabsorption of D-xylose and cyanocobalamin occurs, along with impaired synthesis of the enzymes invertase and lactase [9]. However, these changes are also detrimental to the parasites themselves, leading to a decrease in their numbers [9]. Mechanical damage to the intestinal mucosa and the destruction of the glycocalyx by Giardia contribute to the activation of opportunistic and pathogenic microbiota, leading to dysbiosis. According to data from S. N. Minina, dysbiotic changes are observed in all children with giardiasis: 73.5% show a decrease in the number of lactobacilli and bifidobacteria, 22.5% have a decrease in only lactobacilli, and 4% in only bifidobacteria [10]. A reduction in the number of Escherichia coli is noted in 67% of cases, with 16% of children having no E. coli with normal properties. In 34% of cases, an increase in various opportunistic bacteria is observed, including 28.5% with E. coli exhibiting altered biochemical properties, 10% with Klebsiella pneumoniae, 16% with Staphylococcus aureus, and 10% with Candida species. The metabolic products and death of Giardia, when absorbed from the intestine, cause sensitization of the human body, which can manifest in various forms of allergic reactions [11]. Patients experience unbearable itching, chronic urticaria, and symptoms of allergic rhinitis, conjunctivitis, blepharitis, and more. According to A. V. Sannikova and colleagues, targeted screening of children with atopic dermatitis revealed giardiasis in one out of every four children [12]. In this cohort, atopic dermatitis had a chronic, torpid, continuously relapsing course and was associated with higher levels of immunoglobulin E. The eradication of the parasites contributed to clinical improvement and sustained remission of the allergic condition, accompanied by a decrease in the severity of eosinophilia and total immunoglobulin E levels in the patients [12]. Diagnosing giardiasis requires a laboratory parasitological examination, which is included in the list of mandatory tests performed by clinical diagnostic laboratories. The material for examination consists of stool samples and duodenal contents. In well-formed stool, only cysts are detected (with no time limit for the analysis), while in semi-formed and liquid stools, vegetative forms and (very rarely) cysts are found (with a time limit of 1–2 hours, provided they are stored at +3–15°C). The vegetative stages of the protozoa can also be identified in the duodenal contents. It is important to note that examining duodenal contents does not provide significant advantages over stool analysis, especially in cases where Giardia are only present in the middle and distal sections of the small intestine. A positive result is obtained when vegetative forms and/or cysts of the protozoa are detected. When using any diagnostic method, the intensity of the invasion should also be determined — that is, the number of parasites in one field of view. A negative result is confirmed only after three separate examinations of the material with intervals of 2–3 days. The current parasitological situation in the Russian Federation is characterized by several features that significantly impact the work of diagnostic laboratories: despite the continued widespread prevalence of intestinal parasitosis, there has been a general decline in the intensity of infections, necessitating highly effective diagnostic methods. Currently, the most accessible, informative, and

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effective method is the examination of stool samples using enrichment techniques with reagents from Barrow, Turdiyev, or Safaraliyev—this is considered the "gold standard" for diagnosis. The sensitivity of this method in three separate examinations ranges from 76% to 90%. In most cases, Giardia cysts are detected during the first examination [17]. In 10–15% of patients, the infection is not diagnosed or is only identified after multiple stool tests, which can be explained by prolonged intervals in cyst shedding (ranging from several days to 2 weeks) and the low number of cysts present [17]. Recently, there has been a trend toward ordering serum blood tests using enzyme-linked immunosorbent assay (ELISA) to detect specific antibodies of classes A, M, and G against Giardia antigens without conducting parasitological examinations to identify the pathogen in stool or duodenal contents. This often leads to the overdiagnosis of giardiasis and the unwarranted prescription of specific treatment. Both the absence of specific immunoglobulin class M and the presence of specific IgG cannot be considered direct evidence of active invasion, as antibodies do not appear immediately and can be detected for several months (up to 1.5 years) after the elimination of the parasites [10]. The development and implementation of immunological diagnostic methods based on the detection of specific Giardia antigens (Giardia-specific antigen 65) in stool samples (enzyme-linked immunosorbent assay, indirect immunofluorescence reaction, immunochromatographic analysis) have improved and, in some cases, facilitated the diagnosis of the infection. The sensitivity of the kits offered by various manufacturers ranges from 95% to 100%, while specificity reaches 100%. Molecular genetic analysis methods demonstrate high specificity (up to 89%) and sensitivity (up to 79%), allowing not only for the detection of Giardia DNA in clinical material but also for the identification of their subtype (A or B).

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