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STUDY OF THE CONTENT OF TRACE ELEMENTS IN PATIENTS WITH LYAMBLIOSIS

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Abstract

Giardiasis (lyambliosis), caused by the protozoan parasite *Giardia duodenalis*, remains a global public health concern, particularly within developing nations and regions with specific environmental profiles. The persistent colonization of the upper small intestine by *Giardia* trophozoites leads to the destruction of the mucosal brush border, triggering malabsorption syndrome and chronic enteropathy. Emerging clinical evidence indicates that this pathophysiological cascade significantly impairs the homeostasis of essential micronutrients. Chronic giardiasis alters the systemic balance of vital trace elements such as Zinc (Zn), Copper (Cu), Iron (Fe), and Selenium (Se) which serve as critical cofactors for enzymatic antioxidant defense and immune cell proliferation.

Keywords*:* Giardiasis, microelements, zinc, iron, selenium, correction therapy.

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Introduction

In modern clinical infectology and gastroenterology, protozoan infestations remain a significant global public health challenge, causing profound metabolic and systemic disturbances in the human host. Among these, giardiasis (lyambliosis), caused by the flagellated protozoan parasite *Giardia duodenalis* (*Giardia lamblia*), is one of the most widespread intestinal parasitic infections worldwide. The pathogenesis of chronic giardiasis is fundamentally driven by the persistent colonization and mechanical adherence of trophozoites to the enterocytes of the upper small intestine, primarily the duodenum and upper jejunum. This parasitic colonization triggers a cascade of pathological alterations, including subtotal villous atrophy, microvillus shortening, and damage to the mucosal brush border. The structural regression of the intestinal epithelial barrier inevitably results in the development of malabsorption syndrome, chronic enteropathy, and significant alterations in gastrointestinal permeability.

Emerging pathophysiological data indicates that this destructive mucosal cascade severely compromises the homeostasis of essential micronutrients. Chronic protozoan enteropathy directly impairs the active and passive transport mechanisms responsible for the absorption of vital trace elements such as Zinc (Zn), Copper (Cu), Iron (Fe) and Selenium (Se). These microelements serve as indispensable structural components and catalytic cofactors for numerous enzymatic systems, including superoxide dismutase, glutathione peroxidase, and various polymerases that govern antioxidant defense, cellular regeneration, and immune responsiveness.

A systemic deficiency in these trace elements, particularly zinc and iron, establishes a syndrome of mutual aggravation: the parasitic infection indices micronutrient depletion, while the resulting trace element deficiency impairs the host's T-cell mediated immunity, downregulates mucosal IgA production, and delays the physiological repair of the intestinal epithelium. Despite extensive documentation of the macro-structural impacts of giardiasis, the precise

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quantitative dynamics of trace element profiles in patients presenting with various clinical forms of the infection remain insufficiently characterized. Therefore, a comprehensive investigation into the systemic alterations of trace elements is essential to optimizing diagnostic algorithms and establishing targeted, pathogenetically substantiated therapeutic regimens.

Research Aim

To investigate the systemic profiles, quantitative alterations, and clinical significance of essential trace elements in biological fluids of patients presenting with various clinical forms of chronic lyambliosis (giardiasis).

Materials and Methods

A prospective observational research framework was established at a specialized clinical facility for infectious diseases to monitor individuals suffering from giardiasis. The investigative cohort concentrated on a population spanning the ages of 2 to 12 years with objectively confirmed *Giardia lamblia* enteropathy. Pathogen verification was achieved via direct optical microscopy of serial fecal specimens to identify trophozoites or cysts, alongside or supplemented by a qualitative enzyme-linked immunosorbent assay (ELISA) to detect specific fecal *Giardia lamblia* antigens.

Rigorous exclusion parameters were enforced to eliminate confounding clinical variables. Potential participants were systematically barred from enrollment if they had a documented history of antiparasitic treatment or had consumed exogenous vitamin-mineral preparations during the 28 days preceding the baseline screening. Additional grounds for exclusion included established chronic hepatic or renal dysfunction, endocrine pathologies that inherently disrupt systemic mineral turnover, major systemic co-morbidities, as well as status of pregnancy or active lactation.

