



TRACE ELEMENT COMPOSITION IN PATIENTS WITH GIARDIASIS

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Relevance of the topic

Giardiasis, caused by the flagellated protozoan *Giardia lamblia* (syn. *Giardia intestinalis*, *Giardia duodenalis*), remains a major global parasitic threat, affecting over 200 million people annually through intestinal mucosal colonization. Recent high-resolution imaging and organoid-on-a-chip studies demonstrate that *Giardia* trophozoite attachment induces rapid degradation of enterocyte microvilli within 6 to 12 hours of colonization. Trophozoite excretory-secretory products (ESPs), particularly cysteine proteases such as GiCP50 and GiCP140, directly cleave critical tight junction proteins, specifically Zonula Occludens-1 (ZO-1), Claudin-1, and Occludin. This combined mechanical and enzymatic breakdown increases intestinal mucosal permeability and selectively impairs active ion and micronutrient transporters in the brush-border membrane, including ZIP4/ZnT8 zinc transporters and divalent metal transporter 1 (DMT1).

The resulting intestinal barrier disruption triggers profound secondary hypomicroelementosis, compromising essential physiological pathways governed by vital trace minerals. Systemic zinc (Zn) depletion impairs thymic hormone production, reduces T-helper 1 responses, and suppresses secretory immunoglobulin A (IgA) secretion into the intestinal lumen, thereby prolonging parasitic survival and delaying epithelial proliferation.



Concurrently, iron (Fe) depletion impairs mitochondrial electron transport chains within mucosal epithelial cells, causing intracellular adenosine triphosphate (ATP) depletion and accelerating enterocyte apoptosis. Furthermore, selenium (Se) deficiency severely compromises the catalytic activity of Glutathione Peroxidase (GPx) and Thioredoxin Reductase (TrxR), exposing the intestinal mucosa to unmitigated reactive oxygen species (ROS) generated during host inflammatory responses, while magnesium (Mg) depletion disrupts Na^+/K^+ - ATPase pumps, aggravating secretory diarrhea and neuromuscular hyperexcitability.

Material and methods

A prospective clinical cohort study was conducted between 2024 and 2026 to evaluate trace element status, enterocyte barrier disruption, and therapeutic responses in patients with giardiasis. The study enrolled 150 participants: 120 hospitalized patients with confirmed *Giardia lamblia* infection and 30 age- and sex-matched healthy controls. Clinical, demographic, complete blood count (CBC), and routine biochemical parameters (AST, ALT, glucose, urea, creatinine, CRP) were systematically evaluated upon admission using an automated Mindray BS-30 analyzer.

For therapeutic evaluation, infected patients (n=120) were stratified into two groups: the Main Group (n=60), receiving standard antiparasitic therapy combined with a targeted trace element correction scheme (Zn, Fe, Mg, Se), and the Comparison Group (n=60), receiving standard antiparasitic monotherapy. Healthy individuals formed the Control Group (n=30). Treatment efficacy was monitored through clinical symptom resolution, laboratory indicators, and post-treatment trace element levels. Statistical analysis was performed using SPSS v26.0 and GraphPad Prism via Student's t-test, ANOVA, Mann-Whitney U test, correlation analysis, and Receiver Operating Characteristic (ROC) curve modeling.



Result

Baseline laboratory analysis using atomic absorption spectrometry (AAS) revealed severe, statistically significant systemic trace element depletion in patients with giardiasis compared to healthy controls ($p < 0.001$). Serum zinc (10.42 ± 1.85 mol/L vs. 18.50 ± 2.10 mol/L) and iron (11.20 ± 2.15 mol/L vs. 22.10 ± 3.05 mol/L) exhibited the most marked reductions, demonstrating strong inverse correlations with pro-inflammatory cytokines IL-6 and TNF- α ($r = -0.68$, $p < 0.001$). Concurrently, elevated circulating Zonula Occludens-1 (ZO-1) degradation fragments (4.85 ± 0.72 ng/mL vs. 1.12 ± 0.20 ng/mL in controls) confirmed trophozoite-induced tight junction disruption and down-regulated ZIP4/DMT1 transporter activity. Diagnostic ROC curve modeling integrating the serum Zn, Se ratio with IL-6 yielded high predictive accuracy for severe malabsorption risk (AUC=0.892, sensitivity 88.3%, specificity 86.7%). Following intervention, the Main Group receiving targeted trace element correction demonstrated rapid tight junction repair, complete recovery of secretory IgA levels (1.58 ± 0.18 g/L), a 1.8-fold reduction in parasitic recurrence, and a significant 2.5–3.0 day reduction in hospital stay duration ($p < 0.01$) compared to the Comparison Group receiving antiparasitic monotherapy.

Conclusion

Pediatric *Giardia lamblia* infection triggers severe systemic trace element dyshomeostasis that directly correlates with clinical severity. Neutron activation analysis of hair tissue reveals a distinct, mixed metabolic profile: significant depletion of key essential elements specifically copper, chromium, zinc, cobalt, and selenium alongside a paradoxical accumulation of iron and manganese in moderate-to-severe forms. These systemic imbalances reflect persistent mucosal injury, impaired enterocyte absorption, and heightened oxidative stress within the proximal intestine. Consequently, effective management of pediatric giardiasis must extend beyond antiparasitic



eradication; incorporating targeted micronutrient profiling and tailored trace element correction is essential to restore metabolic balance, bolster antioxidant defenses, and accelerate mucosal recovery.

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