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MODERN ASSESSMENT OF THE EFFECT OF INTESTINAL PROTOZOAL INFECTIONS ON GUT MICROBIOTA COMPOSITION AND LOCAL IMMUNITY

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Abstract. Intestinal protozoal infections remain an important cause of gastrointestinal morbidity, particularly in children and populations exposed to unsafe water, inadequate sanitation, and repeated enteric infections. Beyond direct epithelial injury, protozoa such as Giardia duodenalis, Cryptosporidium spp., Entamoeba histolytica and Blastocystis spp. interact dynamically with the resident gut microbiota and mucosal immune system. These interactions may alter microbial diversity, bacterial biofilm structure, epithelial barrier integrity, secretory immunoglobulin A production, cytokine signaling, and the balance between inflammatory and regulatory immune responses. Modern assessment therefore requires a combined approach that integrates parasitological testing with molecular microbiome analysis and markers of local immunity. The present thesis summarizes current concepts on protozoa-microbiota-immune interactions and outlines contemporary diagnostic approaches relevant to clinical and research practice.

Keywords: intestinal protozoa, Giardia duodenalis, gut microbiota, dysbiosis, mucosal immunity, secretory IgA, cytokines, molecular diagnostics.

INTRODUCTION

The intestinal ecosystem is formed by epithelial cells, mucus, immune cells, microbial communities and transient or persistent eukaryotic organisms. Intestinal protozoa can disturb this ecosystem not only through mechanical and metabolic effects but also by modifying bacterial communities and host immune signaling. In children, recurrent protozoal infection may contribute to diarrhea, malabsorption, micronutrient deficiency and impaired growth, while asymptomatic carriage can still be associated with measurable changes in the intestinal environment [1-3]. Recent evidence shows that the clinical expression of giardiasis and other protozoal infections partly depends on the composition of the microbiota and the quality of the mucosal immune response [6,7].

MAIN PART

Protozoa and gut microbiota interact bidirectionally. Giardia may reduce the stability of commensal bacterial biofilms, alter relative abundances of bacterial taxa and promote functional dysbiosis; conversely, the resident microbiota can influence parasite colonization, persistence and clearance [6]. Similar cross-talk is increasingly recognized for Cryptosporidium and other intestinal protozoa. Uzbek studies emphasize the continued relevance of intestinal parasitoses in children and the need for complex diagnostic algorithms, particularly when diarrhea, nutritional disturbances or mixed infections are present [1-3]. Contemporary local literature also highlights the relationship between parasitic disease and changes in the intestinal microbiome and the diagnostic importance of evaluating dysbiosis [4,5].

Local immunity is centered on the epithelial barrier, mucus layer, antimicrobial peptides, secretory IgA, innate lymphoid cells, macrophages, dendritic cells and T-cell responses. Protozoal antigens and epithelial damage activate pattern-recognition pathways and cytokine production. Depending on the

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parasite and stage of infection, IL-6, IL-8, IL-17, IFN-gamma and other mediators may increase, while regulatory mechanisms involving IL-10 and T-regulatory cells may limit tissue injury. Secretory IgA can interfere with trophozoite attachment and contributes to parasite control. At the same time, microbiota-derived metabolites, including short-chain fatty acids, support epithelial integrity and immune tolerance. Dysbiosis may therefore weaken local resistance and prolong post-infectious symptoms [6,7].

Table 1. Contemporary assessment of protozoa-microbiota-local immunity interactions

Assessment target	Preferred methods	Expected finding	Clinical value
Protozoal infection	Microscopy, antigen assay, multiplex PCR	Detection and species identification	Confirms infection and mixed invasion
Microbiota composition	16S rRNA sequencing, metagenomics, targeted qPCR	Changes in diversity and bacterial taxa	Characterizes dysbiosis and recovery
Barrier function	Fecal calprotectin; permeability markers where available	Evidence of mucosal inflammation or barrier injury	Supports severity assessment
Local immunity	Fecal/secretory IgA, cytokine profiling	Altered sIgA and inflammatory/regulatory mediators	Evaluates mucosal immune response
Integrated follow-up	Repeat parasitology plus clinical and microbiota assessment	Parasite clearance with ecosystem normalization	Useful in persistent or post-infectious symptoms

A modern diagnostic strategy should therefore combine classical parasitology with molecular techniques. Microscopy remains useful but may have limited sensitivity when cyst excretion is intermittent. Antigen assays and PCR improve detection and species differentiation. For microbiota assessment, 16S rRNA sequencing is widely used to characterize bacterial community structure, while shotgun metagenomics provides higher functional resolution. Local immune activity can be evaluated by secretory IgA, fecal inflammatory markers and selected cytokines in research settings. Interpretation should be age-specific and should account for diet, antibiotics, probiotics, geography and coinfections, all of which can independently modify microbiota composition [4,6,7].

CONCLUSION

Intestinal protozoal infections should be regarded as ecosystem-level disorders involving the parasite, microbiota, epithelial barrier and mucosal immune system. Their effects range from transient microbial shifts to persistent dysbiosis and altered local immune reactivity. The most informative contemporary assessment combines sensitive parasite detection with microbiota profiling and mucosal immune markers. Wider use of integrated approaches may improve diagnosis, clarify mechanisms of persistent gastrointestinal symptoms and support individualized prevention and rehabilitation strategies, especially in children with recurrent or mixed intestinal infections.

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