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LIFE CYCLE OF HELMINTH INFECTIONS IN HUMANS

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Abstract
Giardiasis is an intestinal infection primarily affecting the small intestine, caused in humans by the single-celled parasite Giardia intestinalis. There is no international classification for giardiasis; foreign authors focus on different forms of the disease (acute, subacute, chronic) and identify complications associated with giardiasis, including gastrointestinal disorders, extraintestinal manifestations, arthritis, and dermatological and ocular symptoms.

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

Introduction

Helminth infections in humans are a significant issue for medical science and healthcare practice. Giardiasis ranks third in prevalence in the Russian Federation, following enterobiasis and ascariasis. [4] According to WHO experts (1988), giardiasis is defined as:
1) any case of Giardia infection (asymptomatic or with clinical manifestations; diagnostic methods include analysis of stool and duodenal contents)
2) giardiasis with clinical symptoms-diarrhea, abdominal pain, or gastrointestinal discomfort-which resolves after specific treatment (diagnostic methods include analysis of stool and duodenal contents, as well as clinical examination before and after treatment) [1].
Considering the biological nomenclature worldwide, this protozoan is named Giardia intestinalis (also known as Giardia lamblia or Giardia duodenalis). According to WHO experts, approximately 200 million people in Asia, Africa, and Latin America are infected with giardiasis each year [2]. Giardiasis is widespread, Giardia cysts are found in the feces of 20% of the global population [3]. In the Russian Federation, more than 130,000 cases are registered annually, with 70% of these cases occurring in children under 14 years of age [2]. Giardia are among the most primitive representatives of one of the earliest branches of the phylogenetic tree of eukaryotes, characterized

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by anaerobic metabolism and dependence on the utilization of exogenous nucleotides. In the human body, Giardia exist in two forms: the vegetative form (trophozoite) and the cyst form [6]. When cysts enter the body, they easily pass through the acidic gastric barrier and excyst in the duodenal cavity, where they reproduce and colonize the small intestine. In the large intestine, trophozoites develop into cysts. The process of cyst formation from trophozoites takes 12–14 hours, while the formation of trophozoites from cysts (excystation) lasts no more than 10–20 minutes, which facilitates the rapid colonization of the intestine by the protozoa. Cyst excretion from the intestine occurs on days 9–12 post-infection, both continuously (in 4.7% of patients) and intermittently (in 95.3%), with "silent" intervals of 1–17 days [6], which complicates the diagnosis of giardiasis.

The cycle concludes when mature cysts are excreted in feces into the external environment. Giardia reproduce in areas of greatest concentration through simple binary fission. They multiply every 9–12 hours, with carbohydrate-rich food, the presence of yeast-like fungi, and bile in low concentrations promoting this process. The lifespan of Giardia is 30–40 days. The development of clinical manifestations depends on the infective dose and virulence of the pathogen, the functional state of the gastrointestinal tract, and the immune status of the infected individual. Giardia have adapted to parasitize on the brush border of the microvilli in the small intestine [11], where intensive enzymatic breakdown of food substances occurs, and the majority of carbohydrates, proteins, fats, vitamins, mineral salts, and trace elements are absorbed. They obtain nutrients through their central pair of flagella. The result of Giardia's life activities includes impaired epithelial regeneration processes, leading to the destruction of the glycocalyx [5], disruption of the synthesis of certain enzymes (invertase, lactase, amylase, enterases, phosphatases, etc.), multivitamin deficiencies, increased permeability of the intestinal wall, and sensitization of the body to the metabolic products of Giardia. This also triggers pathological viscero-visceral reflexes from the digestive organs due to irritation of nerve endings, contributing to the development of abdominal syndrome, formation of intestinal dysbiosis, and disruption of liver function as a detoxification organ [11]. Some authors believe that these changes are reversible and that after recovery from giardiasis, the absorption process normalizes, as even in areas where Giardia are localized, sections of epithelium with signs of damage alternate with normal sections, despite colonization by the parasites [6].

The colonization of the intestinal mucosa by Giardia is accompanied by the development of an immune response (antibodies, complement, sensitization of immune-competent cells), with secretory antibodies IgA playing a central role in the anti-Giardia defense of the mucosa. The cytotoxic effects of other classes of immunoglobulins (IgG, IgM) on Giardia and their disruption of trophozoite encapsulation processes have also been demonstrated [13]. Possible candidates for protective factors include nitric oxide (the breakdown products of nitric oxide or the oxide itself can damage the vegetative forms of Giardia, inhibiting encapsulation and the excystation of vegetative forms [14]), as well as antimicrobial peptides such as α-defensins from Paneth cells and lactoferrin [14]. In individuals with persistent giardiasis, there is a high frequency of major histocompatibility complex (MHC) antigens HLA B5, B14, DR3, DR4, and DR7 [15]. Haplotypes A9, B5, and A1 are considered markers of predisposition to giardiasis. Non-immune protective factors in humans that damage Giardia include intestinal mucus, bile salts, and digestive enzymes.

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Giardiasis is recognized as one of the markers of immunosuppression. Immunity after a giardiasis infection is non-strong and unstable. To date, strains and isolates of Giardia with varying virulence have been identified, differing in resistance to trypsin and chymotrypsin, and the phenomenon of antigenic variation in Giardia has been observed [11]. Seven genetic groups or assemblages of Giardia intestinalis have been identified, two of which (A and B) are found in both humans and animals [16]. In giardiasis caused by assemblage B, symptomatic disease occurs 2–4 times more frequently [16].

The transmission route for giardiasis is fecal-oral. Transmission factors include water, food, and household items containing cysts. Autoinfection is also possible. The source of giardiasis infection is an infected human or animal, with up to 20 million infectious cysts present in 1 gram of feces. Infection can occur with the ingestion of just 10–100 cysts. In the environment, Giardia cysts remain viable for up to 120 days, while boiling, freezing (down to -13°C or lower), and ultraviolet radiation can kill them [5]. Disinfectants are effective against Giardia only at concentrations 5–10 times higher than those typically used [1,8]. The definitive laboratory diagnostic method for giardiasis is the detection of trophozoites in duodenal contents and/or cysts in feces. In routine practice, it is preferable to examine stool for the detection of Giardia cysts ex tempore or no later than 2–3 hours after defecation, or using some special preservative (Safaraliyev, Turdiyev, Barrow) [6, 8]. If giardiasis is suspected, stool tests should be conducted 3–4 times with intervals of 3–4 days after an initial negative result [8]. The effectiveness of stool testing is around 50% due to the intermittent shedding of cysts. The likelihood of detecting Giardia in duodenal contents is higher than in stool samples, as it is not dependent on the frequency of cyst shedding [8]. Giardia are more frequently detected in portion A.

Serological diagnostic methods are indirect and supplementary for diagnosing giardiasis. The methods for detecting Giardia antigens in feces and biopsies have significantly higher diagnostic potential, especially when using antibodies against whole trophozoites or monospecific antibodies targeting Giardia antigens with a molecular weight of 65 kDa (GSA-65).

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