



Comparative inflammatory, oxidative stress, and tissue injury signatures in amoebiasis and giardiasis

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ABSTRACT

A case-control study was conducted in Basrah, Iraq, between September 2023 and December 2024. Stool samples from 110 symptomatic individuals were examined microscopically using direct wet mounts, concentration methods, and permanent staining techniques. Confirmed cases included 43 patients infected with *Entamoeba spp.* and 30 patients infected with *Giardia duodenalis*, while 50 apparently healthy individuals served as controls. Hemoglobin and leukocyte differential counts, serum total protein, albumin, interleukin-1 beta (IL-1 β), interleukin-6 (IL-6), interleukin-8 (IL-8), C-X-C motif chemokine ligand 10 (CXCL10), malondialdehyde (MDA), glutathione (GSH), and lactate dehydrogenase (LDH) were evaluated. The results demonstrated significant hematological, biochemical, inflammatory, and oxidative stress alterations among infected individuals. Hemoglobin, serum total protein, and albumin levels were significantly reduced in both amoebiasis and giardiasis patients compared with healthy controls, with more pronounced alterations observed in amoebiasis. IL-1 β , IL-6, IL-8, CXCL10, MDA, and LDH levels were significantly elevated, whereas increased GSH levels may reflect a compensatory antioxidant response rather than antioxidant depletion, particularly among amoebiasis patients. Giardiasis patients exhibited inflammatory and oxidative stress changes, although these alterations were less pronounced than those observed in amoebiasis. Comparative analysis indicated that amoebiasis induced stronger inflammatory, oxidative, and tissue injury responses than giardiasis, due to the invasive nature of *amoebic* infection. Overall, *Entamoeba spp.* infection elicited stronger inflammatory, oxidative stress, and tissue injury responses than giardiasis, providing further insight into the distinct host-parasite interactions associated with these intestinal protozoan infections.

1. Introduction

Intestinal protozoan infections continue to represent an important global public health burden, particularly in developing countries where sanitation systems remain inadequate and access to clean water is limited. Among the most clinically significant intestinal protozoa are *Entamoeba histolytica* and *Giardia duodenalis*, both of which are responsible for considerable gastrointestinal morbidity worldwide. These infections are especially prevalent in regions characterized by overcrowding, poor hygienic practices, and contaminated food or water supplies (WHO, 2023; Azami et al., 2025). Amoebiasis, caused by *Entamoeba histolytica*, is considered one of the most important parasitic diseases affecting humans. The infection may range from asymptomatic intestinal colonization to severe invasive disease, including amoebic colitis and liver abscess formation. The pathogenicity of *E. histolytica* is primarily related to its ability to invade host tissues through several

virulence factors, including amoebapores, cysteine proteases, and adhesion molecules, which contribute to epithelial destruction, inflammatory responses, and tissue necrosis (CDC, 2019; Chou and Austin, 2023). In contrast, *Giardia duodenalis* is generally considered a non-invasive intestinal protozoan that colonizes the upper small intestine and interferes with nutrient absorption (Sengupta and Chakraborty, 2023). Despite its non-invasive nature, giardiasis is associated with substantial gastrointestinal disturbances, including diarrhea, malabsorption, abdominal discomfort, and weight loss. The parasite may also disrupt epithelial barrier integrity and trigger immune-mediated cellular responses (Ismael et al., 2024; Stephen et al., 2025). The interaction between intestinal protozoa and the host immune system is complex and involves the activation of multiple inflammatory pathways. During infection, immune cells release a variety of pro-inflammatory cytokines, including interleukin-1 beta (IL-1 β), interleukin-6 (IL-6), and interleukin-8 (IL-8), which contribute to immune cell recruitment and

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