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MORPHOFUNCTIONAL CHANGES OF THE SMALL INTESTINE IN ASCARIASIS

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Abstract

Ascariasis, caused by the parasitization of the nematode Ascaris lumbricoides, is a widespread helminthiasis significantly affecting the morphofunctional state of the small intestine. This article examines the key morphological and functional changes in the small intestine associated with this disease, including inflammatory processes, structural damage to the mucosa, impaired barrier function, and altered digestion and absorption processes. The analysis aims to enhance understanding of the disease's pathogenesis and explore potential approaches to treatment and prevention.

Keywords: Ascariasis, Ascaris lumbricoides, small intestine, morphological changes, inflammation, absorption disorders, helminthiasis, parasitic infections, mucosa, pathogenesis.

Introduction

According to official data from the World Health Organization (WHO), every fifth person on the planet suffers from a parasitic disease, and every third person has had a parasitic disease at some point. By the end of 2019, WHO's official statistics stated that annually 5 million people contract parasitic diseases, and more than 10 million people suffer from various forms of parasitic infections (1).

Considering the polymorphic clinical syndromes associated with parasitosis, WHO experts included most nematodoses in special World Bank Development Programs for combating parasites by 2020 (2).

In recent years, many countries have reported a high frequency of fatal cases from parasitic diseases, often accompanied by the development of severe complications (3).

The pathological impact of helminths on a child's health is multifaceted. Among the mechanisms of helminth impact on the child’s body, significant roles are played by mechanical factors, competition for nutrients with the host organism, and allergization. Helminths cause mechanical damage to the intestinal wall, particularly the mucosal layer, facilitate the entry of infections into the body, and contribute to the development of inflammatory processes. They can induce general intoxication, anemia, hypovitaminosis, digestive dysfunctions, and sensitization of the body, which subsequently leads to the formation of allergic reactions (4).

The clinical picture of the initial phase of many helminth infections is determined by the sensitization of the body, with various local and systemic allergic reactions developing. An incomplete list of pathological effects of parasites on the body includes signs of chronic intoxication, immune dysfunctions, allergization, dyspeptic disorders, pulmonary syndrome, lymphadenopathy, myalgia, iron-deficiency anemia, carcinogenesis, obstruction of bile ducts and

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pancreatic ducts, focal lesions in the liver and pancreas, intestinal obstruction, appendicitis, intestinal perforation, peritonitis, and more (5).

The pathogenesis of ascariasis varies during the migration of larvae in the bloodstream and their presence in respiratory organs, as well as during the parasitism of adult helminths in the human small intestine. From invasive Ascaris eggs in the human small intestine, rhabditiform larvae emerge and penetrate the mucosal layer within 3–4 hours (6).

In cases of intensive invasion, mechanical damage is observed in the walls of the small intestine, blood vessels, liver tissues, and lungs. Clinical manifestations in the late duodenal-intestinal stage are associated with the mechanical impact of helminths and their metabolic products on the intestinal mucosa, leading to digestive dysfunction, motor dysfunction, disruption of nitrogen balance, and hypovitaminosis (7).

Ascaris can migrate beyond the small intestine into bile and pancreatic ducts, the appendix, and respiratory tracts. Occasionally, Ascaris clusters result in blockages, volvulus, or intussusception. Intestinal obstruction is more common with intense invasion, while intussusception occurs with isolated helminths or several individuals of the same sex. Ascaris significantly suppresses the host's immunological reactivity (8).

During the intestinal parasitism of adult Ascaris, the organism's sensitization continues. In the pathogenesis of the intestinal phase, a key role is played by intoxication caused by the toxic products of Ascaris metabolism, resulting in disruptions in the digestive, nervous, reproductive, and other systems. Helminths exert mechanical effects on the intestinal mucosa, causing changes such as impaired parietal digestion, difficulty in absorption and assimilation of proteins, fats, and vitamins, reduced activity of the enzyme lactase, and more (Sagadeyeva Z.I., 2020).

Objective of the Study: To investigate the morphofunctional changes in the mucous membrane of the small intestine under the influence of ascariasis.

Materials and Methods: The study utilized clinical observations, experiments on animals, and histological analysis of small intestine tissues. Microscopy, immunohistochemistry, and biochemical methods were applied to assess structural and functional changes.

Morphological Studies: Histological examinations were conducted on small intestine samples fixed in 10% formalin solution. Tissue sections (5-7 μm thick) were stained with hematoxylin and eosin, along with other methods, such as the PAS reaction for mucoprotein interaction and toluidine blue staining to measure mast cells. Examination of microvilli was carried out using electron microscopy (scanning and transmission).

Functional Studies:

- Enzyme activity of intestinal epithelium (alkaline phosphatase, sucrase, maltase, and amylase) was assessed.
- Absorptive function was evaluated using a glucose tolerance test.
- Levels of inflammatory cytokines (IL-1β, TNF-α, IL-6) in blood serum were determined by ELISA.

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Results of the Study:

Morphological Changes: Animals with ascariasis exhibited structural damage to the mucous membrane of the small intestine, including villous destruction, crypt atrophy, and inflammatory infiltration. An increase in goblet cells indicated mucus hypersecretion as a protective mechanism.

Inflammatory Processes: The parasite triggered an inflammatory response, evidenced by an increased number of lymphocytes, macrophages, and eosinophils. The inflammation was associated with cytokine release, including interleukin-4 and interleukin-13, which enhanced allergic reactions.

Functional Disorders: Disruption of the epithelial barrier function allowed antigens and toxins to penetrate the bloodstream. Reduced enzyme activity (maltase, sucrase) led to impaired digestion and nutrient absorption.

Systemic Consequences: The invasion was accompanied by hypoproteinemia, anemia, and vitamin deficiencies due to malabsorption of nutrients.

Discussion:

The findings demonstrate that ascariasis exerts complex effects on the small intestine. Destruction of the mucous membrane and inflammatory reactions play a key role in the development of pathological changes. It is essential to highlight that timely treatment, including anthelmintic drugs and supportive therapy, can minimize damage and restore intestinal function.

Impairments in Digestion and Absorption:

- Altered enzymatic activity of intestinal juice due to enterocyte damage.
- Reduced absorption of nutrients (proteins, fats, vitamins).

Changes in Small Intestine Motility:

- Increased peristalsis in response to parasitic irritation.
- Risk of developing malabsorption syndrome.

Immune Reactions:

- Effects of ascaris on the immune barrier of the small intestine (IgA secretion).
- Immunopathological responses leading to tissue damage.

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