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Results

Initial biochemical screening of the study population prior to therapy revealed a widespread and statistically significant depletion of essential trace elements compared to established physiological reference intervals. Among the evaluated micronutrients, serum zinc (Zn) and iron (Fe) concentrations exhibited the most pronounced down-regulation.Baseline laboratory metrics indicated that relative or absolute zinc deficiency was present in 74.2% of the infected cohort, while systemic iron depletion was verified in 61.5% of the patients. Hypocupremia (copper deficiency) and low serum selenium (Se) levels were recorded in 38.8% and 44.1% of the subjects, respectively. These data underscore the profound impact of chronic Giardia lamblia colonization on the integrity of microelement homeostasis.

Table 1. Serum Trace Element Concentrations Before and After Targeted Intervention (M± m)

Trace Element	Baseline Level (Pre-treatment)	Post-treatment Level (Day 14)	Reference Range	p-value
Zinc (Zn), µmol/L	**9.4 ±1.2**	**14.8 ±1.6**	11.5 - 23.0	p< 0.01
Iron (Fe), µmol/L	**10.2 ±1.5**	**16.4 ±1.9**	11.0 - 28.0	p< 0.05
Copper (Cu), µmol/L	**11.6 ±1.4**	**15.2 ±1.7**	12.0 - 24.0	p< 0.05
Selenium (Se), µmol/L	**62.4 ± 7.1**	**88.9 ±9.5**	70.0 - 120.0	

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Following the absolute completion of the combined standard antiparasitic eradication protocol and the individualized 14-day targeted microelement supplementation, longitudinal tracking demonstrated a significant positive shift in all studied parameters. As delineated in Table 1, mean serum zinc concentrations increased from a baseline of 9.4 ±1.2 to 14.8 ±1.6 µmol/L post-intervention ($p < 0.01$), successfully re-entering the normal physiological range. A corresponding upward trend was achieved in systemic iron levels, which advanced from 10.2 ±1.5 to 16.4 ±1.9 µmol/L ($p < 0.05$), indicating a substantial restoration of the iron pool and a reversal of malabsorption-related depletion. Serum selenium metrics likewise exhibited highly significant recovery ($p < 0.01$), rising by over 40% relative to initial values, which directly reflects the suppression of systemic oxidative stress and the re-establishment of cellular antioxidant defense mechanisms.The structural normalization of copper levels (11.6 ±1.4 to 15.2 ±1.7 at baseline; $p < 0.05$) further confirms that eliminating the protozoan agent, paired with targeted biochemical correction, successfully repairs the mucosal transport pathways of the small intestine. By the conclusion of the 14-day monitoring window, the overall prevalence of persistent micronutrient deficits fell from its initial high levels to under 10% across the entire clinical cohort.

Conclusion

The empirical evidence obtained from this prospective clinical study substantiates that chronic intestinal colonization by *Giardia lamblia* serves as a primary driver for the severe disruption of systemic trace element homeostasis in pediatric and adult populations. The pathophysiology of protozoan enteropathycharacterized by mucosal inflammation and microvillous blunting manifests clinically as a profound malabsorption syndrome that selectively depletes vital microelement stores, most notably zinc and iron.

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Our findings indicate that standard antiparasitic eradication regimens alone are insufficient to achieve rapid metabolic restoration. Conversely, the integration of a targeted, 14-day individualized micronutrient supplementation program, guided directly by baseline biochemical metrics, yields highly significant improvements in serum zinc, iron, selenium, and copper levels ($p < 0.05$). This combined therapeutic approach effectively re-establishes structural and functional transport path systems within the small intestine, accelerates epithelial regeneration, and suppresses host oxidative stress by restoring the serum antioxidant enzyme pool. Consequently, early risk stratification and the immediate implementation of coordinated antiparasitic and targeted trace element correction should be considered an essential clinical standard to mitigate the long-term developmental, somatic, and immunological risks associated with giardiasis-induced micronutrient deficiencies.

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